Predicting smoking cessation and health risk perceptions:

Exploring the utility of the Liverpool Lung Project risk model



Thesis submitted in accordance with the requirements of the University of Liverpool for the degree of Doctor in Philosophy by Frances Caroline Sherratt

DECLARATION

I certify that all material in this thesis, which is not my own work has been identified.
I further declare that this thesis has not already been accepted in substance for any
degree, and is not concurrently submitted in candidature for any other degree.

Signed	 	
Date	 	
Name of candidate	 	

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ABSTRACT

The Liverpool Lung Project (LLP) risk model predicts an individual's five-year absolute lung cancer risk. Smoking cessation has been identified as the most effective strategy for reducing lung cancer incidence, whilst tailored communications have been considered to be one of the most promising approaches to smoking cessation. The primary aim of this PhD project was therefore to examine whether the LLP intervention was associated with smoking cessation success and lung cancer risk perceptions among Stop Smoking Service (SSS) users. The LLP intervention was developed using the LLP risk model and involves calculation and communication of projected lung cancer risk, based on both smoking and non-smoking behaviour. A number of secondary aims pertaining to risk perceptions and smoking cessation are also considered and described in the main body of the thesis.

The project adopted a mixed methods approach, integrating both quantitative and qualitative research components. In relation to the quantitative component, two randomised controlled trials were employed to evaluate the LLP intervention effect on smoking cessation success and lung cancer risk perceptions; baseline current smokers (n = 302) and baseline recent former smokers (n = 219) were recruited from a SSS in Liverpool, UK. All participants completed a baseline questionnaire, which considered socio-demographics, smoking behaviour, and lung cancer risk perceptions. Two separate single-blinded randomised controlled designs were implemented for baseline current and recent former smokers, whereby participants allocated to the intervention arm received the LLP intervention (based upon the LLP risk model). Follow-up smoking status and lung cancer risk perceptions were established at six months. Bivariate and multivariate analyses were undertaken to explore the strength of any

associations. Qualitative interviews were additionally undertaken with a sub-set of participants derived from the quantitative research component (n = 30). These interviews intended to explore factors implicated in smoking cessation success and smoking-related risk perception, thus complementing the additional findings of the quantitative research component. Interviews were transcribed and analysed using thematic analysis techniques.

The analysis of baseline current smokers revealed that the LLP intervention failed to predict follow-up smoking status or lung cancer risk perceptions; however, the LLP intervention was found to predict follow-up smoking status among baseline former smokers (OR 1.91, 95% CI 1.03-3.55), but not lung cancer risk perceptions. This suggests that those who received the intervention were more likely to be classified as former smokers at follow-up. The qualitative results also provided insight regarding smoking-related risk perception and communication; issues such as perceived lack of control, risk contextualisation, and poor health literacy, were identified to be relevant components to smoking-related risk perception and communication.

The results suggest that the intervention may predict follow-up smoking status among recent former smokers, although the trials entailed insufficient statistical power and therefore, an extension of recruitment or implementation of a larger trial is now required to build upon the results. Nevertheless, the current results contribute towards tobacco control research, practice and policy in various ways. For example, the delivery of the LLP intervention among recent former smokers in SSS, and potentially other healthcare settings, could improve smoking cessation rates and would require

little time and financial resources. In turn, improved smoking cessation success rates would lead to reduced smoking-related disease and associated deaths.

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Chapter 1: Lung cancer risk factors and modelling

1.1 Introduction

Cancer is characterised by a "continuing, purposeless, unwanted, uncontrolled and damaging growth of cells that differ structurally and functionally from the normal cells from which they develop" (Stephens & Aigner, 2009, p. 3). Cancer presents itself in many forms; however, lung cancer is the most commonly diagnosed cancer worldwide. It was estimated that in 2012, there were 1.82 million new cases of lung cancer worldwide and 1.6 million lung cancer deaths (Ferlay et al., 2015). Lung cancer is not considered to be a single disease and rather, is typically accepted as being composed of several diseases, which are pathologically divided into two categories: (1) small cell lung cancer (SCLC); (2) non-small cell lung cancer (NSCLC). NSCLC represents more than 85% of all lung cancers and includes adenocarcinoma, squamous cell carcinoma and large cell carcinoma (National Comprehensive Cancer Network, 2015).

For NSCLC, surgery, radiotherapy, and chemotherapy are the most common treatment modalities, although targeted biological agents are becoming increasingly important (Baldwin, 2016). Comparatively, SCLC is a particularly aggressive malignancy; guidance recommends that localised SCLC should be treated with chemotherapy and radiotherapy, whilst chemotherapy is often considered to be a first-line treatment option for extensive SCLC (Heist, 2015). Unfortunately, most localised lung cancers cause no symptoms, which subsequently results in diagnosis at an advanced stage and sadly, such advanced cases have usually developed beyond curative treatment (Diederich et al., 2002; UyBico et al., 2010). Treatment options continue to evolve (Méry et al., 2015; Scott, Howington, Feigenberg, Movsas, & Pisters, 2007; White &

Pavlakis, 2008) but age-standardised five year survival for patients diagnosed with lung cancer during 2010-2011 in England and Wales, remains substantially low, at only 9.5% (Cancer Research UK [CRUK], 2014c).

The poor prognosis associated with lung cancer has stimulated considerable effort directed towards lung cancer prevention. The most thoroughly documented causal relationship in the history of biomedical research is the positive correlation between tobacco smoking and lung cancer development (Alberg & Samet, 2003). Within the UK, it has been estimated that 86% of lung cancer cases are attributed to tobacco smoking (Parkin, 2011b). In addition to lung cancer, smoking has also been identified as a substantial contributor to a large range of other diseases, including cardiovascular disease (Kenfield, Stampfer, Rosner, & Colditz, 2008; Preston, Glei, & Wilmoth, 2010), chronic obstructive pulmonary disease (COPD) (Forey, Thornton, & Lee, 2011), peptic ulcers (Zhang et al., 2012), and at least a dozen other cancers (Baan et al., 2009). Overall, smoking-related ill health costs the National Health Service (NHS) an estimated £5.2 billion per year; approximately 5.5% of the total NHS budget (Allender, Balakrishnan, Scarborough, Webster, & Rayner, 2009).

Smoking rates among men peaked at 82% in 1948 and among women, 45% in the mid-1960s; however, the health implications of smoking became more widely acknowledged within the UK between the mid-1970s and mid-1990s, resulting in a rapid decline in smoking rates (Action on Smoking and Health [ASH], 2015). Trends in lung cancer incidence rates reflect the trends in smoking prevalence in recent decades (see Figure 1). Consequently, smoking cessation has been highlighted as the

single most effective strategy to reduce lung cancer risk among the 1.3 billion smokers worldwide (Thun, DeLancey, Center, Jemal, & Ward, 2010).

Although smoking cessation has been identified as a key strategy in reducing lung cancer incidence, tobacco control remains a continuous challenge. Smoking cessation success rates stand at a mere 1-5% of smokers per year (F. Song et al., 2002). In 1998, the Government produced a white paper, namely "Smoking Kills" (Department of Health [DH], 1998) in an attempt to address the burden associated with tobacco in the UK. Local Stop Smoking Services (SSS) were proposed as part of this initiative, providing pharmacotherapy products and behavioural support for smokers attempting to quit smoking. Although SSS have proven to be highly effective in reducing smoking prevalence (Ferguson, Bauld, Chesterman, & Judge, 2005; Judge, Bauld, Chesterman, & Ferguson, 2005), innovative and effective interventions would be welcomed to further improve smoking cessation rates.

Risk perception has been described as "the subjective judgement that people make about the characteristics and severity of risk" (Lavino & Neumann, 2010, p. ix). Popular behaviour change theories and models have hypothesised that components, such as perceived vulnerability or risk of illness, contribute towards the implementation or maintenance of health protective behaviours. For example, Weinstein (1983, 1984) proposed that individuals may continue to practise unhealthy behaviours due to inaccurate perceptions of risk and susceptibility, known as optimistic bias (or unrealistic optimism). Previous research has suggested that providing smokers with personalised biomarker feedback in relation to smoking-related disease may help to undermine optimistic bias among smokers, which could

result in enhanced motivation to stop smoking (Young, Hopkins, Smith, & Hogarth, 2010).

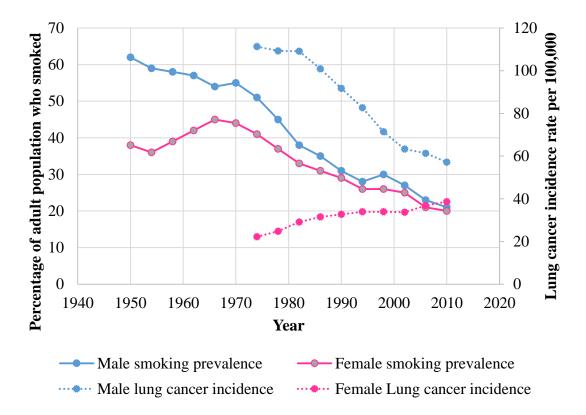


Figure 1. Smoking prevalence and lung cancer incidence, by sex in Great Britain between 1950 and 2010 (Smoking data is weighted as of 1998) (Wald & Nicolades-Bouman, 1991; Office for National Statistics [ONS], 2013a; 2013b)

The LLP risk model was developed based on data from an ongoing molecular-epidemiological case-control study of lung cancer in Liverpool (J. K. Field, Smith, Duffy, & Cassidy, 2005). The risk model incorporates several lung cancer risk factors to predict an individual's absolute five-year lung cancer risk, including smoking, age, gender, asbestos exposure (i.e. occupational exposure), prior malignant disease (other than lung), prior history of pneumonia, and family history of lung cancer. The model has been successfully validated in three independent and external populations from

Europe and North America, demonstrating good discrimination and evidence of predicted benefits for stratifying patients for lung cancer computerised tomography (CT) screening (Raji et al., 2012). The model also provides a means of communicating personalised lung cancer risk to smokers. Despite personalised risk communications being described as one of the most promising approaches to smoking cessation interventions (Velicer, Prochaska, & Redding, 2006), prior research has failed to examine the development, application, and evaluation of lung cancer risk models in the context of smoking cessation.

The current PhD project entailed development of the "LLP intervention", which utilises the LLP risk model to enable calculation and communication of projected lung cancer risk, based on both smoking and non-smoking behaviour. The primary objective of the PhD project was to apply and evaluate the LLP intervention among a population of smokers, to explore the effect on smoking cessation success and lung cancer risk perceptions. In brief, participants were approached during a selection of community groups, overseen by Liverpool's commissioned SSS, Roy Castle FagEnds (RCFE). Participant consent was acquired and baseline questionnaires were completed to establish smoking status, among other factors. Baseline current and recent former smokers were randomised and analysed as two separate datasets, however, the procedure for both trials remained the same. Participants were randomised into one of two groups: (1) the intervention group, in which participants would be provided with generic lung cancer risk information and additionally, the LLP intervention, which entailed delivering personalised lung cancer risk projections based upon the LLP risk model; (2) the control group, in which participants would be provided with generic lung cancer risk information only. The groups were subsequently followed-up at six

months to examine whether any differences in follow-up smoking status and lung cancer risk perceptions occurred between the intervention and control groups. Secondarily, the thesis aimed to explore other factors implicated in smoking-related risk perception and smoking cessation success. The project incorporated quantitative and qualitative research methods to achieve these objectives.

1.2 Thesis organisation

Although the author of the thesis has a background in heath psychology, it was necessary to adopt a multidisciplinary approach to the research project, as the thesis draws upon many research disciplines, including biomedicine, epidemiology, psychology, public health, and sociology. Undertaking a multidisciplinary PhD project enriches the quality of the thesis, as the research can be explored from numerous perspectives; however, an understanding of various methodologies and analytical approaches is required to navigate and interpret a multidisciplinary thesis successfully. The author has therefore endeavoured to ensure that the thesis is clear and comprehendible to individuals from various research disciplines.

The thesis is divided into nine chapters in total. This initial chapter not only introduces the PhD project, but additionally provides an overview of the key known lung cancer risk factors and lung cancer risk prediction models. The aim of this chapter is to identify and provide an understanding of the major risk factors for lung cancer, to appraise the risk prediction models that have been developed to incorporate risk factors, and to explore the application of such models. An awareness of lung cancer risk factors and models is important in demonstrating how the LLP risk model was developed and why this particular model was most relevant to the current project.

Chapter two presents a review of the literature in relation to smoking and risk perception. This chapter provides a profile of smokers in the UK, explores the predictors and associated mechanisms of smoking initiation and cessation, considers tobacco control strategies and interventions, and delivers an overview and review of risk perception theory applicable to the current project. Reviewing the literature not only informs the reader of current knowledge pertaining to tobacco control and behaviour change theory, but it additionally informs the current project design and subsequent analyses.

Chapter three describes the current project methodology. The chapter firstly introduces the study aims and directs the reader towards each of the corresponding results chapters. The project design and justification is subsequently presented, whereby the mixed methods design adopted is discussed. Both quantitative and qualitative research methods are described, including details of participants, materials, and procedures, among other aspects. The analysis plans for both the quantitative and qualitative study components are described in detail within this chapter.

Chapters four, five, six, seven and eight present the project results. Chapter four details the results of the bivariate and multivariate analyses undertaken to investigate the effect of the LLP intervention on follow-up smoking status and lung cancer risk perceptions. This chapter not only has implications for knowledge pertaining to smoking and risk, but additionally, positive results would demonstrate the benefit of delivering the LLP intervention in healthcare settings.

Chapter five presents further quantitative, as well as qualitative results. This chapter focuses on factors implicated in smoking-related risk perception. Quantitative analyses were undertaken to explore the effect of several baseline participant characteristics on perceived personal lung cancer risk, whilst, the qualitative analysis focuses upon exploring factors implicated in smoking-related risk perception. The qualitative results for this chapter discuss two prominent themes associated with smoking-related risk perception: (1) Increased risk awareness; (2) Denying risk. The results are discussed collectively to demonstrate the benefit of mixed methods research. Exploring factors implicated in smoking-related risk perception will contribute towards improved risk communication design, as well as increased knowledge regarding smoking-related risk perception.

Chapter six explores factors implicated in smoking cessation success and refers to the results of the quantitative research component. Predictors of smoking status were explored across three datasets. Firstly, a cross-sectional analysis of all participants recruited in the current project (N = 521) was undertaken. Secondly, an analysis of baseline current smokers was undertaken (n = 297) to explore all potential baseline participant characteristics implicated in follow-up smoking status. Lastly, an analysis of baseline recent former smokers was conducted (n = 216) to explore the predictors of follow-up smoking status. The results are considered collectively to provide a detailed interpretation of potential baseline participant characteristic predictors of smoking status; in doing so, a better understanding of smoking behaviour can be achieved, which in turn may contribute towards improved smoking cessation support.

Chapters seven and eight present the remaining qualitative findings; both chapters explore factors implicated in smoking cessation success. These chapters provide the reader with further insight into the various influential factors associated with smoking cessation and the degree to which smoking cessation is viewed as severely challenging. Chapter seven focuses on psychosocial aspects of smoking and smoking cessation. Participants described motivating factors for quitting and sustaining abstinence, including a range of perceived benefits. Furthermore, participants described the experience of mental conflict upon engaging in a quit attempt. This chapter considers two major themes: (1) Experiences of mental conflict; (2) Perceived benefits of quitting. Chapter eight focuses on issues primarily related to tobacco control practice and policy; the results demonstrate the key features of tobacco control that participants perceived to either encourage or inadvertently deter smoking cessation. This chapter considers two major themes: (1) Reshaping social norms; (2) Attitudes regarding local Stop Smoking Services.

Chapter nine is the final chapter of the PhD thesis. This chapter will draw upon the project results overall and demonstrate how the results of the quantitative and qualitative aspects of the project combine to complement each other and provide a rich understanding of smoking behaviour and risk perception. Each of the study aims are referred to and reflected upon. The research contribution in terms of tobacco control knowledge, practice and policy is explored. Future research directions are considered, followed by a conclusion, in which a summary of the key results and an overview of the project implications are presented.

Having provided an overview of the thesis organisation, the current chapter will now proceed to explore lung cancer incidence and survival, lung cancer risk factors, and lung cancer risk models. Providing insight into these important aspects of research provides the reader with background and justifies the adoption and application of the LLP risk model in the current project.

1.3 Lung cancer incidence and survival

Throughout the 20th century, the importance of lung cancer as a cause of death has increased. In the early 1900s, lung cancer was very rare and caused the deaths of less than 10 men annually per 100,000 (CRUK, 2007). Lung cancer deaths rose six-fold in men and three-fold in women by the 1950s. This increase prompted the first epidemiological study in the UK, which resulted in a confirmed association between smoking and lung cancer (Doll & Hill, 1950). From the early 1970s, lung cancer rates began to decline in the developed world.

In 2012, 35,903 new cases of lung cancer were diagnosed in England, making lung cancer the second most common cancer for both males and females in England; lung cancer accounted for 11.9% of cancer cases among females and 13.6% of cancer cases among males (ONS, 2014b). According to CRUK (2013), lifetime risk of developing lung cancer within the UK in 2008, has been estimated at 1 in 14 for males and 1 in 18 for females. Lung cancer is the primary cause of death in the UK, as reports estimate that 30,437 deaths across England and Wales were attributed to lung cancer in 2013 (ONS, 2014c). Despite some improvements in survival rates over past decades, five-year survival rate in England for 2010-2012 was recently reported at 12.7% (Walters et al., 2015).

1.4 Lung cancer risk factors

A variety of risk factors have been associated with lung cancer. This section will concentrate on the lung cancer risk factors incorporated in the LLP risk model, including age, gender, smoking, occupational exposure (e.g. asbestos), prior malignant disease, prior lung disease (e.g. pneumonia), and family history of lung cancer. Other known epidemiological risk factors not included in the LLP risk model, yet worthy of discussion, will lastly be described. It is important to establish lung cancer risk factors, as such knowledge may enable identification of particularly high-risk groups, prompting implementation of risk prevention strategies.

1.4.1 Age

Lung cancer is strongly associated with age, as older people are more likely to develop the disease. Age-specific incidence of lung cancer rises dramatically from age 40 years, peaking at the 85-89 age range overall (CRUK, 2013). Furthermore, in the UK, approximately three quarters of lung cancer cases were diagnosed among individuals aged 65 years and above in the UK between 2010 and 2012, whilst 87% of cases were diagnosed among individuals aged 60 years and over (CRUK, 2013). The mechanisms for age-related increases in cancer incidence are considered complex; however, the multistep model of cancer, which proposes that several sequential mutations are necessary for a cell to become malignant, advocates that carcinogen exposure duration is a primary cancer risk factor (de Magalhães, 2013).

Age is additionally associated with lung cancer mortality, as deaths are highest among older people. For example, whilst 30% of lung cancer deaths have been reported

among individuals aged 80 years and over, only 27% of lung cancer cases were reported among individuals of the same age range (CRUK, 2014a). Since many lung cancer patients are elderly, they often present with co-morbid difficulties, deeming them unsuitable for radical treatment (Gould & Pearce, 2006); this may therefore contribute towards the increase in age-specific lung cancer deaths among older people.

1.4.2 Gender

Males consistently display higher lung cancer incidence compared to females (Ferlay et al., 2010). Consequently, more males than females die in England and Wales each year due to lung cancer. In 2013, 13,619 female lung cancer deaths were registered, whereas, 16,818 male lung cancer deaths were registered (ONS, 2014c). This significant difference in incidence between genders is likely to be due to males exhibiting higher rates of smoking, among other lifestyle choices, compared with females (e.g. ASH, 2014b).

Despite greater prevalence of smoking and lung cancer among males, it has been suggested that females may be as, if not more, susceptible to lung cancer than males. During the late 1980s and 1990s, a number of case-control studies, were published, providing support for this suggestion (Brownson, Chang, & Davis, 1992; Lubin & Blot, 1984; Osann, Anton-Culver, Kurosaki, & Taylor, 1993; Risch et al., 1993; Schoenberg et al., 1990; Zang & Wynder, 1996). Engeland, Haldorsen, Andersen, and Tretli (1996) published the first prospective cohort study, which examined lung cancer susceptibility between males and females. A large number of participants (N = 26,000) were recruited in 1965 and followed-up from 1966 to 1993. The results suggested that

female current smokers exhibited a relative risk (RR) that was 2.3 times higher, compared with male current smokers (95% CI 1.3-4.1).

More recent cohort studies have contradicted much of the earlier research, as they have tended to report no significant difference in lung cancer risk between males and females (Bain et al., 2004; N. D. Freedman, Leitzmann, Hollenbeck, Schatzkin, & Abnet, 2008; Jemal, Travis, Tarone, Travis, & Devesa, 2003; Kreuzer et al., 2000; Prescott et al., 1998). Furthermore, Y. Yu et al. (2014) recently conducted a systematic review and meta-analysis including 47 articles, containing 404,874 participants. Compared to non-smokers, male to female ratio of RR was 1.61 (95% CI 1.37-1.89) among current smokers. The authors also highlight how this finding conflicts with the traditional perspective that females may be more susceptible to lung cancer that is attributed to smoking tobacco.

Interpretation of such conflicting results has therefore proven challenging. Donington, Le, and Wakelee (2006) highlighted considerations that should be made towards biases in the data, which can substantially impact comparisons of lung cancer risk made in respect to gender, such as inaccurate reporting of smoking habits and the confounding effects of passive smoke.

Regardless of gender differences, Patel (2005) suggested that lung cancer is biologically a different disease among females compared to males. Adenocarcinoma has been found to occur more commonly among females, whereas squamous cell carcinoma has been found to occur more often among males (Ringer, Smith, Engel, Hendy, & Lang, 2005; Thun et al., 1997). The reported lung cancer disparity in relation

to gender, could be attributed to various factors, including genetic and epigenetic differences, gender-specific lifestyle factors, behavioural components, and sex hormones (Paggi, Vona, Abbruzzese, & Malorni, 2010). Further research is necessary to understand the extent to which this disparity exists and the impact of factors that may potentially contribute towards such differences.

1.4.3 Smoking

Doll and Hill (1950) were among the earliest researchers to establish the causal link between smoking and lung cancer. Since this time, numerous epidemiological studies and reviews have consistently identified tobacco smoking as a key risk factor for lung cancer (e.g. Powell, Iyen-Omofoman, Hubbard, Baldwin, & Tata, 2013; Pavlovska et al., 2008; Thun et al., 1997). The causal role of smoking in lung cancer mortality has been irrefutably recognised in longitudinal studies, one of which has spanned 50 years (Stampfer, 2004). Smokers have a 20-fold risk of developing lung cancer, compared to non-smokers (Alberg & Samet, 2003) and it has been estimated that the cumulative risk of death from lung cancer by age 75 among current smokers is 16% (Peto et al., 2000).

Tobacco smoke has been found to contain approximately 4,000 chemical compounds, including at least 69 potent respiratory carcinogenic properties, which damage cells within the lungs and often contribute toward lung cancer (International Agency for Research on Cancer [IARC], 2004). Such carcinogens, including "nicotine-derived nitrosaminoketone" (NNK) and "polycyclic aromatic hydrocarbons" (PAHs) require metabolic activation to apply their carcinogenic effects. The process of metabolic activation leads to the formation of DNA adducts, which are critical in the

carcinogenic process (Hecht, 2003). DNA adducts can potentially be repaired by repair enzymes, whereby DNA can return to its normal undamaged state (Hecht, 1999); however, if the adducts persist during DNA replication, miscoding can occur, which results in a permanent mutation in the DNA sequence (Shrivastav, Li, & Essigmann, 2010). Cells with DNA damage may be removed by apoptosis (programmed cell death) but if a permanent mutation occurs in a critical region of an oncogene (a gene with the potential to cause cancer) or a tumour suppressor gene (a gene that protects a cell from an element of the cancer process), this can lead to activation of an oncogene or deactivation of the tumour suppressor gene (Croce, 2008). Multiple events of this type result in loss of normal cellular growth-control regulation and ultimately, cancer (Wogan, Hecht, Felton, Conney, & Loeb, 2004).

Smoking is a risk factor for all histological types of lung cancer, although the relative risk has been reported as highest for squamous-cell carcinoma (SCC) and SCLC, compared with adenocarcinoma (Simonato et al., 2001). Despite this, there have been dramatic increases in adenocarcinoma incidence within recent decades. For example, in the United States, the ratio of SCC compared with adenocarcinoma was about 18:1 in 1950, whereas it was estimated at 1.2-1.4:1 in 1995 (Wynder & Muscat, 1995). A recent international study demonstrated how adenocarcinoma rates have surpassed those among the previous most frequent sub-type, SCC (Lortet-Tieulent et al., 2014). Such trends have been attributed to changes in smoking behaviour, such as cigarette design and manufacturing technology throughout the late 20th century. For example, average cigarette nicotine and tar delivery decreased respectively from 2.7 and 38mg in 1955, to 0.9 and 12mg in 1997 (Hoffmann, Djordjevic, & Hoffmann, 1997).

Choice of tobacco product used has been found to contribute towards lung cancer risk (Peter N. Lee, 2001; P. N. Lee, Foley, & Coombs, 2012). For example, P. N. Lee et al. (2012) conducted a meta-analysis to investigate epidemiological evidence in the 1900s associating smoking with lung cancer, whereby they identified 287 studies. They found that hand-rolled cigarettes were associated with higher lung cancer risk overall, compared with manufactured cigarettes (random-effects RR 1.29, 95% CI 1.12-1.49). Lung cancer incidence was also lower with use of filtered cigarettes compared to non-filtered cigarettes (RR 0.69, 95% CI 0.61-0.78); however, research suggests that smokers who move from non-filtered cigarettes to filtered cigarettes compensate by increasing the number of cigarettes per day, thus compensation acts as a lung cancer risk factor in itself (Augustine, Harris, & Wynder, 1989). Although unfiltered (or plain) cigarettes have become almost unheard of in England presently, hand-rolled cigarettes have become more prevalent within the past decade (ONS, 2013b).

Cigar and pipe smokers additionally exhibit significantly greater lung cancer risk, compared with never smokers, yet the risk is substantially less than it is among cigarette smokers (Shaper, Wannamethee, & Walker, 2003). This lower risk is believed to be attributed to a lesser amount of tobacco smoked and inhaled among cigar and pipe smokers (Boffetta et al., 1999). It should be noted, however, that cigarettes have remained the most commonly used form of tobacco in the UK since the early 20th century (Wald & Nicolaides-Bouman, 1991) and the popularity of cigars and pipes has dramatically decreased in past decades. In Great Britain, a mere 2% of males have reported smoking at least one cigar a month and less than 0.5% of males

stated they smoked a pipe; cigar and pipe smoking among females is even rarer than it is among males (S. Robinson & Harris, 2011).

In addition to tobacco product use, cigarette design, and manufacturing technology, other smoking variables have been found to contribute towards lung cancer incidence, particularly smoking duration and number of cigarettes smoked per day (S. A. Khuder, 2001; P. N. Lee et al., 2012; S. Yu & Zhao, 1996). For example, S. A. Khuder (2001) conducted a meta-analysis to explore the effect of smoking on major histological types of lung cancer. Heavier smokers (≥ 30 cigarettes per day) exhibited increased risk of adenocarcinoma (odds ratio [OR] 4.10, 95% CI 3.16-5.31) and SCLC (OR 18.3, 95% CI 9.26-36.4), whilst smokers with greater smoking durations (≥ 40 years) additionally exhibited increased risk of adenocarcinoma (OR 3.80, 95% CI 2.35-6.16) and SCLC (OR 38.6, 95% CI 11.9-125).

There is a large amount of research that has demonstrated that ex-smokers convey a lower risk of developing lung cancer, in comparison to current smokers (Ebbert et al., 2003; Gellert, Schottker, & Brenner, 2012; Peto et al., 2000; Pirie, Peto, Reeves, Green, & Beral, 2013). Peto et al. (2000) identified the cumulative risk of lung cancer death up to age 75 years as 10%, 6%, 3% and 2% for men who quit smoking at ages 60, 50, 40 and 30, respectively. Such research identifies that the reduction in risk decelerates as smoking duration and age at smoking cessation increases but additionally, the research suggests that smokers who quit well into late-middle age could avoid a great proportion of their subsequent lung cancer risk. Although many ex-smokers will never return to the risk level of a never smoker due to genetic damage

incurred (Peto et al., 2000), smoking cessation is evidently an effective strategy in significantly reducing lung cancer risk among smokers.

1.4.4 Occupational exposure

Occupational exposure refers to contact with carcinogenic properties in the workplace, such as asbestos. In 2010, it has been estimated that 20.5% of lung cancers among men and 4.3% among women have been found to be attributed to occupational exposures in the UK (Parkin, 2011a). Better insight into the carcinogenicity of chemical and physical agents has been achieved through investigating occupational groups, consisting of individuals who have been heavily exposed to such agents within the workplace. Research has identified that individuals working in specific occupations are at increased lung cancer risk, such as painters (Guha et al., 2011), asbestos cement workers (Magnani et al., 2008), hairdressers (Takkouche, Regueira-Mendez, & Montes-Martinez, 2009), asphalt workers and roofers (Partanen & Boffetta, 1994), printers (Lynge et al., 1995), rubber industry workers (Kogevinas et al., 1998), aluminium smelter workers (Selden, Westberg, & Axelson, 1997), iron and steel workers (Grimsrud, Langseth, Engeland, & Andersen, 1998), and workers exposed to high levels of lead (Anttila et al., 1995). Rushton et al. (2010) found that the three greatest risk factors of occupation-attributable lung cancer registrations in 2005, included asbestos, silica, and diesel exhaust, accounting for 41%, 17%, and 13% of all occupation-attributable cancer registrations, respectively.

Asbestos consists of naturally occurring silicate minerals, often used commercially due to their desirable physical properties. Exposure to asbestos is commonly cited as a risk factor among both non-smokers and smokers (Brown, Darnton, Fortunato, &

Rushton, 2012; Cogliano et al., 2011; P. N. Lee, 2001; Subramanian & Govindan, 2007) and it has been suggested that within Great Britain, asbestos-related lung cancers may have accounted for 2-3% of all lung cancer deaths between 1980 and 2000 (Darnton, McElvenny, & Hodgson, 2006). An interaction between smoking and asbestos has also been documented. For example, Frost (2011) found that among a cohort of asbestos workers who smoked, approximately 3% of lung cancer deaths were attributable to asbestos alone, 66% were due to smoking alone, and 28% were due to the interaction between asbestos and smoking. Asbestos-attributed lung cancer relative risk has, however, been found to be dependent on various factors, including asbestos fibre type (Loomis et al., 2012), asbestos dose (Gustavsson et al., 2002) and asbestos assessment method (Lenters et al., 2011).

Silicosis is a form of occupational lung disease, which develops as a result of inhalation of the chemical compound, silica. Erren, Morfeld, Glende, Piekarski, and Cocco (2011) reported a fixed-effects relative lung cancer risk of 2.1 (95% CI 2.0-2.3) among individuals affected by silicosis, utilising 38 studies. A dose-response relationship has also been documented between silica exposure and lung cancer risk (Lacasse, Martin, Gagne, & Lakhal, 2009), although the existing literature is somewhat ambiguous as to whether silica exposure increases lung cancer risk without silicosis (Brown, 2009). Checkoway and Franzblau (2000) suggested that efforts to fully comprehend the role of silica as a lung cancer risk factor have been hampered due to incomplete or biased ascertainment of silicosis, inadequate exposure assessment, and the inherently strong correlation between silicosis and silica exposure. Furthermore, Kurihara and Wada (2004) undertook a meta-analysis which examined the relationships between silica, smoking and lung cancer. The findings revealed that

cigarette smoking significantly increased lung cancer risk among those previously exposed to silica (RR 4.47, 95% CI 3.17-6.30).

Diesel exhaust was additionally recently classified as a lung cancer risk factor (IARC, 2012). Olsson et al. (2011) pooled information on lifetime work histories and tobacco smoking from 11 case-control studies conducted in Europe and Canada. The findings demonstrated increased lung cancer risk among individuals from the highest quartile of workers who reported diesel exposure, compared to those with no reported exposure (OR 1.31, 95% CI 1.19-1.43). Various professions involving high exposure to diesel have been associated with lung cancer risk, including mining (Silverman et al., 2012), railroad work (Garshick et al., 2004), and truck driving (Jarvholm & Silverman, 2003); however, epidemiological evidence has been assessed as limited, due to few studies reporting on exposure-response relationships and the majority of studies failing to adequately control for potential confounders (Olsson et al., 2011). For example, Larkin et al. (2000) examined the extent to which smoking may confound the relationship between diesel exhaust and lung cancer risk. Workers exposed to diesel exhaust exhibited greater risk prior to smoking adjustment (RR 1.58, 95% CI 1.14-2.20), whilst adjustment for smoking presented significantly lower relative risk estimates (RR 1.44, 95% CI 1.01-2.05). A synergic effect on lung cancer risk between diesel exhaust exposure and smoking has been suggested (e.g. Pintos, Parent, Richardson, & Siemiatycki, 2012) but presently, evidence is limited.

1.4.5 Prior malignant disease

Lung cancer risk is associated with various forms of prior malignant disease, such as breast cancer (Lorigan, Califano, Faivre-Finn, Howell, & Thatcher, 2010), head and

neck cancer (Youlden & Baade, 2011), Hodgkin's lymphoma (Ibrahim et al., 2013; Lorigan, Radford, Howell, & Thatcher, 2005), non-Hodgkin's lymphoma (Morton et al., 2010; Mudie et al., 2006), testicular cancer (Travis et al., 2005), and uterine sarcoma (Koivisto-Korander et al., 2012). For example, Ibrahim et al. (2013) conducted a meta-analysis to examine lung cancer risk among patients who had survived Hodgkin's lymphoma, which incorporated 21 studies and a median follow-up of 11.5 years. Patients who had suffered Hodgkin's lymphoma were significantly more likely to develop lung cancer compared to those who had not developed the disease (RR 4.62, 95% CI 3.18-6.70); however, relative risk varied in association with previous malignancy type.

The exhibited increase in lung cancer risk among individuals with a history of malignant disease, has been primarily attributed to previous cancer treatment, including radiotherapy (Ibrahim et al., 2013; Lorigan et al., 2010; Lorigan et al., 2005; Travis et al., 2005), chemotherapy (Ibrahim et al., 2013; Lorigan et al., 2005; Mudie et al., 2006; Travis et al., 2005), or both (Ibrahim et al., 2013; Travis et al., 2005). For example, Travis et al. (2005) modelled relative risk of lung cancer, by compiling a cohort consisting of 40,576 one-year survivors of testicular cancer. They found that secondary cancer risk increased substantially, as a consequence of radiotherapy alone (RR 2.0, 95% CI 1.9-2.2), chemotherapy alone (RR 1.8, 95% CI = 1.3-2.5), and both (RR 2.9, 95% CI 1.9-4.2). A more recent study (Lorigan et al., 2010) explored the effect of breast cancer treatment on lung cancer risk and argued that radiotherapy only predicted lung cancer among patients who had experienced older treatment techniques. These findings suggest that more modern radiotherapy techniques, may contribute towards little, if any, increase in lung cancer risk.

1.4.6 Prior lung disease

In addition to previous cancer diagnosis, it has also been suggested that prior lung disease is associated with lung cancer risk. A number of recent meta-analyses have confirmed relationships between various prior lung diseases and lung cancer risk, including chronic obstructive pulmonary disease (COPD), chronic bronchitis, emphysema, tuberculosis, and pneumonia (Brenner et al., 2012; Brenner, McLaughlin, & Hung, 2011; Denholm et al., 2014). For example, lung cancer risk is often reported as elevated among individuals with a previous diagnosis of pneumonia. Brenner et al. (2012) conducted a meta-analysis that incorporated 17 studies, including 24,607 cases and 81,829 controls. The studies were primarily conducted across Europe and North America, between 1984 and 2011, to explore the effect of a range of prior lung diseases on lung cancer incidence. Based on 12 studies investigating pneumonia, they found that individuals with a previous history of pneumonia exhibited increased lung cancer risk (RR 1.57, 95% CI 1.22-2.01). Denholm et al. (2014) also conducted a pooled analysis on previous lung disease, utilising 12,739 cases and 14,945 controls from seven case-control studies. They observed a positive effect of pneumonia on lung cancer risk among men (OR 3.31, 95% CI 2.33-4.70), however, the significance of this effect diminished upon including patients with a diagnosis of pneumonia more than two years prior to developing lung cancer.

Schabath et al. (2005) suggested that prior lung disease can create long term inflammation within the bronchi, accompanied by a continual cycle of injury and repair; such damage is believed to contribute towards lung carcinogenesis. More recently, various researchers have highlighted possible methodological limitations,

which may have contributed towards the documented relationship (Brenner et al., 2012; Brenner et al., 2011; Denholm et al., 2014). Brenner et al. (2011) suggested that reverse causality (whereby infections may have been the result of a weakened immune system due to undiagnosed early lung cancer), misdiagnosis (whereby a patient may have exhibited symptoms of early lung cancer, which were misdiagnosed as other lung disease), and detection bias (whereby individuals with disease such as tuberculosis or pneumonia may be more likely to be diagnosed with lung cancer as a result of additional diagnostic tests often used in infections), may partly contribute towards the evident association. The identified methodological limitations corroborate with Denholm et al.'s (2014) aforementioned findings regarding the relationship between pneumonia and lung cancer risk. The true extent of prior lung disease on lung cancer risk and the associated mechanisms of the relationship have yet to be fully understood, highlighting the importance of ongoing research.

1.4.7 Family history

Research suggests that family history of lung cancer is an important predictor of lung cancer risk. A recent pooled analysis (Cote et al., 2012) utilised 24 case-control studies, totalling 24,380 cases and 23,305 controls. Having adjusted for smoking and other potential confounders, they found that individuals with a first-degree relative with lung cancer exhibited increased lung cancer risk (OR 1.51, 95% CI 1.39-1.63); this association was strongest among those with sibling family history of lung cancer (OR 1.82, 95% CI 1.62-2.05).

Matakidou, Eisen, and Houlston (2005) previously conducted a systematic review of 28 case-control, 17 cohort, and seven twin studies, exploring the relationship between

family history and lung cancer risk. The findings suggested that risk is greater among relatives of cases diagnosed at a younger age and among those with multiple affected relatives. Cassidy, Myles, Duffy, Liloglou, and Field (2006) explored the impact of age at diagnosis among a first-degree relative, using 579 cases and 1,157 controls. They found that individuals with a first-degree relative diagnosed with lung cancer below 60 years, exhibited increased risk of developing lung cancer, compared with those without family history (OR 2.08, 95% CI 1.20-3.59). Furthermore, individuals with a first-degree relative diagnosed with lung cancer below the age of 60 years, were substantially more likely to develop lung cancer below 60 years themselves, compared with participants who had no family history (OR 4.89, 95% CI 1.47-16.25).

Matakidou et al. (2005) suggested that the association between family history and lung cancer risk is attributed to genetic susceptibility but additionally, to shared environmental exposures. Further research is required to distinguish the extent to which genetic and environmental factors contribute towards the association between family history of lung cancer and lung cancer risk.

1.4.8 Other known risk factors

The aforementioned lung cancer risk factors are all of relevance to the LLP risk model, which is the focus of the current project; however, several other epidemiological lung cancer risk factors have been acknowledged in the literature, which were not included in the LLP risk model, such as air pollution, involuntary smoking, and radon gas. To provide a fuller overview of all identified lung cancer risk factors, such factors are briefly discussed within this section.

1.4.8.1 Air pollution

Outdoor air pollution, particularly carcinogenic airborne particulate matter, has been identified as a lung cancer risk factor (H. Chen, Goldberg, & Villeneuve, 2008; Hamra et al., 2014). According to CRUK (2014b), approximately 7.8% of lung cancer cases in the UK are attributed to PM2.5 particulate exposure per year. The strongest evidence suggests an increased risk among those exposed to nitrogen oxides (Masri et al., 2005; Vineis et al., 2006), which may be due to nitrogen oxide exposure being easier to measure than many other air pollutants, such as sulphur dioxide (Vineis et al., 2007).

Traffic fumes have been identified as the main source of air pollution in urban areas and IARC (2012) states that diesel exhaust may increase lung cancer risk among the general public; however, the majority of evidence arguing the effect of diesel exhaust is derived from studies examining highly-exposed workers. Hamra et al. (2014) conducted meta-analyses including 18 studies, which explored the relationship between lung cancer and exposure to particulates, PM2.5 and PM10. They estimated increased lung cancer risk by 10 micrograms per cubic metre of air (μ g/m³) increase in PM exposure and found that lung cancer risk increased with exposure to PM2.5 (RR 1.09, 95% CI 1.04-1.14) and PM10 (RR 1.08, 95% CI 1.00-1.17).

Indoor air pollution from solid fuel use for home cooking or heating (i.e. coal and biomass, such as wood) has additionally been associated with increased lung cancer risk (Galeone et al., 2008; Hosgood et al., 2011; Kurmi, Arya, Lam, Sorahan, & Ayres, 2012; Lin, Murray, Cohen, Colijn, & Ezzati, 2008). For example, Kurmi et al. (2012) conducted a meta-analysis of 28 studies, in which they found that users of solid fuels were more likely to develop lung cancer, than non-users (RR 1.70, 95% CI 1.50-1.94);

however, indoor air pollution rarely involves UK samples, as solid fuel is used far less frequently in the UK compared to many other nationalities where excessive use may be problematic (Lissowska et al., 2005).

1.4.8.2 Involuntary smoking

Involuntary smoking (or passive smoking) has been defined as "the exposure of a non-smoker to the smoke produced by active smoking" (Sasco, Secretan, & Straif, 2004, p. S8) and is often referred to as environmental tobacco smoke (ETS) or secondhand smoke (SHS). Oberg, Jaakkola, Woodward, Peruga, and Pruss-Ustun (2011) estimated that worldwide in 2004, 33%, 35% and 40% of male non-smokers, female non-smokers, and children, were exposed to secondhand smoke, respectively. An estimated 14.8% of never smoker lung cancer cases have been attributed to secondhand smoke in the UK (Parkin, 2011b). In January 1981, two studies were published documenting how never smoking women living with a spouse who smoked, exhibited increased risk of lung cancer (Hirayama, 1981; Trichopoulos, Kalandidi, Sparros, & MacMahon, 1981).

Since the early 1980s, a vast body of evidence has continued to demonstrate the carcinogenic effect of passive smoking. R. Taylor, Najafi, and Dobson (2007) conducted a meta-analysis of 55 studies, with the aim of investigating the effect of involuntary smoking on lung cancer risk. This meta-analysis unveiled that the overall relative risk of spousal exposure was 1.27 (95% CI 1.17-1.37) among females. Furthermore, Stayner et al. (2007) also conducted a meta-analysis, which focused on the effect of involuntary smoking on lung cancer, specific to the workplace. The analysis incorporated data from 22 studies and indicated that involuntary smoking in

the workplace was associated with increased lung cancer risk (RR 1.24, 95% CI 1.18-1.29); workers classified as being highly exposed exhibited an even higher level of risk (RR 2.01, 95% CI 1.33-2.60). These findings support previous suggestions of a dose-response relationship between involuntary smoking and lung cancer, similarly to active smoking and lung cancer (A. K. Hackshaw, Law, & Wald, 1997; R. Taylor et al., 2007), demonstrating that there may be no threshold for tobacco carcinogenesis (Alberg & Samet, 2003).

1.4.8.3 Radon gas

Radon is a natural radioactive gas that comes from the minute amounts of uranium that occur naturally in rocks and soils (Health Protection Agency, 2012). The carcinogenicity of radon has been investigated in occupationally exposed populations, particularly among miners (Leuraud et al., 2011). Lung cancer risk has also been associated with residential exposure to radon, whereby radon has filtered into homes from the ground; increasingly so in granite rich areas. Pavia, Bianco, Pileggi, and Angelillo (2003) conducted a meta-analysis of residential exposure to radon gas and lung cancer and reported a pooled OR estimate of 1.24 (95% CI 1.11-1.38) lung cancer risk based on residential exposure at 150 becquerel (a derived unit of radioactivity) per cubic metre (Bq/m³). It should be noted that the majority of homes in the UK have reasonably low radon levels, with an average of approximately 20 Bq/m³ (Bowie & Bowie, 1991; Scivyer, 2001).

Estimates are unclear, but residential radon exposure in the home is likely to be responsible for 0.5% of lung cancer cases in the UK (Parkin, 2011b). Radon exposure is associated with lung cancer risk among both smokers and non-smokers but the

relationship between smoking and radon has been identified as synergistic, in consideration of their effect on lung cancer risk (Lubin & Steindorf, 1995); 3% of lung cancer cases are attributed to the synergic effect of radon and smoking combined (Parkin, 2011b). Darby and Hill (2003) detailed that based on an indoor concentration of 400 Bq/m³, absolute lung cancer risk by the age of 85 was estimated at 2.2% for non-smokers and 49.3% for smokers. The aforementioned research further highlights the damaging effect of tobacco smoking in combination with additional factors.

1.5 Risk modelling

Within recent years, there has been growing interest in the development of methods to predict individual lung cancer risk. Cancer risk prediction models provide an important approach to risk assessment through identifying high-risk individuals, facilitating the design and planning of clinical trials, assisting with the development of benefit-risk indices, improving clinical decision making, and enabling the estimation of cancer burden and cost among a population (A. N. Freedman et al., 2005); this highlights how the utilisation of risk prediction models can contribute towards controlling cancer-related morbidities and deaths.

The number of risk prediction models developed in the field of health has steadily increased since the first risk prediction model for coronary heart disease was published in 1976 (Kannel, McGee, & Gordon, 1976). Gail et al. (1989) developed the first model to predict the likelihood of developing a specific form of cancer (breast) within a given period of time. More recent cancer risk prediction models have been developed to enable the identification of individuals at high-risk of developing various forms of cancer, including bladder, breast, cervical, colorectal, oesophageal, liver, melanoma,

ovarian, pancreatic, prostate, testicular, and lung cancer (National Cancer Institute, 2014).

The accuracy of a cancer risk prediction model relies on a number of factors, including the identification of risk factors, the accuracy of risk factor measurement, the appropriateness of the population, and the statistical techniques used for modelling (Cassidy, Duffy, Myles, Liloglou, & Field, 2007). In comparison to risk models developed in relation to breast cancer and other forms of cancer, few lung cancer risk prediction models had been developed up until the last decade (J. K. Field, 2008); however, lung cancer risk prediction models have advanced substantially within this limited time period, with more recent models incorporating biological and genetic data, in addition to clinical and epidemiological factors (Raji, Agbaje, Duffy, Cassidy, & Field, 2010).

1.5.1 Key lung cancer risk prediction models

Within the past decade, several lung cancer risk prediction models have been developed, incorporating a number of risk factors. Colditz et al. (2000) used grouped consensus data to develop an early lung cancer risk index. Lung cancer risk factors identified included tobacco smoking characteristics, family history, passive smoking, and air pollution, among other components. This index provides a simple estimation of personal cancer risk. The index may help individuals understand applicable risk factors and consider specific lifestyle changes that will reduce risk, but it does not provide precise, individualised information on risk.

Peto et al. (2000) investigated the impact of prolonged smoking and smoking cessation on lung cancer risk. Using case-control data, they estimated the cumulative risk of death from lung cancer by age 75 years among all current smokers and current smokers of at least 25 cigarettes per day to be 16% and 24%, respectively. They further provided the same cumulative lung cancer risk estimates for males who quit smoking at various ages. For example, a male who quits smoking at age 60 years is exposed to a 10% cumulative risk of lung cancer death by age 75, whereas, a male who quits at age 40 years is exposed to only a 3% cumulative risk. Again, this study provides a general guide regarding the relationship between smoking behaviours and lung cancer risk, although it fails to consider other key lung cancer risk factors.

Bach et al. (2003) published the first study to utilise modern statistical modelling in the development of a lung cancer risk model. They used prospective data on 18,172 current and former smokers to determine the extent to which lung cancer risk differentiates predictably among smokers. This risk prediction model calculates 10year lung cancer risk and the model considers age, sex, asbestos exposure and smoking history. A concordance index of 0.72 was reported, which was internally validated, suggesting moderate discrimination. Furthermore, model calibration demonstrated as good. Cronin et al. (2006) assessed the validity of Bach et al.'s (2003) model among smokers (N = 6,239) in the placebo arm of the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study (ATBC). They found that the risk model underestimated lung cancer risk, as expected cases were lower than observed cases (lung cancer cases expected/observed = 0.89, 95% CI 0.80-0.99). Although the model has contributed substantially to the domain of lung cancer risk prediction, it does yield several limitations. The population used to develop the model was derived from

participants involved in the Carotene and Retinol Efficacy Trial (CARET), a chemopreventive trial involving high-risk populations (see Omenn et al., 1994); therefore, the model cannot be applied to individuals within a general population and is only predictive for smokers aged 50-75 years of age, consuming 10-60 cigarettes per day, for 25-55 years.

Spitz et al. (2007) developed three risk prediction models, calculating absolute one-year lung cancer risk by using case-control data, consisting of 1,851 cases and 2,001 controls. Subject data were randomly divided into either training (75%) or validation (25%) groups for never, former, and current smokers, with multivariate models being constructed from the training sets. They aimed to expand the risk factors included within Bach et al.'s (2003) model and therefore, Spitz et al.'s (2007) models incorporate environmental tobacco smoke, family history of cancer, dust exposure, prior respiratory disease and smoking history. The researchers assessed the discriminatory ability of the three models in the validation sets by examining the area under the receiver operating characteristic curve (AUC), which was identified for never, former, and, current smokers, as 0.57, 0.63, and 0.58, respectively, indicating limited discrimination. Hosmer-Lemeshow goodness-of-fit yielded a non-significant result, which is typically associated with good calibration; however, it has been argued that the statistics do not provide convincing evidence of good calibration (Tammemagi, 2015).

The lung cancer risk prediction model most relevant to the current project, is the LLP risk model. Cassidy et al. (2008) developed a lung cancer risk prediction model for five-year lung cancer risk, based on 579 lung cancer cases and 1,157 age and sex

matched population-based controls. Various risk factors were entered into the model, including age, sex, history of pneumonia, asbestos exposure, previous cancer, family history of lung cancer, and smoking duration. The model was applied to the casecontrol population, whereby the AUC was 0.71, suggesting good discrimination between cases and controls. Tammemagi (2015) argued that although the Hosmer-Lemeshow goodness-of-fit was non-significant, the statistics did not provide substantial evidence of good calibration and furthermore, the model's ability to predict risk among never smokers had not been demonstrated. However, the LLP risk model has been externally validated substantial in comparison to the majority of lung cancer risk prediction models. Raji et al. (2012) validated the LLP risk model within three independent populations (UK, Europe and North America). The findings suggested that the model demonstrated good discrimination and support for the utility of the LLP risk model in stratifying individuals for low-dose computerised-tomography (LDCT). More recently, the model has been applied to stratify high-risk individuals for participation in the UK Lung Cancer Screening Trial (UKLS) (see McRonald et al., 2014), demonstrating its suitability for application in such contexts.

D'Amelio et al. (2010) compared the aforementioned three models (Bach et al., 2003; Cassidy et al., 2008; Spitz et al., 2007) in an independent population, using data for 3,197 lung cancer cases and 1,703 controls. The five-year lung cancer risk for each model was ascertained and the discriminatory power, accuracy and clinical utility of each were compared. The Spitz et al. model and the LLP risk model similarly outperformed the Bach et al. model in terms of discrimination between former and current smoking cases and controls, potentially due to the Spitz et al. and LLP models incorporating population-based incidence rates. In terms of accuracy, Spitz et al.'s

model displayed the highest positive predictive values (PPV; The probability of accurately categorising an affected participant), whereas the LLP model demonstrated the highest negative predictive values (NPV; The probability of accurately categorising an unaffected participant). With regard to clinical utility, the LLP risk model was superior in identifying a greater number of lung cancer cases than the other models. These findings are potentially attributed to the importance of tobacco smoking in the LLP risk model, whereas smoking status is a matching variable in Spitz et al.'s model, as opposed to a risk factor. Although, the LLP risk model was superior in identifying a greater number of lung cancer patients, it additionally, incorrectly recognised a large number of controls as lung cancer patients.

When the current project design was originally conceived in 2012, the LLP risk model was selected as the most appropriate lung cancer risk prediction model to base the LLP intervention on for several reasons: the model demonstrates good specificity and accuracy in predicting lung cancer; it has been externally validated among a number of populations; it can be applied among individuals who are asymptomatic; it is not costly in terms of generating output (unlike some other models, which incorporate clinical or biological components); it can be administered simply by non-clinicians.

Several lung cancer risk prediction models have been developed following the LLP risk model and therefore, these will also be discussed to provide a rounded overview of lung cancer risk models. Tammemagi et al. (2011) developed the Prostate, Lung, Colorectal and Ovarian Cancer (PLCO) lung cancer risk models, using data derived from the PLCO screening trial (Gohagan, Prorok, Hayes, & Kramer, 2000). Model 1 consisted of all PLCO controls (N = 70,964), whereas Model 2 was developed among

smokers from the PLCO control group (N = 38,254). Both models incorporated several risk factors including age, socioeconomic status (based on education), family history of lung cancer, COPD, recent chest x-ray, smoking status, pack-years smoked, and smoking duration. The models demonstrate good calibration and discrimination; the AUCs were 0.84 and 0.78, for Model 1 and 2, respectively. The models were, however, limited in that the sample included individuals only between the ages of 55-74 years, who generally had a higher socio-economic status and additionally, data on asbestos exposure and other variables was not available to the researchers. Furthermore, the sample used to externally validate the models was derived from the same population used to develop the model development sample (i.e. the sample consisted of those within the PLCO intervention arm); therefore, the models may be less applicable among more general populations. A subsequent version of the PLCO model has since been developed into the PLCO_{m2012} (Tammemagi et al., 2013). This version includes a further two risk predictors (previous history of malignant disease and race/ethnicity) and omission of a previous predictor (recent chest x-ray). The revised model demonstrated improved discrimination, with an AUC of 0.80 and good calibration. Further external validation of the model would be of benefit.

Hoggart et al. (2012) built separate lung cancer risk prediction models by utilising data acquired from ever smokers (N = 169,035) recruited from the European Prospective Investigation into Cancer and Nutrition (EPIC) (see Riboli et al., 2002). Separate models were developed for current, former and never smokers and the dataset was divided into independent training and test sets. Several risk factors were originally considered during the development of the model but the resulting model incorporated only age and smoking history. The ever smoker model demonstrated good predictive

accuracy, with an AUC reported to be 0.84 (95% CI 0.81-0.88) and also, good calibration. Although a model was considered for never smokers, preliminary analyses displayed substantially poor predictive accuracy, potentially due to missing data and inaccurate recording of some risk factors; therefore, Hoggart et al. (2012) failed to report results for never smokers. Furthermore, external validation of the model is warranted.

A number of lung cancer risk prediction models have recently focused on attempting to identify patients at risk of lung cancer within primary care settings, by incorporating epidemiological and clinical predictors. Hippisley-Cox and Coupland (2011) derived and validated an algorithm, which attempted to identify individuals with suspected lung cancer in primary care, by incorporating various epidemiological and clinical risk factors, including a variety of symptoms (e.g. coughing). The algorithm was developed using 3,785 lung cancer cases, arising from 4,289,282 person-years in the cohort. The algorithms were found to explain 72% of variation and the AUC statistic was 0.92 for both males and females but little is mentioned in terms of calibration. Furthermore, Iyen-Omofoman, Tata, Baldwin, Smith, and Hubbard (2013) developed a lung cancer risk prediction model by incorporating 12,074 lung cancer cases and 120,731 controls, derived from a large general practice database. Socio-demographic and clinical features were ascertained up to two years prior to diagnosis. Upon validation, the model demonstrated good discrimination, with an AUC of 0.88, although information on calibration was not provided Utilising general practice data is clearly useful in developing models of risk, although such models might fail to consider variation in primary care presentation among patients (e.g. Galda, Cheater, & Marshall, 2005).

The Liverpool Lung Project recently developed and internally validated the LLP risk prediction model for lung cancer incidence (or LLPi) (M. W. Marcus, Chen, Raji, Duffy, & Field, 2015). The LLP cohort (N = 8,760) was followed-up between a tenyear period, using hospital episode statistics. Cox proportional hazards models were utilised to identify lung cancer risk predictors. The model incorporates several significant predictors, including age, gender, smoking duration, prior history of COPD, prior diagnosis of malignancy, and family history of early onset of lung cancer (< 60 years). The LLPi model demonstrated a C-statistic of 0.85 (95% CI 0.83-0.87), which was similar to the bias-corrected bootstrap resampling statistic (0.85, 95% CI 0.83-0.87), indicating excellent discrimination (Hosmer, Lemeshow, & Sturdivant, 2013). A non-significant Hosmer-Lemeshow goodness-of-fit test indicated good calibration, although the LLPi model requires further validation.

1.5.2 Clinical and biological risk model expansion

Lung cancer diagnosis has been found to be predicted, in part, by prior lung disease (e.g. Brenner et al., 2012) and several clinical symptoms (e.g. Iyen-Omofoman et al., 2013). Clinical and biological risk factors have been increasingly incorporated into lung cancer risk prediction models, as such measures can provide an objective, immediate measure of one's current status, whilst avoiding the disadvantages of self-reports, such as recall bias and social desirability. For example, several respiratory diseases can be objectively measured using spirometry (FEV1; forced expiratory volume in one second) and FEV1 has been independently associated with lung cancer risk (Fry, Hamling, & Lee, 2012), suggesting that the incorporation of spirometry outputs in risk prediction modelling may improve prediction specificity.

Key lung cancer risk prediction models reflect the strong causal relationship between epidemiological factors, such as smoking and lung cancer risk; however, epidemiological factors cannot account for all variations in lung cancer risk, particularly among never smokers and individuals exhibiting early lung cancer onset (D'Amelio et al., 2010). In such cases, it is likely that the occurrence of lung cancer is attributed to a genetic predisposition and therefore, lung cancer risk prediction models may benefit from considering genetic risk factors where possible. Validated biomarkers identified using biological specimens, such as serum/plasma and bronchial lavage/induced sputum, can also be utilised to ascertain those individuals exhibiting the highest levels of risk (J. K. Field, 2008). Incorporation of biomarkers into lung cancer risk prediction may enhance current methods, although this may also be challenging with regard to cost and feasibility.

Prindiville et al. (2003) found that individuals with cytological atypia (abnormal cells) in sputum are at increased risk of lung cancer, independent of continued smoking. The study demonstrated cumulative lung cancer incidence rates among individuals with moderate or worse cytological atypia in sputum reaches 10% and 20%, at three and six years, respectively. The sample used consisted of heavy smokers only, suggesting that the results cannot be generalised among never and light to moderate smokers. Clinical measures, such as sputum cytology cannot replace the key lung cancer risk prediction models, but introducing such measures may complement them.

Spitz et al. (2008) aimed to enhance their previous models (i.e. Spitz et al., 2007) by incorporating two markers of DNA repair capacity into them. This resulted in a more

precise, albeit limited, discriminatory performance increase when compared to the original model. For example, AUCs for former smokers were 0.67 for the original and 0.70 for the enhanced model. Furthermore, Raji et al. (2010) expanded the original LLP risk model (Cassidy et al., 2008) by incorporating a genetic susceptibility polymorphism (a single nucleotide polymorphism [SNP], in locus SEZ6L) into the model. Raji et al. (2010) reported a significant, yet modest increase in AUC from 0.72 for the original model, to 0.75 for the enhanced model.

In recent years, large genome-wide association studies have enabled the discovery of a number of SNPs linked to lung cancer risk across the entire human genome, urging the potential expansion of genetic-based lung cancer risk prediction. Spitz et al. (2008) and Raji et al. (2010) clearly demonstrate the modest impact of polymorphisms on an individual basis, however, consideration of the impact of SNPs on a collective basis may be considerably more influential (J. H. Park, Gail, Greene, & Chatterjee, 2012). For example, statistical modelling may enable examination of the interactive effects between several SNPs and various epidemiological factors, however, such approaches may also be substantially complex to implement (Janssens et al., 2006; Spitz et al., 2007).

As discussed, the incorporation of individual biomarkers into risk prediction models is currently limited with regard to discriminatory performance (Raji et al., 2010; Spitz et al., 2008). Furthermore, collection and analysis of biological specimens can also be costly both in terms of finance and time, compared with epidemiologically-based models. It should also be considered that for a biomarker to be appropriate for lung cancer risk prediction, it should be measured using non-invasive sampling, such as

using saliva, to enable ease of collection within primary care and similar general population settings. These limitations demonstrate how integrating biomarkers into general population risk prediction may often be inappropriate.

1.5.3 Applications of lung cancer risk models

The National Institute for Health and Care Excellence (NICE) (2011) highlighted the promotion of early detection as one of its key priorities for tackling lung cancer within the UK, suggesting that the public should be better informed of the symptoms of lung cancer through awareness campaigns. Some lung cancer risk prediction models enable the discrimination between individuals exhibiting differing levels of risk and identification of individuals who have not developed lung cancer but are at risk of developing the disease in the future. The utility of risk models may therefore allow more appropriate selection of individuals for chemopreventive interventions or computerised tomography (CT) screening, enabling earlier diagnosis of lung cancer, compared to other clinical practices (Humphrey, Teutsch, & Johnson, 2004).

Currently, there are no studies to our knowledge that have utilised lung cancer risk prediction models to stratify high-risk individuals for chemopreventive interventions; however, in recent years a number of studies have begun to investigate the application of risk models in stratifying individuals for CT screening. Maisonneuve et al. (2011) developed a risk model, designed primarily to quantify individual lung cancer risk based on the individual risk factors used in Bach et al.'s (2003) model, in addition to the factors associated with baseline CT screenings (i.e. lung nodule characteristics and evidence of emphysema), to enable further stratification of high-risk individuals. The study demonstrated that annual CT screening among those identified using the

recalibrated Bach model, as high-risk (predicted annual risk of > 0.3%) and low-risk (predicted annual risk of < 0.3%), resulted in lung cancer detection rates of 90% and 10%, respectively. This model has not, however, been validated using an external population and despite the model being based on age and smoking exposure, little comment is provided with regard to the sensitivity of such an approach (Young & Hopkins, 2012).

More recently, Raji et al. (2012) examined the predictive accuracy of the LLP risk model (Cassidy et al., 2008) for stratifying patients for CT screening. A technique called decision utility analysis was utilised to consider the potential harms and benefits of using the LLP risk model for clinical decision making. This analysis indicated that the model performed better than smoking duration or family history alone in stratifying high-risk individuals for lung cancer CT screening. Furthermore, as previously described, the LLP risk model was utilised to stratify high-risk potential participants as part of UKLS (McRonald et al., 2014). Few studies have been published examining the benefits of stratifying patients for CT screening based on lung cancer risk prediction modelling. Large-scale projects, such as UKLS, may provide future evidence in support of the utilisation of risk models in this context and additionally, may support the establishment of a standardised risk threshold at which to suggest population-based CT screening for lung cancer.

Besides the clinical application, lung cancer risk prediction models could also be useful from a risk communication perspective. Some risk prediction models may be applicable for use within primary care or general population settings, in order to communicate lung cancer risk to the public, thus educating individuals regarding

avoidable risk factors (e.g. smoking) and methods of risk avoidance (e.g. smoking cessation). Hippisley-Cox and Coupland (2011) described how their lung cancer risk algorithm (The QCancer® Lung Algorithm) (previously described), was publically available as a simple web calculator. Furthermore, the feasibility of implementing the algorithm in primary care settings was also explored, although this was aimed at improving diagnostic assessment, rather than communicating risk to patients (Chiang, Glance, Walker, Walter, & Emery, 2015). Y. Chen, Marcus, Niaz, Field, and Duffy (2014) also described the development of a web-based self-assessment tool ("My Lung Risk"), which provided feedback on five-year lung cancer risk based on the original algorithm of the LLP risk model (Cassidy et al., 2008). The web-based self-assessment tool, was designed to provide the public with accessible lung cancer risk information; however, the paper fails to evaluate the efficacy of delivering such risk communication in relation to promoting lifestyle changes to reduce risk. Further research is clearly needed to evaluate the application of lung cancer risk prediction models in health education settings.

1.6 Conclusion

This chapter introduced the PhD project and demonstrated how lung cancer is a dominant and challenging disease to treat, resulting in over 30,000 deaths per year in England and Wales (ONS, 2014c). An extensive number of lung cancer risk factors have been identified and acknowledged within this chapter, with primary focus on those incorporated in the LLP risk model (Cassidy et al., 2008), including age, gender, smoking, occupational exposure, prior malignant disease, prior lung disease, and family history of lung cancer. Among the identified risk factors, smoking is most often attributed to lung cancer development and the synergic effect between smoking and

several other risk factors is also particularly prominent. The extent to which smoking contributes towards the development of lung cancer surpasses that of any other known risk factor and consequently, smoking cessation has been identified as the most effective strategy in addressing the burden of lung cancer.

Various lung cancer risk prediction models were explored within the current chapter. A number of key lung cancer risk prediction models have been developed within the past decade or so, with new and innovative risk models continuing to emerge. Such models traditionally entail the inclusion of epidemiological data, however, more recently, risk models have attempted to integrate clinical and biological data additionally. Although integration of clinical and biological characteristics appears to improve the predictive capabilities of such models, these improvements currently appear limited and can be costly to implement.

Despite several studies acknowledging the application of lung cancer risk models for use in stratifying potential participants or patients for CT screening, a paucity of research was revealed with regards to the application of lung cancer risk prediction models in other health-related domains, such as health education; only one study, to the researcher's knowledge, has acknowledged the potential for the application of lung cancer risk prediction models in health education settings (Y. Chen et al., 2014). The current chapter demonstrated that the LLP risk model (Cassidy et al., 2008) is a particularly appropriate risk model for application within health education settings, especially due to ease of delivery and low implementation costs. The PhD project explores whether the LLP risk model can be applied to promote smoking cessation, however, further understanding of smoking behaviour and risk perception is required

to inform the current project design and to truly appreciate the complexity of the relationship between smoking and risk.

Chapter 2: Smoking and risk

2.1 Introduction

Tobacco is cultivated from two species of tobacco plant named Nicotinana Tabacum and Nicotiana Rustica. The cultivation of tobacco is believed to have originated as early as 5000-3000 BC and it has been suggested that tobacco consumption occurred throughout America by 1492 AD, when Christopher Columbus arrived (Gately, 2001). Presently, tobacco can be consumed in various forms, however, smoked tobacco (i.e. cigarettes) remains the most common form of consumption in the UK since 1919 (Wald & Nicolaides-Bouman, 1991), despite its use declining substantially in recent decades (ONS, 2013b). For example, it was reported that in 1974 most UK males smoked tobacco (51%), whereas in 2011, only 21% reported smoking (ONS, 2013b). It was not until the early 20th century that a growing body of research began to emerge reporting the association between smoked tobacco and smoking-related disease, such as lung cancer (Proctor, 2012).

In recent years, the UK has made commendable efforts in relation to tobacco control policy (Joossens & Raw, 2007) but despite this, smoking-related disease and deaths remain common in the UK to the present day. For example, in 2013, 17% of all deaths among adults aged 35 and over in England were estimated to be associated with smoking (Health and Social Care Information Centre, 2014b). The current chapter explores the literature in relation to smoking and risk; doing so will inform the development of new and innovative tobacco control interventions, which could reduce smoking rates and consequently, smoking-related disease.

This current chapter will firstly explore addiction theory, the prevalence of smoking, smoking aetiology, and smoking-related disease. Factors that influence smoking initiation and common predictors of smoking cessation will also be explored. Exploring smoking behaviour in detail will inform the reader regarding the challenge of reducing smoking rates. This chapter additionally largely focuses on the role of risk perception throughout the smoking lifespan, leading one to consider the contribution of risk perception on smoking initiation and cessation. Tobacco control and policy are discussed and smoking cessation treatments and services are reviewed, which is particularly relevant to the current project, as a sample of Stop Smoking Service (SSS) users were recruited. This chapter will also explore current knowledge pertaining to assessments of risk and harm, especially with regard to lung cancer and lastly, effective risk communication strategies are considered; these aspects are important to incorporate in developing the format by which the Liverpool Lung Project (LLP) risk model (Cassidy et al., 2008) output will be delivered.

2.2 Addiction theory

Addiction is an abstract concept and debate exists as to whether addiction has any general objective structure (Ross & Kincaid, 2010). West and Brown (2013) explain that addiction has been defined in various ways throughout history and many partly overlapping definitions currently remain in the literature. They define addiction as "a chronic condition involving a repeated powerful motivation to engage in a rewarding behaviour, acquired as a result of engaging in that behaviour, that has significant potential for unintended harm. Someone is addicted to something to the extent that they experience this repeated powerful motivation" (p. 15).

The key psychoactive ingredient in smoked tobacco is the stimulant, nicotine, which is typically absorbed through the lungs, bloodstream, and activated in the brain within 15 seconds of inhalation (Attwood et al., 2013); this speed of activation demonstrates why tobacco smoking is a particularly popular choice of tobacco consumption among consumers. Nicotine is the most addictive substance in smoked tobacco and the addictive potential of nicotine is considered high; in line with that of heroin and methadone (West & Brown, 2013).

Nicotine addiction alters behaviour in a complex manner, involving numerous neuronal pathways and neurotransmitters; nicotine inhibits the nicotinic acetylcholine receptors in the central nervous system but also, mesolimbic dopamine, which is a potential neurotransmitter that mediates the reward effect implicated in nicotine use (Jamal, Ameno, Tanaka, Kumihashi, & Kinoshita, 2012). Heishman, Kleykamp, and Singleton (2010) conducted a meta-analysis, which identified that nicotine or smoking contributed towards enhanced motor abilities, attention, and memory. In addition to the conditioning effect of smoking tobacco, Benowitz (2010) described how nicotine addiction occurs as a result of a combination of positive reinforcements, including mood enhancement and withdrawal symptom avoidance. There are a multitude of physical and psychological tobacco withdrawal symptoms, some of which include: tobacco cravings, irritability, anxiety, concentration difficulties, and increased restlessness (Hughes & Hatsukami, 1986); however, the physiological effect of nicotine addiction cannot solely account for the difficulties that many smokers encounter in quitting smoking.

Since the 20th century, numerous new addiction models have emerged as a result of increased knowledge regarding learning, pharmacology, neurophysiology, and neuroanatomy (Westermeyer, 2013). Ross and Kincaid (2010) argue that there is little consensus on the extent to which any one contributing factor can effectively explain aspects of addiction alone and addiction is a multifactorial concept, involving genetics, molecular neurobiological (i.e. biochemical changes at the neuron level), systems neurobiological (e.g. brain regions), picoeconomic dynamics (i.e. sub-personal goalseeking systems, which compete in a manner that explains the individual's whole behaviour), psychological and cognitive processes, and social and cultural influences. They conclude that not one specific model can account for all aspects of the addiction experience. One review, which explored nursing research on smoking cessation published between 1989 and 2008, established that almost half of the studies found were based on explicit formal psychosocial theories or health behaviour models and of these, the Transtheoretical Model of Change (TTM) (J. O. Prochaska & DiClemente, 1983) and the Self Efficacy Theory (Bandura, 1997) were the most frequently used (K. A. O'Connell, 2009). Furthermore, Sutton (2005) suggests that the TTM is one of the most dominant models of behaviour change, which has been applied extensively to smoking behaviour (Atak, 2007; Cahill, Lancaster, & Green, 2010; L. M. Robinson & Vail, 2012).

The TTM proposes and systematically incorporates a number of concepts considered influential to behaviour change, primarily including the stages and processes of change (J. O. Prochaska & DiClemente, 1983; J.O. Prochaska & DiClemente, 1986) (see Figure 2). The TTM stipulates that behaviour change is a dynamic process, which involves progression through five qualitatively distinct stages and the model postulates

that behaviour change is a cyclical, rather than linear process, as individuals may recycle through the stages of change several times before cessation of an addictive behaviour (J. O. Prochaska, DiClemente, & Norcross, 1992). J. O. Prochaska and DiClemente (1984) proposed the five stages, as follows:

- 1. Pre-contemplation, whereby an individual is not currently considering quitting.
- 2. Contemplation, whereby an individual is considering quitting.
- 3. Preparation, whereby an individual is seriously considering quitting within the next 30 days and will prepare to take action to achieve their goal.
- 4. Action, whereby an individual is actively modifying the problematic behaviour.
- 5. Maintenance, whereby an individual strives to prevent relapse and has sustained the action employed to change the behaviour for at least six months.

The TTM postulates that progression through these stages of change is associated with engagement in ten processes of change (J. O. Prochaska & DiClemente, 1983; J.O. Prochaska & DiClemente, 1986). J. O. Prochaska, Velicer, DiClemente, and Fava (1988) stipulate that these processes of change can be either cognitive or behavioural processes. The cognitive processes include consciousness raising, dramatic relief, social liberation, self-re-evaluation, and environmental re-evaluation, whilst the behavioural processes include helping relationships, stimulus control, counterconditioning, reinforcement management, and self-liberation. These processes are defined in greater detail in Appendix A.

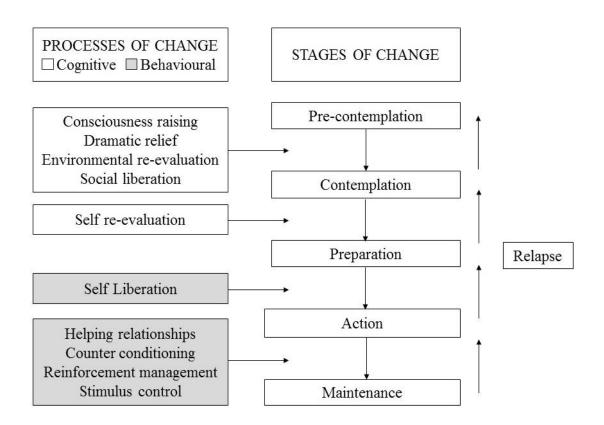


Figure 2. The stages and processes of change stipulated by the Transtheoretical Model of Change (TTM) (J. O. Prochaska & DiClemente, 1983; J.O. Prochaska & DiClemente, 1986)

Despite the common application of the model with regard to smoking (e.g. Atak, 2007; L. M. Robinson & Vail, 2012), researchers have argued that the model is logically flawed in that it uses staging algorithms that are based on arbitrary time periods, it oversimplifies the complexities of behaviour change, and it assumes that individuals generally make coherent and stable plans (Littell & Girvin, 2002; Sutton, 2001; West, 2005). Therefore, the model will not be solely relied upon in regard to informing the current project, although the model provides the reader with an overview of some of the behaviour change mechanisms and the associated strategies potentially employed by smokers. The multifactorial contributors of addiction highlight the complexity and challenge of smoking cessation for so many smokers.

2.3 Epidemiology of smoking

Cigarette smoking varies extensively globally. The World Health Organisation (WHO) (2013) provided data on age-standardised prevalence estimates for cigarette smoking among adults globally in 2011. For males, smoking prevalence ranged from 7% to 67%, whilst for females rates were substantially lower, ranging from 0 to 50%. In the United Kingdom, smoking prevalence was reported at 22% overall. ASH (2014a) provided smoking prevalence statistics in Great Britain by country and stated that among adults in 2012, rates were 20%, 23%, 23% and 24%, in England, Scotland, Wales and Northern Ireland, respectively. As the current study will recruit participants in Liverpool (England) specifically, it was of interest to ascertain smoking prevalence in Liverpool. Smoking prevalence in Liverpool is 24.5%; substantially higher than the average in England (Public Health England, 2014).

Smoking prevalence declined rapidly between the 1970s and 1990s among adults (16 years and over) and continued to fall since the mid-1990s but at a decelerated rate of approximately 0.4% per year (see Table 1) (ONS, 2013b). In addition to smoking prevalence, cigarette consumption per individual has also declined over recent decades in Great Britain. In 1974, average cigarettes smoked per day were estimated to be 18 for males and 14 for females, whereas in 2013, the average number for males was 13 and 11 for females (ONS, 2014a). Furthermore, the number of individuals defined as heavy smokers (those smoking over 20 cigarettes per day) has declined dramatically; 26% of males and 13% of females were defined as heavy smokers in 1974, compared respectively to 5% and 3%, in 2012 (ASH, 2014b).

Table 1. Percentage of adults (16 years and over) who smoke from 1974 to 2012 in Great Britain (Office for National Statistics, 2013b)

Year	Percentage of adults who smoke						
	16-19 years	20-24 years	25-34 years	35-49 years	50-59 years	60 years and over	Total adults
1974	40	48	51	52	51	34	45
1978	34	44	45	45	45	30	40
1982	30	40	38	39	41	27	35
1990	30	39	36	36	35	25	33
1994	30	38	35	34	29	21	27
1998	27	39	32	30	27	17	27
1998	31	40	35	30	27	16	28
2002	31	40	35	31	28	16	26
2006	25	38	34	28	26	15	22
2010	20	31	30	25	22	12	20
2012	19	27	26	24	20	13	20

N.B. Data has been weighted since 1998. Weighted and unweighted data is included for 1988 to display the effect of weighting.

The relationship between smoking and various demographic characteristics will now be explored, with particular focus on sex, age, marital status, ethnicity, and education. ASH (2014b) suggested that there are approximately 10 million smokers in Great Britain. Sex differences have consistently been observed in relation to smoking. For example, the proportion of male adult smokers in Great Britain in 2013, was estimated at 22%, whilst the proportion of females was reported to be 17% (ONS, 2014a).

Smoking prevalence also differentiates by age (see Table 1). Although prevalence is consistently lowest among adults aged 60 and above, individuals within this age group are more likely to be ever-smokers and more likely to have quit smoking (ASH, 2014b). Table 1 suggests that smoking is highest among young people aged 20-24, however, a substantial decline in smoking among young people between 16-19 years

old is also evident from the figures; reflecting the drastic reduction in smoking initiation in recent years.

Differences in smoking status have been identified by marital status (Office for National Statistics, 2014a). In England, the most common marital status overall is Married (46.6%), seconded by Single (34.6%); this is a stark contrast to Liverpool, in which Single is the most common marital status (49.7%), followed by Married (32.3%) (Office for National Statistics, 2014d). Married people are least likely to be current smokers (14%) but more likely to be former smokers (24%), compared with those who are cohabiting (33%) or single (9%). Single people were, however, substantially more likely to be never-smokers (54%), compared with all other groups. These statistics suggest that smoking cessation may be more common among individuals in married relationships and that being single may be a protective factor in relation to smoking initiation.

In terms of ethnicity, the 2011 census revealed that the majority of the general population in England and Wales, classified themselves as White British (80.5%) (ONS, 2012b). White Other was the next largest ethnic group (4.4%), followed thirdly, by Indian (2.5%) and fourthly, Pakistani (2.0%). In contrast, 84.8% of people in Liverpool described themselves as White British, followed by White Other (2.6%), Black African (1.8%) and Chinese (1.7%); cumulatively, 2.5% of residents classified themselves as Mixed (ONS, 2012a). ONS (2011) reported that individuals who described their race as Mixed were most likely to be current smokers (27%), followed by those who were White (21.5%); however, prevalence of former smoker status was highest among White ethnicities (34.3%), followed by Mixed (28.6%). Never smoker

status was highest among Asian or Asian British (75.0%) and secondly, Chinese (71.6%). This information provides insight into the ethnic groups most likely to smoke and suggests that some ethnic minorities may also be at increased risk of smoking.

Smoking is also a health inequalities concern, as smoking rates are higher among lower socio-economic groups in most developed countries (Barnett, Pearce, & Moon, 2009; Cavelaars et al., 2000; A. C. Marcus, Shopland, Crane, & Lynn, 1989). In 2011, in Great Britain, smoking prevalence among those with Managerial and Professional, Intermediate, and Routine/Manual statuses was 13%, 20%, and 28%, respectively ONS (2013b). These findings are particularly relevant in the present project as Liverpool remains the most deprived local authority in England (Liverpool City Council, 2011), which may have contributed towards the aforementioned increased prevalence of smoking in Liverpool, compared to the average in England (Public Health England, 2014).

2.4 Smoking-related disease

Smoking was associated with 17% of all deaths (79,700) in England alone in 2013 and the financial burden of smoking on the NHS has been estimated in England to be £2.7 billion in 2006 (Callum, Boyle, & Sandford, 2011). In Liverpool, smoking related deaths are among the highest rates in England; Liverpool's directly age standardised rate is 456 per 100,000 population, aged 35 years and above, 2010-2012, whereas England's worst level is 480 and average in England is 292 (Public Health England, 2014).

In 2013, it was estimated that smoking was linked with 28% of all cancers (Health and Social Care Information Centre, 2014b). The widely documented relationship between smoked tobacco and lung cancer is discussed elsewhere (see Section 1.3.3). Baan et al. (2009) conducted a review, in which it established a variety of cancers associated with tobacco smoking in addition to lung cancer, including nose and sinus, mouth and upper throat, larynx, oesophagus, liver, pancreas, stomach, kidney, bowel, ovary, bladder, leukaemia, and cervical cancer.

Smoking was also responsible for approximately 35% of all respiratory disease deaths in 2013 (Health and Social Care Information Centre, 2014b). For example, based on random-effects meta-analyses of most-adjusted relative risks (RR) or odds ratio (OR), Forey et al. (2011) found that the effect estimates for ever smokers compared to never smokers were 2.89 (95% CI 2.63-3.17) for chronic obstructive pulmonary disease (COPD) using 129 studies, 2.69 (95% CI 2.50-2.90) using 114 studies for chronic bronchitis, and 4.51 (95% CI 3.38-6.02) for emphysema using 28 studies.

Lastly, in 2013, smoking was attributed to 13% of cardiovascular disease (CVD) deaths (Health and Social Care Information Centre, 2014b). According to Lakier (1992), smoking is the most preventable cause of cardiovascular morbidity and mortality. There is a large body of research from epidemiological studies demonstrating the relationship between smoking and CVD (Kenfield et al., 2008; Preston et al., 2010). A recent study examined the impact of smoking on CVD among males (Ehteshami-Afshar, Momenan, Hajshekholeslami, Azizi, & Hadaegh, 2014). The study recruited 3,059 men (without CVD at baseline) and participants were followed-up for a median of 9.3 years. Former smokers were at increased risk of CVD

events (Hazard Ratio [HR] 2.42, 95% CI 1.28–0.56), although the results suggested that smoking had no effect on coronary heart disease (CHD) events and total/CVD mortality. Current smokers of over 10 cigarettes per day and 20 cigarettes per day were at cumulatively increased risk of CVD/CHD events and total/CVD mortality, although smoking fewer than 10 cigarettes per day was only related to lower levels of increased CVD risk (HR 2.12, 95% CI 1.14–3.95) and its mortality (HR 4.57, 95% CI 1.32–15.79).

Smoking has additionally been associated with other diseases, such as diabetes. The Health Professionals' Follow-up Study displayed that diabetes risk among males was increased among smokers of 25 cigarettes per day or more (RR 1.94, 95% CI 1.25-3.03), in comparison to non-smokers (Rimm, Chan, Stampfer, Colditz, & Willett, 1995). Additionally, one Japanese prospective study followed-up 6,250 non-diabetic males between the ages of 30 and 60 years old for 60,904 person-years and for those who smoked over 30 cigarette per day, they reported an increased risk (RR 1.73, 95% CI 1.20-2.48) (Uchimoto et al., 1999). Another study followed 21,068 US male physicians aged 40-84 for 255,830 person-years and the study reported an increased relative risk for current smokers of 20 cigarettes per day or more (RR 1.7, 95% CI 1.3-2.3), as well as for current smokers of less than 20 cigarettes per day (RR 1.5 (95% CI 1.0-2.2), after multivariate adjustment for body mass index, physical activity, and other risk factors (Manson, Ajani, Liu, Nathan, & Hennekens, 2000). Lastly, Ko, Chan, Tsang, Critchley, and Cockram (2001) examined the relationship between smoking and diabetes in 3,718 Chinese participants. They found that smoking was associated with diabetes in males (OR 1.71, 95% CI 1.11-2.63) but not females. Despite these findings, research has typically reported an association between smoking and diabetes among females (Rimm et al., 1995; Sairenchi et al., 2004).

The aforementioned studies demonstrate the role of smoking in the aetiology of a variety of diseases. The following section will explore contributing factors of smoking initiation and cessation, in order to identify particularly vulnerable groups and to understand the complexity of smoking behaviour; doing so, could potentially inform health interventions and subsequently, reduce the excessive number of smoking-related morbidities and deaths previously described.

2.5 Smoking initiation and cessation

Having explored addiction, epidemiology and smoking-related disease, this section will explore risk factors for initiation and common predictors of smoking cessation. Section 2.5.1 will consider patterns in smoking initiation and various factors, which could lead to some individuals being more vulnerable to smoking initiation. Section 2.5.2 will consider the common predictors of smoking cessation and cessation maintenance; doing so will enable identification of various groups for whom smoking cessation may be more or less challenging. Consideration of such factors will also inform the development of a statistical analysis plan for the current project.

2.5.1 Risk factors for initiation

It has been estimated that approximately 207,000 children aged 11-15 years old start smoking each year in the UK (Hopkinson, Lester-George, Ormiston-Smith, Cox, & Arnott, 2014). The legal age of sale of cigarettes in Britain rose from 16 years old to 18 years old in 2007. Despite the enforcement of such legislation, in 2011 it was

revealed that the majority of British adult current or former smokers reported starting smoking before they were 18 years old (66%), whilst 40% started before the age of 16 (ONS, 2013b). The proportion of children regularly smoking in England has, however, dropped dramatically in recent decades; this has been particularly evident within the past decade, whereby the percentage of regular smokers between the ages of 11-15 years has decreased from 9% in 2003, to 3% in 2013 (Health and Social Care Information Centre, 2014a). Exploration of the predictors of smoking initiation may highlight particular groups more likely to smoke, enabling smoking initiation and consequently, smoking cessation interventions to be tailored towards such groups.

Over recent decades, research has documented a variety of risk factors for smoking initiation: genetic factors; personality; socio-economic status and relationships; media; advertising. In the past decade or so, a number of reviews have documented the association between genetic factors and smoking initiation and maintenance, whilst acknowledging the interaction with environmental factors (Al Koudsi & Tyndale, 2005; Baler & Volkow, 2011; Chatkin, 2006; Davies & Soundy, 2009; M. Munafo, Clark, Johnstone, Murphy, & Walton, 2004; M. R. Munafo & Johnstone, 2008; Russo et al., 2011; Schnoll, Johnson, & Lerman, 2007; Turner, Mermelstein, & Flay, 2004). Genetic variants can impact dopamine receptor functioning and liver enzymes that metabolise nicotine, which in turn may contribute towards increased risk of nicotine addiction (Moolchan, Ernst, & Henningfield, 2000) and it has been estimated that 56% of variance in smoking initiation is accounted for by genetic effects (Sullivan & Kendler, 1999).

Components of personality have also previously been associated with smoking initiation. There are two broad models of personality that are frequently implemented in health research: (1) The Five-Factor Model (FFM) (Costa, Fagan, Piedmont, Ponticas, & Wise, 1992); (2) Eysenck & Eysenck's (1975) Three-Factor Model. The FFM suggests that personality traits are stable behaviours over time and postulates that personality is defined by five specific dimensions, including openness to experience, conscientiousness, extraversion, agreeableness, and neuroticism. Eysenck & Eysenck's (1975) three-factor model was informed by genetics and physiology and suggests that personality differences can be attributed to brain function, stipulating three specific dimensions, including extraversion, neuroticism, and psychoticism (which also entails aggression).

One of the most commonly reported relationships pertaining to smoking initiation and personality relates to the association between sensation-seeking, often incorporated in measures of extraversion and neuroticism. Numerous studies have identified that smokers are more likely to score highly on scales of extraversion, neuroticism, and psychoticism (Ajdacic-Gross et al., 2009; Arai, Hosokawa, Fukao, Izumi, & Hisamichi, 1997; Brook et al., 2008; Cherry & Kiernan, 1976; Forgays, Bonaiuto, Wrzesniewski, & Forgays, 1993; Lipkus, Barefoot, Feaganes, Williams, & Siegler, 1994; Spielberger & Jacobs, 1982; van Loon, Tijhuis, Surtees, & Ormel, 2005). A recent review also concluded that sensation-seeking or risk-taking traits predict smoking but that the evidence for such a relationship is far stronger in self-reports, compared to real-life studies (Bloom, Matsko, & Cimino, 2014). Research also suggests that personality works as a mediator between genetic factors (e.g. increased

extracellular levels of dopamine in the ventral striatum i.e. reward centre) and personality (e.g. the trait of novelty seeking) (Boileau et al., 2003; Leyton et al., 2002).

As described, smoking rates are higher among lower socio-economic groups (ONS, 2013b), which highlights how socio-economic status is often associated with smoking initiation; however, there are a number of factors often associated with socioeconomic status that have also been found to independently relate to smoking initiation. Firstly, educational attainment is one aspect commonly associated with smoking initiation. One review summarised that smoking initiation has consistently been associated with school performance and additional evidence suggests that smoking is related to educational aspirations and commitment to school (Tyas & Pederson, 1998). A more recent study (Gilman et al., 2008) sought to examine whether the relationship between educational attainment and smoking initiation is causal. They explored smoking behaviours among a birth cohort, which began recruitment in 1959 (N = 1311). They found that the number of pack years was higher among people with lower than high school education (RR 1.58, 95% CI 1.31-1.91); however, a repeat analysis, which incorporated having siblings reduced the relative risk considerably (RR 1.23, 95% CI 0.80 -1.93). They subsequently suggested that a large proportion of the education discrepancy in smoking initiation is attributable to aspects shared by siblings.

There is a great deal of literature exploring not only the relationship between having siblings who smoke and smoking initiation, but also the relationship between having parents who smoke and smoking initiation. For example, Leonardi-Bee, Jere, and Britton (2011) undertook a systematic review and meta-analysis to assess the extent

of the effects of having other smokers in the family on smoking initiation. In total, 58 studies were included in the meta-analyses and ORs for smoking initiation in childhood were significantly increased if the child had at least one parent who smoked (OR 1.72, 95% CI 1.59-1.86); however, having a sibling who smoked particularly increased the likeliness of smoking initiation (OR 2.30, 95% CI 1.85-2.86). A number of reviews have further explored the association between smoking among peers and smoking initiation (Bindah & Othman, 2011; Hoffman, Sussman, Unger, & Valente, 2006; Seo & Huang, 2012; Simons-Morton & Farhat, 2010). Bindah and Othman (2011) surmised that there is good supportive evidence that peer smoking is attributed to adolescent smoking behaviour and that adolescents who hold more positive attitudes towards their peers smoking will more likely be influenced to smoke. It has also been suggested that having a teacher who smokes can influence smoking initiation (Wold, Torsheim, Currie, & Roberts, 2004); this is likely to be less problematic within England currently because smoke-free schools guidance has been introduced within England in the past decade, which has become increasingly prominent nationwide and is aimed at both students and staff (e.g. NICE, 2010) and therefore, students should be less likely to observe teachers smoking.

Many studies have attempted to ascertain the impact of tobacco advertising on smoking initiation but the magnitude of the impact can be challenging to assess, due to problematic data and the inability to analyse substantial changes in advertising (Levy, Chaloupka, & Gitchell, 2004). Lovato, Linn, Stead, and Best (2003) undertook a Cochrane review which identified nine longitudinal studies that followed up a total of 12,000 adolescents aged 18 or below, who were not regular smokers at baseline. All studies included assessed smoking behaviour at baseline and follow-up, exposure

to advertising, and receptivity or attitudes to tobacco advertising, or brand awareness at baseline. The review concluded that in all studies, increased awareness of, or receptivity to, tobacco advertising was associated with cigarette experimentation and smoking initiation. This established association is now less of a concern currently in the UK, because although tobacco advertising expenditure in the UK exceeded £100 million per year in the 1980s and 1990s (ASH, 2006), the Tobacco Advertising and Promotion Act was introduced in 2002, which made tobacco advertising illegal, exclusive of point of sale displays. Following this, the 2009 Health Act was introduced, which additionally prohibited tobacco products to be displayed at point of sale; tobacco display prohibition was implemented for large shops from April 2012 and from April 2015 for all other retail outlets (DH, 2011).

2.5.2 Cessation and maintenance of cessation

Recent research suggests that the majority of British current smokers (63-67%) would like to quit smoking (Lader 2009; Robinson and Harris, 2011). Furthermore, 26% of all current smokers are estimated to make at least one serious quit attempt per year (Lader, 2009). Despite these statistics, successful smoking cessation rates are very low. West (2006) estimates that the historic cessation rate in England over the past 40 years is 1.2% of smokers and the cessation rate in England for 2006 was between 2.4-2.8%; however, West (2006) recommends interpreting the data with caution as estimates were based on a number of assumptions and a limited sample size. Smoking cessation is valuable throughout the lifespan; quitting at 60, 50, 40, or 30 years old gains an individual approximately 3, 6, 9, or 10 years of life expectancy, respectively (Doll, Peto, Boreham, & Sutherland, 2004).

Reviewing common smoking cessation predictors will enable exploration of possible predictors or confounding variables in regard to the current project. More generally, doing so could inform the development of effective interventions and strategies to enable the identification of "high-risk" groups that may need more intensive support. Common smoking cessation predictors will be discussed within the following three themes: socio-demographics; smoking behaviour; and psychological factors.

Common socio-demographic variables associated with smoking cessation success, including age, sex and marital status, will now be explored. A number of studies have found that older people are more likely to achieve smoking cessation success at follow-up (Fidler, Brown, Stapleton, West, & Ferguson, 2013; Hymowitz et al., 1997; C. Lee & Kahende, 2007; Monsó, Campbell, Tønnesen, Gustavsson, & Morera, 2001). Abdullah et al. (2006) suggests intention to quit among older smokers is attributed to factors such as having health problems in the past, smoking duration, and cigarettes per day.

Although females typically smoke less than males (ONS, 2014a), females have been found to be less successful in quitting smoking than males. Scharf and Shiffman (2004) conducted a meta-analysis, which found that women are less successful at quitting, regardless of treatment, whilst, further meta-analyses have suggested that these sex differences are evident, particularly in relation to the use of nicotine replacement therapy (NRT) (Cepeda-Benito, Reynoso, & Erath, 2004; Perkins & Scott, 2008). More recently, Vangeli, Stapleton, Smit, Borland, and West (2011) reviewed the evidence and concluded that there was no consistent relationship between sex and quit attempts or quit attempt success. It has been suggested that such

inconsistencies may be due to differences in risk factors for failure between real-life settings and randomised controlled trials (RCTs) (Caponnetto & Polosa, 2008). Nevertheless, Torchalla, Okoli, Hemsing, and Greaves (2011) suggested several underlying mechanisms as to why these sex differences might exist, such as genetic variants, hormonal influences, increased responsiveness to non-pharmacological support among females, increased vulnerability to depression and negative mood among females, weight concerns among females, and receipt of less effective social support among females.

There are also several social factors that have been implicated in smoking cessation success. The smoking status of family or friends can have implications for an individual's quit attempt success. For example, being married to a non- or former smoker has been found to enhance the likelihood of cessation (Coppotelli & Orleans, 1985; Hanson, Isacsson, Janzon, & Lindell, 1990; McBride et al., 1998). There are, however, mixed findings in regard to the relationship between marital status and smoking cessation success, with some studies implicating the positive effect of marriage on smoking cessation (Broms, Silventoinen, Lahelma, Kaprio, & Koskenvuo, 2004; Gourlay, Forbes, Marriner, Pethica, & McNeil, 1994), whilst others found no association (Hagimoto, Nakamura, Morita, Masui, & Oshima, 2010; Hellman, Cummings, Haughey, Zielezny, & O'Shea, 1991; West, McEwen, Bolling, & Owen, 2001; Zhou et al., 2009). Research also suggests that social support behaviour is of great importance to successful cessation. For example, supportive behaviour, including co-operative support or reinforcement of an individual's efforts to stop smoking, has been found to enhance smoking cessation success (Coppotelli & Orleans, 1985; Mermelstein, Lichtenstein, & McIntyre, 1983). Conversely, negative social behaviour, such as nagging, regulating, or complaining about behaviour may increase the likeliness of relapse (Cohen & Lichtenstein, 1990; Roski, Schmid, & Lando, 1996).

Research also suggests that greater affluence is related to smoking cessation success. Kotz and West (2009) found that 20.4% of participants from a higher social grade remained abstinent after one year compared to 11.4% of those from the lowest social grade; several other studies using populations of British smokers have found a relationship between socio-economic predictors and smoking cessation success (Chandola, Head, & Bartley, 2004; Fidler & West, 2011; West et al., 2001). Hiscock, Bauld, Amos, Fidler, and Munafo (2012) suggest that less affluent groups are less likely to successfully quit smoking due to several factors, including inadequate social support, lower motivation, higher levels of addiction, increased exposure to stress or psychological differences, poorer adherence to cessation treatment, and increased vulnerability to tobacco company marketing exposure.

Common smoking-related predictors of cessation success will now be considered, including age at smoking initiation, tobacco dependence, and quit attempts. A number of studies suggest that age at smoking initiation is predictive of smoking cessation success later in life (Breslau & Peterson, 1996; J. Chen & Millar, 1998; Ellickson, McGuigan, & Klein, 2001; Hymowitz et al., 1997; Sadik A. Khuder, Dayal, & Mutgi, 1999; Tucker, Ellickson, & Klein, 2002). For example, Hymowitz et al. (1997) conducted a large cohort study (N = 13,415), whereby smokers between 25 and 34 years old were recruited. The study found that individuals who were over 20 years old

when they started smoking, were more likely to have quit smoking at five-year follow-up (RR 1.16, 95% CI 1.01-1.32).

Smokers who start smoking at an earlier age are also more likely to exhibit increased nicotine dependence (Breslau & Peterson, 1996; Lando et al., 1999; Taioli & Wynder, 1991). Nicotine dependence is typically measured using the Fagerström Test for Nicotine Dependence (FTND) (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991); a reliable measure of nicotine dependence that has been applied extensively across various populations (e.g. Fagerström & Furberg, 2008; Meneses-Gaya, Zuardi, Loureiro, & Crippa, 2009). Research suggests that dependence severity predicts less successful smoking cessation attempts (Breslau & Peterson, 1996; Dale et al., 2001; Fagerström, Russ, Yu, Yunis, & Foulds, 2012; Fidler & West, 2011; Hagimoto et al., 2010; Harris et al., 2004; Killen, Fortmann, Kraemer, Varady, & Newman, 1992; Richmond, Kehoe, & Webster, 1993; Rohde, Kahler, Lewinsohn, & Brown, 2004; Scherphof et al., 2013; Stapleton et al., 1995; West et al., 2001; Zhou et al., 2009). Cigarettes per day (CPD) (a component of FTND) has also been found to predict less successful quit attempts, as individuals who smoke a greater number of cigarettes per day tend to find it more difficult to quit (Haug, Schaub, & Schmid, 2014; Hellman et al., 1991; Hymowitz, Sexton, Ockene, & Grandits, 1991; Li et al., 2010; Li et al., 2011; West et al., 2001).

It has also been suggested that quit attempt history is a predictor of smoking cessation success among adults and adolescents (Andrews, Yeh, Pao, & Horn, 2011; Farkas et al., 1996; Hymowitz et al., 1991; Li et al., 2010; Li et al., 2011; R. P. Murray et al., 2000; Zhou et al., 2009; Zhu, Sun, Billings, Choi, & Malarcher, 1999), as several

studies propose that smokers with shorter previous quit attempts are less likely to quit smoking successfully (Borrelli et al., 2002; Garvey, Bliss, Hitchcock, Heinold, & Rosner, 1992; Hagimoto et al., 2010; Li et al., 2010; Li et al., 2011; Zhou et al., 2009). Furthermore, a number of recent studies have suggested that planning a quit date is a less successful strategy for smoking cessation than spontaneously quitting (Cooper et al., 2010; Hughes & Callas, 2011; Sendzik, McDonald, Brown, Hammond, & Ferrence, 2011); however, Vangeli et al. (2011) advises that quit attempt history is not a consistent predictor of smoking cessation and therefore, the extent to which quit attempt history is indicative of smoking cessation success is unclear.

Various psychological factors, including mental health, alcoholism, and motivation, have also been associated with smoking cessation success. Successful smoking cessation has been found to be inhibited by the presence of mental health disorders, such as major depression and anxiety disorder. Hitsman et al. (2013) conducted a recent systematic review and meta-analysis of 42 trials, which assessed the effect of having past major depression on short and long term smoking cessation. They found that smokers with past major depression were less likely to remain abstinent at three months (OR 0.83, 95% CI 0.72–0.95) and six months (OR 0.81, 95% CI 0.67–0.97). Morrell and Cohen (2006) also conducted a systematic review, in which they concluded that anxiety can reduce smoking cessation success; however, the authors advise that there are few studies to support this hypothesis due to most studies of affect and smoking cessation focusing on depressive symptoms and negative affect, rather than anxiety. One factor which may mediate the effect is the experience of severe withdrawal symptoms among individuals with mental health disorders during the cessation process, such as induced mood disturbances and increased anxiety (Breslau,

Kilbey, & Andreski, 1992; Covey, Glassman, & Stetner, 1997), although the interaction between mental health disorders and smoking cessation is believed to be highly complex (Leventhal & Zvolensky, 2015).

Alcohol misuse is also a factor linked with smoking cessation. Research suggests that although current and former alcohol dependent individuals exhibit greater nicotine dependence and are less likely to quit smoking in their lifetime, smokers with a past history of alcohol misuse are not disadvantaged with respect to quitting smoking on a given attempt (Hughes & Kalman, 2006; Leeman, Huffman, & O'Malley, 2007). Furthermore, greater length of alcohol abstinence has been shown to predict successful smoking cessation (Heffner, Barrett, & Anthenelli, 2007). More generally, the use of alcohol has been linked to increasing urges to smoke among abstinent smokers (Epstein, Sher, Young, & King, 2007; Kirchner & Sayette, 2007). One review suggests that sequential, rather than simultaneous alcohol and smoking treatment programmes are preferable to support behaviour change (Kodl, Fu, & Joseph, 2006).

A number of studies suggest that intention to quit or motivation is a predictor of smoking cessation success (Li et al., 2010; Li et al., 2011). For example, Li et al. (2011) found that smokers who intended to quit smoking within the following month were more than twice as likely to remain abstinent at follow-up (OR 2.18, 95% CI 1.46-3.27); however, results regarding motivation are inconsistent. One review highlighted that although motivation to quit is a strong predictor of quit attempt, evidence suggesting that motivation to quit is a predictor of smoking cessation is weak (Vangeli et al., 2011). Despite this, therapeutic approaches to smoking cessation such as motivational interviewing, an approach that assumes motivation to change is

elicited from the client (Rollnick & Miller, 1995), have been found to increase abstinence rates by 2.3% (Hettema & Hendricks, 2010).

Relapse has been described as a "breakdown or failure in a person's attempt to change or modify any target behaviour" (Marlatt & George, 1984) (p. 261). Polosa and Caponetto (2013) described smoking cessation as "not an event but a process, and relapse is an ordinary component of this process" (p. 118); an estimated 4 out of 5 smokers who make a quit attempt relapse within a month of quitting, whilst it has been estimated that only 3-5% of smokers who have made a quit attempt remain abstinent at 6 months (Hughes, 2007). Traditionally, the literature focuses largely on the initial behaviour change of smoking cessation, as opposed to maintenance of the behaviour change.

In addition to the previously described factors that have been found to be strongly associated with smoking cessation, some clinical factors have been found to influence relapse. For example, one study found that the pharmaceutical intervention, Varenicline has been found to improve cessation rates when examined as a long term maintenance intervention, whereas, other behavioural and pharmaceutical interventions have not been found to be effective in reducing the risk of relapse (Tonstad, Tonnesen, Hajek, Williams, Billing, & Reeves, 2006). A handful of health behaviour models have, however, highlighted the role of maintenance and relapse in the smoking cessation process.

The health behaviour model, the TTM (J. O. Prochaska & DiClemente, 1983; J.O. Prochaska & DiClemente, 1986), was described earlier in this chapter. The TTM

highlights the application of alternate processes of change throughout differing stages of change. This might suggest that certain processes are of greater or lesser importance among individuals at differing stages of smoking cessation. For example, some processes may be of particular importance to individuals initially embarking on a quit attempt, compared to those continuing to maintain abstinence. The model constructs highlight the importance of longer term relapse prevention research, as well as research focusing on initial predictor smoking cessation.

Marlatt & Gordon (1985) specifically developed a model for relapse prevention. The model was developed to teach individuals to effectively anticipate and cope with potential relapse situations, using behavioural skill-training procedures with cognitive intervention techniques. Figure 3 displays the key features of the model and demonstrates how various factors can influence the result of a high-risk situation. The model proposes that immediate determinants (e.g. high-risk situations, coping skills, outcome expectancies, and the abstinence violation effect), as well as covert antecedents (e.g. lifestyle factors and urges and cravings) can influence outcomes. Again, this model will not be solely relied upon in informing the current project, but alongside the aforementioned literature, it does provide insight into understanding smoking cessation and maintenance.

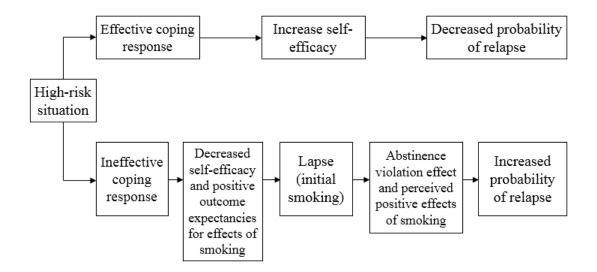


Figure 3. The Relapse Prevention Model (Marlatt & Gordon, 1985)

2.6 Tobacco control in England

Having explored specific groups who may exhibit increased risk of smoking initiation, and having considered potential predictors or inhibitors of smoking cessation, the current section explores governmental efforts to address the aforementioned inequalities and to reduce the financial and health burden of tobacco overall. Section 2.6.1 explores tobacco control policy in England, with particular reference to the white paper, "Healthy Lives, Healthy People: A Tobacco Control Plan for England" (DH, 2011); this section provides an overview of the approaches and strategies implemented by the Government, which aim to tackle smoking prevalence. Following this, Section 2.6.2 explores one of the key tobacco control strategies, which has been widely implemented within the past decade or so, SSS. Section 2.6.2 will provide an overview of SSS and explore pharmacotherapy and behavioural interventions recommended for delivery within such services; exploring these aspects will introduce the setting for the current project, whilst informing the reader of current smoking cessation interventions offered to smokers across England and reported efficacy in relation to such approaches.

2.6.1 Tobacco control policy in England

In 2011, DH (2011) published the white paper, "Healthy Lives, Healthy People: A Tobacco Control Plan for England". The document describes how the Government has set three national ambitions to concentrate tobacco control work as a whole. These include: (1) reducing smoking prevalence among adults in England; (2) reducing smoking prevalence among young people in England; (3) reducing smoking during pregnancy in England. Through the proposed tobacco control plan, the Government pronounced their support for a comprehensive approach to tobacco control in England across six internationally recognised elements.

Firstly, the Government proposed the intention to stop the promotion of tobacco, which entails various sub-components including proposing consultation around the options to reduce the promotional impact of tobacco product packaging (i.e. plain packaging). More recently, increasing steps have been made to introduce regulations for standardised tobacco product packaging (DH, 2014); a recent systematic review (M. Stead et al., 2013) found that although study results were somewhat mixed, standardised packaging reduces the appeal of cigarettes and smoking.

Secondly, the Government announced its intention to make tobacco less affordable through strategies including continuation of the ongoing policy to use tax to sustain the high price of tobacco; one review concluded that there is sufficient evidence for the effectiveness of increased tobacco excise taxes and costs in reducing tobacco consumption and smoking prevalence (Chaloupka, Straif, & Leon, 2011).

Thirdly, the Government detailed intentions to effectively regulate tobacco products. For example, they state how they aim to co-ordinate scientific and market research on the use of nicotine-containing products, such as electronic cigarettes (or e-cigarettes), via the Medicines and Healthcare Products Regulatory Agency (MHRA). The recently approved European Tobacco Product Directive (TPD) subjects e-cigarettes that make medicinal claims regarding smoking cessation or harm reduction and/or products containing above 20mg/ml nicotine, to a medicinal regulatory regime (European Commission, 2014). Products classified as medicinal will be licenced by the Medicines and Healthcare products Regulatory Agency (MHRA) by 2016 (MHRA, 2013).

Fourthly, helping tobacco users to quit was cited by the Government as another priority. This component focuses particularly on the continued provision of Local SSS. The white paper, "Smoking Kills" (DH, 1998) proposed the initiation of Local SSS. SSS in the UK deliver evidence based behavioural and pharmaceutical interventions to support smokers in quitting and such services have proven to be highly effective in reducing smoking prevalence (Ferguson et al., 2005; Judge et al., 2005).

Fifthly, the Government announced intentions to reduce exposure to secondhand smoke, such as encouraging smokers to alter their behaviour, to deter smokers from smoking in family homes or cars, primarily because secondhand smoke is attributed to 165,000 new cases of disease among children each year in the UK (Royal College of Physicians, 2010). Efforts were recently proposed for smoking in cars carrying children to become illegal, this policy was implemented in October 2015 in England (DH, 2014).

Effective communication for tobacco control is the sixth and final element of the recent tobacco control plan. This entails sub-components, such as providing clear and consistent information to support smokers to stop smoking and signposting smokers to resources which may support quitting, such as the NHS smoke-free website (http://www.nhs.uk/smokefree).

This section has provided an overview of current Government tobacco control policy priorities and the comprehensive approach to tobacco control that is adopted. The following section will focus on the aforementioned fourth strand of the "Healthy Lives, Healthy People" tobacco control plan (DH, 2011), "helping tobacco users to quit", of which SSS provision is an important component.

2.6.2 Stop Smoking Services in England

The provision of local SSS in England is a key feature of the current national tobacco control plan (DH, 2011) and a network of services has existed in England since 1999 (DH, 1998). Services aim to support smokers within local communities to quit by providing a range of pharmacotherapy products and behavioural therapies (NICE, 2008) and they have proven to be consistently effective, both nationally (Ferguson et al., 2005; Godfrey, Parrott, Coleman, & Pound, 2005; Judge et al., 2005; West, May, West, Croghan, & McEwen, 2013) and among particularly disadvantaged groups (Bauld, Judge, & Platt, 2007; Chesterman, Judge, Bauld, & Ferguson, 2005). Self-reported four-week quit rates in 2013-14 were 51% overall in services (National Centre for Smoking Cessation and Training [NCSCT], 2014) and it has been estimated that 36,000 premature deaths were prevented due to provision of services in 2012-13

(Bauld, 2014 as cited in NCSCT, 2014). The Smoking Toolkit Study used no medication or support as a reference point to identify that SSS support and medication was a considerably more effective approach to smoking cessation (OR 3.53, 95% CI 2.12-5.88), than medication on prescription (OR 1.73, 95% CI 1.39-2.15), and nicotine replacement therapy (NRT) over-the-counter (OR 0.97, 95% CI 0.81-1.16) (West & Fidler, 2011).

NICE (2008) and the NCSCT (2014) are key sources of service provision guidance for NHS professionals and others who have either a direct or indirect position in SSS provision. Both organisations advise on the use and evidence base of pharmacotherapy products and behavioural support; research regarding these recommendations will now be explored to provide an overview of the SSS settings and treatment approaches adopted.

NICE (2008) recommends the use of a number of pharmacotherapy products, including nicotine replacement therapy, varenicline (Champix, Pfizer), and bupropion (or Zyban). Various forms of NRT are widely available within SSS. A Cochrane review established that NRT overall has been found to be substantially more effective at six-month follow-up compared to non-NRT control groups (RR 1.60, 95% CI 1.53-1.68) (Stead et al., 2012). Furthermore, the review explored whether specific forms of NRT were more or less effective than others. They found that nasal spray was particularly effective (RR 2.02, 95% CI 1.49-2.73), in comparison to tablets/lozenges (RR 1.95, 95% CI 1.61-2.36), inhalers (RR 1.90, 95% CI 1.36-2.67), patches (RR 1.64, 95% CI 1.52-1.78), and gum (RR 1.49, 95% CI 1.40-1.60). Lastly, the review revealed that combination of a slow-release nicotine containing product (i.e. nicotine patch)

with a fast-delivery form of NRT (e.g. inhalers) was a substantially more effective treatment approach than use of a single NRT product (RR 1.34, 95% CI 1.18-1.51).

Varenicline (or Champix) is a non-nicotine drug, which binds to high affinity and selectivity of the $\alpha 4\beta 2$ neuronal nicotinic acetylcholine receptor, acting as a partial agonist; thus, craving and withdrawal symptoms are alleviated and reinforcing reward effects of smoking are reduced by preventing nicotine binding to the receptors (NICE, 2007). A recent Cochrane review assessed the efficacy of varenicline, measuring sixmonth abstinence (Cahill, Stead, & Lancaster, 2012). Based on 14 trials, the pooled relative risk for sustained abstinence at standard dose compared to placebo was 2.27 (95% CI 2.02-2.55). Mills et al. (2012) also conducted a meta-analysis, which explored the efficacy of varenicline. They found that at three months, varenicline was more effective than placebo (RR 2.26, 95% CI 1.69-3.02) or standard dose NRT (RR 1.29, 95% CI 1.11-1.49), yet at six months, varenicline remained more effective than placebo (RR 2.24, 95% CI 1.75-2.88) but it was not significantly more effective than standard NRT (RR 1.18, 95% CI 0.95-1.48). It has previously been theorised that varenicline may impact mood and suicidal ideation, as it interacts with the central nervous system and dopamine release (Hays & Ebbert, 2008; Hughes, 2008); however, a recent systematic review concluded that there is consistent evidence among the more valid study designs, that varenicline either does not contribute towards increased suicide rates or if it does, the effect is extremely minimal (Hughes, 2015).

Bupropion (or Zyban) is an anti-depressant drug, which is believed to enhance smoking cessation by inhibiting dopamine re-uptake and reducing the effect of nicotine on nicotinic acetylcholine receptors (Wilkes, 2008). Bupropion has been

found to be significantly effective in enhancing smoking cessation success. One Cochrane review revealed how the pooled analysis of 44 trials suggested that bupropion used independently is significantly more effective than placebo for long term abstinence (RR 1.62, 95% CI 1.49-1.76), yet they concluded that there was insufficient evidence to support the use of bupropion alongside NRT (Hughes, Stead, Hartmann-Boyce, Cahill, & Lancaster, 2014). Another Cochrane review suggested that bupropion and NRT were equally effective (RR 1.01, 95% CI 0.87-1.18) but combination of bupropion and NRT achieved significantly better abstinence rates (RR 1.24, 95% CI 1.06-1.45) (Stead et al., 2012); however, bupropion has been found to be overall less effective for smoking cessation than varenicline (Cahill et al., 2012; Hughes et al., 2014).

The NCSCT (2014) suggests that the most effective treatment method involves combination of smoking cessation pharmacotherapy and behavioural support provided by SSS, such as individual behavioural counselling, group behaviour therapy, and telephone support. NICE (2008) provide recommendations regarding individual behavioural counselling and group behaviour therapy. They suggest that individual counselling should involve scheduled face-to-face appointments between smoking counsellor and smoker, which typically involves weekly sessions over a minimum of four weeks following the quit date. With regard to group behaviour therapy, they suggest that therapy should comprise of scheduled meetings in which smokers receive information, advice, support and some form of behavioural intervention, such as cognitive behavioural therapy. As recommended for individual support, NICE also suggest that group behavioural therapy should be offered for at least the first four weeks following a quit date.

A number of studies have assessed the efficacy of individual and group support for smoking cessation. Lancaster and Stead (2005) conducted an analysis which incorporated 22 trials of individual behavioural therapy. They found that individual counselling was more effective than control in enhancing smoking cessation at long-term follow-up (RR 1.39, 95% CI 1.24-1.57); however, no difference in effect was displayed between intensive and brief individual counselling (RR 0.96, 95% CI 0.74-1.25). A Cochrane review on group therapy was also undertaken, in which 13 trials were included in an analysis of group therapy compared with self-help (Stead & Lancaster, 2005). The authors revealed that group support was significantly more effective than self-help (RR 1.98, 95% CI 1.60-2.46), yet no significant difference in effectiveness was detected between group therapy and individual counselling; nevertheless, one large study (N = 126,890) found that group therapy was significantly more effective than individual support within SSS (OR 1.43, 1.16-1.76) (Brose et al., 2011).

NCSCT (2014) dichotomise group therapy into open (or rolling) and closed group programmes. Open (or rolling) group programmes enable smokers to join a programme spontaneously, resulting in a group of smokers at varying stages of their cessation attempt, whereas closed group programmes require smokers to start the programme simultaneously, encouraging service users to continue attending sessions for a specific number of weeks. NICE (2008) fail to provide guidance on the format of group therapy, although a recent study (N = 202,084) which used individual behaviour as a reference point found that open group programmes were most effective

at 4-week quit (OR 1.26, 95% CI 1.12-1.41), compared to closed group programmes (OR 1.11, 95% CI 0.98-1.26) (Hiscock et al., 2013).

The NCSCT (2014) categorises telephone support into proactive telephone support, reactive telephone support and text-based support. Telephone support (or Quitlines) typically entail providing support and encouragement to smokers who intend to quit or are actively quitting. Proactive telephone counselling involves smoking counsellors contacting the client, whereas reactive telephone counselling involves the client contacting the service (NICE, 2008). A Cochrane review into telephone counselling for smoking cessation found that cessation success was higher among smokers who had received a number of sessions of proactive telephone counselling (RR 1.37, 95% CI 1.26-1.50) (Stead, Hartmann-Boyce, Perera, & Lancaster, 2013). The authors also suggest that there is evidence to support the efficacy of reactive telephone counselling but that it is limited in comparison, as fewer studies have been conducted in this area of research, presumably because reactive telephone counselling RCTs would entail a control group of callers, who would be refused telephone support.

One strategy adopted by some SSS, although not currently recommended by NICE (2008), is the use of text-messaging to support service users. Y. F. Chen et al. (2012) suggested that the use of computer and other electronic aids can enhance prolonged abstinence rates, in comparison to no intervention or generic self-help materials (RR 1.32, 95% CI 1.21-1.45). Kong, Ells, Camenga, and Krishnan-Sarin (2014) conducted a narrative review of the literature regarding text-message support for smoking cessation. Of the studies they included, all used motivational messages, which were grounded in either socio-cognitive behavioural theories, behaviour change techniques,

and/or individually tailored messages. One pooled analysis, which included 9,000 participants concluded that mobile-phone based interventions are significantly associated with improved six-month outcomes, compared to control programmes (RR 1.71, 95% CI 1.47-1.99) (Whittaker et al., 2012).

2.7 Introducing risk perception

Having considered the literature regarding smoking and tobacco control, the remaining focus of the current chapter is risk. Risk may be regarded as "uncertainties: possibilities, chances, or likelihoods of events, often as consequences of some activity or policy" (Taylor-Gooby & Zinn, 2006, p.1); however, risk is not solely considered in terms of statistical probabilities but, it is explored extensively in psychosocial research and practice. We have always been exposed to risks but previous to modernity, risks were typically viewed as a result of non-human factors, such as natural disasters such as famine or earthquakes. Modernity (characterised by technological advancement and the development of governing institutions) has consequentially resulted in exposure of other risks, such as nuclear/chemical warfare or newly discovered diseases, leading our "risk society" to become increasingly aware and fearful of risks (Beck, 1992). Indeed, the term risk is now used in every day and professional discourses to denote danger or hazard (Douglas, 1992).

Douglas (1992) also emphasised the shift towards a blame culture, in line with modernity, whereby any occurrence of death, accidents or misfortune is attributed to someone or something. Furthermore, Lupton and Tulloch (2002) described how within Western societies risk avoidance is linked closely with the ideal of the "civilised" body, an increasing willingness to seize control over one's life, rationalise and regulate

the self and body, and ultimately, avoid the vicissitudes of fate. The ideal of the civilised body links closely with cognitive theory regarding health risk behaviour. For example, the Health Belief Model (HBM) (Rosenstock, 1966) is one of the most dominant theories in health behaviour research (Carpenter, 2010) and stipulates that health beliefs (e.g. perceived susceptibility of disease) predict health behaviour engagement. It has been argued that such approaches represent human behaviour in a computational manner, whereby risk avoidance is considered rational and risk-taking is viewed as irrational (Bloor, 1995 as cited in Lupton, 1999). Douglas (1992) further argues that such approaches depict humans as "hedonic calculators calmly seeking to pursue private interested. We are said to be risk-aversive, but alas, so inefficient in handling information that we are unintentional risk-takers; basically we are fools." (p. 13). Similarly to addiction theory, not one specific theory or model can account for interpretation and responses to risk and therefore, the current thesis will explore risk by incorporating a range of perspectives, as discussed.

Risk perception is the subjective, individualised assessment of risk. It is conceptualised as a multi-factorial construct, integrating perceived vulnerability, unrealistic optimism, and precaution effectiveness (Borrelli, Hayes, Dunsiger, & Fava, 2010). Perceived vulnerability reflects the perceived likelihood that an individual will suffer health consequences as a result of engaging in the risk behaviour (Gerrard, Gibbons, Benthin, & Hessling, 1996; Gerrard, Gibbons, & Bushman, 1996; Weinstein, 1999; Weinstein, Rothman, & Sutton, 1998). Unrealistic optimism refers to the inaccurate underestimation of the occurrence of a negative event, relative to other people in similar circumstances (Weinstein & Klein, 1996). Lastly, precaution

effectiveness denotes the perceived likelihood that engaging in a precautionary behaviour will result in benefit to health (Weinstein, 1988).

Cognitive dissonance theory has received substantial attention in the literature and may provide some explanation as to why some smokers quit and many continue to smoke despite the risks. In the context of smoking, cognitive dissonance can occur when a smoker holds at least two opposing but related cognitions (or perceptions), which can result in mental conflict (Festinger, 1957, 1962). Consequentially, a smoker will act in various ways in an attempt to reduce dissonance and mental conflict. For example, a heavy smoker may become increasingly aware of lung cancer risk, creating dissonance between willingness to smoke and fear of disease. The smoker may attempt to reduce cognitive dissonance in a number of ways, such as stopping smoking or rationalising or justifying continued smoking (e.g. being critical of a risk communication source). A number of studies have suggested that smokers reduce cognitive dissonance by rationalising or justifying smoking, which in turn has been associated with continued smoking and relapse behaviour (Dijkstra, 2009; Fotuhi et al., 2013; Gibbons, Eggleston, & Benthin, 1997; Halpern, 1994; Jenks, 1992; Kleinjan, van den Eijnden, Dijkstra, Brug, & Engels, 2006; Kleinjan, van den Eijnden, & Engels, 2009; McMaster & Lee, 1991; Peretti-Watel, Halfen, & Grémy, 2007; C. A. Robinson, Bottorff, Smith, & Sullivan, 2010).

The following sections aim to explore what makes some risks more acceptable than others, whether risk perception predicts smoking initiation, and how influential risk perception is in smoking maintenance and cessation. Understanding these components

will enable us to inform the development of effective risk communications; thus, preventing smoking initiation and promoting smoking cessation.

2.7.1 Factors implicated in risk perception

Whether an individual perceives a risk as threatening or not ultimately affects how the individual reacts towards the risk and the degree to which an individual perceives a risk as threatening can rely upon several factors, including voluntariness, controllability, familiarity, sociocultural factors, and the media. A pioneering paper by Starr (1969) detailed the evaluation of risk acceptability, by comparing risk information with risk behaviour. Starr suggested that people are prepared to accept voluntary risks approximately 1,000 times greater than involuntary risks; therefore, if an individual perceives a risk as voluntary (e.g. smoking), the risk is likely to be more acceptable compared with an involuntary risk (e.g. chemical exposure). Voluntary risk-taking is also associated with risk exposure benefit. Voluntary risk-taking has been described as engagement in a behaviour that is perceived as risky, yet undertaken intentionally and by choice (Lupton & Tulloch, 2002). Lupton (1999) describes how risk-taking can also be reported more positively and that to some, voluntary risk-taking may enhance a sense of personal agency, opportunity to discover a communal spirit with other like-minded individuals, and can provide "temporary liberation from routine constraints" (p. 166). This highlights how risks may not only be perceived as something disastrous or threatening, but to some, a risk may also be viewed as potentially beneficial.

The concept of voluntariness links closely with controllability of risk. Compared with other health beliefs, perceptions associated with control have often been found to contribute largely to variance when tested across a number of health behaviour theories (e.g. Godin & Kok, 1996). People will often perceive a risk as more threatening if the risk is perceived as uncontrollable, whilst a risk that is perceived as controllable is likely to be perceived as less threatening (Slovic, 2000a). For example, individuals tend to view their risk as lower if they are the driver of a car (and perceive themselves to be in control), rather than the passenger in a car (potentially perceiving themselves to be out of control) (McKenna, 1993). Sjoberg (2000) also describes how perceived personal risk is typically lower compared to perceived risk of others' risk. Since it is not possible for the majority of us to be below average risk, this would suggest that many of us are unrealistically optimistic about our personal risk. The concept of unrealistic optimism in relation to smoking cessation is described further in Section 2.7.3.

Familiarity or habituation is also an important factor with regard to risk perception. Risks perceived as new or unknown are often perceived as more threatening (e.g. E. C. Smith, Burkle, & Archer, 2011). Being exposed to a risk over time can result in perceiving the risk to be less threatening, despite the objective level of risk remaining the same (Slovic, 2000a); this illustrates the role of habituation, which is particularly relevant to smoking, as smoking can become routine, mundane and ultimately habitual. Furthermore, Lupton (1999) suggests that risk responses may not occur on a conscious level, despite models of rationality emphasising the role of conscious behaviour. For example, a passenger may automatically fasten a seat-belt as a response to habituation, as opposed to a response of rational risk avoidance.

Sociocultural variables have also been found to have a profound impact on risk perception. Douglas and Wildavsky (1982) contend that risk perception is itself a social process and they critique cognitive approaches to risk, arguing that such approaches fail to acknowledge social influence. For example, research suggests that white males consistently perceive risks as lower compared to other demographic groups (Finucane, Slovic, Mertz, Flynn, & Satterfield, 2000); this finding has been attributed to socio-political factors rather than biological factors. Risk perception has also been found to differentiate across socio-economic status. Lupton (1999) highlights how white males from particularly affluent groups more often perceive risks generally as less threatening; in considering smoking, however, recent research suggests that those from the most deprived households tend to perceive smoking-related health risks as less threatening (Peretti-Watel, Seror, et al., 2014). It has been argued that insecurity and risk is concentrated among the poorest in society and since access to material resources determines action, deprivation may ultimately implicate risk acceptance or avoidance (A. Jones, Abbott, & Quilgars, 2006).

Mass media might also play a role in the formation and adaptation of risk perception. It is challenging to escape media in contemporary society; we consistently seek or are exposed to information from various media sources, including the Internet, newspapers, television, and radio. Wahlberg and Sjoberg (2000) reviewed the evidence for a relationship between media and risk perception. They summarised that media is not a strong causal influence of risk perception, particularly personal risk perception; however, they do suggest that increased media exposure may affect risk perception but that the effects are weakened by impersonal impact. The media has also been accused of misrepresenting health threats. Bomlitz and Brezis (2008) argue that

emerging health threats are over-reported in mass media, compared with common threats. For example, the very limited outbreak of Ebola in America received an arguably disproportionate amount of media coverage for a number of weeks following the first imported case (Towers et al., 2015), especially when compared with other common, ongoing threats, such as smoking. With regard to smoking specifically, a more recent review suggested that the media plays a significant role in forming perceptions towards smoking among youths (Wakefield, Flay, Nichter, & Giovino, 2003) and the media has been suggested as one of the primary sources of cancer risk related knowledge among smokers (Dowding, 2006).

This section has identified various factors, which may impact the extent to which a risk is perceived as harmful. The following two sections will explore the literature in relation to the impact of risk perception on smoking behaviour, specifically smoking initiation and smoking cessation. In exploring these concepts, the reader will be provided with a more in-depth understanding of the role of risk perception in smoking behaviour and potentially, considering such aspects will aid the development of the LLP intervention.

2.7.2 Risk perception and smoking initiation

As the vast majority of smokers start smoking prior to the age of 18 years old (ONS, 2013b), much of the literature regarding risk perception and smoking initiation tends to focus on initiation among children and adolescents. Determining whether perceptions of risk are important in smoking initiation is important; if young people's perceptions of risk do not relate to smoking initiation, health campaigns focused towards educating young people about the harms of smoking may be ineffective. Furthermore, individuals who are more dismissive regarding the risks of smoking at

the point of initiation, might potentially be less receptive to risk communication encouraging smoking cessation at a later stage.

Earlier research argued that young people are aware of the risks. For example, Viscusi (1990) found that young people greatly overestimate the effect of smoking on lung cancer, indicating that young people are particularly informed of the risks associated with smoking. In response to these findings, Jamieson and Romer (2001b) suggested that despite such findings, a large proportion of smokers are dissatisfied with their decision to start smoking; thus, arguably invalidating claims that young people are fully informed of smoking-related risks. They highlight that the first Annenberg Tobacco Survey results revealed that although again, young people tend to overestimate lung cancer risk, far fewer overestimate smoking-related mortality.

A number of surveys have also found that smoking youths or those susceptible to smoking tend to underestimate the effect of smoking on health, compared to non-smoking youths or those less susceptible (Aryal, Petzold, & Krettek, 2013; Halpern-Felsher, Biehl, Kropp, & Rubinstein, 2004; Nichter, Nichter, Vuckovic, Quintero, & Ritenbaugh, 1997; Romer & Jamieson, 2001; Slovic, 2000b; Virgili, Owen, & Sverson, 1991). For example, Romer and Jamieson (2001) found that young non-smokers were more likely to perceive the risk of lung cancer as high (79.1%), compared to smokers (69.6%); however, Greening and Dollinger (1991) found no difference in perceived risk estimates regarding cancer, emphysema, and stroke between smokers and non-smokers.

The second Annenberg Tobacco Survey revealed that health risk perceptions predict smoking among 14-22 years old youths who believed that smoking cessation is more difficult to achieve and disease onset happens sooner, yet this was not apparent among young people who did not hold the aforementioned views (Gerking & Khaddaria, 2012). A. V. Song et al. (2009) conducted a longitudinal study in which risk perceptions would be considered as continuous variables. They found that individuals who held the lowest perceptions of long-term and short-term smoking-related risks were approximately 3.64 and 2.68 times more likely to start smoking at follow-up, respectively, compared to those who perceived the risks as highest. Another longitudinal study, which followed participants (N = 477) over three years, also found that perceptions towards health and safety predicted smoking initiation (Gerrard, Gibbons, Benthin, et al., 1996).

Despite the aforementioned findings, the literature suggests that perceptions of addiction may be stronger predictors of smoking initiation. Young people tend to overestimate their ability to quit smoking and underestimate the magnitude of addiction. One study suggests that young people are more likely to believe that they can smoke for a few years and then quit (60%), compared to adults (48%) (Arnett, 2000). O'Loughlin, Karp, Koulis, Paradis, and DiFranza (2009) recruited 877 participants and found that non-daily smokers at baseline, were more likely to become daily smokers at follow-up if they exhibited inflated perceptions of personal mental and physical addiction. Notably, Okoli, Richardson, Ratner, and Johnson (2009) recruited youths (N = 5051) and found that those who perceived greater personal mental and physical addiction were identified as being more susceptible to smoking,

despite participants being non-smokers. This suggests that potentially non-smokers form predisposed ideas around addiction and self, even before they begin to smoke.

Lastly, it should also be noted that the relationship between health risk perceptions and smoking initiation may also be mediated by other factors, such as tobacco product options. For example, Brennan, Gibson, Momjian, and Hornik (2015) found that 13% and 23% of young people viewed menthol cigarettes as less harmful and addictive, respectively, compared with regular cigarettes. Furthermore, lower perceptions of harm were associated with intention to use.

2.7.3 Risk perception and smoking cessation

Understanding the role of risk perception in smoking maintenance and cessation is important in the development of effective smoking-related risk communications. A review of the literature regarding motivating factors that contribute towards smoking cessation found that smoking cessation was primarily predicted by concerns for health (McCaul et al., 2006), suggesting that health risk perceptions may play an important role in the process of smoking cessation. A number of studies have detailed how health risk perceptions in regard to smoking may be associated with variables including age, sex, ethnicity, education, smoking behaviour (e.g. cigarettes per day), and family history of disease (Ayanian & Cleary, 1999; Borrelli et al., 2010; E. J. Hahn, Rayens, Hopenhayn, & Christian, 2006; Strecher, Kreuter, & Kobrin, 1995).

In comparison to their non-smoking counterparts, current smokers have been found to typically acknowledge personal risk of smoking-related disease as higher (Chassin, Presson, Sherman, & Kim, 2002; Eisinger, 1971; A. Hahn & Renner, 1998; E. J. Hahn

et al., 2006; Honda & Neugut, 2004; Horwitz, Hindi-Alexander, & Wagner, 1985; C. Lee, 1989; McKenna, Warburton, & Winwood, 1993; Peretti-Watel et al., 2007; Reppucci, Revenson, Aber, & Dickon Reppucci, 1991; Rise, Strype, & Sutton, 2002; Rutten, Blake, Hesse, Augustson, & Evans, 2011; Strecher et al., 1995; Weinstein, Marcus, & Moser, 2005; Williams & Clarke, 1997; Zlatev, Pahl, & White, 2010). For example, Rutten et al. (2011) undertook a survey (N = 1765) and identified that personal perceived lung cancer risk was most often perceived as "very high" by current smokers (15.2%), followed by former (1.9%) and never smokers (1.6%). They also established, however, that current smokers more often reported feeling reluctant to get checked for lung cancer, in case they had it (23.4%), compared to former (13.3%) and never smokers (9.8%). Furthermore, 18.4% of current smokers described themselves as worrying about lung cancer all of the time, compared to 3.1% and 1.8% of former and never smokers, respectively. This research suggests that although current smokers acknowledge and worry about their increased risk, they may exhibit some denial behaviours e.g. avoidance of screening, due to concerns around disease acquisition.

A review by Weinstein (1998) concluded that smokers consistently acknowledge that smoking is responsible for increased health risks, yet they tend to regard the level of risk as lower in comparison to non-smokers. The review also highlighted how smokers underestimate personal health risks when considered relative to other smokers, potentially demonstrating unrealistic optimism (Weinstein & Klein, 1996). In recent decades, the domain of unrealistic optimism has received a great deal of attention in relation to smoking. Smokers are also more likely to underestimate the role of smoking in the acquisition of disease (Arnett, 2000; Chapman, Wong, & Smith, 1993; C. Lee, 1989; Peretti-Watel et al., 2007; Reppucci et al., 1991; Ruchlin, 1999) and also more

likely to emphasise the likelihood that disease occurs due to factors other than smoking behaviour, such as chance or genetic factors (Chapman et al., 1993; Horwitz et al., 1985; Rutten et al., 2011; Santos, Tonstad, Montgomery, Paalani, & Faed, 2011; Weinstein et al., 2005).

A number of studies have also demonstrated how smokers exhibit substantial differences between how they perceive personal risk and general risk (Dillard, McCaul, & Klein, 2006; A. Hahn & Renner, 1998; McKenna et al., 1993; Reppucci et al., 1991; Rise et al., 2002; Segerstrom, McArthy, Caskey, Gross, & Jarvik, 1993; Strecher et al., 1995; Weinstein et al., 2005; Williams & Clarke, 1997; Zlatev et al., 2010). More specifically, Weinstein et al. (2005) conducted a survey (N = 6369) consisting of current, former and never smokers. They found that 37.9%, 43.2% and 47.1% of current, former, and never smokers, respectively, correctly estimated lung cancer survival to be below a quarter, suggesting that current smokers are unrealistically optimistic in their perceptions around lung cancer survival. Furthermore, the results suggested that despite absolute lung cancer risk increasing substantially with cigarette consumption per day, perceptions of relative and absolute lung cancer risk were similar across variations in cigarette consumption. Again, this suggests that smokers are either unaware of the reported increase in lung cancer risk in line with cigarettes per day, or consumers of greater numbers of cigarettes per day may underestimate the strength of this relationship.

Unrealistic optimism has independently been found to be associated with other myths and beliefs about personal smoking. Dillard et al. (2006) compared objective and personal perceived risk, to stratify unrealistic optimists from a sample of smokers (N

= 377). They found that individuals who were unrealistically optimistic regarding personal lung cancer risk were more likely to believe that a greater number of lung cancers are cured and less likely to identify smoking cessation as a means of reducing lung cancer risk or to have plans to quit smoking. It has been suggested that when forming relative risk perceptions, smokers often characterise the behaviour of an abstract individual, a "risk stereotype", whereby estimates of smoking duration, cigarettes per day and cigarette nicotine content are considered and incorporated (A. Hahn & Renner, 1998). A. Hahn and Renner (1998) suggested that smokers use these risk stereotypes when evaluating their relative personal risk; this could provide some explanation as to why smokers consistently underestimate comparative risk, as they have created an extreme example for comparison to themselves.

The majority of the aforementioned research focuses on exploring differences in risk perception by smoking status. More in-depth exploration of risk perception throughout the process of smoking cessation is required to further establish the causal role of risk perception. Chapman et al. (1993) found that a number of self-exempting risk perceptions (e.g. "Many smokers live until a ripe old age so it's not that bad for you") were significantly more prevalent among individuals pre-contemplating smoking cessation, compared to those in the action stage of behaviour change, as stipulated by the TTM (see Section 2.2). E. J. Hahn et al. (2006) found that lung cancer risk perceptions were positively related to stage of change, although recent former smokers classified in the highest stage of change (maintenance), perceived their risk as lower than that of current smokers. Furthermore, Borrelli et al. (2010) found that individuals in the preparation stage of change perceived vulnerability of disease as higher and exhibited lower unrealistic optimism compared to those in preceding stages of change.

Again, this suggests that those embarking on smoking cessation may be more realistic in their judgements of risk and the consequences of smoking, compared to precontemplators.

Generally, research suggests that perceived risk increases with intention to quit (Borland et al., 2009; Dillard et al., 2006; Magnan, Koblitz, Zielke, & McCaul, 2009; Norman, Conner, & Bell, 1999; Williams & Clarke, 1997), although much of these findings are based on cross-sectional surveys and a number of studies have failed to find a strong relationship (Segerstrom et al., 1993; Umeh & Barnes, 2011). Several studies have also suggested that risk perceptions predict quit attempts (Borland et al., 2009; Jacobson, Catley, Lee, Harrar, & Harris, 2014; Jamieson & Romer, 2001a) but again, a number of studies failed to find an association (Norman et al., 1999; Segerstrom et al., 1993).

Arguably, the strongest evidence for an association between risk perception and smoking cessation, may derive from longitudinal studies that include a measure of cessation. Borland et al. (2009) found that risk perceptions were associated with intention to quit and making a quit attempt, yet they found no consistent predictive effect on sustained smoking cessation. Despite this, a small number of recent studies have found an association between health risk perceptions and smoking cessation. Among medically ill smokers (N = 237), increases in future perceived vulnerability have been found to predict continuous abstinence at two-months follow-up (OR 3.39, 95% CI 1.09-10.55), whilst unrealistic optimism was also found to relate to continued smoking (Borrelli et al., 2010). Hayes and Borrelli (2013) conducted a study in which they dichotomised Latino smokers into light and heavy smokers (N = 131). They found

that for heavier smokers, health beliefs significantly predicted abstinence at two and three month follow-ups, but only among heavier smokers. Most recently, Jacobson et al. (2014) explored risk perception among college students who smoked (N = 243) and found that higher perceived vulnerability to disease at baseline predicted smoking cessation at 3-month follow-up (OR 1.41, 95% CI 1.15-1.72). These longitudinal studies demonstrate some promising results, although there is clearly a limited number of well-designed longitudinal studies exploring the impact of risk perceptions on smoking cessation.

In summary, research suggests that risk perceptions differ by smoking status and that smokers typically underestimate their personal risk, when comparing themselves to other smokers. The majority of the research also suggests that risk perceptions are associated with stages of change, intentions to quit, and quit attempts; however, there are few longitudinal studies to support the notion that risk perception influences smoking cessation specifically. Attempts to explore this concept may have been hampered by the fact that risk perceptions are challenging to measure; they entail the fluid assessment of risk and can be influenced by many factors, at any time. Despite this, the risk perception literature will have undoubtedly influenced the inception of many risk communication tools aimed at enhancing smoking cessation. In general, the literature suggests a relationship between risk perception and smoking cessation. The following section will explore the literature in relation to smoking-related risk communication, as the delivery of communications based on objective assessments of smoking-related risk and harm, could undermine unrealistic optimism and potentially, enhance smoking cessation success.

2.8 Risk communication

This section will explore risk communication, processing of risk communication, recommendations around communicating smoking-related risk, and previously investigated assessments of risk and harm in relation to smoking. Consideration of such aspects is essential when informing the design and implementation of a new intervention.

2.8.1 Processing risk communication

Risk communication has been described as "any purposeful exchange of information about risks between interested parties" (World Health Organisation, 2001) (p. 317). It is an essential, instrumental and purposeful process, which is typically conceptualised by a one-way flow process consisting of a sender, a message, and a receiver, but it can also sometimes include additional components, such as total understanding by the receiver and feedback to the communicator (Corcoran, 2007).

Although risk perception can provide some explanation as to why individuals may engage in behaviour change, risk perception cannot solely account for behaviour change influenced by risk communications. There are various theories and models that have been used to describe the influence of risk communication on health behaviour and therefore, this section will provide a brief overview of three key health behaviour theories and models: (1) The Health Belief Model; (2) The Protection Motivation Theory; (3) The Extended Parallel Processing Model.

The Health Belief Model (HBM) was based on work by Rosenstock (1966) and developed by Becker (1974). The model emphasises the role of cognitions in

processing risk information and describes how such constructs can influence behaviour change. Risk perception holds a key role in the model, as perceived seriousness and susceptibility of disease are said to influence perceived threat of disease, which in turn contributes towards likeliness of engagement in behaviour change. This would suggest that informing a smoker of their increased lung cancer risk may influence how threatening they perceive smoking-related risk to be, which in turn, may influence smoking cessation. Although the HBM has been described as one of the most dominant theories in health behaviour research (Carpenter, 2010), its main components have been criticised for exhibiting weak effect sizes and its predictive capacity has been demonstrated to be limited in comparison to that of other heath behaviour models (Harrison, Mullen & Green, 1992; Zimmerman & Vernberg, 1994).

The Protection Motivation Theory (PMT) was developed by Rogers (1975; 1983). The model aims to provide understanding regarding the mechanisms by which fear appeals are processed. Fear appeals have been described as "communications presenting the threat of impending danger to motivate compliance with a proposed recommendation" (p. 233) (Keller & Lehmann, 2008, as cited in Orazi & Pizzetti, 2015). The model proposes that intentions to engage in a behaviour change, as a result of exposure to a risk message, rely upon a number of factors: (1) perceived threat severity (e.g. lung cancer); (2) perceived probability of the occurrence (e.g. personal perceived risk of developing lung cancer); (3) perceived response efficacy (e.g. perceived extent to which a preventive behaviour, such as smoking cessation, might reduce lung cancer risk), and; (4) perceived self-efficacy (one's own perceived confidence in undertaking the preventive behaviour). The HBM and the PMT differ primarily in the way the models are organised. More specifically, the PMT is organised into two processes: (1)

threat-appraisal processes, and; (2) coping-appraisal processes. The PMT has been applied extensively to smoking cessation (Farhangmehr, Jalali, & Silva, 2015; Pechmann, Zhao, Goldberg, & Reibling, 2003; Smerecnik, Quaak, van Schayck, van Schooten, & de Vries, 2011; Wright, French, Weiman, & Marteau, 2006; Yan et al., 2014); however, unlike the HBM, the PMT does not account for additional environmental and cognitive factors (e.g. social norms).

One model that has particular relevance in the context of the current project is the Extended Parallel Process Model (EPPM) (Witte, 1992; 1994). The EPPM integrates aspects of previous theoretical perspectives, such as The Protection Motivation Theory (Rogers, 1975) and danger control/fear control framework developed by Leventhal (1970). The EPPM suggests that cognitions contribute to fear appeal success (e.g. changes in attitudes and behaviour) via danger control processes, whilst the emotion fear contributes to fear appeal failure (e.g. defensive avoidance) via fear control processes. Within the model, perceived threat of the risk and perceived efficacy of risk communication response are both particularly important components in processing risk communication. A small number of studies have also successfully explored smoking-related risk communication within the framework of the EPPM (Emery, Szczypka, Abril, Kim, & Vera, 2014; Gharlipour et al., 2015; Gould, Watt, Cadet-James, Clough, 2015; Greening, 1997; Ho, 1992; Wong & Cappella, 2009; Wright, French, Weiman, & Marteau, 2006).

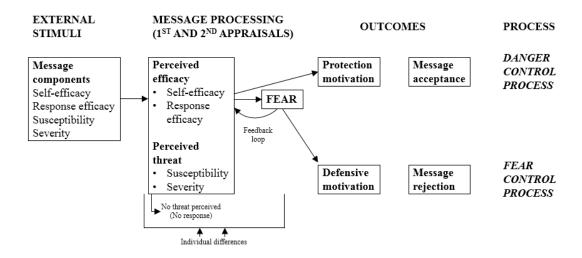


Figure 4. The Extended Parallel Processes Model (Witte, 1992; 1994)

2.8.2 Optimising smoking-related risk communication

The manner in which risk communication is presented is crucial and can influence the extent to which perceptions of risk affect behaviour change (Ahmed, Naik, Willoughby, & Edwards, 2012). Various factors attributed to risk communication presentation can contribute towards risk communication salience, including numerical format, the use of absolute versus relative risk, the use of visual imagery, message framing, and individualising communications. As the current project intends to transform statistical output from the LLP risk model (Cassidy et al., 2008) into a format appropriate for a general population, guidance pertaining to effective delivery of risk communications should be considered.

One of the challenges in delivering effective risk communication is the difficulty that a vast proportion of society has in comprehending numbers and statistics (Gigerenzer, Gaissmaier, Kurz-Milcke, Schwartz, & Woloshin, 2007). Poorer health literacy has been associated with higher nicotine levels, more positive attitudes towards smoking, less knowledge regarding smoking related risks, and lower risk perceptions

(independent of socio-economic status) (Stewart et al., 2013). Furthermore, Lipkus, Samsa, and Rimer (2001) demonstrated how even highly educated participants (N = 463) are frequently unable to correctly answer fairly simplistic numeracy questions; for example, only 15-21% correctly recalled that 1 in 1000 was the equivalent of 0.1%. This raises concerns regarding how numerical risk information is communicated to the public. One review recommended that health risk communication should be consistent in the use of numerical formats, use the same numeric denominator, and present round numbers rather than decimals, if possible (Lipkus, 2007). Reyna and Brainerd (2008) also highlights the importance of ratio bias, a phenomenon whereby higher frequencies bias probability judgements, such as 1 in 10 being perceived as fewer than 10 in 100, despite the probabilities being equal. This should also be incorporated into the presentation of numerical risk estimates among smokers.

Another influential factor regarding risk communication salience is the use of absolute versus relative risk estimates. An earlier review suggested that presenting relative risk reduction information is much more persuasive than providing absolute risk reduction information (Adrian Edwards, Elwyn, Covey, Matthews, & Pill, 2001). Subsequent research suggested that relative risks are easily misinterpreted and information should preferably be communicated using absolute probabilities (O'Doherty & Suthers, 2007). More recently, a Cochrane review was undertaken, which evaluated the effect of statistical presentation of information for the same risks on risk understanding (Akl et al., 2011). The review concluded that there was no significant difference in risk understanding between absolute and relative risk reduction (Standardised Mean Difference [SMD] 0.02, 0.39-0.43), although relative risk was perceived to be larger (SMD 0.41, 0.03-0.79) and more persuasive (SMD 0.66, 0.51-0.81).

Another favourable attribute of risk communication is the incorporation of graphical information, such as charts, graphs, and illustrations. Lipkus and Hollands (1999) emphasised how graphical imagery can enhance the understanding of numerical risk and suggested that graphical imagery possesses at least three attributes for effective risk communication: (1) graphics can convey patterns in data which may have otherwise gone unnoticed; (2) specific types of graph could evoke specific mathematical operations; (3) graphs may attract and sustain attention, unlike statistics, potentially. Hill et al. (2010) conducted three focus groups (N = 37) consisting of both health practitioners and consumers. They explored 16 formats of the same risk information and established that graphical formats, the use of colour to represent risk, and comparative risk information (e.g. smoking status), were preferable risk communication attributes.

The manner in which a risk message is framed has also been found to be substantially important in the communication of risk. A recent review assessed the most appropriate methods of framing risk messages with regard to smoking cessation (Toll et al., 2014). The authors concluded that loss-framed messages (e.g. "Smoking will harm your health by causing problems like lung cancer") are less effective than gain-framed messages (e.g. "Quitting smoking will benefit your health by preventing problems like lung cancer") in promoting smoking cessation. This is supported by the results of a recent meta-analysis, which incorporated 198 effect sizes from 94 studies (Gallagher & Updegraff, 2012). The results of the correlation analysis suggested that gain-framed messages were more likely to encourage prevention behaviours than loss-framed messages (r = 0.083, p = 0.002). The analysis incorporated a range of behaviours,

however, sub-analysis including only smoking cessation studies also found the result to be particularly significant (r = 0.198, p < 0.001). If applicable, it has also been suggested that messages should be framed in a number of ways (e.g. the chance of developing cancer, the chance of not developing cancer) (O'Doherty & Suthers, 2007).

Lastly, one of the most promising approaches to smoking cessation interventions for entire populations are tailored (or personalised) risk communications (Velicer et al., 2006). Tailored risk communications can take many forms, such as providing personalised feedback concerning risk status or genetic vulnerability to disease, and assessing readiness to change behaviour (Gerrard, Gibbons, & Reis-Bergan, 1999). A recent Cochrane review, suggested that personalised risk information was associated with increased informed choice, increased knowledge, more accurate risk perceptions, and increased likelihood of screening uptake (A. Edwards et al., 2013).

2.8.3 Assessments of smoking-related risk and harm

Providing smokers with individualised information in relation to markers of harm may be helpful to undermine smokers' unrealistic optimism, resulting in enhanced motivation to quit (Young et al., 2010). Assessments of risk or harm in relation to smoking and focusing on lung cancer risk, might include lung cancer screening, measurement of exhaled carbon monoxide (CO), lung function, or genetic susceptibility to lung cancer. This section will evaluate whether undertaking assessments of risk and harm can influence smoking cessation or risk perceptions.

Screening for lung cancer using low-dose computed tomography (CT) has been suggested as a means of reducing lung cancer deaths through the early detection and

treatment of lung cancers (J. K. Field, Oudkerk, Pedersen, & Duffy, 2013); however, a number of studies have also explored the impact of screening on smoking behaviour. A number of studies have attempted to identify whether participation in a lung cancer screening programme is associated with increased motivation to quit smoking (Gomez & LoBiondo-Wood, 2013; Munshi & McMahon, 2013; Poghosyan, Kennedy Sheldon, & Cooley, 2012; Slatore, Baumann, Pappas, & Humphrey, 2014). The results of such studies suggest that it is unclear whether undergoing screening is associated with smoking cessation as findings are inconsistent, although smoking cessation has been associated most often with screening results per se (e.g. abnormal scans) and overall screening trial participation. A number of studies suggest that individuals who participate in lung cancer screening trials or express interest in them, typically have increased perceptions of lung cancer risk (E. J. Hahn et al., 2006; E. R. Park et al., 2014), which could explain the increased rate of smoking cessation among trial participants.

Various forms of biomedical risk assessment have also been examined in relation to smoking cessation. A Cochrane review was undertaken to establish the effect of biomedical risk assessment on smoking cessation, which included fifteen trials (Bize et al., 2012). The authors highlight the scarcity of high quality studies and suggest that there is no evidence to confirm that feedback in relation to exhaled CO measurement (RR 1.06, 95% CI 0.85-1.32) or spirometry (RR 1.18, 95% CI 0.77-1.81) in primary care, are effective aids in promoting smoking cessation; however, these pooled results were based on only two trials each. Of the 11 remaining trials, two were statistically significant; one study assessed the effect of lung age feedback (RR 2.12, 95% CI 1.24-3.62), whilst the other evaluated the effect of feedback on smoking cessation in regard

to ultrasonography (i.e. an ultrasound-based diagnostic imaging technique) of specific arteries (RR 2.77, 1.04-1.74).

Since Bize et al. (2012) undertook the aforementioned Cochrane review, a number of other studies have been undertaken, providing further insight into the role of biomedical risk assessment in promoting smoking cessation. In relation to expired CO feedback, few studies have subsequently been published. Ripoll et al. (2012) published a protocol for a clinical trial, which will examine the efficacy of exhaled CO feedback for smoking cessation but the results are not yet apparent. Furthermore, Grant, Ashton, and Phillips (2014) undertook qualitative interviews with SSS users (N = 23) and found that almost all participants who received expired CO feedback found the information motivational, although most participants in this study were former smokers. This suggests that feedback pertaining to exhaled CO may be motivational for recent former smokers continuing to abstain, as low readings may reinforce behaviour change, although it is unclear whether current smokers in receipt of higher readings may be motivated by such information; the literature currently would suggest little effect, if any.

Recent findings in relation to the effect of providing smokers with spirometry or "lung age" information have also been limited. One small scale study explored the effect of providing "lung age" information on smoking cessation among a sample of injection drug users (N = 100) (Drummond et al., 2014). They found no significant difference in cessation between the intervention and control groups, although the sample size was substantially low. The protocol for a larger study (N = 444) has been published (Irizar-

Aramburu et al., 2013), which aims to measure the effect of spirometry feedback on smoking cessation but results have yet to be published.

The aforementioned Cochrane review (Bize et al., 2012) included only three trials that involved delivering smokers risk information pertaining to genetic susceptibility of lung cancer. A review undertaken at a similar time included seven studies and identified a short-term effect of genetic notification on short-term smoking cessation (RR 1.55, 95% CI 1.09-2.21) (de Viron et al., 2012). One recent study required smokers to watch an educational video regarding the tumour suppressor protein p16 and subsequently provided participants with results of a test for methylated p16 presence in sputum (an indicator of lung cancer risk) (Shofer et al., 2014). The test failed to predict changes in lung cancer risk perceptions or cessation at follow-up, although the sample was substantially low (N = 35). Kammin, Fenton, and Thirlaway (2014) conducted a survey among SSS users in the UK (N = 139) and identified that service users expressed an interest in learning about their personal genetic susceptibility of lung cancer and reported that a high-risk result would increase motivation to quit. Lastly, a large-scale study protocol has been published, which also aims to establish the effect on smoking cessation of providing a gene-based test (Nichols, Grob, de Lusignan, Kite, & Williams, 2014). Although again results have been inconsistent in relation to the provision of gene-based risk information for smokers, this is a developing area of research that has shown some promise.

It should lastly be noted that previous literature in this area has focused upon the effect of communicating lung health risk on the initial act of smoking cessation, as opposed to smoking cessation maintenance; therefore, future research should endeavour to explore the effect of such risk communication on smoking cessation maintenance also, as smokers in various stages of behaviour change may employ differing cognitive and experiential processes to facilitate change (e.g. J. O. Prochaska & DiClemente, 1983; J.O. Prochaska & DiClemente, 1986). Potentially, some risk communication methods maybe more appropriate for smoking cessation, compared with smoking cessation maintenance, and vice versa.

2.9 Conclusion

Within recent decades, tobacco control policy and practice in the UK has resulted in substantial advances in relation to smoking initiation and cessation rates. In particular, SSS provide an effective and accessible service, aimed at enhancing smoking cessation rates throughout England; despite this, smoking cessation rates in SSS have remained similar for the past three years, demonstrating the need for new and innovative interventions.

The literature suggests that health risk perceptions may predict smoking initiation and cessation, although further research is needed, particularly longitudinal studies. Furthermore, research suggests that unrealistic optimism regarding smoking-related risk perception could be undermined by the delivery of personalised smoking-related risk communication; this could potentially lead to increased smoking cessation rates.

This review of the literature regarding smoking and risk contextualises the current project and informs the reader of present findings regarding the domains relevant to the PhD project. Furthermore, this literature review aids aspects of the methodology

section, including the development of the LLP intervention and the statistical analyses plans, which consider common smoking cessation and risk perception predictors.

Chapter 3: Methodology

3.1 Introduction

This chapter details the project development and methodology of the PhD project. Firstly, the chapter will describe the study aims and objectives, as informed by the literature discussed in the preceding chapters. The chapter will subsequently specify the development of the PhD project intervention (or the Liverpool Lung Project [LLP] intervention). The LLP intervention was developed using the LLP risk model (Cassidy et al., 2008) and involves calculation and communication of projected lung cancer risk, based on both smoking and non-smoking behaviour. Subsequently, the research design and ethical considerations are described and justified. Participants, design, procedure, and analysis plans for both the quantitative and qualitative research components are subsequently discussed. Finally, data analysis preparations are considered prior to the results chapters.

3.2 Aims and objectives

The primary aim of the thesis is to examine whether the LLP intervention (based on the LLP risk model) can be used to promote smoking cessation rates among Stop Smoking Service (SSS) users. Secondary aims of the thesis include investigating whether application of the LLP intervention influencesfollow-up lung cancer risk perception, exploring other associations with smoking cessation, and improving understandings regarding smoking-related risk perception and communication. The objectives of the thesis are as follows:

1. To investigate whether application of the LLP intervention is associated with smoking cessation success and lung cancer risk perceptions.

- 2. To explore factors implicated in smoking-related risk perceptions.
- 3. To explore factors implicated in smoking cessation success.

By conducting this study, it is anticipated that the results will build upon current knowledge pertaining to smoking cessation, risk perception, and risk communication but additionally, the results could have important implications for tobacco control. For example, if the LLP intervention is found to be associated with follow-up smoking cessation success, it could potentially be implemented within various SSS. Implementation of an effective smoking cessation intervention would reduce smoking rates and in turn, reduce the burden of smoking-related disease and deaths.

3.3 LLP intervention development

Advanced statistical techniques are required to calculate lung cancer risk using the LLP risk model (as detailed in Cassidy et al., 2008) and in its standard form the output from the model is not appropriate for delivery among the general population. As described in the previous chapter, presentation is crucial in developing effective methods of risk communication (e.g. Ahmed et al., 2012).

The aim in developing the LLP intervention, was to produce a risk communication tool, which incorporated an individualised (A. Edwards et al., 2013), gain-framed message (Toll et al., 2014), which could be framed in a number of ways (O'Doherty & Suthers, 2007). Furthermore, it was preferable for the intervention to communicate risk, whilst avoiding decimals (Lipkus, 2007) and incorporating imagery, to enhance the understanding of numerical risk (Lipkus & Hollands, 1999). It was therefore necessary to develop the LLP risk model output via several phases.

The screenshots of the user interfaces included within this section reflect the details of an example participation cases. This example case considers a 43 year old male, with 33 years smoking duration and a previous diagnosis of pneumonia, with no previous malignancies, family history of lung cancer, or previous asbestos exposure (it is noteworthy that although smoking initiation at 10 years old seems unusual, this example information was a based on the details of a real study participant in the PhD project. The current project required the calculation and provision of output from the LLP risk model, among hundreds of participants with varying histories, in a time-limited setting and therefore, it was essential to develop a means of achieving this objective. A desktop database was developed by an IT technician based within the LLP, using the program, Microsoft Access. This database incorporated the risk calculation formulae associated with the model (for details, see Cassidy et al., 2008). The original user interface for the database presented five-year lung cancer risk by incorporating the LLP risk model formulae (see Figure 5).

Despite the development of this database easing risk calculations, presentation of the output in this format was deemed unhelpful to participants, as it failed to demonstrate the benefit of quitting smoking. The user interface was therefore developed further to enable presentation of projected risk at age 70 years, by comparing two scenarios: (1) continued smoking; (2) continued abstinence. This second user interface again, incorporated the LLP risk model formulae but manipulated the output to enable presentation of five-year projected risk scores at age 70 (see Figure 6).

Age:		43	
Gender	Gender		•
Pneumonia	Pneumonia		•
Asbestos		No	•
Previous Cancer		No	•
Family History		No	•
Years smoked:		33	
	Calculate		
Result:		0.75	

Figure 5. The original risk calculator user interface

	Now		Risk at age 70 - If Continued smoking		Risk at age 70 - If quit smoking now	
Age:	43		70		70	
Gender	Male	•				
Pneumonia	Yes	•				
Asbestos	No	•				
Previous Cancer	No	•				
Family History	No	•				
Years smoked:	33		60		33	
			Calculate			
Risk Score			12.19		5.23	2

Figure 6. The second risk calculator user interface

Risk projections were projected at the age of 70 years among all participants for several reasons. Firstly, the researcher aimed to ensure that all participants received consistently presented results, therefore avoiding the undesirable effects of extraneous variables. Secondly, life expectancy in Liverpool is considerably low. In the UK, overall average life expectancy for males and females has been estimated at 79 and 83 years, respectively (WHO, 2015); whereas, in Liverpool, average life expectancy for males and females has been estimated at 75 and 79 years, respectively (ONS, 2012c). The researcher aimed to select an age preceding life expectancy, yet requiring an age old enough to exhibit a substantial level of risk if the participant continued to smoke, as almost 90% of lung cancer cases occur in those above age 60 (ONS, 2013a). This also ensured that the project inclusion criteria regarding age, refrained from focusing simply on older participants, as all participants could be provided with risk projections based on their current circumstances.

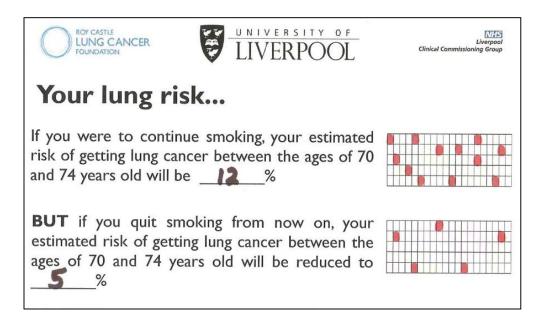


Figure 7. The finalised LLP intervention

The second user interface presented addressed the design issues associated with the original user interface, however, the format was not appropriate for delivery among a general population visually. Output acquired from the user interface was therefore presented to participants in a more simplistic and visually appealing manner. The finalised LLP intervention (see Figure 7) achieved the objectives of clarifying the numerical estimates by avoiding decimals (Lipkus, 2007) and incorporating imagery to enhance the understanding of numerical risk (Lipkus & Hollands, 1999). Lastly, the LLP intervention could be delivered by a researcher, who would also provide verbal risk communication, reiterating the results and answering participant questions.

3.4 Research design and justification

A mixed methods research design was adopted in order to achieve the project objectives, which involved incorporation of both quantitative and qualitative approaches. Johnson, Onwuegbuzie, and Turner (2007) suggested that mixed methods research has become increasingly identified as the third key research paradigm, alongside quantitative and qualitative research. Variation exists in relation to the definition of mixed methods research; however, Tashakkori & Creswell (2007b, as cited in Creswell & Plano Clark, 2011) described mixed methods research as "research in which the investigator collects and analyses data, integrates the findings, and draws inferences using both qualitative and quantitative approaches or methods in a single study or a program of inquiry" (p. 4).

Pragmatism is often considered the primary epistemological approach with regard to mixed methods research and several authors have identified pragmatism to be the most appropriate justification for adoption of such a research design (Johnson et al., 2007).

For example, a pragmatic approach stipulates that the research question should take precedence over the research methodology and the underpinning research paradigm, meaning that the research question ultimately dictates the research methodology (Tashakkori & Teddlie, 2010). A pragmatic perspective was adopted by the researcher in the design of the current project. The primary research objective for the current project was defined prior to determining the appropriate research methodology and underpinning research paradigm; consequently, the research question has informed the mixed methods design, which primarily aims to address the primary research objective.

Some researchers contend that it is inappropriate to mix methodologies due to fundamental differences between philosophical paradigms, yet it is widely argued that combining research methods is appropriate in various settings (Teddlie & Tashakkori, 2010). In relation to tobacco control research, it has been suggested that mixed methods research can be highly beneficial and may capitalise upon the strengths of both quantitative and qualitative research methods (Mathie & Carnozzi, 2005). Mathie and Carnozzi (2005) identified several motives for undertaking mixed methods tobacco control research, including triangulation, complementarity, conceptual development, and expansion. Triangulation involves applying the same research question across differing methodologies, to verify findings or biases used. Complementarity refers to how the results of one method may be elaborated, illustrated or clarified by the results of an alternate method. Expansion involves the implementation of mixed methods to increase the scope and depth of the research. The mixed methods approach adopted in the current project incorporated a quantitative exploration of the predictors of smoking status and lung cancer risk perceptions

(including an investigation of the LLP intervention effect), alongside a complementary and expansive qualitative investigation, whereby factors implicated in smoking cessation success and smoking-related risk perception were explored.

3.5 Quantitative research methods

The quantitative component of the research was designed to address all current project objectives, although the primary objective dictated the quantitative research design. In order to address the primary objective (i.e. to investigate whether application of the LLP intervention is associated with smoking cessation success and lung cancer risk perceptions) a randomised controlled trial (RCT) design was adopted. The effect of the LLP intervention was tested by randomly allocating participants to one of two groups: a control group; or, an intervention group. Following collation of baseline measures, all participants were followed-up in an identical manner. RCTs are considered the gold standard for assessing intervention efficacy, as they can eliminate selection bias (Moher et al., 2010). Data was collected at baseline and six months, in line with recommendations on best practice (West, Hajek, Stead, & Stapleton, 2005). The subsequent sections will provide comprehensive detail regarding the appropriate power calculation, participants, materials, procedure, and piloting for the quantitative component of the research.

3.5.1 Sample size calculation

Prior to the project implementation, a sample size calculation was generated using the computer program, G * Power (Faul, Erdfelder, Buchner, & Lang, 2009; Faul, Erdfelder, Lang, & Buchner, 2007). This calculation incorporated the anticipated smoking cessation success rate in control arm; the six-month smoking cessation rate

of 26% was estimated and communicated by Roy Castle Fagends SSS (RCFE) (Appendix B). Furthermore, a 10% difference in smoking cessation rate between control and intervention groups was considered, as guided by the literature (Parkes, Greenhalgh, Griffin, & Dent, 2008). The resulting power calculation indicated that a sample size consisting of 673 current smokers was required to detect a 10% difference (using χ^2 test), considering 80% power for a 5% two-sided type 1 error. Lastly, it should be noted that this calculation incorporated 1:1 randomisation allocation.

Quit rates are typically measured for SSS at 4-weeks and 12-months and these have been reported at 51% (NCSCT, 2014) and 15% (Ferguson et al., 2005); however, one study identified 12-month quit rates for RCFE, at 21% (Owen & Springett, 2004). Local correspondence with RCFE informed the power calculation but regional differences should be noted. Furthermore, smoking cessation trials of a similar design utilised more general populations than the current study, which may also influence the difference that the power calculation aimed to detect. Both aspects should be considered when interpreting the results.

3.5.2 Participants

Between May 2013 and December 2013, a total of 521 participants between the ages of 18-60 years were recruited via 55 RCFE community drop-in sessions. Host locations for the drop-in sessions varied but included: GP surgeries; libraries; SureStart children's centres; churches; council services; hospitals; sports centres; and community centres. Appendix C displays a full list of the drop-in centres in Liverpool at the time recruitment commenced, as an example. The finalised inclusion criteria for

the project required participants to be between 18-60 years old, to have no previous diagnosis of lung cancer, and to be engaged with RCFE.

It was originally intended for current smokers only to be recruited and followed up at six months; however, it became apparent at the beginning of the recruitment phase that it would not be possible to recruit the estimated 673 current smokers required to achieve appropriate statistical power (see Section 3.5.1). This challenge was primarily due to the lack of current smokers available in RCFE drop-in sessions, partly attributed to the delivery of a rolling programme, consisting of both current and recent former smokers. It was subsequently decided to recruit both current and recent former smokers as two separate RCTs and two respective datasets; however, all participants were followed up at six months. By adopting this design, the impact of the LLP intervention on follow-up smoking cessation success could be analysed among baseline current smokers, as well as baseline recent former smokers, albeit using limited sample sizes.

The initial sample of baseline current smokers consisted of 302 participants, whereas, the initial sample of baseline recent former smokers consisted of 219 participants; throughout the thesis, separate datasets for baseline current and recent former smokers are therefore referred to.

Recent former smokers were defined as individuals who reported abstinence for at least seven days but remained engaged with SSS. Figure 8 demonstrates the distribution of quit duration across the sample of recent former smokers recruited at baseline (N.B. Figures do not equate to 219 due to some missing data). The median

number of days abstinence among the sample was 39.0 days (IQR = 21.0-75.0), with a minimum and maximum reported number of seven and 600 days abstinence, respectively. Quit duration varied among the samplebut it was recorded wherever possible and controlled for throughout the quantitative analyses (e.g. regression modelling).

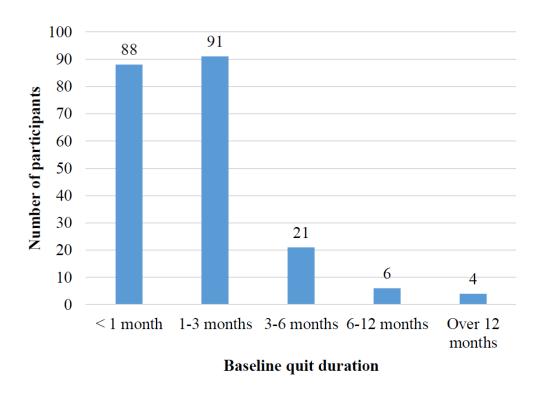


Figure 8. Self-reported baseline quit duration among recent former smokers

3.5.3 Materials

The design of the project materials was carefully considered. Printed project materials, included the participant information sheet (PIS) (Appendix D), consent form (Appendix E), baseline questionnaire (Appendix F), control leaflet (Appendix G), LLP intervention (see Figure 7), follow-up questionnaire (Appendix H), and a follow-up

letter (Appendix I). The questionnaires entailed several measures, which will be detailed later in this chapter (see Section 3.5.3.1).

In developing the project materials, the researcher considered published recommendations on questionnaire development (Kelley, Clark, Brown, & Sitzia, 2003; Rattray & Jones, 2007); doing so, would encourage service user involvement in the research, ease of questionnaire completion, and enhanced questionnaire reliability and validity. Questionnaire content, layout, and the use of consistent graphical imagery (e.g. inclusion of the University of Liverpool logo), were all key considerations. Pens and stamped addressed envelopes were also required to enable participants to complete the study and return questionnaires, in cases where telephone follow-ups were not possible.

With regard to technological equipment, a password-protected laptop was required during recruitment, to enable the timely calculation of lung cancer risk projections based on the LLP risk model. Furthermore, an encrypted USB drive was utilised throughout recruitment to store a secure record of participants recruited, avoiding participant recruitment duplication. A telephone was required for questionnaire follow-ups.

Several computer software packages were required to complete the project. The LLP database, "SB" was used to store participants' details and the RCFE database, "Quit with us" was used to access additional, relevant service user information, with participant consent. University of Liverpool survey software was utilised to ensure that the follow-up questionnaire was accessible online via the URL, http://survey.liv.ac.uk/smoking-study. Microsoft Access and Excel were installed on

the project laptop. Microsoft Access facilitated the development and use of a risk calculator, which incorporated formulae based on the LLP risk model. Microsoft Excel was used to develop and securely store a record of participants recruited. Lastly, the computer program IBM-SPSS statistical software version 21.0 (New York, NY) was utilised to support data analyses.

3.5.3.1 Questionnaire measures

The baseline questionnaire (Appendix F) and follow-up questionnaire (Appendix H) included a wide range of measures; however, this section will focus on the measures of interest to the current project (see Table 2) and these measures cover three broad areas: (1) socio-demographics; (2) smoking behaviour; (3) lung cancer risk perceptions.

Measures of interest pertaining to socio-demographics, included age, gender, ethnicity, marital status, highest educational attainment, and socio-economic status (using postcode). All of these measures (except socio-economic status) were taken from the established LLP questionnaire and have been previously validated (Cassidy et al., 2008). Postcodes were collated to establish English Index of Multiple Deprivation (IMD) ranks, which represent socio-economic status. IMD is a robust index that uses 38 separate indicators, based on weighted data from seven domains: income deprivation (22.5%); employment deprivation (22.5%); health deprivation and disability (13.5%); education, skills and training deprivation (13.5%); barriers to housing and services (9.3%); crime (9.3%); and, living environment deprivation (9.3%) (Deas, Robson, Wong, & Bradford, 2003). IMD information was obtained using a website developed by Mimas at the University of Manchester (Mimas, 2014).

IMD data are typically reported as ranks within quintiles, based on national population data: quintile 1 (most deprived) = IMD ranks 1-6,496; quintile 2 (above average deprivation) = IMD ranks 6,497-12,993; quintile 3 (average) = IMD rank 12,994-19,489; quintile 4 (below average deprivation) = 19,490-25,986; quintile 5 (least deprived) = IMD rank 25,987-32,482 (e.g. McRonald et al., 2014). These quintiles will therefore be referred to in relation to socio-economic status.

Table 2. Questionnaire variables of interest in the current project

Variable	Data type		
<u>Demographics</u>			
Age	Continuous		
Gender	Categorical		
Ethnicity	Categorical		
Marital status	Categorical		
Highest educational attainment	Categorical		
Socio-economic status (using postcode)	Categorical		
Smoking characteristics			
Smoking status (7-day point prevalence)†	Categorical		
Quit duration †	Continuous		
Age started smoking	Continuous		
Living with another smoker	Categorical		
Nicotine dependence	Continuous		
Cigarettes per day	Continuous		
Lung cancer risk perceptions			
Perceived personal lung cancer risk †	Ordinal		
Perceived average smoker lung cancer risk †	Ordinal		
Perceived relative risk of lung cancer †	Ordinal		
Lung cancer worry †	Ordinal		
Perceived lung cancer survival	Ordinal		

[†]Repeated measure

Measures of interest relating to smoking behaviour, included smoking status (i.e. 7-day point prevalence [PP]), quit duration (applicable to former smokers only), age started smoking, living with another smoker, nicotine dependence, and cigarettes per day. Smoking status was measured using 7-day PP. Seven-day PP is commonly used in smoking cessation trials and has a number of advantages. Velicer and Prochaska (2004) suggested that 7-day PP can be advantageous, as measurement at six or 12

months can include smokers who delayed action and quit, capturing the dynamic, reallife process of smoking cessation and additionally, the measure can be potentially validated by biochemical measures.

Smoking duration was calculated by deducting age started smoking from cessation age (having considered any gaps in smoking duration with the participant). Nicotine dependence was also measured using the Fagerström Test for Nicotine Dependence (FTND) (Heatherton et al., 1991). For participants who identified themselves as former smokers, cigarettes per day and the FTND were adapted to reflect habits prior to quitting, as previously described (Sherratt, Marcus, Robinson, Newson, & Field, 2015). Finally, whether or not the participant lived with another smoker was recorded.

Measures of interest regarding lung cancer risk perception, included perceived personal lung cancer risk, perceived average smoker lung cancer risk, perceived relative risk of lung cancer, lung cancer worry, and perceived lung cancer survival. Three of the measures regarding lung cancer risk perceptions were adapted from a previous study (Weinstein et al., 2005) and explored perceived personal lung cancer risk, perceived lung cancer risk of the average smoker, and perceived relative risk of lung cancer. Perceived personal lung cancer risk was asked with the question, "Would you say your chances of getting lung cancer in the future are..." Participants were provided with five response options: Very low; Somewhat low; Moderate; Somewhat high; Very high. Perceived lung cancer risk of the average smoker was ascertained with the question, "Would you say the chances of the average smoker getting lung cancer in future are..." Participants were offered five response options: Very low; Somewhat low; Moderate; Somewhat high; Very high. Perceived relative risk of a

smoker compared with a non-smoker was ascertained using the question, "Would you say the average smoker has..." Participants were provided with five response options: About the same lung cancer risk as a non-smoker; A little higher lung cancer risk than a non-smoker; Twice the lung cancer risk of a non-smoker; Five times the lung cancer risk of a non-smoker.

The remaining two items regarding lung cancer risk perceptions were adapted from a previous study (Rutten et al., 2011); one of which measured lung cancer worry with the question, "How often do you worry about getting lung cancer?" Participants were offered four response options: Rarely or never; Sometimes; Often; All the time. The other measure examined perceived lung cancer survival with the question, "Overall, how many people who get lung cancer do you think will live at least five years?" Participant were provided with five response options: Less than a quarter; About a quarter; About half; About three quarters; Nearly all.

3.5.4 Procedure

As described earlier, the quantitative research component effectively entailed two separate RCTs; one RCT investigated whether application of the LLP intervention is associated with smoking cessation success and lung cancer risk perceptions among baseline current smokers, whilst the other RCT investigated whether application of the LLP intervention is associated with smoking cessation success and lung cancer risk perceptions among baseline recent former smokers. Figures 7 and 8 display the flow of participants through the RCTs, for baseline current smokers and baseline recent former smokers, respectively. Although the current project entailed two separate

RCTs, the procedure for both RCTs remained the same. The study flow will now be described in full.

The researcher attended a wide range of RCFE drop-in sessions, across Liverpool. Upon arrival at an RCFE drop-in session, service users were provided with a PIS (Appendix D) and introduced to the researcher (via an RCFE advisor), who was available for questions. All service users were provided with time to consider participation and following advisor consultation, service users were offered the opportunity to participate. Those who chose to participate were asked to complete a consent form (Appendix E), requiring participants to provide their contact details and sign each consent box that they agreed with. Participants were asked to retain the PIS and photocopies of consent forms were forwarded to participants within one week of recruitment. Participants completed a baseline questionnaire (Appendix F).

Participants for both trials were randomised to either an intervention or control group. Randomisation software was utilised to allocate participants to one of the two groups (via the URL, http://www.randomization.com/). Double-blinding was not possible to implement due to the funding restrictions of the project, as the researcher was responsible for recruitment, randomisation, and delivering the intervention; however, participants were blinded to group allocation, as the PIS did not explicitly describe the information participants would receive. Schulz and Grimes (2002) described several benefits for participant blinding in RCTs, such as participants being less likely to have biased psychological or physical responses to the intervention.

Participants in both arms were provided with the control leaflet (Appendix G), which provided some brief, generic lung cancer risk information, whilst participants in the intervention group were additionally provided with the LLP intervention (Figure 7). The researcher was able to immediately calculate risk projections for participants, by inputting participant questionnaire responses into the desktop database using Microsoft Access. Participants in the intervention arm were also provided with a verbal explanation of the LLP intervention. Following the completion of the baseline questionnaire, participants were contacted at six-month follow-up to complete a follow-up questionnaire (Appendix H).

Participants were contacted at follow-up via telephone firstly. Options to enhance the response rate were explored. If participants were not contactable via telephone (the researcher would attempt to contact them morning, afternoon and evening), the follow-up questionnaire was posted to the participant with a stamped addressed envelope and a letter (Appendix I), which requested the participant to complete the follow-up questionnaire and return it to the researcher. The researcher also adapted the follow-up online questionnaire for completion, via the URL, http://survey.liv.ac.uk/smoking-study. The URL was included in the follow-up letter. All participants were fully debriefed regarding the project and control participants were offered the LLP intervention following debrief.

Follow-up Analys is Randomis ation Recruitment (6-month) completed (n = 95)questionnaire Included in RCT analysis intervention arm † Follow-up Allocated to LLP intervention (n = 154)All participants included in RCT analysis of baseline current smokers † (n = 151)address (n = 3)Deceased or untraceable No response (n = 56) Lost to follow-up (n = 59)questionnaire completed (n = 302)Service users provided with PIS Consented and baseline Randomisation (n = 297)completed (n = 92)questionnaire Follow-up Allocated to control (n = 148)Included in RCT analysis control arm † (n = 146)address(n=2)Deceased or untraceable No response (n = 54)Lost to follow-up (n = 56)

Figure 9. The flow of baseline current smokers through the trial

† = Intention to treat analysis (see West et al., 2005)

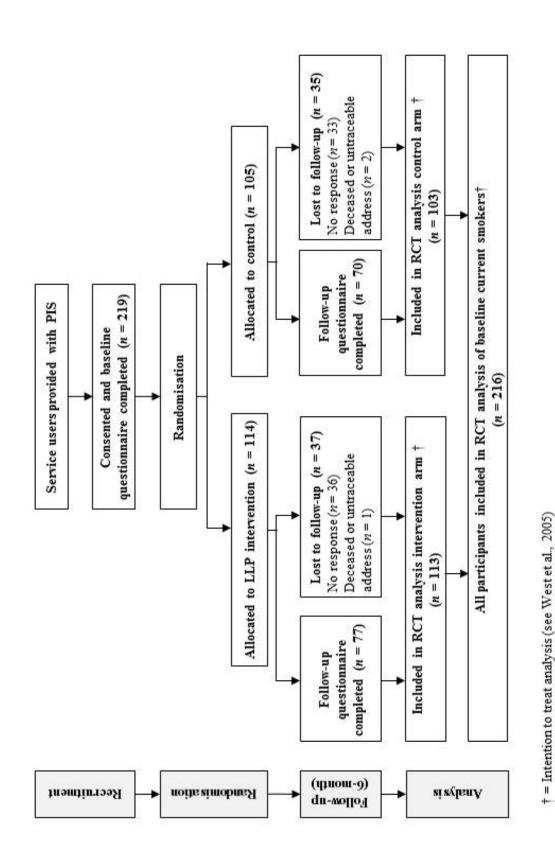


Figure 10. The flow of baseline recent former smokers through the trial

Completed paper consent forms and questionnaires were stored in a locked cabinet, at the University of Liverpool Cancer Research Centre. A unique study identification number (MPI) was assigned to participants and all project forms were labelled with the study identifier and barcoded to facilitate handling of the data. All questionnaire data and consent options were inputted into the LLP database, "SB" (accessible to nominated LLP staff, requiring two passwords) and consent forms were scanned for electronic storage on a secure drive located on the LLP computer network. The accuracy of data collection was checked at regular intervals. Participant names, corresponding drop-in session attended, and treatment group allocation were recorded to avoid recruitment duplication. Microsoft Excel was used to record this information and the document was saved as a password-protected document on an encrypted USB memory stick.

3.5.5 Piloting the questionnaire

The questionnaire was pre-piloted with several members of the Roy Castle Lung Cancer Research Programme. This allowed the researcher to gain feedback on the questionnaire prior to presenting it to advisors and service users at RCFE. Minimal feedback was, however, generated from this meeting with LLP employees. Subsequently, the researcher attended RCFE drop-in sessions for one week, to observe the format of the drop-in sessions, explore project feasibility, and discuss questionnaire development with advisors and some service users. Piloting the study was necessary as it provided valuable feedback from potential members of the study population, thus offering an opportunity to improve questionnaire format and measures (Creswell, 2014).

Most RCFE advisors and service users were satisfied with the content and layout of the questionnaire but some commented on the language used in the project materials. For example, one of the validated measures was phrased "Overall, how many people who get lung cancer do you think will survive at least five years?" One service user commented that she was unclear what was meant by "survive". As a result of such feedback, some questions were amended to ensure that questionnaire language used was suitable for individuals of varying literacy levels. Furthermore, advisors reported low levels of literacy among many service users, highlighting the importance of using uncomplicated, plain English language in the questionnaires. This also guided the delivery of questionnaires, as the researcher subsequently ensured that participants were consistently supported to undertake the questionnaire.

Time was also an important factor to consider, which was identified subsequently to the questionnaire piloting. Service users were encouraged to drop-in sporadically within a pre-advertised time. In observing the drop-in sessions, it became apparent that some service users would engage in longer support sessions, whilst others attended simply to acquire a prescription and leave immediately. Time-burden was therefore an important factor to consider. To utilise time effectively, it was decided that service users would receive a PIS upon arrival at the venue, providing potential participants with the opportunity to consider engagement in the project. Furthermore, it was necessary to review the length of the questionnaire and RCFE management requested for completion to take no more than approximately ten minutes; this was adhered to.

Lastly, some of the drop-in session host locations had limited facilities (e.g. simply a desk and two chairs). The researcher was often required to book a separate room for

the research or arrive early to acquire a small area away from the advisor. This would enable service users who did not wish to take part in the research to maintain their confidential relationship with RCFE, whilst enhancing confidentiality for the current project, and avoiding contamination of participant blinding. The researcher remained conscious of participant confidentiality and sensitivity at all times.

3.6 Qualitative research methods

The qualitative component of the research complements the aforementioned RCTs and was intended to address project objectives two and three (i.e. to explore factors implicated in smoking-related risk perceptions and smoking cessation success). Risk is a complex phenomenon and previous research has failed to utilise a lung cancer risk prediction model as a smoking cessation intervention; therefore, a qualitative approach was deemed most appropriate to address the project objectives, as qualitative research is "especially useful when the research is concerned with either a novel domain or where the issues are complex or dilemmatic" (p. 132-133) (J. A. Smith, Michie, Stephenson, & Quarrell, 2002).

The qualitative research component involved conducting semi-structured telephone interviews with a sub-sample derived from the larger quantitative research component. Semi-structured interviewing is a qualitative method, which combines pre-determined questions, usually using an interview schedule, with the opportunity for the interviewer to examine certain themes or responses in greater detail. Semi-structured interviews have previously been implemented to explore smoking experiences among current and former smokers (L. Hackshaw, Bauld, & McEwen, 2012; Rachael L. Murray, McNeill, Lewis, Britton, & Coleman, 2010; Schofield, Kerr, & Tolson, 2007;

Vangeli & West, 2012). Furthermore, semi-structured interviews are recommended when the researcher is aware of the questions they wish to ask but they are not aware of the responses they are likely to receive (Morse, 2012). For example, at the point of undertaking the qualitative interviews, it was unclear as to whether the LLP intervention would have an effect on smoking cessation rates; therefore, semi-structured interviews seemed the most appropriate methodology to gauge responses and experiences regarding smoking and risk. Open-ended questions were posed and related to the LLP intervention, attitudes to risk, and experiences of smoking and smoking cessation.

Telephone interviews have been criticised because they lack face-to-face contact, which can facilitate the development of rapport; however, telephone interviews can be beneficial in that they increase anonymity in regard to examining sensitive issues and they also reduce costs in relation to time and finances (Irvine, Drew, & Sainsbury, 2013).

3.6.1 Participants

Qualitative interviews were conducted between May 2013 and January 2014. Participants (N = 30) were sought via the larger quantitative research component. The sample consisted of both current (40%) and recent former (60%) smokers between the ages of 18-60 years old, who had attended RCFE within the previous two months. The sub-sample primarily consisted of males, who were classified as White British, and the median age was 49.0 years (IQR 39.5-55.3), whilst ages ranged from 25-59 years (see Table 2).

Table 3. Participant characteristics among the qualitative sample

Pseudonym	Age	Gender	Ethnicity	Smoking	Treatment group
-			·	status	
1. Michael	48	Male	White British	Former	Control
2. Robert	34	Male	White British	Current	Control
3. Jean	59	Female	White British	Former	Intervention
4. Jacob	59	Male	Mixed Caribbean	Former	Control
5. Alex	56	Male	White British	Current	Control
6. Stuart	45	Male	White British	Former	Control
7. Ronald	40	Male	White British	Former	Control
8. Patricia	50	Female	White British	Former	Control
9. David	37	Male	White British	Current	Control
10. Andrew	34	Male	White British	Current	Control
11. Emily	59	Female	White British	Former	Control
12. John	53	Male	White British	Former	Control
13. Julia	49	Female	White British	Former	Intervention
14. Oliver	54	Male	White British	Former	Intervention
15. Jack	49	Male	White British	Current	Control
Charlotte	26	Female	White British	Former	Intervention
17. Matilda	33	Female	White British	Former	Intervention
18. Paul	38	Male	White British	Former	Intervention
19. Sophie	55	Female	White British	Former	Intervention
20. Peter	50	Male	White British	Current	Control
21. Joel	44	Male	White British	Current	Control
22. Luke	54	Male	White British	Current	Intervention
23. Charles	40	Male	White British	Current	Control
24. Gavin	45	Male	White British	Former	Control
25. Shaun	48	Male	White British	Current	Intervention
26. Sarah	55	Female	White British	Former	Intervention
27. Patrick	56	Male	White British	Former	Intervention
28. Timothy	58	Male	White British	Former	Intervention
29. Gemma	57	Female	White British	Current	Intervention
30. Eric	25	Male	White British	Current	Intervention

The sampling method was effectively opportunistic, dependent upon whether participants were available for interview, although participants did need to be stratified based on treatment group as part of the quantitative research design. Efforts were therefore made to achieve even numbers of participants from both the control and intervention groups, among both the baseline current and recent former smoker samples, in order to eliminate the extraneous effect of undertaking qualitative interviews on the RCT analyses. Despite these documented efforts, it was not possible to achieve an equal number of qualitative participants from both treatment groups across both samples due to the availability of participants. The majority of baseline

current and recent former smokers recruited for the qualitative sub-study were allocated to the intervention group; n = 8 (66.7%) and n = 10 (55.6%), respectively.

3.6.2 Materials

Printed study materials relevant to the qualitative component of the research, included the PIS (Appendix D) and the consent form (Appendix E). In relation to technological equipment, an encrypted USB memory stick was utilised to stratify participants for interviews and record participants who had completed qualitative interviews. The researcher used a landline speakerphone to contact participants and a Dictaphone to record interviews. A remote office was available to ensure that calls could be made confidentially to participants. Transcripts were electronically stored on the secure University of Liverpool "M:/" drive within the PhD researcher's account. Interviews were transcribed in Microsoft Word. Microsoft Excel was used to store the aforementioned record of participants recruited, and NVIVO 10.0 (QSR International) was used to aid the analysis.

3.6.3 Procedure

An interview schedule was developed (Appendix J), which aimed to achieve the project objectives and provoke open responses and fruitful dialogue between researcher and participant. As the qualitative research component utilised a subsample of participants derived from the RCT, the procedure regarding recruitment has previously been described (see Section 3.5.4); however, all participants were provided with the opportunity to sign an optional consenting statement as part of the consent from (detailed on Appendix E), which would grant permission to be contacted regarding the qualitative research component. The consent form stated: "I give

permission for the researcher to contact me by telephone, to ask me my views about receiving risk information on lung cancer and quitting smoking. I understand that these telephone interviews will be recorded and that the audio recording will be destroyed, once it has been transcribed."

Using Microsoft Excel, individuals who provided consent to be contacted in relation to the qualitative interviews were identified and contacted chronologically and opportunistically by recruitment date. Interviews were undertaken by telephone within two months of recruitment. Upon contacting participants via telephone, the researcher reiterated the aims of the qualitative study and confirmed whether or not the participant continued to consent to undertake the interview and whether it was a convenient time to be interviewed. The researcher informed participants that the call would be recorded, transcribed and subsequently, anonymised. Telephone interviews were guided by the interview schedule (Appendix K) and typically lasted between 20 and 25 minutes but ranged from 15 to 60 minutes. Following the interview, participants were thanked for their involvement and informed that they would be contacted again in future, as part of the quantitative follow-up.

Following each interview, the interview recording was immediately uploaded to the University of Liverpool M:/ and transcribed by the researcher in Microsoft Word. The accuracy of transcription was checked at regular intervals. All transcripts were anonymised and pseudonyms were generated. A separate Microsoft Word file was created, in which study identifiers were listed alongside pseudonyms, age, gender, ethnicity, smoking status, and treatment group (sourced from the quantitative study questionnaire [Appendix F] or the interview itself). This file and the anonymised

transcripts were saved as password protected documents on the secure University of Liverpool M:/ drive on the researcher's account. Participant audio recordings were subsequently deleted, further ensuring participant anonymity.

3.7 Ethical considerations

An ethics application was submitted to Liverpool Research Ethics Committee, which detailed the full protocol for the PhD project. Full ethical approval was obtained on 12th February 2013 (Appendix K). The researcher also undertook a National Institute of Health Research (NIHR) Good Clinical Practice course, to enhance knowledge regarding ethical considerations (Appendix L). Some of the pertinent ethical considerations in relation to the current project are noted in the following sections, including informed consent, voluntary participation, anonymity, appropriate handling of participants, and debriefing.

3.7.1 Informed consent

All participants received a PIS (Appendix D) and were given the opportunity to ask questions. Following this, participants signed a consent form prior to completing the baseline questionnaire. The PIS also included project contact details. Participants who consented to undertake a qualitative interview were contacted by telephone and were interviewed at a mutually convenient time over the telephone. Participants who agreed to complete a telephone interview were informed that interviews would be recorded, but that recordings would be deleted following transcription and anonymisation.

3.7.2 Voluntary participation

Upon completing the consent form, participants were informed verbally and in writing of their option to withdraw from the study. It was highlighted that withdrawal from either the quantitative or qualitative research components would not affect participants' treatment or within RCFE or any other health service provider. To avoid service users feeling coerced to participate, service users were informed that the researcher worked independently to RCFE, both verbally and via the PIS. Furthermore, participants were informed that it was not mandatory for them to answer all questionnaire measures or qualitative interview questions, should they wish to exclude any information.

3.7.3 Anonymity

Although full anonymity was considered initially, it was not appropriate to ensure participant anonymity throughout the project. This was due to two reasons; firstly, participants were to be contacted at six-month follow-up and therefore, contact details were required, and secondly, participant data contributed towards the larger LLP dataset, which required personal details for further follow-up. These aspects were relayed to participants in the consent form; however, at the point of data inputting, participants were assigned a study identifier number (MPI), ensuring that personal details were referred to minimally. The file linking the study identifier with identifiable participant details was kept under strict security, with access by authorised personnel only. All data were handled in accordance with the Data Protection Act 1998. All interview transcripts were anonymised by assigning pseudonyms and names of people and places were omitted from the transcripts to further enhance anonymity. Lastly, interview recordings were deleted upon transcription.

3.7.4 Appropriate handling of participants

It was necessary to remain sensitive to participant needs throughout both the quantitative and qualitative research components, as the questionnaire and qualitative interviews addressed some sensitive and emotive issues, such as lung cancer, risk, and blame; therefore, participants were informed that some questions could be omitted within the questionnaire and additionally, it was deemed inappropriate for the researcher to fully probe participants for responses in circumstances whereby participants presented in an uncomfortable or distressed manner. Furthermore, participants were referred to RCFE advisors if they had any further concerns regarding smoking cessation.

3.7.5 Debriefing

Participants were blinded to randomisation; following completion of the project, all participants were debriefed regarding the aims of the project and blinding (with the exception of those who were lost to follow-up). The researcher offered to deliver the LLP intervention to all control participants, who would not have received it at baseline. Participants who had since relapsed at follow-up were also encouraged to seek the support of RCFE.

3.8 Quantitative research analyses

This section describes the data analyses undertaken in regard to the quantitative research component. A variety of statistical tests were performed as part of the quantitative investigation. Study data were transferred from the program used to store LLP data, "SB" to IBM-SPSS statistical software version 21.0 (New York, NY), which was a more manageable format suitable for statistical analysis. Prior to devising

a data analysis plan, data preparation was necessary; this involved adapting the datasets to allow for intention to treat analyses, as well as transforming some variables, to ensure the dataset was suitable for multivariate analyses. Following data preparation, the data analysis plan was developed, whereby various statistical tests were selected to explore the data; these are described.

The datasets needed to be adapted to account for an intention to treat approach to the analysis. West et al. (2005) recommended common standards for smoking cessation trials (The Russell Standard). As part of these recommendations, West et al. (2005) proposed that smoking cessation trials should adopt an intention to treat strategy, which stipulates that data from all randomised participants should be included in follow-up analyses, unless participants have died or have relocated to an untraceable address. If these circumstances are not applicable but smoking status cannot be determined, the participant is regarded as a current smoker when analysing the data. Figures 7 and 8 display the flow of baseline current smokers and baseline recent former smokers and demonstrate how the intention to treat approach was integrated throughout the trials.

A number of steps were taken to prepare the dataset for the main analyses. Firstly, data transformation was considered. Cell frequencies for variable levels among all variables were calculated initially to explore the distribution of the data and to prepare the data for additional analyses. A number of variables were subsequently transformed to avoid low cell frequencies and therefore, develop robust regression model, in line with recommendations (A. Field, 2013). Appendix M displays the original cell frequencies for all variables of interest, which were subsequently recoded and the

transformation for each of the identified variables is described. These reformed variables and associated levels are consequently incorporated throughout the quantitative results chapters.

3.8.1 Data analysis plans

Following data preparation, data analysis plans were developed. The current section identifies the key research objectives associated with the quantitative research component and considers the appropriate statistical tests to address each of the objectives. It should be noted that the distribution of all study variables was considered prior to the implementation of any given test. In checking the distribution of variables, histograms and Q-Q plots were generated, whilst the Shapiro-Wilk test was implemented to examine the P-value (p < 0.05 suggests non-normal distribution) (A. Field, 2013); however, all baseline project variables considered were not-normally distributed, which ultimately influenced the choice of statistical tests that were conducted.

A listwise deletion method was adopted for multivariate analyses, whereby cases with missing data were excluded from analyses. Efforts were, however, made to avoid missing data. For example, participants were contacted by telephone in cases where random questionnaire responses were missing; however, in many cases missing data was unavoidable. A number of participants requested not to provide responses in relation to perceived personal lung cancer risk, as they felt uncomfortable predicting their own risk and some participants chose not to provide some demographic details, such as ethnicity, as they disapproved of being categorised.

3.8.1.1 Objective one analysis

It was necessary to undertake various analyses to address the first project objective, "To investigate whether application of the LLP intervention is associated with smoking cessation success and lung cancer risk perceptions". This objective incorporated the use of both bivariate and multivariate analyses and all tests were undertaken firstly, for the dataset consisting of baseline current smokers and secondly, for the dataset consisting of baseline recent former smokers. Treatment group was the key predictor variable of interest, whilst the outcome variables of interest, included follow-up smoking status, perceived personal lung cancer risk, perceived average smoker lung cancer risk, perceived relative risk of lung cancer, and lung cancer worry.

Bivariate tests were undertaken between baseline participant characteristics and treatment groups, to explore differences among treatment groups, in order to explore any potential confounding effects. The choice of bivariate test was largely dependent on the type of data the variable entailed and the number of variable levels included within the variable, as none of the baseline participant characteristic variables were normally distributed. Associations between treatment group and baseline participant characteristic variables with two or more categorical levels, were measured using Chisquared tests (or Fisher's Exact test when expected cell frequencies were less than five). Associations between treatment group and continuous baseline participant characteristic variables were examined using Mann Whitney U-tests.

Baseline characteristics significantly associated with treatment group at the level of p < 0.25 were highlighted and considered for inclusion in the development of several multivariate models to explore the previously described outcome measures. It should

be noted that p < 0.25 was identified as the appropriate level of significance for covariate selection, in line with guidance from Hosmer et al. (2013), regarding purposeful selection of covariates in logistic regression; by following this guidance, potentially confounding variables would be controlled for.

Forced entry binary logistic regression was subsequently deemed the most appropriate method to guide multivariate model development to explore any associations between treatment groups and the aforementioned outcome variables, whilst controlling for potentially confounding variables. Studenmund and Cassidy (as cited in A. Field, 2013) recommend forced entry over stepwise modelling for theory testing, as stepwise techniques can be influenced by random data variation and subsequently, stepwise modelling rarely produce replicable output if the model is retested.

Binary logistic regression entails a number of assumptions and therefore, further tests were required to ensure that these assumptions were not violated. Logistic regression assumes that each case is independent, that the model should be fitted correctly, that there is no multicollinearity, and that there is linearity of predictor variables and log odds (A. Field, 2013). In cases where these assumptions are violated, this is detailed throughout the results chapters but these aspects will now be discussed.

All observations for the current quantitative investigation were independent. With regard to the model fit, logistic regression assumes that the model should be fitted correctly. The Hosmer-Lemeshow goodness-of-fit statistic was used to assess how well the model fitted the data; a non-significant statistic (p > 0.05) suggests that the model fits the data well (Hosmer et al., 2013). The goodness-of-fit statistic is stated in

regard to all relevant logistic regression analyses within the results chapters. Residual statistics were also explored to examine the model fit.

Binary logistic regression also assumes that predictor variables should not be highly correlated with each other (i.e. multicollinearity). Variables to be included in multivariate models were explored by implementing a Spearman's Rho correlation matrix to identify closely related variables. Following this, VIF and tolerance statistics were ascertained. To ensure the assumption of multicollinearity was not violated, VIF values needed to be below 10 (Bowerman & O'Connell, 1990; Myers, 1990, as cited in A. Field, 2013), whilst tolerance values needed to be below 0.1 (Menard, 1995, as cited in A. Field, 2013); however, no cases of multicollinearity were evident in the current project.

Logistic regression also assumes that there is linearity of continuous predictor variables and log of the data (or logit). In order to explore linear relationships, the interaction term between the predictor and its log transformation were investigated; a significant result (p < 0.05) suggests that the main effect has violated the linearity of the logit assumption (A. Field, 2013). These tests were undertaken for all regression models and in cases where the assumption is violated, this is detailed in the results chapters.

Lastly, logistic regression requires substantial sample sizes. A. Field (2013) proposes that goodness-of-fit tests for logistic regression assume that no cells have an expected count below one and no more than 20% should be below five; therefore, several variables were transformed, as previously described (see Appendix M).

3.8.1.2 Objective two analysis

A selection of bivariate and multivariate analyses were undertaken to address the second project objective, "To explore factors implicated in smoking-related risk perceptions". For these analyses, a dataset was utilised, which consisted of all project participants recruited (regardless of smoking status) who responded to the baseline perceived personal lung cancer risk measure (N = 502); the aim in undertaking these analyses was to investigate cross-sectional differences overall by perceived personal lung cancer risk. The predictive capacity of all baseline participant characteristics was considered and baseline perceived personal lung cancer risk was the key outcome variable of interest.

Bivariate tests were undertaken between baseline participant characteristics and baseline perceived personal lung cancer risk, to explore differences among perceived personal lung cancer risk levels, in order to identify potential predictors and to explore any potentially confounding effects. Again, the choice of bivariate tests implemented was largely dependent on the type of data the variable consisted of and the number of variable levels included within each variable because as detailed, all baseline participant characteristic variables were not normally distributed. Relationships between baseline perceived personal lung cancer risk and predictor variables with two categorical levels, were measured using Mann Whitney U-tests, to ascertain differences in means between the two levels. Kruskal Wallis H-test was implemented to identify associations between baseline perceived personal lung cancer risk and predictor variables with more than two categorical levels. Lastly, relationships

between baseline perceived personal lung cancer risk and variables consisting of continuous or ordinal data were tested using Spearman's Rho correlation analysis.

Again, guidance proposed by Hosmer et al. (2013) pertaining to purposeful selection of covariates for the development of multivariate models was considered; therefore, predictor variables deemed to be significant at the level of p < 0.25 were considered in the development of further multivariate analyses. Hosmer et al. (2013) additionally recommended refining initial multivariate models to develop a more parsimonious final model, which typically involves removing non-significant variables. A parsimonious model is preferable because developing a model with too many independent variables can lead to a mathematically unstable outcome, with reduced generalisability beyond the current project sample (Stoltzfus, 2011).

As the outcome variable was ordinal, ordinal regression, more specifically, a proportional odds (PO) regression model, was developed to examine predictors in greater detail. PO modelling has a number of key assumptions, similar to binary logistic regression; however, PO modelling assumes that the outcome variable includes interval levels and that the odds ratio (OR) is equal across each interval threshold. The score test for the PO assumption (or test of parallel lines) can be conducted to examine whether the assumption of PO is violated for a PO model; p < 0.05 suggests that the assumption of PO is violated (A. A. O'Connell, 2005). The results of tests of parallel lines are detailed in the results chapter where applicable.

3.8.1.3 Objective three analysis

It was necessary to undertake various analyses to address the third project objective, "To explore factors implicated in smoking cessation success". This objective employed the use of both bivariate and multivariate analyses and all analyses were undertaken across three datasets: (1) baseline current smokers; (2) baseline recent former smokers; (3) all project participants recruited at baseline. Predictors of follow-up smoking status were explored among the first two datasets, whilst predictors of baseline smoking status were explored using the sample of participants overall. To successfully achieve the current project objective, it was necessary to build a multivariate model for each of the described datasets; however, the analysis plan for each of the datasets remained almost identical.

Bivariate tests were undertaken between baseline participant characteristics and smoking status, to explore potential predictors of smoking status and to identify any potentially confounding effects. As described, the choice of bivariate test was dependent on the variable data type and the number of variable levels included within the variable; no baseline participant characteristic variables were normally distributed. Associations between smoking status and baseline participant characteristic variables with two or more categorical levels, were measured using Chi-squared tests (or Fisher's Exact test when expected cell frequencies were less than 5). Associations between smoking status and continuous baseline participant characteristic variables, were examined using Mann Whitney U-tests.

Again, Hosmer et al.'s (2013) recommendations regarding purposeful selection of covariates in logistic regression guided the development of the multivariate models.

Therefore, all baseline participant characteristics associated with smoking status at the level of p < 0.25 were considered for inclusion in the development of multivariate models and efforts were made to refine initial models to ensure that they were parsimonious.

Again, forced entry binary logistic regression was subsequently deemed the most appropriate method to guide multivariate model development to explore any associations between baseline participant characteristics and smoking status, whilst controlling for any potentially confounding variables. In conducting binary logistic regression analyses, the assumptions associated with binary logistic regression were noted; each case should be independent, the model should be fitted correctly, there should be no multicollinearity, and there should be linearity of predictor variables and log odds (A. Field, 2013). These aspects of binary logistic regression were previously discussed (see Section 3.8.1.1) but again, in cases where these assumptions are violated, details are provided within the respective results chapter.

3.9 Qualitative research analyses

This section describes the qualitative data analyses undertaken with regard to qualitative research component. Thematic analysis (TA) was selected as the most appropriate approach to analysing the dataset. Braun and Clarke (2006) explained that TA provides a flexible, useful research tool, capable of providing detailed and complex accounts of data with theoretical freedom. Furthermore, adopting an inductive TA approach to the study would enable us to explore and recognise frequently recurrent themes, primarily at a semantic level (i.e. basing analysis on the surface meanings). A number of studies have also implemented TA to explore

qualitative datasets involving service users engaged with SSS (Henderson, Memon, Lawson, Jacobs, & Koutsogeorgou, 2011; S. E. Jones & Hamilton, 2013).

A step-by-step guide to TA helped to inform the process of analysis (Braun & Clarke, 2006) and as previously described, the computer program, Nvivo 10 (QSR International) was used to aid the analysis. Braun and Clarke (2006) described the process of TA through six phases: (1) Familiarising oneself with the data; (2) Generating initial codes; (3) Searching for themes; (4) Reviewing themes; (5) Naming and defining themes; (6) Producing the report. These phases will now be explored in greater detail with regard to the current project.

Firstly, the researcher aimed to familiarise themselves with the data. The researcher collated the data and transcribed the recordings in Microsoft Word. As the researcher personally transcribed the data, this provided an opportunity for the researcher to familiarise themselves with the data, whilst ensuring that the written transcripts accurately reflected the verbal recordings. Following transcription, the researcher actively read the transcripts at least three times to further familiarise themselves with the data. Furthermore, the researcher began to note prominent findings at this stage.

Secondly, initial codes were generated. This was done firstly by annotating the paper transcripts themselves; however, as time progressed, this method proved challenging to develop and maintain, so transcripts were exported to Nvivo 10 (QSR International), which eased data organisation. Initial codes were identified by examining the data on a line-by-line basis. Figure 11 provides an example of a data extract and how this was coded initially.

Data extract	Coded for
"It's just the fact that it's been going on so long now, do you know what I mean?	1. Considers smoking duration as extensive.
It's like starting to do me head in, in a	2. Starting to feel frustrated about
way, it's not like, it's not a joke anymore now, you know what I mean, it's starting	continued smoking.
to proper do me head in, you know what	
I mean?"	

Figure 11. Qualitative data extract with applied initial codes

Thirdly, the researcher searched for broader themes. This involved the researcher analysing the codes to consider how these codes could contribute towards the development of overarching themes. In doing so, the researcher developed several mind maps. These mind maps included key major themes (i.e. overarching themes) and sub-themes (collections of codes that contribute towards an overarching theme).

Fourthly, themes were reviewed by the researcher. This involved reading all collective data extracts for each potential theme and considering whether they fit appropriately. Furthermore, themes were reviewed in consideration of the dataset as a whole and whether the thematic map reflected the meanings derived from the data. The review process was extensive and iterative, as initially some sub-themes did not fit well with the major themes developed; therefore, themes were reviewed until a satisfactory thematic map was developed. One of the PhD project supervisors was also an experienced social anthropologist, with vast experience in qualitative research; validation of themes occurred through face-to-face and online discussions between the respective PhD supervisor and PhD researcher.

The fifth stage entailed the researcher defining the themes and further refining the themes that were presented. The researcher produced a detailed analysis for each theme. Six major themes were finalised: (1) Increased risk awareness; (2) Disregarding risk; (3) Experience of mental conflict; (4) Perceived benefits of quitting; (5) Reshaping social norms; (6) Attitudes regarding local Stop Smoking Services.

Some of the themes or sub-themes were inter-related and such cases are acknowledged in the results chapters. Figure 12 displays an example finalised thematic map for Chapter 5, which explores the major themes, "Increased risk awareness" and "Disregarding risk". Qualitative results are presented in Chapters 5, 7 and 8.

Lastly, the researcher wrote up the results, which entailed concisely and coherently providing an account of the data; as part of this, sufficient evidence of the themes derived from the data (i.e. relevant quotes) were required to demonstrate the prevalence of the theme.

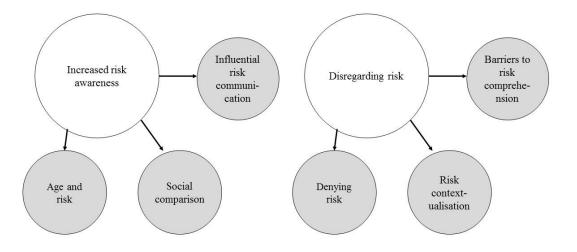


Figure 12. A finalised thematic map for two major qualitative themes

3.10 Conclusion

The current chapter documented the development of the LLP intervention and presented the methodology for the PhD project. The project utilised a mixed methods research design to primarily investigate whether application of the LLP intervention was associated with follow-up smoking cessation success and lung cancer risk perceptions. The application of a lung cancer risk prediction model in the context of smoking cessation has not previously been investigated and this chapter highlights how adopting a mixed methods approach could be particularly beneficial in achieving the project objectives.

The quantitative research component not only entails examination of the LLP intervention effect but additionally, it explores factors implicated in smoking cessation success and lung cancer risk perceptions. If application of the LLP intervention is found to be associated with follow-up smoking cessation success, this finding would have important implications for tobacco control practice; if effective, the LLP intervention could be delivered within SSS, thus reducing smoking rates and associated smoking-related disease. This component will also contribute towards understanding of smoking-related risk perception and smoking cessation success, which in turn could inform the delivery of SSS and the development of future lung cancer risk communication tools.

The qualitative research component complements the quantitative research in that it aims to explore factors implicated in smoking cessation success and smoking-related risk perceptions; as part of this, the perceived efficacy of the LLP intervention can also

be investigated, as well as the potential mechanisms for the LLP intervention effect, if applicable. Again, the results of the qualitative investigation will inform both SSS and risk communication development, as well as contributing towards improved knowledge regarding how smokers perceive smoking-related risk and the extent to which this influences smoking behaviour. The discussion chapter of the thesis will draw upon the mixed methods approach in particular and detail all project results collectively. The results of the research are presented in the following chapters.

Chapter 4: Evaluating the LLP intervention

4.1 Introduction

This chapter aims to explore whether application of the LLP intervention is associated with smoking cessation success and lung cancer risk perceptions. The results discussed in this chapter will help to determine the extent to which application of the LLP intervention is associated with smoking cessation success and lung cancer risk perceptions. If the LLP intervention is found to be associated with smoking cessation, this has important implications for tobacco control practice. Furthermore, exploration of the relationships between the LLP intervention and lung cancer risk perceptions may enhance understandings regarding risk perception and the mechanisms by which the intervention may or may not be associated with smoking cessation success. The current chapter addresses one key thesis objective:

 To investigate whether application of the LLP intervention is associated with smoking cessation success and lung cancer risk perceptions.

In order to achieve the above objective, statistical analyses were conducted. Firstly, a list of baseline participant characteristics was compiled, which was informed by the literature (see Section 2.5.2). Analyses were undertaken to ascertain significant differences between the intervention and control groups; by undertaking these analyses potential confounding variables could be recognised and incorporated into further analyses. Multivariate analyses were undertaken to explore the extent to which application of the LLP intervention is associated with each of the follow-up outcome variables: (1) 7-day point prevalence (i.e. smoking status); (2) perceived personal lung

cancer risk; (3) perceived average smoker lung cancer risk; (4) perceived relative risk of lung cancer, and; (5) lung cancer worry.

The current project originally aimed to recruit only baseline current smokers (i.e. those who had smoked in the previous seven days at baseline); however, both baseline current smokers and recent former smokers (i.e. those who had quit for at least one week but remained engaged in SSS) were ultimately recruited, due to the limited availability of baseline current smokers. Bivariate and multivariate tests, which examined the LLP intervention effect are therefore explored across two separate datasets: (1) baseline current smokers only (n = 297); (2) baseline former smokers only (n = 216). It is necessary to analyse these two datasets separately as some statistical associations could remain hidden or may not be applicable if both samples were analysed collectively as one.

4.2 Evaluating the intervention effect among baseline current smokers

The current section will utilise a sample of baseline current smokers (n = 297) to investigate whether application of the LLP intervention is associated with follow-up 7-day point prevalence, perceived personal lung cancer risk, perceived average smoker lung cancer risk, perceived relative risk of lung cancer, and lung cancer worry. The results of all bivariate and multivariate analyses are described. Several bivariate tests were undertaken to explore significant differences between the treatment groups. Table 4 displays the distribution of baseline participant characteristics between the two treatment groups, as well as indicating significant differences of note. Hosmer et al. (2013) suggest incorporating all potential confounders, significant at the level of 25% in further multivariate analyses.

The bivariate tests revealed that the baseline variables age (p = 0.154), socio-economic status (p = 0.003), and perceived relative risk of lung cancer (p = 0.024) significantly differed by treatment group. No significant effects were observed between treatment groups and the remaining baseline variables, including gender, ethnicity, marital status, highest educational attainment, age started smoking, living with another smoker, FTND, cigarettes per day, perceived personal lung cancer risk, perceived average smoker lung cancer risk, lung cancer worry, and perceived lung cancer survival. It should be noted that the identified relationship between treatment group and socio-economic status and perceived relative risk were not anticipated. It is unclear as to why these relationships occurred; these differences could be due to chance or limited cell numbers but highlighting them as significant to the level of p < 0.25 ensures that any extraneous variable effects can be controlled for.

To compare 7-day point prevalence abstinence, perceived personal lung cancer risk, perceived average smoker lung cancer risk, perceived relative risk of lung cancer, and lung cancer worry, logistic regression analyses were conducted to adjust for baseline between-group differences (i.e. age, socio-economic status, and perceived relative risk of lung cancer). Table 5 displays that no significant effects were detected between treatment groups and any of the outcome variables, including 7-day point prevalence (p=0.658), perceived personal lung cancer risk (p=0.785), perceived average smoker lung cancer risk (p=0.950), perceived relative risk of lung cancer (p=0.580), and lung cancer worry (p=0.455), even after adjusting for the relevant baseline characteristics.

Table 4. Participant characteristics by treatment group among baseline current smokers

Baseline variable	Control	Intervention	<i>P</i> -value
Buseline variable	(n = 146, 49.2%)	(n = 151, 50.8%)	§
Age (Median, IQR)	42.5 (33.0-51.0)	41.0 (30.0-50.0)	0.154*
Gender			0.343
Female	83 (56.8)	94 (62.3)	
Male	63 (43.2)	57 (37.7)	
Ethnicity†			0.802
White	134 (91.8)	137 (92.6)	
Other	12 (8.2)	11 (7.4)	
Marital status†			0.823
Other	21 (14.4)	23 (15.4)	
Single	76 (52.1)	81 (54.4)	
Married and living together	49 (33.6)	45 (30.2)	
Highest educational attainment†			0.803
Basic or no qualifications	66 (46.2)	70 (47.6)	
Higher qualifications	77 (53.8)	77 (52.4)	
Socio-economic status†			0.003*
Most deprived	117 (80.1)	138 (92.0)	
Least deprived	29 (19.9)	12 (8.0)	
Age started smoking (Median, IQR)	15.0 (13.0-17.0)	15.0 (13.0-17.0)	0.600
Living with another smoker†			0.423
No	51 (34.9)	58 (39.5)	
Yes	95 (65.1)	89 (60.5)	
<u>FTND</u> (Median, IQR)	6.0 (4.0-7.0)	6.0 (4.0-7.0)	0.854
Cigarettes per day (Median, IQR)	20.0 (11.3-25.0)	20.0 (14.5-20.0)	0.668
Perceived personal lung cancer risk†			0.514
Low	23 (16.4)	28 (19.2)	
Moderate	53 (37.9)	61 (41.8)	
High	. 64 (45.7)	57 (39.0)	0.400
Perceived average smoker lung cancer risk			0.688
Very low to moderate	33 (23.2)	39 (26.9)	
Somewhat high	54 (38.0)	49 (33.8)	
Very high	55 (38.7)	57 (39.3)	0.0044
Perceived relative risk of lung cancer†	2 5 (4 0 0)	24 (24 1)	0.024*
About the same to a little higher risk	26 (18.8)	31 (21.4)	
Twice as high risk	17 (12.3)	29 (20.0)	
Five times higher risk	37 (26.8)	48 (33.1)	
Ten times higher risk	58 (42.0)	37 (25.5)	0.006
Lung cancer worry†	42 (20 1)	44 (20 5)	0.986
Rarely or never	43 (30.1)	44 (29.7)	
Sometimes	53 (37.1)	54 (36.5)	
Often or all the time	47 (32.9)	50 (33.8)	0.060
Perceived lung cancer survival†	51 (05.1)	54 (25.0)	0.869
Less than a quarter	51 (36.4)	54 (37.8)	
About a quarter	44 (31.4)	39 (27.3)	
About half	32 (22.9)	37 (25.9)	
About three quarters to nearly all	13 (9.3)	13 (9.1)	

[†] Figures do not equate to 297 due to some missing data, FTND = Fagerström Test of Nicotine Dependence, § Categorical variables were analysed using χ^2 test and continuous variables with Mann Whitney U-test, * p < 0.25

Table 5. Outcome variables at six-month follow-up among current smokers

0-4	Gartal	T	Odd Data (OSO) Cha	D1
Outcome variables	n (%)	$n\left(\%\right)$	Odds Kallo (93% C1)	r-value
Primary outcome variable				
Seven-day point prevalence				0.658
Current	116 (79.5)	123 (81.5)	1.00~(Base)	
Former	30 (20.5)	28 (18.5)	0.87 (0.47-1.62)	
Secondary outcome variables				
Perceived personal lung cancer risk?				0.785
Low	11 (13.1)	9 (10.2)	1.00~(Base)	
Moderate	42 (50.0)	42 (47.7)	1.37 (0.49-3.82)	
High	31 (36.9)	37 (42.0)	1.45 (0.51-4.17)	
Perceived average smoker lung cancer risk†				0.950
Very low to moderate	16 (18.4)	15 (16.7)	1.00~(Base)	
Somewhat high	31 (35.6)	35 (38.9)	1.17 (0.45-3.06)	
Very high	40 (46.0)	40 (44.4)	1.11 (0.43-2.85)	
Perceived relative risk of lung cancer				0.580
About the same to a little higher risk	8 (9.1)	13 (15.1)	1.00~(Base)	
Twice as high risk	15 (17.0)	10 (11.6)	0.42(0.12-1.49)	
Five times higher risk	21 (23.9)	24 (27.9)	0.72 (0.22-2.30)	
Ten times higher risk	44 (50.0)	39 (45.3)	0.65 (0.21-1.99)	
Lung cancer worry				0.455
Rarely or never	37 (42.0)	37 (41.1)	1.00~(Base)	
Sometimes	33 (37.5)	28 (31.1)	1.01 (0.48-2.13)	
Often or all the time	18 (20.5)	25 (27.8)	1.62 (0.71-3.67)	

Tigures do not equate to 297 due to some missing data, "Multivariate logistic regression: adjusted for baseline age, socio-economic status, and perceived relative risk, * p < 0.05

4.3 Evaluating the intervention effect among baseline recent former smokers

The present section utilises a sample of baseline recent former smokers (n = 216) to investigate whether application of the LLP intervention is associated with the outcome variables, follow-up 7-day point prevalence, perceived personal lung cancer risk, perceived average smoker lung cancer risk, perceived relative risk of lung cancer, and lung cancer worry.

Numerous bivariate tests were conducted to investigate significant differences between the intervention and control groups. Table 6 shows the distribution of baseline participant characteristics between the two treatment groups. Again, Hosmer et al. (2013) suggest incorporating all potential confounders, significant at the level of 25%, in further multivariate analyses; therefore, all variables of relevance to further multivariate analyses are indicated.

A number of significant differences were revealed between the intervention and control groups (Table 6). Age (p = 0.122), gender (p = 0.243), ethnicity (p = 0.241), marital status (p = 0.178), highest educational attainment (p = 0.001), and quit duration (p = 0.156) significantly differed by treatment group. No significant effects were revealed between treatment groups and the baseline variables, socio-economic status, age started smoking, living with another smoker, FTND, cigarettes per day, perceived personal lung cancer risk, perceived average smoker lung cancer risk, perceived relative risk of lung cancer, lung cancer worry, and perceived lung cancer survival.

Table 6. Participant characteristics by treatment group among recent former smokers

Age (Mediam, IQR)	Baseline variable	Control	Intervention	<i>P</i> -value
Age (Median, IQR)	Buseline variable			
Gender 52 (50.5) 66 (58.4) Ala Female Male 51 (49.5) 47 (41.6) 147 (41.6) Ethnicity† 0.241* 0.241* White 92 (89.3) 105 (93.8) 0.241* White 92 (89.3) 105 (93.8) 0.78* Other 11 (10.7) 7 (6.3) 7 Other 16 (15.5) 22 (19.8) 18 (36.9) Single 51 (49.5) 41 (36.9) 48 (43.2) Married and living together 36 (35.0) 48 (43.2) 0.001* Highest educational attainment† 0 (058.3) 41 (36.6) 0.001* Basic or no qualifications 60 (58.3) 41 (36.6) 0.937 Higher qualifications 60 (58.3) 41 (36.6) 0.937 Most deprived 87 (84.5) 95 (84.1) 0.937 Least deprived 16 (15.5) 18 (15.9) 0.567 Age started smoking (Median, IQR) 20 (70.10.10.0) 6.0 (40.10.10.0) 0.812 Living with another smoker† 6.0 (50.0-7.0) 6.0 (40.8.0)	Age (Median, IOR)			
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Ethnicity		, ,	, ,	
White Other 92 (89.3) 105 (93.8) 0 colors Other Other 11 (10.7) 7 (6.3) 0.178* Other Single Other 16 (15.5) 22 (19.8) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9) 4 (36.9)			,	0.241*
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Higher qualifications	Highest educational attainment†			0.001*
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FTND (Median, IQR) 6.0 (5.0-7.0) 6.0 (4.0-8.0) 0.943 Cigarettes per day (Median, IQR) 20.0 (20.0-30.0) 20.0 (15.0-30.0) 0.461 Quit duration (days) (Median, IQR) 35.0 (21.0-70.0) 46.0 (26.0-76.0) 0.156* Perceived personal lung cancer risk† 0.987 Low 33 (33.0) 35 (32.1) Moderate 43 (43.0) 47 (43.1) High 24 (24.0) 27 (24.8) Perceived average smoker lung cancer risk† 0.277 Very low to moderate 19 (18.6) 22 (20.2) Somewhat high 51 (50.0) 43 (39.4) Very high 32 (31.4) 44 (40.4) Perceived relative risk of lung cancer† 0.447 About the same to a little higher risk 14 (13.9) 21 (19.3) Twice as high risk 18 (17.8) 24 (22.0) Five times higher risk 29 (28.7) 23 (21.1) Ten times higher risk 40 (39.6) 41 (37.6) Lung cancer worry† 0.937 Rarely or never 36 (35.3) 37 (33.0) Sometimes <td>No</td> <td>80 (77.7)</td> <td></td> <td></td>	No	80 (77.7)		
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Derceived personal lung cancer risk Company				0.461
Low 33 (33.0) 35 (32.1) Moderate 43 (43.0) 47 (43.1) High 24 (24.0) 27 (24.8) Perceived average smoker lung cancer risk† 0.277	Quit duration (days) (Median, IQR)	35.0 (21.0-70.0)	46.0 (26.0-76.0)	0.156*
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About three quarters to nearly all 8 (8.1) 7 (6.4)	-	` '	, ,	
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[†] Figures do not equate to 216 due to some missing data, FTND = Fagerström Test of Nicotine Dependence, § Categorical variables were analysed using χ^2 test and continuous variables with Mann Whitney U-test, * p < 0.25

Table 7. Outcome variables at six-month follow-up among recent former smokers

Outcome variables	Control	Intervention	Odds Ratio (95% CI) ^a	P-value
	n (%)	n (%)		
Primary outcome variable				
Seven-day point prevalence				0.040*
Current	66 (64.1)	56 (49.6)	1.00~(Base)	
Former	37 (35.9)	57 (50.4)	1.91 (1.03-3.55)	
Secondary outcome variables				
Perceived personal lung cancer risk*				0.711
Low	17 (25.8)	23 (29.5)	1.00~(Base)	
Moderate	27 (40.9)	34 (43.6)	0.76 (0.30-1.92)	
High	22 (33.3)	21 (26.9)	0.66 (0.24-1.79)	
Perceived average smoker lung cancer risk†				0.567
Very low to moderate	11 (16.2)	10 (12.8)	1.00~(Base)	
Somewhat high	20 (29.4)	25 (32.1)	1.69 (0.54-5.26)	
Very high	37 (54.4)	43 (55.1)	1.77 (0.61-5.17)	
Perceived relative risk of lung cancer				0.874
About the same to a little higher risk	6 (9.4)	8 (10.5)	1.00~(Base)	
Twice as high risk	10 (15.6)	8 (10.5)	0.82 (0.18-3.67)	
Five times higher risk	15 (23.4)	17 (22.4)	1.24 (0.31-4.95)	
Ten times higher risk	33 (51.6)	43 (56.6)	1.26 (0.37-4.32)	
Lung cancer worry*				0.274
Rarely or never	29 (42.6)	33 (42.3)	1.00~(Base)	
Sometimes	27 (39.7)	26 (33.3)	1.01 (0.46-2.21)	
Often or all the time	12 (17.6)	19 (24.4)	2.18 (0.79-6.00)	
				1

†Figures do not equate to 216 due to some missing data, ^a Multivariate logistic regression: adjusted for baseline age, gender, ethnicity, marital status, highest educational attainment, and quit duration, * p < 0.05

To compare the outcome variables logistic regression analyses were undertaken, to ensure that identified between-group differences at baseline were adjusted for (i.e. age, gender, ethnicity, marital status, highest educational attainment, and quit duration).

Table 7 demonstrates a significant relationship between treatment groups and 7-day point prevalence (p=0.040), which indicates that individuals appointed to the intervention group were 1.91 (95% CI 1.03-3.55) times more likely to be classified as former smokers at follow-up compared to those in the control group. No significant effects were evident between treatment groups and lung cancer risk perceptions, including perceived personal lung cancer risk (p=0.711), perceived average smoker lung cancer risk (p=0.567), perceived relative risk of lung cancer (p=0.874), and lung cancer worry (p=0.274), despite adjusting for the aforementioned baseline characteristics.

4.4 Discussion

The current chapter primarily investigated whether application of the LLP intervention is associated with follow-up smoking cessation success. Furthermore, the effect of the LLP intervention on lung cancer risk perceptions was investigated. Exploring these concepts helps to establish the efficacy of the LLP intervention in promoting smoking cessation and additionally improves knowledge regarding the mechanisms by which risk communication often influences health behaviour change. This chapter intended to investigate whether application of the LLP intervention is associated with smoking cessation success and lung cancer risk perceptions; the results, potential strengths and limitations, as well as implications will now be discussed in greater detail.

4.4.1 Exploration of chapter findings

Application of the LLP intervention was found to be associated with follow-up 7-day point prevalence (i.e. smoking status) among baseline recent former smokers, however, the intervention was not found to be associated with follow-up 7-day point prevalence among baseline current smokers. These differences in LLP intervention effect between the two datasets might indicate differences in psychological mechanisms implicated between current and recent former smokers in relation to the responses to the LLP intervention.

Behaviour change theories, such as the Transtheoretical Model of Change (TTM) (J. O. Prochaska & DiClemente, 1983; J.O. Prochaska & DiClemente, 1986), suggest that some processes of change are more applicable than others to individuals at varying stages throughout a quit attempt; this might provide some explanation regarding the differences in intervention effect between current and recent former smokers that occurred in the present project. For example, the TTM (J. O. Prochaska & DiClemente, 1983; J.O. Prochaska & DiClemente, 1986) stipulates that processes, such as "reinforcement management" are more applicable among individuals actively quitting (i.e. recent former smokers), whereas, the process "self-liberation" could be more applicable to current smokers progressing from the preparation to action stage of change (see Figure 2).

No significant effects were identified regarding the relationships between LLP intervention and lung cancer risk perceptions but again, the theoretical underpinnings of behaviour change theories, such as the TTM (J. O. Prochaska & DiClemente, 1983;

J.O. Prochaska & DiClemente, 1986), may provide explanation as to why the LLP intervention failed to be associated with follow-up lung cancer risk perceptions. For example, the LLP intervention may be particularly suited to the process of change, "consciousness raising", which is implicated among individuals progressing from precontemplation to contemplation (see Figure 2).

4.4.2 Potential strengths and weaknesses

The current chapter describes the results of the two RCTs implemented to explore the effect of the LLP intervention on follow-up smoking status among baseline current and recent former smokers. A range of questionnaire variables were explored, allowing exploration of various predictors of smoking cessation, including several perceptions of lung cancer risk. Furthermore, efforts were made to ensure that participants were blinded as to which treatment group they had been allocated to and a six-month follow-up was implemented in order to provide greater confidence that abstinence will continue long-term and that a degree of health benefit can be achieved (West et al., 2005). There are, however, some potential study limitations, which should be noted.

The original power calculation indicated that at least 673 current smokers were required to achieve appropriate statistical power (see Section 3.5.1) and it was therefore intended for only current smokers to be recruited. Recruitment was hampered by the lack of time and financial resources available to the PhD project; this led to an insufficient sample size to fully conclude the findings in relation to current smokers. In light of the lack of available current smokers, an exploratory study of recent former smokers was set-up to further explore differences in smoking behaviour and risk perception between the groups. Future research might benefit from inclusion

of a longer recruitment period, employment of more than one researcher, and, potentially expansion of the number or format of SSS recruitment sites included in the project.

Seven-day point prevalence was used to measure smoking status at baseline and follow-up and the measure has been argued to be highly advantageous, as measurement at six months can include smokers who delayed action and quit, enabling the dynamic, real-life process of smoking cessation to be captured (Velicer & Prochaska, 2004); however, some researchers recommend the use of prolonged abstinence (i.e. self-reported continuous abstinence since quit attempt) in addition to 7-day point prevalence to enhance reliability (Hughes, Carpenter, & Naud, 2010). If the trial was to be replicated, prolonged abstinence might be a favourable additional measure.

The Russell Standard, a standard outcome criteria for smoking cessation trials, also promotes the use of biochemical verification of smoking status at follow-up (e.g. exhaled carbon monoxide [CO]) (West et al., 2005); however, it was not possible to collate such data in the current project, again due to the lack of PhD project resources. Although biochemical verification may have been preferable, the value of self-reported smoking status should not be underestimated. Connor Gorber, Schofield-Hurwitz, Hardt, Levasseur, and Tremblay (2009) undertook a review, in which the accuracy of self-reports was explored. They identified that whilst self-reported smoking status can occasionally be under reported, it may not always be feasible to ascertain biochemical verification of smoking status in some studies. Furthermore, one review, which explored 26 studies found that self-reports of smoking were accurate in

most studies. The review surmised that sensitivity means and specificity means of self-report were both high when compared with biochemical indices; 87.5% and 89.2%, respectively (Patrick et al., 1994). Nevertheless, if the study was to be repeated, biochemical verification of follow-up smoking status should be considered, although this would require increased financial resources.

Future research may also benefit from implementation of a clustered randomised controlled trial design, as participants were recruited within RCFE drop-in sessions and some service users may have therefore provided others with details of project participation, which could contaminate treatment blinding. Clustered randomised controlled trials do control for "contamination" across participants, although they involve a more complex design and analysis, and require a greater number of participants to achieve a similar level of statistical power, compared to individual randomised controlled trials (Campbell, Elbourne, & Altman, 2004). Efforts were made to avoid contamination in the current RCTs, as participants were blinded regarding randomisation and participants were seen by the researcher on a 1:1 basis, in a confidential setting.

The current study established p < 0.25 as the appropriate variable entry level for multivariate analysis (Hosmer et al., 2013); p < 0.25 was deemed the most appropriate level, as traditional levels such as p < 0.05 can fail in identifying important variables (Bendel & Afifi, 1977; Mickey & Greenland, 1989). This criterion allowed for extraneous effects to be controlled for, however, future efforts could incorporate interaction terms in the analysis, to enable the identification of mediating and moderating variable effects, which may have been controlled for in the current study.

Lastly, the use of the LLP risk model in itself, as a risk communication tool, does yield some limitations. The risk model measures smoking duration, as opposed to age since quitting smoking (e.g. Peto et al., 2000). This may potentially influence the predictive accuracy of applying the model in this context, as although smoking duration and age since quit are likely to be highly associated, smoking duration alone cannot fully reflect quit behaviour. Furthermore, as risk was projected and some individuals were particularly young (i.e. 20-30 years), they may be exposed to further risk factors prior to reaching the age at which risk is projected for individuals (i.e. 70 years old). For these reasons, lung cancer risk may therefore be underestimated for some individuals.

4.4.3 Implications for knowledge, practice and policy

The current chapter findings have a number of important implications in relation to tobacco control knowledge, practice and policy. Despite the limitations of the study, the findings suggest that the LLP intervention promoted follow-up smoking cessation among recent former smokers, but not among current smokers. A recruitment extension or the implementation of a larger trial might address the current project limitations and provide further support for the use of the LLP intervention among certain populations.

If further research was able to replicate the current project findings, a cost-benefit analysis may be beneficial to consider the implementation of the LLP intervention within SSS. Quit rates reported by SSS have remained fairly consistent in recent years (NCSCT, 2014) and therefore, a new cost-effective intervention that is deliverable within SSS would certainly be welcomed. Furthermore, the current intervention is

designed in a manner that enables non-clinicians to communicate risk and therefore, SSS advisors would be able to deliver the intervention to service users, with basic training. Alternatively, the present intervention could be developed to enable delivery via post or electronically via SSS, thus potentially reinforcing quit attempts.

Future research could additionally explore the application of the LLP intervention among alternate populations, such as non-help seeking smokers. Considering the results alongside the literature, the LLP intervention might be successfully delivered among smokers who are pre-contemplating or contemplating behaviour change, rather than those already engaging in behaviour change. This is an area that certainly warrants further research, as interventions of this kind, may be more or less applicable to various audiences.

4.4.4 Conclusion

In summary, the current chapter presented the bivariate and multivariate analyses results, pertaining to the quantitative investigation. The chapter aimed to explore the utility of the LLP risk model in the context of smoking cessation and more specifically, the extent to which the LLP intervention is associated with follow-up smoking cessation success and lung cancer risk perceptions.

The results suggest that the LLP intervention may be associated with follow-up smoking status among individuals who have recently quit smoking but not among current smokers; however, further research may be required to address some of the current project limitations. If future research can provide further support for the application of the LLP intervention, there are a number of implications for tobacco

control practice particularly. Furthermore, the results of the current chapter have important implications in terms of researcher understandings pertaining to smoking and risk perception, providing further insight into the mechanisms by which smokers perceive risk and the subsequent impact on smoking cessation success. The following chapter will expand upon the findings in the current chapter and will consider additional predictors of risk perception more broadly, thus providing further insight into the relationship between smoking behaviour and risk perception.

Chapter 5: An exploration of factors implicated in smoking-related risk perception

5.1 Introduction

Whilst the previous chapter demonstrated that the LLP intervention was not associated with follow-up lung cancer risk perceptions, the current chapter aims to explore predictors of lung cancer risk perception more broadly. Furthermore, participants' attitudes and experiences regarding risk perception and communication are extensively investigated. By exploring the correlates and predictors of risk perception (including smoking behaviour), improved knowledge regarding risk perception and behaviour change can be achieved. In turn, a better understanding of the relationship between risk perception and smoking behaviour could inform the development of future risk communications. The present chapter addresses one key thesis objective:

• To explore factors implicated in smoking-related risk perceptions.

To achieve this objective, quantitative and qualitative analytic techniques were employed. Firstly, questionnaire data is referred to, as statistical analyses were conducted to explore associations by baseline perceived personal lung cancer risk. Bivariate and multivariate analyses were undertaken to establish associations. Several statistical models are described, which were developed to ascertain the extent to which baseline characteristics predict personal perceived lung cancer risk. Secondly, part of the results pertaining to the qualitative analysis are explored, with particular focus on service users' experiences and perceptions regarding smoking-related risk. Considering the results of both the quantitative and qualitative methodologies collectively will provide an enriched insight into the extent to which smoking-related

risk perception can be predicted and will inform the development of future smokingrelated risk communications.

5.2 Correlates and predictors of perceived personal lung cancer risk

The current section will explore the predictors of baseline perceived personal lung cancer risk. The aim of conducting these analyses is to explores differences in baseline participant characteristics, such as smoking status, by perceived personal lung cancer risk; therefore, all participants recruited for the PhD project who responded to the baseline perceived personal lung cancer risk measure (N = 502) were included in the described analyses. Bivariate analyses were conducted to identify potential associations with perceived personal lung cancer risk and subsequently, multivariate tests were undertaken to develop a parsimonious model for the prediction of perceived personal lung cancer risk.

Several bivariate analyses were undertaken firstly to ascertain which baseline participant characteristics may have differentiated across baseline perceived personal lung cancer risk levels. Table 8 provides an overview of baseline participant characteristics by baseline perceived personal lung cancer risk. All variables for which significance levels are below 25% are highlighted; these variables were deemed suitable to be entered in a multivariate model to predict baseline perceived personal lung cancer risk, as recommended (Hosmer et al., 2013).

The bivariate tests unveiled several baseline participant characteristics that were significantly associated with baseline perceived personal lung cancer risk, at the described level of 25%, including age (p = 0.007), ethnicity (p = 0.026), smoking

status (p < 0.001), age started smoking (p = 0.147), nicotine dependence (p = 0.045), perceived average smoker lung cancer risk (p < 0.001), perceived relative risk of lung cancer (p < 0.001), and lung cancer worry (p = 0.005). These variables will therefore be included in the development of a multivariate model for the prediction of baseline perceived personal lung cancer risk. No significant relationships were observed between baseline perceived personal lung cancer risk and the remaining variables, including gender, marital status, highest educational attainment, socio-economic status, living with another smoker, cigarettes per day, and perceived lung cancer survival.

Multivariate analyses were subsequently conducted to further examine the predictors of baseline perceived personal lung cancer risk. All aforementioned baseline variables at the level of p < 0.25 in Table 8, were included in an initial proportional odds (PO) regression model. The results regarding the initial PO regression model for predictors of baseline perceived personal lung cancer risk are displayed in Appendix N. The initial model was statistically significant, $\chi^2(12) = 90.932$, p < 0.001. The model explained 19.7% (Nagelkerke R^2) of the variance in baseline perceived personal lung cancer risk; however, the model was refined in line with recommendations (Hosmer et al., 2013) in order to create a more parsimonious model, which typically involves removing non-significant variables. The final PO model for the prediction of baseline perceived lung cancer risk is presented (Table 9). The final model was statistically significant, $\chi^2(7) = 88.113$, p < 0.001. The model explained 18.8% (Nagelkerke R^2) of the variance in baseline perceived personal lung cancer risk.

Table 8. Participant characteristics by baseline perceived personal lung cancer risk

Baseline per	ceived personal lun	g cancer risk	P-value §
Low		~	
(n = 123.			
,	,	,	
	43.0 (35.0-50.3)		0.007*
(, , ,	(,	0.709
72 (58.5)	115 (55.8)	97 (56.1)	
- ()		, ,	0.026*
104 (84.6)	195 (96.1)	160 (93.0)	
- (- · ·)	- ()	(****)	0.533
18 (14.8)	27 (13.2)	33 (19.3)	
		, ,	
05 (02.0)	,, (5,10)	00 (0217)	0.963
60 (50.0)	103 (50.2)	86 (50.3)	0.500
, ,	, ,	, ,	
00 (00.0)	102 (1510)	00 (1517)	0.433
106 (86.2)	177 (86.3)	144 (83.2)	0
17 (1010)	20 (1017)	2) (1010)	p<0.001*
53 (43.1)	116 (56 3)	121 (69 9)	p (0.001
	, ,		0.147*
13.0 (11.0 10.0)	13.0 (13.0 17.0)	13.0 (13.0 17.0)	0.117
			0.710
91 (74.0)	134 (65.0)	120 (70.6)	
, ,	, ,	, ,	
, ,	, ,	, ,	0.045*
, ,	, ,	, ,	0.746
,			p<0.001*
	54 (26.6)	14 (8.1)	P
, ,			
, ,		, ,	
, ,	()	(,	p<0.001*
	45 (22.6)	19 (11.0)	r
_, (,,	(==.0)	-> ()	
20 (16.8)	49 (24.6)	18 (10.4)	
(0-1.5)	(=)	, , (==,,	0.005*
47 (38.5)	63 (30.7)	48 (27.7)	
20 (23.0)	55 (20.5)	05 (50.1)	0.289
44 (37.6)	72 (35.8)	73 (42.9)	0.207
, ,			
		` '	
13 (11.1)	15 (0.5)	10 (7.7)	
	-	Low ($n = 123$, 24.5%)Moderate ($n = 206$, 41.0%) $40.0 (30.0-51.0)$ $43.0 (35.0-50.3)$ $72 (58.5)$ $51 (41.5)$ $115 (55.8)$ $91 (44.2)$ $104 (84.6)$ $19 (15.4)$ $195 (96.1)$ $19 (15.4)$ $18 (14.8)$ $65 (53.3)$ $39 (32.0)$ $27 (32.2)$ $77 (37.6)$ $60 (50.0)$ $60 (50.0)$ $103 (50.2)$ $102 (49.8)$ $106 (86.2)$ $17 (13.8)$ $17 (86.3)$ $17 (13.8)$ $28 (13.7)$ $37 (56.9)$ $90 (43.7)$ $15.0 (14.0-18.0)$ $15.0 (13.0-17.0)$ $91 (74.0)$ $32 (26.0)$ $6.0 (4.0-7.0)$ $20.0 (15.0-30.0)$ $134 (65.0)$ $20 (15.0-30.0)$ $20.0 (15.0-25.0)$ $\frac{1}{2} (22.7)$ $\frac{1}{2} (22.7)$ $\frac{1}{2} (22.6)$ $\frac{1}{2} (22.6)$ $\frac{20}{2} (16.8)$ $34 (28.6)$ $34 (28.6)$ $34 (28.6)$ $34 (28.6)$ $34 (28.6)$ $34 (28.6)$ $34 (28.6)$ $34 (28.6)$ $34 (28.6)$ $35 (28.6)$ $38 (31.9)$ $48 (24.1)$ $\frac{47}{3} (35.1)$ $47 (38.5)$ $47 (38.5)$ $48 (24.1)$ $49 (24.6)$ $41 (34.5)$ $48 (24.1)$ $\frac{47}{3} (35.1)$ $47 (38.5)$ $48 (24.1)$ $48 (24.1)$ $\frac{47}{3} (35.1)$ $48 (23.0)$ $58 (28.9)$ $23 (19.7)$ $58 (28.9)$ $23 (19.7)$ $58 (28.9)$	Low (n = 123, 24.5%) Moderate (n = 206, 24.5%) High (n = 173, 34.5%) 40.0 (30.0-51.0) 43.0 (35.0-50.3) 46.0 (35.0-53.0) 72 (58.5) 115 (55.8) 97 (56.1) 51 (41.5) 91 (44.2) 76 (43.9) 104 (84.6) 195 (96.1) 160 (93.0) 19 (15.4) 8 (3.9) 12 (7.0) 18 (14.8) 27 (13.2) 33 (19.3) 65 (53.3) 101 (49.3) 82 (48.0) 39 (32.0) 77 (37.6) 56 (32.7) 60 (50.0) 103 (50.2) 86 (50.3) 60 (50.0) 102 (49.8) 85 (49.7) 106 (86.2) 177 (86.3) 144 (83.2) 17 (13.8) 28 (13.7) 29 (16.8) 53 (43.1) 116 (56.3) 121 (69.9) 70 (56.9) 90 (43.7) 52 (30.1) 15.0 (14.0-18.0) 15.0 (13.0-17.0) 15.0 (13.0-17.0) 91 (74.0) 134 (65.0) 120 (70.6) 32 (26.0) 72 (35.0) 50 (29.4) 6.0 (4.0-7.0) 6.0 (4.0-7.0) 6.0 (4.5-7.0) 20.0 (15.0-30.0) </td

[†] Figures do not equate to 502 (i.e. those who completed the baseline perceived personal lung cancer risk measure) due to some missing data, FTND = Fagerström Test of Nicotine Dependence, \S Categorical variables were analysed using Kruskal Wallis H-test, however, binary categorical variables were analysed using Mann Whitney U-test, and continuous variables with Spearman's Rho, * p < 0.25

Table 9. Final proportional odds model for the prediction of baseline perceived personal lung cancer risk

Odds	Lower	Upper	<i>P</i> -value
(95% CI)	(95% CI)	(95% CI)	
1.030	1.013	1.047	p<0.001*
			p<0.001*
0.336	0.234	0.482	
<u>isk</u>			
2.569	1.588	4.154	p<0.001*
3.210	1.937	5.320	p<0.001*
1.103	0.621	1.961	0.738
1.314	0.772	2.236	0.314
2.157	1.273	3.658	0.004*
	(95% CI) 1.030 0.336 risk 2.569 3.210 1.103 1.314	(95% CI) (95% CI) 1.030 1.013 0.336 0.234 risk 2.569 1.588 3.210 1.937 1.103 0.621 1.314 0.772	(95% CI) (95% CI) (95% CI) 1.030 1.013 1.047 0.336 0.234 0.482 cisk 2.569 1.588 4.154 3.210 1.937 5.320 1.103 0.621 1.961 1.314 0.772 2.236

^{*} p < 0.05

The Wald criterion displayed that age (p < 0.001), smoking status (p < 0.001), and perceived average smoker lung cancer risk (p < 0.001) were significant predictors of baseline perceived lung cancer risk. Furthermore, participants who perceived the relative risk of lung cancer among smokers to be ten times higher than a non-smoker's risk, were significantly more likely to perceive their personal lung cancer risk as higher (OR 2.16, 95% CI 1.27-3.66), compared to those who perceived a smoker's relative risk of lung cancer to be about the same to a little higher than a non-smoker; however, no other levels were significant for the variable of perceived relative risk of lung cancer.

Subsequently, goodness-of-fit and parallel lines tests were conducted to assess the model in greater detail. The Deviance goodness-of-fit test was not significant, χ^2 (677) = 680.151, p = 0.459 and the Pearson goodness-of-fit test was not significant either, χ^2 (677) = 719.553, p = 0.125, indicating that the model fitted the data well; however,

the test of parallel lines was significant, $\chi^2(7) = 21.224$, p = 0.003, suggesting that the assumption of proportional odds among the levels within the outcome variable, had been violated (A. A. O'Connell, 2005).

The test of parallel lines has been described as being anti-conservative because it often results in rejection of the PO assumption, particularly when the number of explanatory variables is large (Brant, 1990) or there is a continuous explanatory variable in the model (Allison, 1999 as cited in A. A. O'Connell, 2005). It was therefore necessary to examine the data using several separate logistic regression analyses to explicitly investigate how the odds ratios (ORs) for the baseline predictor variables differentiate at varying thresholds.

Baseline perceived personal lung cancer risk was dichotomised at two levels, namely "Moderate or above" (Vs. "Low") and "High" (Vs. "Moderate or below"). Two separate binary logistic regression analyses were conducted to explore the ORs for the independent variables, across the separate levels in the data to assess the consistency (see Table 10). Furthermore, Table 10 demonstrates the results of individual tests of parallel lines that were conducted for each independent and dummy variable (where applicable), to further explore the PO assumption.

The first logistic regression analysis compared "Moderate or above" with "Low" baseline perceived personal lung cancer risk (Table 10). The model was statistically significant, $\chi^2(7) = 42.761$, p < 0.001, explained 12.7% (Nagelkerke R^2) of the variance, and the Hosmer-Lemeshow goodness-of-fit test indicated that the model fitted the data well, $\chi^2(8) = 3.431$, p = 0.904. Several variables were identified as

significant predictors, including age (p = 0.006), smoking status (p < 0.001), and both levels of perceived average smoker lung cancer risk (both p = 0.001); however, none of the levels attributed to perceived relative risk of lung cancer were significant.

The second logistic regression analysis compared "High" with "Moderate or below" baseline perceived personal lung cancer risk (Table 10). The model was statistically significant, $\chi^2(7) = 88.367$, p < 0.001, explained 22.9% (Nagelkerke R^2) of the variance, and the Hosmer-Lemeshow goodness-of-fit test indicated that the model fitted the data well, $\chi^2(8) = 8.616$, p = 0.376. The results of the second logistic regression analysis differed in some respects to the first. Age (p = 0.001) and smoking status (p < 0.001) were both found to be significant. In addition, for perceived average smoker lung cancer risk, both dummy variables, "Somewhat high" and "Very high" were significant (p = 0.002 and p < 0.001, respectively), whilst the perceived relative risk of lung cancer dummy variable, "Ten times higher" was highly significant (p < 0.001) but other levels were non-significant.

Separate tests of parallel lines were conducted to isolate variables which may have contributed towards the violation of the proportional odds assumption. Test of parallel lines were non-significant for age (p=0.927) and smoking status (p=0.998), indicating that the assumption of proportional odds was upheld for these variables.

Table 10. Results of two logistic regression analyses for "Moderate or above" and "High" baseline perceived personal lung cancer risk

Baseline variable	Moderate or above perceived personal	above percei	ved personal	High p	High perceived personal	ersonal	Test of parallel
	ll	lung cancer risk	sk	lul	lung cancer risk	isk	lines
	В	OR	P-value	В	OR	P-value	P-value
Age	0.029	1.030	*900.0	0.033	1.033	0.001*	0.927
Baseline smoking status							0.998
Former	-1.080	0.339	p < 0.001*	-1.078	0.340	p < 0.001*	
(Base = Current)							
Perceived average smoker lung cancer risk							
Somewhat high	0.990	2.690	p < 0.001*	1.074	2.927	0.002*	0.154
Very high	1.001	2.721	p < 0.001*	1.377	3.964	p < 0.001*	0.028*
(Base = Very low to moderate)							
Perceived relative risk of lung cancer							
Twice as high risk	0.198	1.219	0.597	-0.054	0.947	0.891	0.899
Five times higher risk	0.035	1.035	0.920	0.588	1.801	0.089	0.002*
Ten times higher risk	0.108	1.114	0.754	1.219	3.385	p < 0.001*	0.090
(Base = About the same to a little higher risk)							
JO 0							

* p < 0.05

With regard to perceived average smoker lung cancer risk, the PO assumption (p > 0.05) was upheld for "Somewhat high" but not for "Very high" (p = 0.028). On inspection of the OR for the level "Somewhat high", the assumption of PO is plausible; however, the OR for "Very high" is particularly high in comparison to the "Moderate or above" analysis. The results suggest that the cumulative OR for "Very high" (OR 3.21) slightly underestimates the under-representation of "Very high" in the "Moderate to above" level (OR 2.72) and somewhat overestimates the under-representation in the "High" level (OR 3.96); however, the directions of the coefficients are consistent across the data divides and the result is statistically significant across both models. This might suggest that the OR from the ordinal model may provide a fair representation of the overall pattern of individuals who perceive average smoker lung cancer risk as "Very High".

The PO assumption was also upheld for all levels of perceived relative lung cancer risk, except "Five times higher risk" (p = 0.002). "Five times higher risk" failed to display a significant value in neither the ordinal nor logistic regression models; however, the OR for "Five time higher risk" is particularly high in the binary logistic regression model for "High" perceived personal lung cancer risk (OR 1.80), compared with the model for "Moderate or above" (OR 1.04). This suggests that the cumulative OR for "Five times higher risk" (OR 1.31) somewhat underestimates the underrepresentation of "Five times higher risk" in the "Moderate to above" level. Again, the variable level "Twice as high risk" failed to display a significant value in neither the ordinal nor logistic regression models; however, the test of parallel lines and the OR for "Twice as high risk" would suggest that the assumption of PO is plausible.

Perhaps most notably, the test of parallel lines was non-significant for "Ten times higher risk", which was a significant predictor in the ordinal model (p = 0.004); however, the logistic regression analyses revealed that the dummy variable was non-significant for the "Moderate or above" model (p = 0.754), yet highly significant for the "High" model (p < 0.001). Upon inspection of the ORs, these differences became increasingly apparent; the OR for "Ten times higher" were substantially lower in the "Moderate or above" model (OR 1.11), compared with the model for "High" (OR 3.39). The cumulative OR, therefore, largely underestimates the under-representation of "Ten times higher" in the "Moderate or above" comparison.

Consideration of the individualised ORs for a number of binary logistic regression models suggests that the PO model (Table 9) is a reasonable overview of the data patterns in regard to baseline perceived personal lung cancer risk; however, it should be noted that a small number of the variable levels may be under-represented in regard to high risk perception, including perceived average smoker lung cancer risk and perceived relative risk of lung cancer (particularly the level, "Ten times higher") and therefore, this should be considered prior to generalising across all cumulative levels.

5.3 A qualitative exploration of smoking-related risk perception

The current section will explore the results of the qualitative investigation pertaining to smoking-related risk perception and communication. This section aims to better understand smoking-related risk perception among smokers, by considering participants' experiences of risk perception more broadly in relation to smoking and by examining responses to the study intervention, with the intention of establishing facilitators and barriers to risk communication salience. Two major themes emerged

from the analysis: (1) Increased risk awareness; (2) Disregarding risk. Both themes will now be detailed.

5.3.1 Increased risk awareness

"Increased risk awareness" was one of the prominent themes identified throughout the investigation of smoking-related risk perception. The theme explores the factors that participants identified as influential in their experiences of smoking-related risk perception. This section also documents examples of how risk communication may have enhanced smoking-related risk awareness among participants. This major theme consisted of three prominent sub-themes, which included "Age and risk", "Social comparison", and "Influential risk communication". These sub-themes will now be described and explored.

5.3.1.1 Age and risk

Nineteen participants described how they started smoking in their youth and how they were rarely concerned regarding smoking-related risk at the time of initiation; this was reflected across the full age range of participants. One participant said, "It never even passed me mind I don't think. Well, what would happen to meself and others around me, I never took it into consideration, I just done it" (Robert, 34, Current). Another participant also described how even with an acknowledged increase in public risk awareness, this failed to deter smoking initiation.

... not when I was younger, I never thought about it at all. Even when they started putting the warnings on the packets, I never thought about it. I should

have done I suppose, in hindsight but I didn't. I was young, foolish. (Patrick, 56, Former)

Several participants described how they had previously considered smoking-related risk upon initiating smoking, but that these concerns had been minimal. The results suggested that these low perceptions of perceived risk were attributed to two primary concepts: (1) youthful smokers failing to experience the immediate negative effects of smoking; (2) optimistic bias during youth regarding personal perceived risk of smoking-related disease. The term optimistic bias was referred to in Chapter 2 but in brief, optimistic bias may occur when individuals continue to practice unhealthy behaviours due to inaccurate perceptions of risk and susceptibility (Weinstein, 1983, 1984).

Three participants believed that smoking was not associated with any negative health consequences in youth, and the results suggest that this belief had contributed towards smoking initiation or continued smoking in youth. One participant explained, "When you're young, you're fit and healthy, it doesn't really affect you, you think, 'I don't know what they're talking about, I smoke and I'm fine' " (Sarah, 55, Former). Another participant described, "I was young, erm, and the body is more resilient when you're young and you just don't worry about it" (Luke, 54, Current).

Three participants demonstrated optimistic bias in youth regarding personal perceived risk of smoking-related disease. The results suggested that young people often fail to consider the development of smoking-related disease, or potentially they do consider it but they are not greatly concerned. Furthermore, observations of older smokers who

appear healthy provided reassurance for smokers during their youth regarding the development of disease. One participant explained, "... 'cause I wouldn't even expect nothin' like that, you know, when I first started ... when I was younger, I wouldn't even expect nothin' like that to happen, do you know what I mean?" (David, 37, Current). Another participant described how he underestimated personal perceived smoking-related risk in youth, as he observed older smokers with seemingly no health concerns; this appeared to reinforce his sense of optimistic bias and encouraged him to continue smoking.

In my opinion, young people who smoke, they just keep smoking. I don't think health warnings are going to frighten them. 'Cause they look around themselves and if they're only twenty and they see people in their sixties and seventies smoking, they're going to be thinking, oh, there's nothing wrong with them and they just carry on smoking like. I used to think that meself. (Patrick, 56, Former)

The majority of participants described how public awareness of smoking-related risk has substantially increased in recent decades. Two participants provided examples of how the landscape of tobacco control has changed over recent decades. One participant commented on the change in advertising regulations since he started smoking, highlighting how society's attitude towards smoking-related risk has changed.

I think then because I'm what, forty-five, so I think, when I was eleven, the adverts on the TV were for, erm, not just cigarettes but for pipes, smoking

pipes and cigars, adverts were on the billboards and in the papers. (Gavin, 45, Former)

The lack of tobacco control legislation and risk communication appeared to enhance the normalisation of smoking in previous decades and therefore, promote uptake. Another participant also stated that when he started smoking, fewer people were aware of smoking-related risk. Furthermore, the participant suggested that smoking had been previously prescribed to alleviate symptoms associated with poor health.

It wasn't something that the general populous was aware of, never mind a seven year old, bearing in mind I'm forty years old now, so we're talking thirty-odd years ago. The dangers of smoking were not as widely known among the scientific community, never mind the general populous ... back in them days, that time, if you were asthmatic, this is more applicable to the adults than as a child, but if you were an asthmatic and had been admitted to hospital with an asthma attack, they would actually give you a fag in the morning. The whole point was being that it'd help cough up the mucus on your chest. (Charles, 40, Current)

Several participants attributed limited risk awareness to smoking initiation, highlighting the lack of tobacco control and risk communication strategies in previous decades. Such comments were also irrespective of age, suggesting that smokers were viewing risk awareness comparatively to the present, rather than to a specific time point whereby certain legislation or guidance was introduced. For example, one older participant explained, "There was no like health scares or things like that back at that time, to what there is now. Compared to what they know now about smoking, there

wasn't that much on it back in them days" (Alex, 56, Current), whilst, a younger participant commented, "It was early nineties, so no one really knew anything about it, well they knew but they were like, it's just a myth and stuff like that, as I got older, I sort of realised it was an issue" (Matilda, 33, Former). These comments highlight the lack of risk awareness pertaining to smoking among service users upon initiating smoking. Although youth is commonly associated with risky behaviours, such as illicit drug usage, alcohol abuse, and smoking (see France, 2000), the present results suggest that poor risk awareness appeared to exacerbate smoking initiation in youth.

The findings suggest that smoking-related risk perceptions are dynamic throughout the lifespan, as risk perception changes were observed among participants from youth to present day. Ten participants broadly described how with age, their personal perceived smoking-related risk had increased, making them more conscientious of smoking and risk. Although such comments were made by participants of varying ages, the majority of participants (n = 7) were over fifty years of age. One participant described, "I mean it does give you cancer, smoking, and erm, I mean, as you're getting older and things, you tend to think about it more" (Michael, 48, Former). Another participant explained how they had experienced declining health with age, and that with increasing exposure to smoking-related health campaigns, they related their poor health to smoking.

Erm, I think it's when you get older and you start to feel the effects of smoking and when you see advertisements about how side effects of what smoking can do, then you can relate to them as you're getting older. (Sarah, 55, Former)

More specifically, some age milestones or life events were additionally observed as being influential in the transformation of risk perception. Four participants described how "milestone" birthdays influenced the way in which they perceived smoking-related risk, with many participants describing how ageing promoted intentions to quit.

Well it was age. It was one of those things. I said I'm getting too old to smoke now. I decided about a year ago I wanted to quit but I never got round to it ... well I'm fifty-five on me next birthday, which is a couple of weeks and I wanted to use that as a target to say I've quit smoking. (Luke, 54, Current)

This comment demonstrates how milestone birthdays may facilitate intentions to quit, providing smokers with a target timeframe to work within. Another participant commented, "... also I think it is, I'm getting older now and I'll be sixty next year and I think, oh god, I haven't got long left now! (laughs) What's going to kill me?! (laughs)" (Jean, 59, Former). This comment also demonstrates how the increased perception of risk associated with ageing, appears to be underpinned by fear of disease or mortality. This was apparent among a number of other participants. For example, one participant described how they intended to quit smoking with the aim of reducing risk of smoking-related disease and prolonging life.

Well getting lung cancer and all that now. That's what it is, I'm petrified to death, and that's what goes through my head, I know it all comes to us one day but I am, I'm, I'm, trying all kinds of stuff so I can have a longer life ... (Sophie, 55, Former)

Three participants additionally described how significant others were an important aspect of the reported fear of disease and mortality, which many participants attributed to motivation to quit. One participant described how she was responsible for the care of her daughter who had special needs, and expressed concerns that smoking could contribute towards deterioration of her health, which could ultimately impact her daughter's care, "Well I've been trying to cut down for months and months and, erm, I was worrying because I'm getting older and I've got me daughter there to look after" (Gemma, 57, Current). Another participant described how she had young grandchildren and her concerns regarding smoking and mortality appeared to be partially attributed to her wishes to be present for her grandchildren's upbringings.

I look at them and I think, you know, "I wanna be here when you're older" ... you know, to watch them growing up. Think the main thing is these three new babies 'cause they're so close together and they're only babies and I'm thinking, that was what I said, I thought, I want to see these grow up. (Jean, 59, Former)

5.3.1.2 Social comparisons of risk

The majority of participants (n = 17) provided examples of others whom they believed to have developed smoking-related disease and these experiences appeared to heighten perceived personal risk. Half of participants described significant others who had been affected by smoking-related disease. Participants typically described cases of smoking-related disease whereby significant others had developed cancer, particularly lung cancer. One participant described how lung cancer was common among members of his family, "Yeah well, I've known people in my own family that have died because

of smoking. Like me granddad, three of my uncles have died of lung cancer, they were all like smokers, they all died" (Michael, 48, Former). Another participant described how a family friend suffering with disease attributed to smoking, had advised them to stop smoking.

My mate's mum died of lung cancer and I still think of that and that was years ago, years and years ago and she always used to say to us, "all pack in smoking" and every now and again that goes through your head. (Sophie, 55, Former)

Such comments demonstrate how the experiences of significant others, whether first degree relatives or more distant family or friends, can resonate and have a lasting impact on smokers' perceptions of risk and worry.

A small number of participants explicitly described how significant others' experiences of smoking-related disease influenced their intention to quit smoking. One current smoker described how his cousin had been in hospital, with complications the participant described as attributed to smoking. The participant suggested that this event had influenced his intention to quit smoking.

I only have to look at my cousin in hospital, look at her, she stopped smoking, she stopped smoking for 12 months and look at her, so "Hello!" If I don't have the wake-up call now, I'll never have the wake-up call, will I? You know? (Jack, 49, Current)

Another participant described how her parents had died from cancer, which she had attributed to smoking; she described how this incentivised her to quit smoking, "A big incentive for me to stop because both parents died of that, I don't like mentioning the word, that thing that begins with C" (Timothy, 58, Former). This comment also demonstrates how some participants preferred not to speak about smoking-related diseases, such as cancer, presumably due to fear of disease.

5.3.1.3 Influential risk communication

As previously described within this chapter, participants described how public awareness of risk had increased in recent decades; in addition to this, participants observed a substantial increase in risk communication regarding smoking alongside the increased public awareness of risk. At least four participants felt that risk communication focusing on smoking had become increasingly prevalent, with some describing such communication as almost ubiquitous. These participants also suggested that increased risk communication exposure had promoted their motivation to quit smoking. One participant stated, "Yeah, I think what it is, everywhere you go, everywhere you turn around, there is some sort of information about smoking but it's more in your face and it does make you think hard about it. You know?" (John, 53, Former). Another participant explained, "Hopefully, it's all the publicity and all the stuff that's written about it. You know? That's basically what it is, isn't it? ... It's changing people's minds and the way they view it isn't it?" (Shaun, 48, Current).

Three key vehicles for risk communication were prominently discussed throughout the interviews: cigarette packaging; television; and, the LLP intervention. It should be noted that the interview schedule included questions pertaining to the LLP intervention, whereas, cigarette packaging and television were not original components of the interview schedule.

Eleven participants discussed their opinions regarding the use of cigarette packs in communicating risk to smokers. One participant referred to the messages conveyed on cigarettes packs, "It's there on the (cigarette) packet, it's telling you, you're killing yourself or you're doing serious damage to yourself, one of them, whichever way you want to put it" (Jack, 49, Current). All other participant comments referred to the images displayed on cigarette packaging. One participant explained, "They put scary things on cigarette boxes didn't they? ... Those things on where there's lungs and what it could do to you, I thought that was a good idea" (Timothy, 58, Former). Another participant suggested that they found the images repelling and described efforts to ignore such images whilst smoking, "All those pictures on it ... it's just not nice. Even I've done, when I have a smoke, I tell them to turn the packet upside down but it stays in your mind what you're doing to yourself" (Jacob, 59, Former).

The majority of participants who discussed risk communication and cigarette packaging believed the images of smoking-related disease to be an effective method of promoting risk communication and discouraging smoking. One participant described how the images led him to question his personal perception of smoking-related damage and encouraged him to stop smoking.

I think some of the adverts actually hit home and make you want to stop, like when you see these pictures on the back of cigarette boxes, like people with half a throat missing and pictures of a black lung and stuff like that. That's when you, you start thinking, like what do my lungs look like? What's it doing to me? (Robert, 34, Current)

Another participant described the images as unpleasant and seemingly repelling, "I know when they started putting the lung things on ... I used to look at that and go "Urgh! Oh my god!" You know? To meself" (Sophie, 55, Former). Other participants described efforts to ignore or deny risk messages on cigarette packs and this will be discussed in greater detail in the subsequent section (see Section 5.3.2.1).

Television was also frequently described as a noted medium for smoking risk communication by several participants (n = 11). One participant was able to recall the details of a recent television campaign, suggesting that the imagery was memorable, "Well, that last one advert, you know with the cigarette, the pigment in the skin, that was a really, really horrible one, you know?" (John, 53, Former). One participant described an advertisement they had viewed, which depicted the association between cardiovascular disease and smoking, "Every time there's an advert, there was an advert where they'd have a drag of a cigarette and it was the artery blocking up ..." (Paul, 38, Former). The participant continued to explain how such television campaigns had prompted him to consider his personal risk, "... and you know things like that make you think 'Eeeh, why do I smoke?' You know? There's no need for it. So I do, I think those adverts definitely do work" (Paul, 38, Former). Another participant described how a similar campaign had influenced his friend's children to question their mother's smoking, which he described as thought provoking for the mother.

A friend of mine, her children had ... seen the propaganda on TV and the mum was out having a cigarette in the backyard ... and the children actually came out and were speaking to the mum and said, "Mum, do you want to die?" She was very puzzled and taken aback by that and she turned around and said "What do you mean?" and the children went "Well, you're smoking and we all know cigarettes kill you, so do you want to die?" and I mean, I think things like that are more, certainly more motivators for families. (Charles, 40, Current)

Although several participants commented on the prevalence and efficacy of television campaigns in promoting smoking cessation, all participants appeared to approve of them and no participants suggested the campaigns were ineffective.

Lastly, the LLP intervention was discussed among participants who had received the intervention. The intention of the intervention was to improve smoking cessation success and increase risk awareness. The qualitative analysis suggested that for some participants, the LLP intervention had been memorable, as five participants were able to recall the projections they had been provided and some participants described how they still possessed the written piece of risk communication they were provided. One participant reiterated, "Yeah, I do (remember the LLP intervention); 10% if I carried on smoking and 5% if I quit" (Luke, 54, Current), whilst another participant stated, "I've still got it upstairs somewhere in me drawer. I think mine was 3% ..." (Patrick, 56, Former).

The majority of participants (n = 10) who received the LLP intervention in the qualitative sub-study, approved of the intervention, with comments suggesting that the LLP intervention might be beneficial as a motivational tool for smoking cessation. A small number of current smokers described how they felt the intervention may have contributed towards reinforcing their motivation to quit smoking, "It shed some light, as I say, I wasn't too sure what figures I'd get back from you but as I say, it's shed some light, it's reinforced me target that I should quit smoking" (Luke, 54, Current). Another current smoker suggested that the intervention had an effect, albeit small, on his motivation to quit, "Erm, I suppose it affected it a little bit, you do think about those things, so obviously it affects it a bit" (Shaun, 48, Current).

Some recent former smokers additionally provided similar accounts regarding the study intervention. One recent former smoker described how the study intervention reinforced her quit attempt, similarly to receiving feedback pertaining to exhaled CO when attending RCFE, "Oh yeah, that does (affect motivation) and the same as when I get the, erm, you know when I blow into the thing (the CO monitor)? Yeah, it just gave me a boost" (Gemma, 57, Current). Another recent former smoker additionally described how the risk projections he had received reinforced his quit attempt.

Oh it's a big difference, a massive difference if you've got that little bit of a scare in the back of your brain. That's another incentive to pack it in ... there's no way I want to cop to that is there? (Timothy, 58, Former)

As previously described, fear appeals have been described as "communications presenting the threat of impending danger to motivate compliance with a proposed recommendation" (p. 233) (Keller & Lehmann, 2008, as cited in Orazi & Pizzetti,

2015). The above comment also demonstrates how the LLP intervention incorporated aspects of fear appeals, as the aforementioned participant expressed fear or vulnerability to lung cancer and suggested that the LLP intervention would enhance his motivation to maintain abstinence for this reason. Several other participants commented on the notion of fear appeals. For example, one participant described how she found the risk communication to be provocative and frightening.

Yeah, well when you look at it and you was like, if you carried on smoking and this, you know, what your percentages were like, it is frightening because I think anyone in their seventies now, that's not old ... You just don't realise what cigarettes are doing to your body." (Julia, 49, Former)

Despite some positive remarks, several participants responded apathetically to the risk communication. Notably, no participants suggested that the risk communication discouraged their quit attempt; however, five participants suggested that the risk communication had little, if any, effect on their motivation and awareness of risk. Primarily, participants described how they were satisfied with their current quit attempt progress and therefore, felt that external factors had little impact.

Erm, well, I sort of knew about most of it anyway ... It didn't really come as a surprise in anyway, you know, it was like a sort of update for me really, that was it really ... I'm happy with the situation, like where I'm up to now, so I'm happy with the way things are going (laughs). (Oliver, 54, Former)

Another participant was asked whether they felt the risk communication influenced their motivation either positively or negatively, and they responded apathetically, "I don't know really because I've never ever been for anything like that or heard anything like that, or tested anything like that. That's the first time I've been tested for anything like that, lungs or anything like that" (Sophie, 55, Former). This comment also demonstrates how some participants highlighted that they were already substantially aware of smoking-related risk, which potentially reflects well on current tobacco control campaigns to improve risk awareness.

It should be noted that participants additionally described limiting features of the LLP intervention and detailed barriers to risk communication more broadly; this is explored further throughout the following theme, "Disregarding risk".

5.3.2 Disregarding risk

"Disregarding risk" is the other prominent key qualitative theme explored in the current chapter. Similarly to the previous theme, this theme explores risk perception and communication but it focuses on factors that may contribute towards reduced concerns regarding smoking-related risk, which could potentially promote continued smoking. The barriers to effective risk communication are considered within this section, with particular focus on the LLP intervention. This major theme consists of three key sub-themes, which include "Denying risk", "Risk contextualisation", and "Barriers to risk comprehension".

5.3.2.1 Denying risk

As described earlier in this chapter, participants acknowledged increased awareness of smoking-related risk and observed increases in smoking-related risk communication within society. Despite these observed increases, some participants described the experience of being aware of smoking-related risks, yet simply endeavouring not to focus on them. One participant described, "I've always known the risks to be honest but you don't want to think about the risk while you're smoking, do you?" (Robert, 34, Current). Another participant described how he experienced only fleeting thoughts whereby he considered smoking-related risk whilst smoking.

I have always felt and thought about things like that but it never ever stopped me from smoking at the time. One minute it goes through your head and the next minute, in the blink of an eye, it's forgotten. (Sophie, 55, Former)

These comments demonstrate how smokers attempted to ignore the risks associated with smoking whilst smoking, potentially as a means of dealing with cognitive dissonance. Cognitive dissonance was described in greater detail in Chapter 2; in brief, cognitive dissonance might occur when a smoker holds at least two opposing but related cognitions, which can result in mental conflict (Festinger, 1957, 1962). For example, dissonance may develop when a heavy smoker continues to receive smoking-related risk communication; the smoker may ignore or deny further risk communications, in an effort to reduce mental conflict.

Some participants explored the barriers to acknowledging risk further. Participants (*n* = 6) attributed the experience of addiction to the lack of risk acknowledgement whilst

smoking, as addiction was perceived as overpowering. One participant explained, "I think the nicotine has such a strong hold on you, that it puts, the danger of it doesn't come across, do you know what I mean? Because you're so addicted to it" (Stuart, 45, Former). Another participant suggested that smokers are aware of the risks of smoking and often want to quit but simultaneously, the strength of the addiction to cigarettes is overwhelming and creates challenges in quitting.

You know it's wrong and you know you shouldn't be doing it but it's not as easy as saying, you shouldn't be buying it anymore ... smoking is a disease, it is an illness, once you get hooked, it is, takes a while to get it out the system, to like come off it, especially for some people. (Gavin, 45, Former)

One current smoker actively demonstrated this conflict between risk awareness and addiction, as smoking-related disease was discussed, "Well now we're talking about cigarettes, has made me feel I want one, isn't that mad?" The interviewer responded, "It's interesting because you're telling me about all the health side of things..." and the participant responded, "And I want one." The interviewer continued to ask, "And how do you explain that?" The participant attributed this response to the intense withdrawals and psychological mechanisms.

It's a craving ... you know it's doing you no good but you still want it, it doesn't make sense. It's withdrawals, you're addicted to nicotine, so your body is just screaming out for it. I think a lot of it is mind over matter because until I've started talking to you, I wasn't thinking of one. (Joel, 44, Current)

Finally, the analysis identified conflicting statements among two participants with regard to risk perception, suggesting that the participants were potentially minimising smoking-related risk. Both examples were among current smokers. One participant was initially asked what he felt had motivated him to stop smoking, the participant replied, "Since I got this COPD. Err, like, chest infection thing you know" (Alex, 56, Current); however, later in the interview, the participant explained, "If (cigarettes) were still round about £3 or £4, I'd still probably be a smoker but erm, erm, with them putting the prices up all the time, it's forced my end" (Alex, 56, Current). This comment displays how initially the participant attributed his motivation to quit to ill health, yet later he acknowledges that he would have continued to smoke, if not for the increasing price of cigarettes.

Another participant, who had suffered strokes, which his doctor had attributed to smoking, also demonstrated conflicting cognitions. The participant initially associated his smoking with the occurrence of strokes; however, the participant continued to suggest that he was not exhibiting any effects associated with smoking, "Yeah, me strokes are related to the smoking, yeah, ... well, I still don't think I'm suffering any effects from smoking at the moment but I know I will in the end" (Joel, 44, Current). These conflicting statements suggest that some current smokers minimise the damage attributed to smoking; minimising the associated mental conflict may aid continued smoking.

5.3.2.2 Contextualising risk

Perceptions of smoking-related risk were often contextualised among participants, alongside other external factors deemed significant by participants, such as

uncertainty, perceived lack of control, and other risks more widely. The analysis demonstrated how uncertainty could play an important role in the formation of smoking-related risk perceptions. This chapter previously discussed how the majority of participants described significant others whom they believed to have developed smoking-related disease; however, some participants questioned whether the disease described was smoking-related.

I've found that I'm going to more funerals that what I'm going to Christenings and it's people that when I was younger. They were all older than me and now they're all dying and a lot of them were smoking-related. Well ok, you can't say for definite it was smoking-related ... (Ronald, 40, Former)

One participant explained, "I've heard of people who've had breast cancer and you think, is this down to cigarettes? The smoking? You know?" (Julia, 49, Former). Despite strong associations between smoking and various diseases, it is often not possible to definitively attribute smoking to the development of a given disease; this uncertainty is communicated by participants but it is unclear as to the extent to which uncertainty influences perceptions of smoking-related disease in the current study.

Perceived lack of control was another important component of risk perception discussed, which was underpinned by uncertainty. Five participants denoted a perceived lack of control in relation to the development of disease. Participants provided examples of seemingly healthy, non-smokers, who had developed diseases often associated with smoking, or alternatively, examples of older smokers who were

disease-free. For example, one participant referred to a non-smoking athlete who had developed lung cancer.

People will get lung cancer regardless of whether they smoke or not, down to various environmental factors. One great athlete was someone called Lilian Boyd and she died of lung cancer but she didn't smoke a day in her life. (Luke, 54, Current)

Another participant described how a number of her non-smoking, significant others had died at a relatively young age, "I've known loads of people, I mean, my Uncle was 44 and he's died, and he's never smoked in his life. Me Aunty, she was only 50 and she's never smoked in her life" (Charlotte, 26, Former).

In addition to the aforementioned accounts, at least four participants explicitly described a perceived lack of control with regards to smoking-related disease. One participant explained how she reacted to individuals who were critical of her smoking, "If anyone said anything I'd say 'Look, when your number's up, your number's up, no matter what you do" (Patricia, 50, Former), whilst, another participant described the inevitability of disease, "It was kind of one of these, you know, well if you're gonna get it," (Charlotte, 26, Former).

Specifically to lung cancer risk, the analysis also revealed how participants often failed to consider the importance of lung cancer risk, as the risk of lung cancer was contextualised among other risks that participants described exposure to. The results of the analysis also suggested that quit attempts did not take precedence when

considered in the context of additional stressors. One participant described how he had previously been homeless and alcohol dependent, deprioritising smoking cessation in favour of addressing other difficulties.

I lost my home and everything through drink, do I want to go back to that? I don't think so! So, I've had to go on the streets and everything to wake up and smell the coffee, building your life, and I did say when I was getting me life back and all that, the next thing that's going is them stupid ciggies. (Jack, 49, Current)

Another participant explained how he started to suffer with depression after a relationship breakdown. The participant emphasised how he concentrated his resources in addressing other lifestyle stressors, which were considered a priority, rather than focusing on smoking cessation.

I think that I was, I was coming down with, err, at the time, I didn't know I was starting to suffer with depression so, the likes of things like that (smoking cessation), you don't really worry about things like that ... I wasn't sleeping and everything else ... so for me it wasn't about packing the cigarettes in, it was about changing me whole lifestyle. (Gavin, 45, Former)

Two other participants also demonstrated how smoking and lung cancer risk were contextualised among other potential risks, demonstrating how resources can often be focused on broader areas of risk, as opposed to lung cancer alone, for example. One participant described over-saturation of information pertaining to risk factors, leading

her to dismiss communication regarding other risk factors, "It doesn't really matter, I mean everyone says, 'Oh don't have that because that can give you cancer' and 'Don't have that because, don't go on the sunbeds because that can give you cancer'. Like, there's always something" (Charlotte, 26, Former). One recent former smoker was asked how she felt about her lung cancer risk being reduced as a result of stopping smoking and she responded, "I've never give much thought about when you pack in smoking, about it all going down, I haven't given it much thought, all I was concerned about was the amount of people that do die because they smoke" (Jean, 59, Former). The latter comment suggests that the participant was primarily concerned regarding overall disease and mortality in relation to smoking, as opposed to lung cancer specifically, which again demonstrates the range of potential risks smokers might consider. This concept also interlinks with the following section, "Barriers to risk comprehension", as risk contextualisation may result in some sources of risk communication being neglected or being viewed as unimportant.

5.3.2.3 Barriers to risk communication saliency

The current section focuses on the barriers to risk communication saliency. The majority of this section refers to the LLP intervention, as participants who received the intervention were posed a number of questions regarding their response to the LLP intervention. Furthermore, some participants described additional barriers to risk interpretation and comprehension, which are discussed.

A number of participants (n = 4) provided comments, which demonstrated a lack of understanding regarding the LLP intervention. One participant thought that the two risk projection scores (see Figure 7) compared a smoker and a non-smoker, rather than

the participant's comparative scores between continued smoking and smoking cessation, "It was quite a shock, erm, especially when you see a person's lungs who hadn't smoked compared to a person who had smoked. The difference between them is quite alarming!" (Alex, 56, Current). Three of the four participants also demonstrated a level of misunderstanding, as they suggested that with smoking cessation, their risk could have been reduced further than the risk projections suggested. One participant explained, "Well, I'm hoping that I can get lower (laughs)" (Sophie, 55, Former). Another participant felt that her risk may have declined more so since being delivered the intervention, as she had since cut down the number of cigarettes consumed per day.

Mine said if you continue smoking, at the age of 70-74 years old, your risk will be 12% and then, if you quit smoking from now on, your estimated risk of getting lung cancer will be 9%, so I've reduced them again since then, so hopefully it'll go down. (Gemma, 57, Current)

This suggests that some participants failed to understand that the LLP intervention provided a comparison of projected lung cancer risk, based on continued smoking or smoking cessation. This finding also fits well with the aforementioned results regarding uncertainty and risk, as often participants remained potentially unrealistic regarding risk, despite being provided with an arguably objective measure of risk. Furthermore, although the LLP risk model has demonstrated good discrimination between cases and controls (AUC 0.71) (Cassidy et al., 2008), it is not possible to account for all potential lung cancer risk factors (e.g. genetic factors), meaning that deviation may occur between projected and actual risk; this knowledge may have

further contributed towards the ideal adopted by some participants, that lung cancer should be substantially lower than projected with smoking cessation.

One participant additionally demonstrated a lack of understanding in relation to the imagery displayed on the LLP intervention (see Figure 7), which was included to demonstrate the risk projections visually, "To tell you the truth, I didn't understand it. I'm just looking for it now ... I didn't understand all the little square things ... Yeah, I just didn't understand what they were" (Gemma, 57, Current). This also suggests that the visual component of the LLP intervention may have been difficult to comprehend for some participants.

One of the key issues that emerged in relation to the study intervention, was the perception that the levels of risk projected were very low. The median risk projection for participants overall who continued to smoke was 6.46% (IQR = 5.52-6.69), whilst the average risk projection for participants who continued to abstain from smoking was 1.66% (IQR = 1.11-3.40); several participants anticipated the percentages associated with continued smoking to be considerably higher. One participant commented, "Well higher, yeah ... when I was smoking, I expected it to be higher, I think it was only 7, whatever it was, it was only really low, like I didn't expect it to be so low" (Charlotte, 26, Former). Another participant additionally felt that her risk projection for continued smoking was particularly low, "I actually expected my lung cancer chances to be higher because everyone's like, 'Oh, you're going to get lung cancer if you smoke' and I think mine was 6% which I thought, well, that's still quite low" (Matilda, 33, Former). The participant continued to provide her estimation of lung cancer risk, if she had continued smoking, "You know what I expected it to be

like up in the 20, maybe 30% range, that was like the figure I had imagined in my head. If I carry on smoking, there's a very good chance I will die of lung cancer ..." (Matilda, 33, Former). One participant suggested that the risk projections were so low that they could encourage some smokers to continue smoking.

My sister's a hardcore smoker and reason is, it would validate her to carry on smoking, "That's a minimal percentage, oh, I'll carry on". So, 'cause they'll want an excuse anyway and you're kind of, the University of Liverpool are saying I've got a 6% chance of getting lung cancer between 70 and 74. I've known people in their 70s die anyway and it's all to do with the quality of life and people do think they are getting the quality of life they need through smoking and then not smoking and going through all the hassle and all that and I'm only going to get 6%; it's a bit crap that! (Sarah, 55, Former)

This comment also demonstrates how some smokers may perceive smoking to add to their quality of life and therefore, if such risk projections portray a minimal reduction in lung cancer risk from quitting, some smokers may not perceive quitting to be beneficial, as it could reduce quality of life.

Some participants (n = 3) described feeling oversaturated with risk messages, which contributed to a desensitising effect in relation to further risk communication. This led participants to be dismissive towards further communication; this is also relevant to the previous section (see Section 5.3.2.2), which explored risk contextualisation, as the results suggested that participants consider smoking-related risk in the context of other risks. One participant explained, "If you listened to everything, you wouldn't,

you wouldn't move, you'd just be wrapped up in cotton wool (laughs)" (Charlotte, 26, Former). Another participant similarly commented how he had observed other smokers who seemingly appeared desensitised by the graphic images on cigarette packs, which depict smoking-related disease.

Now, in work, there's a few lads that do smoke, and when they started putting pictures on of the diseases and things like that, it didn't really bother them, it become sort of blasé with them, like a swap shop, like what have you got on your packet? (Gavin, 45, Former)

This quote not only demonstrates how ubiquitous risk communication could lead to desensitised attitudes towards smoking-related risk, but additionally, this was the only comment that conflicts with the aforementioned accounts in the previous section (see Section 5.3.1.3), which demonstrated how cigarette packs were considered an effective vehicle for risk communication messages.

5.4 Discussion

The current chapter explored smoking-related risk perception in greater detail by employing both quantitative and qualitative research methods. The quantitative research component allowed for the investigation of correlates and predictors of perceived personal lung cancer risk, whilst the qualitative research component explored smoking-related risk perception and communication in rich detail. This chapter aimed to address the thesis objective, to explore factors implicated in smoking-related risk perception. By undertaking the aforementioned research and achieving this project objective, researcher knowledge regarding smoking-related risk perception

will be improved upon, potential groups that demonstrate inaccurate risk perceptions can be identified and potentially targeted for future health campaigns, and future risk communications can be better informed to improve efficacy. The current chapter findings also provide further explanation regarding the results of the previous chapter, in which the effect of the LLP intervention on follow-up smoking cessation and lung cancer risk perceptions was investigated. This section will explore the findings in relation to previous research, consider the strengths and potential limitations of the research methodology adopted in relation to this chapter, and lastly, discuss the implications of the chapter results.

5.4.1 Exploration of chapter findings

Several predictors of perceived personal lung cancer risk were identified in relation to the quantitative analysis, most notably, age, smoking status, perceived average smoker lung cancer risk, and perceived relative risk of lung cancer. The results of the quantitative research indicated that older participants were more likely to perceive their baseline perceived personal lung cancer risk as higher. Furthermore, the results of the qualitative analysis corroborated with the positive correlation between age and perceived personal lung cancer risk, identified as part of the quantitative analysis. The results suggested that throughout youth, concerns regarding the long-term effects of smoking were minimal, whilst with age, participants became increasingly fearful of disease and mortality, prompting them to consider smoking cessation in an effort to prolong life.

Previous studies have suggested that with continued smoking and increased age, perceptions of risk increase (Arnett, 2000; C. Lee, 1989). Furthermore, research

suggests that younger people with lower perceptions of smoking-related risk are more likely to start smoking, whilst younger smokers are also more likely to perceive their ability to quit smoking as higher, compared with older smokers (A. V. Song et al., 2009).

Several participants in the current study described how, during their youth, they would employ self-exempting perceptions of risk (e.g. "There's nothing wrong with them (older people) and they just carry on smoking"); this is consistent with previous research that has identified how young smokers tend to be optimistic and hold self-exempting beliefs regarding smoking (Mantler, 2013).

The current findings also suggest that baseline smoking status predicts baseline perceived personal lung cancer risk; the bivariate results suggest that current smokers more frequently perceived their personal perceived lung cancer risk as high. Previous research has also identified differences in perceived personal risk perception by smoking status, with current smokers being more likely to perceive their personal risk as very high, followed by former, and never smokers (Rutten et al., 2011). The current study is novel, in that it identified significant differences in perceived personal lung cancer risk between current and recent former smokers, despite all participants engaging with SSS.

The result regarding the relationship between smoking status and personal perceived lung cancer risk also coincides with previous research. There is a common misconception among the general population that the lung cancer incidence rate declines among former smokers; on the contrary, upon smoking cessation, the

precipitous rise in risk does cease but generally risk remains constant (Peto, 2011). The current results might suggest that recent former smokers are less likely to suspect lung cancer in themselves if they were to develop symptoms and therefore, recent former smokers may benefit from further education in this respect, in order to increase symptom awareness. Ali et al. (2015) found that current smokers were less likely to engage in a lung cancer screening trial, compared to former smokers. However, E. J. Hahn et al. (2006) identified that former smokers were more likely to be interested in receiving screening information or being screened than current smokers not interested in quitting smoking, but less likely to be interested in screening information or being screened compared to current smokers who were thinking about or preparing to quit. Further research is required to better understand the relationship between smoking status and help-seeking and potentially implement better education, particularly across socio-economic status (as the current study results focused almost entirely on individuals from more deprived backgrounds).

The quantitative results also revealed that perceived average smoker lung cancer risk and perceived relative risk of lung cancer were predictive of perceived personal lung cancer risk. This suggests that smokers who perceive the objective risk of lung cancer to be high, are more likely to perceive their personal risk of lung cancer to be high also. Previous research has additionally found perceptions of lung cancer to be correlated with other perceptions regarding lung cancer risk (e.g. Dillard et al., 2006).

The results of the qualitative analysis provide further insight into certain factors implicated in smoking-related risk perceptions, which the quantitative survey neglected to investigate. For example, the qualitative component considered responses

to the LLP intervention and barriers to objectively perceiving personal smoking-related risk. The qualitative investigation proved fruitful in identifying perceived efficacy of the LLP intervention in promoting smoking cessation, as several participants suggested that receiving the intervention promoted motivation to quit. The results suggest that the LLP intervention incorporated aspects of fear appeals, which have been described as "communications presenting the threat of impending danger to motivate compliance with a proposed recommendation" (p. 233) (Keller & Lehmann, 2008, as cited in Orazi & Pizzetti, 2015).

One meta-analysis described how fear appeals motivate adaptive behaviour change (e.g. message acceptance) but also, maladaptive behaviour (e.g. defensive avoidance) (Witte & Allen, 2000). Previous research has found that strong visual fear appeals (e.g. images of diseased lungs) influence smoking cessation intention and success (Gallopel-Morvan, Gabriel, Le Gall-Ely, Rieunier, & Urien, 2011; Hammond, Fong, McDonald, Brown, & Cameron, 2004). The results of the qualitative analysis provide evidence for both adaptive and maladaptive behaviours in relation to fear appeals. Witte's (1992; 1994) Extended Parallel Process Model (EPPM) (see Figure 4) provides some explanation as to the mechanisms by which the LLP intervention may have been processed by some participants. The model highlights how cognitions can contribute to fear appeal success, whilst the emotion fear can contribute towards fear appeal failure. Potentially, participants who exhibited low self-efficacy or those deemed the impact of smoking cessation as minimal in reducing lung cancer risk, may have been less likely to adopt a protective behaviour (i.e. smoking cessation), yet many participants who continued to smoke appeared able to accept this message (as participant recall of the LLP intervention was high), as the EPPM suggests.

Fear appeals were also prominently discussed by participants with regards to other common platforms of smoking-related risk communication, such as those found on cigarette packs or televised tobacco control campaigns. Such methods were found to evoke fear and promote intention to quit smoking; however, at least one participant account supported the notion that fear appeals can evoke avoidance behaviours.

The qualitative findings also corroborate with the quantitative findings that identified differing LLP intervention effects on follow-up smoking status, between baseline current and former smokers. The qualitative results revealed that current smokers described how receiving the LLP intervention provided additional risk information that was viewed as motivational in quitting smoking, whilst former smokers explained that receiving the information provided further reinforcement of their decision to quit. This finding fits with the notion that smokers at alternate stages of change may be more receptive to this kind of risk communication, as stipulated by the behavioural model, the Transtheoretical Model of Change (J. O. Prochaska & DiClemente, 1983; J.O. Prochaska & DiClemente, 1986). The model suggests that reinforcement management in itself is a process of change found to be implicated among recent former smokers, to help maintain long term cessation. In contrast with the quantitative results, the LLP intervention was described as having a motivational effect among baseline current smokers; potentially, some current smokers may have provided a socially desirable response, which could explain the discrepancy in results across methods or alternatively, too few participants were sampled in the trial of current smokers to detect a significant result.

Some participants failed to understand that the LLP intervention provided a comparison of projected lung cancer risk based on continued smoking and smoking cessation. Furthermore, a number of participants perceived the projected levels of risk provided as unexpectedly low; potentially this may have been due to limited understandings regarding the concept of five-year risk. Such limited understanding could be attributed to poor health literacy (Gigerenzer et al., 2007; Stewart et al., 2013) or the means by which the levels of risk were presented in the study intervention (Akl et al., 2011). Furthermore, one participant described how she failed to understand what the visual image on the written LLP intervention represented (see Figure 7). Previous research typically supports the use of visual imagery on written risk communication (Lipkus & Hollands, 1999), which suggests that potentially, the graphics included in the LLP intervention could benefit from further refinement.

In addition to exploring participant responses regarding the LLP intervention, the results of the qualitative analysis in this chapter also revealed factors associated with risk perception, which could potentially undermine or enhance broader risk communication efforts. Factors, including uncertainty or perceived lack of control over disease, risk contextualisation, and experiences of addiction, were found to contribute towards participants' responses to smoking-related risk communication.

Uncertainty and perceived lack of control were key overlapping components considered by participants. Lindbladh and Lyttkens (2003) describe how genuine uncertainty could implicate risk preventative behaviours. They argue that an individual who has less confidence in probabilistic information, is less likely to engage in information gathering and behaviour change. Familiarity and habituation may also

contribute towards perceived lack of control, as being exposed to a risk over time can result in perceiving the risk to be less threatening, despite the objective level of risk remaining constant (Slovic, 2000a). Furthermore, a number of participants additionally revealed self-exempting risk perceptions associated with perceived lack of control. Self-exempting risk perceptions have been associated with stage of change (Chapman et al., 1993), however, the current findings suggest that these perceptions were independent of smoking status.

Risk contextualisation was another important aspect considered by participants. Peretti-Watel, Seror, et al. (2014) suggested that those from the most deprived households tend to perceive smoking-related health risks as less threatening, whilst Lindbladh and Lyttkens (2003) described how individuals with fewer resources, fewer opportunities to mitigate poor outcomes, and greater exposure to social and material risk are less likely to be able to devote adequate resources to cope with health-related risk communication. This is relevant to the present thesis, as socio-economic disadvantage is closely associated to smoking and additionally, Liverpool is considered to be the most deprived local authority in England (Liverpool City Council, 2011). Participants considered lung cancer risk alongside other risks in society, which appeared to overwhelm some participants, leading to prioritisation of risk preventative behaviours, dependent on the value attached to a specific risk. This was also true with regards to risk communication, as participants described over-saturation of risk messages, which led to sources of information being ignored; the quantitative results corroborate the qualitative findings in this instance, as lung cancer worry was considered to be substantially low, despite all participants demonstrating smoking history (see Section 5.2).

Lastly, participants often attributed failure to engage in smoking-related risk preventative behaviours to the experience of addiction. This notion fits well with the health behaviour theory, PRIME theory (West & Brown, 2013), which not only considers plans, intentions, and motives in addiction, but it also emphasises the importance of impulses; whereas, it has been argued that traditional models of behaviour change, such as the HBM (Rosenstock, 1966), represent human behaviour in a computational manner, whereby risk avoidance is considered rational and risk-taking is viewed as irrational (Bloor, 1995 as cited in Lupton, 1999). A number of participants described an awareness of smoking-related risk but a simultaneous inability to quit smoking, due to the experience of addiction.

5.4.2 Potential strengths and limitations

The current chapter particularly draws on the mixed methods approach adopted throughout the PhD project and demonstrates how mixing methods can not only be complementary, but it can also increase the scope and depth of the research (Mathie & Carnozzi, 2005); this is a key strength, which is particularly relevant to the current chapter.

The semi-structured design of the qualitative interviews combined pre-determined questions with the opportunity for the interviewer to examine various themes and responses in greater detail. This proved highly beneficial in relation to the current chapter results, as the researcher was unaware of the types of responses they were likely to receive (Morse, 2012), particularly as complex discussions regarding smoking-related risk perception ensued. Participation selection biases were

particularly relevant in relation to the qualitative component of this chapter, as some individuals are more likely to engage with cancer risk research compared to others (Loon, Tijhuis, Picavet, Surtees, & Ormel, 2003); presumably, individuals who tend to deny or ignore cancer risk were less likely to engage in the interviews.

Another key strength in the current chapter is the use of both ordinal (or proportional odds [PO]) and logistic regression modelling in relation to the quantitative analysis. As the outcome variable of interest (baseline perceived personal lung cancer risk) was ordinal, PO regression was deemed most suitable for the analysis. In comparison to frequently used approaches for binary and nominal data, ordinal regression models benefit from making full use of ranked data, yet such approaches are often underutilised in biomedical and epidemiological studies (Thornicroft, Rose, Kassam, & Sartorius, 2007). The use of PO regression was, however, limited due to the assumption of PO often being violated. To further strengthen the analysis, binary regression models were additionally developed to complement the results and address any weaknesses in relation to the PO model; a multinomial regression model could have been considered, although this approach would have prevented the ordinal nature of the outcome variable from being detected.

The correlational analysis in the current chapter included all participants recruited in the quantitative aspect of the PhD project, who responded to the baseline perceived personal lung cancer risk measure (N = 502); this allowed for a novel and in-depth analysis of risk perception among both current and recent former smokers engaged with RCFE. It should be noted that the quantitative findings in this chapter emanate from a correlational analysis and therefore, only causal inferences can be conveyed

(Bowen & Wiersema, 1999; Weinstein et al., 1998). Currently, it is unclear as to whether smoking status predicts perceived personal lung cancer risk, perceived personal lung cancer risk predicts smoking status, or potentially, the relationship is bidirectional; the following chapter will attempt to address this paucity in the research further.

If a similar topic is to be researched in future, it would be beneficial to conduct the qualitative and quantitative methods sequentially. The qualitative research could therefore be conducted firstly to establish barriers and facilitators to communicating lung cancer risk, which could inform the design of the LLP intervention future and ultimately, improve LLP intervention efficacy.

5.4.3 Implications for knowledge, practice and policy

The current chapter findings have a number of implications for knowledge, practice and policy. The quantitative analysis revealed several results, which were often corroborated by the qualitative analysis. For example, age was consistently associated with risk perception. Some participants described how milestone birthdays prompted quit attempts. This has implications for tobacco control practice and policy, as potentially smoking cessation campaigns could target specific age groups. One recent review explored smoking cessation interventions aimed at smokers aged over 50 years old and concluded that only a small number of smoking cessation trials examined older smokers and that further research was required (D. Chen & Wu, 2015). Age-targeted smoking cessation interventions could easily be implemented in SSS, as service users provide their date of birth upon registration.

Associations were also identified between perceived personal lung cancer risk and smoking status, as well as objective perceptions of risk (e.g. perceived risk of the average smoker). These reported correlations suggest that potentially, smokers who are better educated or have more realistic perceptions regarding smoking-related risk of lung cancer, are more likely to perceive their personal lung cancer risk as higher. Health campaigns communicating the objective risk of lung cancer (particularly the relative risk) might therefore increase perceived personal lung cancer risk among smokers, which in turn, could promote smoking cessation.

Furthermore, future research might consider developing an intervention that incorporates lifetime risk (as opposed to five-year risk) and several smoking-related outcomes (as opposed to lung cancer alone); statistical presentation of risk information can greatly impact risk message persuasiveness (Akl et al., 2011).

With regard to broader smoking-related risk perception, the current chapter results have several implications. Many participants described an overwhelming experience of exposure to several risks, whereby certain risk preventative behaviours (e.g. smoking cessation) were deprioritised in favour of other risk preventative behaviours. This emphasises the importance of the development of interventions aimed at improving the resources available to individuals from particularly deprived households, who may be exposed to a greater number of risks.

Lastly, smokers who demonstrate high levels of risk awareness and acceptance, yet continue to smoke, would certainly be a potential sample to focus upon in future. For example, one study identified several barriers to smoking cessation success among smokers with COPD (Eklund, Hedman, Nilsson, & Lindberg, 2012). Further

investigation into smoking cessation barriers and facilitators among hard-to-reach smokers and individuals suffering with smoking-related disease might offer further knowledge regarding risk perception and smoking cessation theory. This could, in turn, lead to the development of more effective and targeted interventions.

5.4.4 Conclusion

In summary, the current chapter presented much of the results pertaining to both the quantitative and qualitative investigations. Key predictors of perceived personal lung cancer risk, such as age and smoking status, were discussed and potential implications regarding tobacco control research and practice were considered (e.g. age-specific smoking cessation interventions). Further factors implicated in risk perception were identified, which may inhibit the salience or efficacy of smoking-related risk communications; uncertainty of risk, risk contextualisation, and experiences of addiction were considerably notable aspects. This chapter also provides insight regarding participants' responses regarding the LLP intervention more specifically; potential areas for improvement were highlighted and discussed.

This chapter also demonstrates how implementation of a mixed methods design can strengthen a project, resulting in an in-depth exploration of risk perception among smokers; the quantitative results were able to qualify qualitative findings and vice versa, whilst the results of the qualitative analysis were able to provide meaning and explanation to associations established as part of the quantitative investigation. The following chapter will explore the predictors of smoking cessation through statistical analysis; in doing so, the relationship between lung cancer risk perceptions and

smoking status can be explored further using both cross-sectional and longitudinal methods.

Chapter 6: Predicting smoking status

6.1 Introduction

Whilst Chapter 4 intended to examine whether application of the LLP intervention was associated with follow-up smoking cessation success, this chapter aims to explore additional association with follow-up smoking cessation success. The results discussed in this chapter will help to determine a better understanding of the associations with follow-up smoking cessation among both baseline current and recent former smokers. By undertaking this research, groups struggle to quit or are more prone to relapse might be identified. Not only will these results inform the literature regarding smoking behaviour and risk perception but subsequently, new strategies might be developed and implemented to support identified vulnerable groups to successfully quit smoking. The current chapter addresses one key thesis objective:

• To explore factors implicated in smoking cessation success.

Statistical analyses were undertaken to achieve this objective. Both cross-sectional and longitudinal predictors of smoking status were examined. Firstly, differences in baseline participant characteristics by baseline smoking status were explored across all project participants recruited at baseline. Secondly, differences in baseline participant characteristics by follow-up smoking status were explored among baseline current smokers. Finally, differences in baseline participant characteristics by follow-up smoking status were explored among recent former smokers. For all three investigations, bivariate analyses were conducted to inspect significant group differences and to select variables for inclusion in multivariate models. Logistic

regression models were subsequently developed to explore the extent to which variables predicted smoking behaviour.

6.2 Differences between baseline current and recent former smokers

The present section refers to analyses whereby the overall sample of participants recruited in the PhD project (N = 521) were utilised to investigate whether baseline participant characteristics predict baseline 7-day point prevalence. The results of all bivariate and multivariate analyses are described.

A range of bivariate tests were undertaken to explore significant differences between baseline current and recent former smokers. Table 11 displays the distribution of baseline participant characteristics between the two smoking status groups; key differences of note are indicated. Hosmer et al. (2013) suggested incorporating all covariates significant at the level of 25%, in further multivariate analyses; therefore, this guidance was adhered to.

The bivariate analyses revealed that the baseline variables age (p = 0.002), marital status (p = 0.052), highest educational attainment (p = 0.142), living with another smoker (p = 0.002), FTND (p = 0.007), cigarettes per day (p < 0.001), perceived personal lung cancer risk (p < 0.001), perceived average smoker lung cancer risk (p = 0.109), perceived relative risk of lung cancer (p = 0.232), and lung cancer worry (p = 0.232) significantly differed by baseline smoking status at the level of 25%. No significant effects were observed between baseline smoking status and the baseline participant characteristics, including gender, ethnicity, socio-economic status, age started smoking, and perceived lung cancer survival.

Table 11. Participant characteristics by baseline smoking status

Baseline variable	Baseline current smokers former smok $(n = 302, 58.0\%)$ $(n = 219, 42.0\%)$		P-value §
Age (Median, IQR)	41.5 (31.0-51.0)	44.0 (37.0-52.0)	0.002*
Gender Gender	11.5 (51.6 51.6)	1110 (3710 3210)	0.308
Female	179 (59.3)	120 (54.8)	0.300
Male	123 (40.7)	99 (45.2)	
Ethnicity†	123 (40.7)	77 (43.2)	0.925
White	275 (92.0)	200 (91.7)	0.728
Other	24 (8.0)	18 (8.3)	
Marital status†	21 (0.0)	10 (0.0)	0.052*
Other	44 (14.7)	38 (17.5)	0.002
Single	161 (53.7)	93 (42.9)	
Married or living together	95 (31.7)	86 (39.6)	
Highest educational attainment†) (S1.7)	00 (25.0)	0.142*
Basic or no qualifications	139 (47.1)	117 (53.7)	011 . _
Higher qualifications	156 (52.9)	101 (46.3)	
Socio-economic status†	150 (52.5)	101 (10.5)	0.616
Most deprived	259 (86.0)	185 (84.5)	0.010
Least deprived	42 (14.0)	34 (15.5)	
Age started smoking (Median, IQR)	15.0 (13.0-17.0)	15.0 (14.0-17.0)	0.655
Living with another smoker†	15.0 (15.0 17.0)	15.0 (1.10 17.0)	0.002*
No	189 (63.4)	167 (76.3)	0.002
Yes	109 (36.6)	52 (23.7)	
FTND (Median, IQR)	6.0 (4.0-7.0)	6.0 (5.0-8.0)	0.007*
Cigarettes per day (Median, IQR)	20.0 (12.0-20.0)	20.0 (15.0-30.0)	p<0.001*
Perceived personal lung cancer risk†	2010 (1210 2010)	2010 (1210 2010)	p<0.001*
Low	53 (18.3)	70 (33.0)	P
Moderate	116 (40.0)	90 (42.5)	
High	121 (41.7)	52 (24.5)	
Perceived average smoker lung cancer r	· · ·	- (= ····)	0.109*
Very low to moderate	73 (25.0)	42 (19.6)	0.100
Somewhat high	105 (36.0)	96 (44.9)	
Very high	114 (39.0)	76 (35.5)	
Perceived relative risk of lung cancer†	11. (65.0)	, 6 (66.6)	0.232*
About the same to a little higher	58 (20.2)	35 (16.4)	
risk	0 0 (=0.=)	(-0.1)	
Twice as high risk	48 (16.7)	43 (20.2)	
Five times higher risk	86 (30.0)	52 (24.4)	
Ten times higher risk	95 (33.1)	83 (39.0)	
Lung cancer worry;	()	(,	0.138*
Rarely or never	89 (30.1)	75 (34.6)	
Sometimes	108 (36.5)	87 (40.1)	
Often or all the time	99 (33.4)	55 (25.3)	
Perceived lung cancer survival†	()		0.524
Less than a quarter	105 (36.5)	88 (41.7)	
About a quarter	84 (29.2)	63 (29.9)	
About half	72 (25.0)	44 (20.9)	
About three quarters to nearly all	27 (9.4)	16 (7.6)	
# Figures do not equate to 521 due to som			CNT: 4

[†] Figures do not equate to 521 due to some missing data, FTND = Fagerström Test of Nicotine Dependence, § Categorical variables were analysed using χ^2 test and continuous variables with Mann Whitney U-test, * p < 0.25

Multivariate analyses were undertaken to explore the differences by baseline smoking status further. All aforementioned potential predictors of baseline smoking status at the level of p < 0.25 were included in the development of a binary logistic regression model. Appendix O provides the results pertaining to the first multivariate model for the prediction of baseline smoking status. The first model was statistically significant, $\chi^2(16) = 71.458$, p < 0.001. The model explained 19.1% (Nagelkerke R^2) of the variance in baseline smoking status and correctly classified 63.9% of cases. Furthermore, the Hosmer-Lemeshow goodness-of-fit test indicated that the model fitted the data well, $\chi^2(8) = 8.643$, p = 0.373.

As previously described, Hosmer et al. (2013) provide guidance for model building strategies and purposeful selection of covariates. They recommend refining the initial multivariate model to develop a more parsimonious final model, which typically involves removing non-significant variables. Furthermore, Hosmer-Lemeshow goodness-of-fit tests are conducted and compared throughout the development process to explore model fit.

The finalised model was statistically significant, $\chi^2(9) = 58.734$, p < 0.001 (Table 12). The model explained 15.3% (Nagelkerke R^2) of the variance in baseline smoking status and correctly classified 62.8% of cases. Furthermore, the Hosmer-Lemeshow goodness-of-fit test indicated that the model fitted the data well, $\chi^2(8) = 6.638$, p = 0.576, demonstrating a considerable improvement in fit compared to the initial model.

The Wald criterion displayed that age (p < 0.001), living with another smoker (p = 0.022), and both "Moderate" (p = 0.026) and "High" (p < 0.001) levels of perceived

personal lung cancer risk were significant predictors. Furthermore, perceived relative risk of lung cancer level, "Ten times higher risk" (p = 0.003) and the lung cancer worry level, "Often or all the time" (p = 0.002) were both significant.

Table 12. Final logistic regression model for the prediction of baseline former smoker status

Baseline variable	Odds	Lower	Upper	<i>P</i> -value
	(95% CI)	(95% CI)	(95% CI)	
Age	1.040	1.021	1.060	p<0.001*
Living with another smoker				0.022*
Yes	0.605	0.393	0.930	
(Base = No)				
Perceived personal lung cancer risk				
Moderate	0.576	0.354	0.936	0.026*
High	0.246	0.144	0.420	p<0.001*
(Base = Low)				
Perceived relative risk of lung cancer				
Twice as high risk	1.648	0.872	3.115	0.124
Five times higher risk	1.468	0.809	2.664	0.206
Ten times higher risk	2.441	1.361	4.380	0.003*
(Base = About the same to a little)				
higher risk)				
Lung cancer worry				
Sometimes	0.846	0.536	1.334	0.471
Often or all the time	0.548	0.328	0.916	0.022*
Base = Rarely or never				

FTND = Fagerström Test of Nicotine Dependence, * p < 0.05

6.3. Predicting follow-up smoking status among baseline current smokers

The current section utilises baseline current smokers (n = 297) to examine whether baseline participant characteristics predict follow-up smoking status. Again, the results of all bivariate and multivariate analyses are described.

Bivariate tests were undertaken to explore the association between baseline participant characteristics and follow-up smoking status, among baseline current smokers only (N = 297). Table 13 displays the distribution of baseline participant characteristics

between follow-up smoking status groups; key differences are indicated in the table. Again, guidance produced by Hosmer et al. (2013) suggested incorporation of all covariates significant at the level of 25% in further multivariate analyses.

The bivariate analyses revealed that baseline variables, including ethnicity (p = 0.179), socio-economic status (p = 0.198), age started smoking (p = 0.159), living with another smoker (p = 0.247), FTND (p = 0.079), and perceived relative risk of lung cancer (p = 0.048) significantly differed by baseline smoking status at the level of 25%. No significant effects were observed between follow-up smoking status and the baseline participant characteristics, including age, gender, marital status, highest educational attainment, cigarettes per day, perceived personal lung cancer risk, perceived average smoker lung cancer risk, lung cancer worry, perceived lung cancer survival, and treatment group.

Appendix P displays the results regarding the initial multivariate model for predictors of follow-up smoking status, among baseline current smokers. The original model was statistically significant, $\chi^2(8) = 22.856$, p = 0.004. The model explained 12.6% (Nagelkerke R^2) of the variance in follow-up smoking status and correctly classified 81.0% of cases. Furthermore, the Hosmer-Lemeshow goodness-of-fit test indicated that the model fitted the data adequately, $\chi^2(8) = 9.661$, p = 0.290.

Table 13. Participant characteristics by follow-up smoking status among baseline current smokers

Baseline variable	Follow-up	Follow-up	P-value §
	current smokers	former smokers	
	(n = 239, 80.5%)	(n = 58, 19.5%)	
Age (Median, IQR)	42.0 (31.0-50.0)	43.0 (30.8-52.0)	0.711
<u>Gender</u>			
Female	142 (59.4)	35 (60.3)	0.897
Male	97 (40.6)	23 (39.7)	

Ethnicity†			
White	220 (93.2)	51 (87.9)	0.179*
Other	16 (6.8)	7 (12.1)	
Marital status†			0.594
Other	35 (14.7)	9 (15.8)	
Single	130 (54.6)	27 (47.4)	
Married or living together	73 (30.7)	21 (36.8)	
Highest educational attainment†			0.950
Basic or no qualifications	110 (46.8)	26 (47.3)	
Higher qualifications	125 (53.2)	29 (52.7)	
Socio-economic status†			0.198*
Most deprived	202 (84.9)	53 (91.4)	
Least deprived	36 (15.1)	5 (8.6)	
Age started smoking (Median, IQR)	15.0 (13.0-17.0)	15.0 (14.0-18.0)	0.159*
Living with another smoker†	,	,	
No	152 (64.4)	32 (56.1)	0.247*
Yes	84 (35.6)	25 (43.9)	
FTND (Median, IQR)	6.0 (4.0-7.0)	6.0 (4.0-7.0)	0.079*
Cigarettes per day (Median, IQR)	20.0 (12.5-20.0)	20.0 (10.0-20.0)	0.300
Perceived personal lung cancer risk†	,	()	0.581
Low	39 (16.8)	12 (22.2)	
Moderate	95 (40.9)	19 (35.2)	
High	98 (42.2)	23 (42.6)	
Perceived average smoker lung cancer risl		_= (,	0.909
Very low to moderate	58 (24.9)	14 (25.9)	
Somewhat high	85 (36.5)	18 (33.3)	
Very high	90 (38.6)	22 (40.7)	
Perceived relative risk of lung cancer†	70 (30.0)	22 (10.7)	0.048*
About the same to a little higher risk	51 (22.4)	6 (10.9)	0.010
Twice as high risk	40 (17.5)	6 (10.9)	
Five times higher risk	68 (29.8)	17 (30.9)	
Ten times higher risk	69 (30.3)	26 (47.3)	
Lung cancer worry†	07 (30.3)	20 (47.3)	0.427
Rarely or never	74 (31.6)	13 (22.8)	0.427
Sometimes	84 (35.9)	23 (40.4)	
Often or all the time	76 (32.5)	21 (36.8)	
Perceived lung cancer survival†	70 (32.3)	21 (30.6)	0.663
Less than a quarter	89 (39.2)	16 (28.6)	0.003
About a quarter	61 (26.9)	22 (39.3)	
About a quarter About half	55 (24.2)	14 (25.0)	
	, ,		
About three quarters to nearly all	22 (9.7)	4 (7.1)	0.662
Treatment group	116 (70.5)	102 (01 5)	0.663
Control	116 (79.5)	123 (81.5)	
Intervention	30 (20.5)	28 (18.5)	

[†] Figures do not equate to 297 due to some missing data, FTND = Fagerström Test of Nicotine Dependence, § Categorical variables were analysed using χ^2 test (except for those with expected cell frequencies < 5, which were analysed using Fisher's Exact test) and continuous variables with Mann Whitney U-test, * p < 0.25

Again, Hosmer et al.'s (2013) guidance pertaining to model building was followed in an effort to achieve a parsimonious model. Few variables remained of statistical or clinical relevance, resulting in the final model demonstrated in Table 14.

The finalised model was statistically significant, $\chi^2(3) = 8.173$, p = 0.043. The model explained 4.5% (Nagelkerke R^2) of the variance in follow-up smoking status and correctly classified 80.6% of cases. The Hosmer-Lemeshow goodness-of-fit test was not applicable to the finalised model, as only one independent variable was included. The Wald criterion displayed that the perceived relative risk of lung cancer level, "Ten times higher risk" was significant (p = 0.017).

Table 14. Finalised logistic regression model for the prediction of follow-up former smoker status among baseline current smokers

Baseline variable	Odds (95% CI)	Lower (95% CI)	Upper (95% CI)	<i>P</i> -value
Perceived relative risk of lung cancer				
Twice as high risk	1.275	0.382	4.254	0.693
Five times higher risk	2.125	0.783	5.771	0.139
Ten times higher risk	3.203	1.228	8.354	0.017*
(Base = About the same to a little)				
higher risk)				

^{*} p < 0.05

6.4 Predicting follow-up smoking status among baseline recent former smokers

The present section utilises baseline recent former smokers (n = 216) to examine whether baseline participant characteristics predict follow-up smoking status. The results of all bivariate and multivariate analyses conducted are again described.

Bivariate analyses were conducted to examine the associations between baseline participant characteristics and follow-up smoking status, among baseline recent former smokers. Section 3.5.2 provides further details regarding the definition of recent former smokers.

The distribution of baseline participant characteristics are displayed between follow-up smoking status groups (Table 15). The key differences are indicated in the table. Again, Hosmer et al. (2013) suggested incorporation of all covariates significant at the level of 25% into an initial multivariate model.

The bivariate results revealed that the baseline variables age (p = 0.029), marital status (p = 0.034), quit duration (p = 0.002), perceived lung cancer survival (p = 0.154), and treatment group (p = 0.032) significantly differed by baseline smoking status at the level of 25%. No significant effects were identified between follow-up smoking status and the baseline participant characteristics, including gender, ethnicity, highest educational attainment, socio-economic status, age started smoking, living with another smoker, FTND, cigarettes per day, perceived personal lung cancer risk, perceived average smoker lung cancer risk, perceived relative risk of lung cancer, and lung cancer worry.

Multivariate analyses were conducted to explore the predictors of follow-up smoking status among baseline recent former smokers. All aforementioned potential predictors of baseline smoking status at the level of p < 0.25 were included in the development of a binary logistic regression model.

Table 15. Participant characteristics by follow-up smoking status among baseline recent former smokers

Baseline variable	Follow-up current smokers	Follow-up former smokers	P-value §
	(n = 122, 56.5%)	(n = 94, 43.5%)	
Age (Median, IQR)	42.0 (36.0-52.0)	47.5 (39.0-53.0)	0.029*
<u>Gender</u>			0.517
Female	69 (54.6)	49 (52.1)	
Male	53 (43.4)	45 (47.9)	

Ethnicity†			0.666
White	110 (90.9)	87 (92.6)	
Other	11 (9.1)	7 (7.4)	
Marital status†			0.034*
Other	25 (20.8)	13 (13.8)	
Single	57 (47.5)	35 (37.2)	
Married or living together	38 (31.7)	46 (48.9)	
Highest educational attainment†			0.965
Basic or no qualifications	64 (52.9)	57 (47.1)	
Higher qualifications	57 (47.1)	44 (46.8)	
Socio-economic status†			0.939
Most deprived	103 (84.4)	79 (84.0)	
Least deprived	19 (15.6)	15 (16.0)	
Quit duration (Median, IQR)	35.0 (20.0-57.0)	50.0 (25.5-90.0)	0.002*
Age started smoking (Median, IQR)	15.0 (14.0-17.0)	15.0 (13.0-17.0)	0.766
Living with another smoker†			0.840
No	92 (75.4)	72 (76.6)	
Yes	30 (24.6)	22 (23.4)	
FTND (Median, IQR)	6.0 (5.0-8.0)	6.0 (4.0-7.3)	0.305
Cigarettes per day (Median, IQR)	20.0 (15.0-30.0)	20.0 (15.0-30.0)	0.797
Perceived personal lung cancer risk†			0.590
Low	35 (29.9)	33 (35.9)	
Moderate	51 (43.6)	39 (42.4)	
High	31 (26.5)	20 (21.7)	
Perceived average smoker lung cancer risk	†		0.324
Very low to moderate	26 (21.8)	15 (16.3)	
Somewhat high	55 (46.2)	39 (42.4)	
Very high	38 (31.9)	38 (41.3)	
Perceived relative risk of lung cancer†			0.383
About the same to a little higher risk	24 (20.2)	11 (12.1)	
Twice as high risk	25 (21.0)	17 (18.7)	
Five times higher risk	27 (22.7)	25 (27.5)	
Ten times higher risk	43 (36.1)	38 (41.8)	
Lung cancer worry†			0.910
Rarely or never	42 (35.0)	31 (33.0)	
Sometimes	49 (40.8)	38 (40.4)	
Often or all the time	29 (24.2)	25 (26.6)	
Perceived lung cancer survival†	` '	, ,	0.154*
Less than a quarter	50 (42.4)	37 (41.1)	
About a quarter	40 (33.9)	22 (24.4)	
About half	23 (19.5)	21 (23.3)	
About three quarters to nearly all	5 (4.2)	10 (11.1)	
Treatment group	` '	` /	0.032*
Control	66 (64.1)	56 (49.6)	
Intervention	37 (35.9)	57 (50.4)	
† Figures do not equate to 216 due to sor		. ,	est of Nicotine

[†] Figures do not equate to 216 due to some missing data, FTND = Fagerström Test of Nicotine Dependence, § Categorical variables were analysed using χ^2 test and continuous variables with Mann Whitney U-test, * p < 0.25

An initial model was developed but in line with recommendations (Hosmer et al., 2013), model refinement was considered; the initial model developed was deemed most satisfactory following further exploration and was therefore, classified as the final model (Table 16). The final model was statistically significant, $\chi^2(8) = 35.170$, p < 0.001. The model explained 21.8% (Nagelkerke R^2) of the variance in follow-up

smoking status and correctly classified 66.7% of cases. Furthermore, the Hosmer-Lemeshow goodness-of-fit test indicated that the model fitted the data well, $\chi^2(8) = 7.427$, p = 0.491.

Table 16. Final logistic regression model for the prediction of follow-up former smoker status among baseline former smokers

Baseline variables	Odds	Lower	Upper	<i>P</i> -value
	95% CI	95% CI	95% CI	
Age	1.042	1.005	1.080	0.026*
Marital status				
Single	1.917	0.712	5.164	0.198
Married and living together	3.242	1.275	8.247	0.014*
(Base = Other)				
Quit duration (days)	1.009	1.003	1.014	0.003*
Perceived lung cancer survival				
About a quarter	0.926	0.441	1.943	0.838
About half	1.467	0.646	3.330	0.360
About three quarters to nearly all	4.600	1.230	17.202	0.023*
$(Base = Less\ than\ a\ quarter)$				
Treatment group				0.046*
Intervention	1.888	1.013	3.521	
(Base = Control)				

^{*} p < 0.05

The Wald criterion indicated that age (p=0.026), quit duration (p=0.003), and treatment group (p=0.046) were significant predictors overall. Furthermore, the marital status level, "Married and living together" (p=0.014) and the perceived lung cancer survival level, "About three quarters to nearly all" (p=0.023) were both significant.

6.5 Discussion

The present chapter explored the predictors of smoking status. Analyses were undertaken to explore differences between current and recent former smokers at baseline and subsequently, analyses were conducted to investigate the predictors of follow-up smoking status among: (1) baseline current smokers; (2) baseline recent

former smokers. By investigating these concepts, a more in-depth understanding of the predictors of smoking cessation can be achieved. Furthermore, the findings will enable identification of potentially vulnerable groups, who may find smoking cessation more challenging. These groups could subsequently be targeted for additional support or intervention. The results, potential strengths and limitations in relation to this chapter, and implications will now be discussed sequentially.

6.5.1 Exploration of chapter findings

The current chapter results identified a number of predictors of baseline smoking status and follow-up smoking cessation. Predictors of baseline smoking status included age, marital status, living with another smoker, perceived personal lung cancer risk, perceived relative risk of lung cancer, and lung cancer worry. Predictors of follow-up smoking status among baseline current smokers included the perceived relative risk of lung cancer level, "Ten times higher". Lastly, predictors of follow-up smoking status among baseline recent former smokers included age, marital status, quit duration, and perceived lung cancer survival. Some of the key predictors will now be discussed.

The cross-sectional analysis demonstrated a positive relationship between age and former smoker status. This positive relationship remained at follow-up among baseline recent former smokers, but not among baseline current smokers; it is unclear why age failed to predict follow-up smoking status among baseline current smokers but potentially, the limited number of former smokers at follow-up within the analysis of baseline current smokers may have reduced power to detect a difference. A number of studies have found that older people are more likely to achieve smoking cessation

success, compared to younger smokers (Fidler et al., 2013; Hymowitz et al., 1997; C. Lee & Kahende, 2007; Monsó et al., 2001).

Previous research has additionally found that marital status is associated with smoking cessation success, as smokers who are married are more likely to quit compared to those who are single (Broms et al., 2004; Gourlay et al., 1994). The current results suggest that individuals who were married or living together were more likely to be classified as a former smoker at follow-up, compared to those who were divorced, widowed, or other, in respect of the baseline former smokers analysis. Again, the analysis of baseline current smokers failed to detect a significant difference here. Furthermore, no difference in marital status was detected between smoking status groups in regards to the cross-sectional analysis; however, the cross-sectional analysis did reveal that participants who lived with a smoker were less likely to be categorised as a former smoker. This finding also corroborates with previous research that suggests that being married to a non- or former smoker enhances smoking cessation success (Coppotelli & Orleans, 1985; Hanson et al., 1990; McBride et al., 1998).

The current study also identified that baseline quit duration was indicative of follow-up smoking status among baseline recent former smokers. Previous research has also highlighted how baseline quit duration can be an important predictor of follow-up smoking cessation success. For example, Gilpin, Pierce, and Farkas (1997) explored follow-up abstinence rates among former smokers who had achieved varying quit durations at baseline. They estimated that 12%, 25%, and 52% of baseline former smokers who had quit for less than one month, 1-3 months, and 3-6 months, remained abstinent at follow-up, respectively.

Lastly, the analyses within this chapter revealed that several lung cancer risk perceptions were predictive of smoking status, although the specific lung cancer risk perceptions of relevance differed across each of the three datasets analysed. Previous research has provided cross-sectional support for the relationship between smoking status and lung cancer risk perceptions (e.g. Weinstein et al., 2005), whilst a number of recent studies have displayed a positive relationship between risk perceptions and follow-up smoking cessation success (Borrelli et al., 2010; Hayes & Borrelli, 2013; Jacobson et al., 2014). The identified differences between lung cancer risk perceptions and smoking status in the current project were most prevalent among variables tests across the cross-sectional results, compared to the longitudinal results. Furthermore, bivariate analyses demonstrated that overall, the majority of participants underestimated relative risk, compared to traditional values of relative risk (e.g. Pesch et al., 2012).

6.5.2 Potential strengths and weaknesses

The present chapter describes the results pertaining to the predictors of smoking status. Cross-sectional analyses were undertaken to explore differences between baseline smoking status groups, whilst longitudinal analyses were conducted to examine differences between follow-up smoking status groups: (1) baseline current smokers; (2) baseline recent former smokers. Not only does the chapter combine the results of cross-sectional and longitudinal analyses, to develop an improved understanding of smoking behaviour, but the extensive number of baseline characteristic variables allowed an in-depth analysis of the predictors of smoking behaviour. There are, however, some potential study limitations, which should be acknowledged.

The current project employed a self-report measure for smoking status, 7-day point prevalence. Although 7-day point prevalence can be highly advantageous (Velicer & Prochaska, 2004) and the sensitivity and specificity of self-reported smoking status has previously been identified as high when compared with biochemical indices (Patrick et al., 1994), inclusion of a measure of prolonged abstinence and biochemical verification of smoking status may have been preferable (West et al., 2005). This aspect was described in greater detail earlier in the thesis (see Section 4.4.2).

Although the sample size was modest in relation to the cross-sectional analysis, the analysis of follow-up smoking status in relation to baseline current smokers was limited in particular, as 58 participants were classified as former smokers at followup. This resulted in some data analysis challenges, such as the presence of low cell frequencies and the subsequent need for variable transformation (Appendix M). Furthermore, some associations highlighted in Table 11 were found to contradict the literature. For example, education was found to be significant at the level of p < 0.25and the data suggested that individuals with lower levels of education were more likely to be former smokers, yet the literature contradicts this. It should be noted that some associated relationships were identified at the level of p < 0.25, which is nontraditional, for regression modelling. Furthermore, some variable levels were combined, which may mean that discreet differences among some variables were not possible to detect. A larger sample size could have addressed such issues and could have increased the number of significant results, as several variables were found to be non-significant that had previously been deemed significant in the literature. For example, one meta-analysis found that females are less successful in quitting smoking

than males (Scharf & Shiffman, 2004), whereas, the current project failed to establish this association.

6.5.3 Implications for knowledge, practice and policy

This section highlights a number of implications in relation to tobacco control knowledge, practice and policy. Several predictors of smoking status were identified within this chapter; however, key variables of interest differed across the three samples explored. Implications with regards to the key variables of interest, age, marital status, quit duration, and lung cancer risk perceptions, will be discussed.

As described, a positive correlation was identified between age and smoking cessation. This might suggest that younger smokers find smoking cessation to be more challenging, compared to older smokers. This highlights the need to develop new and innovative interventions tailored towards younger people and potentially to explore the barriers to smoking cessation success among this population.

The results also demonstrated how baseline recent former smokers who were single were less likely to be classified as a former smoker at follow-up, whilst the cross-sectional results revealed that participants who reported living with a smoker were also less likely to be classified as a former smoker than a current smoker. As appropriate social support can enhance smoking cessation (Coppotelli & Orleans, 1985; Mermelstein et al., 1983), future efforts should explore methods of improving social support and coping strategies, specifically among such groups to further improve smoking cessation rates.

The current project suggests that quit duration predicts follow-up smoking cessation success among baseline former smokers. NICE (2008) recommend that exploring the most effective and cost effective methods of preventing relapse among individuals who have already quit should be a research priority. The current findings further support this recommendation; potentially, extended provision of behavioural smoking cessation support could be a cost-effective method of improving long term smoking cessation attempts. Further research is required.

The results also demonstrate how various lung cancer risk perceptions predict smoking behaviour, as the differences associated with perceived relative risk of lung cancer were of particular significance. Potentially, communicating risk in terms of relatives may be the most appropriate method of delivering information on cancer-related risk. Improving the methods by which risk is communicated could, in turn, improve smoking cessation rates.

6.5.4 Conclusion

The current chapter presented bivariate and multivariate results pertaining to the prediction of smoking status. The chapter aimed to utilise both cross-sectional and longitudinal datasets to explore the predictors of smoking behaviour in greater detail. Several key predictors were identified, including age, marital status, living with another smoker, quit duration (among baseline recent former smoker), and lung cancer risk perceptions; however, the results often differed across datasets, potentially due to sample size limitations

The results highlight a number of potential groups that may be particularly vulnerable to relapse. Further research should focus upon exploring such populations further and considering opportunities for delivering more intensive and potentially, tailored support to the identified groups. Furthermore, several lung cancer risk perceptions were found to predict smoking status; this finding indicates the need for further research and may be used to inform the development of future risk communications. The following two chapters will build upon the findings in the present chapter and will consider additional factors implicated in smoking cessation success, by implementing qualitative methods.

Chapter 7: Conquering smoking cessation: Accentuating the positives and eliminating the negatives

7.1 Introduction

The previous chapter involved a quantitative investigation of the predictors of smoking behaviour, whereas, the current and following chapter explore factors implicated in smoking cessation success using qualitative techniques. The current chapter results explore service users' personal experiences of quitting smoking, with focus on various aspects that participants deemed helpful or unhelpful in quitting smoking. By undertaking this qualitative component, a better understanding of smoking and smoking cessation can be achieved, specific groups who experience difficulties quitting smoking can be identified, and the results could inform the development of tobacco control practice and policy. The present chapter addresses one key thesis objective:

• To explore factors implicated in smoking cessation success.

This chapter will firstly explore the experience of mental conflict, which many participants described upon attempting to quit smoking. The notion of mental conflict refers to the psychological struggle described among participants with respect to the choice between stopping smoking and continuing to smoke. Secondly, anticipated and experienced benefits of quitting smoking are discussed. Health, financial, and image or functionality benefits were all noted and participants would commonly refer to the benefits of quitting smoking, presumably to promote motivation and reinforce a quit attempt. The two major themes identified in this chapter include: (1) Experiences of

mental conflict; (2) Perceived benefits of quitting. These themes will be described and the respective sub-themes for each of these themes are discussed in turn.

7.2 Experiences of mental conflict

"Experiences of mental conflict" is the first major theme considered in this current chapter. Participants provided accounts of this experience, often referring to a psychological "battle" or "fight". More specifically, participants described an experience of internal conflict, whereby they would psychologically battle conflicting intentions, motives, and impulses in relation to smoking cessation. This major theme consists of four prominent sub-themes, including "Motivation and self-efficacy", "Regaining control", "Self-doubt" and "Smoker identity", which will subsequently be described.

7.2.1 Motivation and self-efficacy

"Motivation and self-efficacy" was the first prominent sub-theme associated with the major theme, "Experiences of mental conflict". Participants consistently advocated that motivation is essential for any smoker embarking on a quit attempt, "...but I think it's, it comes back to that old fashioned thing, you've got to want to do it, know what I mean?" (Paul, 38, Former) and "The thing is, you've got to want to do it, haven't you? You've got to want to give up. If you don't want to do it, it's not going to work. You've got to in your mind though, that you want to do it." (Shaun, 48, Current); however, such strong opinions regarding motivation were most common among recent former smokers.

Many participants additionally suggested that smoking cessation could only be sustained if the smoker demonstrated an autonomous willingness to quit smoking (i.e. quitting out of personal choice), as opposed to quitting to appease others, "Oh well, I think you've got to want to do it yourself like. It's no good like other people telling you. It's something you've got to do yourself" (Michael, 48, Former).

Yeah, well, it's got to be yourself, you've got to do it for yourself. There's no use saying there's FagEnds, you know, you can go to your doctor, chemist, Lloyds chemist do the no smoking. It's ok saying that to people but they've got to do it for themselves. (Julia, 49, Former)

Again, these comments were most prominent among recent former smokers, who provided insight into their quit. One current smoker attributed his quit attempt to a doctor's recommendation, however, his subsequent comments portrayed him as less determined, "Well, I've thought it was time anyway but just the doctors telling me all the time anyway, you know, I thought I might as well stop (pause) or try to" (Joel, 44, Current).

A central component of Bandura's social learning theory (1977, 1982) is self-efficacy, which refers to an individual's assessment of their effectiveness of competency to perform a behaviour successfully. Self-efficacy also appeared to be important to many participants. Many recent former smokers illustrated high levels of self-efficacy, with several expressing self-congratulatory, and often repetitive comments, presumably to demonstrate confidence in one's ability to maintain change and to reinforce the behaviour change. For example, one recent former smoker commented, "I am

stopping, I am stopping, I have stopped and I have no intention now of coming this far and going back to how I did and I know that for a fact, that I'm not going back" (Jacob, 59, Former), whilst another stated, "I went to watch Everton on Sunday and it never even bothered me, I didn't even want a ciggy, it just didn't bother me..." (Timothy, 58, Former).

A number of researchers have demonstrated that self-talk (i.e. inner speech), may assist cognitive and self-regulatory functions (Diaz & Berk, 1992; D. G. MacKay, 1992). Participants described how they implemented positive self-talk, presumably to promote self-efficacy and reinforce change. Participants provided various examples of positive self-talk, seemingly to enhance self-efficacy and to combat temptation in situations whereby they might have felt vulnerable to smoking.

I just thought come on (*participant's name*) if you want to have any kind of quality of life much longer, you're going to have to start looking after yourself a bit better, and that's what I proposed to do, yeah, try to pack in smoking. (Peter, 50, Current)

One recent former smoker described a situation whereby they experienced a craving but subsequently implemented self-talk to reinforce smoking cessation and reassure themselves of their ability to remain abstinent.

I start getting really agitated and have to get up and walk out the room, 'cause me body's just had that little thing going "You want a ciggy!" and I'm like "No... bugger off! You're not getting one!" (Matilda, 33, Former)

7.2.2 Regaining control

The majority of participants (n = 21) described the experience of smoking as habitual. Fourteen participants detailed how smoking had effectively dictated their daily routine, which for some had contributed towards their motivation to quit. Several participants described an explicit sense of being controlled by cigarettes, as opposed to having control over cigarettes. Current smokers appeared particularly frustrated, presumably because they still felt enslaved by smoking, as the following comments suggest.

I decided about a year ago I wanted to quit but I never got round to it. I finally got round to it and obviously, the next stage is to quit smoking and get on with the rest of me life but as I say, I really am a slave to cigarettes at the moment, and that's got to stop. (Luke, 54, Current)

It's just the fact that it's been going on so long now, do you know what I mean? It's like starting to do me head in, in a way, it's not like, it's not a joke anymore now, you know what I mean? It's starting to proper do me head in, you know what I mean? (Andrew, 34, Current)

A number of recent former smokers also commented on the notion of regaining control. One participant was asked what he disliked about smoking and he responded, "Erm, being hooked on them really. Having something like a cigarette having so much control over me" (Sarah, 55, Former). Whilst, another recent former smoker stated,

"... being in control of meself and not having the ciggies controlling me. Feeling more in control" (Stuart, 45, Former).

A number of participants from more deprived households described how dependence on cigarettes and concerns regarding cost, resulted in frequent consideration and planning in relation to acquiring cigarettes; this also seemed to contribute towards the perceived lack of control many participants experienced and appeared to contribute towards motivation to quit. One current smoker expressed hatred towards cigarettes and seemingly condemned his frequent pursuit of cigarettes.

First thing I used to do as well, if I didn't have a ciggy, if I did have money, I'd be at that shop at seven o'clock, when it opens, for a pack of ciggies. The last few ciggies that I have had, I don't even enjoy them. I don't even enjoy them anymore, horrible, nasty, I want yous gone, I want yous gone out me life. (Jack, 49, Current)

One recent former smoker detailed how she felt the need to plan ahead for cigarettes and that this was a constant burden, which motivated her to quit smoking.

Like, I was thinking, where am I going to get my ciggy, like these are going to do me until Friday, and then what do I do? And it was just, I thought, I don't want to live like this ... I just thought, I don't want to plan my life around it anymore. I just didn't want to, I just woke up and I thought, I just didn't want to do it. I don't want to smoke anymore. (Julia, 49, Former)

The financial relief attached to smoking cessation may contribute in part to the sense of liberation identified by many participants, especially among more deprived service users.

Participants also frequently described examples whereby smoking contributed towards routine formation and how smoking restricted the choices and opportunities available to them often. One participant described how this aspect of smoking disrupted family life.

Yeah well, you know, it dictates your life doesn't it smoking? And you know, like when we were going out on days out with the kids, you would erm, the kids were all eager to go, you know they're ready aren't they before yous and they're sat in the car and I'm standing out the door, going hang on just let me have a ciggy because can't smoke 'cause yous are in the car. They're like "Dad, come on!" (Robert, 34, Current)

Another recent former smoker who had previously smoked sixty cigarettes per day, described how smoking had interfered with participation in certain leisure activities.

Last time I went to the pictures was, I think it was, '89, 1989 because, I wouldn't go, because, erm, it's when they banned all the smoking in the cinemas and all that and, erm, but now I feel like I can go. I can do all the stuff that I couldn't do before because of the ciggies. (Stuart, 45, Former)

Although many participants described the importance of habit and routine dictation in regard to regaining control, a substantial component was addiction. Addiction was viewed as a major barrier to smoking cessation success and an inhibitor of self-efficacy. Seventeen participants described smoking as an addiction, although it should be noted that participants weren't directly asked about addiction or habit, and rather they were asked why they felt they had continued to smoke since initiation. Although a small number of participants described the enjoyment of smoking, most explained that they no longer enjoyed smoking and that they continued to smoke due to the addiction.

It's like I said, it's just the addiction really, you just want one. Like, you don't smoke because you enjoy it, you smoke because you need the nicotine in it. If you could get injections where you could get that nicotine then that would just be the same wouldn't it? 'Cause when you're smoking you just get that nicotine into your body and it's nothing to do with anything else. (Shaun, 48, Current)

Another current smoker described how their dependency had developed as they became accustomed to smoking, "It was just erm, it was like you're addicted to them. That's all it was, just smoking, smoking and smoking, that's all it was, 'cause you just got used to it' (David, 37, Current). Participants also often used the terms habit and addiction interchangeably, suggesting that some participants viewed the terms effectively as synonyms for dependency, "And I didn't realise how hard it was to get out of that habit 'cause it's a drug, it's a drug going through my body isn't it? Going through my system?" (Julia, 49, Former). One current smoker described, "I suppose,

I suppose it was just like a habit, like you're addicted to it then, so it wasn't easy just to stop... 'cause by that time you're addicted to it aren't you?" (Andrew, 34, Current).

Two participants compared smoking to other addictions, seemingly to highlight how severe an addiction they perceived smoking to be and to demonstrate how challenging smoking behaviour is to regulate. One participant compared smoking to heroin use, "It's like a heroin addict and when they inject, the risk of HIV and that, because they know all these things but it's the addiction isn't it?" (Sarah, 55, Former), whilst another participant compared smoking to alcohol dependency.

Well I think it's the same with anyone who's an alcoholic, they could have liver failure or kidney failure or whatever and you could say to them look, your literally drowning yourself and your going to end up killing yourself in the next six months. (Gavin, 45, Former)

The above participant continued to describe how he felt that some members of society were ignorant to the difficulties that smokers endure in attempting to quit smoking and how often non-smokers fail to empathise with how challenging smoking cessation can be.

I think, you'll probably find a lot of the ones that would say yeah, they feel sort of like interrogated, like the ones who you know, are mums, and when you see them pushing prams, because I've been out and I've seen people looking and the likes of having a baby in a pram and they're smoking and you see them, giving them funny looks like, you know, "You shouldn't be doing that"; you

know it's wrong and you know you shouldn't be doing it but it's not as easy as saying, you shouldn't be buying it anymore. (Gavin, 45, Former)

Some participants discussed how withdrawal symptoms were viewed as a barrier to successfully quitting smoking, again demonstrating how some participants felt powerless in steering or controlling a quit attempt. One recent former smoker described how he suffered severe physical withdrawal symptoms, which he found difficult to endure.

The withdrawals, I didn't like them, I used to get pains in me gums, severe pains then but now I'm over all that and I never want to go back on them again. To come this far, and just to go back to them, honest to god, I'd never forgive meself. (Jacob, 59, Former)

Another recent former smoker explained how his previous quit attempts had been limited due to the experience of severe withdrawals, "I've never packed in long enough 'cause the longest I'd packed in for was 12 hours ... Oh, it was horrible. It was the worst feeling ever" (Stuart, 45, Former).

7.2.3 Self-doubt

Participants expressed concerns regarding how challenging they often viewed quitting to be and how they feared they were incapable of quitting. The majority of participants admitted that quitting smoking had been challenging and often quit attempts were described as a struggle. This complements the sub-theme, "Motivation and self-

efficacy" (see Section 7.2.1), as participants described how self-doubt effectively antagonised perceived self-efficacy.

Although the majority of participants acknowledged the difficulties of quitting smoking (n = 24), current smokers were particularly more likely to portray quitting as a struggle and emphasise the difficulty of quitting, "I just wanted to quit, that's all it is really, just erm, finding it hard all those years of smoking, I just wanna give it up now, I'm finding it a bit hard like" (David, 37, Current). Another current smoker commented, "Well that's the aims of it (to quit) but erm, it is still a struggle trying to get off them... especially with people (are) smoking around you" (Alex, 56, Current).

Recent former smokers who had quit for longer periods tended to acknowledge the difficulty of quitting, although their concerns often regarded the earlier stages of quitting and the previous difficulties they had experienced, "Well every time I've had the urge, I always think, I'm not going through another week like that... apparently, I was unapproachable that week!" (Ronald, 40, Former). One recent former smoker described how his present concerns regarded cessation of a pharmacotherapy product, rather than smoked cessation per se, "I've still got, I haven't reached the stage where I'm comfortable coming off the lozenges yet, erm, but I think that'll be the hardest part" (Stuart, 45, Former), thus highlighting the importance of nicotine cessation overall to some service users.

Several participants also suggested that they had feared that they would be incapable of quitting smoking. This was most prevalent among recent former smokers who reflected on feelings prior to quitting. Recent former smokers described how they had

previously doubted their ability to quit, as they often expressed disbelief in the success of their quit attempt, "Yeah, I always assumed, I always thought I'd never, erm, have the courage to stop smoking" (Timothy, 58, Former). Another participant described the concept of smoking cessation as somewhat overwhelming prior to quitting.

I went to the doctors and it was him who said you know, "Wouldn't you think about it? Wouldn't you consider it?" and I was like, "Well I have tried a few times and I went back on them." and I said "I don't think I could, I haven't got the willpower." (Patricia, 50, Former)

This comment also demonstrates that previous failed quit attempts may contribute towards self-doubt, which appears to inhibit intentions to quit. Recent former smokers in the study willingly described the doubts they experience regarding smoking cessation prior to quitting smoking, yet current smokers failed to describe such experiences. This may have been due to social desirability bias, or potentially, self-doubt may be more prominent among current smokers prior to service engagement.

7.2.4 Smoker identity

A behaviour-specific identity may be formed when a given behaviour is gradually internalised over time, as a defining aspect of a person (e.g. identity as a smoker); this is of importance, as there is a positive bi-directional relationship between behaviour-specific identity and behaviour (Hertel & Mermelstein, 2012). The current results demonstrated that a sense of smoker identity was evident, as at least eight participants referred to the social-construction of smoker classification, how smoker identity

related to self-perception and quitting, or how smoker identity may have inhibited the reported sense of liberation from smoking that many participants portrayed.

Although smoker identity was most commonly referred to by recent former smokers, two current smokers described how they perceived their identity as a smoker engaging with support services, in relation to other smokers. One current smoker often described themselves as a non-smoker throughout the interview, for example, "I'll go outside and have a smoke but now I don't have to do anything, them things anymore, 'cause I don't smoke' (Jack, 49, Current); however, the participant subsequently described a situation whereby an RCFE advisor challenged the participant's perception of themselves as a non-smoker.

I went on Monday to the Royal and Keith (RCFE advisor), Keith wasn't there, Roy (RCFE advisor), the other guy, he said to me, 'cause ... I was having the odd few ciggies, and he said, "It doesn't matter if I smoke 30 and you smoke 10; you're still classed as a smoker, what you need to try and do if you can, he said, is ... just make a date and when they're going, they're going altogether" ... I'll tell him about this 'cause I'm still having the odd one you see Fran, so I'm still classed as a smoker, aren't I? (Jack, 49, Current)

Another current smoker, also refrained from describing himself as a smoker, suggesting that his alternate outlook positioned him separately to the way in which he viewed other smokers, "It's something I've felt recently but the thing is we're not all the same kind of people and my outlook on smoking is going to be different to somebody else's and I'm at the point of giving cigarettes up and that to me is a

different outlook to a smoker." (Luke, 54, Current). Such comments suggest that some current smokers adopt alternate or extended smoker identities, potentially to detach themselves from their traditional perception of a smoker and to promote successful smoking cessation.

Smoker identity was referred to primarily by recent former smokers. One recent former smoker described how smoking was viewed as a part of her life, which she recognised as an initial barrier to quitting smoking.

I always thought I'd never erm, have the courage to stop smoking, you know, because I'd smoked for that long and it was part of my life. That's how I looked at it you know; I smoke, I'm a smoker." (Julia, 49, Former).

Another participant described a conversation with a friend who smoked, in which her friend proposed that she was not capable of quitting smoking, as it had been part of her life for such a long period. The participant viewed this as a challenge to stop smoking, and subsequently quit for several months.

And I said, "Oh I'd like to quit", just in passing conversation and she said "You can't quit" and I said "Why can't I quit?" and she said because you've been smoking for the past 20, 11 years ..." and I said "I can quit ... I'm quitting!" (Charlotte, 26, Former)

This suggests that not only do participants view smoking as part of their own identity but additionally, some smokers view smoking as part of the identity of other smokers, and this could potentially be viewed as a barrier or facilitator to quitting. In the above example, the participant appeared to challenge the constrictions of a smoker identity.

Two participants also suggested that smoking would be a definitive aspect of identity among former smokers, throughout life. One participant described how former smokers should continue to be vigilant regarding cues to smoking, as a lack of vigilance could result in relapse. This also highlights how some service users viewed smoking to be part of their identity throughout the lifespan, regardless of quit duration.

... there's something about drinking alcohol that triggers something in your brain, even if you've given up smoking, all the ex-smokers, once they quit smoking, they'll go out, have a couple of drinks, see someone else smoking, it takes that to go back for an ex-smoker. They pick up a cigarette again. Once you're a smoker, you're always a smoker. It's like being an alcoholic, once an alcoholic, you're always an alcoholic. A smoker has to avoid that one cigarette. (Sarah, 55, Former)

Another recent former smoker described a conversation she had with a longer-term former smoker, in which she considered how smoking might remain part of her identity for life and this appeared to be initially disconcerting for the participant.

... one of the women I worked with, she's got breast cancer and that's how she's stopped and ... she's been stopped for years and she said "Oh god (participant's name)! I've been stopped for years and every now and again I

want one and then it just goes." and I thought, oh god, have I got that for the rest of me life? (Patricia, 50, Former)

The participant continued to describe how she began to accept that smoking could remain an ongoing aspect of her life despite quitting. She continued to describe strategies that other long term former smokers had employed to avoid smoking.

But they sort of talk me round 'cause as quick as you've thought about them, you know, as quick as you think about them, you know it goes, that smell, I mean one of them kept a dirty ash tray and every times she felt like a ciggy, she'd have a smell of the ash tray, and that was enough to put you off, so you're never bothered (laughs). You know little things like that? ... You can still exist and do things that you normally do, just without smoking. (Patricia, 50, Former)

It should be noted that both of the aforementioned participants were aged 50 years and over and therefore, it is unclear whether these perspectives are still relevant to younger recent former smokers throughout their lifespan.

In summary, although participants described a sense of freedom from the mundane and restrictive routine and addiction that entailed smoking, many recent former smokers simultaneously realised that smoking could potentially be part of their lives long-term; a number of former smokers described how they might have to remain vigilant of cravings and cues to avoid smoking. Despite this, the final comment

suggests that former smokers are satisfied to accept this as a condition of remaining smoke-free.

7.3 Perceived benefits of quitting

"Perceived benefits of quitting" is the other key major theme considered in the current chapter. This theme explores current smokers' perceptions and expectations regarding the benefits of quitting, whilst recent former smokers' accounts focused on their experiences and future expectations regarding the benefits of quitting. Both current and former smokers focused on the positive aspects of quitting, such as the perceived benefits, presumably to promote motivation and to reinforce smoking cessation success. This major theme consists of three prominent sub-themes, including "Improved health", "Improved finances", and "Image and functionality".

7.3.1 Improved health

Nearly all participants (n = 27) acknowledged an association between improved health and smoking cessation. The results demonstrate how most participants (n = 19) identified specific physical symptoms, which they typically attributed to smoking. Common symptoms identified to be associated with smoking, included coughing, breathlessness, and chest pain. Coughing was the most frequently cited symptom to be associated with smoking, "...it's the same now. I'm getting up every morning, coughing, coughing" (Gemma, 57, Current). Another participant described, "...you tend to think about it more. Like your breathing and things like that, like your chest. I've had a lot of trouble you know, you know with me chest? Coughing all the time" (Michael, 48, Former).

Several participants (n = 14) directly attributed the experience of physical health symptoms to motivation to quit smoking, as participants' comments tended to suggest that they sought relief from the physical symptoms associated with immediate health concerns through smoking cessation.

Just getting puffed out, start getting, you know, a bit out of breathe, it's just, it's just, I could see it having an effect on me, using me inhaler more, erm, like I said, mobility issues, so I just thought sod it, kick it on the head while I can. (Matilda, 33, Former)

Another participant explained how the occurrence of various symptoms had influenced them to consider smoking cessation, "It's been everything, it's been a health scare; I had high blood pressure, so I had to get that down" (Ronald, 40, Former).

A number of recent former smokers (n = 4) also described how previous health diagnoses had influenced them to quit smoking. The nature of the diagnosis varied in regard to length of condition and severity. One participant described how recurrent coughs and chest infections had influenced his decision to quit smoking, "Erm, the health reasons 'cause that's what caused me to pack it in. 'Cause I was forever having bronchitis, chesty coughs and things like that, so I had to pack it in' (Patrick, 56, Former), whilst another participant described how diagnosis of a cardiovascular condition had contributed towards her decision to quit, "Well I wanted to quit for ages but I couldn't but then I was told I've got a blocked artery in me leg and I thought, this is it now. I've had me warning, I've got to do something' (Gemma, 57, Current).

Several current smokers (n = 5) described how they too had experienced ill health and that this had influenced their decision to quit. Sadly, at least three of these current smokers, described how they had suffered particularly ill health in recent years and none of the three participants reported previous quit attempts that had lasted beyond a few weeks. One participant explained how he had been diagnosed with a number of respiratory diseases and expressed frustration at himself for continuing to smoke.

Then when I started with the asthma, I wasn't diagnosed with asthma until I was about 30, if not younger, 29, 30, so, still smoked then and I couldn't pack up even with me health ... Now I've got the COPD as well, now, I'm like "ah hello!" (Jack, 49, Current)

One participant who was slightly younger reported having suffered a number of strokes, "In the past, I've had two strokes and me doctors have told me smoking really doesn't help" (Joel, 44, Current). These comments demonstrate how, many current smokers suffering with ill health do perceive the benefits of quitting and associate improved health with smoking cessation, yet they often fail to sustain a quit attempt.

Improved health appeared to be a key anticipated benefit of quitting, however, the majority of participants also described health improvements subsequent to quitting. This was relevant to some current smokers, as well as former smokers, as some current smokers were able to recall experienced health improvements associated with previous quit attempts; this further enhanced anticipated perceived benefits of quitting among current smokers.

The most common health improvement associated with smoking cessation was an alleviation of respiratory symptoms, such as coughing or breathlessness. One recent former smoker described how their cough had improved since quitting.

You tend to think about it more. Like your breathing and things like that, like your chest. I've had a lot of trouble you know, you know with me chest? Coughing all the time, but it has eased up a little but since I have quit. It has like, sort of eased off (Michael, 48, Former).

One current smoker described improvements he had observed from cutting down, "Everything, you feel better in yourself, even when I'm walking, with me asthma and me COPD, I'm finding I can walk up the stairs better and everything" (Jack, 49, Current).

Participants tended to focus on respiratory improvements they had observed, although one participant focused on anticipated improvements in cardiovascular health as a result of quitting, "With me heart, I'm on heart tablets and blood pressure tablets now. Well I was, I don't know if I'll still be on them now because it's gone down an awful lot" (Sophie, 55, Former).

Although most participants described improvements in health as a consequence of quitting, one participant described how she had not identified any health benefits as a result of quitting, although she was not suffering from any health concerns prior to quitting, "I'm not like one of these who's feeling fit as a fiddle and all that like. I don't

feel any different that way but I didn't ever feel unwell or whatever" (Emily, 59, Former). The lack of health improvements did not appear to affect motivation, perhaps as her perceptions regarding the benefits of quitting whilst smoking were not of concern.

7.3.2 Improved finances

Comments regarding the cost of cigarettes featured heavily throughout the interviews. Several participants described being particularly conscientious regarding how much money they had spent on cigarettes, either currently or prior to quitting. Such participants were often able to recall and reflect on money spent on cigarettes, "If I was looking to smoke now though, it'd be costing me like £16.00 a day and I just couldn't afford it anyway. 'Cause they're like £7.00 to £8.00 per pack aren't they?" (Patrick, 56, Former). One participant had calculated the combined cost of his own and his spouse's smoking per year, potentially as a strategy to further enhance motivation to quit.

Yeah, because me and me wife smoked at the time and when we sat down and spoke about it, erm, I go to work in one direction and she goes to work in the other direction, and we were both taking £10.00 out of the machine, which was £140.00 a week, which £140.00 times 52, is £7280.00, just on cigarettes. Do you know what I mean? (Robert, 34, Current).

The rising price of cigarettes was a particularly significant issue revealed in the analysis. Participants frequently commented regarding the increased cost of cigarettes, "'Cause they're getting more and more expensive aren't they?" (Julia, 49, Former).

One recent former smoker stated, "I'd be a fool to go back to them now and with the money as well, I mean, they're not going to make them cheaper, they're just going to keep going up and up and up, aren't they?" (Patricia, 50, Former). Furthermore, participants did not seem to be opposed to the increasing cost of cigarettes, as most commented how increasing the cost of cigarettes would likely be the most effective way to encourage people to quit smoking. This also fits well with the next chapter, which explores influential aspects of tobacco control practice and policy.

I mean I think like, the way the government goes about it, I mean because it is expensive, it does put people off, you know what I mean? You think like, you know it's costing so much money and you're not getting anything from it back, you've only got so much money to pay with so, it is to your benefit, so if you can pack in, pack it in. (Michael, 48, Former)

Another participant commented, "I think it makes people think more about packing in and especially the prices now, that's one of the issues of a lot of people now" (Sophie, 55, Former). Indeed, one participant directly attributed quitting to the increased cost of cigarettes, suggesting that for some service users, money may be a particularly important motivating factor, "If they were still 'round about £3.00 or £4.00, I'd still probably be a smoker ..." (Alex, 56, Current).

Although increasing the cost of cigarettes was typically viewed as an effective strategy to encourage smokers to quit, the analysis additionally revealed some aspects of this approach that could be problematic. The increasing cost of cigarettes appeared to put financial pressure on a number of participants, which is of particular relevance as most

participants were unemployed and in receipt of benefits. One recent former smoker explained how they had previously struggled to fund smoking, which was a source of stress, "Ok, you know, cigarettes, the price of them, and I was thinking, you know, where am I going to get my next pack of ciggies from?" (Julia, 49, Former), whilst another participant similarly described their concerns.

'Cause I know when I'm smoking and I have to say to meself, I can't get that because I need to make sure I have enough money for ciggies or I can't get that because I'd rather have 20 ciggies than that. (Charlotte, 26, Former)

This is concerning, as although increasing the cost of tobacco can make smoking less affordable and therefore, reduce smoking rates, some participants from particularly deprived households may potentially prioritise cigarettes over essential items, which could jeopardise health and wellbeing. This aspect also inter-links with a previous section in this chapter, which explored regaining control (Section 7.2.2). Furthermore, some participants (n = 3) suggested how the increasing cost of cigarettes may lead some current smokers to consider other alternatives, including criminal activity or options that could potentially be worse for health. One participant suggested that increased cigarettes costs in the UK may influence smokers to obtain cheap cigarettes abroad, which could induce further damage to health.

You can go to places like say Turkey and places like that where you can get cheap cigarettes... as soon as you have a cigarette, it burns the back of your throat and you know it's a dodgy cigarette and that's the other thing that people

are going to go down the line, they're going to start just smoking anything.

(Gavin, 45, Former)

One current smoker suggested that increased cigarette prices could influence increases in crime, as smokers may feel that cigarettes are essential.

The only way you can get a person to stop smoking is just to keep pricing it up and up and up, until they can't afford it and the only option then is to give up... but then I don't know, do people turn to crime then to go and get them? (Alex, 56, Current)

Participants were typically forthcoming in explaining how they had saved money since quitting or cutting down. As demonstrated in previous quotes, many participants reflected on the amount of money they would save over a given time period if they quit. A number of participants reported saving money since quitting or cutting down. One current smoker commented, "And the money, as well, I'm only on benefits as well, so I'm finding the extra money comes in handy, well handy" (Peter, 50, Current). One recent former smoker reflected on the money saved since quitting.

At the moment now, I'm thinking of how much money I'm going to save and how much I'm going to have in me little tin that I've bought to put me money in (laughs). Oh, only it's less than what you pay for your cigarettes but only like, up until now I think I've got £6.00 in there. It's only like a pound or two pound, you know whatever I've got in me purse; I just put it in a little tin. (Jean, 59, Former)

Participants also detailed how they intended to spend, or had spent, the money saved since quitting or cutting down. Although the nature of purchases or desired purchases varied, five participants described how they intended to go on holiday using the money saved. One recent former smoker commented, "Oh yeah, we're going on holiday. Erm, for the first time in 10 years, we're going abroad" (Charlotte, 26, Former). One current smoker described how the prospect of saving money and going on holiday with his family promoted his motivation to quit smoking.

Yeah, so it was a no-brainer for me. I just wanted to quit and quit as soon as possible to give me children maybe that extra bit of money, to maybe go down the caravan more often or take them out a little bit more often." (Robert, 34, Current)

Four participants also described how they had spent, or intended to spend, money saved since quitting or cutting down on home improvements. One participant described how they felt they were no longer constrained financially since quitting, enabling them to feel less concerned about spending money on items other than cigarettes.

And the money part of it as well. Say I used to go the shop, erm, any shop, say you went in B&M for instance, and seen like a bedding set or you know, something for the house and I'd go "Aww look at that!" and then I'd look at me money and think, aww that'd be ciggy money, so I wouldn't buy it,

whereas, now I go in and if I see something, I'll go "Look at that!" and I'll just buy it, don't even think about it. (Patricia, 50, Former)

Another participant detailed how they were presently redecorating their home with the money saved over the three months since quitting.

Well you know, the money I've saved, I've moved into me new flat, that's what I'm doing right now, I'm painting it all, having a break, so, I've saved a hell of a lot of money. I've saved up and bought all new furniture and that's just three months, so, you know, so, I'm made up with that, and deservedly am. (Paul, 38, Former)

Overall, the cost of cigarettes was not only a motivating factor for quitting, but the money saved from quitting was perceived as a reward for the behaviour change, and this was acknowledged by several participants.

It's just the money now, because the money you spend on cigarettes, you know stopping and cutting down on it, I've been buying things around, you know the apartment where I am living and you know ... instead of just going out to buy ciggies, you can just put money to one side. I'm buying things now, I've got things to show for it. (David, 37, Current)

Whilst, one recent former smoker stated, "Well, you know, if you can buy yourself something and then you know, think, well I bought that 'cause I didn't smoke, you know... I think it gives you a bit of a boost as well" (Patricia, 50, Former).

7.3.3 Image and functionality

Participants were concerned with various components associated with self-image. Self-image may be described as the collection of subjective perceptions regarding oneself, one's body, mental functioning, social attitudes, and adjustment in various aspects of life (Offer, Ostrov, Howard, & Dolan, 1989). With respect to self-image, participants in the present study commented most often regarding aspects of physical image, which they anticipated or experienced improvements in following smoking cessation.

Seven participants acknowledged that weight gain was associated with smoking cessation. Two participants perceived weight gain as a benefit of quitting smoking, although both participants were male. One recent former smoker described how he had gained weight since quitting,

It's been four weeks that I've stopped and I've gone from 12 stone to 13 stone, which is like, 'cause I'm 6 foot but I want to get my weight back up to like 13 and a half, so I've got another half a stone to go and then I'm happy. That's only within four weeks of not smoking." (Jacob, 59, Former)

One current smoker was also asked which benefits they associated smoking cessation with, to which they responded, "If I quit smoking, I'd probably put on more weight and things like that" (Eric, 25, Current). Contrary to these comments, five participants perceived weight gain as a disadvantage of quitting smoking, rather than a benefit; however, these participants made light of the degree to which weight gain was viewed

as disadvantageous, often with humour, as smoking cessation was viewed as a priority, "Instead of going for a ciggy, I go for a sandwich and I don't care if I do go like a big roly pole (laughs)" (Jack, 49, Current). One recent former smoker described, "Yeah, although I have gained a little bit of weight, which I'm not happy about, but I'll pedal that off in the nice warm sunshine (laughs)" (Charlotte, 26, Former).

Several participants also commented on the effect smoking has on skin (n = 5), perceiving improvements in skin appearance as a key benefit of quitting. One younger female spoke extensively regarding the association between skin deterioration and smoking, suggesting that concerns regarding skin deterioration motivated her to quit smoking,

I think last year ... I'd seen me Aunty and she walks round with horrible wrinkles on her and it was all, that was the main reason and I think this time, even though I still don't want wrinkles ... I just think to meself, oh well you know, I'm only 26 and you know, these people are not 'til, they're like 50, 60 when they find themselves getting wrinkles ... but it still all adds up doesn't it? (Charlotte, 26, Former)

The remaining four participants of the five, were also recent former smokers, who described how they had observed an improvement in their skin condition since quitting smoking, with males and females commenting equally. One female stated, "I think me skin's getting better as well ... on me face; pity about me arms and me legs like! (laughs)" (Emily, 59, Former), whilst one male participant described how improvements in skin condition were considered a perceived benefit to quitting,

"Getting healthy again and seeing me skin, going back to, you know, the way it should be and stuff like that" (Jacob, 59, Former).

A small number of participants expressed concerns regarding the associations between smoking and oral health issues, such as teeth discolouring and gum disease (n = 3). One participant also suggested that their teeth were whiter since quitting smoking, "...me teeth are a lot whiter" (Stuart, 45, Former), demonstrating that one perceived benefit of quitting was improvements in teeth colouring. Two of the participants who made the same association described teeth discolouring and gum disease as a deterrent for smoking, "(When you smoke) your teeth go yellow, like problems with your teeth and all that... all your gums start disease in. Do you know what I mean?" (Michael, 48, Former). Another participant suggested that dental issues had previously motivated her last quit attempt.

My reasons last year that I wanted to quit, me reasons last year were because I went the dentist and I've always had perfect white teeth and she said "Oh you've got a bit of build-up." or something like that and I just went "Oh that is it! That is it! No more!" Because it was just horrible, I always said, I liked me teeth the way they are. (Charlotte, 26, Former)

Although few comments focused on oral health, the above statements suggest that some service users may perceive improvements in oral health as a benefit of quitting, suggesting that such perceptions could promote motivation to quit smoking.

Lastly, but most significantly with regard to image, over two-thirds of participants commented on the odour associated with smoking (n = 21). This is relevant to self-image in particular, as participants often described how they perceived themselves whilst smoking or how they felt others might have perceived them. Generally, participants disapproved of the smell of smoke, "You know when you go by someone who's been smoking and I think 'Oh my god, the smell is just vile!' and I think I must have smelt like that meself, but you can't smell it on yourself." (Patricia, 50, Former). Another participant exclaimed, "Do you know what? When I go around to someone's house and it stinks of it, and I cannot stand the smell, I cannot stand the smell!" (Jack, 49, Current). All participants who commented on the smell disapproved of it, except one recent former smoker, who found the smell to be pleasurable and considered it a trigger.

I do like the smell of people when they're smoking when I walk past them, I do like that! (laughs) Bizarre! ... when I'm walking past a shop ... and I get a quick whiff of it, it's like, "Ooh! That's nice!" (laughs). (Ronald, 40, Former)

Several recent former smokers explicitly perceived no longer smelling of smoke as a benefit of quitting smoking, "I smell a lot better, I don't have to use as much aftershave (laughs)" (Stuart, 45, Former). Another recent former smoker noted, "Well, (when you smoke) you smell for a start. You're conscious of your breath. I mean, you can brush your teeth ten times a day and you've sweaty breath because you're smoking cigarettes" (Matilda, 33, Former).

Participants also commented on improved physical functionality in a number of areas. Firstly, eight participants described how they associated smoking with a loss of taste function. One participant described how smoking had affected their ability to taste, yet whilst they smoked, they had been unable to recognise this, "... the taste, you know when you have a cup of tea and then a ciggy, oh, your mouth just tastes horrible, because when I quit, I realised how bad that actually is" (Charlotte, 26, Former). Another recent former smoker described how their ability to taste had returned since quitting and how the extent of the improvement was unanticipated, "You don't realise when you're doing it, until you've stopped and all the, your taste buds and everything are back, how bloody idiotic it was ..." (Timothy, 58, Former).

Secondly, with regard to physical functionality, a number of participants also perceived an improved sense of smell as a benefit of quitting smoking, as smoking had dulled their sense of smell. One recent former smoker described, "Like you can smell things around you more, like flowers and all that, different types ..." (Michael, 48, Former). Another participant was asked what they viewed as the best things about quitting, responded in part, "One, better smell, you can smell things better" (Shaun, 48, Current).

Thirdly in relation to improved functionality, and relevant to improved taste and weight gain, a number of participants (n = 5) also briefly described how they had observed an improved appetite since quitting smoking, "Yeah, I'm eating like a horse! I've just spent a week in Egypt all-inclusive and I've never eaten so much in me life!" (Ronald, 40, Former). Another recent former smoker stated, "Yeah, well it improves your health, improves your appetite" (Jacob, 59, Former).

The last component observed in regard to functionality, which was expressed among both current and recent former smokers, was an overall sense of improved vitality and quality of life (QOL). Seven participants identified this benefit, since they had quit or cut down. A number of participants described how they had exhibited improved energy levels.

I had no energy. And since I have stopped smoking, I feel as though, erm, I'm not as tired. I think that when I was smoking, not all the time but I'd feel tired, I felt drained. But now I don't, I feel like, because I keep saying to meself, I say, "God, where am I getting the energy from?" and I think, is this because I stopped smoking or is it just me? (Julia, 49, Former)

Another participant also commented on feeling more energetic, "I feel, I was feeling healthy anyway because after feeling so big (the participant refers to previous weight loss) but I do feel a lot fitter. And me energy, I find me energy picked up as well" (Gavin, 45, Former).

Several participants described a general sense of improved QOL, incorporating psychological and physical components of wellbeing, "Everything! Everything! You feel better in yourself ... just everything, everyday life, just a better quality of life as well" (Jack, 49, Current). Another recent former smoker stated, "I feel younger (laughs) ... I'm not young like but I feel it ... I felt like, 90 before I packed in smoking ... I'm 21 now!" (Sophie, 55, Former).

7.4 Discussion

The current chapter explored factors implicated in smoking cessation success. Qualitative methods were employed to explore aspects relevant to service users' personal experiences of quitting smoking. In particular, this chapter identified two key themes, including "Experiences of mental conflict" and "The benefits of quitting". Participants appeared to draw upon psychological resources to focus upon the positive implications of quitting; this was apparent as they extensively described the key benefits of quitting smoking, as well as discussing efforts to combat negative cognitions regarding aspects such as self-doubt and temptation. The results of the current chapter, the potential strengths and limitations, and the implications are now discussed.

7.4.1 Exploration of chapter findings

The findings in relation to the theme, "Experiences of mental conflict" will be explored firstly. This particular major theme demonstrated the confliction of intentions, motives, and impulses that service users described encountering upon quitting smoking. The sub-themes associated with this major theme, included "Motivation and self-efficacy", "Regaining control", "Self-doubt" and "Smoker identity".

Motivation and self-efficacy were both identified by participants as important components for smoking cessation success. Motivation is a well-established predictor of smoking cessation success (Li et al., 2010; Li et al., 2011), whilst self-efficacy has also been found to predict smoking cessation success (Hendricks, Delucchi, & Hall,

2010; Schnoll et al., 2011). More specifically, several participants described how smoking cessation should be an autonomous decision, motivated by self, rather than others. This links closely with behaviour change theory, such as Self-determination Theory (SDT) (Deci & Ryan, 1985), which stipulates that smoking cessation (or behaviour change more broadly) is most likely when individuals feel that the choice is autonomous (i.e. the individual chooses to change behaviour), rather than the choice being forced upon them.

In relation to motivation, self-efficacy and additionally, self-doubt, participants described implementation of positive self-talk. As described, self-talk (i.e. inner speech), has been found to assist cognitive and self-regulatory functions (Diaz & Berk, 1992; D. G. MacKay, 1992). Furthermore, a number of studies have implicated the effect of self-talk in regulating smoking behaviour (Kelly, Zuroff, Foa, & Gilbert, 2010; Merchant, Pulvers, Brooks, & Edwards, 2013; Naughton, McEwen, & Sutton, 2013). The current findings provides further evidence for cognitive-specific strategies, as participants described how they would implicate self-talk to regulate smoking behaviour.

Participants described an experience of regaining control over cigarettes and over their lives more broadly, upon quitting smoking. In some contexts, smoking has previously been adopted as an integral aspect of a liberated identity; for example, the cigarette was previously promoted as a symbol of emancipation among women, a "torch of freedom" (Amos & Haglund, 2000). The current results demonstrate how smokers, including women, no longer associate a sense of liberation with the opportunity to smoke, but rather a sense of liberation was associated with smoking cessation. Many

participants felt that their lives had been dictated by smoking and therefore, smoking cessation was associated with regaining control, as recent former smokers were able to live their lives without the constraints of smoking. One recent review (Goldenberg, Danovitch, & IsHak, 2014) concluded that there is a negative relationship between smoking and QOL; the current results support these findings and demonstrate the mechanisms by which smoking can impact QOL.

The concept of a smoker identity was also frequently discussed among participants. A recent meta-ethnography (Tombor et al., 2015) suggested that identity plays a role in smoking cessation, as identity appears to influence intentions to quit. The current results provide further support for this notion and demonstrate how this relationship may be facilitated. A number of recent former smokers in the present project expressed concerns that smoking would always be part of their identity; one previous study found that identity progression to that of a "non-smoker" was not necessary in order to achieve long-term abstinence (Vangeli & West, 2012), whilst the current findings appear to corroborate this result.

"Perceived benefits of quitting" was the other key theme identified in the current chapter, which consisted of the sub-themes "Improved health", "Improved finances", and "Image and functionality". Anticipated and experienced perceived benefits of smoking cessation were consistently discussed across the interviews; participants appeared to focus largely on the perceived benefits of smoking cessation in order to promote motivation and to reinforce behaviour change.

Health concerns featured prominently throughout the interviews as one of the key incentives for quitting. Previous research has found health concerns to be a key implicating factor in smoking cessation motivation (McCaul et al., 2006), whilst health improvement is one of the key aspects that former smokers find helpful in remaining abstinent (Katz & Singh, 1986). The vast majority of participants described how the experience of physical symptoms and development of disease attributed to smoking prompted their decision to quit smoking. This finding also complements the results of the present quantitative analysis, which indicated differences in health risk perceptions between current and recent former smokers.

Previous research suggests that many current smokers in poor health often fail to internalise or personalise the health effects of smoking (e.g. Bock et al., 2001); some current smokers in the present study described poor health and limited smoking cessation success previously, yet they often fully acknowledged the effects of smoking on their health. This example demonstrates how risk perception alone cannot fully explain smoking cessation behaviour, as addiction is a complex, multifaceted phenomenon (Ross & Kincaid, 2010).

Participants also commonly noted how improved finances were perceived as a beneficial outcome of smoking cessation. The increasing cost of cigarettes appeared to motivate some participants to quit smoking, a notion which supports previous research. For example, Hsieh, Chen, Lee, and Yeh (2014) examined the effect of cigarette price levels on smoking prevalence; simulated results suggested that a 10% increase in cigarette price would result in a reduction in smoking prevalence of 1.42% in the UK. Simultaneously, the current findings also generated some concerns, as some

participants from more deprived backgrounds described how they had previously prioritised cigarettes over other general needs. This suggests that participants' overall health and wellbeing could have been jeopardised, as cigarettes were viewed as essential. This demonstrates how some tobacco control strategies may result in inadvertent consequences, which could result in marginalisation of smokers in society.

Improvements in self-image and functionality were also considered to be a beneficial outcome associated with smoking cessation, albeit, not to the extent improved health and finances were. In particular, physical appearance was of greatest concern. Improvements in image have previously been cited as one of the key components that former smokers found helpful in remaining abstinent (Katz & Singh, 1986). The majority of research that considers the relationship between physical appearance and smoking cessation, refers to young females (e.g. Grogan, Fry, Gough, & Conner, 2009; Hysert, Mirand, Giovino, Cummings, & Kuo, 2003; Riedel, Robinson, Klesges, & McLain-Allen, 2002); however, the current project identified that perceived benefits of quitting regarding image were valued irrespective of age or sex, except in relation to weight-gain.

A number of components of functionality were also acknowledged as a perceived benefit of smoking cessation. A recent meta-analysis identified that former smokers exhibited improvements in mental health, as they no longer experienced negative affect associated with acute nicotine withdrawal between cigarettes (G. Taylor et al., 2014), whilst, further research found that recent former smokers often exhibit changes in energy expenditure (Allen, Brintnell, Hatsukami, & Reich, 2004; Perkins, Epstein, & Pastor, 1990; Vander Weg, Klesges, Eck Clemens, Meyers, & Pascale, 2001); such

factors could contribute towards the denoted perceived improvements in energy levels and overall QOL described among recent former smokers in the current project.

It should be noted that participants would freely describe the perceived benefits of smoking cessation in great detail, with little focus on the perceived barriers to smoking cessation, regardless of smoking status. This might suggest that participants relayed such benefits in an effort to reinforce behaviour change, in line with behaviour change theories, such as the Transtheoretical Model of Change (TTM) (J. O. Prochaska & DiClemente, 1983; J.O. Prochaska & DiClemente, 1986); again, recent former smokers were most forthcoming with regards to listing perceived benefits, which fits well with the stages and processes of change stipulated in the model.

7.4.2 Potential strengths and weaknesses

As described earlier in the thesis, the semi-structured design of the qualitative interview schedule allowed for both the inclusion of pre-determined questions and the opportunity for the interviewer to explore varying themes and responses more indepth. This approach enabled the identification of several sub-themes and concepts in this chapter, such as smoker identity and positive self-talk, which might not have been identified if a semi-structured design had not been implemented.

The current qualitative investigation does, however, prevent the inference of causal relationships between smoking behaviour and potentially motivating factors, such as perceived benefits of quitting; Some qualitative designs have integrated a number of waves to interviews, to explore the effects of various potential predictors of smoking cessation over time (Hargreaves et al., 2010; Ritchie, Amos, & Martin, 2010a, 2010b);

doing so, could have further enriched understandings of smoking behaviour. Recent former smokers were, however, able to retrospectively recall their experiences of quitting at various stages, which enriched the analysis and provided justification for some suggested causal relationships.

7.4.3 Implications for tobacco control knowledge, practice and policy

The current chapter findings have several implications for tobacco control knowledge, practice and policy. The analysis aided the development of the prominent theme, "Experiences of mental conflict", which generated various sub-themes and concepts of interest. Areas for future development include positive self-talk, inadvertent effects of tobacco control campaigns, and smoker identity.

There is a paucity of research in regards to the application of positive self-talk among smokers attempting to quit. The current project revealed that this method of self-regulation was common among smokers, yet, previous research has failed to explore the development and evaluation of interventions to promote positive self-talk among smokers. Further research is warranted, as the results of the current qualitative analysis deem positive self-talk to be a helpful strategy in promoting smoking cessation success.

The results of the current chapter also identified the concept of a smoker identity. Some recent former smokers expressed concerns that attributes associated with a smoker identity might remain following long-term smoking cessation and this appeared to be accepted as a condition of abstinence. There is currently a paucity of research in relation to the concept of smoker identity, especially in respect to

quantitative research. Further studies could explore this concept in greater detail and establish whether the existence of a smoker identity inhibits or promotes smoking cessation success.

The current chapter also demonstrated how some tobacco control strategies (e.g. cigarette price increases) could inadvertently potentially marginalise and jeopardise the health and wellbeing of some smokers. This concept is considered further in the following chapter, which focuses upon influential aspects of tobacco control practice and policy; however, future efforts should remain sensitive to the impact of tobacco control policies on some current smokers.

The present chapter results also demonstrate how many participants underestimated the improvement smoking cessation would have on functionality, such as QOL; further research should explore this concept further, as improved awareness of such benefits could encourage more smokers to quit.

7.4.4 Conclusion

In summary, the current chapter presented the results of part of the qualitative analysis, which aimed to examine factors implicated in smoking cessation success. The results of the current chapter demonstrated how participants would focus greatly upon the positive aspects of smoking cessation, such as the perceived benefits of quitting, and only briefly, were the perceived barriers considered. Furthermore, the experience of mental conflict was described consistently among participants, demonstrating the psychological battle which ensued upon attempting to quit smoking and sustain

abstinence; again, participants described efforts to promote positive coping strategies and counter negative cognitions in an attempt to remain smoke-free.

This chapter demonstrates the benefits of implementing a semi-structured design, as much of the chapter implications emerged from sub-themes and concepts, which would not have been identified if the interviews had been designed in a strictly structured manner. Various opportunities for future research and development were also considered in relation to aspects, such as positive self-talk, inadvertent effects of tobacco control campaigns, and smoker identity. The following chapter will further explore factors implicated in smoking cessation success, focusing on influential aspects of tobacco control practice and policy.

Chapter 8: Influential aspects of tobacco control practice and policy

8.1 Introduction

Similarly to the last chapter, the current chapter investigates factors implicated in smoking cessation success. The present chapter results explore service users' experiences of smoking and smoking cessation, with focus on components associated with tobacco control practice and policy. By conducting such research, it is possible to consider aspects related to policy and practice that service users might have found helpful or potentially unhelpful in promoting smoking cessation. The findings can be used to inform future service development and policy-making. The present chapter addresses one key thesis objective:

• To explore factors implicated in smoking cessation success.

The current chapter will firstly considers participants' experiences regarding the transformation of smoking-related social norms in recent decades. Participants suggested that smokers had become increasingly marginalised within society, whilst several described feelings of judgement or stigma, which were primarily attributed to tobacco control policy. Secondly, attitudes regarding local SSS are explored. SSS were greatly valued by participants; key aspects of services are detailed and potential service improvements are also described. The two major themes identified in this chapter include: (1) Reshaping social norms; (2) Attitudes regarding local Stop Smoking Services. These two themes will be described in greater detail and the respective sub-themes for each will be discussed subsequently.

8.2 Reshaping social norms

"Reshaping social norms" is the first major theme considered in this chapter. Lapinski and Rimal (2005) suggested that collectively, "Norms serve as prevailing codes of conduct that either prescribe or proscribe behaviours that members of a group can enact" (p. 129). The white paper, "Healthy Lives, Healthy People: A Tobacco Control Plan for England" (DH, 2011) described how tobacco control efforts aimed to encourage communities across England to reshape social norms, with the intention of making smoking less desirable, acceptable and accessible. This desired change in social norms was evident in the results of the current chapter, as participants reported transformation with regards to societal attitudes towards smoking and considered how such changes impact smoking behaviour. This major theme consisted of two prominent sub-themes, including "A sense of belonging" and "Judgement and stigma", which will now be described.

8.2.1 A sense of belonging

"A sense of belonging" was the first prominent sub-theme associated with the major theme, "Reshaping social norms". Participants were firstly asked what they felt triggered them to start smoking initially and primarily, participants described how smoking was a normal behaviour among family and friends throughout youth. Most participants (n = 23) described how many of their relatives smoked and a number of participants additionally attributed smoking initiation to the culture of smoking that was prevalent within their families. One older participant described how most of her first and second-degree relatives smoked at the time she started smoking, "My grandmother smoked, all my aunties smoked, my father smoked; it was one of the things that you did back in the day" (Sarah, 55, Former). It was anticipated that this

account would be most prevalent among older participants, as smoking prevalence has declined for a number of decades; however, this association appeared to be evident irrespective of age. One younger participant described how her mother and step-father both smoked, which she attributed to her own smoking initiation.

Yeah, err, it was because of me mum, me mum had smoked for all me life and then she quit for like 4 years, and er, I walked in one day and caught her smoking after like four years ... I think it was just like payback like oh I'll try it 'cause there must be something to it (laughs). I was only 14, so you don't know much then do you? ... I think 'cause me stepdad was smoking as well, I used to pinch his ciggies, I think that was it (laughs). (Charlotte, 26, Former)

At least nine participants suggested that smoking was typical within their friendship groups, at the time of smoking initiation. Several participants provided accounts of how they experimented with smoking among friends in school.

Yeah, just friends. I always remember, we all started at the same age, which was about 12 and we were sitting behind a coach, having a ciggy each and then throwing up, and then having another ciggy, and then throwing up and having another ciggy! And then before we knew it, we got hooked on them. (Stuart, 45, Former)

Another participant commented, "Start smoking? It's just that all my friends were smoking at the time. I was young ... yeah, everyone smoked" (Gemma, 57, Current).

The analysis also identified the mechanisms by which social norms effect smoking initiation. Two participants suggested that smoking was learnt by observing significant others smoking. For example, one participant attributed his daughter's non-smoking status to his own non-smoking status throughout the time she grew up.

I think it's changed now with the young ones 'cause the likes of me daughter, she doesn't, I put that down because of her growing up, I didn't smoke and while she was growing up I never smoked. I put that down to she never saw us smoking, so she never wanted to try it or copy it. (Gavin, 45, Former)

At least seven participants, however, suggested that smoking initiation was attributed to a willingness to feel a sense of acceptance or belonging within a particular social group, in which smoking was considered normal. One participant described how they had started smoking in an attempt to fit in with new friends,

Erm, all me friends smoked when I was 13, so I just like joined in, everyone else was doing it ... I joined a youth group and everyone else smoked, so it was just sort of like, to fit in with everyone else, 'cause everyone else smoked. (Matilda, 33, Former)

Another participant described how they conformed to smoking seemingly to seek group acceptance, "I was about twelve when I started. Everyone at school was doing it so you just get in with the gang, don't you?" (Emily, 59, Former).

Social norms were not only important in contributing towards smoking initiation but they also appeared to greatly implicate smoking cessation and relapse. In the mid-1970s, almost half of the British population smoked, whereas currently, smokers consist of only a fifth of the British population (ONS, 2013b). Six participants highlighted observed reductions in smoking prevalence within recent decades, which implicated social norms around smoking. Three of these participants explicitly attributed changes in social norms to motivation to quit.

Erm, I was about twenty when I started, and I think it's err, I think it's the way the world's going, everyone smoking not become sociable no more. Do you know what I mean? I think that's why I gave up because everyone had stopped around me and I'm glad I did because I feel much better in meself. (John, 53, Former)

One participant highlighted how being the remaining smoker in a social group not only motivated her to quit, but knowing of others who had successfully quit enhanced her perceived self-efficacy to quit. The participant also described how a smoking habit is easier to maintain when others smoke.

I had friends that smoked and it was just so easy when they were smoking, you know, "have one". You know but it's different now 'cause the girls I actually work with they've quit for 9 months and the caretaker at the school, where I work, he packed in smoking about two years ago, and we all used to go for a ciggy and me other friend who used to smoke as well, she's left but she used to go and have a smoke, but then he packed in and she left so that's just left

me and I thought, oh well, and I'd just said, aww, if they can pack in then I must be able to. (Patricia, 50, Former)

Although several participants described how smoking became atypical within their social networks and this had motivated some to quit, conversely, many more participants described how smoking was normal among their present social networks (n = 17), which often antagonised attempts to quit smoking. One participant described how he found it difficult to refrain from smoking in the presence of his girlfriend smoking, "Yeah, me girlfriend does smoke. I do find it hard with her, I do have the odd cig but that's about it ... in my view, it's when I'm sitting 'round people' (David, 37, Current), whilst, another participant described how smoking was normal within his family, which posed a barrier to quitting.

Yeah, being around people smoking ... it really is, especially around meal times, around meal times and that, you know your family and everyone else and you start lighting up ... it is still a struggle trying to get off them ... especially with people smoking around you. (Alex, 56, Current)

The results demonstrate how social norms can be viewed as either a facilitator or a barrier to quitting, dependent on the situation; if a smoker is part of a social group in which members are predominantly non-smokers, this could promote smoking cessation, whereas, if a smoker is part of a social group in which members are predominantly smokers, this could impede attempts to quit smoking. Furthermore, three participants described how they associated social smoking with a kind of

camaraderie and even a sense of belonging, which meant that becoming a non-smoker led to feelings of social isolation.

Erm, probably feeling pushed out, because I'm in an atmosphere where everyone smokes, everyone who comes to my house smokes, and 'cause I feel like I'm not involved in the atmosphere, I think that's going to be, especially this time as well, I think that's going to be the hardest 'cause I'm the only one who doesn't smoke, I'm the only one who goes in the living room because everyone else sits at the table and as soon as they put a cig out, they'll be lighting another one ... (I'm) just trying to get out of the way, but that's not helping 'cause I feel isolated, like I don't belong at the minute but if I remember rightly, I went through this last time because I could go and sit in the kitchen and not want one. (Charlotte, 26, Former)

Another recent former smoker described a similar experience, whereby she felt uncomfortable and out of place in the early stages of smoking cessation, but that this stage passed eventually.

Most of my friends and family are smokers, so I found that I had a stigma because I wasn't smoking ... because I didn't feel like I was part of the group. At the local pub, everyone goes outside for one so if you're a non-smoker, you're sat at the table, minding the drinks while they're all outside having a ciggy, and when they come back in, you feel like you've missed out. At first I found it difficult and then, you know, I just went outside with them for a

cigarette but I didn't smoke, just to be part of the conversation ... (Matilda, 33, Former)

These comments further highlight the impact of social norms on smoking behaviour and demonstrate how feelings of social isolation could potentially promote or impede quit attempts; group membership and acceptance is clearly a primary concern for many smokers.

Despite many participants expressing concerns regarding social isolation in the early stages of quitting, two recent former smokers described how they had no concerns regarding significant others smoking in their presence, "Me dad's seventy-seven and he smokes and all that, and he smokes in front of me, and it doesn't bother me in the slightest" (Stuart, 45, Former). Another recent former smoker explained, "People still smoke around me now but you know, it doesn't bother me" (Sophie, 55, Former). These comments, alongside the aforementioned accounts, suggest that the extent to which social smoking antagonises quitting potentially diminishes over time.

8.2.2 Judgement and stigma

"Judgement and stigma" was the second prominent sub-theme associated with the major theme, "Reshaping social norms". Stigma occurs "when elements of labelling, stereotyping, separating, status loss, and discrimination co-occur in a power situation that allows these processes to unfold" (Link & Phelan, 2001, p.382). The majority of participants (n = 17) reported feeling judged and even stigmatised for smoking. One recent former smoker described, "They're looked down upon now aren't they by a lot

of people, people who smoke. You are really stigmatised if you're a smoker in some places" (Patrick, 56, Former).

Participants suggested that the reported stigma attached to smoking had become increasingly apparent within recent years, potentially due to increased implementation of tobacco control strategies, such as the smoking ban in England. Feeling uncomfortable or judged for smoking outside public places was common among participants. One participant described, "You know, you felt like, once the smoking ban came in, kind of a leper, that you had to go outside and all that" (Emily, 59, Former), whilst, another participant explained, "... there's definitely a stigma now, it's terrible ... if you go to a restaurant or a bar or anything, you get looks off people and that" (Gemma, 57, Current).

Several participants described how the introduction of policies such as the smoking ban and the increasing sense of marginalisation may have promoted motivation to quit, "I suppose in a way it's good because it does make you think more, it actually does because it's like with the weather, you used to stand out there, smoking, a couple of pints, you're having a cigarette and people are looking at you and you think well, what am I doing?" (Michael, 48, Former). Another recent former smoker also described how he previously felt judged and subsequently, guilty for smoking outside public places despite it being legal, "...sort of like people staring at me and I was like oh, well I was outside but I felt guilty, you know what I mean? I can understand it, you know what I mean?" (John, 53, Former). The aforementioned comments suggest that increased feelings of guilt and discomfort associated with smoking outside public places may potentially promote motivation to quit.

In addition to participants reporting feeling judged or stigmatised for smoking, the analysis suggested that many participants (n = 9) made judgements regarding other smokers, which were typically disapproving. Furthermore, all of the participants who commented regarding other smokers were recent former smokers. One participant described how his daughter smoked; he describes how his daughter did not smoke in his presence, yet, he deemed his daughter to be antisocial and selfish, as she often smelt of smoke in his presence.

... when one of our girls walks in, I can smell it a mile away and they're sucking mints before they come in here and you can still get on it. It's very, what's the word I'm looking for, very unsociable and selfish is the word ... selfish yeah, I'm not saying every smoker's selfish, well I am really aren't I? (Timothy, 58, Former)

Another recent former smoker reflected on how she felt observing others smoking since quitting. The participant appears to view the image of smoking as unpleasant and undesirable; such negative judgements could potentially further reinforce her smoking cessation success.

I look at people with their ciggies and I'm thinking it might sound strange but, the other week I was just, I was sitting outside, the weather was lovely and there was just like two women, just gabbing a bit and I was looking at them, and they were smoking and I felt like saying to them, "Do you enjoy that?" 'Cause to me, looking at them, it didn't look as if they were enjoying it. They

were more like talking and I thought, that used to be me with a ciggy in me hand; it doesn't look nice to be honest with you. (Julia, 49, Former)

Three recent former smokers additionally made judgements regarding current smokers who were attempting to quit but had yet to become abstinent; participants demonstrated little empathy for such smokers. One recent former smoker described how some of his neighbours who were gradually reducing their cigarette consumption continued to smoke whilst using nicotine patches.

I've got people in the street and they were trying to pack in, they had erm, they were a couple like, they had patches but the ones you cut down gradually, and I thought well, I couldn't see the point of that, you either smoke or you don't and like using the patches as well, they were still smoking. (Patricia, 50, Former)

Additionally, another recent former smoker appeared somewhat confounded considering other RCFE service users who had been engaged in services for a substantial time but continued to smoke.

That's why I went to FagEnds and erm, I'm here to stop, not to cut down. But there's still people going down there, and they're still smoking - they've been going since I've been going, I, I can't get me head round it. (Timothy, 58, Former)

Several participants described encounters with other former smokers who expressed anti-smoking attitudes; participants condemned this characteristic apparent among some former smokers, yet many of the participants continued to express similar, conceited attitudes. One recent former smoker suggested that she wanted to remain non-judgemental regarding others smoking, yet she described how she often felt "superior" as a non-smoker.

Yeah, but I don't want to turn into one of these ex-smokers who goes "Ooo! (makes a noise to suggest condescending) Oh my god!" Do you know what I mean? Don't make me sound like that! ... Yeah, when you smell it on other people, it makes me feel a bit superior really (laughs). (Emily, 59, Former)

One recent former smoker explained how during his previous quit he became strongly opposed to smoking, yet following a relapse, he continued to judge other current smokers negatively. The comment also suggests that the participant may have identified himself separately to other current smokers, having adopted conceited attitudes associated with his previous quit,

To be honest, I've been on both sides ... when I, I did smoke ... I didn't care less what people thought, but when I did pack in smoking, I became the biggest anti-smoker, I could ever think of and even when I started smoking again I still hated the smell of smoke. Even though I smoked, if someone was standing next to me smoking, and it was coming towards me, I'd be giving them dirty looks and I'd be moving away and then I'd start lighting a cigarette up. (Gavin, 45, Former)

Although the majority of comments from recent former smokers were negative towards smoking and current smokers, one participant expressed empathy for current smokers.

I know what it's like to smoke, so I wouldn't preach to anyone, for the simple reason that is, if they want to pack in smoking, they will do. You can only advise someone who wants to take it further, do you know what I mean? (Stuart, 45, Former)

8.3 Attitudes regarding local Stop Smoking Services

"Attitudes regarding local Stop Smoking Services" is the second key major theme considered in this current chapter. The development and implementation of Stop Smoking Services (SSS) were proposed in the white paper, "Smoking Kills" (DH, 1998), as part of a wider initiative aimed at addressing the burden associated with tobacco in the UK. The value of SSS was evident among participants in the current project. Participants described their personal experience of engaging with RCFE, with focus on particularly valued components of the service and notable areas for service development. This major theme consists of four prominent sub-themes, including "Pharmacotherapy", "Recording progress", "Behavioural support", and "Accessibility and awareness".

8.3.1 Pharmacotherapy

"Pharmacotherapy" was the first prominent sub-theme associated with the major theme, "Attitudes regarding local Stop Smoking Services". All participants were advised and typically prescribed pharmacotherapy products via RCFE. Further, participants described how RCFE promoted a wide range of licenced pharmacotherapy products, including nicotine patches, Nicorette inhalators, varenicline (Champix), lozenges (often described as mints), oral spray, nasal spray, and chewing gum. Nicotine patches were the most popular choice of pharmacotherapy for the most recent quit attempt reported by participants (n = 15), although, it should be noted that a small number of participants did not disclose pharmacotherapy choice throughout the interviews (n = 5) and therefore, it cannot be assumed that half of all participants had used or intended on using nicotine patches for their most recent quit attempt. Most participants who reported using nicotine patches, also detailed the use of a fast-delivery form of nicotine replacement therapy (NRT), most often Nicorette oral spray.

Several participants identified barriers to use of certain pharmacotherapy products, which were most commonly identified in relation to nicotine patches and varenicline. With regard to nicotine patches, five participants reported reacting badly to nicotine patches, whereby use resulted in nausea or allergic reactions and participants described how this deterred use, "I can't have the patches because the glue; I'm allergic to it" (Jack, 49, Current). One participant described a skin reaction to nicotine patches that discouraged use and the participant expressed concerns regarding possible medical contraindications associated with using nicotine patches. The participant describes how such concerns could deter smokers from quitting.

I've had a really difficult time this week, cause I don't know if it's bein' on pencillin, 'cause I had a really bad infection and had to take 2 tablets 4 times a day but the patches, where they've been on me skin today, they're all blistering

up, I don't know if it's a reaction to the penicillin or what I don't know, so that's put me off a bit. (Gemma, 57, Current)

Two participants also described barriers to using varenicline. One participant explained how they were unable to use varenicline, as they were already using anti-depressants, demonstrating additional concerns regarding contraindications, "I can't have the tablets (varenicline) because I'm on anti-depressants and I might have a reaction to it..." (Jack, 49, Current). Another participant complained regarding the difficulty they encountered in accessing a prescription for varenicline. Unlike NRT, RCFE advisors were unable to prescribe varenicline, although advisors could issue services users with a letter recommending a prescription of varenicline, which service users were required to present to their GP. This particular comment suggests that some service users may perceive the process of obtaining a prescription for varenicline as inconvenient, which could potentially deter use.

I've just gone through the rigmarole of trying to get onto Champix (varenicline). Now, I got given a letter from FagEnds to take to the doctor and I got told they'd give you a prescription in 48 hours but it turns out what I've got to do first is get an appointment because they won't give out. They said ... you need an appointment with the doctor for the first prescription. (Luke, 54, Current)

Barriers to pharmacotherapy use was closely related to pharmacotherapy perceived efficacy. Participants who cited more barriers to use were less likely to perceive various products as effective and were more likely to be a current smoker. For

example, one current smoker explained, "Erm, I think they should have made stronger patches... they were ok at first, I went on the patches, they didn't seem to work" (Eric, 25, Current). Another current smoker commented, "... sometimes the patches just don't give you the right amount of nicotine that your body usually takes and that makes you go back on to them, or, just things like that" (Shaun, 48, Current). Such comments suggest that perceived barriers and efficacy regarding pharmacotherapy may contribute towards use and potentially, smoking cessation success.

As anticipated, recent former smokers were more likely to perceive the pharmacotherapy products they were using as effective, compared with current smokers. There was no prominent pattern among recent former smokers regarding product choice, although the perceived efficacy of the product was typically based on personal experience, "The patch helps as well, yeah. That's sort of like, that's sort of like, well you're not putting it up to your mouth, so that must help you a lot, mustn't it? The patch?" (Michael, 48, Former). Another participant favoured the lozenges (mints), "The gum worked for her but I couldn't, the gum, the chewing gum. I had the little mints. They seemed to have worked for me" (Timothy, 58, Former).

8.3.2 Recording progress

"Recording progress" was the second prominent sub-theme associated with the major theme, "Attitudes regarding local Stop Smoking Services". Quit duration was consistently recorded and recalled among recent former smokers, despite variations in duration length. The accounts suggest that keeping a record of quit duration was a means of recording progress. One participant described how she recorded quit duration on a daily basis; this method of recording progress enabled her to break down the quit

attempt and reinforced behaviour change, "Well, I've never told anyone this but I've got a calendar and every day I've packed in I cross off, so when you look at it you go 'Wow!'" (Patricia, 50, Former). Another participant also described how they recorded progress by mentally acknowledging each week they had quit, which also appeared to reinforce the quit attempt, "... like four to five weeks now isn't it? So, you kind of go, 'another week!' You know what I mean?" (Michael, 48, Former).

Engagement with RCFE created additional opportunities to record progress, as service users were required to attend regular drop-in sessions. Participants described how they attended RCFE on a weekly or bi-weekly basis. Regular appointments appeared to add further structure to quit attempts, enhancing opportunities to record and consider progress, "With having FagEnds ... they say ... 'There's another week', and then your week turns into a month" (Paul, 38, Former). Another participant similarly explained, "Well going to FagEnds helped me ... you've gone another fortnight without smoking" (Patrick, 56, Former).

As part of engagement with RCFE, service users were required to provide a breath sample to ascertain exhaled CO and following this, the RCFE advisor would provide feedback regarding the reading and record it on the service user's record card. Four recent former smokers described receiving feedback regarding exhaled CO and felt that this method of recording progress was also beneficial. One participant commented, "I like the fact that when you go to FagEnds, you can see the results when you blow into the tube and stuff like that, you can see that nicotine's not in your system" (Jacob, 59, Former). One participant demonstrated how the reading could have a direct impact on mood,

I'll go down there and be like that "Give me a reading!" (laughs). You know, it's funny, I went down there today and Keith (RCFE advisor) said "Do you want your reading?" and I said "It should be nought, 'cause it's been nought for ages." and Keith said "It's one." and I said "Where's the one come from?" and he could see me, I got a little bit of a cob on. He said "Unless you come in with a mask on, it's a brilliant reading, we know you haven't had a ciggy, it's car fumes and things like that", so it was one, normally it's nought ... (Timothy, 58, Former)

These comments suggest that exhaled CO measurement feedback is beneficial for recent former smokers as it provides an objective measure of carbon monoxide, which indicates non-smoker status; however, current smokers failed to comment on this aspect, which might suggest that this method of recording progress is only beneficial to service users who have recent quit.

8.3.3 Behavioural support

"Behavioural support" was the third prominent sub-theme associated with the major theme, "Attitudes regarding local Stop Smoking Services". Nine participants praised RCFE advisors for the behavioural support they had received and explained how the service had been beneficial, whilst no participants described negative experiences as a result of engaging in the service.

A number of participants (n = 4) expressed favourable opinions regarding the advice and support they received from RCFE advisors. One participant described a previous discussion with an RCFE advisor, whereby the advisor provided recommendations regarding gradual NRT reduction and this advice was deemed beneficial,

I said to him, "I must have forgot to put a couple on (nicotine patches)", 'cause I still had them, and he said "Well, that's 'cause you've forgot about it and you've thought you had one on, 'cause you've seen the patch you see" and he said, "Well, try and do it, you know you haven't put one on, you know, so leave one off on purpose and say to yourself, you haven't put one on". So, I got another packet of seven days supply off him and they're still in the cupboard. (Patricia, 50, Former)

Another participant described how he had previously accessed a local chemist, which failed to offer more comprehensive behavioural support. The participant continued to describe how he valued RCFE and the behavioural support they offered, as he suggested that this component of support enhanced his quit attempt,

I don't think I would have done it without FagEnds, without going to that meeting because there was another thing I done, I went to a Lloyds chemist a while back, and it was just handing things out, just giving me these little lozenges. I was just going in having them and still having a ciggy but with FagEnds, there's a meeting, they have a little talk to you, there's more advice. (Timothy, 58, Former)

Advisor feedback was also an important component of behavioural support considered by five participants, as rewarding comments were deemed beneficial in reinforcing the behaviour change. One recent former smoker described,

I think the whole thing with FagEnds has been really good, certainly for someone like me, I'm a bit like a dog getting a reward. On a Friday when I go there, I get me chewing gum and I get me "Well done, there's a pat on the back" and you know, I think doing it on my own would have been more difficult ... (Ronald, 40, Former)

Another participant also described the benefit of receiving positive feedback from advisors whilst attending drop-ins, "It's nice to go back and they say well done ..." (Paul, 38, Former).

Finally, RCFE additionally offers proactive telephone support, which was referred to by two participants; both participants felt that proactive telephone support was beneficial in that telephone advisors helped to reinforce smoking cessation and provided service users with reassurance that support was regularly available.

Yeah and you see like the likes of these phone calls you get, really makes you feel proud that you've achieved something, do you understand what I'm ... I know people might find it a bit patronising but it's nice for someone to say you're doing good and you are doing good if you've packed it in and that. (Jacob, 59, Former)

Another participant explained, "... the help is there and it's there for me, you know, even if they phone up, it helps you through the phone and talking, stuff like that. You know, there will always be somebody there to help you" (John, 53, Former).

8.3.4 Accessibility and awareness

"Accessibility and awareness" was the fourth prominent sub-theme associated with the major theme, "Attitudes regarding local Stop Smoking Services". The accessibility of SSS was considered by several participants (n = 9). Participants commented on how support services had become increasingly prevalent and accessible within Liverpool more recently. One participant described how local support services were unheard of in the 1990s and how his current quit attempt had been aided by the increased availability of support services,

'cause when I first packed in, the patches had just come out and I was buying them meself but I didn't have them, like support or anyone to talk to ... I think it's easier this time around because there's more help and support and there's more people doing it. (Gavin, 45, Former)

Another participant also highlighted the changing landscape of SSS in Liverpool, within the past decade or so, "Yeah, there's loads of changes now, there's loads of help now as well. You wouldn't even ... five years ago, really, well really since FagEnds come on the scene" (Jack, 49, Current).

With regard to accessibility, the frequency and locality of community drop-in sessions appeared to be of particular importance. One participant explained, "... and you've

got that now; they're coming to your area. Do you know what I mean? So there's no need for you to go travelling anyway, they come to you, so I think that's a good thing" (John, 53, Former). Another participant expressed approval regarding the availability of support in Liverpool and described how RCFE drop-in sessions were available within workplaces in addition to community settings, which he was already accessing, "I just think there's loads and loads of support out there for people ... There's lots of help, like I believe people (RCFE advisors) were coming to our work I was told..." (Paul, 38, Former). Such comments indicated that accessibility was an important attribute of SSS; this suggests that if there were fewer accessible community-based services, fewer smokers would be likely to engage with services.

Awareness of services was another component of this sub-theme, which a small number of participants (n = 4) identified in relation to service engagement. Participants highlighted the importance of service publicity in engaging smokers with services. One participant suggested, "I think like, more awareness, like groups, like FagEnds, where other stop smoking things, I think if people knew, and they're a bit like, sort of like determined, they would try and pack in" (Michael, 48, Former). Another participant advised increasing advertising expenditure for support services, to encourage smokers to engage and quit, "I suppose what would help people is, I suppose, with the advertising side of things, like more, FagEnds and stuff" (Andrew, 34, Current). Despite such comments, two participants described how they had previously postponed engagement with RCFE, despite knowing of the service, as they felt that they could initially quit without the additional support. One participant explained how eventual engagement in services propelled her motivation to quit.

... you just keep saying, "Aww I'm giving up, so I'm not doing this", "I'm going to give up after this packet", so no, that went on for a while, so I don't really know what made it, I think the fact that I actually went to FagEnds definitely helped. (Emily, 59, Former)

Another participant described how they had avoided engagement with services and this coincided with repeated quit attempts; the participant continued to emphasise the significance of support in quitting.

... I never went to FagEnds. I'd been on and off for a few years, and just gone for the odd two weeks and not gone back, but you can't do the smoking on your own, you do need to get the help that's available now for you, so use it. (Jack, 49, Current)

8.4 Discussion

The present chapter explored the factors implicated in smoking cessation success, using qualitative methods. Participants' experiences of smoking and smoking cessation were explored in relation to aspects associated with tobacco control policy and practice. In particular, this chapter identified two key themes, including "Reshaping social norms" and "Attitudes regarding local Stop Smoking Services". Participants observed a transformation in smoking-related social norms, in line with the introduction of tobacco control policy and these observed changes in societal norms were often attributed to motivation to quit. Furthermore, participants highly valued RCFE, the SSS. Various components of the service were discussed, whilst some participant proposed opportunities for service improvement. The results of the

current chapter, the potential strengths and limitations, and the implications will now be discussed.

8.4.1 Exploration of chapter findings

The results in relation to the theme, "Reshaping social norms" will be explored firstly. This particular major theme demonstrated the impact that changing smoking-related social norms has on smoking. More specifically, participants expressed a need to belong within society and the results indicated that this need could potentially inhibit or promote smoking cessation success. Feelings of judgement or stigma were also described and attributed to smoking. The sub-themes associated with this major theme included "A sense of belonging" and "Judgement and stigma".

The results provided evidence in support of a relationship between changing social norms and smoking cessation behaviour. The majority of participants described how smoking no longer remained a social norm within their closer social networks and also, society overall. A large number of studies have found that individuals are more likely to start or continue to smoke if smoking is normative within their local social networks (Bindah & Othman, 2011; Hoffman et al., 2006; Leonardi-Bee et al., 2011; Seo & Huang, 2012; Simons-Morton & Farhat, 2010), whilst, research also suggests that smokers are more likely to quit if their significant others are non- or former smokers (Coppotelli & Orleans, 1985; Hanson et al., 1990; McBride et al., 1998).

Contrary to the above finding, a small number of participants described how smoking had remained socially normal within their local social networks, which created some difficulties in attempting to quit smoking. Many participants, who were affiliated with

social groups of smokers, attached a sense of belonging to smoking and therefore, these participants felt socially isolated by quitting smoking; this could potentially impede smoking cessation success.

Judgement and stigma were also important concerns, as many participants described feeling judged and even stigmatised by others in society. This finding is supported by previous research, which suggests that non-smokers do stigmatise smokers. For example, Peretti-Watel, Legleye, Guignard, and Beck (2014) conducted a survey of non-smokers (N = 3091). A cluster analysis revealed four contrasting profiles, one of which represented respondents whereby a strong sense of moral condemnation and social rejection of smokers was demonstrated.

Feelings of judgement and stigma were particularly associated with the introduction of tobacco control policy (e.g. the smoking ban in England). A number of studies have found that the implementation of smoke-free legislation in public places is associated with feelings of stigmatisation among smokers (Betzner et al., 2012; Hargreaves et al., 2010; Ritchie et al., 2010a, 2010b), whilst several studies have identified a relationship between the implementation of smoke-free legislation and increased quit attempts and smoking cessation success within the UK (Lock et al., 2010; D. F. Mackay, Haw, & Pell, 2011; Nagelhout et al., 2012). The current findings suggest that the implementation of smoke-free legislation contributes towards uncomfortable feelings of judgement and stigma, which in turn contributes towards motivation to quit.

Participants not only described concerns regarding being judged and stigmatised by others, but many participants appeared to project similar attitudes towards other, current smokers. Ritchie et al. (2010a) suggested that some smokers attempt to ameliorate smoking-related stigma by participating in the stigmatisation of other smokers; the current research supports this notion, as participants, albeit typically recent former smokers, often referred to other smokers in a conceited manner. Furthermore, Vangeli and West (2012) additionally described how some recent former smokers form negative, albeit unintentional, judgements towards current smokers. The current findings further support for this notion.

The results in relation to the theme, "Attitudes regarding local Stop Smoking Services" will now be explored. This major theme explored the components of SSS, which were deemed particularly valuable to participants, whilst some recommendations were proposed regarding service improvements. The sub-themes associated with this major theme included "Pharmacotherapy", "Recording progress", "Behavioural support", and "Accessibility and awareness".

The current study findings suggest that all participants favoured the use of SSS and deemed engagement as beneficial in aiding a smoking cessation attempt. Previous research suggests that SSS have been proven to provide effective stop smoking support to smokers intending on quitting (Ferguson et al., 2005; Godfrey et al., 2005; Judge et al., 2005; West et al., 2013), which supports the current findings.

With regards to pharmacotherapy, nicotine patches were used most often to aid quit attempts among participants, whilst a combination of fast and slow release NRT was frequently favoured. A recent Cochrane review (Stead et al., 2012) found that combination NRT was substantially more effective compared to use of a single NRT

product and the current results suggest that this approach was promoted by RCFE advisors.

Perceived barriers to pharmacotherapy use were also considered. Few studies have explored perceived barriers to NRT use, although one small qualitative study (Silla, Beard, & Shahab, 2014) interviewed 15 smokers and identified key concerns regarding NRT use, including addiction, long-term use, efficacy, health consequences, and delays with quitting process. The present results identified a number of barriers to use in relation to nicotine patches (including skin reactions and nausea) and varenicline (including accessibility and contraindication concerns); such barriers were found to deter some smokers from using various smoking cessation aids.

Participants also noted the opportunities that SSS provided them with regards to recording smoking cessation progress, such as the provision of feedback pertaining to exhaled CO. A recent Cochrane review (Bize et al., 2012) suggested that there was no evidence to confirm that feedback in relation to exhaled CO enhanced smoking cessation; however, the review pooled only two studies in the analysis regarding providing exhaled CO, the studies pooled reported delivering feedback pertaining to exhaled CO measurement on only one occasion (SSS typically relay exhaled CO weekly or bi-weekly), and the authors additionally highlighted the scarcity of high quality studies. A previous, smaller qualitative study (Grant et al., 2014) found that almost all participants who received exhaled CO feedback found the information motivational. The current findings build upon this research and highlight how, for recent former smokers at least, the provision of exhaled CO feedback is deemed to be a method of reinforcing behaviour change.

Participants listed several potential aspects of behavioural support, which were considered beneficial in promoting motivation to quit or sustain abstinence. One particular aspect of note was the delivery of proactive telephone counselling. A recent Cochrane review supported the implementation of proactive telephone counselling, rather than reactive telephone counselling (L. F. Stead et al., 2013); however, the review also noted a paucity of evidence with regard to the efficacy of proactive telephone counselling. The current findings support the implementation of proactive telephone counselling in SSS, as a small number of participants described how receiving telephone calls was reassuring and reinforcing; further research is needed.

Accessibility and awareness were also identified as important aspects of SSS delivery, as some participants suggested that increased knowledge and opportunities regarding accessing services would enhance engagement and potentially smoking cessation success. One recent review (R. L. Murray, Bauld, Hackshaw, & McNeill, 2009), explored methods of improving access to SSS among disadvantaged groups, such as proactive case finding; however, the evidence on effective strategies to increase access was found to be limited. The current findings suggest that increasing the breadth of SSS drop-in session locations would be preferable but further research is needed.

8.4.2 Potential strengths and weaknesses

In parallel to the previous chapter, the semi-structured design of the qualitative interview schedule allowed for pre-determined questions, as well as providing the opportunity for the interviewer to explore varying areas of interest. This was particularly apparent in the current chapter, as participants described important aspects

related to tobacco control policy and practice, which were deemed to be influential in promoting motivation to quit or smoking cessation success, such as the impact of the smoking ban in England or valued attributes of SSS. There are, however, some potential limitations of note, in relation to the current chapter results.

Potential participation selection biases should be considered in relation to the current chapter results (e.g. Loon et al., 2003). Participants who favoured RCFE may have been more willing to participate in the qualitative investigation, compared to those who were dissatisfied with RCFE; this may have resulted in some biased perspectives and should be considered when interpreting the results. Nevertheless, the current chapter results present a number of implications relevant to SSS development.

Barriers regarding the use of various pharmacotherapy products were identified and attributed to current smokers, more often than recent former smokers. Similarly to the previous chapter, this chapter demonstrates how qualitative research can be used to identify potential associations, although the design prevents the inference of causal relationships. For example, a qualitative design incorporating a number of waves could have enabled a more extensive exploration of the relationships between barriers to pharmacotherapy use and smoking cessation success; however, the exploration of both current and recent former smokers' experiences adopted in the current project provides in-depth insight into the experiences of service users overall.

8.4.3 Implications for tobacco control knowledge, practice and policy

The current chapter presented several key implications for tobacco control knowledge, practice and policy. The qualitative analysis resulted in the development of the

prominent theme, "Reshaping social norms", which generated various sub-themes and concepts of interest. Potential areas for future development include exploration of topics, including social isolation, stigma, and former smokers' judgements.

In relation to social isolation, a small number of participants associated with a social group of smokers described feeling isolated and alone throughout quitting, which was often deemed to be challenging. The finding highlighted the need for future research among such individuals who feel socially isolated, which in turn could inform the development of further interventions to support such individuals.

The current findings also indicate an association between smoke-free legislation and smoking cessation, with stigma seeming to mediate this relationship. This potentially provides support for the implementation of smoke-free legislation; however, the findings regarding stigma also fit well with the previous chapter findings, which demonstrated how some tobacco control strategies (e.g. increasing cigarette costs) may inadvertently impact aspects of smoker QOL. Future tobacco control policies could potentially marginalise smokers further and therefore, means of avoiding such effects should be considered prior to policy dissemination.

A final implication of interest in relation to reshaping social norms relates to the finding that many participants described arguably negative attitudes regarding other current smokers. It is presently unclear whether adopting such judgements towards other smokers promotes smoking cessation success, however, further research would certainly be of interest and might contribute towards understandings of behaviours

associated with sustained abstinence. Potentially, the adoption of such attitudes could reinforce smoking cessation success.

The qualitative analysis also resulted in the development of the prominent theme, "Attitudes regarding local Stop Smoking Services", which generated various subthemes and highlighted several aspects for research and practice development. Some of the key areas for future development, include exploration of barriers to varenicline, the benefits of providing exhaled CO feedback, and the efficacy of proactive telephone counselling.

In relation to the barriers to using varenicline, the current results highlighted several reasons as to why some participants refrained from using varenicline. Whilst barriers proposed may certainly have been valid among some participants, the findings highlight the need for further research. To the researcher's knowledge, previous research has failed to explore this concept. Potentially, misconceptions and misinterpretations regarding varenicline may deter some smokers from using the product; further research could contribute towards improved knowledge regarding barriers to use of varying pharmacotherapy products and subsequently, work towards addressing any concerns that may be prevalent among smokers.

As mentioned, many participants described the benefit of receiving exhaled CO feedback during SSS drop-in sessions. There is a currently limited evidence regarding the efficacy of providing exhaled CO feedback as a tool for promoting smoking cessation, however, this is primarily due to the lack of relevant and quality research. A well-designed randomised controlled trial could examine the effect of receiving

regular exhaled CO feedback on smoking cessation success and would enable researchers to better understand the extent to which this approach might be effective.

The last key implication in relation to SSS delivery regards the efficacy of delivering proactive telephone counselling. Whilst the current study demonstrated a number of participants' accounts in favour of proactive telephone counselling, there is presently limited evidence to support its efficacy due to a lack of quality research. The implementation of a qualitative study focusing on telephone support specifically may provide further guidance on the implementation of telephone support services, whilst avoiding the limitations of implementing an RCT (see L. F. Stead et al., 2013).

8.4.4 Conclusion

In summary, the present chapter discussed the results of part of the qualitative investigation, which aimed to examine factors implicated in smoking cessation success. The current chapter discussed several concepts relevant to tobacco control policy and practice, which participants deemed helpful and sometimes unhelpful in promoting smoking cessation. In particular, the relationship between smoking-related social norms and smoking behaviour was explored and the results indicated that a sense of belonging and experiences of stigma were potential inhibitors of smoking cessation success. Furthermore, participants discussed aspects of SSS, which they deemed to be beneficial and opinions regarding areas for service development were offered.

Again, this chapter demonstrates the benefits of conducting semi-structured qualitative research, as aspects relating to tobacco control practice and policy were not originally

intended as part of the interview schedule, yet the majority of participants discussed issues regarding social norms and support services, thus demonstrating the importance of these issues to participants. Several opportunities for future research and development were discussed, such as exploring strategies for supporting smokers who feel isolated as a result of quitting and considering potential barriers and facilitating factors associated with use of varying pharmacotherapy products. The next and final chapter will consider the PhD project results as a whole by reflecting upon the thesis objectives, considering the overall research contribution of the PhD thesis, and finally exploring future directions.

Chapter 9: Discussion

9.1 Introduction

This PhD thesis describes a mixed methods project, combining quantitative and qualitative research methodologies, which primarily aimed to investigate whether application of the Liverpool Lung Project (LLP) intervention (based on the LLP risk model) was associated with follow-up smoking cessation success and lung cancer risk perceptions. Factors implicated in smoking-related risk perception and smoking cessation success were additionally explored. The project findings will: contribute towards improved knowledge regarding smoking behaviour and risk perception; identify potential improvements regarding the delivery of Stop Smoking Services (SSS); and, inform the development of future smoking-related risk communications. All of the aforementioned implications could contribute towards improved smoking cessation rates and subsequently, reduced smoking-related disease and deaths.

The present chapter is organised into several sections. Firstly, the key research findings are presented for each of the thesis objectives. Secondly, the project research contribution overall in relation to tobacco control research, practice and policy is explored. Thirdly, potential future directions for research are considered. Lastly, conclusions are drawn regarding the overall project.

9.2 Key findings

Three project objectives were determined, which were achieved using a combination of quantitative and qualitative research methods. This section will denote each project objective and describe the corresponding key findings pertaining to each of the given objectives.

9.2.1 Objective one

The first objective investigated whether application of the LLP intervention (based on the LLP risk model) was associated with follow-up smoking cessation success and lung cancer risk perceptions. Statistical analyses were undertaken to evaluate the effect of the LLP intervention on six-month follow-up smoking status and several lung cancer risk perceptions.

Baseline current smokers and recent former smokers were recruited as two separate samples. The results suggested that application of the LLP intervention was not associated with follow-up smoking status among baseline current smokers but was significantly associated with follow-up smoking status among baseline former smokers only. Baseline former smokers who received the intervention were almost twice as likely to be classified as a former smoker at follow-up compared to those in the control group (OR 1.91, 95% CI 1.03-3.55).

Statistical analyses were undertaken to assess the effect of the LLP intervention on lung cancer risk perceptions at six months. The results of the analysis suggested that application of the LLP intervention was associated with follow-up perceived personal lung cancer risk, perceived average smoker lung cancer risk, perceived relative risk of lung cancer, and lung cancer worry, among baseline current smokers. The analyses of baseline recent former smokers also failed to find an association with the aforementioned four lung cancer risk perceptions at six months.

These results were of interest as the LLP intervention was associated with follow-up smoking status among baseline recent former smokers, but not baseline current smokers, and additionally, the LLP intervention was not associated with any of the four follow-up lung cancer risk perceptions across both of the datasets. Potentially, this suggests that other psychosocial mechanisms may have been responsible for the LLP intervention effect on follow-up smoking status, independent to lung cancer risk perceptions.

To the researcher's knowledge, this is the first time such an analysis has been undertaken and it is of particular note that a relationship was established between the LLP intervention and follow-up smoking status among baseline former smokers; however, it is necessary to be cognisant of the fact that this project was not fully statistically powered as a result of sample size limitations, primarily due to recruitment being undertaken solely by the PhD researcher.

9.2.2 Objective two

The second objective entailed exploring factors implicated in smoking-related risk perceptions. This objective was addressed using both quantitative and qualitative research methods. Figure 13 summarises the key factors implicated in personal perceived smoking-related risk, as identified in the current project. The results of the quantitative investigation identified age, baseline smoking status, perceived average smoker lung cancer risk, and the perceived relative risk of lung cancer level "Ten times higher risk", as predictors of baseline perceived personal lung cancer risk. Cross tabulation results suggested that objective risk perceptions were closely related to

personal risk perceptions, whilst older participants, classified as current smokers were more likely to perceive their risk as higher.

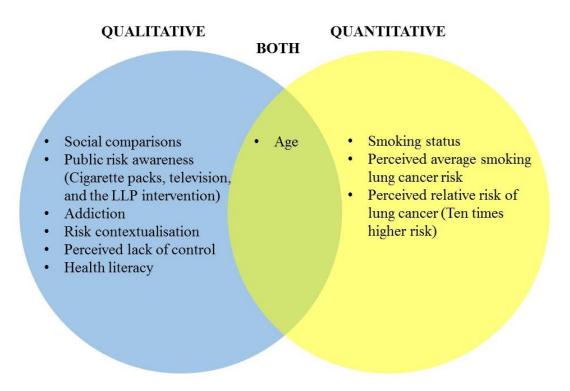


Figure 13. Key identified factors implicated in smoking-related risk perceptions

The qualitative analysis further explored factors implicated in smoking-related risk perceptions. As discussed, the quantitative results identified age as a predictor of baseline perceived personal lung cancer; the qualitative results provided further support for this finding, as many individuals who participated in the qualitative research component described how their personal perception of smoking-related risk had increased with age. Several other key factors implicated in smoking-related risk perception were identified in relation to the qualitative research component; the qualitative results greatly emphasised the importance of an individual's personal experiences on personal perceptions of risk. Important factors, which appeared to be implicated in smoking-related risk perception, included having significant others who

had experienced smoking-related disease, public risk awareness campaigns and interventions, addiction, risk contextualisation, perceived lack of control, and health literacy.

9.2.3 Objective three

The third objective entailed exploring factors implicated in smoking cessation success. This objective was also addressed using a combination of quantitative and qualitative research methods. The quantitative research component utilised three separate datasets to achieve this objective: (1) a sample of baseline current smokers, to explore predictors of follow-up smoking status among those who had not yet quit; (2) a sample of baseline recent former smokers, to explore predictors of follow-up smoking status among those who had already quit; (3) a combined sample of both baseline current and recent former smokers, to explore cross-sectional associations with smoking status at baseline. Figure 14 summarises the identified predictors of smoking status across the three datasets.

The predictors of baseline current smokers were limited (potentially, due to the low number of participants classified as recent former smokers at follow-up within this dataset); the perceived relative risk of lung cancer level, "Ten times higher risk" was the only significant result following model refinement. A greater number of baseline participant characteristics were found to predict follow-up smoking status with regards to the dataset consisting of baseline recent former smokers; significant variables included LLP intervention (as discussed), age, marital status, quit duration, the perceived lung cancer survival level, "About three quarters to nearly all". Furthermore, the cross-sectional analysis of baseline smoking status, which combined all current

smokers and recent former smokers recruited during the project, identified several predictors, including age, living with another smoker, perceived personal lung cancer risk, the perceived relative risk of lung cancer level, "Ten times higher risk", and the lung cancer worry level, "Often or all the time".



Figure 14. Predictors of smoking status at baseline and follow-up, across the three quantitative datasets

Some of the aforementioned quantitative results were corroborated by the results of the qualitative analysis. For example, the quantitative results displayed how age was positively correlated with smoking cessation success (among some of the datasets), whilst the qualitative results described how participants had little desire to quit smoking throughout their youth and that with age, they had become more determined to quit smoking. The qualitative analysis enabled the identification of a wide range of variables, which appeared to be implicated in smoking cessation success. Some of the relevant psychosocial factors included motivation, self-efficacy and self-doubt, self-regulatory behaviour (e.g. self-talk), smoker identity, and perceived benefits of quitting smoking, whilst factors associated with tobacco control practice and policy implicated in smoking cessation success, included social norms, stigma, and various components of local SSS.

9.3 Research contribution

The overall findings of the current project contribute towards three key areas of tobacco control, including knowledge, practice and policy. The results pertaining to each of these three aspects will now be discussed.

9.3.1 Contribution to tobacco control knowledge

The PhD project findings contribute towards tobacco control knowledge in numerous ways; key contributions will now be explored. Previously, lung cancer risk prediction models have been considered or utilised to stratify high-risk patients for CT screening (Maisonneuve et al., 2011; McRonald et al., 2014; Raji et al., 2012) and adapted to deliver web-based self-assessment tools (Y. Chen et al., 2014). This project is innovative, as to the researcher's knowledge, it is the first project, to explore the adaptation, delivery, and evaluation of a lung cancer risk prediction model in the context of smoking cessation. Application of the LLP intervention was found to be associated with follow-up smoking status among baseline former smokers but not

among baseline current smokers, which provides insight into the mechanisms by which the LLP intervention was effective; the findings provide support for the behavioural model, the Transtheoretical Model of Change (TTM) (J. O. Prochaska & DiClemente, 1983), which stipulates that behavioural processes of change differentiate by stage of change. Furthermore, the LLP intervention was found to have no significant effect on lung cancer risk perceptions at follow-up; this finding corroborates with the notion that the significant relationship between the LLP intervention and follow-up smoking status among baseline former smokers, might have been due to the LLP intervention being viewed as a reinforcing message.

The qualitative findings in relation to the LLP intervention effect also contribute towards risk communication literature more widely. Gigerenzer et al. (2007) described how the vast proportion of society have difficulty in comprehending numbers and statistics, which can have implications for effective risk communication delivery. Research has also explored the effect of communicating risk in absolute or relative terms; findings suggest relative risk has been found to be perceived as larger and more persuasive (Akl et al., 2011). The present project contributes towards further knowledge, as the results demonstrated that five-year lung cancer risk was perceived as low, even among those who were deemed particularly high-risk. Furthermore, the results suggested that many participants failed to understand the concept of five-year risk, with some participants often suggesting that the figures pertained to life-time risk. This highlights the benefit of producing multiple risk scores that would provide a range of risks with continued smoking and smoking cessation; a more rounded figure could potentially be better communicated to patients and the precision of the LLP risk model alone could potentially be misinterpreted, as evidence by the qualitative results.

Another potential research opportunity might be to develop a lung cancer risk model, which specifically aims to communicate lung cancer risk attributed to smoking; current lung cancer risk models are typically developed with the aim of identifying high-risk individuals (A. N. Freedman et al., 2005), often to stratify individuals for screening. A model that integrates various smoking-related predictors of lung cancer would certainly be of value and would be far more applicable for application in this context compared risk models already available.

Previous research has suggested that perceived personal lung cancer risk differentiates based on smoking status (Rutten et al., 2011), a finding which was corroborated in the current project. Despite this, there are only a handful of studies in support of a relationship between risk perception and smoking cessation success, at varying long-term follow-up periods (Borrelli et al., 2010; Hayes & Borrelli, 2013; Jacobson et al., 2014). The current project addresses this paucity of research, in that the results demonstrate a relationship between some aspects of lung cancer risk perceptions and follow-up smoking status.

Baseline current smokers who perceived a smoker to exhibit "Ten times higher risk" of developing lung cancer than a non-smoker, were more likely to be classified as recent former smokers at follow-up, compared to those who perceived a smoker to exhibit "About the same to a little higher" lung cancer risk as a non-smoker; however, other lung cancer risk perceptions failed to predict follow-up smoking cessation among baseline current smokers. This finding provides some support for the relationship between baseline lung cancer risk perceptions and follow-up smoking status.

Another key finding, which contributes towards the literature, was the documented association between age, risk perception, and smoking cessation. Previous research has suggested that older smokers are more likely to achieve smoking cessation at follow-up compared to younger smokers (Fidler et al., 2013; Hymowitz et al., 1997; C. Lee & Kahende, 2007; Monsó et al., 2001). In the current project, age was not found to predict follow-up smoking cessation among baseline current smokers, but it did predict follow-up smoking cessation among baseline former smokers, which suggests that older smokers may be more capable of maintaining abstinence in comparison to their younger counterparts. The qualitative results also contributed towards improved knowledge of the relationship between age, risk perception, and smoking cessation. The qualitative results suggested that, with age, participants appeared to become increasingly concerned regarding the development of disease and mortality; older participants would often perceive smoking cessation as an opportunity to prolong life. These findings demonstrate how the qualitative results shed light on the quantitative relationship between age, risk perception and smoking cessation.

It has been suggested that medically unwell current smokers often fail to internalise or personalise the effects of smoking (Bock et al., 2001). The results of the current project highlight the multifaceted manner of addiction (Ross & Kincaid, 2010), as many current smokers in the present project were fully aware of the risks of smoking and attributed smoking to their own poor health, yet they continued to smoke; the present project explores participants' responses to this experience and highlights the challenges that service users experience in overcoming smoking-related impulses, as stipulated in the health behaviour theory, PRIME theory (see West & Brown, 2013).

A recent review (Goldenberg et al., 2014) described a negative relationship between smoking and quality of life (QOL). The current findings contribute towards improved understandings of the mechanisms by which this relationship occurs. For example, in addition to the documented impact of smoking on health, the introduction of smoke-free legislation, such as the smoking ban in public places, led some smokers to feel marginalised. Furthermore, participants described how increased cigarette prices often failed to deter smoking and, alternatively, increased deprivation. These findings also demonstrate how smoking cessation was subsequently associated with a sense of liberation.

Previous research has also found that self-efficacy has been found to predict smoking cessation (Hendricks et al., 2010; Schnoll et al., 2011). Regarding self-efficacy, the current project was able to contribute towards knowledge regarding behaviour change techniques associated with increased self-efficacy. Self-talk (i.e. inner speech) may assist cognitive and self-regulatory functions (Diaz & Berk, 1992; D. G. MacKay, 1992) and a small number of studies have implicated the effect of self-talk in regulating smoking behaviour (Kelly et al., 2010; Merchant et al., 2013; Naughton et al., 2013). Despite this, previous research has failed to explore the application of self-talk in the context of SSS. The current findings suggest that positive self-talk can combat temptation to smoke, and ultimately, improve self-efficacy and smoking cessation success.

The present findings also increased research knowledge with regards to the association between smoke-free legislation and smoking cessation. Previous research has found

that smoke-free legislation has been found to increase quit attempts and smoking cessation success within the UK (Lock et al., 2010; D. F. Mackay et al., 2011; Nagelhout et al., 2012), whilst smoke-free legislation in public places has been associated with feelings of stigmatisation among smokers (Betzner et al., 2012; Hargreaves et al., 2010; Ritchie et al., 2010a, 2010b). The current findings contribute towards the literature, as the qualitative results corroborate with previous findings regarding the association between stigma and smoke-free legislation but further to this, participants were found to directly attribute stigma and the de-normalisation of smoking, to their own motivation to quit; thus, the current findings provides further understanding and support regarding the relationship between smoke-free legislation and smoking cessation.

Smokers have also been found to be more likely to quit if their significant others are non- or former smokers (Coppotelli & Orleans, 1985; Hanson et al., 1990; McBride et al., 1998). The present results provided further support and understanding regarding this relationship. Not only was living with another smoker a significant predictor of baseline smoking status as part of the quantitative research component, but smoking-related social norms were found to be implicated in smoking cessation success as part of the qualitative research component. More specifically, the results of the qualitative interviews demonstrated how social smoking can be associated with a sense of belonging and therefore, smoking cessation can lead to feelings of social isolation, which could impede smoking cessation attempts.

A number of key research contributions were identified with regards to the delivery of SSS. Previous research has been limited in relation to exploring barriers to use of

pharmacotherapy products. One previous study explored barriers to nicotine replacement therapy use among a small number of smokers (Silla et al., 2014); however, the present study contributed to understandings of pharmacotherapy choice, as a number of barriers to the use of nicotine patches, as well as varenicline were identified. Furthermore, previous research argues that there is limited support to suggest that the delivery of feedback pertaining to exhaled CO is effective in enhancing smoking cessation success (Bize et al., 2012). The current project, however, suggests that SSS users perceive regular exhaled CO feedback as beneficial in promoting their smoking cessation attempt, among former smokers, at least.

9.3.2 Contribution to tobacco control practice

Much of the current findings also contribute towards tobacco control practice, with particular emphasis on the delivery of SSS. Potential implications for tobacco control practice will now be explored. Quit rates have remained fairly consistent in previous years within SSS (NCSCT, 2014), and therefore, an innovative and low-cost intervention to promote smoking cessation success would certainly be welcomed. As described, the present findings suggest that the LLP intervention may be appropriate for delivery within SSS, as the delivery of the LLP intervention would be timely and easily communicated by non-clinicians, with basic training. More specifically, recent former smokers could benefit from receiving the LLP intervention, as it may be viewed as a means of reinforcing smoking cessation success. Other possible settings for the delivery of the LLP intervention, include GP surgeries, hospital settings, and electronic health resources available to the public.

As previously described, perceived relative risk of lung cancer was found to predict follow-up smoking status among baseline current smokers; however, most participants were found to underestimate perceived relative risk of lung cancer, whilst the majority of participants overestimated lung cancer survival and lung cancer worry was typically low. Differences in risk perception across smoking status might suggest that better education is required as former smokers might underestimate their personal perceived lung cancer risk, however, further research is required to fully understand the relationship between smoking status and risk perception. Inclusion of figures pertaining to relative risk could be an effective means of communicating lung cancer risk in future health campaigns; previous research suggests that relative risk is often viewed as higher compared with absolute risk (Akl et al., 2011) and the present findings suggest that perceived relative risk of lung cancer is implicated in follow-up smoking status.

The findings pertaining to the association between age and smoking cessation also have important implications for tobacco control practice. For example, participants described how milestone birthdays were often used as target quit dates or knowledge of such dates promoted motivation to quit; therefore, age targeted campaigns may prove particularly effective in promoting smoking cessation success. This is particularly feasible in relation to promoting re-engagement with SSS, as re-invitation letters could be issued at specific times of the year relevant to service users who have disengaged with the service, thus potentially prompting re-engagement.

Self-talk was consistently employed by participants in an effort to avoid temptation to smoke and to promote smoking cessation success. Previous research suggests that selftalk may be beneficial in regulating smoking behaviour (Kelly et al., 2010; Merchant et al., 2013; Naughton et al., 2013). Currently, NCSCT (2014) guidance pertaining to behaviour change techniques fails to acknowledge the role self-talk in self-regulation associated with smoking. The current findings, alongside the aforementioned studies, demonstrate the potential benefit of delivering cognitive and self-regulatory function training within SSS, as implementation could further promote smoking cessation success.

The reported sense of social isolation among recent former smokers affiliated with social networks of smokers, also has implications for tobacco control practice. Recent former smokers in such situations may benefit from more intensive SSS support, which might include training in coping mechanisms specific to situations whereby service users may be particularly vulnerable to feelings of isolation. Furthermore, the current results also suggest that delivering exhaled CO feedback in SSS is beneficial in promoting smoking cessation success among recent former smokers, as it presumably reinforces the behaviour change; the current findings support the continued implementation of feedback in SSS and other primary or secondary care services, in which it may be applicable.

9.3.3 Contribution to tobacco control policy

The current project findings were most relevant to tobacco control knowledge and practice, although there were some key implications in relation to tobacco control policy additionally. For example, issues regarding the impact of smoke-free legislation on smoking cessation success and the delivery of smoking-related public health campaigns were noted.

The qualitative results demonstrated a relationship between smoke-free legislation and smoking cessation, which supports the implementation of tobacco control policies that intend to make smoking less desirable, acceptable and accessible (DH, 2011); however, the present findings also demonstrate how many smokers appeared marginalised in society as a consequence of the smoke-free legislation. Several smokers described how they were excluded from some social and leisure opportunities, whilst those from poorer backgrounds suffered greater deprivation as a consequence of increased cigarette prices. Future efforts should remain sensitive to the effect of tobacco control legislation on current smokers, whilst delivering effective tobacco control legislation.

Previous research also suggests that fear appeals (e.g. images of diseased lungs) influence smoking cessation intention and success (Gallopel-Morvan et al., 2011; Hammond et al., 2004). Fear appeals are often implemented as part of tobacco control campaigns and graphic images depicting smoking-related disease are frequently featured as part of such campaigns. The current results corroborate previous findings regarding the effect of fear appeals on smoking cessation, as participants in the current project commonly referred and responded to such fear appeals, whilst some participants attributed fear appeals to smoking cessation.

9.4. Future research directions

Future research directions were discussed in greater detail throughout the discussion sections of the previous results chapters; however, this current section considers the key directions for future research in consideration of the PhD project overall.

One of the essential research directions would be to either extend recruitment for the current project or to devise a large-scale trial to test the efficacy of the LLP intervention further, due to the current project entailing insufficient statistical power to conclude the findings; a sample size of at least 673 current smokers would be required to achieve appropriate statistical power (see Section 3.5.1). Further research should also attempt to address the current project limitations, which were discussed in greater detail following each of the results chapters. In particular, consistent collection of exhaled CO measurements at follow-up may have been a beneficial attribute to the project design (West et al., 2005), whilst the inclusion of a measure of self-reported prolonged abstinence may have also increased the validity of the results (Hughes et al., 2010). Future research directions might also consider the application of the LLP intervention among other populations, such as current smokers from primary and secondary care settings, as well as non-help-seeking populations. The LLP intervention effect upon individuals who are not preparing to change behaviour would certainly be of interest.

Some issues were identified in undertaking the qualitative and quantitative methods concurrently. Although efforts were made to purposively sample patients for the qualitative study, across treatment groups, this was not always possible. As some participants described the qualitative work as "therapeutic", participation in the qualitative research may in itself have had an extraneous effect on the quantitative investigation. If a similar design is to be implemented, it would be beneficial to conduct the qualitative and quantitative methods sequentially. For example, the qualitative research could be conducted firstly to establish barriers and facilitators to

communicating lung cancer risk, which could inform the design of the quantitative study. This, however, was not possible in the current project, due to time and financial constraints.

The results of the qualitative analysis demonstrated how the majority of participants felt that the lung cancer risk projections they received as part of the LLP intervention were perceived as being particularly low. Although participant reactions to the LLP intervention might illustrate maladaptive behaviour in response to fear appeals (Witte & Allen, 2000), it is likely that these reactions were due to the delivery of risk information pertaining to five-year risk, as the findings suggest that some participants failed to understand the concept of five-year risk. It would therefore be of interest to develop and evaluate the effect on smoking cessation success, of an intervention based on an alternate lung cancer risk prediction model that calculates lifetime risk. Furthermore, the development of a risk model, which considers and incorporates several smoking-related diseases, would also be of value. Potentially, integration of such factors would generate increased risk projections, making the intervention more salient and in turn, increasing smoking cessation rates.

The current project also demonstrates how risk perceptions fail to fully explain behaviour change, as the predictive value of risk perceptions in the quantitative research component was inconsistent and the qualitative research component revealed how many medically unwell smokers described being fully aware of the implications of smoking, yet they continued to smoke; these experiences fit well with new addiction theories, such as PRIME theory (West & Brown, 2013). Simultaneously, researchers suggest that not one particular theory can explain addiction (Ross & Kincaid, 2010).

Future research should endeavour to consider studying the experiences of medically unwell smokers specifically, to improve understandings regarding addictive behaviours.

The current project not only highlighted the impact of the LLP intervention on smoking behaviour but the qualitative results provided further evidence for the implementation of several other tobacco control strategies. Health and financial implications of smoking were widely discussed throughout the qualitative interviews. Although it is hugely important to develop and explore new and innovative interventions designed to promote smoking cessation, the impact established tobacco control approaches, such as increasing tobacco costs, continues to be exhibited. The current project highlights the importance of adopting a holistic approach to tobacco control research, practice, and policy in future.

9.5 Final conclusion

This was the first project, to the researcher's knowledge, to explore the utility of a lung cancer risk prediction model in the context of smoking cessation. A mixed methods approach was adopted to primarily explore whether application of the LLP intervention was associated with follow-up smoking status and lung cancer risk perceptions. The LLP intervention was developed using the LLP risk model and involved calculation and communication of projected lung cancer risk, based on both smoking and non-smoking behaviour. Factors implicated in smoking cessation success and smoking-related risk perceptions were also explored as part of the PhD thesis.

The key finding of the current project was that application of the LLP intervention was associated with follow-up smoking status among baseline former smoker but not

among baseline current smokers. Furthermore, the LLP intervention had no significant effect on any of the follow-up lung cancer risk perceptions. Aspects of the health behaviour model, the TTM (J. O. Prochaska & DiClemente, 1983) were found to provide some explanation for the exhibited difference in effect between current and recent former smokers and the absence of an effect on follow-up risk perceptions, as the model suggests that different processes of change (such as, reinforcement management) are associated with different stages of behaviour change (such as, the maintenance stage). It should be noted, however, that there were few former smokers at follow-up in relation to the analysis of baseline current smokers; further research involving a larger sample size could help to identify further significant results.

As previously described, a recruitment extension or implementation of a large trial would now be required to confirm the current results, as the present project resulted in a limited sample size. Further research would also benefit from inclusion of a measure exploring prolonged abstinence and biochemical verification of self-reported smoking status. If further research can validate the results of the current project, the LLP intervention could be feasibly delivered within SSS, particularly as it would incur little cost or time to deliver. Ultimately, this could result in improvements in smoking cessation rates or sustained abstinence, which would subsequently reduce smoking-related disease and deaths.

A review of the literature suggested that perceived personal lung cancer risk might have been predicted by several baseline participant characteristics; in particular, age and smoking status were found to be strong predictors. The qualitative research component complemented the quantitative results, providing further support for an association between age and risk perception, in addition to identifying several other factors implicated in smoking-related risk perception. These findings provide detail regarding the role that risk perception plays in smoking behaviour and demonstrate the complexity of risk perception. A better understanding of risk perception will ultimately inform the development of risk communication tools.

An extensive range of factors implicated in smoking cessation success were also derived from both quantitative and qualitative research components, including age, marital status, living with another smoker, baseline quit duration, lung cancer risk perceptions, motivation, self-efficacy and self-doubt, self-regulation, smoker identity, perceived benefits of quitting, social norms, stigma, and various aspects of SSS. The wide variation in factors identified demonstrates how smoking is a highly complex addiction, for which there is little consensus on the extent to which any single contributing factor can effectively explain addictive behaviours (Ross & Kincaid, 2010). Future research should continue to endeavour to understand addiction, as new theories and addiction models continue to emerge (Westermeyer, 2013); doing so, will better inform tobacco control knowledge, practice and policy, which will ultimately, enhance smoking cessation rates.

References

- Abdullah, A. S., Ho, L. M., Kwan, Y. H., Cheung, W. L., McGhee, S. M., & Chan, W. H. (2006). Promoting smoking cessation among the elderly: What are the predictors of intention to quit and successful quitting? *J Aging Health*, *18*(4), 552-564. doi: 10.1177/0898264305281104
- Action on Smoking and Health. (2006). Factsheet No. 16: The economics of tobacco. Retrieved from http://ash.org.uk/files/documents/ASH_temp_0a1e.pdf
- Action on Smoking and Health. (2014a). Smoking Statistics. Retrieved from http://www.ash.org.uk/files/documents/ASH 93.pdf
- Action on Smoking and Health. (2014b). Smoking statistics: Who smokes and how much. Retrieved from http://ash.org.uk/files/documents/ASH_106.pdf
- Action on Smoking and Health (ASH). (2015). Smoking statistics: Who smokes and how much. Retrieved from http://ash.org.uk/files/documents/ASH_106.pdf
- Ahmed, H., Naik, G., Willoughby, H., & Edwards, A. G. (2012). Communicating risk. *BMJ*, *344*, e3996. doi: 10.1136/bmj.e3996
- Ajdacic-Gross, V., Landolt, K., Angst, J., Gamma, A., Merikangas, K. R., Gutzwiller, F., & Rossler, W. (2009). Adult versus adolescent onset of smoking: How are mood disorders and other risk factors involved? *Addiction*, *104*(8), 1411-1419. doi: 10.1111/j.1360-0443.2009.02640.x
- Akl, E. A., Oxman, A. D., Herrin, J., Vist, G. E., Terrenato, I., Sperati, F., . . . Schunemann, H. (2011). Using alternative statistical formats for presenting risks and risk reductions. *Cochrane Database Syst Rev*(3), Cd006776. doi: 10.1002/14651858.CD006776.pub2
- Al Koudsi, N., & Tyndale, R. F. (2005). Genetic influences on smoking: A brief review. *Ther Drug Monit*, 27(6), 704-709. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16404798
- Alberg, A. J., & Samet, J. M. (2003). Epidemiology of lung cancer. *Chest*, *123*(S1), 21S-49S. doi: 10.1378/chest.123.1_suppl.21S
- Ali, N., Lifford, K. J., Carter, B., McRonald, F., Yadegarfar, G., Baldwin, D. R., . . . Brain, K. (2015). Barriers to uptake among high-risk individuals declining participation in lung cancer screening: a mixed methods analysis of the UK Lung Cancer Screening (UKLS) trial. BMJ Open, 5(e008254). doi: 10.1136/bmjopen-2015-008254
- Allen, S. S., Brintnell, D. M., Hatsukami, D., & Reich, B. (2004). Energy intake and physical activity during short-term smoking cessation in postmenopausal women. *Addictive Behaviors*, 29(5), 947-951. doi: 10.1016/j.addbeh.2004.02.041
- Allender, S., Balakrishnan, R., Scarborough, P., Webster, P., & Rayner, M. (2009). The burden of smoking-related ill health in the UK. *Tobacco Control*, 18(4), 262-267. doi: 10.1136/tc.2008.026294
- Amos, A., & Haglund, M. (2000). From social taboo to "torch of freedom": The marketing of cigarettes to women. *Tobacco Control*, 9(1), 3-8. doi: 10.1136/tc.9.1.3
- Andrews, J., Yeh, P., Pao, W., & Horn, L. (2011). Molecular predictors of response to chemotherapy in non-small cell lung cancer. *Cancer Journal*, 17(2), 104-113. doi: 10.1097/PPO.0b013e318213f3cf
- Anttila, A., Heikkila, P., Pukkala, E., Nykyri, E., Kauppinen, T., Hernberg, S., & Hemminki, K. (1995). Excess lung cancer among workers exposed to lead. *Scand J Work Environ Health*, 21(6), 460-469. doi: 10.5271/sjweh.62

- Arai, Y., Hosokawa, T., Fukao, A., Izumi, Y., & Hisamichi, S. (1997). Smoking behaviour and personality: a population-based study in Japan. *Addiction*, 92(8), 1023-1033. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9376772
- Arnett, J. J. (2000). Optimistic bias in adolescent and adult smokers and nonsmokers. *Addictive Behaviors*, 25(4), 625-632. doi: 10.1016/S0306-4603(99)00072-6
- Aryal, U. R., Petzold, M., & Krettek, A. (2013). Perceived risks and benefits of cigarette smoking among Nepalese adolescents: A population-based cross-sectional study. *BMC Public Health*, *13*(1), 1-9. doi: 10.1186/1471-2458-13-187
- Atak, N. (2007). A transtheoretical review on smoking cessation. *International Quarterly of Community Health Education*, 28(2), 165-174. doi: 10.2190/IQ.28.2.f.
- Attwood, A., Aveyard, P., Bauld, L., Britton, J., Hajek, P., Hastings, G., . . . West, R. (2013). Chapter 77 Tobacco. In P. M. Miller (Ed.), *Principles of Addiction* (pp. 767-776). San Diego: Academic Press.
- Augustine, A., Harris, R. E., & Wynder, E. L. (1989). Compensation as a risk factor for lung cancer in smokers who switch from nonfilter to filter cigarettes. *Am J Public Health*, *79*(2), 188-191. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1349931/
- Ayanian, J. Z., & Cleary, P. D. (1999). Perceived risks of heart disease and cancer among cigarette smokers. *Journal of the American Medical Association*, 281(11), 1019-1021. doi: 10.1001/jama.281.11.1019
- Baan, R., Grosse, Y., Straif, K., Secretan, B., El Ghissassi, F., Bouvard, V., . . . Cogliano, V. (2009). A review of human carcinogens--Part F: chemical agents and related occupations. *Lancet Oncol*, 10(12), 1143-1144. doi: http://dx.doi.org/10.1016/S1470-2045(09)70358-4
- Bach, P. B., Kattan, M. W., Thornquist, M. D., Kris, M. G., Tate, R. C., Barnett, M. J., . . . Begg, C. B. (2003). Variations in lung cancer risk among smokers. *J Natl Cancer Inst*, 95(6), 470-478. doi: 10.1093/jnci/95.6.470
- Bain, C., Feskanich, D., Speizer, F. E., Thun, M., Hertzmark, E., Rosner, B. A., & Colditz, G. A. (2004). Lung cancer rates in men and women with comparable histories of smoking. *J Natl Cancer Inst*, *96*(11), 826-834. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15173266
- Baler, R. D., & Volkow, N. D. (2011). Addiction as a systems failure: focus on adolescence and smoking. *J Am Acad Child Adolesc Psychiatry*, *50*(4), 329 -339. doi: 10.1016/j.jaac.2010.12.008
- Baldwin, D. R. (2016). Non small cell lung cancer. BMJ Best Practice. Retrieved from http://bestpractice.bmj.com/best-practice/monograph/1082.html Accessed 5 March 2016.
- Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, 84(2), 191-215. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/847061
- Bandura, A. (1982). Self-efficacy mechanism in human agency. *American Psychologist*, 37(2), 122-147. Retrieved from http://psycnet.apa.org/psycinfo/1982-25814-001
- Bandura, A. (1997). *Self-efficacy: The exercise of control.* New York: W.H. Freeman & Co.

- Barnett, R., Pearce, J., & Moon, G. (2009). Community inequality and smoking cessation in New Zealand, 1981-2006. *Soc Sci Med*, 68(5), 876-884. doi: 10.1016/j.socscimed.2008.12.012
- Bauld, L., Judge, K., & Platt, S. (2007). Assessing the impact of smoking cessation services on reducing health inequalities in England: observational study. *Tobacco Control*, 16(6), 400-404. doi: 10.1136/tc.2007.021626
- Beck, U. (1992). *Risk society: Towards a new modernity*. London: Sage Publications Ltd.
- Becker, M. H. (1974). The Health Belief Model and personal health behavior. *Health Education Monographs*, 2, 324-473.
- Bendel, R. B., Afifi, A. A. (1977). Comparison of stopping rules in forward regression. *Journal of the American Statistical Association*, 72(357), 46-53. doi: http://www.tandfonline.com/doi/abs/10.1080/01621459.1977.10479905
- Benowitz, N. L. (2010). Nicotine Addiction. *N Engl J Med*, *362*(24), 2295-2303. doi: 10.1056/NEJMra0809890
- Betzner, A. E., Boyle, R. G., Luxenberg, M. G., Schillo, B. A., Keller, P. A., Rainey, J., . . . Saul, J. E. (2012). Impact of indoor-air policies: Experience of Smokers and Recent Quitters with Smokefree Regulations and Quitting. *American Journal of Preventive Medicine*, 43(S3), S163-S170. doi: 10.1016/j.amepre.2012.08.005
- Bindah, E. V., & Othman, M. N. (2011). The Role of Parental and Peer Smoking Influences in the Development of Adolescents' Smoking Behavior: A Review. *Australian Journal of Basic and Applied Sciences*, 5(11), 1054-1061. Retrieved from http://repository.um.edu.my/16474/1/Bindah%20%26%20Othman%20AJBA S.pdf
- Bize, R., Burnand, B., Mueller, Y., Rege-Walther, M., Camain, J. Y., & Cornuz, J. (2012). Biomedical risk assessment as an aid for smoking cessation. *Cochrane Database Syst Rev*, 12, Cd004705. doi: 10.1002/14651858.CD004705.pub4
- Bloom, E. L., Matsko, S. V., & Cimino, C. R. (2014). The relationship between cigarette smoking and impulsivity: A review of personality, behavioral, and neurobiological assessment. *Addiction Research & Theory*, 22(5), 386-397. doi: 10.3109/16066359.2013.867432
- Bock, B. C., Becker, B., Monteiro, R., Partridge, R., Fisher, S., & Spencer, J. (2001). Physician intervention and patient risk perception among smokers with acute respiratory illness in the emergency department. *Prev Med*, *32*(2), 175-181. doi: 10.1006/pmed.2000.0799
- Boffetta, P., Pershagen, G., Jockel, K. H., Forastiere, F., Gaborieau, V., Heinrich, J., . . . Simonato, L. (1999). Cigar and pipe smoking and lung cancer risk: A multicenter study from Europe. *J Natl Cancer Inst*, *91*(8), 697-701. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10218507
- Boileau, I., Assaad, J. M., Pihl, R. O., Benkelfat, C., Leyton, M., Diksic, M., . . . Dagher, A. (2003). Alcohol promotes dopamine release in the human nucleus accumbens. *Synapse*, 49(4), 226-231. doi: 10.1002/syn.10226
- Bomlitz, L. J., & Brezis, M. (2008). Misrepresentation of health risks by mass media. *Journal of Public Health*, 30(2), 202-204. doi: 10.1093/pubmed/fdn009
- Borland, R., Yong, H. H., Balmford, J., Fong, G. T., Zanna, M. P., & Hastings, G. (2009). Do risk-minimizing beliefs about smoking inhibit quitting? Findings from the International Tobacco Control (ITC) Four-Country Survey. *Prev Med*, 49(2-3), 219-223. doi: 10.1016/j.ypmed.2009.06.015

- Borrelli, B., Hayes, R. B., Dunsiger, S., & Fava, J. L. (2010). Risk perception and smoking behavior in medically ill smokers: a prospective study. *Addiction*, 105(6), 1100-1108. doi: 10.1111/j.1360-0443.2010.02900.x.
- Borrelli, B., Hogan, J. W., Bock, B., Pinto, B., Roberts, M., & Marcus, B. (2002). Predictors of quitting and dropout among women in a clinic-based smoking cessation program. *Psychol Addict Behav*, *16*(1), 22-27. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11934082
- Bowen, H. P., & Wiersema, M. F. (1999). Matching method to paradigm in strategy research: limitations of cross-sectional analysis and some methodological alternatives. *Strategic Management Journal*, 20(7), 625-636. doi: 10.1002/(SICI)1097-0266(199907)20:7<625::AID-SMJ45>3.0.CO;2-V
- Bowie, C., & Bowie, S. H. (1991). Radon and health. *Lancet*, *337*(8738), 409-413. doi: http://dx.doi.org/10.1016/0140-6736(91)91177-V
- Brant, R. (1990). Assessing proportionality in the proportional odds model for ordinal logistic regression. *Biometrics*, 46(4), 1171-1178. Retrieved from http://www.jstor.org/stable/2532457
- Braun, V., & Clarke, V. (2006). Using thematic analysis in psychology. *Qualitative Research in Psychology*, 3(2), 77-101. doi: 10.1191/1478088706qp063oa
- Brennan, E., Gibson, L., Momjian, A., & Hornik, R. C. (2015). Are young people's beliefs about menthol cigarettes associated with smoking-related intentions and behaviors? *Nicotine & Tobacco Research*, *17*(1), 81-90. doi: 10.1093/ntr/ntu134
- Brenner, D. R., Boffetta, P., Duell, E. J., Bickeboller, H., Rosenberger, A., McCormack, V., . . . Hung, R. J. (2012). Previous lung diseases and lung cancer risk: A pooled analysis from the International Lung Cancer Consortium. *Am J Epidemiol*, *176*(7), 573-585. doi: 10.1093/aje/kws151
- Brenner, D. R., McLaughlin, J. R., & Hung, R. J. (2011). Previous lung diseases and lung cancer risk: A systematic review and meta-analysis. *PLoS One*, 6(3), e17479. doi: 10.1371/journal.pone.0017479
- Breslau, N., Kilbey, M. M., & Andreski, P. (1992). Nicotine withdrawal symptoms and psychiatric disorders: Findings from an epidemiologic study of young adults. *Am J Psychiatry*, *149*(4), 464-469. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1554030
- Breslau, N., & Peterson, E. L. (1996). Smoking cessation in young adults: Age at initiation of cigarette smoking and other suspected influences. *Am J Public Health*, 86(2), 214-220. doi: 10.2105/AJPH.86.2.214
- Broms, U., Silventoinen, K., Lahelma, E., Kaprio, J., & Koskenvuo, M. (2004). Smoking cessation by socioeconomic status and marital status: The contribution of smoking behavior and family background. *Nicotine and Tobacco Research*, 6(3), 447-455. doi: 10.1080/14622200410001696637
- Brook, D. W., Brook, J. S., Zhang, C., Whiteman, M., Cohen, P., & Finch, S. J. (2008). Developmental trajectories of cigarette smoking from adolescence to the early thirties: Personality and behavioral risk factors. *Nicotine Tob Res*, *10*(8), 1283-1291. doi: 10.1080/14622200802238993
- Brose, L. S., West, R., McDermott, M. S., Fidler, J. A., Croghan, E., & McEwen, A. (2011). What makes for an effective stop-smoking service? *Thorax*, 66(10), 924-926. doi: 10.1136/thoraxjnl-2011-200251
- Brown, T. (2009). Silica exposure, smoking, silicosis and lung cancer--complex interactions. *Occup Med (Lond)*, 59(2), 89-95. doi: 10.1093/occmed/kqn171

- Brown, T., Darnton, A., Fortunato, L., & Rushton, L. (2012). Occupational cancer in Britain. Respiratory cancer sites: Larynx, lung and mesothelioma. *Br J Cancer*, 107(S1), S56-70. doi: 10.1038/bjc.2012.119
- Brownson, R. C., Chang, J. C., & Davis, J. R. (1992). Gender and histologic type variations in smoking-related risk of lung cancer. *Epidemiology*, *3*(1), 61-64. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1313311
- Cahill, K., Lancaster, T., & Green, N. (2010). Stage-based interventions for smoking cessation. *Cochrane Database Syst Rev*(11), Cd004492. doi: 10.1002/14651858.CD004492.pub4
- Cahill, K., Stead, L. F., & Lancaster, T. (2012). Nicotine receptor partial agonists for smoking cessation. *Cochrane Database Syst Rev*, 4, Cd006103. doi: 10.1002/14651858.CD006103.pub6
- Callum, C., Boyle, S., & Sandford, A. (2011). Estimating the cost of smoking to the NHS in England and the impact of declining prevalence. *Health Econ Policy Law*, 6(4), 489-508. doi: 10.1017/s1744133110000241
- Campbell, M. K., Elbourne, D. R., & Altman, D. G. (2004). CONSORT statement: Extension to cluster randomised trials. *BMJ*, 328(7441), 702-708. doi: 10.1136/bmj.328.7441.702
- Cancer Research UK. (2007). CancerStats: Lung cancer and smoking UK. Retrieved from:

 http://publications.cancerresearchuk.org/downloads/Product/cs_pdf_lung_july_2007.pdf
- Cancer Research UK. (2013). Lung cancer incidence statistics. Retrieved from: http://www.cancerresearchuk.org/health-professional/cancer-statistics/statistics-by-cancer-type/lung-cancer/incidence
- Cancer Research UK. (2014a). Lung cancer mortality statistics. Retrieved from http://www.cancerresearchuk.org/health-professional/cancerstatistics/statistics-by-cancer-type/lung-cancer/mortality
- Cancer Research UK. (2014b). Lung cancer risk factors. Retrieved from http://www.cancerresearchuk.org/health-professional/cancer-statistics/statistics-by-cancer-type/lung-cancer/risk-factors
- Cancer Research UK. (2014c). Lung cancer survival statistics. Retrieved from http://www.cancerresearchuk.org/health-professional/cancerstatistics/statistics-by-cancer-type/lung-cancer/survival
- Caponnetto, P., & Polosa, R. (2008). Common predictors of smoking cessation in clinical practice. *Respiratory Medicine*, 102(8), 1182-1192. doi: 10.1016/j.rmed.2008.02.017
- Carpenter, C. J. (2010). A meta-analysis of the effectiveness of Health Belief Model variables in predicting behavior. *Health Communication*, 25(8), 661-669. doi: 10.1080/10410236.2010.521906
- Cassidy, A., Duffy, S. W., Myles, J. P., Liloglou, T., & Field, J. K. (2007). Lung cancer risk prediction: a tool for early detection. *Int J Cancer*, 120(1), 1-6. doi: 10.1002/ijc.22331
- Cassidy, A., & Field, J. (2007). Environmental and genetic risk factors of lung cancer. In A. F. G. Taktak & A. C. Fisher (Eds.), *Outcome prediction in cancer* (pp. 67-100). Oxford: Elsevier.
- Cassidy, A., Myles, J. P., Duffy, S. W., Liloglou, T., & Field, J. K. (2006). Family history and risk of lung cancer: Age-at-diagnosis in cases and first-degree relatives. *Br J Cancer*, *95*(9), 1288-1290. doi: 10.1038/sj.bjc.6603386

- Cassidy, A., Myles, J. P., van Tongeren, M., Page, R. D., Liloglou, T., Duffy, S. W., & Field, J. K. (2008). The LLP risk model: An individual risk prediction model for lung cancer. *Br J Cancer*, *98*(2), 270-276. doi: 10.1038/sj.bjc.6604158
- Cavelaars, A. E., Kunst, A. E., Geurts, J. J., Crialesi, R., Grotvedt, L., Helmert, U., . . . Mackenbach, J. P. (2000). Educational differences in smoking: International comparison. *BMJ*, 320(7242), 1102-1107. doi: http://dx.doi.org/10.1136/bmj.320.7242.1102
- Cepeda-Benito, A., Reynoso, J. T., & Erath, S. (2004). Meta-analysis of the efficacy of nicotine replacement therapy for smoking cessation: Differences between men and women. *J Consult Clin Psychol*, 72(4), 712-722. doi: 10.1037/0022-006X.72.4.712
- Chaloupka, F. J., Straif, K., & Leon, M. E. (2011). Effectiveness of tax and price policies in tobacco control. *Tobacco Control*, 20(3), 235-238. doi: 10.1136/tc.2010.039982
- Chandola, T., Head, J., & Bartley, M. (2004). Socio-demographic predictors of quitting smoking: how important are household factors? *Addiction*, 99(6), 770-777. Retrieved from: http://www.ncbi.nlm.nih.gov/pubmed/15139875
- Chapman, S., Wong, W. L., & Smith, W. (1993). Self-exempting beliefs about smoking and health: differences between smokers and ex-smokers. *Am J Public Health*, 83(2), 215-219. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1694573/
- Chassin, L., Presson, C. C., Sherman, S. J., & Kim, K. (2002). Long-term psychological sequelae of smoking cessation and relapse. *Health Psychol*, 21(5), 438-443. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12211510
- Chatkin, J. M. (2006). The influence of genetics on nicotine dependence and the role of pharmacogenetics in treating the smoking habit. *J Bras Pneumol*, 32(6), 573-579. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17435909
- Checkoway, H., & Franzblau, A. (2000). Is silicosis required for silica-associated lung cancer? *Am J Ind Med*, 37(3), 252-259. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10642414
- Chen, D., & Wu, L.-T. (2015). Review: Smoking cessation interventions for adults aged 50 or older: A systematic review and meta-analysis. *Drug and Alcohol Dependence*, 154, 14-24. doi: 10.1016/j.drugalcdep.2015.06.004
- Chen, H., Goldberg, M. S., & Villeneuve, P. J. (2008). A systematic review of the relation between long-term exposure to ambient air pollution and chronic diseases. *Rev Environ Health*, 23(4), 243-297. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/19235364
- Chen, J., & Millar, W. J. (1998). Age of smoking initiation: implications for quitting. *Health reports*, 9(4), 39-46. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9836879
- Chen, Y., Marcus, M. W., Niaz, A., Field, J. K., & Duffy, S. W. (2014). MyLungRisk: A user-friendly, web-based calculator for risk assessment of lung cancer based on the validated Liverpool Lung Project risk prediction model. *International Journal of Health Promotion and Education*, 52(3), 144-152. doi: 10.1080/14635240.2014.888814
- Chen, Y. F., Madan, J., Welton, N., Yahaya, I., Aveyard, P., Bauld, L., . . . Munafo, M. (2012). Effectiveness and cost-effectiveness of computer and other electronic aids for smoking cessation: A systematic review and network meta-analysis. *Health Technol Assess*, 16(38), 1-205. doi: 10.3310/hta16380

- Cherry, N., & Kiernan, K. (1976). Personality scores and smoking behaviour: A longitudinal study. *Br J Prev Soc Med*, *30*(2), 123-131. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC478950/
- Chesterman, J., Judge, K., Bauld, L., & Ferguson, J. (2005). How effective are the English smoking treatment services in reaching disadvantaged smokers? *Addiction*, 100(S2), 36-45. doi: 10.1111/j.1360-0443.2005.01026.x
- Chiang, PP-C., Glance, D., Walker, J., Walter, F. M. & Emery, J. D. (2015). Implementing a QCancer risk tool into general practice consultations: an exploratory study using simulated consultations with Australian general practitioners. British Journal of Cancer, 112, S77-S83. doi: 10.1038/bjc.2015.46
- Cogliano, V. J., Baan, R., Straif, K., Grosse, Y., Lauby-Secretan, B., El Ghissassi, F., . . . Wild, C. P. (2011). Preventable exposures associated with human cancers. *J Natl Cancer Inst*, 103(24), 1827-1839. doi: 10.1093/jnci/djr483
- Cohen, S., & Lichtenstein, E. (1990). Partner behaviors that support quitting smoking. *J Consult Clin Psychol*, 58(3), 304-309. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/2365893
- Colditz, G. A., Atwood, K. A., Emmons, K., Monson, R. R., Willett, W. C., Trichopoulos, D., & Hunter, D. J. (2000). Harvard report on cancer prevention volume 4: Harvard cancer risk index. Risk index working group, Harvard Center for Cancer Prevention. *Cancer Causes Control*, 11(6), 477-488. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10880030
- Connor Gorber, S., Schofield-Hurwitz, S., Hardt, J., Levasseur, G., & Tremblay, M. (2009). The accuracy of self-reported smoking: A systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine Tob Res*, 11(1), 12-24. doi: 10.1093/ntr/ntn010
- Cooper, J., Borland, R., Yong, H. H., McNeill, A., Murray, R. L., O'Connor, R. J., & Cummings, K. M. (2010). To what extent do smokers make spontaneous quit attempts and what are the implications for smoking cessation maintenance? Findings from the International Tobacco Control Four country survey. *Nicotine Tob Res*, *12(S)*, S51-57. doi: 10.1093/ntr/ntq052
- Coppotelli, H. C., & Orleans, C. T. (1985). Partner support and other determinants of smoking cessation maintenance among women. *J Consult Clin Psychol*, *53*(4), 455-460. doi: 10.1037/0022-006X.53.4.455
- Corcoran, N. (2007). Chapter 1: Theories and models in communicating health messages. In N. Corcoran. *Communicating health: Strategies for health promotion* (pp. 5-31). London: Sage Publications Ltd.
- Costa, P. T., Jr., Fagan, P. J., Piedmont, R. L., Ponticas, Y., & Wise, T. N. (1992). The five-factor model of personality and sexual functioning in outpatient men and women. *Psychiatr Med*, 10(2), 199-215. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1615160
- Cote, M. L., Liu, M., Bonassi, S., Neri, M., Schwartz, A. G., Christiani, D. C., . . . Hung, R. J. (2012). Increased risk of lung cancer in individuals with a family history of the disease: a pooled analysis from the International Lung Cancer Consortium. *Eur J Cancer*, 48(13), 1957-1968. doi: 10.1016/j.ejca.2012.01.038
- Covey, L. S., Glassman, A. H., & Stetner, F. (1997). Major depression following smoking cessation. *Am J Psychiatry*, 154(2), 263-265. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9016279

- Creswell, J. W. (2014). Research design: Qualitative, quantitative, and mixed methods approaches (4th ed.). Thousand Oaks, California: Sage Publications Inc.
- Creswell, J. W., & Plano Clark, V. L. (2011). *Designing and conducting mixed methods research*. Los Angeles: Sage Publications.
- Croce, C. M. (2008). Oncogenes and cancer. *New England Journal of Medicine*, 358(5), 502-511. doi: 10.1056/NEJMra072367
- Cronin, K. A., Gail, M. H., Zou, Z., Bach, P. B., Virtamo, J., & Albanes, D. (2006). Validation of a model of lung cancer risk prediction among smokers. *J Natl Cancer Inst*, 98(9), 637-640. doi: 10.1093/jnci/djj163
- D'Amelio, A. M., Jr., Cassidy, A., Asomaning, K., Raji, O. Y., Duffy, S. W., Field, J. K., . . . Etzel, C. J. (2010). Comparison of discriminatory power and accuracy of three lung cancer risk models. *Br J Cancer*, *103*(3), 423-429. doi: 10.1038/sj.bjc.6605759
- Dale, L. C., Glover, E. D., Sachs, D. P., Schroeder, D. R., Offord, K. P., Croghan, I. T., & Hurt, R. D. (2001). Bupropion for smoking cessation: predictors of successful outcome. *Chest*, 119(5), 1357-1364. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11348939
- Darby, S. C., & Hill, D. C. (2003). Health effects of residential radon: a European perspective at the end of 2002. *Radiat Prot Dosimetry*, 104(4), 321-329. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14579888
- Darnton, A. J., McElvenny, D. M., & Hodgson, J. T. (2006). Estimating the number of asbestos-related lung cancer deaths in Great Britain from 1980 to 2000. *Ann Occup Hyg*, 50(1), 29-38. doi: 10.1093/annhyg/mei038
- Davies, G. E., & Soundy, T. J. (2009). The genetics of smoking and nicotine addiction. *S D Med*, *Spec No*, 43-49. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/19363894
- de Magalhães, J. P. (2013). How ageing processes influence cancer. *Nature Reviews Cancer*, 13(5), 357-365. doi: 10.1038/nrc3497
- de Viron, S., Van der Heyden, J., Ambrosino, E., Arbyn, M., Brand, A., & Van Oyen, H. (2012). Impact of genetic notification on smoking cessation: systematic review and pooled-analysis. *PLoS One*, *7*(7), e40230. doi: 10.1371/journal.pone.0040230
- Deas, I., Robson, B., Wong, C., & Bradford, M. (2003). Measuring neighbourhood deprivation: A critique of the Index of Multiple Deprivation. *Environment and Planning C: Government and Policy*, 21(6), 883-903. doi: 10.1068/c0240
- Deci, E. L., & Ryan, R. M. (1985). *Intrinsic motivation and self-determination in human behavior*. New York: Plenum.
- Denholm, R., Schuz, J., Straif, K., Stucker, I., Jockel, K. H., Brenner, D. R., . . . A, C. O. (2014). Is previous respiratory disease a risk factor for lung cancer? *Am J Respir Crit Care Med*, 190(5), 549-559. doi: 10.1164/rccm.201402-0338OC
- Department of Health. (2014). Consultation on the introduction of regulations for standardised packaging of tobacco products. London, UK: Retrieved from https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/323922/Cons_doc.pdf.
- Department of Health [DH]. (1998). Smoking kills: A white paper on tobacco. Retrieved from https://www.gov.uk/government/uploads/system/uploads/attachment_data/fil e/260754/4177.pdf
- Department of Health [DH]. (2011). Healthy lives, healthy people: A tobacco control plan for England. Retrieved from

- https://www.gov.uk/government/publications/the-tobacco-control-plan-for-england
- Department of Health [DH]. (2014). Government response to the consultation on smoking in private vehicles carrying children. Retrieved from https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/384319/smoking_in_vehicles-_govresponse.pdf
- Diaz, R. M., & Berk, L. E. (1992). *Private speech: From social interaction to self-regulation*. Hillsdale, NJ: Lawrence Erlbaum Associates.
- Diederich, S., Wormanns, D., Semik, M., Thomas, M., Lenzen, H., Roos, N., & Heindel, W. (2002). Screening for early lung cancer with low-dose spiral CT: Prevalence in 817 asymptomatic smokers. *Radiology*, 222(3), 773-781. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11867800
- Dijkstra, A. (2009). Disengagement beliefs in smokers: Do they influence the effects of a tailored persuasive message advocating smoking cessation? *Psychology & Health*, 24(7), 791-804. doi: 10.1080/08870440801998962.
- Dillard, A. J., McCaul, K. D., & Klein, W. M. (2006). Unrealistic optimism in smokers: implications for smoking myth endorsement and self-protective motivation. *J Health Commun*, 11(S1), 93-102. doi: 10.1080/10810730600637343
- Doll, R., & Hill, A. B. (1950). Smoking and carcinoma of the lung; preliminary report. *Br Med J*, 2(4682), 739-748. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14772469
- Doll, R., Peto, R., Boreham, J., & Sutherland, I. (2004). Mortality in relation to smoking: 50 years' observations on male British doctors. *BMJ*, *324*(1519). doi: http://dx.doi.org/10.1136/bmj.38142.554479.AE
- Donington, J. S., Le, Q. T., & Wakelee, H. A. (2006). Lung cancer in women: Exploring sex differences in susceptibility, biology, and therapeutic response. *Clin Lung Cancer*, 8(1), 22-29. doi: 10.3816/CLC.2006.n.029
- Douglas, M. (1992). Risk and Blame: Essays in Cultural Theory. Oxon: Routledge.
- Douglas, M., & Wildavsky, A. (1982). Risk and culture: An essay on the selection of technical and environmental dangers. Berkeley: University of California Press.
- Dowding, D. (2006). Personal beliefs, experiences, and emotions influenced smokers' perceptions of their cancer risk. *Evidence Based Nursing*, 9(3), 92-92.
- Drummond, M. B., Lambert, A. A., Stitzer, M. L., Merlo, C. A., Rand, C. S., Wise, R. A., . . . Goldberg, S. (2014). A randomized study of contingency management and spirometric lung age for motivating smoking cessation among injection drug users. *BMC Public Health*, *14*(1). doi: 10.1186/1471-2458-14-761
- Ebbert, J. O., Yang, P., Vachon, C. M., Vierkant, R. A., Cerhan, J. R., Folsom, A. R., & Sellers, T. A. (2003). Lung cancer risk reduction after smoking cessation: Observations from a prospective cohort of women. *J Clin Oncol*, *21*(5), 921-926. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12610194
- Edwards, A., Elwyn, G., Covey, J., Matthews, E., & Pill, R. (2001). Presenting risk information a review of the effects of framing and other manipulations on patient outcomes. *J Health Commun*, 6(1), 61-82. doi: 10.1080/10810730150501413
- Edwards, A., Naik, G., Ahmed, H., Elwyn, G. J., Pickles, T., Hood, K., & Playle, R. (2013). Personalised risk communication for informed decision making about taking screening tests. *Cochrane Database Syst Rev, 2*, Cd001865. doi: 10.1002/14651858.CD001865.pub3

- Ehteshami-Afshar, S., Momenan, A., Hajshekholeslami, F., Azizi, F., & Hadaegh, F. (2014). The impact of smoking status on 9.3 years incidence of cardiovascular and all-cause mortality among Iranian men. *Annals of Human Biology*, 41(3), 249-254. doi: 10.3109/03014460.2013.853834
- Eisinger, R. A. (1971). Psychosocial predictors of smoking recidivism. *J Health Soc Behav*, 12(4), 355-362. Retrieved from http://www.jstor.org/stable/2137080
- Eklund, B. M., Hedman, L., Nilsson, S., & Lindberg, I. (2012). Why do smokers diagnosed with COPD not quit smoking? A qualitative study. *Tob Induc Dis*, *10*(1). doi: 10.1186/1617-9625-10-17
- Ellickson, P. L., McGuigan, K. A., & Klein, D. J. (2001). Predictors of late-onset smoking and cessation over 10 years. *Journal of Adolescent Health*, 29(2), 101-108. doi: http://dx.doi.org/10.1016/S1054-139X(00)00199-3
- Emery, S. L., Szczypka, G., Abril, E. P., Kim, Y., Vera, L. (2014). Are you scared yet? Evaluating messages in tweets about the Tips Campaign. *Journal of Communication*, 64(2), 278-295. doi: 10.1111/jcom.12083
- Engeland, A., Haldorsen, T., Andersen, A., & Tretli, S. (1996). The impact of smoking habits on lung cancer risk: 28 years' observation of 26,000 Norwegian men and women. *Cancer Causes Control*, 7(3), 366-376. Retrieved from http://link.springer.com/article/10.1007/BF00052943
- Epstein, A. M., Sher, T. G., Young, M. A., & King, A. C. (2007). Tobacco chippers show robust increases in smoking urge after alcohol consumption. *Psychopharmacology (Berl)*, 190(3), 321-329. doi: 10.1007/s00213-006-0438-8
- Erren, T. C., Morfeld, P., Glende, C. B., Piekarski, C., & Cocco, P. (2011). Metaanalyses of published epidemiological studies, 1979-2006, point to open causal questions in silica-silicosis-lung cancer research. *Med Lav, 102*(4), 321-335. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/21834269
- European Commission. (2014). European Commission's draft Tobacco Products. European Commission's Directive. Retrieved from http://ec.europa.eu/health/tobacco/docs/dir_201440_en.pdf
- Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual of the Eysenck Personality Questionnaire*. London: Hodder and Stoughton.
- Fagerström, K., & Furberg, H. (2008). A comparison of the Fagerström Test for Nicotine Dependence and smoking prevalence across countries. *Addiction*, 103(5), 841-845. doi: 10.1111/j.1360-0443.2008.02190.x
- Fagerström, K., Russ, C., Yu, C.-R., Yunis, C., & Foulds, J. (2012). The Fagerström test for Nicotine Dependence as a predictor of smoking abstinence: A pooled analysis of varenicline clinical trial data. *Nicotine & Tobacco Research*, 14(12), 1467-1473. doi: 10.1093/ntr/nts018
- Farhangmehr, M., Jalali, M., & Silva, C. (2015). Anti-smoking themes what works best for adolescents? *International Review On Public & Non Profit Marketing*, 12(1), 17. doi:10.1007/s12208-014-0124-1
- Farkas, A. J., Pierce, J. P., Zhu, S. H., Rosbrook, B., Gilpin, E. A., Berry, C., & Kaplan, R. M. (1996). Addiction versus stages of change models in predicting smoking cessation. *Addiction*, *91*(9), 1271-1280; discussion 1281-1292. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8854358
- Faul, F., Erdfelder, E., Buchner, A., & Lang, A. G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behav Res Methods*, 41(4), 1149-1160. doi: 10.3758/brm.41.4.1149

- Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods*, 39(2), 175-191. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17695343
- Ferguson, J., Bauld, L., Chesterman, J., & Judge, K. (2005). The English smoking treatment services: one-year outcomes. *Addiction*, 100(S2), 59-69. doi: 10.1111/j.1360-0443.2005.01028.x
- Ferlay, J., Shin, H. R., Bray, F., Forman, D., Mathers, C., & Parkin, D. M. (2010). GLOBOCAN 2008 v1.2, Cancer Incidence and Mortality Worldwide: IARC CancerBase No. 10. Retrieved from http://globocan.iarc.fr/
- Ferlay, J., Soerjomataram, I., Dikshit, R., Eser, S., Mathers, C., Rebelo, M., . . . Bray, F. (2015). Cancer incidence and mortality worldwide: Sources, methods and major patterns in GLOBOCAN 2012. *International Journal of Cancer*, *136*(5), E359-E386. doi: 10.1002/ijc.29210
- Festinger, L. (1957). A theory of cognitive dissonance by Leon Festinger. Stanford (Calif.): Stanford University Press.
- Festinger, L. (1962). Cognitive dissonance. *Scientific American*, 207(4), 93-107. doi: 10.1038/scientificamerican1062-93
- Fidler, J., Brown, J., Stapleton, J., West, R., & Ferguson, S. G. (2013). How does rate of smoking cessation vary by age, gender and social grade? Findings from a population survey in England. *Addiction*, 108(9), 1680-1685. doi: 10.1111/add.12241
- Fidler, J., & West, R. (2011). Enjoyment of smoking and urges to smoke as predictors of attempts and success of attempts to stop smoking: A longitudinal study. *Drug and Alcohol Dependence*, 115, 30-34. doi: 10.1016/j.drugalcdep.2010.10.009
- Field, A. (2013). *Discovering statistics using IBM SPSS Statistics* (4th ed.). London: Sage Publications Inc.
- Field, J. K. (2008). Lung cancer risk models come of age. *Cancer Prev Res (Phila)*, 1(4), 226-228. doi: 10.1158/1940-6207.CAPR-08-0144
- Field, J. K., Oudkerk, M., Pedersen, J. H., & Duffy, S. W. (2013). Prospects for population screening and diagnosis of lung cancer. *Lancet*, 382(9893), 732-741. doi: 10.1016/s0140-6736(13)61614-1
- Field, J. K., Smith, D. L., Duffy, S., & Cassidy, A. (2005). The Liverpool Lung Project research protocol. *Int J Oncol*, 27(6), 1633-1645. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16273220
- Finucane, M. L., Slovic, P., Mertz, C. K., Flynn, J., & Satterfield, T. A. (2000). Gender, race, and perceived risk: The 'white male' effect. *Health, Risk & Society*, 2(2), 159-172. doi: 10.1080/713670162
- Forey, B. A., Thornton, A. J., & Lee, P. N. (2011). Systematic review with metaanalysis of the epidemiological evidence relating smoking to COPD, chronic bronchitis and emphysema. *BMC Pulmonary Medicine*, 11, 36-36. doi: 10.1186/1471-2466-11-36
- Forgays, D. G., Bonaiuto, P., Wrzesniewski, K., & Forgays, D. K. (1993). Personality and cigarette smoking in Italy, Poland, and the United States. *Int J Addict*, 28(5), 399-413. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8478154
- Fotuhi, O., Fong, G. T., Zanna, M. P., Borland, R., Yong, H., & Cummings, K. M. (2013). Patterns of cognitive dissonance-reducing beliefs among smokers: a longitudinal analysis from the International Tobacco Control (ITC) Four

- Country Survey. *Tobacco Control*, 22(1), 52-58.doi: 10.1136/tobaccocontrol-2011-050139
- France, A. (2000). Towards a Sociological Understanding of Youth and their Risk-taking. *Journal of Youth Studies*, *3*(3), 317-331. doi: 10.1080/713684380
- Freedman, A. N., Seminara, D., Gail, M. H., Hartge, P., Colditz, G. A., Ballard-Barbash, R., & Pfeiffer, R. M. (2005). Cancer risk prediction models: a workshop on development, evaluation, and application. *J Natl Cancer Inst*, 97(10), 715-723. doi: 10.1093/jnci/dji128
- Freedman, N. D., Leitzmann, M. F., Hollenbeck, A. R., Schatzkin, A., & Abnet, C. C. (2008). Cigarette smoking and subsequent risk of lung cancer in men and women: analysis of a prospective cohort study. *Lancet Oncol*, *9*(7), 649-656. doi: 10.1016/S1470-2045(08)70154-2
- Frost, G. (2011). RR833 The joint effect of asbestos exposure and smoking on the risk of lung cancer mortality for asbestos workers (1971-2005). 62. Retrieved from http://www.hse.gov.uk/research/rrpdf/rr833.pdf
- Fry, J. S., Hamling, J. S., & Lee, P. N. (2012). Systematic review with meta-analysis of the epidemiological evidence relating FEV1 decline to lung cancer risk. *BMC Cancer*, 12. doi: 10.1186/1471-2407-12-498
- Gail, M. H., Brinton, L. A., Byar, D. P., Corle, D. K., Green, S. B., Schairer, C., & Mulvihill, J. J. (1989). Projecting individualized probabilities of developing breast cancer for white females who are being examined annually. *J Natl Cancer Inst*, 81(24), 1879-1886. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/2593165
- Galda, P. M., Cheater, F., Marshall, P. (2005). Men and health help-seeking behaviour: literature review. *Journal of Advanced Nursing*, 49(6), 616-623. doi: 10.1111/j.1365-2648.2004.03331.x
- Galeone, C., Pelucchi, C., La Vecchia, C., Negri, E., Bosetti, C., & Hu, J. (2008). Indoor air pollution from solid fuel use, chronic lung diseases and lung cancer in Harbin, Northeast China. *Eur J Cancer Prev, 17*(5), 473-478. doi: 10.1097/CEJ.0b013e328305a0b9
- Gallagher, K. M., & Updegraff, J. A. (2012). Health message framing effects on attitudes, intentions, and behavior: A meta-analytic review. *Annals of Behavioral Medicine*, 43(1), 101-116. doi: 10.1007/s12160-011-9308-7
- Gallopel-Morvan, K., Gabriel, P., Le Gall-Ely, M., Rieunier, S., & Urien, B. (2011). The use of visual warnings in social marketing: The case of tobacco. *Journal of Business Research*, 64(1), 7-11. doi: http://dx.doi.org/10.1016/j.jbusres.2009.09.012
- Garshick, E., Laden, F., Hart, J. E., Rosner, B., Smith, T. J., Dockery, D. W., & Speizer, F. E. (2004). Lung cancer in railroad workers exposed to diesel exhaust. *Environ Health Perspect*, 112(15), 1539-1543. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15531439
- Garvey, A. J., Bliss, R. E., Hitchcock, J. L., Heinold, J. W., & Rosner, B. (1992). Predictors of smoking relapse among self-quitters: A report from the Normative Aging Study. *Addict Behav*, *17*(4), 367-377. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1502970
- Gately, I. (2001). *Tobacco: The story of how tobacco seduced the world.* New York: Grove Press.
- Gellert, C., Schottker, B., & Brenner, H. (2012). Smoking and all-cause mortality in older people: Systematic review and meta-analysis. *Arch Intern Med*, 172(11), 837-844. doi: 10.1001/archinternmed.2012.1397

- Gerking, S., & Khaddaria, R. (2012). Perceptions of Health Risk and Smoking Decisions of Young People. *Health Economics*, 21(7), 865-877. doi: 10.1002/hec.1760
- Gerrard, M., Gibbons, F. X., Benthin, A. C., & Hessling, R. M. (1996). A longitudinal study of the reciprocal nature of risk behaviors and cognitions in adolescents: What you do shapes what you think, and vice versa. *Health Psychol*, *15*(5), 344-354. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8891713
- Gerrard, M., Gibbons, F. X., & Bushman, B. J. (1996). Relation between perceived vulnerability to HIV and precautionary sexual behavior. *Psychol Bull*, *119*(3), 390-409. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8668745
- Gerrard, M., Gibbons, F. X., & Reis-Bergan, M. (1999). The effect of risk communication on risk perceptions: The significance of individual differences. *J Natl Cancer Inst Monogr*(25), 94-100. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10854464
- Gharlipour, Z., Hazavehei, S. M., Moeini, B., Nazari, M., Beigi, A. M., Tavassoli, E., & ... Barkati, H. (2015). The effect of preventive educational program in cigarette smoking: Extended Parallel Process Model. *Journal of Education and Health Promotion*, 44. doi:10.4103/2277-9531.151875
- Gibbons, F. X., Eggleston, T. J., & Benthin, A. C. (1997). Cognitive reactions to smoking relapse: The reciprocal relation between dissonance and self-esteem. *Journal of Personality and Social Psychology*, 72(1), 184-195. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9008380
- Gigerenzer, G., Gaissmaier, W., Kurz-Milcke, E., Schwartz, L. M., & Woloshin, S. (2007). Helping Doctors and Patients Make Sense of Health Statistics. *Psychological Science in the Public Interest*, 8(2), 53-96. doi: 10.1111/j.1539-6053.2008.00033.x
- Gilman, S. E., Martin, L. T., Abrams, D. B., Kawachi, I., Kubzansky, L., Loucks, E. B., . . . Buka, S. L. (2008). Educational attainment and cigarette smoking: a causal association? *Int J Epidemiol*, *37*(3), 615-624. doi: 10.1093/ije/dym250
- Gilpin, E. A., Pierce, J. P., & Farkas, A. J. (1997). Duration of smoking abstinence and success in quitting. *J Natl Cancer Inst*, 89(8), 572-576. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9106646
- Godfrey, C., Parrott, S., Coleman, T., & Pound, E. (2005). The cost-effectiveness of the English smoking treatment services: evidence from practice. *Addiction*, 100(S2), 70-83. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15844290
- Godin, G., & Kok, G. (1996). The theory of planned behavior: A review of its applications to health-related behaviors. *Am J Health Promot*, 11(2), 87-98. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10163601
- Gohagan, J. K., Prorok, P. C., Hayes, R. B., & Kramer, B. S. (2000). The Prostate, Lung, Colorectal and Ovarian (PLCO) Cancer Screening Trial of the National Cancer Institute: history, organization, and status. *Control Clin Trials*, *21*(S6), 251S-272S. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11189683
- Goldenberg, M., Danovitch, I., & IsHak, W. W. (2014). Quality of life and smoking. *The American Journal on Addictions*, 23(6), 540-562. doi: 10.1111/j.1521-0391.2014.12148.x
- Gomez, M. M., & LoBiondo-Wood, G. (2013). Lung Cancer Screening With Low-Dose CT: Its Effect on Smoking Behavior. *Journal of the Advanced Practitioner in Oncology*, 4(6), 405-414. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/25032020

- Gould, G., & Pearce, A. (2006). Assessment of Suitability for Lung Resection. Continuing Education in Anaesthesia, Critical Care & Pain, 6(3), 97-100. doi: 10.1093/bjaceaccp/mkl016
- Gould, G. S., Watt, K., Cadet-James, Y., Clough, A. R. (2015). Using the risk behaviour diagnosis scale to understand Australian Aboriginal smoking: A cross-sectional validation survey in regional New South Wales. *Preventive Medicine Reports*, 2(1), 4-9. doi: 10.1016/j.pmedr.2014.10.004
- Gourlay, S. G., Forbes, A., Marriner, T., Pethica, D., & McNeil, J. J. (1994). Prospective study of factors predicting outcome of transdermal nicotine treatment in smoking cessation. *BMJ*, *309*(6958), 842-846. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7950614
- Grant, A., Ashton, K., & Phillips, R. (2014). Foucault, surveillance, and carbon monoxide testing within stop-smoking services. *Qualitative Health Research*, 25(7): 912–922. doi: 10.1177/1049732314553992
- Greening, L. (1997). Adolescents' cognitive appraisals of cigarette smoking: An application of the protection motivation theory. *Journal of Applied Social Psychology*, 27(22), 1972-1985. doi: 10.1111/j.1559-1816.1997.tb01635.x
- Greening, L., & Dollinger, S. J. (1991). Adolescent smoking and perceived vulnerability to smoking-related causes of death. *Journal of Pediatric Psychology*, *16*(6), 687-699. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1798008
- Grimsrud, T. K., Langseth, H., Engeland, A., & Andersen, A. (1998). Lung and bladder cancer in a Norwegian municipality with iron and steel producing industry: population based case-control studies. *Occup Environ Med*, 55(6), 387-392. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9764098
- Grogan, S., Fry, G., Gough, B., & Conner, M. (2009). Smoking to stay thin or giving up to save face? Young men and women talk about appearance concerns and smoking. *Br J Health Psychol*, *14*(Pt 1), 175-186. doi: 10.1348/135910708x327617
- Guha, N., Merletti, F., Steenland, N. K., Altieri, A., Cogliano, V., & Straif, K. (2011). Lung cancer risk in painters: A meta-analysis. *Cien Saude Colet*, 16(8), 3613-3632. doi: 10.1289/ehp.0901402
- Gustavsson, P., Nyberg, F., Pershagen, G., Scheele, P., Jakobsson, R., & Plato, N. (2002). Low-dose exposure to asbestos and lung cancer: dose-response relations and interaction with smoking in a population-based case-referent study in Stockholm, Sweden. *Am J Epidemiol*, *155*(11), 1016-1022. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12034580
- Hackshaw, A. K., Law, M. R., & Wald, N. J. (1997). The accumulated evidence on lung cancer and environmental tobacco smoke. *BMJ*, 315(7114), 980-988. doi: http://dx.doi.org/10.1136/bmj.315.7114.980
- Hackshaw, L., Bauld, L., & McEwen, A. (2012). Stop smoking service clients' views following the introduction of smoke-free legislation in england. *Journal of Smoking Cessation*, 7(1), 47-54. doi: 10.1017/jsc.2012.4
- Hagimoto, A., Nakamura, M., Morita, T., Masui, S., & Oshima, A. (2010). Smoking cessation patterns and predictors of quitting smoking among the Japanese general population: a 1-year follow-up study. *Addiction*, *105*(1), 164-173. doi: 10.1111/j.1360-0443.2009.02735.x
- Hahn, A., & Renner, B. (1998). Perception of health risks: How smoker status affects defensive optimism. *Anxiety, Stress and Coping, 11*(2), 93-112. doi: 10.1080/10615809808248307

- Hahn, E. J., Rayens, M. K., Hopenhayn, C., & Christian, W. J. (2006). Perceived risk and interest in screening for lung cancer among current and former smokers. *Res Nurs Health*, 29(4), 359-370. doi: 10.1002/nur.20132
- Halpern-Felsher, B. L., Biehl, M., Kropp, R. Y., & Rubinstein, M. L. (2004). Perceived risks and benefits of smoking: Differences among adolescents with different smoking experiences and intentions. *Preventive Medicine*, *39*, 559-567. doi: 10.1016/j.ypmed.2004.02.017
- Halpern, M. T. (1994). Effect of smoking characteristics on cognitive dissonance in current and former smokers. *Addictive Behaviors*, 19(2), 209-217. doi: http://www.ncbi.nlm.nih.gov/pubmed/8036967
- Hammond, D., Fong, G. T., McDonald, P. W., Brown, K. S., & Cameron, R. (2004). Graphic Canadian cigarette warning labels and adverse outcomes: Evidence from Canadian smokers. *Am J Public Health*, *94*(8), 1442-1445. doi: http://www.ncbi.nlm.nih.gov/pubmed/15284057
- Hamra, G. B., Guha, N., Cohen, A., Laden, F., Raaschou-Nielsen, O., Samet, J. M., . . . Loomis, D. (2014). Outdoor particulate matter exposure and lung cancer: A systematic review and meta-analysis. *Environ Health Perspect*, 122(9), 906-911. doi: 10.1289/ehp.1408092
- Hanson, B. S., Isacsson, S.-O., Janzon, L., & Lindell, S.-E. (1990). Social support and quitting smoking for good. Is there an association? Results from the population study, "Men born in 1914," Malmö, Sweden. *Addictive Behaviors*, *15*(3), 221-233. doi: http://dx.doi.org/10.1016/0306-4603(90)90065-6
- Hargreaves, K., Amos, A., Highet, G., Martin, C., Platt, S., Ritchie, D., & White, M. (2010). The social context of change in tobacco consumption following the introduction of 'smokefree' England legislation: A qualitative, longitudinal study. *Soc Sci Med*, 71(3), 459-466. doi: 10.1016/j.socscimed.2010.04.025
- Harris, K. J., Okuyemi, K. S., Catley, D., Mayo, M. S., Ge, B., & Ahluwalia, J. S. (2004). Predictors of smoking cessation among African-Americans enrolled in a randomized controlled trial of bupropion. *Prev Med*, *38*(4), 498-502. doi: 10.1016/j.ypmed.2003.12.008
- Harrison, J. A., Mullen, P. D., Green, L. W. (1992). A meta-analysis of studies of the Health Belief Model with adults. *Health Education Research*, 7(1), 107-116. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10148735
- Haug, S., Schaub, M. P., & Schmid, H. (2014). Smoking Cessation: Predictors of adolescent smoking cessation and smoking reduction. *Patient Educ Couns*, 95, 378-383. doi: 10.1016/j.pec.2014.03.004
- Hayes, R. B., & Borrelli, B. (2013). Differences between Latino daily light and heavier smokers in smoking attitudes, risk perceptions, and smoking cessation outcome. *Nicotine Tob Res*, *15*(1), 103-111. doi: 10.1093/ntr/nts095
- Hays, J. T., & Ebbert, J. O. (2008). Varenicline for tobacco dependence. *N Engl J Med*, *359*(19), 2018-2024. doi: 10.1056/NEJMct0800146
- Health and Social Care Information Centre. (2014a). *Smoking, drinking and drug use among young people in England in 2013*. London: Health and Social Care Information Centre. Retrieved from http://www.hscic.gov.uk/catalogue/PUB14579.
- Health and Social Care Information Centre. (2014b). Statistics on smoking: England 2014. Retrieved from http://www.hscic.gov.uk/catalogue/PUB14988/smokeng-2014-rep.pdf

- Health Protection Agency. (2012). Radon. Retrieved from http://www.hpa.org.uk/Topics/Radiation/UnderstandingRadiation/UnderstandingRadiationTopics/Radon/
- Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerstrom, K. O. (1991). The Fagerstrom Test for Nicotine Dependence: a revision of the Fagerstrom Tolerance Questionnaire. *Br J Addict*, 86(9), 1119-1127. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1932883
- Hecht, S. S. (1999). Tobacco smoke carcinogens and lung cancer. *J Natl Cancer Inst,* 91(14), 1194-1210. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10413421
- Hecht, S. S. (2003). Tobacco carcinogens, their biomarkers and tobacco-induced cancer. *Nat Rev Cancer*, *3*(10), 733-744. doi: 10.1038/nrc1190
- Heffner, J. L., Barrett, S. W., & Anthenelli, R. M. (2007). Predicting alcohol misusers' readiness and ability to quit smoking: A critical review. *Alcohol and Alcoholism*, 42(3), 186-195. doi: http://www.ncbi.nlm.nih.gov/pubmed/17526628
- Heishman, S. J., Kleykamp, B. A., & Singleton, E. G. (2010). Meta-analysis of the acute effects of nicotine and smoking on human performance. *Psychopharmacology*, 210(4), 453-469. doi: 10.1007/s00213-010-1848-1
- Heist, R. S. (2015). Small cell lung cancer. BMJ Best Practice. Retrieved from http://bestpractice.bmj.com/best-practice/monograph/1081.html Accessed 5 March 2016.
- Hellman, R., Cummings, K. M., Haughey, B. P., Zielezny, M. A., & O'Shea, R. M. (1991). Predictors of attempting and succeeding at smoking cessation. *Health Education Research*, *6*(1), 77-86. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10148727
- Henderson, H. J., Memon, A., Lawson, K., Jacobs, B., & Koutsogeorgou, E. (2011). What factors are important in smoking cessation amongst deprived communities? A qualitative study. *Health Education Journal*, 70(1), 84-91. doi: 10.1177/0017896910373170
- Hendricks, P. S., Delucchi, K. L., & Hall, S. M. (2010). Mechanisms of change in extended cognitive behavioral treatment for tobacco dependence. *Drug Alcohol Depend*, 109(1-3), 114-119. doi: 10.1016/j.drugalcdep.2009.12.021
- Hertel, A. W., & Mermelstein, R. J. (2012). Smoker Identity and Smoking Escalation Among Adolescents. *Health Psychol*, 31(4), 467-475. doi: 10.1037/a0028923
- Hettema, J. E., & Hendricks, P. S. (2010). Motivational Interviewing for Smoking Cessation: A Meta-Analytic Review. *J Consult Clin Psychol*, 78(6), 868-884. doi: 10.1037/a0021498
- Hill, S., Spink, J., Cadilhac, D., Edwards, A., Kaufman, C., Rogers, S., . . . Tonkin, A. (2010). Absolute risk representation in cardiovascular disease prevention: comprehension and preferences of health care consumers and general practitioners involved in a focus group study. *BMC Public Health*, 10, 108. doi: 10.1186/1471-2458-10-108
- Hippisley-Cox, J., & Coupland, C. (2011). Identifying patients with suspected lung cancer in primary care: Derivation and validation of an algorithm. *Br J Gen Pract*, 61(592), e715-723. doi: 10.3399/bjgp11X606627
- Hirayama, T. (1981). Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. *Br Med J (Clin Res Ed)*, 282(6259), 183-185. doi: http://www.ncbi.nlm.nih.gov/pubmed/6779940

- Hiscock, R., Bauld, L., Amos, A., Fidler, J. A., & Munafo, M. (2012). Socioeconomic status and smoking: a review. *Ann N Y Acad Sci*, 1248, 107-123. doi: 10.1111/j.1749-6632.2011.06202.x
- Hiscock, R., Murray, S., Brose, L. S., McEwen, A., Bee, J. L., Dobbie, F., & Bauld, L. (2013). Behavioural therapy for smoking cessation: The effectiveness of different intervention types for disadvantaged and affluent smokers. *Addictive Behaviors*, 38(11), 2787-2796. doi: 10.1016/j.addbeh.2013.07.010
- Hitsman, B., Papandonatos, G. D., McChargue, D. E., Demott, A., Herrera, M. J., Spring, B., . . . Niaura, R. (2013). Past major depression and smoking cessation outcome: A systematic review and meta-analysis update. *Addiction*, 108(2), 294-306. doi: 10.1111/add.12009
- Hoffman, B. R., Sussman, S., Unger, J. B., & Valente, T. W. (2006). Peer influences on adolescent cigarette smoking: A theoretical review of the literature. *Subst Use Misuse*, *41*(1), 103-155. doi: 10.1080/10826080500368892
- Ho, R. (1992). Cigarette health warnings: The effects of perceived severity, expectancy of occurrence, and self-efficacy on intentions to give up smoking. *Australian Psychologist*, 27(2), 109-113. doi:10.1080/00050069208257590
- Hoffmann, D., Djordjevic, M. V., & Hoffmann, I. (1997). The changing cigarette. *Prev Med*, 26(4), 427-434. doi: 10.1006/pmed.1997.0183
- Hoggart, C., Brennan, P., Tjonneland, A., Vogel, U., Overvad, K., Ostergaard, J. N., .
 . . Vineis, P. (2012). A Risk Model for Lung Cancer Incidence. *Cancer Prev Res (Phila)*. doi: 10.1158/1940-6207.capr-11-0237
- Honda, K., & Neugut, A. I. (2004). Associations between perceived cancer risk and established risk factors in a national community sample. *Cancer Detect Prev*, 28(1), 1-7. doi: 10.1016/j.cdp.2003.12.001
- Hopkinson, N. S., Lester-George, A., Ormiston-Smith, N., Cox, A., & Arnott, D. (2014). Child uptake of smoking by area across the UK. *Thorax*, 69(9), 873-875. doi: 10.1136/thoraxjnl-2013-204379
- Horwitz, M. B., Hindi-Alexander, M., & Wagner, T. J. (1985). Psychosocial mediators of abstinence, relapse, and continued smoking: A one-year follow-up of a minimal intervention. *Addict Behav*, *10*(1), 29-39. doi: http://www.ncbi.nlm.nih.gov/pubmed/4003135
- Hosgood, H. D., 3rd, Wei, H., Sapkota, A., Choudhury, I., Bruce, N., Smith, K. R., . . . Lan, Q. (2011). Household coal use and lung cancer: systematic review and meta-analysis of case-control studies, with an emphasis on geographic variation. *Int J Epidemiol*, 40(3), 719-728. doi: 10.1093/ije/dyq259
- Hosmer, D. W., Lemeshow, S., & Sturdivant, R. X. (2013). *Applied logistic regression* (5th ed.). Hoboken: Wiley.
- Hsieh, C. J., Chen, S. H., Lee, J. M., & Yeh, C. Y. (2014). Is there the threshold effect of cigarette price on smoking prevalence? A cross-country panel data analysis. *Applied Economics*, 46(21), 2534-2544. doi: 10.1080/00036846.2014.904494
- Hughes, J. R. (2007). Effects of abstinence from tobacco: valid symptoms and time course. *Nicotine and Tobacco Research*, *9*(3), 315–327. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17365764
- Hughes, J. R. (2008). Smoking and suicide: A brief overview. *Drug and Alcohol Dependence*, 98(3), 169-178. doi: 10.1016/j.drugalcdep.2008.06.003
- Hughes, J. R. (2015). Varenicline as a Cause of Suicidal Outcomes. *Nicotine Tob Res.* doi: 10.1093/ntr/ntu275

- Hughes, J. R., & Callas, P. W. (2011). Is delaying a quit attempt associated with less success? *Nicotine Tob Res*, 13(12), 1228-1232. doi: 10.1093/ntr/ntr207
- Hughes, J. R., Carpenter, M. J., & Naud, S. (2010). Do point prevalence and prolonged abstinence measures produce similar results in smoking cessation studies? A systematic review. *Nicotine Tob Res*, 12(7), 756-762. doi: 10.1093/ntr/ntq078
- Hughes, J. R., & Hatsukami, D. (1986). Signs and symptoms of tobacco withdrawal. *Arch Gen Psychiatry*, 43(3), 289-294. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3954551
- Hughes, J. R., & Kalman, D. (2006). Review: Do smokers with alcohol problems have more difficulty quitting? *Drug and Alcohol Dependence*, 82, 91-102. doi: 10.1016/j.drugalcdep.2005.08.018
- Hughes, J. R., Stead, L. F., Hartmann-Boyce, J., Cahill, K., & Lancaster, T. (2014). Antidepressants for smoking cessation. *Cochrane Database Syst Rev, 1*, Cd000031. doi: 10.1002/14651858.CD000031.pub4
- Humphrey, L. L., Teutsch, S., & Johnson, M. (2004). Lung cancer screening with sputum cytologic examination, chest radiography, and computed tomography: An update for the U.S. Preventive Services Task Force. *Ann Intern Med, 140*(9), 740-753. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15126259
- Hymowitz, N., Cummings, K. M., Hyland, A., Lynn, W. R., Pechacek, T. F., & Hartwell, T. D. (1997). Predictors of smoking cessation in a cohort of adult smokers followed for five years. *Tobacco Control*, *6*(S2), S57. doi: 10.1136/tc.6.suppl_2.S57
- Hymowitz, N., Sexton, M., Ockene, J., & Grandits, G. (1991). Baseline factors associated with smoking cessation and relapse. MRFIT Research Group. *Prev Med*, 20(5), 590-601. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1758840
- Hysert, P., Mirand, A., Giovino, G., Cummings, K., & Kuo, C. (2003). "At Face Value": Age progression software provides personalised demonstration of the effects of smoking on appearance. *Tobacco Control*, *12*(2), 238-238. doi: 10.1136/tc.12.2.238
- Ibrahim, E. M., Kazkaz, G. A., Abouelkhair, K. M., Al-Mansour, M. M., Al-Fayea, T. M., Al-Foheidi, M., . . . Elmasri, O. A. (2013). Increased risk of second lung cancer in Hodgkin's lymphoma survivors: a meta-analysis. *Lung*, *191*(1), 117-134. doi: 10.1007/s00408-012-9418-4
- International Agency for Research on Cancer (IARC). (2004). Tobacco smoking and involuntary smoking. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans, 83. Retrieved from http://monographs.iarc.fr/ENG/Monographs/vol83/index.php
- International Agency for Research on Cancer (IARC). (2012). IARC: Diesel engine exhaust carcinogenic. Retrieved from http://www.iarc.fr/en/mediacentre/pr/2012/pdfs/pr213_E.pdf
- Irizar-Aramburu, M. I., Martínez-Eizaguirre, J. M., Pacheco-Bravo, P., Diaz-Atienza, M., Galparsoro-Goikoetxea, M., Aguirre-Arratibel, I., . . . Alba-Latorre, M. (2013). Effectiveness of spirometry as a motivational tool for smoking cessation: A clinical trial, the ESPIMOAT study. *BMC Family Practice*, 14. doi: 10.1186/1471-2296-14-185
- Irvine, A., Drew, P., & Sainsbury, R. (2013). 'Am I not answering your questions properly?' Clarification, adequacy and responsiveness in semi-structured

- telephone and face-to-face interviews. *Qualitative Research*, *13*(1), 87-106. doi: 10.1177/1468794112439086
- Iyen-Omofoman, B., Tata, L. J., Baldwin, D. R., Smith, C. J., & Hubbard, R. B. (2013). Using socio-demographic and early clinical features in general practice to identify people with lung cancer earlier. *Thorax*, 68(5), 451-459. doi: 10.1136/thoraxjnl-2012-202348
- Jacobson, J. D., Catley, D., Lee, H. S., Harrar, S. W., & Harris, K. J. (2014). Health risk perceptions predict smoking-related outcomes in Greek college students. *Psychology of Addictive Behaviors*, 28(3), 743-751. doi: 10.1037/a0037444
- Jamal, M., Ameno, K., Tanaka, N., Kumihashi, H., & Kinoshita, H. (2012). Nicotine addiction and treatment: Recent advances. In G. Di Giovanni (Ed.), *Nicotine addiction, prevention, health effects and treatment options*. N.Y.: Nova Science Publishers, Inc.
- Jamieson, P., & Romer, D. (2001a). The role of perceived risk in starting and stopping smoking. In P. Slovic (Ed.), *Smoking: Risk, perception, & policy*. Thousand Oaks, California: Sage Publications Inc.
- Jamieson, P., & Romer, D. (2001b). What do young people think they know about smoking? In P. Slovic (Ed.), *Smoking: risk, perception, & policy*. Thousand Oaks, California: Sage Publications Inc.
- Janssens, A. C., Aulchenko, Y. S., Elefante, S., Borsboom, G. J., Steyerberg, E. W., & van Duijn, C. M. (2006). Predictive testing for complex diseases using multiple genes: Fact or fiction? *Genet Med*, 8(7), 395-400. doi: 10.109701.gim.0000229689.18263.f4
- Jarvholm, B., & Silverman, D. (2003). Lung cancer in heavy equipment operators and truck drivers with diesel exhaust exposure in the construction industry. *Occup Environ Med*, 60(7), 516-520. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12819286
- Jemal, A., Travis, W. D., Tarone, R. E., Travis, L., & Devesa, S. S. (2003). Lung cancer rates convergence in young men and women in the United States: Analysis by birth cohort and histologic type. *Int J Cancer*, *105*(1), 101-107. doi: 10.1002/ijc.11020
- Jenks, R. J. (1992). Attitudes, perceptions, and risk-taking behaviors of smokers, exsmokers, and nonsmokers. *Journal of Social Psychology*, *132*(5), 569-575. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1453690
- Johnson, R. B., Onwuegbuzie, A. J., & Turner, L. A. (2007). Toward a definition of mixed methods research. *Journal of Mixed Methods Research*, 1(2), 112-133. doi: 10.1177/1558689806298224
- Jones, A., Abbott, D., & Quilgars, D. (2006). Social inequality and risk. In P. Taylor-Gooby & J. Zinn (Eds.), *Risk in social science* (pp. 228-249). Oxford: Oxford University Press.
- Jones, S. E., & Hamilton, S. (2013). Introducing a new stop smoking service in an acute UK hospital: A qualitative study to evaluate service user experience. *European Journal of Oncology Nursing*, 17, 563-569. doi: 10.1016/j.ejon.2013.01.011
- Joossens, L., & Raw, M. (2007). Progress in tobacco control policies in 30 European countries, 2005 to 2007. Retrieved from http://www.europeancancerleagues.org/images/stories/pdf/290_30_european _countries_text_final.pdf

- Judge, K., Bauld, L., Chesterman, J., & Ferguson, J. (2005). The English smoking treatment services: short-term outcomes. *Addiction*, 100(S2), 46-58. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15844289
- Kammin, T., Fenton, A. K., & Thirlaway, K. (2014). A genetic lung cancer susceptibility test may have a positive effect on smoking cessation. *Journal of Genetic Counseling*. doi: 10.1007/s10897-014-9766-8
- Kannel, W. B., McGee, D., & Gordon, T. (1976). A general cardiovascular risk profile: the Framingham Study. *Am J Cardiol*, 38(1), 46-51. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/132862
- Katz, R. C., & Singh, N. N. (1986). Reflections on the ex-smoker: Some findings on successful quitters. *J Behav Med*, 9(2), 191. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3712429
- Kelley, K., Clark, B., Brown, V., & Sitzia, J. (2003). Good practice in the conduct and reporting of survey research. *International Journal for Quality in Health Care*, 15(3), 261-266. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12803354
- Kelly, A. C., Zuroff, D. C., Foa, C. L., & Gilbert, P. (2010). Who benefits from training in self-compassionate self-regulation? A study of smoking reduction. *Journal* of Social & Clinical Psychology, 29(7), 727-755. doi: 10.1521/jscp.2010.29.7.727
- Kenfield, S. A., Stampfer, M. J., Rosner, B. A., & Colditz, G. A. (2008). Smoking and smoking cessation in relation to mortality in women. *JAMA Journal of the American Medical Association*, 299(17), 2037-2047. doi: 10.1001/jama.299.17.2037
- Khuder, S. A. (2001). Effect of cigarette smoking on major histological types of lung cancer: A meta-analysis. *Lung Cancer*, 31(2-3), 139-148.
- Khuder, S. A., Dayal, H. H., & Mutgi, A. B. (1999). Age at smoking onset and its effect on smoking cessation. *Addictive Behaviors*, 24(5), 673-677. doi: http://dx.doi.org/10.1016/S0306-4603(98)00113-0
- Killen, J. D., Fortmann, S. P., Kraemer, H. C., Varady, A., & Newman, B. (1992). Who will relapse? Symptoms of nicotine dependence predict long-term relapse after smoking cessation. *J Consult Clin Psychol*, 60(5), 797-801. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1401396
- Kirchner, T. R., & Sayette, M. A. (2007). Effects of smoking abstinence and alcohol consumption on smoking-related outcome expectancies in heavy smokers and tobacco chippers. *Nicotine Tob Res*, *9*(3), 365-376. doi: 10.1080/14622200701188893
- Kleinjan, M., van den Eijnden, R. J. J. M., Dijkstra, A., Brug, J., & Engels, R. C. M. E. (2006). Excuses to continue smoking: The role of disengagement beliefs in smoking cessation. *Addictive Behaviors*, 31(12), 2223-2237. doi: http://dx.doi.org/10.1016/j.addbeh.2006.02.012
- Kleinjan, M., van den Eijnden, R. J. J. M., & Engels, R. C. M. E. (2009). Adolescents' rationalizations to continue smoking: The role of disengagement beliefs and nicotine dependence in smoking cessation. *Addictive Behaviors*, *34*, 440-445. doi: 10.1016/j.addbeh.2008.12.010
- Ko, G. T., Chan, J. C., Tsang, L. W., Critchley, J. A., & Cockram, C. S. (2001). Smoking and diabetes in Chinese men. *Postgrad Med J*, 77(906), 240-243. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11264486
- Kodl, M., Fu, S. S., & Joseph, A. M. (2006). Tobacco cessation treatment for alcohol-dependent smokers: when is the best time? *Alcohol Research & Health*, 29(3),

- 203-207. Retrieved from http://pubs.niaaa.nih.gov/publications/arh293/203-207.pdf
- Kogevinas, M., Sala, M., Boffetta, P., Kazerouni, N., Kromhout, H., & Hoar-Zahm, S. (1998). Cancer risk in the rubber industry: a review of the recent epidemiological evidence. *Occup Environ Med*, *55*(1), 1-12. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9536156
- Koivisto-Korander, R., Scelo, G., Ferro, G., Mellemkjaer, L., Hemminki, K., Weiderpass, E., . . . Pukkala, E. (2012). Second primary malignancies among women with uterine sarcoma. *Gynecol Oncol*, *126*(1), 30-35. doi: 10.1016/j.ygyno.2012.04.002
- Kong, G., Ells, D. M., Camenga, D. R., & Krishnan-Sarin, S. (2014). Text messaging-based smoking cessation intervention: A narrative review. *Addictive Behaviors*, 39, 907-917. doi: 10.1016/j.addbeh.2013.11.024
- Kotz, D., & West, R. (2009). Explaining the social gradient in smoking cessation: It's not in the trying, but in the succeeding. *Tobacco Control*, 18(1), 43-46. doi: 10.1136/tc.2008.025981
- Kreuzer, M., Boffetta, P., Whitley, E., Ahrens, W., Gaborieau, V., Heinrich, J., . . . Simonato, L. (2000). Gender differences in lung cancer risk by smoking: A multicentre case-control study in Germany and Italy. *Br J Cancer*, 82(1), 227-233. doi: 10.1054/bjoc.1999.0904
- Kurihara, N., & Wada, O. (2004). Silicosis and smoking strongly increase lung cancer risk in silica-exposed workers. *Ind Health*, 42(3), 303-314. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15295901
- Kurmi, O. P., Arya, P. H., Lam, K. B., Sorahan, T., & Ayres, J. G. (2012). Lung cancer risk and solid fuel smoke exposure: A systematic review and meta-analysis. *Eur Respir J*, 40(5), 1228-1237. doi: 10.1183/09031936.00099511
- Lacasse, Y., Martin, S., Gagne, D., & Lakhal, L. (2009). Dose-response meta-analysis of silica and lung cancer. *Cancer Causes Control*, 20(6), 925-933. doi: 10.1007/s10552-009-9296-0
- Lader, D. (2009). Opinions Survey Report No. 40 Smoking-related behaviour and attitudes, 2008/09. Retrieved from http://www.ons.gov.uk/ons/rel/lifestyles/smoking-related-behaviour-and-attitudes/2008-09/index.html
- Lakier, J. B. (1992). Smoking and cardiovascular disease. *Am J Med*, *93*(1S), 8S-12S. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1497005
- Lancaster, T., & Stead, L. F. (2005). Individual behavioural counselling for smoking cessation. *Cochrane Database Syst Rev*(2), Cd001292. doi: 10.1002/14651858.CD001292.pub2
- Lando, H. A., Thai, D. T., Murray, D. M., Robinson, L. A., Jeffery, R. W., Sherwood, N. E., & Hennrikus, D. J. (1999). Age of initiation, smoking patterns, and risk in a population of working adults. *Preventive Medicine*, *29*(6), 590-598. doi: http://dx.doi.org/10.1006/pmed.1999.0590
- Lapinski, M. K., & Rimal, R. N. (2005). An explication of social norms. *Communication Theory*, 15(2), 127-147. doi: 10.1111/j.1468-2885.2005.tb00329.x
- Larkin, E. K., Smith, T. J., Stayner, L., Rosner, B., Speizer, F. E., & Garshick, E. (2000). Diesel exhaust exposure and lung cancer: Adjustment for the effect of smoking in a retrospective cohort study. *Am J Ind Med*, *38*(4), 399-409. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10982980

- Lavino, J. G., & Neumann, R. B. (2010). *Psychology of risk perception*. New York: Nova Science Publishers.
- Lee, C. (1989). Perceptions of immunity to disease in adult smokers. *J Behav Med*, *12*(3), 267-277. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/2634103
- Lee, C., & Kahende, J. (2007). Factors associated with successful smoking cessation in the United States, 2000. *Am J Public Health*, 97(8), 1503-1509. doi: 10.2105/AJPH.2005.083527
- Lee, P. N. (2001). Lung cancer and type of cigarette smoked. *Inhal Toxicol*, *13*(11), 951-976. doi: 10.1080/089583701753210353
- Lee, P. N. (2001). Relation between exposure to asbestos and smoking jointly and the risk of lung cancer. *Occup Environ Med*, 58(3), 145-153.
- Lee, P. N., Foley, B. A., & Coombs, K. J. (2012). Systematic review with metaanalysis of the epidemiological evidence in the 1900s relating smoking to lung cancer. *BMC Cancer*, 12(1), 385-474. doi: 10.1186/1471-2407-12-385
- Leeman, R. F., Huffman, C. J., & O'Malley, S. S. (2007). Alcohol history and smoking cessation in nicotine replacement therapy, bupropion sustained release and varenicline trials: A review. *Alcohol and Alcoholism*, 42(3), 196-206. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17526629
- Lenters, V., Vermeulen, R., Dogger, S., Stayner, L., Portengen, L., Burdorf, A., & Heederik, D. (2011). A meta-analysis of asbestos and lung cancer: is better quality exposure assessment associated with steeper slopes of the exposure-response relationships? *Environ Health Perspect*, 119(11), 1547-1555. doi: 10.1289/ehp.1002879
- Leonardi-Bee, J., Jere, M. L., & Britton, J. (2011). Exposure to parental and sibling smoking and the risk of smoking uptake in childhood and adolescence: A systematic review and meta-analysis. *Thorax*, 66(10), 847-855. doi: 10.1136/thx.2010.153379
- Leuraud, K., Schnelzer, M., Tomasek, L., Hunter, N., Timarche, M., Grosche, B., . . . Laurier, D. (2011). Radon, smoking and lung cancer risk: results of a joint analysis of three European case-control studies among uranium miners. *Radiat Res*, 176(3), 375-387. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/21714633
- Leventhal, H. (1970). Findings and theory in the study of fear communications. In L. Berkowitz (Ed.), *Advances in experimental social psychology: Volume 5.* (pp. 119-186). New York: Academic Press.
- Leventhal, A. M., & Zvolensky, M. J. (2015). Anxiety, depression, and cigarette smoking: A transdiagnostic vulnerability framework to understanding emotion–smoking comorbidity. *Psychol Bull, 141*(1), 176-212. doi: 10.1037/bul0000003
- Levy, D. T., Chaloupka, F., & Gitchell, J. (2004). The effects of tobacco control policies on smoking rates: a tobacco control scorecard. *J Public Health Manag Pract*, 10(4), 338-353. doi: http://www.ncbi.nlm.nih.gov/pubmed/15235381
- Leyton, M., Boileau, I., Benkelfat, C., Diksic, M., Baker, G., & Dagher, A. (2002). Amphetamine-induced increases in extracellular dopamine, drug wanting, and novelty seeking: a PET/[11C]raclopride study in healthy men. *Neuropsychopharmacology*, 27(6), 1027-1035. doi: 10.1016/S0893-133X(02)00366-4
- Li, L., Borland, R., Yong, H., Fong, G. T., Quah, A. C. K., Zanna, M. P., . . . Omar, M. (2010). Predictors of smoking cessation among adult smokers in Malaysia

- and Thailand: Findings from the International Tobacco Control Southeast Asia survey. *Nicotine and Tobacco Research*, *12*(S1), S34-S44. doi: 10.1093/ntr/ntq030
- Li, L., Feng, G., Jiang, Y., Yong, H., Borland, R., & Fong, G. T. (2011). Prospective predictors of quitting behaviours among adult smokers in six cities in China: Findings from the International Tobacco Control (ITC) China Survey. *Addiction*, 106(7), 1335-1345. doi: 10.1111/j.1360-0443.2011.03444.x
- Lin, H. H., Murray, M., Cohen, T., Colijn, C., & Ezzati, M. (2008). Effects of smoking and solid-fuel use on COPD, lung cancer, and tuberculosis in China: a time-based, multiple risk factor, modelling study. *Lancet*, *372*(9648), 1473-1483. doi: 10.1016/S0140-6736(08)61345-8
- Lindbladh, E., & Lyttkens, C. H. (2003). Polarization in the reaction to health-risk information: A question of social position? *Risk analysis: An official publication of the society for risk analysis, 23*(4), 841-855. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12926576
- Link, B. G., & Phelan, J. C. (2001). Conceptualizing stigma. *Annual Review of Sociology*, (27), 363-385. doi: 10.1146/annurev.soc.27.1.363
- Lipkus, I. M. (2007). Numeric, verbal, and visual formats of conveying health risks: Suggested best practices and future recommendations. *Medical Decision Making*, 27(5), 696-713. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17873259
- Lipkus, I. M., Barefoot, J. C., Feaganes, J., Williams, R. B., & Siegler, I. C. (1994). A short MMPI scale to identify people likely to begin smoking. *J Pers Assess*, 62(2), 213-222. doi: 10.1207/s15327752jpa6202_4
- Lipkus, I. M., & Hollands, J. G. (1999). The visual communication of risk. *JNCI: Journal of the National Cancer Institute*(25), 149-163. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10854471
- Lipkus, I. M., Samsa, G., & Rimer, B. K. (2001). General performance on a numeracy scale among highly educated samples. *Medical Decision Making*, 21(1), 37-44. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11206945
- Lissowska, J., Bardin-Mikolajczak, A., Fletcher, T., Zaridze, D., Szeszenia-Dabrowska, N., Rudnai, P., . . . Boffetta, P. (2005). Lung cancer and indoor pollution from heating and cooking with solid fuels: The IARC international multicentre case-control study in Eastern/Central Europe and the United Kingdom. *Am J Epidemiol*, 162(4), 326-333. doi: 10.1093/aje/kwi204
- Littell, J. H., & Girvin, H. (2002). Stages of change: A critique. *Behavior Modification*, 26(2), 223-273. doi: 10.1177/0145445502026002006
- Liverpool City Council. (2011). The Index of Multiple Deprivation 2010: A Liverpool Analysis. Retrieved from http://liverpool.gov.uk/media/129441/Full-Report-2010.pdf
- Lock, K., Adams, E., Pilkington, P., Duckett, K., Gilmore, A., & Marston, C. (2010). Evaluating social and behavioural impacts of English smoke-free legislation in different ethnic and age groups: Implications for reducing smoking-related health inequalities. *Tobacco Control*, 19(5), 391-397. doi: 10.1136/tc.2009.032318
- Loomis, D., Dement, J. M., Elliott, L., Richardson, D., Kuempel, E. D., & Stayner, L. (2012). Increased lung cancer mortality among chrysotile asbestos textile workers is more strongly associated with exposure to long thin fibres. *Occup Environ Med*, 69(8), 564-568. doi: 10.1136/oemed-2012-100676

- Loon, A. J. M. v., Tijhuis, M., Picavet, H. S. J., Surtees, P. G., & Ormel, J. (2003). Survey non-response in the Netherlands: Effects on prevalence estimates and associations. *Annals of Epidemiology*, *13*(2), 105-110. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12559669
- Lorigan, P., Califano, R., Faivre-Finn, C., Howell, A., & Thatcher, N. (2010). Lung cancer after treatment for breast cancer. *Lancet Oncol*, 11(12), 1184-1192. doi: 10.1016/s1470-2045(10)70056-5
- Lorigan, P., Radford, J., Howell, A., & Thatcher, N. (2005). Lung cancer after treatment for Hodgkin's lymphoma: A systematic review. *Lancet Oncol*, 6(10), 773-779. doi: 10.1016/S1470-2045(05)70387-9
- Lortet-Tieulent, J., Soerjomataram, I., Ferlay, J., Rutherford, M., Weiderpass, E., & Bray, F. (2014). International trends in lung cancer incidence by histological subtype: Adenocarcinoma stabilizing in men but still increasing in women. *Lung Cancer*, 84(1), 13-22. doi: 10.1016/j.lungcan.2014.01.009
- Lovato, C., Linn, G., Stead, L. F., & Best, A. (2003). Impact of tobacco advertising and promotion on increasing adolescent smoking behaviours. *Cochrane Database Syst Rev*(4), CD003439. doi: 10.1002/14651858.CD003439
- Lubin, J. H., & Blot, W. J. (1984). Assessment of lung cancer risk factors by histologic category. *J Natl Cancer Inst*, 73(2), 383-389. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/6087006
- Lubin, J. H., & Steindorf, K. (1995). Cigarette use and the estimation of lung cancer attributable to radon in the United States. *Radiat Res*, 141(1), 79-85. doi: 10.2307/3579093
- Lupton, D. (1999). Risk. London: Routledge.
- Lupton, D., & Tulloch, J. (2002). 'Life would be pretty dull without risk': voluntary risk-taking and its pleasures. *Health, Risk & Society, 4*(2), 113-124. doi: 10.1080/13698570220137015
- Lynge, E., Rix, B. A., Villadsen, E., Andersen, I., Hink, M., Olsen, E., . . . Silfverberg, E. (1995). Cancer in printing workers in Denmark. *Occup Environ Med*, 52(11), 738-744. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8535493
- Mackay, D. F., Haw, S., & Pell, J. P. (2011). Impact of Scottish smoke-free legislation on smoking quit attempts and prevalence. *PLoS One*, *6*(11), e26188. doi: 10.1371/journal.pone.0026188
- MacKay, D. G. (1992). Constraints on theories of inner speech. In D. Reisberg (Ed.), *Auditory imagery* (pp. 121-149). Hillsdale, NJ: Erlbaum.
- Magnan, R. E., Koblitz, A. R., Zielke, D. J., & McCaul, K. D. (2009). The effects of warning smokers on perceived risk, worry, and motivation to quit. *Annals of Behavioral Medicine*, *37*(1), 46-57. doi: 10.1007/s12160-009-9085-8
- Magnani, C., Ferrante, D., Barone-Adesi, F., Bertolotti, M., Todesco, A., Mirabelli, D., & Terracini, B. (2008). Cancer risk after cessation of asbestos exposure: A cohort study of Italian asbestos cement workers. *Occup Environ Med*, 65(3), 164-170. doi: 10.1136/oem.2007.032847
- Maisonneuve, P., Bagnardi, V., Bellomi, M., Spaggiari, L., Pelosi, G., Rampinelli, C., . . . Veronesi, G. (2011). Lung cancer risk prediction to select smokers for screening CT--a model based on the Italian COSMOS trial. *Cancer Prev Res* (*Phila*), 4(11), 1778-1789. doi: 10.1158/1940-6207.CAPR-11-0026
- Manson, J. E., Ajani, U. A., Liu, S., Nathan, D. M., & Hennekens, C. H. (2000). A prospective study of cigarette smoking and the incidence of diabetes mellitus

- among us male physicians. *Am J Med*, 109(7), 538-542. doi: http://dx.doi.org/10.1016/S0002-9343(00)00568-4
- Mantler, T. (2013). A systematic review of smoking Youths' perceptions of addiction and health risks associated with smoking: Utilizing the framework of the health belief model. *Addiction Research & Theory*, 21(4), 306-317. doi: 10.3109/16066359.2012.727505
- Marcus, A. C., Shopland, D. R., Crane, L. A., & Lynn, W. R. (1989). Prevalence of cigarette smoking in the United States: Estimates from the 1985 current population survey. *J Natl Cancer Inst*, 81(6), 409-414. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/2783978
- Marcus, M. W., Chen, Y., Raji, O. Y., Duffy, S. W., & Field, J. K. (2015). LLPi:Liverpool Lung Project risk prediction model for lung cancer incidence. *Cancer Prev Res (Phila)*, 8(6), 570-575. doi: 10.1158/1940-6207.capr-14
- Marlatt, G. A. & George, W. H. (1984). Relapse prevention: Introduction and overview of the model. *British Journal of Addiction*, 79(3), 261-273. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/6595020
- Marlatt, G. A. & Gordon, J. R. (1985). Relapse prevention: Maintenance strategies in the treatment of addictive behaviors. New York: Guilford Press.
- Masri, F. A., Comhair, S. A., Koeck, T., Xu, W., Janocha, A., Ghosh, S., . . . Aulak, K. S. (2005). Abnormalities in nitric oxide and its derivatives in lung cancer. *Am J Respir Crit Care Med*, 172(5), 597-605. doi: 10.1164/rccm.200411-1523OC
- Matakidou, A., Eisen, T., & Houlston, R. S. (2005). Systematic review of the relationship between family history and lung cancer risk. *Br J Cancer*, *93*(7), 825-833. doi: 10.1038/sj.bjc.6602769
- Mathie, A., & Carnozzi, A. (2005). Qualitative research for tobacco control: A howto introductory manual for researchers and development practitioners. Ottawa, Canada: IDRC Books.
- McBride, C. M., Curry, S. J., Grothaus, L. C., Nelson, J. C., Lando, H., & Pirie, P. L. (1998). Partner smoking status and pregnant smoker's perceptions of support for and likelihood of smoking cessation. *Health Psychology*, *17*(1), 63-69. doi: 10.1037/0278-6133.17.1.63
- McCaul, K. D., Hockemeyer, J. R., Johnson, R. J., Zetocha, K., Quinlan, K., & Glasgow, R. E. (2006). Motivation to quit using cigarettes: A review. *Addictive Behaviors*, 31(1), 42-56. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15916861
- McKenna, F. P. (1993). It won't happen to me: Unrealistic optimism or illusion of control? *British Journal of Psychology*, 84(1), 39. doi: 10.1111/j.2044-8295.1993.tb02461.x
- McKenna, F. P., Warburton, D. M., & Winwood, M. (1993). Exploring the limits of optimism: the case of smokers' decision making. *Br J Psychol*, *84* (*Pt 3*), 389-394. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8401990
- McMaster, C., & Lee, C. (1991). Brief report: Cognitive dissonance in tobacco smokers. *Addictive Behaviors*, 16, 349-353. doi: 10.1016/0306-4603(91)90028-G
- McRonald, F. E., Yadegarfar, G., Hands, C. J., Williamson, P. R., Field, J. K., Holemans, J. A., . . . Weller, D. (2014). The UK Lung Screen (UKLS): Demographic profile of first 88,897 approaches provides recommendations for

- population screening. *Cancer Prevention Research*, 7(3), 362-371. doi: 10.1158/1940-6207.CAPR-13-0206
- Medicines and Healthcare Products Regulator Agency [MHRA]. (2013). Nicotine Containing Products. Retrieved from http://www.webcitation.org/6I89OvSYh
- Meneses-Gaya, I. C. d., Zuardi, A. W., Loureiro, S. R., & Crippa, J. A. d. S. (2009). Psychometric properties of the Fagerström Test for Nicotine Dependence. *Jornal Brasileiro de Pneumologia*, 35(1), 73-82. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/19219334
- Merchant, G., Pulvers, K., Brooks, R. D., & Edwards, J. (2013). Coping with the urge to smoke: A real-time analysis. *Res Nurs Health*, 36(1), 3-15. doi: 10.1002/nur.21520
- Mermelstein, R., Lichtenstein, E., & McIntyre, K. (1983). Partner support and relapse in smoking-cessation programs. *J Consult Clin Psychol*, *51*(3), 465-466. doi: 10.1037/0022-006X.51.3.465
- Méry, B., Guy, J.-B., Swalduz, A., Vallard, A., Guibert, C., Almokhles, H., ... Magné, N. (2015). The evolving locally-advanced non-small cell lung cancer landscape: Building on past evidence and experience. *Critical Reviews in Oncology / Hematology*. doi: 10.1016/j.critrevonc.2015.05.020
- Mickey, R. M., & Greenland, S. (1989). A study of the impact of confounder selection criteria on effect estimation. *American Journal of Epidemiology*, 129(1), 125-137. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/2910056
- Mills, E. J., Wu, P., Lockhart, I., Thorlund, K., Puhan, M., & Ebbert, J. O. (2012). Comparisons of high-dose and combination nicotine replacement therapy, varenicline, and bupropion for smoking cessation: A systematic review and multiple treatment meta-analysis. *Ann Med*, 44(6), 588-597. doi: 10.3109/07853890.2012.705016
- Mimas. (2014). GeoConvert. Retrieved from http://geoconvert.mimas.ac.uk/index.htm
- Moher, D., Hopewell, S., Schulz, K. F., Montori, V., Gøtzsche, P. C., Devereaux, P. J., . . . Altman, D. G. (2010). CONSORT 2010 Explanation and Elaboration: updated guidelines for reporting parallel group randomised trials. *BMJ*: *British Medical Journal*, *340*, c869. doi: 10.1136/bmj.c869
- Monsó, E., Campbell, J., Tønnesen, P., Gustavsson, G., & Morera, J. (2001). Sociodemographic predictors of success in smoking intervention. *Tobacco Control*, 10(2), 165-169. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11387538
- Moolchan, E. T., Ernst, M., & Henningfield, J. E. (2000). A review of tobacco smoking in adolescents: Treatment implications. *J Am Acad Child Adolesc Psychiatry*, 39(6), 682-693. doi: 10.1097/00004583-200006000-00006
- Morrell, H. E. R., & Cohen, L. M. (2006). Cigarette smoking, anxiety, and depression. *Journal of Psychopathology and Behavioral Assessment*, 28(4), 283-297.
- Morse, J. M. (2012). *Qualitative health research: Creating a new discipline*. Walnut Creek, CA: Left Coast Press.
- Morton, L. M., Curtis, R. E., Linet, M. S., Bluhm, E. C., Tucker, M. A., Caporaso, N., . . . Fraumeni, J. F., Jr. (2010). Second malignancy risks after non-Hodgkin's lymphoma and chronic lymphocytic leukemia: Differences by lymphoma subtype. *J Clin Oncol*, 28(33), 4935-4944. doi: 10.1200/jco.2010.29.1112
- Mudie, N. Y., Swerdlow, A. J., Higgins, C. D., Smith, P., Qiao, Z., Hancock, B. W., . . . Linch, D. C. (2006). Risk of second malignancy after non-Hodgkin's

- lymphoma: A British Cohort Study. *J Clin Oncol*, 24(10), 1568-1574. doi: 10.1200/JCO.2005.04.2200
- Munafo, M., Clark, T., Johnstone, E., Murphy, M., & Walton, R. (2004). The genetic basis for smoking behavior: A systematic review and meta-analysis. *Nicotine Tob Res*, 6(4), 583-597. doi: 10.1080/14622200410001734030
- Munafo, M. R., & Johnstone, E. C. (2008). Genes and cigarette smoking. *Addiction*, 103(6), 893-904. doi: 10.1111/j.1360-0443.2007.02071.x
- Munshi, V., & McMahon, P. (2013). Importance of smoking cessation in a lung cancer screening program. *Curr Surg Rep, 1*(4). doi: 10.1007/s40137-013-0030-1
- Murray, R. L., Bauld, L., Hackshaw, L. E., & McNeill, A. (2009). Improving access to smoking cessation services for disadvantaged groups: A systematic review. *Journal of Public Health*, *31*(2), 258-277. doi: 10.1093/pubmed/fdp008
- Murray, R. L., McNeill, A., Lewis, S., Britton, J., & Coleman, T. (2010). Unplanned attempts to quit smoking: a qualitative exploration. *Addiction*, 105(7), 1299-1302. doi: 10.1111/j.1360-0443.2010.02980.x
- Murray, R. P., Gerald, L. B., Lindgren, P. G., Connett, J. E., Rand, C. S., & Anthonisen, N. R. (2000). Characteristics of participants who stop smoking and sustain abstinence for 1 and 5 years in the Lung Health Study. *Prev Med*, 30(5), 392-400. doi: 10.1006/pmed.2000.0642
- Nagelhout, G. E., de Vries, H., Boudreau, C., Allwright, S., McNeill, A., van den Putte, B., . . . Willemsen, M. C. (2012). Comparative impact of smoke-free legislation on smoking cessation in three European countries. *European Journal of Public Health*, 22(S1), 4-9. doi: 10.1093/eurpub/ckr203
- National Cancer Institute. (2014). Cancer risk prediction and assessment. Retrieved from http://epi.grants.cancer.gov/cancer_risk_prediction/#risk
- National Comprehensive Cancer Network. (2015). NCCN clinical practice guidelines in oncology: Non-small cell lung cancer. Retrieved from http://www.nccn.org/Accessed 11 January 2016.
- National Centre for Smoking Cessation and Training (NCSCT). (2014). Local Stop Smoking Services. Service and delivery guidance 2014. Retrieved from http://www.ncsct.co.uk/usr/pub/LSSS_service_delivery_guidance.pdf
- National Centre for Smoking Cessation and Training (NCSCT). (2014). Standard treatment programme: A guide to providing behavioural support for smoking cessatin.

 Retrieved from http://www.ncsct.co.uk/usr/pub/standard_treatment_programme.pdf
- National Institute for Health and Care Excellence. (2010). *NICE Public health guidance 23. School-based interventions to prevent smoking*. Manchester, UK: NICE. Retrieved from http://www.nice.org.uk/guidance/ph23.
- National Institute for Health and Care Excellence (NICE). (2011). Lung cancer: The diagnosis and treatment of lung cancer. Clinical guideline 121. Manchester, UK: NICE. Retrieved from https://www.nice.org.uk/guidance/cg121/resources/guidance-lung-cancerpdf.
- National Institute for Health and Care Excellence (NICE). (2007). *NICE Technology appraisal guidance 123*. Manchester, UK: NICE. Retrieved from https://www.nice.org.uk/guidance/ta123/resources/guidance-varenicline-for-smoking-cessation-pdf
- National Institute for Health and Clinical Excellence. (2008). NICE Public health guidance 10. Smoking cessation services in primary care, pharmacies, local authorities and workplaces, particularly for manual working groups, pregnant

- women and hard to reach communities. London: NICE. Retrieved from http://guidance.nice.org.uk/PH10/Guidance/pdf/English.
- Naughton, F., McEwen, A., & Sutton, S. (2013). Use and effectiveness of lapse prevention strategies among pregnant smokers. *Journal of Health Psychology*, 20(11), 1427-1433. doi: 10.1177/1359105313512878
- Nichols, J. A. A., Grob, P., de Lusignan, S., Kite, W., & Williams, P. (2014). Genetic test to stop smoking (GeTSS) trial protocol: Randomised controlled trial of a genetic test (Respiragene) and Auckland formula to assess lung cancer risk. *BMC Pulmonary Medicine*, *14*(77). doi: 10.1186/1471-2466-14-77
- Nichter, M., Nichter, M., Vuckovic, N., Quintero, G., & Ritenbaugh, C. (1997). Smoking experimentation and initiation among adolescent girls: qualitative and quantitative findings. *Tobacco control*, 6(4), 285-295. doi: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1759584/
- Norman, P., Conner, M., & Bell, R. (1999). The theory of planned behavior and smoking cessation. *Health Psychol*, 18(1), 89-94. doi: 10.1037/0278-6133.18.1.89
- O'Connell, A. A. (2005). *Logistic regression models for ordinal response variables*. Thousand Oaks, CA: SAGE Publications, Inc.
- O'Connell, K. A. (2009). Theories used in nursing research on smoking cessation. Annual Review Of Nursing Research, 27, 33-62. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/20192099
- O'Doherty, K., & Suthers, G. K. (2007). Risky communication: Pitfalls in counseling about risk, and how to avoid them. *Journal of Genetic Counseling*, *16*(4), 409-417. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17473963
- O'Loughlin, J., Karp, I., Koulis, T., Paradis, G., & DiFranza, J. (2009). Determinants of first puff and daily cigarette smoking in adolescents. *Am J Epidemiol*, 170(5), 585. doi: 10.1093/aje/kwp179
- Oberg, M., Jaakkola, M. S., Woodward, A., Peruga, A., & Pruss-Ustun, A. (2011). Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. *Lancet*, *377*(9760), 139-146. doi: 10.1016/S0140-6736(10)61388-8
- Offer, D., Ostrov, E., Howard, K. I., & Dolan, S. (1989). A manual for the offer self-image questionnaire for adolescents (OSIQ) (4th ed.). Chicago, Illinois: Michael Reese Hospital.
- Office for National Statistics (ONS). (2011). Integrated household survey April 2010 to March 2011: Experimental statistics. Retrieved from http://www.ons.gov.uk/ons/dcp171778_227150.pdf
- Office for National Statistics (ONS). (2012a). 2011 Census: Key statistics for England and wales, March 2011. Retrieved from http://www.ons.gov.uk/ons/dcp171778_290685.pdf
- Office for National Statistics (ONS). (2012b). Ethnicity and national identity in England and Wales 2011. Retrieved from http://www.ons.gov.uk/ons/dcp171776_290558.pdf
- Office for National Statistics (ONS). (2012c). Life Expectancy in the United Kingdom: 1991-2010, by 2009 local authority district. Retrieved from http://www.ons.gov.uk/ons/interactive/life-expectancy-in-the-uk/index.html
- Office for National Statistics (ONS). (2013a). Cancer statistics registrations, England (Series MB1), No. 42, 2011. Retrieved from http://www.ons.gov.uk/ons/rel/vsob1/cancer-statistics-registrations--england-series-mb1-/no--42--2011/index.html

- Office for National Statistics (ONS). (2013b). Chapter 1 Smoking (General Lifestyle Survey overview: A report on the 2011 General Lifestyle Survey). Retrieved from http://www.ons.gov.uk/ons/dcp171776_302558.pdf
- Office for National Statistics (ONS). (2014a). Adult smoking habits in Great Britain, 2013. Retrieved from http://www.ons.gov.uk/ons/dcp171778_386291.pdf
- Office for National Statistics (ONS). (2014b). Cancer registration statistics, England, 2012. Retrieved from http://www.ons.gov.uk/ons/dcp171778_367563.pdf
- Office for National Statistics (ONS) (2014c). Deaths registered in England and Wales (Series DR), 2013. Retrieved from http://www.ons.gov.uk/ons/dcp171778_381807.pdf
- Office for National Statistics (ONS). (2014d). How have living arrangements and marital status in England and Wales changed since 2001? Retrieved from http://www.ons.gov.uk/ons/dcp171776_356002.pdf.
- Ogden, J. (2012). *Health psychology: A textbook* (5th ed.). Berkshire, England: Open University Press.
- Okoli, C. T. C., Richardson, C. G., Ratner, P. A., & Johnson, J. L. (2009). Non-smoking youths' "perceived" addiction to tobacco is associated with their susceptibility to future smoking. *Addictive Behaviors*, *34*, 1010-1016. doi: 10.1016/j.addbeh.2009.06.010
- Olsson, A. C., Gustavsson, P., Kromhout, H., Peters, S., Vermeulen, R., Bruske, I., . . . Straif, K. (2011). Exposure to diesel motor exhaust and lung cancer risk in a pooled analysis from case-control studies in Europe and Canada. *Am J Respir Crit Care Med*, *183*(7), 941-948. doi: 10.1164/rccm.201006-0940OC
- Omenn, G. S., Goodman, G., Thornquist, M., Grizzle, J., Rosenstock, L., Barnhart, S., . . . et al. (1994). The beta-carotene and retinol efficacy trial (CARET) for chemoprevention of lung cancer in high risk populations: smokers and asbestos-exposed workers. *Cancer Res*, *54*(S7), 2038s-2043s. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8137335
- Orazi, D. C., & Pizzetti, M. (2015). Revisiting fear appeals: A structural re-inquiry of the protection motivation model. *International Journal of Research in Marketing*, 32(2), 223-225. doi: http://dx.doi.org/10.1016/j.ijresmar.2015.02.003
- Osann, K. E., Anton-Culver, H., Kurosaki, T., & Taylor, T. (1993). Sex differences in lung-cancer risk associated with cigarette smoking. *Int J Cancer*, *54*(1), 44-48. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8386708
- Owens, C. & Springett, J. (2006). The Roy Castle Fag Ends Stop Smoking Service: A successful client-led approach to smoking cessation. *Journal of Smoking Cessation*, 1(1), 13-18. doi: 10.1375/jsc.1.1.13
- Paggi, M. G., Vona, R., Abbruzzese, C., & Malorni, W. (2010). Gender-related disparities in non-small cell lung cancer. *Cancer Letters*, 298(1), 1-8. doi: http://dx.doi.org/10.1016/j.canlet.2010.08.009
- Park, E. R., Streck, J. M., Gareen, I. F., Ostroff, J. S., Hyland, K. A., Rigotti, N. A., . . . Nichter, M. (2014). A qualitative study of lung cancer risk perceptions and smoking beliefs among national lung screening trial participants. *Nicotine Tob Res*, *16*(2), 166-173. doi: 10.1093/ntr/ntt133
- Park, J. H., Gail, M. H., Greene, M. H., & Chatterjee, N. (2012). Potential usefulness of single nucleotide polymorphisms to identify persons at high cancer risk: an evaluation of seven common cancers. *J Clin Oncol*, *30*(17), 2157-2162. doi: 10.1200/JCO.2011.40.1943

- Parkes, G., Greenhalgh, T., Griffin, M., & Dent, R. (2008). Effect on smoking quit rate of telling patients their lung age: the Step2quit randomised controlled trial. *BMJ*, 336(7644), 598-600. doi: 10.1136/bmj.39503.582396.25
- Parkin, D. M. (2011a). 14. Cancers attributable to occupational exposures in the UK in 2010. *Br J Cancer*, 105(S2), S70-72. doi: 10.1038/bjc.2011.487
- Parkin, D. M. (2011b). Tobacco-attributable cancer burden in the UK in 2010. *Br J Cancer*, *105*(S2), S6-S13. doi: 10.1038/bjc.2011.475
- Partanen, T., & Boffetta, P. (1994). Cancer risk in asphalt workers and roofers: review and meta-analysis of epidemiologic studies. *Am J Ind Med*, 26(6), 721-740. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7892824
- Patel, J. D. (2005). Lung cancer in women. *J Clin Oncol*, 23(14), 3212-3218. doi: 10.1200/JCO.2005.11.486
- Patrick, D. L., Cheadle, A., Thompson, D. C., Diehr, P., Koepsell, T., & Kinne, S. (1994). The validity of self-reported smoking: a review and meta-analysis. *Am J Public Health*, 84(7), 1086-1093. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8017530
- Pavia, M., Bianco, A., Pileggi, C., & Angelillo, I. F. (2003). Meta-analysis of residential exposure to radon gas and lung cancer. *Bull World Health Organ*, 81(10), 732-738. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14758433
- Pavlovska, I., Orovchanec, N., & Zafirova-Ivanovska, B. (2008). Lung cancer and the smoking habit case control study. *Prilozi*, 29(2), 269-280. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/19259052
- Pechmann, C., Zhao, G., Goldberg, M. E., & Reibling, E. T. (2003). What to convey in antismoking advertisements for adolescents: The use of protection motivation theory to identify effective message themes. *Journal of Marketing*, 67(2), 1-18. doi: http://dx.doi.org/10.1509/jmkg.67.2.1.18607
- Peretti-Watel, P., Halfen, S., & Grémy, I. (2007). Risk denial about smoking hazards and readiness to quit among French smokers: An exploratory study. *Addictive Behaviors*, 32(2), 377-383. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16750305
- Peretti-Watel, P., Legleye, S., Guignard, R., & Beck, F. (2014). Research paper: Cigarette smoking as a stigma: Evidence from France. *International Journal of Drug Policy*, 25, 282-290. doi: 10.1016/j.drugpo.2013.08.009
- Peretti-Watel, P., Seror, V., Verger, P., Guignard, R., Legleye, S., & Beck, F. (2014). Smokers' risk perception, socioeconomic status and source of information on cancer. *Addictive Behaviors*, 39, 1304-1310. doi: 10.1016/j.addbeh.2014.04.016
- Perkins, K. A., Epstein, L. H., & Pastor, S. (1990). Changes in energy balance following smoking cessation and resumption of smoking in women. *J Consult Clin Psychol*, 58(1), 121-125. doi: 10.1037/0022-006X.58.1.121
- Perkins, K. A., & Scott, J. (2008). Sex differences in long-term smoking cessation rates due to nicotine patch. *Nicotine & Tobacco Research*, 10(7), 1245-1251. doi: 10.1080/14622200802097506
- Pesch, B., Kendzia, B., Gustavsson, P., Jockel, K. H., Johnen, G., Pohlabeln, H., . . . Bruning, T. (2012). Cigarette smoking and lung cancer--relative risk estimates for the major histological types from a pooled analysis of case-control studies. *Int J Cancer*, *131*(5), 1210-1219. doi: 10.1002/ijc.27339
- Peto, J. (2011). That lung cancer incidence falls in ex-smokers: misconceptions 2. *British Journal of Cancer*, 104(3), 389. doi:10.1038/sj.bjc.6606080

- Peto, R., Darby, S., Deo, H., Silcocks, P., Whitley, E., & Doll, R. (2000). Smoking, smoking cessation, and lung cancer in the UK since 1950: combination of national statistics with two case-control studies. *BMJ*, *321*(7257), 323-329. doi: http://dx.doi.org/10.1136/bmj.321.7257.323
- Pintos, J., Parent, M. E., Richardson, L., & Siemiatycki, J. (2012). Occupational exposure to diesel engine emissions and risk of lung cancer: Evidence from two case-control studies in Montreal, Canada. *Occupational & Environmental Medicine*, 69(11), 787-792. doi: 10.1136/oemed-2012-100964
- Pirie, K., Peto, R., Reeves, G. K., Green, J., & Beral, V. (2013). The 21st century hazards of smoking and benefits of stopping: A prospective study of one million women in the UK. *Lancet*, *381*(9861), 133-141. doi: 10.1016/s0140-6736(12)61720-6
- Poghosyan, H., Kennedy Sheldon, L., & Cooley, M. E. (2012). The impact of computed tomography screening for lung cancer on smoking behaviors: a teachable moment? *Cancer Nurs*, 35(6), 446-475. doi: 10.1097/NCC.0b013e3182406297
- Polosa, R. & Caponetto, P. (2013). Advances in Smoking Cessation. London: Future Medicine Ltd.
- Powell, H. A., Iyen-Omofoman, B., Hubbard, R. B., Baldwin, D. R., & Tata, L. J. (2013). The association between smoking quantity and lung cancer in men and women. *Chest.* 143(1), 123-129. doi: 10.1378/chest.12-1068
- Prescott, E., Osler, M., Hein, H. O., Borch-Johnsen, K., Lange, P., Schnohr, P., & Vestbo, J. (1998). Gender and smoking-related risk of lung cancer. The Copenhagen Center for Prospective Population Studies. *Epidemiology*, *9*(1), 79-83. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9430273
- Preston, S. H., Glei, D. A., & Wilmoth, J. R. (2010). A new method for estimating smoking-attributable mortality in high-income countries. *Int J Epidemiol*, 39(2), 430-438. doi: 10.1093/ije/dyp360
- Prindiville, S. A., Byers, T., Hirsch, F. R., Franklin, W. A., Miller, Y. E., Vu, K. O., . . . Bunn, P. A. (2003). Sputum cytological atypia as a predictor of incident lung cancer in a cohort of heavy smokers with airflow obstruction. *Cancer Epidemiol Biomarkers Prev*, 12(10), 987-993. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14578133
- Prochaska, J. O., & DiClemente, C. C. (1983). Stages and processes of self-change of smoking: Toward an integrative model of change. *J Consult Clin Psychol*, 51(3), 390-395. doi: 10.1037/0022-006X.51.3.390
- Prochaska, J. O., & DiClemente, C. C. (1984). *The transtheoretical approach: Crossing traditional boundaries of therapy*. Homewood, IL: Dow Jones Irwin.
- Prochaska, J. O., & DiClemente, C. C. (1986). Toward a comprehensive model of change. In W. R. Miller & N. Heather (Eds.), *Treating addictive behaviors* (pp. 3-27). New York: Plenum.
- Prochaska, J. O., DiClemente, C. C., & Norcross, J. C. (1992). In search of how people change. Applications to addictive behaviors. *Am Psychol*, 47(9), 1102-1114. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1329589
- Prochaska, J. O., Velicer, W. F., DiClemente, C. C., & Fava, J. (1988). Measuring processes of change: applications to the cessation of smoking. *J Consult Clin Psychol*, 56(4), 520-528. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3198809

- Proctor, R. N. (2012). The history of the discovery of the cigarette–lung cancer link: Evidentiary traditions, corporate denial, global toll. *Tobacco Control*, 21(2), 87-91. doi: 10.1136/tobaccocontrol-2011-050338
- Public Health England. (2014). Liverpool Health Profile 2014. Retrieved from http://www.apho.org.uk/resource/item.aspx?RID=142116
- Raji, O. Y., Agbaje, O. F., Duffy, S. W., Cassidy, A., & Field, J. K. (2010). Incorporation of a genetic factor into an epidemiologic model for prediction of individual risk of lung cancer: The Liverpool Lung Project. *Cancer Prev Res* (*Phila*), 3(5), 664-669. doi: 10.1158/1940-6207.CAPR-09-0141
- Raji, O. Y., Duffy, S. W., Agbaje, O. F., Baker, S. G., Christiani, D. C., Cassidy, A., & Field, J. K. (2012). Predictive accuracy of the liverpool lung project risk model for stratifying patients for computed tomography screening for lung cancer: A case-control and cohort validation study. *Ann Intern Med*, 157(4), 242-250. doi: 10.7326/0003-4819-157-4-201208210-00004
- Rattray, J., & Jones, M. C. (2007). Essential elements of questionnaire design and development. *Journal of Clinical Nursing*, 16(2), 234-243. doi: 10.1111/j.1365-2702.2006.01573.x
- Reppucci, J. D., Revenson, T. A., Aber, M., & Dickon Reppucci, N. (1991). Unrealistic optimism among adolescent smokers and nonsmokers. *The Journal of Primary Prevention*, 11(3), 227. Retrieved from http://link.springer.com/article/10.1007%2FBF01326505
- Reyna, V. F., & Brainerd, C. J. (2008). Numeracy, ratio bias, and denominator neglect in judgments of risk and probability. *Learning and Individual Differences*, 18, 89-107. doi: 10.1016/j.lindif.2007.03.011
- Riboli, E., Hunt, K. J., Slimani, N., Ferrari, P., Norat, T., Fahey, M., . . . Saracci, R. (2002). European Prospective Investigation into Cancer and Nutrition (EPIC): Study populations and data collection. *Public Health Nutr*, *5*(6B), 1113-1124. doi: 10.1079/phn2002394
- Richmond, R. L., Kehoe, L. A., & Webster, I. W. (1993). Multivariate models for predicting abstention following intervention to stop smoking by general practitioners. *Addiction*, 88(8), 1127-1135. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8401167
- Riedel, B. W., Robinson, L. A., Klesges, R. C., & McLain-Allen, B. (2002). What motivates adolescent smokers to make a quit attempt? *Drug Alcohol Depend*, 68(2), 167-174. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12234646
- Rimm, E. B., Chan, J., Stampfer, M. J., Colditz, G. A., & Willett, W. C. (1995). Prospective study of cigarette smoking, alcohol use, and the risk of diabetes in men. *BMJ*, 310(6979), 555-559. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7888928
- Ringer, G., Smith, J. M., Engel, A. M., Hendy, M. P., & Lang, J. (2005). Influence of sex on lung cancer histology, stage, and survival in a midwestern United States tumor registry. *Clin Lung Cancer*, 7(3), 180-182. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16354312
- Ripoll, J., Girauta, H., Ramos, M., Medina-Bombardó, D., Pastor, A., Alvarez-Ossorio, C., . . . Torres, E. (2012). Clinical trial on the efficacy of exhaled carbon monoxide measurement in smoking cessation in primary health care. *BMC Public Health*, 12, 322. doi: 10.1186/1471-2458-12-322
- Risch, H. A., Howe, G. R., Jain, M., Burch, J. D., Holowaty, E. J., & Miller, A. B. (1993). Are female smokers at higher risk for lung cancer than male smokers?

- A case-control analysis by histologic type. *Am J Epidemiol*, *138*(5), 281-293. Retrieved from http://aje.oxfordjournals.org/content/138/5/281
- Rise, J., Strype, J., & Sutton, S. (2002). Comparative risk ratings and lung cancer among Norwegian smokers. *Addiction Research & Theory*, 10(3), 313-320. doi: 10.1080/16066350290025690
- Ritchie, D., Amos, A., & Martin, C. (2010a). "But it just has that sort of feel about it, a leper": Stigma, smoke-free legislation and public health. *Nicotine Tob Res*, 12(6), 622-629. doi: 10.1093/ntr/ntq058
- Ritchie, D., Amos, A., & Martin, C. (2010b). Public places after smoke-free: A qualitative exploration of the changes in smoking behaviour. *Health Place*, *16*(3), 461-469. doi: 10.1016/j.healthplace.2009.12.003
- Robinson, C. A., Bottorff, J. L., Smith, M. L., & Sullivan, K. M. (2010). "Just because you've got lung cancer doesn't mean I will": Lung cancer, smoking, and family dynamics. *J Fam Nurs*, 16(3), 282-301. doi: 10.1177/1074840710370747
- Robinson, L. M., & Vail, S. R. (2012). An integrative review of adolescent smoking cessation using the transtheoretical model of change. *Journal of Pediatric Health Care*, 26(5), 336-345. doi: 10.1016/j.pedhc.2010.12.001
- Robinson, S., & Harris, H. (2011). Smoking and drinking among adults, 2009. A report of the 2009 General Lifestyle Survey. Retrieved from http://www.ons.gov.uk/ons/index.html.
- Rogers, R. W. (1975). A protection motivation theory of fear appeals and attitude change. *Journal of Psychology*, 91(1), 93-114. doi: 10.1080/00223980.1975.9915803
- Rogers, R. W. (1983). Cognitive and physiological processes in fear appeals and attitude change: A revised theory of protection motivation. In J. T. Cacioppo & R. E. Petty (Eds.), Social psychophysiology: A source book. New York: Guilford Press.
- Rohde, P., Kahler, C. W., Lewinsohn, P. M., & Brown, R. A. (2004). Psychiatric disorders, familial factors, and cigarette smoking: III. Associations with cessation by young adulthood among daily smokers. *Nicotine & Tobacco Research*, 6(3), 509-522. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15203785
- Rollnick, S., & Miller, W. R. (1995). What is motivational interviewing? *Behavioural and Cognitive Psychotherapy*, 23(4), 325-334. doi: http://dx.doi.org/10.1017/S135246580001643X
- Romer, D., & Jamieson, P. (2001). Original article: Do adolescents appreciate the risks of smoking? Evidence from a national survey. *Journal of Adolescent Health*, 29, 12-21. doi: 10.1016/S1054-139X(01)00209-9
- Rosenstock, I. M. (1966). Why people use health services. *Milbank Mem Fund Q*, 44(S3), 94-127. doi: 10.1111/j.1468-0009.2005.00425.x.
- Roski, J., Schmid, L. A., & Lando, H. A. (1996). Long-term associations of helpful and harmful spousal behaviors with smoking cessation. *Addictive Behaviors*, 21(2), 173-185. doi: 10.1016/0306-4603(95)00047-X
- Ross, D., & Kincaid, H. (2010). Introduction: What is addiction? In D. Ross, Kincaid, H., Spurrett, D., Collins, P. (Ed.), *What Is Addiction?* (pp. vii-xi). Cambridge, MA, USA: MIT Press.
- Royal College of Physicians. (2010). Passive smoking and children: A report by the Tobacco Advisory Group. Retrieved from https://www.rcplondon.ac.uk/sites/default/files/documents/passive-smoking-and-children.pdf

- Ruchlin, H. S. (1999). An Analysis of Smoking Patterns among Older Adults. *Medical Care*, 37(6), 615-619. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10386573
- Rushton, L., Bagga, S., Bevan, R., Brown, T. P., Cherrie, J. W., Holmes, P., . . . Hutchings, S. J. (2010). Occupation and cancer in Britain. *Br J Cancer*, 102(9), 1428-1437. doi: 10.1038/sj.bjc.6605637
- Russo, P., Cesario, A., Rutella, S., Veronesi, G., Spaggiari, L., Galetta, D., . . . Greenberg, D. S. (2011). Impact of genetic variability in nicotinic acetylcholine receptors on nicotine addiction and smoking cessation treatment. *Curr Med Chem*, *18*(1), 91-112. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/21110812
- Rutten, L. J. F., Blake, K. D., Hesse, B. W., Augustson, E. M., & Evans, S. (2011). Illness representations of lung cancer, lung cancer worry, and perceptions of risk by smoking status. *Journal of Cancer Education*, 26(4), 747-753. doi: 10.1007/s13187-011-0247-6
- Sairenchi, T., Iso, H., Nishimura, A., Hosoda, T., Irie, F., Saito, Y., . . . Fukutomi, H. (2004). Cigarette smoking and risk of type 2 diabetes mellitus among middleaged and elderly Japanese men and women. *Am J Epidemiol*, *160*(2), 158-162. doi: 10.1093/aje/kwh183
- Santos, H. d., Tonstad, S., Montgomery, S., Paalani, M., & Faed, P. (2011). Smoking cessation behavior in male Portuguese Californians. *Californian Journal of Health Promotion*, 9(2), 68-76. Retrieved from http://www.cjhp.org/Volume9_2011/Issue2/68-76dossantos.pdf
- Sasco, A. J., Secretan, M. B., & Straif, K. (2004). Tobacco smoking and cancer: A brief review of recent epidemiological evidence. *Lung Cancer*, 45(S2), S3-9. doi: 10.1016/j.lungcan.2004.07.998
- Schabath, M. B., Delclos, G. L., Martynowicz, M. M., Greisinger, A. J., Lu, C., Wu, X., & Spitz, M. R. (2005). Opposing effects of emphysema, hay fever, and select genetic variants on lung cancer risk. *Am J Epidemiol*, *161*(5), 412-422. doi: 10.1093/aje/kwi063
- Scharf, D., & Shiffman, S. (2004). Are there gender differences in smoking cessation, with and without bupropion? Pooled- and meta-analyses of clinical trials of Bupropion SR. *Addiction*, *99*(11), 1462-1469. doi: http://www.ncbi.nlm.nih.gov/pubmed/15500599
- Scherphof, C. S., van den Eijnden, R. J. J. M., Harakeh, Z., Raaijmakers, Q. A. W., Kleinjan, M., Engels, R. C. M. E., & Vollebergh, W. A. M. (2013). Effects of nicotine dependence and depressive symptoms on smoking cessation: A longitudinal study among adolescents. *Nicotine & Tobacco Research*, *15*(7), 1222-1229. doi: doi: 10.1093/ntr/nts260
- Schnoll, R. A., Johnson, T. A., & Lerman, C. (2007). Genetics and smoking behavior. *Curr Psychiatry Rep*, 9(5), 349-357. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17915073
- Schnoll, R. A., Martinez, E., Tatum, K. L., Glass, M., Bernath, A., Ferris, D., & Reynolds, P. (2011). Increased self-efficacy to quit and perceived control over withdrawal symptoms predict smoking cessation following nicotine dependence treatment. *Addictive Behaviors*, 36(1-2), 144-147. doi: 10.1016/j.addbeh.2010.08.024
- Schoenberg, J. B., Klotz, J. B., Wilcox, H. B., Nicholls, G. P., Gil-del-Real, M. T., Stemhagen, A., & Mason, T. J. (1990). Case-control study of residential radon

- and lung cancer among New Jersey women. *Cancer Res*, *50*(20), 6520-6524. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/2208111
- Schofield, I., Kerr, S., & Tolson, D. (2007). An exploration of the smoking-related health beliefs of older people with chronic obstructive pulmonary disease. *Journal of Clinical Nursing*, 16(9), 1726-1735. doi: 10.1111/j.1365-2702.2007.01701.x
- Schulz, K. F., & Grimes, D. A. (2002). Blinding in randomised trials: Hiding who got what. *Lancet*, *359*(9307), 696-700. doi: 10.1016/s0140-6736(02)07816-9
- Scivyer, C. R. (2001). Radon protection for new buildings: A practical solution from the UK. *Sci Total Environ*, 272(1-3), 91-96. doi:10.1016/S0048-9697(01)00670-2
- Scott, W. J., Howington, J., Feigenberg, S., Movsas, B., & Pisters, K. (2007). Treatment of non-small cell lung cancer stage I and stage II: ACCP evidence-based clinical practice guidelines (2nd edition). *Chest*, *132*(S3), 234S-242S. doi: 10.1378/chest.07-1378
- Segerstrom, S. C., McArthy, W. J., Caskey, N. H., Gross, T. M., & Jarvik, M. E. (1993). Optimistic bias among cigarette smokers. *Journal of Applied Social Psychology*, 23(19), 1606-1618. doi: 10.1111/j.1559-1816.1993.tb01051.x
- Selden, A. I., Westberg, H. B., & Axelson, O. (1997). Cancer morbidity in workers at aluminum foundries and secondary aluminum smelters. *Am J Ind Med*, *32*(5), 467-477. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9327070
- Sendzik, T., McDonald, P. W., Brown, K. S., Hammond, D., & Ferrence, R. (2011). Planned quit attempts among Ontario smokers: Impact on abstinence. *Addiction*, 106(11), 2005-2013. doi: 10.1111/j.1360-0443.2011.03498.x
- Seo, D. C., & Huang, Y. (2012). Systematic review of social network analysis in adolescent cigarette smoking behavior. *J Sch Health*, 82(1), 21-27. doi: 10.1111/j.1746-1561.2011.00663.x
- Shaper, A. G., Wannamethee, S. G., & Walker, M. (2003). Pipe and cigar smoking and major cardiovascular events, cancer incidence and all-cause mortality in middle-aged British men. *Int J Epidemiol*, *32*(5), 802-808. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14559754
- Sherratt, F. C., Marcus, M. W., Robinson, J., Newson, L., & Field, J. K. (2015). Electronic cigarette use and risk perception in a Stop Smoking Service in England. *Addiction Research & Theory*, 23(4), 336-342. doi: doi:10.3109/16066359.2015.1006629
- Shofer, S., Beyea, M., Li, S., Bastian, L. A., Wahidi, M. M., Kelley, M., & Lipkus, I. M. (2014). Feasibility of using an epigenetic marker of risk for lung cancer, methylation of p16, to promote smoking cessation among US veterans. *BMJ Open Respiratory Research*, 1(1), e000032. doi: 10.1136/bmjresp-2014-000032
- Shrivastav, N., Li, D., & Essigmann, J. M. (2010). Chemical biology of mutagenesis and DNA repair: Cellular responses to DNA alkylation. *Carcinogenesis*, 31(1), 59-70. doi: 10.1093/carcin/bgp262
- Silla, K., Beard, E., & Shahab, L. (2014). Nicotine replacement therapy use among smokers and ex-smokers: Associated attitudes and beliefs: A qualitative study. *BMC Public Health*, *14*, 1311-1311. doi: 10.1186/1471-2458-14-1311
- Silverman, D. T., Samanic, C. M., Lubin, J. H., Blair, A. E., Stewart, P. A., Vermeulen, R., . . . Attfield, M. D. (2012). The diesel exhaust in miners study: A nested case-control study of lung cancer and diesel exhaust. *J Natl Cancer Inst*, 104(11), 855-868. doi: 10.1093/jnci/djs034

- Simonato, L., Agudo, A., Ahrens, W., Benhamou, E., Benhamou, S., Boffetta, P., . . . Zambon, P. (2001). Lung cancer and cigarette smoking in Europe: an update of risk estimates and an assessment of inter-country heterogeneity. *Int J Cancer*, *91*(6), 876-887.
- Simons-Morton, B. G., & Farhat, T. (2010). Recent findings on peer group influences on adolescent smoking. *J Prim Prev*, 31(4), 191-208. doi: 10.1007/s10935-010-0220-x
- Sjoberg, L. (2000). Factors in risk perception. *Risk Analysis: An Official Publication Of The Society For Risk Analysis*, 20(1), 1-11. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10795334
- Slatore, C. G., Baumann, C., Pappas, M., & Humphrey, L. L. (2014). Smoking behaviors among patients receiving computed tomography for lung cancer screening. Systematic review in support of the U.S. preventive services task force. *Ann Am Thorac Soc*, 11(4), 619-627. doi: 10.1513/AnnalsATS.201312-460OC
- Slovic, P. (2000a). *The perception of risk*. London: Earthscan.
- Slovic, P. (2000b). What does it mean to know a cumulative risk? Adolescents' perceptions of short-term and long-term consequences of smoking. *Journal of Behavioral Decision Making*, 13(2), 259-266. doi: 10.1002/(SICI)1099-0771(200004/06)13:2<259::AID-BDM336>3.0.CO;2-6
- Smerecnik, C., Quaak, M., van Schayck, C. P., van Schooten, F., & de Vries, H. (2011). Are smokers interested in genetic testing for smoking addiction? A socio-cognitive approach. Psychology & Health, 26(8), 1099-1112. doi:10.1080/08870446.2010.541909
- Smith, E. C., Burkle, F. M., Jr., & Archer, F. L. (2011). Fear, familiarity, and the perception of risk: A quantitative analysis of disaster-specific concerns of paramedics. *Disaster Medicine and Public Health Preparedness*, *5*(1), 46-53. doi: 10.1001/dmp.10-v4n2-hre10008
- Smith, J. A., Michie, S., Stephenson, M., & Quarrell, O. (2002). Risk perception and decision-making processes in candidates for genetic testing for Huntington's disease: An interpretative phenomenological analysis. *Journal of Health Psychology*, 7(2), 131-144. doi: 10.1177/1359105302007002398
- Song, A. V., Morrell, H. E. R., Cornell, J. L., Ramos, M. E., Biehl, M., Kropp, R. Y., & Halpern-Felsher, B. L. (2009). Perceptions of smoking-related risks and benefits as predictors of adolescent smoking initiation. *Am J Public Health*, *99*(3), 487-492. doi: 10.2105/AJPH.2008.137679
- Song, F., Raftery, J., Aveyard, P., Hyde, C., Barton, P., & Woolacott, N. (2002). Cost-effectiveness of pharmacological interventions for smoking cessation: a literature review and a decision analytic analysis. *Med Decis Making*, 22(S5), S26-37. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12369228
- Spielberger, C. D., & Jacobs, G. A. (1982). Personality and smoking behavior. *J Pers Assess*, 46(4), 396-403. doi: 10.1207/s15327752jpa4604_11
- Spitz, M. R., Etzel, C. J., Dong, Q., Amos, C. I., Wei, Q., Wu, X., & Hong, W. K. (2008). An expanded risk prediction model for lung cancer. *Cancer Prev Res* (*Phila*), 1(4), 250-254. doi: 10.1158/1940-6207.CAPR-08-0060
- Spitz, M. R., Hong, W. K., Amos, C. I., Wu, X., Schabath, M. B., Dong, Q., . . . Etzel, C. J. (2007). A risk model for prediction of lung cancer. *J Natl Cancer Inst*, 99(9), 715-726. doi: 10.1093/jnci/djk153
- Stampfer, M. (2004). New insights from the British doctors study. *BMJ*, 328(7455), 1507. doi: 10.1136/bmj.328.7455.1507

- Stapleton, J. A., Russell, M. A., Feyerabend, C., Wiseman, S. M., Gustavsson, G., Sawe, U., & Wiseman, D. (1995). Dose effects and predictors of outcome in a randomized trial of transdermal nicotine patches in general practice. *Addiction*, 90(1), 31-42. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7888977
- Starr, C. (1969). Social benefit versus technological risk. *Science*, *165*(3899), 1232-1238. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/5803536
- Stayner, L., Bena, J., Sasco, A. J., Smith, R., Steenland, K., Kreuzer, M., & Straif, K. (2007). Lung cancer risk and workplace exposure to environmental tobacco smoke. *Am J Public Health*, *97*(3), 545-551. doi: 10.2105/ajph.2004.061275
- Stead, L. F., Hartmann-Boyce, J., Perera, R., & Lancaster, T. (2013). Telephone counselling for smoking cessation. *Cochrane Database Syst Rev*, 8, Cd002850. doi: 10.1002/14651858.CD002850.pub3
- Stead, L. F., & Lancaster, T. (2005). Group behaviour therapy programmes for smoking cessation. *Cochrane Database Syst Rev*(2), Cd001007. doi: 10.1002/14651858.CD001007.pub2
- Stead, L. F., Perera, R., Bullen, C., Mant, D., Hartmann-Boyce, J., Cahill, K., & Lancaster, T. (2012). Nicotine replacement therapy for smoking cessation. *Cochrane Database Syst Rev*, 11, Cd000146. doi: 10.1002/14651858.CD000146.pub4
- Stead, M., Moodie, C., Angus, K., Bauld, L., McNeill, A., Thomas, J., . . . Bryce, S. L. (2013). Is consumer response to plain/standardised tobacco packaging consistent with framework convention on tobacco control guidelines? A systematic review of quantitative studies. *PLoS One*, 8(10), e75919. doi: 10.1371/journal.pone.0075919
- Stephens, F. O., & Aigner, K. R. (2009). Basics of oncology. New York: Springer.
- Stewart, D. W., Adams, C. E., Cano, M. A., Correa-Fernandez, V., Li, Y., Waters, A. J., . . . Vidrine, J. I. (2013). Associations Between Health Literacy and Established Predictors of Smoking Cessation. *Am J Public Health*, *103*(7), e43-e49. doi: 10.2105/AJPH.2012.301062
- Stoltzfus, J. C. (2011). Logistic regression: A brief primer. *Acad Emerg Med*, *18*(10), 1099-1104. doi: 10.1111/j.1553-2712.2011.01185.x
- Strecher, V. J., Kreuter, M. W., & Kobrin, S. C. (1995). Do cigarette smokers have unrealistic perceptions of their heart attack, cancer, and stroke risks? *J Behav Med*, *18*(1), 45-54. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7595951
- Subramanian, J., & Govindan, R. (2007). Lung cancer in never smokers: A review. *J Clin Oncol*, 25(5), 561-570. doi: 10.1200/JCO.2006.06.8015
- Sullivan, P. F., & Kendler, K. S. (1999). The genetic epidemiology of smoking. *Nicotine Tob Res*, 1(S2), S51-57; discussion S69-70. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11768187
- Sutton, S. (2001). Back to the drawing board? A review of applications of the transtheoretical model to substance use. *Addiction*, *96*(1), 175-186. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11177528
- Sutton, S. (2005). Stage theories of health behaviour. In M. Conner & P. Norman (Eds.), *Predicting health behaviour: Research and practice with social cognition models* (2nd ed. pp. 223-275). Maidenhead: Open University Press.
- Taioli, E., & Wynder, E. L. (1991). Effect of the age at which smoking begins on frequency of smoking in adulthood. *New England Journal of Medicine*, 325(13), 968-969. doi: doi:10.1056/NEJM199109263251318

- Takkouche, B., Regueira-Mendez, C., & Montes-Martinez, A. (2009). Risk of cancer among hairdressers and related workers: A meta-analysis. *Int J Epidemiol*, 38(6), 1512-1531. doi: 10.1093/ije/dyp283
- Tammemagi, M. (2015). Application of risk prediction models to lung cancer screening. *Journal of Thoracic Imaging*, 30(2), 88-100. doi: 10.1097/RTI000000000000142
- Tammemägi, M. C., Katki, H. A., Hocking, W. G., Church, T. R., Caporaso, N., Kvale, P. A., & ... Berg, C. D. (2013). Selection criteria for lung-cancer screening. New England Journal Of Medicine, 368(8), 728-736 9p. doi:10.1056/NEJMoa1211776
- Tammemagi, C. M., Pinsky, P. F., Caporaso, N. E., Kvale, P. A., Hocking, W. G., Church, T. R., . . . Prorok, P. C. (2011). Lung cancer risk prediction: Prostate, Lung, Colorectal And Ovarian Cancer Screening Trial models and validation. *J Natl Cancer Inst*, 103(13), 1058-1068. doi: 10.1093/jnci/djr173
- Tashakkori, A., & Teddlie, C. (2010). Sage handbook of mixed methods in social & behavioral research (2nd ed.). London: Sage Publications.
- Taylor-Gooby, P., & Zinn, J. (2006). The current significance of risk. In P. Taylor-Gooby & J. Zinn (Eds.), *Risk in social science* (pp. 1-19). Oxford, UK: Oxford University Press.
- Taylor, G., McNeill, A., Girling, A., Farley, A., Lindson-Hawley, N., & Aveyard, P. (2014). Change in mental health after smoking cessation: systematic review and meta-analysis. *BMJ*, *348*. doi: http://dx.doi.org/10.1136/bmj.g1151
- Taylor, R., Najafi, F., & Dobson, A. (2007). Meta-analysis of studies of passive smoking and lung cancer: Effects of study type and continent. *Int J Epidemiol*, *36*(5), 1048-1059. doi: 10.1093/ije/dym158
- Teddlie, C., & Tashakkori, A. (2010). Overview of contemporary issues in mixed methods research. In C. Teddlie & A. Tashakkori (Eds.), *SAGE Handbook of mixed methods in social & behavioral research* (2nd ed., pp. 1-44). Thousand Oaks, California: SAGE Publications, Inc.
- Thornicroft, G., Rose, D., Kassam, A., & Sartorius, N. (2007). Stigma: Ignorance, prejudice or discrimination? *The British Journal of Psychiatry*, 190(3), 192-193. doi: 10.1192/bjp.bp.106.025791
- Thun, M. J., DeLancey, J. O., Center, M. M., Jemal, A., & Ward, E. M. (2010). The global burden of cancer: Priorities for prevention. *Carcinogenesis*, *31*(1), 100-110. doi: 10.1093/carcin/bgp263
- Thun, M. J., Lally, C. A., Flannery, J. T., Calle, E. E., Flanders, W. D., & Heath, C. W., Jr. (1997). Cigarette smoking and changes in the histopathology of lung cancer. *J Natl Cancer Inst*, 89(21), 1580-1586. doi: http://www.ncbi.nlm.nih.gov/pubmed/9362155
- Toll, B. A., Rojewski, A. M., Duncan, L. R., Latimer-Cheung, A. E., Fucito, L. M., Boyer, J. L., . . . Herbst, R. S. (2014). "Quitting smoking will benefit your health": The evolution of clinician messaging to encourage tobacco cessation. *Clin Cancer Res*, 20(2), 301-309. doi: 10.1158/1078-0432.ccr-13-2261
- Tombor, I., Shahab, L., Herbec, A., Neale, J., Michie, S., & West, R. (2015). Smoker identity and its potential role in young adults' smoking behavior: A meta ethnography. *Health Psychol.*, *34*(10), 992-1003. doi: 10.1037/hea0000191
- Tonstad, S., Tonnesen, P., Hajek, P., Williams, K. E., Billing, C. E., & Reeves, K. E. (2006). Effect of maintenance therapy with varenicline for smoking cessation. *JAMA*, 296, 64–71. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16820548

- Torchalla, I., Okoli, C. T. C., Hemsing, N., & Greaves, L. (2011). Gender Differences in Smoking Behaviour and Cessation. *Journal of Smoking Cessation*, 6(1), 9-16. doi: 10.1375/jsc.6.1.9
- Towers, S., Afzal, S., Bernal, G., Bliss, N., Brown, S., Espinoza, B., . . . Castillo-Chavez, C. (2015). Mass media and the contagion of fear: The case of ebola in america. *PLoS One*, 10(6), 1-13. doi: 10.1371/journal.pone.0129179
- Travis, L. B., Fossa, S. D., Schonfeld, S. J., McMaster, M. L., Lynch, C. F., Storm, H., . . . Gilbert, E. S. (2005). Second cancers among 40,576 testicular cancer patients: Focus on long-term survivors. *J Natl Cancer Inst*, 97(18), 1354-1365. doi: 10.1093/jnci/dji278
- Trichopoulos, D., Kalandidi, A., Sparros, L., & MacMahon, B. (1981). Lung cancer and passive smoking. *Int J Cancer*, 27(1), 1-4. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7251227
- Tucker, J. S., Ellickson, P. L., & Klein, D. J. (2002). Smoking cessation during the transition from adolescence to young adulthood. *Nicotine and Tobacco Research*, 4(3), 321-332. doi: 10.1080/14622200210142698
- Turner, L., Mermelstein, R., & Flay, B. (2004). Individual and contextual influences on adolescent smoking. *Ann N Y Acad Sci*, 1021, 175-197. doi: 10.1196/annals.1308.023
- Tyas, S. L., & Pederson, L. L. (1998). Psychosocial factors related to adolescent smoking: A critical review of the literature. *Tobacco Control*, 7(4), 409-420. doi:10.1136/tc.7.4.409
- Uchimoto, S., Tsumura, K., Hayashi, T., Suematsu, C., Endo, G., Fujii, S., & Okada, K. (1999). Impact of cigarette smoking on the incidence of Type 2 diabetes mellitus in middle-aged Japanese men: The Osaka Health Survey. *Diabetic Medicine*, 16(11), 951-955. doi: 10.1046/j.1464-5491.1999.00173.x
- Umeh, K., & Barnes, J. (2011). Cognitive Appraisals and Smoking Intentions: The Role of Decision Making Competence. *Journal of Smoking Cessation*, 6(02), 144-151. doi: doi:10.1375/jsc.6.2.144
- UyBico, S. J., Wu, C. C., Suh, R. D., Le, N. H., Brown, K., & Krishnam, M. S. (2010). Lung cancer staging essentials: The new TNM staging system and potential imaging pitfalls. *Radiographics*, 30(5), 1163-1181. doi: 10.1148/rg.305095166
- van Loon, A. J., Tijhuis, M., Surtees, P. G., & Ormel, J. (2005). Determinants of smoking status: Cross-sectional data on smoking initiation and cessation. *Eur J Public Health*, 15(3), 256-261. doi: 10.1093/eurpub/cki077
- Vander Weg, M. W., Klesges, R. C., Eck Clemens, L. H., Meyers, A. W., & Pascale, R. W. (2001). The relationships between ethnicity, gender, and short-term changes in energy balance following smoking cessation. *International Journal of Behavioral Medicine*, 8(2), 163-177. doi: 10.1207/S15327558IJBM0802 06
- Vangeli, E., Stapleton, J., Smit, E. S., Borland, R., & West, R. (2011). Predictors of attempts to stop smoking and their success in adult general population samples: A systematic review. *Addiction*, 106(12), 2110-2121. doi: 10.1111/j.1360-0443.2011.03565.x
- Vangeli, E., & West, R. (2012). Transition towards a 'non-smoker' identity following smoking cessation: An interpretative phenomenological analysis. *British Journal of Health Psychology*, 17(1), 171-184. doi: 10.1111/j.2044-8287.2011.02031.x

- Velicer, W. F., & Prochaska, J. O. (2004). A comparison of four self-report smoking cessation outcome measures. *Addictive Behaviors*, 29, 51-60. doi: 10.1016/S0306-4603(03)00084-4
- Velicer, W. F., Prochaska, J. O., Fava, J. L., Norman, G. J., & Redding, C. A. (1998). Smoking cessation and stress management: Applications of the transtheoretical model of behavior change. *Homeostasis in Health and Disease*, 38(5-6), 216-233.
- Velicer, W. F., Prochaska, J. O., & Redding, C. A. (2006). Tailored communications for smoking cessation: Past successes and future directions. *Drug Alcohol Rev*, 25(1), 49-57. doi: 10.1080/09595230500459511
- Vineis, P., Hoek, G., Krzyzanowski, M., Vigna-Taglianti, F., Veglia, F., Airoldi, L., . . . Riboli, E. (2006). Air pollution and risk of lung cancer in a prospective study in Europe. *Int J Cancer*, 119(1), 169-174. doi: 10.1002/ijc.21801
- Vineis, P., Hoek, G., Krzyzanowski, M., Vigna-Taglianti, F., Veglia, F., Airoldi, L., . . . Riboli, E. (2007). Lung cancers attributable to environmental tobacco smoke and air pollution in non-smokers in different European countries: A prospective study. *Environ Health*, 6, 7. doi: 10.1186/1476-069X-6-7
- Virgili, M., Owen, N., & Sverson, H. H. (1991). Adolescents' smoking behavior and risk perceptions. *Journal Of Substance Abuse*, *3*(3), 315-324. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/1821288
- Viscusi, W. K. (1990). Do smokers underestimate risks? *Journal of Political Economy*, 98, 1253-1269.
- Wahlberg, A. A. F., & Sjoberg, L. (2000). Risk perception and the media. *Journal of Risk Research*, 3(1), 31-50. doi: 10.1080/136698700376699
- Wakefield, M., Flay, B., Nichter, M., & Giovino, G. (2003). Role of the media in influencing trajectories of youth smoking. *Addiction*, 98, 79-103. doi: 10.1046/j.1360-0443.98.s1.6.x
- Wald, N., & Nicolaides-Bouman, A. (1991). *UK smoking statistics* (2nd ed.). Oxford: Oxford University Press.
- Walters, S., Benitez-Majano, S., Muller, P., Coleman, M. P., Allemani, C., Butler, J., . . . Bernard, R. (2015). Is England closing the international gap in cancer survival? *British Journal of Cancer*, 113(5), 848-860. doi: 10.1038/bjc.2015.265
- Weinstein, N. D. (1983). Reducing unrealistic optimism about illness susceptibility. *Health Psychology*, 2(1), 11-20. Retrieved from http://psycnet.apa.org/index.cfm?fa=buy.optionToBuy&id=1988-10759-001
- Weinstein, N. D. (1984). Why it won't happen to me: Perceptions of risk factors and susceptibility. *Health Psychol*, *3*(5), 431-457. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/6536498
- Weinstein, N. D. (1988). The precaution adoption process. *Health Psychol*, 7(4), 355-386. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3049068
- Weinstein, N. D. (1998). Accuracy of smokers' risk perceptions. *Annals of Behavioral Medicine*, 20(2), 135-140. Retrieved from http://link.springer.com/article/10.1007%2FBF02884459#page-1
- Weinstein, N. D. (1999). What does it mean to understand a risk? Evaluating risk comprehension. *J Natl Cancer Inst Monogr*(25), 15-20. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10854451
- Weinstein, N. D., & Klein, W. M. (1996). Unrealistic optimism: Present and future. *Journal of Social and Clinical Psychology*, 15(1), 1-8. doi: 10.1521/jscp.1996.15.1.1

- Weinstein, N. D., Marcus, S. E., & Moser, R. P. (2005). Smokers' unrealistic optimism about their risk. *Tobacco Control*, 14(1), 55-59. doi: 10.1136/tc.2004.008375
- Weinstein, N. D., Rothman, A. J., & Sutton, S. R. (1998). Stage theories of health behavior: Conceptual and methodological issues. *Health Psychol*, *17*(3), 290-299. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9619480
- West, R. (2005). Time for a change: Putting the Transtheoretical (Stages of Change) Model to rest. *Addiction*, 100(8), 1036-1039. doi: 10.1111/j.1360-0443.2005.01139.x
- West, R. (2006). Background smoking cessation rates in England. Retrieved from http://www.smokinginengland.info/Ref/paper2.pdf
- West, R., & Brown, J. (2013). A theory of addiction. John Wiley & Sons, Ltd.
- West, R., & Fidler, J. (2011). Smoking and smoking cessation in England 2010: Findings from the smoking toolkit study. Retrieved from www.smokinginengland.info
- West, R., Hajek, P., Stead, L. F., & Stapleton, J. (2005). Outcome criteria in smoking cessation trials: Proposal for a common standard. *Addiction*, 100(3), 299-303. doi: 10.1111/j.1360-0443.2004.00995.x
- West, R., May, S., West, M., Croghan, E., & McEwen, A. (2013). Performance of English stop smoking services in first 10 years: Analysis of service monitoring data. *BMJ*, 347, f4921. doi: 10.1136/bmj.f4921
- West, R., McEwen, A., Bolling, K., & Owen, L. (2001). Smoking cessation and smoking patterns in the general population: A 1-year follow-up. *Addiction*, 96(6), 891-902. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11399220
- Westermeyer, J. (2013). Chapter 1 Historical understandings of addiction. In P. M. Miller (Ed.), *Principles of addiction* (pp. 3-12). San Diego: Academic Press.
- White, S., & Pavlakis, N. (2008). Evolution of biological therapies in non-small cell lung cancer. *Cancer Forum*, 32(3), 147. Retrieved from http://cancerforum.org.au/forum/2008/november/evolution-of-biological-therapies-in-non-small-cell-lung-cancer/
- Whittaker, R., McRobbie, H., Bullen, C., Borland, R., Rodgers, A., & Gu, Y. (2012). Mobile phone-based interventions for smoking cessation. *Cochrane Database Syst Rev*, 11, Cd006611. doi: 10.1002/14651858.CD006611.pub3
- Wilkes, S. (2008). The use of bupropion SR in cigarette smoking cessation. *Int J Chron Obstruct Pulmon Dis*, 3(1), 45-53. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/18488428
- Williams, T., & Clarke, V. A. (1997). Optimistic bias in beliefs about smoking. *Australian Journal of Psychology*, 49(2), 106-112. doi: 10.1080/00049539708259861
- Witte, K. (1992). Putting the fear back into fear appeals: The extended parallel process model. *Communication Monographs*, 59, 329-349. doi:10.1080/03637759209376276
- Witte, K. (1994). Fear control and danger control: A test of the extended parallel process model (EPPM). *Communication Monographs*, 61, 113-134. doi: 10.1080/03637759409376328
- Witte, K., & Allen, M. (2000). A meta-analysis of fear appeals: Implications for effective public health campaigns. *Health Education & Behavior*, 27(5), 591-615. doi: 10.1177/109019810002700506

- Wogan, G. N., Hecht, S. S., Felton, J. S., Conney, A. H., & Loeb, L. A. (2004). Environmental and chemical carcinogenesis. *Semin Cancer Biol*, 14(6), 473-486. doi: 10.1016/j.semcancer.2004.06.010
- Wold, B., Torsheim, T., Currie, C., & Roberts, C. (2004). National and school policies on restrictions of teacher smoking: A multilevel analysis of student exposure to teacher smoking in seven European countries. *Health Educ Res*, 19(3), 217-226. doi: 10.1093/her/cyg029
- Wong, N. C. H. & Cappella, J. N (2009). Antismoking threat and efficacy appeals: effects on smoking cessation intentions for smokers with low and high readiness to quit. *Journal of Applied Communication Research*, 37(1), 1-20. doi: 10.1080/00909880802593928
- World Health Organization (WHO). (2001). Water Quality: Guidelines, Standards and Health. L. Fewtrell & J. Bartram (Ed.). London: IWA Publishing.
- World Health Organisation (WHO). (2015). Global health observatory data repository: life expectancy. Data by country. Geneva, Switzerland: WHO. Retrieved from http://apps.who.int/gho/data/node.main.688?lang=en
- World Health Organization (WHO). (2013). Who report on the global tobacco epidemic, 2013: Enforcing bans on tobacco advertising, promotion and sponsorship. Geneva, Switzerland: WHO. Retrieved from http://www.who.int/tobacco/global_report/2013/en/.
- Wright, A. J., French, D. P., Weinman, J., & Marteau, T. M. (2006). Can genetic risk information enhance motivation for smoking cessation? An analogue study. *Health Psychology*, 25(6), 740-752. doi:10.1037/0278-6133.25.6.740
- Wynder, E. L., & Muscat, J. E. (1995). The changing epidemiology of smoking and lung cancer histology. *Environ Health Perspect*, 103(S8), 143-148. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8741774
- Yan, Y., Jacques-Tiura, A. J., Chen, X., Xie, N., Chen, J., Yang, N., & ... MacDonell, K. K. (2014). Application of the Protection Motivation Theory in predicting cigarette smoking among adolescents in China. *Addictive Behaviors*, 39(1), 181-188. doi:10.1016/j.addbeh.2013.09.027
- Youlden, D. R., & Baade, P. D. (2011). The relative risk of second primary cancers in Queensland, Australia: A retrospective cohort study. *BMC Cancer*, 11, 83. doi: 10.1186/1471-2407-11-83
- Young, R. P., & Hopkins, R. J. (2012). Lung cancer risk prediction to select smokers for screening CT--letter. *Cancer Prev Res (Phila)*, *5*(4), 697-698; author reply 699. doi: 10.1158/1940-6207.CAPR-11-0531
- Young, R. P., Hopkins, R. J., Smith, M., & Hogarth, D. K. (2010). Smoking cessation: The potential role of risk assessment tools as motivational triggers. *Postgrad Med J*, 86(1011), 26-33; quiz 31-22. doi: 10.1136/pgmj.2009.084947
- Yu, S., & Zhao, N. (1996). Combined analysis of case-control studies of smoking and lung cancer in China. *Lung Cancer*, 14(S1), S161-S170. doi: 10.1016/S0169-5002(96)90221-4
- Yu, Y., Liu, H., Zheng, S., Ding, Z., Chen, Z., Jin, W., . . . Zhang, R. (2014). Gender susceptibility for cigarette smoking-attributable lung cancer: A systematic review and meta-analysis. *Lung Cancer*, 85, 351-360. doi: 10.1016/j.lungcan.2014.07.004
- Zang, E. A., & Wynder, E. L. (1996). Differences in lung cancer risk between men and women: Examination of the evidence. *J Natl Cancer Inst*, 88(3-4), 183-192. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8632492

- Zhang, L., Ren, J. W., Wong, C. C., Wu, W. K., Ren, S. X., Shen, J., . . . Cho, C. H. (2012). Effects of cigarette smoke and its active components on ulcer formation and healing in the gastrointestinal mucosa. *Curr Med Chem*, *19*(1), 63-69. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/22300077
- Zhou, X., Nonnemaker, J., Sherrill, B., Gilsenan, A. W., Coste, F., & West, R. (2009). Attempts to quit smoking and relapse: Factors associated with success or failure from the ATTEMPT cohort study. *Addictive Behaviors*, *34*, 365-373. doi: 10.1016/j.addbeh.2008.11.013
- Zhu, S. H., Sun, J., Billings, S. C., Choi, W. S., & Malarcher, A. (1999). Predictors of smoking cessation in U.S. adolescents. *Am J Prev Med*, 16(3), 202-207. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10198659
- Zimmerman, R. S., & Vernberg, D. (1994). Models of preventive health behavior: Comparison, critique, and meta-analysis. In G. Albrecht (Ed.), *Advances in medical sociology, volume 4: health behavior models: A reformulation*. Greenwich, CT: JAI Press.
- Zlatev, M., Pahl, S., & White, M. (2010). Perceived risk and benefit for self and others as predictors of smokers' attitudes towards smoking restrictions. *Psychology & Health*, 25(2), 167-182. doi: 10.1080/08870440802372449

Appendix A. Processes of change implicated in the health behaviour model, the Transtheoretical Model of Change (TTM)

The ten processes of change are defined as follows (Velicer, Prochaska, Fava, Norman, & Redding, 1998):

- Consciousness raising: Becoming increasingly aware of the causes, consequences and treatment of the problematic behaviour. Interventions that may encourage awareness can include feedback, education, confrontation, interpretation, bibliotherapy, and media campaigns.
- Dramatic relief: Increased emotional experiences, whereby reduced affect may
 follow if appropriate action is accomplished. Psychodrama, role playing,
 grieving, and personal testimonies, are all examples of techniques that can
 induce emotional experiences.
- 3. Environmental re-evaluation: Combination of cognitive and affective assessments regarding the impact of the individual's behaviour upon their social environment e.g. the impact of drinking alcohol on others. Reassessments can be induced through empathy training, documentaries, and family interventions.
- 4. Social liberation: Increased social opportunities or alternatives for nonproblematic behaviours. Advocacy, empowerment procedures, and

appropriate policies can provide increased opportunities, particularly for those who have experienced deprivation or oppression.

- 5. Self-re-evaluation: Combination of cognitive and affective assessments regarding the individual's self-image, whether that be with the problematic behaviour or without. Value clarification, healthy role models, and imagery, are all strategies that may encourage self-re-evaluation.
- 6. Stimulus control: Removal of cues that may prompt the problematic behaviour or adding prompts for healthy alternatives to the behaviour. Avoidance, environmental re-engineering, and self-help groups can be utilised as a means of stimulus control.
- Helping relationships: Experiencing caring, accepting, trusting and supportive relationships. Sources of social support can vary from family members to counsellor support.
- 8. Counterconditioning: Learning and developing new, healthier behaviours in replacement of the problematic behaviour. Useful strategies to enforce counterconditioning may include learning relaxation techniques for stress, or developing assertion techniques to counter peer pressure.
- 9. Reinforcement management: Implementation of punishment and rewards for enforcing certain behaviours, whether they are implemented by the individual

or by others. Procedures for reinforcement can include contingency contracts, positive self-statements, and group recognition.

10. Self-liberation: Often regarded as willpower, self-liberation refers to one's belief in their ability to change, and commitment to act on their belief. Self-liberation has been found to be enhanced by multiple rather than single choices, such as New Year's resolutions and public testimonies.

Appendix B. Roy Castle FagEnds (RCFE) e-mail communication regarding service cessation rates

E-mail communication removed as contains personal contact details for RCFE staff.

200			He hay easile serificion se way, wayer tree Teamhordy Failly wayer tree, Elon D
L	ROY CASILE		
L	ragends		Email:tobaccocontrol@roycastle.org
	MONDAY	GROUPS	Freephone 0800 195 2131
AM	9:00am - 10.15am	Norris Green	Ellergreen Community & Early Years Centre, Ellergreen Road L11 2RY
	10.00am - 11.15am	Wavertree	Wavertree Library, The High Street, Wavertree, L15 4LP
	10:00am - 11:15am	West Derby 1-2-1appointment only	Lloyds Pharmacy, 66 Muirhead Avenue East, Liverpool, L11 1EN
	9:15am - 10.30am	Mossley Hill 1-2-1 appointment only	Greenbank Road Surgery, 1b Greenbank Road, L18 1HG
	12.00pm - 12.45pm	Brownlow Group Practice	Brownlow Group Practice, The Infirmary, 70 Pembroke Place Liverpool Merseyside L69 3GF
	1:00pm - 2.15pm	Breckfield	Breck Road Library, Breck Road, L5 6PX
	1:30pm - 2.45pm	Wavertree 1-2-1 appointment only	Asda Smithdown Road, Wavertree, L15 2LF
	1:30pm -2.15pm	Old Swan	Old Swan One Stop Shop, Derby Lane, Old Swan, Liverpool, L13 6QA
	2:00pm - 3.15pm	Fazakerley	Long Lane Health Centre, Long Lane, Fazakerley, L9 6DQ
	2:00pm - 3.15pm	Garston	Garston Library, Bowden Road, Garston, L19 1QN
	4:00pm - 4:45pm	Toxteth	The Florrie, 377 Mill Street, Toxteth, Liverpool, L8 4RF
	4:15pm-6:00pm	Speke	Neighbourhood Health centre, 75 South Parade, Speke, L24 2XP
	5.00pm-6.15pm	City Centre	Royal Liverpool Hospital, J&K Clinic, Prescot Street, L7 8XP
	5.15pm - 6.15pm	Dingle	The Elms Medical Centre, The Elms, Liverpool, L8 3SS
	6.00pm - 6.45pm	Croxteth	Croxteth Community Fire Station, L11 9AP
	TUESDAY	GROUPS	
AM	9:00am - 10.45am	Belle Vale	Belle Vale Health Centre, Hedgefield Road, L25 2XE
	9:00am - 10.15am	Dovecot	Yewtree Childrens Centre, Berryford Road L14 4ED
	9:30am - 10:45am	Wavertree	Sefton Park Medical Centre, Smithdown Road, Liverpool, L15 2LQ
	9:45am - 11.00am	Old Swan	Salvation Army, Prescot Road, Old Swan, L13 3DA
	10:00am - 11:15am	Everton	Everton Road Health Centre, 45 Everton Road, Liverpool, L6 2EW
	12.00pm-1.15pm	Anfield	Townsend Neighbourhood Health Centre, 98 Townsend Lane, L6 0BB
PM	12:30pm - 1.45pm	Walton	The Breeze Inn, The Longmoor Lane Methodist Church, opposite Black Bull Pub, L9 0EA
	1:00pm - 2.15pm	Toxteth	Princes Park Health centre, Bentley Road, Liverpool, L8 0SY
	1:30pm - 2.45pm	Fairfield 1-2-1appointment only	Halls & Stevens Chemist, 18 Prescot Road, L7 0JA
	2:00pm - 3.15pm	Netherley	Netherley Health Centre, Middlemass Hey, L27 7AF
	2:00pm - 2:45pm	Baycliff	Baycliff Family Health Centre, 73 Baycliff Road, West Derby, L12 6QT
	2:00pm - 3:45pm	Norris Green	Norris Green Library, Townsend Avenue, Liverpool, L11 5AF
	1:00pm - 1.45pm	Walton	Walton Library, One Stop, Evered Avenue, Walton L9 2AF
	3:00pm - 3.45pm	Kirkdale	Bousfield Surgery, Westminister Road, L4 4PP
	3:30pm - 4.45pm	Wavertree	Earle Road Childrens Centre, 139 Earl Road, L7 6HD
	4:00pm - 5.15pm	Allerton	Allerton Library, Allerton Road, L18 6HG
	4:00pm - 5.45pm	Belle Vale	Belle Vale Health Centre, Hedgefield Road, L25 2XE
	5.20pm - 6.15pm	Toxteth	Abercromby Health Centre, Grove Street, L7 7HG
	5:30pm - 6:45pm	West Derby	St Mary's Millennium Centre, Meadow Lane, L12 5EA

	WEDNESDAT	GROOFS	
₹	AM 10:00am - 11.15am	Vauxhall	Vauxhall Medical Centre, Limekiln Lane, Vauxhall, L5 8XR
	10:00am - 11.15am	Norris Green	Ellergreen Medical Centre, 24 Carr Lane, Liverpool, L11 2YA
	10:00am - 11.15am	Speke	Margaret Thompson Medical Centre, East Millwood Road, Speke, L24 6TH
	10.15am-11.30am	Toxteth	Abercromby Health Centre, Grove Street, L7 7HG
	10:30am - 11.15am	Hunts Cross 1-2-1appointment only	Hunts Cross Health Centre, Hillfooot Road, Hunts Cross L25 0ND
	10.00am - 11.15am	Tuebrook 1-2-1 appointment only	Lloyds Pharmacy, 629-631 West Derby Road, L13 8AG
	11:00am - 12:30pm	Wavertree	Asylum Link, 7 Overbury Street, Liverpool, L7 3HJ
ā	PM 1:00pm - 2.45pm	Dovecot 1-2-1 no app. needed	Dovecot MAC, Dovecot Parade, L14 9BA
	1:00pm - 3.15pm	Speke	Neighbourhood Health centre, 75 South Parade, Speke, L24 2SF
	1:00pm - 2:15pm	Kirkdale	Kirkdale Community Centre, 238 Stanley Road, L5 7QP
	2:00pm-3.30pm	Norris Green 1-2-1 no app. needed	Broadway One Stop, Broad Lane, Liverpool, L11 1JB
	2:00pm - 3:45pm	Aintree 1-2-1 appointment only	Aintree Group Practice, Moss Lane, Liverpool, L9 8AL
	3.00pm - 4.45pm	Walton	Spellow Library, County Road, L4 3QF
	3:30pm - 4.45pm	Toxteth	Archer Hall, 68 Upperhill Street, Liverpool, L8 1YR
	4:00pm - 5:15pm	Childwall	Childwall Library, Childwall Fiveways, Liverpool, L15 6YG
	4:00pm - 5.00pm	Norris Green	Clubmoor Children's Centre, Utting Avenue East, L11 1DY
	5:15pm - 6.45pm	Walton	Breeze Hill Neighbourhood Health Centre, 3 Rice Lane, Liverpool, L9 1AD
	5.30pm - 7.00pm	Toxteth	Princes Park Health centre, Bentley Road, Liverpool, L8 0SY
	THURSDAY	GROUPS	
	12:00pm - 12.45pm	Walton	Walton Sports Centre, Walton Hall Avenue, L4 9XP
	1:00pm - 2:15pm	Garston	Mather Avenue Surgery, 584 Mather Avenue, Liverpool, L19 4UG
	1:00pm - 2:15pm	Anfield	Mere Lane Medical Centre, Anfield, Liverpool, L5 0QW
	FRIDAY	GROUPS	
₹	AM 9:00am - 10.15am	Dingle	Mattew Arnold Primary School, Dingle Lane Childrens Centre, L8 9UB
	9:00am - 10.15am	Gateacre 1-2-1appointment only	Gateacre Brow Surgery, 1 Gateacre Brow, L25 3PA
	10:00am - 11.15am	Croxteth	Croxteth Family Health Clinic, Altcross Road, L11 0BS
	10:00am - 10:45am	Kensington	Kensington Park Neighbourhood Centre, 157 Edge Lane, Liverpool, L7 2PF
	10.45am - 11.45am	City Centre 1-2-1 appointment only	St James Health Centre, 29 Great George Street, Liverpool L1 5DZ
ď	PM 1:00pm - 2.15pm	Sefton Park	Sefton Park Library, Aigburth Road, L17 4JS
	1:30pm - 2.45pm	Fazakerley	Church Lounge, St Pauls Church, Formosa Drive, Fazakerley, L10 7LB
	2:00pm - 3:30pm	Kirkdale	Stanley Medical Centre, 60 Stanley Road, Kirkdale, Liverpool, L5 2QA
	2:30pm-3:45pm	Netherley	Netherley Health Centre, Middlemass Hey, L27 7AF
	2:30pm-3:45pm	Tuebrook	Green Lane Medical Centre, Tuebrook, L13 7DY
	2:30pm - 3.45pm	Woolton	Woolton House Medical Centre, Woolton Street, L25 5JA
	SATURDAY	GROUPS	
₹	AM 10:00am - 11.45am	Old Swan	Old Swan NHS Walk in Centre, St Oswald's Street, Old Swan, L13 5XG

Appendix D. Participant information sheet







ROY CASTLE LUNG CANCER RESEARCH PROGRAMME

200 London Road, Liverpool L3 9TA, UK
Tel No. 0151 794 8957
Mobile No. 07******** (tbc)

Lung disease risk awareness and future smoking behaviour Participant Information Sheet

You are being invited to take part in a research project but before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully.

What is the purpose of this research?

The main purpose of this study is to find out whether talking to people who smoke about the risk of developing lung disease, motivates them to quit smoking. The findings of the study could help researchers learn more about ways of supporting people to quit smoking, which could reduce lung disease risk among people in the future. Secondly, with your permission, we would also like to use the information you give us to prepare a risk-assessment model, using lifestyle information, which will help us find out an individual's risk of developing certain lung diseases. This can help identify disease earlier, making treatment more effective.

Why have I been chosen?

We are approaching and inviting people to participate in the study who are aged 18-60 years old, without a current diagnosis of lung cancer, and attend FagEnds drop-in centres in Liverpool.

Do I have to take part?

No. It is up to you to decide whether or not to take part. If you do, you will be given this information sheet to keep and asked to sign a consent form.

What will happen to me if I take part?

We would like you to complete two questionnaires. The first questionnaire will ask you questions about your lifestyle, your smoking habits and your thoughts about quitting smoking and lung disease. You will also be provided with information regarding the risk of lung disease. We will contact you in six months time either by telephone or post, to request you to complete a second questionnaire, similar to the first but shorter. A small number of participants will additionally be randomly asked to take part in a telephone interview with the researcher, whereby the researcher will ask you a number of questions about receiving information on lung disease and your views on quitting smoking. However, this is completely optional.

What are the possible benefits and risks of taking part?

We cannot promise that you will benefit from taking part, although the information you give us might help improve the treatment and services provided for people who are either currently smoking or giving up smoking. Everyone who takes part will be given information regarding lung disease risk, which may be of interest to some people wishing to stop smoking.

Will my taking part in the study be kept confidential?

Yes. All the information that is collected about you during the course of the study will be kept strictly confidential in accordance with the Data Protection Act 1998. Throughout the length of the Liverpool Lung Project, we would also like to see how you are by accessing information from your medical records, your GP, NHS Central Register and local medical registers, but only with your permission.

What will happen if I do not want to carry on with the study?

You are free to withdraw at any time and without giving a reason. A decision to withdraw or a decision not to take part, will not affect the standard of care or advice you receive.

What will happen to the results of the research study?

The results of this research will be published in scientific journals but all information will be used anonymously.

Who is funding the research?

This research is funded by the Roy Castle Lung Cancer Foundation and Liverpool Primary Care Trust.

Who has reviewed the study?

This study is sponsored by the University of Liverpool and has been approved by Liverpool Research Ethics Committee.

Contact Details

If you would like to discuss the study in more detail or have any concerns, please contact the lead researcher (Frances Sherratt) on 0151 794 8957 or 07*** *** *** (to be confirmed). Alternatively, you can email the project at quitrisk@liverpool.ac.uk

Thank you for considering taking part and taking the time to read this information sheet.

Appendix E. Consent form







MPI:

ROY CASTLE LUNG CANCER RESEARCH PROGRAMME

200 London Road, Liverpool, L3 9TA, UK Tel No. 0151 794 8920

Liverpool Lung Project: Lung Cancer Risk Awareness and Future Smoking Behaviour

Consent Form

Please read carefully and initial each box	if you agree
I have read the Participant Information Sheet (Version 3.0, dated 30.04.13) for and have been given a copy to keep. I have had an opportunity to discuss understand why the research is being done. I know how to contact the research	the study, ask questions and
I also understand that my participation is voluntary and that I can withdraw without giving a reason and without it affecting the standard of care I receive.	v from the study at any time,
I agree to provide information about myself and I understand that all confidentially and stored securely at the University of Liverpool Cancer Building).	
I give permission to be contacted by the Liverpool Lung Project (LLP) with resprojects.	gard to other, future research
I give permission for the researcher to contact me by telephone, to ask me information on lung cancer and quitting smoking. I understand that these recorded and that the audio recording will be destroyed, once it has been written.	telephone interviews will be
I agree and understand that information held and maintained by The Heal Centre and other agencies keeping patient medical records may be used to be my health status.	
I agree and understand that some of the research projects may be carried or commercial organisations.	ut by researchers working for
Name (BLOCK CAPITALS):	Date:
Address:	Telephone (Home):
	Telephone (Mobile):
Signature:	Best time to telephone you (please tick): Morning Afternoon Evening
Consented by (BLOCK CAPITALS):	Date:
Signature:	
The contact details of the lead researcher are:	la Laca Cara Davida

Name: Roy Castle Lung Cancer Research Programme, Fran Sherratt Address: Tel: 0151 794 8957 The University of Liverpool Cancer Research Centre, Email: quitrisk@liverpool.ac.uk 200 London Road, Liverpool, L3 9TA

Appendix F. Baseline questionnaire







QUESTIONNAIRE

The Liverpool Lung Project:

Lung Cancer Risk Awareness and Future Smoking Behaviour

1. What is your r	ame? (pleas	e write your n	ame here)			
2. Are you? (plea ☐ Male (1) ☐	se tick one b Female (2)	ох)				
3. What is your	date of birth	d d r	m m y y]		
4. Are you? (plead ☐ Single (1) ☐		ox) □ Living togetl	ner ⑶ 🔲 Widov	ved ⑷ Divo	rced/separated	(5) Other (6)
5. How would yo ☐ White British (1) ☐ Black African (5) ☐ Asian Pakistani ☐ Mixed Caribbe	☐ White ☐ Black	Other (6) Chinese (10)	☐ White Other (3 ☐ Asian British	☐ Asian In		ck Caribbean (4) ian Other (9) ixed Asian (13)
6. What is the high None (88) □ 1st Degree (5) □ NVQ 4-5/HNC/	l 1-5 O Levels l Higher Degr	s/GCSEs (1) ee (6)	u have? (please 5+ O Levels/G NVQ 1 (7) Professional q	CSEs (2)	1 A Level (3) C	12+ A Levels (4) 1 NVQ 3 (9) 1 Other (77)
7. Can you think (please tick on ☐ Yes (1)		activities you	've done in whic	h you've been i	n contact with a	asbestos?
8. Have you ever	been diagno	sed with the f	following? (pleas	e tick for each)		
Pneumonia			☐ Yes (1)		No (2)	
Chronic obstructi	ve pulmonary	/ disease (COP			No (2)	
Lung Cancer			☐ Yes (1)		No (2)	
Cancer, other tha	n lung cance		☐ Yes (1)		No (2)	
9. Have any of you			ee relatives had rite the age at w			YES, please
Family member:	Father (1)	Mother (2)	Brother (3)	Sister (4)	Son (5)	Daughter (6)
Age diagnosed:						
10.Have you smo ☐ Yes (1)	ked one or m	ore cigarettes	s within the past	week? (please	tick one)	
11. If you have al	ready quit sn	noking, please	write your quit	date here:	d d m	m y y

12. In your own words, please describe your smoking status (e.g. current or former smoker)
13. How old were you when you started smoking? (please write age in the box)
14. Do you live with any other smokers? (please tick one box) ☐ Yes (1) ☐ No (2)
15. Are you thinking about giving up smoking? (please tick one box) □ I've already quit (1) □ Yes, definitely (2) □ Yes, possibly (3) □ Not really (4)
16. DURING THE TIME PERIOD YOU SMOKED THE MOST
a) How soon after you woke up did you smoke your first cigarette? (please tick one) ☐ Within 5 minutes (1) ☐ 6-30 minutes (2) ☐ 31-60 minutes(3) ☐ After 60 minutes (4)
b) Did you find it difficult not to smoke in places where it isn't allowed, such as in the library? ☐ Yes (1) ☐ No (2)
c) Which cigarette would you have hated most to give up? (please tick one) ☐ The first one in the morning (1) ☐ All others (2)
d) Did you smoke more often during the first hours after waking than during the rest of the day? \square Yes $_{^{(1)}}$ \square No $_{^{(2)}}$
e) Did you smoke if you were so ill that you were in bed most of the day? ☐ Yes (1) ☐ No (2)
f) How many cigarettes per day did you smoke? (please write number in box)
17. Would you say the chances of the average smoker getting lung cancer in future are: (please tick one) ☐ Very low (1) ☐ Somewhat low (2) ☐ Moderate (3) ☐ Somewhat high (4) ☐ Very high (5)
18. How often do you worry about getting lung cancer? (please tick one box) ☐ Rarely or never (1) ☐ Sometimes (2) ☐ Often (3) ☐ All the time (4)
19. Would you say the average smoker has: (please tick one box) ☐ About the same lung cancer risk as a non-smoker? (1) ☐ A little higher lung cancer risk than a non-smoker? (2) ☐ Twice the lung cancer risk than a non-smoker? (3) ☐ Five times the lung cancer risk than a non-smoker? (4) ☐ Ten times the lung cancer risk than a non-smoker? (5)
20. Overall, how many people who get lung cancer do you think will live at least 5 years? (please tick one) ☐ Less than a quarter (1) ☐ About a quarter (2) ☐ About half (3) ☐ About three quarters (4) ☐ Nearly all (5)
21. Would you say your chances of getting lung cancer in the future are: (please tick one box) ☐ Very low (1) ☐ Somewhat low (2) ☐ Moderate (3) ☐ Somewhat high (4) ☐ Very high (5)
Thank you very much for taking the time to complete this questionnaire
FOR OFFICE USE ONLY MPI No: Location: Advisor:

FOR OFFICE USE ONLY MPI No:		Location: Advis	sor:
Baseline Risk Projection:	5-year risk score:	Age 70 risk score - smoking:	Age 70 risk score - cessation:
Yes (A) / No (B)			
Permission given to request information from FagEnds:	Fagerstrom:	CO reading:	Previous attendances in past 6 months:
Yes / No			







Thank you for taking part in this study

The study is funded by the Roy Castle Lung Cancer Foundation and Liverpool Primary Care Trust, and it aims to improve stop-smoking services available to people who want to quit smoking. Quitting smoking is the best thing you can do to avoid many serious diseases, including lung cancer. Lung cancer is the most commonly diagnosed cancer worldwide. Around 41,400 people are diagnosed with this disease in the UK each year and survival rates from lung cancer are very low.

the benefits of quitting smoking, you could speak to your FagEnds advisor or visit the FagEnds Around 9 out of 10 lung cancer cases are due to smoking cigarettes. For more information on website at www.stopsmoking.org.uk

Version 2.0 11.03.13

Appendix H. Follow-up questionnaire







MPI:

SIX-MONTH FOLLOW-UP QUESTIONNAIRE The Liverpool Lung Project:

Lung Cancer Risk Awareness and Future Smoking Behaviour

PLEASE COMPLETE THIS FORM AND RETURN IN THE PRE-PAID ENVELOPE PROVIDED THANK YOU FOR TAKING PART IN THIS STUDY

	The state of the s	one or more cigarett	es within the past wee	ek? (please tick o	ne)			
2.	If you have already	quit smoking, pleas	e write your quit date	here:	d	m r	m y	У
	and the second second	bout giving up smoki Yes, definitely (2)	ng? (please tick one b ☐ Yes, possibly (3)	ox) Not really (4)	I	⊐ Defi	nitely n	ot (5)
		chances of the avera omewhat low (2)	age smoker getting lui ☐ Moderate ⑶	ng cancer in futur Somewhat hi			tick or ery hig	, , , , , , , , , , , , , , , , , , ,
	How often do you Rarely or never (1)	worry about getting	lung cancer? (please to S (2)	•	[⊐ All th	he time	! (4)
	About the same lur A little higher lung Twice the lung cand Five times the lung	e average smoker han ng cancer risk as a now cancer risk than a now cer risk than a non-sn cancer risk than a no cancer risk than a no	n-smoker? (2) noker? (3) n-smoker? (4))				
	one)	5	g cancer do you think					
	5 5 5	ur chances of getting omewhat low (2)	lung cancer in the fut ☐ Moderate (3)	ure are: (please t			ery high) (5)
	Have you ever used Yes, currently (1)		ette? (please tick <u>one</u>) s, in the past (2)	□ No (3)				
10 <u>on</u>	727	gular cigarettes, hov	v harmful do you thin	k electronic cigare	ettes aı	re? (ple	ease tic	:k
	Less harmful (1)	☐ As harmful (2)	☐ More h	armful (3)	□ Do	n't kno	OW (4)	

PLEASE COMPLETE THE REMAINING QUESTIONS, <u>ONLY IF YOU HAVE EVER USED AN</u> <u>ELECTRONIC CIGARETTE</u>:

11. When did you last use an	electronic cigarette, ev	en one puff? (p	lease tick <u>o</u>	<u>ne</u>)
\square Within the past month (1)	☐ Within the past 1-6			than 6 months ago (3)
12. How many times a day do	/did you use an electro	onic cigarette? (please tick	one)
	-15 times a day (2)	☐ 16-25 times		☐ More than 25 times (4)
13. Do/did you use an electro	(57)	10 70.70		
☐ A long term or permanent i				
☐ A stop-smoking aid, which		y for a short tim	e? (2)	
☐ Other, please tell use your	view: (3)			
14. What best describes your		100		
		•••••		
15. Do/did you find yourself s	moking the electronic	cigarette anywh	nere that yo	u wouldn't normally
15. Do/did you find yourself s smoke a regular cigarette? Pl		_	nere that yo	u wouldn't normally
160 V V				_
160 V V	ease tell us your view:			_
160 V V	ease tell us your view:			_
160 V V	ease tell us your view:			_
160 V V	ease tell us your view:			_

PLEASE RETURN THIS COMPLETED FORM IN THE PRE-PAID ENVELOPE PROVIDED THANK YOU AGAIN FOR TAKING PART IN THIS STUDY

Appendix I. Follow-up letter





Liverpool Clinical Commissioning Group

ROY CASTLE LUNG CANCER RESEARCH PROGRAMME 200 London Road, Liverpool, L3 9TA, UK Tel No. ____

Date:
Dear
You may recall completing a questionnaire within a FagEnds drop-in about 6 months ago, which was associated with the Roy Castle Lung Cancer Research Programme. You may remember at the time, I explained I would be contacting all participants 6 months after completing the questionnaire. The main aim of this is to find out more about your experience of quitting smoking over the past six months, whether you have successfully quit or whether you have found quitting smoking challenging. By doing this, we hope to find out more about the things that people find helpful or difficult when trying to quit smoking.
I did try to get in contact with you by telephone to ask you these few and final questions but have unfortunately been unsuccessful. Therefore, I have enclosed the follow-up questionnaire for you with a pre-paid envelope. If you could please complete the one page questionnaire and return it to me in the pre-paid envelope provided, this would be greatly appreciated. As before, if there are any questions you feel uncomfortable answering, you can just skip to the next question.
If you have any questions or you would prefer to go through these questions with me over the telephone at a time that's convenient for you, please feel free to contact me on any time between 9am and 4.30pm Tuesdays or Thursdays.
I look forward to hearing from you and thank you again for taking part in this study.
Yours sincerely,
Frances Sherratt PhD Student

Appendix J. Interview schedule

- For some people, there are lots of reasons to quit smoking but there can also be plenty of barriers and challenges too – tell me about how this is for you and your experiences.
- 2. Have you tried to quit in the past? If yes, when and what do you think triggered you to start smoking again? Do you feel that anything has changed this time?
- 3. If no, what made you decide to quit smoking this time?
- 4. How did you react when you were given the information on lung cancer risk?
 What kind of feelings or emotions did you experience?
- 5. (Recap results with participant) Did you expect your risk to be this level if you carried on smoking? Why do you think you felt like that?
- 6. (Recap results with participant) Did you expect your risk to be this level if you quit smoking? Why do you think you felt like that?
- 7. Has receiving the lung risk information affected your motivation to quit smoking? Do you feel more or less motivated? In what ways?
- 8. How did you feel about the way the information was presented to you?

Appendix K. Ethical Approval



NRES Committee North West - Liverpool Central

HRA NRES Centre - Manchester 3rd Floor Barlow House 4 Minshull Street Manchester M1 3DZ

> Telephone: 0161 625 7818 Facsimile: 0161 625 7299

12 February 2013

PROFESSOR JOHN FIELD
UNIVERSITY OF LIVERPOOL
ROY CASTLE LUNG CANCER RESEARCH PROGRAMME, ROY CASTLE BUILDING, 200
LONDON ROAD
LIVERPOOL
L3 9TA

Dear PROFESSOR FIELD

Study title: Lung Cancer Risk Awareness and Future Smoking

Behaviour

REC reference: 13/NW/0087
Protocol number: UoL000942
IRAS project ID: 123793

The Research Ethics Committee reviewed the above application at the meeting held on 06 February 2013. Thank you for attending to discuss the application.

We plan to publish your research summary wording for the above study on the NRES website, together with your contact details, unless you expressly withhold permission to do so. Publication will be no earlier than three months from the date of this favourable opinion letter. Should you wish to provide a substitute contact point, require further information, or wish to withhold permission to publish, please contact the Co-ordinator Mrs Carol Ebenezer, nrescommittee.northwest-liverpoolcentral@nhs.net.

Ethical opinion

The Chair welcomed you and Mrs Sherratt to the REC and thanked you for attending to discuss the study. You agreed to the presence of the observer for the discussion of the application. The Committee told you that this is a very worthwhile study.

You explained that this is linked to the Liverpool Lung project but is a separate application as you submitted it as an amendment and were asked to submit as a fresh application.

The Committee asked whether FAGENDS has suitable staff to deal with any participants who become distressed, and you explained that they are trained in dealing with people who are trying to give up smoking. The Committee enquired what would happen if a participant had a bad prognosis and you explained that this is not how it is put to them They will be given information such as "if you continue to smoke, you may be at more risk in 20 years' time", and "if you stop the risk will be xxxx" The risk model is run in several places,

including in GP surgeries. The participants attend because they know already that they are at risk. They will be given information in a gentle and supportive manner and will be told that if they develop certain symptoms, they should see their GP. You confirmed that you are au fait with other smoking related diseases to warn against. FAGENDS is set up around lung cancer but participants are told of the whole range of possible diseases.

You confirmed for the Committee that interpreters are not available for this study but acknowledged that studies should be done on non-English speakers in the future.

The Committee asked whether the questionnaire has been validated or piloted. You explained that it is the questionnaire from the Liverpool Lung Project as approved by this Committee. It has been adapted over the years after interactions with the Committee. You go through the questionnaires with the participants so if there is any difficulty in understanding you can explain.

The Committee asked for clarification of the consenting process. You explained that attendees will be given a Participant Information Sheet when they come in and will then go through the FAGENDS process. They will then meet with the researcher. The Committee asked whether they could be given at least 24 hours to decide whether to participate, and you explained that you might never see them again as the drop-out rate is quite high. The Committee accepted this.

The Committee queried retaining the data for future projects, and you explained that you have kept all of your Consent Forms the same. It is fully explained and they can say no if they wish. If, in the future, you research into biomarkers, the data will be useful.

The Committee asked who will estimate the risk, and you explained that the researcher will enter the data into a computer programme so the data goes through a validated risk model. You have two statisticians who have done a power calculation for the study and you want to demonstrate that telling people that they are at risk will help them to give up. You hope to get sufficient evidence to use this in the FAGENDS project, and the more participants you get, the higher the power calculation will be, and the more chance of implementing the change.

You had no questions for the Committee

The members of the Committee present gave a favourable ethical opinion of the above research on the basis described in the application form, protocol and supporting documentation, subject to the conditions specified below.

Ethical review of research sites

NHS Sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations

It is responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The documents reviewed and approved at the meeting were:

Document	Version	Date
Covering Letter		21 January 2013
Evidence of insurance or indemnity		02 August 2012
Investigator CV	Field	
Investigator CV	Jude	
Investigator CV	Sherratt	
Letter from Sponsor		21 January 2013
Other: Letter from funder		19 December 2012
Other: Information for both groups	1	01 January 2013
Other: Risk Calculation cards	1	01 January 2013
Other: List of acronyms	1	01 January 2013
Other: Evidence of peer review		16 January 2013
Other: Confirmation letter from FagEnds		22 November 2012
Other: LLP fav op of Amend 23		28 November 2012
Other: LLP amend 3 protocol pg 4		08 November 2012
Other: sponsor study submission request		10 January 2013
Participant Consent Form	1.1	01 January 2013
Participant Information Sheet	1.1	01 January 2013
Protocol	1.1	01 January 2013
Questionnaire: Baseline	1.1	01 January 2013
Questionnaire: follow up	1.1	01 January 2013
REC application	3.4	21 January 2013

Membership of the Committee

The members of the Ethics Committee who were present at the meeting are listed on the attached sheet.

Carmel Dersch knows Professor Field

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Reporting requirements

The attached document "After ethical review – guidance for researchers" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- · Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

<u>Feedback</u>

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

Further information is available at National Research Ethics Service website > After Review

13/NW/0087

Please quote this number on all correspondence

We are pleased to welcome researchers and R & D staff at our NRES committee members' training days - see details at http://www.hra.nhs.uk/hra-training/

With the Committee's best wishes for the success of this project.

Yours sincerely

Mrs Julie Brake

Chair

Email: nrescommittee.northwest-liverpoolcentral@nhs.net

List of names and professions of members who were present at the Enclosures:

meeting and those who submitted written comments

"After ethical review - guidance for researchers"

Mrs Lindsay Carter Copy to:

Eldenezh.

Mrs Heather Rogers

NRES Committee North West - Liverpool Central

Attendance at Committee meeting on 06 February 2013

Committee Members:

Name	Profession	Present	Notes
Mrs Julie Brake	Specialist Diabetes Nurse / Chair	Yes	
Dr Murthy Burra	Consultant Anaesthetist	Yes	
Mrs Hannah Chambers	Lay Member	Yes	
Miss Carmel Dersch	Lay Member	Yes	
Mr Derek Hollingsbee	Pharmacist	Yes	
Professor Donald Kelly	Retired Vet	Yes	
Mr Frank Killen	Lay Member	No	
Dr Charles King	Lay Member	Yes	
Miss Karen Knowles	Biomedical Scientist	Yes	
Mr Duane Mellor	Dietitian	Yes	
Mrs Jennifer Newman	Consumer Liaison Manager	No	
Dr Stephen Pennefather	Consultant Anaesthetist	No	
Dr Lyvonne Tume	Senior Nursing Research Fellow Paediatric ICU	Yes	
Dr Helen Wong	Statistician	Yes	

Also in attendance:

Name	Position (or reason for attending)
Miss Anna Bannister	Assistant Co-ordinator
Mrs Carol Ebenezer	Committee Co-ordinator
Rebecca Thompson	Observer



Certificate of Attendance

Frances Sherratt

attended

Good Clinical Practice (GCP) Refresher: A practical guide to ethical and scientific quality standards in clinical research

on 21/02/2014

Sessions include:
GCP: the standards and why we have them
Study overview
Informed consent
Essential documents
GCP recent changes



Appendix M. Variable transformation

This appendix describes some of the questionnaire variables, which were transformed due to low cell frequencies. In cases whereby low cell frequencies exist, it is preferable to transform the data to avoid low cell frequencies and therefore, enable the development of robust regression models (A. Field, 2013). The reformed variables are referred to throughout the thesis results; this appendix simply highlights the frequencies in relation to the original variables.

Firstly, socio-demographic variables were addressed. Table 17 displays the list of socio-demographic variables selected for recoding due to low cell frequencies, including ethnicity, marital status, highest educational attainment, and socio-economic status. The method of ethnicity and highest educational attainment recoding has been previously adopted elsewhere (Sherratt et al., 2015). Ethnicity was transformed into two values: (1) White (White British, White Irish, and White Other); (2) Other (Black British, Black Caribbean, Black African, Black Other, Asian British, Asian Indian, Asian Other, Asian Pakistani, Ethnic Chinese, Ethnic Other, Mixed African, Mixed Asian, Mixed Caribbean, Mixed Other, and Other).

The variable levels for highest educational attainment were recoded into the following two groups: (1) Basic or no qualifications (i.e. General Certificate of Secondary Education level [GCSE] or below) (None, 1-5 O Levels/GCSEs, or 5+ O Levels/GCSEs); (2) Higher qualifications and other (1 A level, 2+ A levels, First degree, Higher degree, NVQ 1, NVQ 2, NVQ 3, NVQ 4-5/HNC/HND, Professional qualification, and Other).

Table 17. Original values for socio-demographic variables among follow-up datasets

Baseline variable	Baseline current smokers $(n = 297)$	Baseline recent former smokers $(n = 216)$
Ethnicity †	(n-297)	(n-210)
White British	260 (88.4)	192 (89.3)
White Irish	6 (2.0)	4 (1.9)
White Other	5 (1.7)	1 (0.5)
Black British	0 (0.0)	0 (0.0)
Black Caribbean	0 (0.0)	0 (0.0)
Black African	0 (0.0)	2 (0.9)
Black Other	7 (2.4)	5 (2.3)
Asian British	0 (0.0)	0 (0.0)
Asian Indian	0 (0.0)	0 (0.0)
Asian Other	0 (0.0)	· · · ·
Asian Odiei Asian Pakistani	• •	1 (0.5) 0 (0.0)
Ethnic Chinese	2 (0.7)	· · ·
Ethnic Other	0 (0.0)	0 (0.0) 1 (0.5)
	0 (0.0)	· · ·
Mixed African	2 (0.7)	0 (0.0)
Mixed Asian	0 (0.0)	2 (0.9)
Mixed Caribbean	3 (1.0)	4 (1.9)
Mixed Other	1 (0.3)	1 (0.5)
Other	8 (2.7)	0 (0.0)
Marital status †	1 (2)	00 (10 0)
Single	157 (53.2)	92 (43.0)
Married	50 (16.9)	57 (26.6)
Living together	44 (14.9)	27 (12.6)
Divorced/separated	33 (11.2)	34 (15.9)
Other	5 (1.7)	3 (1.4)
Widowed	6 (2.0)	1 (0.5)
Highest educational attainment		
None	55 (19.0)	43 (20.0)
1-5 O Levels/GCSEs	66 (22.8)	56 (26.0)
5+ O Levels/GCSEs	15 (5.2)	15 (7.0)
1 A level	5 (1.7)	2 (0.9)
2+ A levels	17 (5.9)	5 (2.3)
First degree	20 (6.9)	11 (5.1)
Higher degree	6 (2.1)	5 (2.3)
NVQ 1	6 (2.1)	3 (1.4)
NVQ 2	25 (8.6)	22 (10.2)
NVQ 3	25 (8.6)	14 (6.5)
NVQ 4-5/HNC/HND	13 (4.5)	14 (6.5)
Professional Qualification	15 (5.2)	10 (4.7)
Other	22 (7.6)	15 (7.0)
Socio-economic status †		
Most deprived	255 (86.1)	182 (84.3)
Above average	24 (8.1)	25 (11.6)
Average	15 (5.1)	8 (3.7)
Below average	2 (0.7)	1 (0.5)
Least deprived	0 (0.0)	0 (0.0)

[†] Figures do not equate to 297 and 216, in the respective baseline current smokers and baseline recent former smokers datasets, due to some missing data

It was also necessary to recode marital status, due to several low cell frequencies. Three levels were defined, as follows: (1) Other (Divorced, Separated, Other, and Widow); (2) Single (Single), and; (3) Married or living together (Married, Living together). The distribution in regards to socio-economic status was additionally uneven, with a number of low cell frequencies. It was therefore necessary to recode socio-economic status into two categories: (1) Most deprived (Most deprived); (2) Least deprived (Above average, Average, Below average, Least deprived).

All variables associated with lung cancer risk perception were additionally reformed due to low cell frequencies. Table 18 provides the frequencies among lung cancer risk perception variables with low cell frequencies, prior to transformation. Transformation of lung cancer risk perceptions were considered by exploring both baseline and follow-up lung cancer risk perceptions across both follow-up datasets. It was necessary to reform repeated measure variables at baseline and follow-up to ensure consistency. Again, the reformed variables are referred to throughout the thesis results.

Firstly, perceived personal lung cancer risk was recoded. The results displayed that the cell frequencies for "Very low" and "Very high" were considerably low in comparison to some other levels. The variable levels were therefore reduced to the following three levels: (1) Low (Very low, Low); (2) Moderate (Moderate); (3) High (High, Very high).

Secondly, perceived average smoker lung cancer risk levels were recoded, as few participants responded to a number of the lower levels, particularly at follow-up. The

variable levels were therefore recoded into three levels: (1) "Very low to moderate" (Very low, Low, Moderate); (2) Somewhat high (Somewhat high); (3) Very high (Very high).

Thirdly, perceived relative risk of lung cancer was recoded, as few follow-up participants viewed relative risk as "About the same as a non-smoker". The five levels were recoded into four, including: (1) About the same to a little higher than a non-smoker (About the same risk as a non-smoker, A little higher risk that a non-smoker); (2) Twice as high risk than a non-smoker (Twice as high risk than a non-smoker); (3) Five times higher risk than a non-smoker (Five times higher risk than a non-smoker); (4) Ten times higher risk than a non-smoker (Ten times higher risk than a non-smoker).

Fourthly, lung cancer worry was considered. A limited number of cell frequencies were apparent in regard to the level, "All the time" among follow-up participants. The variables levels were therefore recoded into three levels, as follows: (1) Rarely or never (Rarely or never); (2) Sometimes (Sometimes); (3) Often to all the time (Often, All the time).

Lastly, perceived lung cancer survival was investigated. The final level for perceived lung cancer survival had a substantially low frequency of cells and therefore, the five variable levels were combined into four levels, as follows: (1) Less than three quarters (Less than three quarters); (2) About a quarter (About a quarter); (3) About half (About half); (4) About three quarters to nearly all (About three quarters, Nearly all).

Table 18. Original values for lung cancer risk perception variables among follow-up datasets

Baseline variable	Baseline cur	Baseline current smokers	Baseline recen	Baseline recent former smokers
	Respondents at baseline	Respondents at follow-up	Respondents at baseline	Respondents at follow-up
	(n = 302)	(n = 187)	(n = 219)	(n = 147)
Perceived personal lung cancer risk †				
Very low	17 (5.9)	6 (3.5)	17 (8.1)	15 (10.4)
Somewhat low	34 (11.9)	14 (8.1)	51 (24.4)	25 (17.4)
Moderate	114 (39.9)	84 (48.8)	90 (43.1)	61 (42.4)
Somewhat high	93 (32.5)	45 (26.2)	46 (22.0)	28 (19.4)
Very high	28 (9.8)	23 (13.4)	5 (2.4)	15 (10.4)
Perceived average smoker lung cancer risk †				
Very low	3 (1.0)	2 (1.1)	0 (0.0)	0 (0.0)
Somewhat low	5 (1.7)	1 (0.6)	2 (0.9)	1 (0.7)
Moderate	64 (22.3)	28 (15.8)	39 (18.5)	20 (13.7)
Somewhat high	103 (35.9)	66 (37.3)	94 (44.5)	45 (30.8)
Very high	112 (39.0)	80 (45.2)	76 (36.0)	80 (54.8)
Perceived relative risk of lung cancer +				
About the same risk as a non-smoker	26 (9.2)	5 (2.9)	11 (5.2)	4 (2.9)
A little higher risk that a non-smoker	31 (11.0)	16 (9.2)	24 (11.4)	10 (7.1)
Twice as high risk than a non-smoker	46 (16.3)	25 (14.4)	42 (20.0)	18 (12.9)
Five times higher risk than a non-smoker	85 (30.0)	45 (25.9)	52 (24.8)	32 (14.8)
Ten times higher risk than a non-smoker	95 (33.6)	83 (47.7)	81 (38.6)	76 (54.3)
Lung cancer worry †				
Rarely or never	87 (29.9)	74 (41.6)	73 (34.1)	62 (42.5)
Sometimes	107 (36.8)	61 (34.3)	87 (40.7)	53 (36.3)
Often	62 (21.3)	30 (16.9)	37 (17.3)	20 (13.7)
All the time	35 (12.0)	13 (7.3)	17 (7.9)	11 (7.5)
Perceived lung cancer survival +				
Less than a quarter	105 (37.1)	n/a	87 (41.8)	n/a
About a quarter	83 (29.3)	n/a	62 (29.8)	n/a
About half	69 (24.4)	n/a	44 (21.2)	n/a
About three quarters	18 (6.4)	n/a	12 (5.8)	n/a
Nearly all	8 (2.8)	n/a	3 (1.4)	n/a
+ Figures do not senists to respondent totals due to some missing date	some missing data			

† Figures do not equate to respondent totals due to some missing data

Appendix N. Initial proportional odds (PO) regression model for prediction of baseline perceived personal lung cancer risk

Baseline variable	Odds	Lower	Upper	P value
	(95% CI)	(95% CI)	(95% CI)	
Age	1.026	1.009	1.043	0.003*
Ethnicity				0.227
Other	0.656	0.331	1.300	
(base = White)				
Smoking status				p<0.001*
Former	0.344	0.238	0.498	
(base = Current)				
Age started smoking	0.983	0.943	1.024	0.418
FTND	1.053	0.974	1.139	0.193
Perceived average smoker lung cancer ri	<u>.sk</u>			
Somewhat high	3.094	1.846	5.187	p<0.001*
Very high	2.545	1.557	4.161	p<0.001*
$(base = Very\ low\ to\ moderate)$				
Perceived relative risk of lung cancer				
Twice as high risk	2.162	1.258	3.715	0.005*
Five times higher risk	1.356	0.787	2.336	0.273
Ten times higher risk	1.091	0.609	1.957	0.769
(base = About the same to a little)				
higher risk)				
Lung cancer worry				
Sometimes	1.292	0.816	2.045	0.275
Often or all the time	1.116	0.734	1.696	0.607
(base = Rarely or never)				

FTND = Fagerström Test of Nicotine Dependence, * p < 0.05

Appendix O. Initial binary logistic regression model for prediction of baseline smoking status

Baseline variable	Odds	Lower	Upper	<i>P</i> -value
	(95% CI)	(95% CI)	(95% CI)	
Age	1.031	1.009	1.054	0.006*
Marital status				
Single	1.586	0.992	2.538	0.054
Married or living together	1.306	0.708	2.410	0.394
(Base = Other)				
Highest educational attainment				0.438
Higher qualifications	0.848	0.559	1.286	
(Base = Basic or no qualifications)				
Living with another smoker				0.018*
Yes	0.572	0.360	0.908	
(Base = No)				
<u>FTND</u>	1.103	0.981	1.241	0.100
<u>Cigarettes per day</u>	0.850	1.002	0.978	1.028
Perceived personal lung cancer risk				
Moderate	0.513	0.304	0.866	0.013*
High	0.208	0.115	0.374	p<0.001*
(Base = Low)				
Perceived average smoker lung cancer ri	<u>sk</u>			
Somewhat high	1.498	0.835	2.688	0.176
Very high	1.027	0.551	1.917	0.932
(Base = Very low to moderate)				
Perceived relative risk of lung cancer				
Twice as high risk	1.669	0.844	3.299	0.141
Five times higher risk	1.525	0.799	2.912	0.201
Ten times higher risk	2.570	1.351	4.888	0.004*
(Base = About the same to a little)				
higher risk)				
Lung cancer worry				
Sometimes	0.758	0.468	1.227	0.260
Often or all the time	0.574	0.334	0.985	0.044*
(Base = Rarely or never)				

 $\overline{\text{FTND}} = \text{Fagerstr\"{o}m}$ Test of Nicotine Dependence, * p < 0.05

Appendix P. Initial binary logistic regression model for prediction of follow-up smoking status among baseline current smokers

Baseline variable	Odds	Lower	Upper	<i>P</i> -value
	(95% CI)	(95% CI)	(95% CI)	
Ethnicity				0.028*
White	3.223	1.137	9.134	
(Base = Other)				
Socio-economic status				0.105
Most deprived	0.429	0.154	1.193	
$(Base = Least \ deprived)$				
Age started smoking	1.042	0.975	1.114	0.222
Living with another smoker				0.114
Yes	0.595	0.312	1.134	
(Base = No)				0.079
FTND	0.886	0.775	1.014	
Perceived relative risk of lung cancer				
Twice as high risk	1.301	0.375	4.510	0.679
Five times higher risk	2.200	0.786	6.157	0.133
Ten times higher risk	4.212	1.550	11.445	0.005*
(Base = About the same to a little)				
higher risk)				

FTND = Fagerström Test of Nicotine Dependence, * p < 0.05

Appendix Q: List of publications and conference abstracts

Sherratt, F., Marcus, M. W., Field, J. K., & Robinson, J. (2016). Application of a lung cancer risk prediction model for the promotion of smoking cessation. *Manuscript under review in American Journal of Health Promotion*.

Purpose. The current project sought to examine whether delivery of lung cancer risk projections (calculated using the Liverpool Lung Project [LLP] risk model) predicted follow-up smoking status. Design. Two single-blinded randomised controlled trials (RCTs). Setting. Stop Smoking Services in Liverpool (UK). Subjects. Baseline current smokers (N = 297) and baseline recent former smokers (N = 216) were recruited. Intervention. Participants allocated to treatment groups were provided with personalised lung cancer risk projections, calculated using the LLP risk model. Measures. Baseline and follow-up questionnaires explored socio-demographics, smoking behavior and lung cancer risk perceptions. Analysis. Bivariate analyses identified significant differences between treatment groups and logistic regression models were developed to investigate the treatment effect on the outcome variables. Results. Lung cancer risk projections were not found to predict follow-up smoking status in the trial of baseline current smokers; however, they did predict follow-up smoking status in the trial of baseline recent former smokers (OR 1.91, 95% CI 1.03 3.55). Conclusion. The current study suggests that lung cancer risk projections may promote abstinence among individuals who have quit smoking, but not motivate smokers to quit.

Sherratt, F., Chen, Y., Field, J. K., & Robinson, J. (2014, Jul). Exploring reactions to risk and uncertainty in the context of smoking and lung cancer. Paper presented at the

XVIII International Sociology Association World Congress of Sociology, Yokohama, Japan.

Research examining the sociology of diagnosis has demonstrated how the provision of a clinical diagnosis from a health professional can promote identification of illness or disease, facilitate behaviour change, and enhance adoption or resistance of illness identities (Jutel and Nettleton, 2011). Further examination of such frameworks could contribute towards achieving a richer understanding of health communication compliancy, thus enabling better management or avoidance of ill health and disease. The current study examines such frameworks within the context of a Stop Smoking Service (FagEnds, Liverpool) and the study has been designed to replicate a recognised model of diagnosis. However, rather than a clinical diagnosis, smokers receive a personalised lung cancer risk assessment – essentially providing them with a diagnosis of uncertainty. The primary aim of this study will be to ascertain whether provision of personalised lung cancer risk information alongside health advice, will smoking cessation. This mixed-method paper will disseminate the enhance findings of a randomised controlled study consisting of ~300 smokers, in which the control arm receive generalised lung cancer risk information and the intervention arm receive a personalised lung cancer risk assessment, using the Liverpool Lung Project Risk Model (Cassidy et al., 2008). It is anticipated that provision of personalised lung cancer risk information may encourage behaviour change i.e. smoking cessation and long-term maintenance of this change at six-month follow-up. In support of the aforementioned quantitative survey, qualitative interviews will also be conducted with a selection of participants (N = 30), which aims to provide further explanation as to the impact of receipt of a diagnosis of uncertainty and the subsequent behaviour and attitudes of which it may entail. Overall, it is anticipated that the findings will contribute towards our understanding of the sociology of diagnosis and may help to inform the development of future health risk communications.

Sherratt, F., Marcus, M., Robinson, J., & Field, J. K. (2014, Apr). Do health risk perceptions influence smoking cessation? Poster session presented at the University of Liverpool Postgraduate Poster Day 2014, Liverpool, UK.

Research examining health risk perceptions and smoking cessation has rendered mixed results regarding the strength of this relationship. A better understanding of this relationship could inform effective health risk communication among smokers. This poster presents preliminary results from two aspects of the PhD: questionnaires (N = 521) and telephone interviews (N = 24), both with smokers. Although results highlighted the complexity of the relationship, they suggested that health risk perceptions were a smoking cessation contributor. The final component of the PhD - six-month follow-up questionnaires, will enhance our understanding of the impact of risk perceptions on smoking cessation, and help to inform future risk communications.

Sherratt, F., Chen, Y., Marcus, M., Robinson, J., & Field, J. K. (2013, Nov). Smokers' lung cancer risk perceptions and smoking cessation success: A longitudinal study. Poster session presented at the 9th National Cancer Research Institute (NCRI) Cancer Conference, Liverpool, UK.

Background. In the UK, lung cancer accounts for 6% of all deaths (Office for National Statistics, 2011) and smoking is responsible for 86% of lung cancer incidence (Parkin,

2011). Thus, smoking cessation has been highlighted as the most effective strategy to reduce lung cancer risk (Thun, 2010). One suggested explanation as to why individuals continue to smoke despite being informed of the potential harms, is due to individuals underestimating personal risk of smoking-related disease (e.g. lung cancer); known as unrealistic optimism (Weinstein, 1983, 1984). Cross-sectional surveys demonstrated an association between unrealistic optimism and lower have motivation to quit smoking (Dillard, McCaul, & Klein, 2006; Weinstein, Marcus, & Moser, 2005). The current study aims to examine the role and impact of unrealistic optimism for smokers attending stop-smoking services, by utilising a longitudinal design. Methodology. Smokers (N=600) will be recruited from Roy Castle FagEnds stop-smoking services across Liverpool, currently contemplating or actively quitting smoking. Baseline and six-month follow-up questionnaires will be completed, whereby the following will be measured: smoking status, lung cancer risk perceptions, and objective lung cancer risk (calculated using the validated Liverpool Lung Project lung cancer risk prediction model [Cassidy et al., 2006; Raji et al., 2012]). Univariate statistics and multivariate linear regression models will be used to analyse the results. Results. Preliminary results utilising the current sample of 39 participants, consisting of 46% males and 54% females, revealed the median participant age as 43 years (IQR = 31-51). All participants will be classified as high risk of lung cancer if they continue to smoke, however, 64% perceived their risk of lung cancer as lower than the average smoker. The poster will present further results based on the complete sample (N=600). Conclusions. By conducting this study, a better understanding of the

relationship between lung cancer risk perceptions and smoking cessation success is

likely to be achieved. The findings from this study will contribute towards researchers'

understanding of smoking cessation processes and could potentially inform future smoking cessation interventions.

Sherratt, F., Robinson, J., & Field, J. K. (2013, Sep). The significance of lung cancer risk perceptions among individuals quitting smoking: A mixed-method study. Poster session presented at the British Sociological Association Medical Sociology Annual Conference, York, UK.

It is often assumed that individuals will engage in health behaviour change, at which point they perceive their risk as reasonably high; this is frequently advocated by effective risk communication literature (Fischhoff, 1998) and many health behaviour models, such as the Health Belief Model (Rosenstock, 1966). Although some studies support the relationship between risk perceptions and health behaviours (Brewer et al., 2007), others suggest a substantially weaker association (Gerrard, Gibbons, & Bushman, 1996). It has been suggested that this may be due to methodological shortfalls, such as utilising cross-sectional, correlational designs to examine the association, alongside inadequate knowledge of potentially moderating factors (Klein, Zajac, & Monin, 2009). The present study incorporates qualitative and quantitative methods to investigate how relevant and fluid lung cancer risk perceptions can be in the process of smoking cessation; it examines their influence on smoking cessation and the extent to which lung cancer risk communication can be effective in facilitating cessation. Current and former smokers (N=30) will be recruited from Roy Castle FagEnds smoking cessation services across Liverpool. Participants will initially complete a psychosocial questionnaire, measuring lung cancer risk perceptions. Subsequently, they will be provided with objective lung cancer risk calculations, based on risk factors personal to them, by utilising the Liverpool Lung Project (LLP) Risk Model (Cassidy et al., 2008). Lastly, participants will be requested to complete a semi-structured interview, exploring the nature of risk perceptions relevant to smoking cessation and reactions to the provision of lung cancer risk information. The study protocol and the results will be discussed in relation to their implications for smoking cessation services and campaigns.

Sherratt, F., Chen, Y., Field, J. K., & Robinson, J. (2013, May). The impact of providing individualised lung cancer risk projections on smoking cessation success (LCRSC). Poster session presented at the Liverpool Cancer Research UK Centre Annual Meeting, Liverpool, UK.

In the UK, lung cancer accounts for 6% of all deaths (Office for National Statistics, 2011) and smoking is responsible for 86% of lung cancer cases (Parkin, 2011). In the context of smoking, unrealistic optimism is the underestimation of one's own risk of a harmful outcome in comparison to the average smoker; an attribute associated with lower motivation to quit smoking (Dillard, McCaul, & Klein, 2006). LCRSC is a randomised controlled study which aims to examine whether providing smokers with individualised, lung cancer risk projections (using the Liverpool Lung Project Risk Model [Cassidy et al., 2008]) alongside standard care, reduces the frequent discrepancy between perceived and objective risk, thus enhancing smoking cessation. Participants (N=700) will be recruited from Roy Castle FagEnds smoking cessation services across Liverpool and smoking status will be recorded at baseline and 6-month follow-up. The poster will present the study protocol and the potential outcomes will be discussed in relation to their implications for smoking cessation treatment.

Sherratt, F., Chen, Y., Hyde. R., Field, J. K., & Robinson, J. (2012, Nov). Smoking and attitudes to lung cancer: A review of the literature. Poster session presented at the 8th National Cancer Research Institute (NCRI) Cancer Conference, Liverpool, UK.

Background. Smoking cessation has been highlighted as the single most effective strategy to reduce lung cancer risk among the 1.3 billion smokers worldwide (Thun et al., 2010). Despite 63% of smokers in the UK reporting a willingness to quit smoking (Robinson and Harris, 2011) and NICE producing evidence-based guidance on effective smoking cessation strategies, smoking cessation success rates stand at a mere 1-5% of smokers per year (Song et al., 2002). It is clearly necessary to consider alternative strategies to motivate smokers to quit. *Methods*. Here we report the findings from a systematic review of the literature, designed to further explore the relationship between smoking and attitudes towards lung cancer. We consider gaining a greater understanding of the associations between variables and their contribution towards differences in attitudes towards lung cancer risk, and uptake of smoking cessation services and/or quitting. Results. Perceived individual risk has been found to correlate with motivation to quit smoking (Tessaro et al., 1997), which in turn is associated with smoking cessation (Boardman et al., 2005). Perceived risk of lung cancer is elevated among smokers (Rutten et al., 2011), yet smokers have been found to underestimate their personal risk of developing lung cancer when compared to other smokers (Weinstein et al., 2005). Furthermore, current smokers are more likely to attribute lung cancer to smoking-independent factors, such as inheritance (Kaphingst et al., 2009), which may adversely affect perceived risk and cessation motivation. Conclusions. The outcome of this review could potentially inform future smoking cessation

interventions within the Liverpool Lung Project in conjunction with the Roy Castle smoking cessation service (http://www.stopsmoking.org.uk/) and contribute to the reduction of future lung cancer incidence.