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**The biomedicalisation of smoking cessation: A mixed methods  
study of lay perspectives on nicotine addiction**

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## **Abstract**

Biomedical models of addiction are becoming increasingly prevalent in public discourse about cigarette smoking, however there is some concern that smoking is becoming overly medicalised. Neuroscience research has revealed that nicotine use is associated with long-lasting changes in the brain, and these findings are being presented in the media and in advertising campaigns for smoking cessation pharmacotherapies. The “brain disease model of addiction”, which posits that chronic drug use leads to long-term changes in brain networks that make it very difficult to quit, is increasingly being applied to tobacco dependence. This is controversial in the tobacco control field, where population-based strategies have been successful in reducing the prevalence of smoking. Proponents of a biomedical model of tobacco dependence believe that it will lead to increased treatment seeking, more efficacious treatments, and a reduction in stigma. Critics of a biomedical model of nicotine addiction have expressed concerns that it will reduce individual responsibility for smoking, increase stigma, and undermine individuals’ beliefs in their ability to quit. While these competing views on the impacts of biomedical models of addiction have been debated in the academic literature, it is unknown to what extent members of the public endorse a biomedical model of tobacco dependence, and have incorporated it into their everyday understandings of smoking.

This research examined the influence of biomedical discourses of smoking on lay beliefs of Australian smokers and non-smokers about tobacco dependence and its treatment. It also explored daily smokers’ attitudes to the labelling of nicotine addiction as a “brain disease.” A mixed methods approach was used to ascertain the extent to which biomedical understandings of smoking have been incorporated into lay discourse on smoking and quitting; how Australian smokers understand nicotine addiction and the role of the brain; and how endorsement of the brain disease model of addiction may be related to smokers’ attitudes towards smoking cessation and preferences for quitting methods.

The thesis is comprised of three studies. Firstly, a secondary analysis of data from interviews with 55 members of the general public, including never smokers, ex-smokers and current smokers. The data was analysed to ascertain public attitudes on the best methods for quitting smoking. Results revealed that while cessation medications were frequently described as a helpful means to quit, the role of willpower, choice, and motivation were seen as central.

Medications were often described as a “second line of defence”, or as aids to be used alongside counselling or behavioural strategies.

A second qualitative study involved in-depth interviews with 29 daily smokers about their views on smoking cessation and their understanding of the role of the brain in smoking. The results revealed positive attitudes to quitting without assistance. Cessation medications were not perceived as magic bullets, and willpower and personal responsibility were emphasised. While many smokers were aware that smoking affected their brain, few agreed it was a brain disease. Participants expressed concerns that biomedical understandings of smoking could increase the stigma of smoking, diminish personal responsibility for cessation, and deter smokers from seeking treatment.

These qualitative results informed the design of a quantitative survey completed by 1538 Australian smokers. This survey examined endorsement of neurobiological explanations of smoking, and their relationship to self-efficacy and treatment preferences. Approximately one third of participants agreed that smoking was a brain disease and more than half agreed that smoking changes the chemistry of the brain. Endorsement of the brain disease label, and agreement that smoking changes the chemistry of the brain, were associated with greater intention to use cessation medications. However the effect sizes were small, suggesting that beliefs about smoking and the brain may have modest real world impact. Contrary to the claim that promotion of brain-based explanations of smoking will increase feelings of fatalism, this survey shows that agreement with the claim that smoking is a brain disease was associated with higher self-efficacy.

A medical, or chronic disease, model that emphasises the role of neurochemistry in tobacco dependence, while becoming more dominant in academic discourse, was not accepted by most Australian smokers. Concerns about negative consequences of describing smoking in this way, and wariness about the motives behind public health anti-smoking campaigns, were reasons behind this rejection. Consistent with existing literature on lay understandings of health and illness, alternatives discourses that describe it as a matter of willpower, choice, and a habit, remain strongly rooted in public dialogue about smoking. This means that smokers are unlikely to see the terminology of smoking as a “brain disease” positively. However, smokers were interested in information about the effects of smoking on the brain, and further research should investigate constructive ways of presenting this information in health promotion material and

clinical interactions. Biomedical explanations of smoking should acknowledge the agency of individuals and the complexity of addiction to cigarettes in order to be believable and acceptable to smokers.

## **Declaration by author**

This thesis *is composed of my original work, and contains* no material previously published or written by another person except where due reference has been made in the text. I have clearly stated the contribution by others to jointly-authored works that I have included in my thesis.

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## **Publications during candidature**

### **Peer-reviewed papers**

Morphett, K., Lucke, J., Gartner, C. E., Carter, A., Meurk, C., & Hall, W. (2013). Public attitudes toward the treatment of nicotine addiction. *Nicotine and Tobacco Research*, 15(9), 1617-1622.

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Meurk, C., Partridge, B., Carter, A., Hall, W., Morphett, K., & Lucke, J. (2014). Public attitudes in Australia towards the claim that addiction is a (brain) disease. *Drug and Alcohol Review*, 33(3), 272-279.

Morphett, K., Partridge, B., Gartner, C., Carter, A., & Hall, W. (2015). Why Don't Smokers Want Help to Quit? A Qualitative Study of Smokers' Attitudes towards Assisted vs. Unassisted Quitting. *International Journal of Environmental Research and Public Health*, 12(6), 6591.

Morphett, K., Carter, A., Hall, W., & Gartner, C. (2016). A qualitative study of smokers views on brain-based explanations of tobacco dependence. *International Journal of Drug Policy*, 29, 41-48.

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### **Contributions by others to the thesis**

Coral Gartner was involved in the project design and assisted with data interpretation and editing of all chapters. Wayne Hall was involved in project design and assisted with editing of all chapters. Brad Partridge was involved in the study design, assisted with coding of data for Chapter 5, and provide feedback on the associated manuscript. Adrian Carter was involved in the study design and assisted with coding of data for Chapters 7 and 8. He also assisted with data interpretation and editing of all chapters. Jayne Lucke contributed to the project design and was involved in data interpretation and editing of Chapter 5. Carla Meurk assisted with the methods section of Chapter 5.

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tobacco dependence, nicotine addiction, smoking, smoking cessation, lay understandings, attitudes, brain disease model of addiction, medicalization.

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## **Table of Contents**

<b>Abstract.....</b>	<b>ii</b>
<b>Declaration by author.....</b>	<b>v</b>
<b>Publications during candidature .....</b>	<b>vi</b>
<b>Publications included in this thesis.....</b>	<b>viii</b>
<b>Contributions by others to the thesis .....</b>	<b>x</b>
<b>Table of Contents .....</b>	<b>xiii</b>
<b>List of Tables .....</b>	<b>xvii</b>
<b>List of Figures.....</b>	<b>xviii</b>
<b>List of abbreviations used in the thesis .....</b>	<b>xix</b>
<b>Chapter 1. Introduction.....</b>	<b>1</b>
<b>1. Changing public portrayals of smoking.....</b>	<b>1</b>
1.1. Public perceptions of smoking and quitting.....	2
1.2. Overview of the thesis .....	2
1.3. Novel contribution of the thesis.....	5
<b>Chapter 2. The bio/medicalisation of tobacco dependence: Evidence and Arguments</b>	<b>7</b>
2.1. Medicalisation: A brief background .....	7
2.2. Conceptions of tobacco dependence: From “bad habit” to “nicotine addiction” ....	10
2.3. Pharmacotherapy for smoking cessation and the role of the health professional ....	13
2.4. Smoking as a chronic disease .....	16
2.5. The neuroscience of nicotine addiction .....	17
2.6. The brain disease model of addiction .....	20
2.7. Criticisms of the biomedicalisation of tobacco smoking.....	21
2.8. Limits to the biomedicalisation of smoking.....	24
2.9. The role of nicotine addiction in the tobacco “endgame” .....	26
2.10. Lay understandings of smoking and nicotine addiction: the missing link? .....	27
<b>Chapter 3. Lay understandings of smoking and the role of the brain in addiction....</b>	<b>29</b>
3.1. Lay understandings of health and illness: A brief review of the literature .....	29
3.2. Lay understandings of addiction.....	31
3.3. Smokers’ understandings of nicotine addiction.....	35
3.4. Lay beliefs about the brain and behaviour .....	40
3.5. The place of neurobiology in public understandings of addiction.....	44

3.6.	Smoking and the brain: Public portrayals .....	44
3.7.	The place of neurobiology in lay conceptions of smoking .....	50
3.8.	Original contribution of literature review chapters.....	51
<b>Chapter 4.</b>	<b>Method .....</b>	<b>53</b>
4.1.	Mixed methods research: Introduction and justification .....	53
4.2.	Methodological orientation .....	55
4.3.	Overview of study design .....	56
4.4.	Study 1: Public attitudes towards smoking cessation treatments .....	58
4.5.	Study 2: Smokers attitudes towards quitting strategies and neurobiological explanations of smoking .....	63
4.6.	Study 3 – Survey of Australian smokers’ endorsement of brain-based explanations of smoking and relationship to treatment intentions and self-efficacy .....	67
4.7.	Summary .....	73
<b>Chapter 5.</b>	<b>Public attitudes toward the treatment of nicotine addiction .....</b>	<b>74</b>
5.1.	Introduction.....	75
5.2.	Method .....	78
5.3.	Data analysis .....	79
5.4.	Results.....	79
5.5.	Discussion.....	84
5.6.	Concluding remarks on Chapter 5 .....	86
5.7.	Original contribution of Chapter 5.....	88
<b>Chapter 6.</b>	<b>Why don’t smokers want help to quit? A qualitative study of smokers’ attitudes towards assisted versus unassisted quitting.....</b>	<b>89</b>
6.1.	Introduction.....	91
6.2.	Method .....	93
6.3.	Results.....	94
6.4.	Discussion.....	102
6.5.	Concluding remarks on Chapter 6 .....	107
6.6.	Original contribution of Chapter 6.....	110
<b>Chapter 7.</b>	<b>A qualitative study of smokers’ views of brain-based explanations of tobacco dependence. ....</b>	<b>111</b>
7.1.	Introduction.....	112
7.2.	Method .....	115

7.3.	Results.....	117
7.4.	Discussion .....	127
7.5.	Concluding remarks on Chapter 7 .....	130
7.6.	Original contribution of Chapter 7.....	133
<b>Chapter 8. “I’m already bad enough” – A qualitative exploration of smokers reactions to labelling nicotine addiction as a brain disease. ....</b>		<b>135</b>
8.1.	Introduction.....	135
8.2.	Method .....	136
8.3.	Results.....	137
8.4.	Discussion .....	145
<b>Chapter 9. Do neurobiological understandings of smoking influence quitting self-efficacy or treatment intentions? .....</b>		<b>150</b>
9.1.	Introduction.....	150
9.2.	Method .....	151
9.3.	Survey development.....	152
9.4.	Measures .....	153
9.5.	Statistical data analysis .....	156
9.6.	Results.....	157
9.7.	Discussion.....	172
<b>Chapter 10. Discussion.....</b>		<b>177</b>
10.1.	Original contribution of the current research.....	177
10.2.	To what extent has a medical discourse of tobacco dependence been incorporated into lay understandings of smoking and cessation in Australia? .....	178
10.3.	Do smokers believe tobacco dependence is based in their brain, and what are the implications for treatment choices and quitting self-efficacy? .....	181
10.4.	Strengths and limitations of this research .....	183
10.5.	Clinical and public policy implications of the findings .....	186
10.6.	Directions for future research .....	190
<b>References.....</b>		<b>193</b>
<b>Appendix A – Powerpoint presentation provided to participants in Study 2 .....</b>		<b>221</b>
<b>Appendix B – Information sheet and informed consent for Study 2 .....</b>		<b>225</b>
<b>Appendix C – Interview schedule and demographic survey for Study 2 .....</b>		<b>227</b>

<b>Appendix D - Quantitative survey questions.....</b>	<b>231</b>
<b>Appendix E - Taverner Research Privacy Policy.....</b>	<b>243</b>
<b>Appendix F – Additional statistical analyses.....</b>	<b>245</b>



## **List of Tables**

Table 2.1 - The “5 A’s” model for treating tobacco use and dependence .....	15
Table 2.2 - Potential social effects of biomedical explanations of addiction .....	24
Table 4.1 - The purposes of mixed methods research .....	55
Table 4.2 - Braun and Clarke’s steps for thematic analysis.....	62
Table 5.1 - Sample characteristics for Study 1 .....	79
Table 6.1 - Participant demographics for Study 2 .....	95
Table 6.2 - Strategies used on previous quit attempts .....	96
Table 9.1 - Participant demographics for survey .....	158
Table 9.2 - Smoking-related variables .....	159
Table 9.3 - Reported reasons for smoking .....	160
Table 9.4 - Endorsement of brain-based explanations of smoking.....	161
Table 9.5 - Acceptance of Robert West’s explanation of smoking .....	162
Table 9.6 - Change in agreement that smoking is a brain disease after being presented with Robert West explanation.....	163
Table 9.7 - Expectations of consequences of brain-based explanations of smoking.....	164
Table 9.8 - Proportion of smokers who endorsed brain-based explanations of smoking stratified by demographics and smoking characteristics.....	166
Table 9.9 - Binary logistic regression model regressing intention to use cessation medications (yes versus no) on socio-demographic and smoking characteristics .....	169
Table 9.10 - Binary logistic regression model regressing level of self-efficacy on socio-demographic and smoking characteristics .....	171

## **List of Figures**

Figure 2-1 The reward pathway of the brain .....	18
Figure 3-1 Image comparing non-drug user and cocaine user brain scans.....	41
Figure 3-2. Examples of media reporting on the neuroscience of smoking in Australia.....	46
Figure 3-3. Image from the “Tobacco Stories” resource by Menzies School of Health .....	48
Figure 3-4. Image of a smoking brain from Pfizer’s “Nail Quitting” website .....	49
Figure 4-1. Research design.....	58
Figure 9-1 - Recruitment process for survey .....	152

## **List of abbreviations used in the thesis**

AA .....	Alcoholics Anonymous
BDMA.....	Brain disease model of addiction
DSM.....	Diagnostic and Statistical Manual
FTND .....	Fagerstrom Test for Nicotine Dependence
HSI .....	Heaviness of Smoking Index
ICD.....	International Classification of Diseases
nAChR .....	Nicotinic acetylcholine receptor
NIDA.....	National Institute of Drug Abuse
NRT.....	Nicotine replacement therapy
ORU .....	Online Research Unit
RACGP .....	Royal Australian College of General Practitioners
PBS .....	Pharmaceutical Benefits Scheme
PUS .....	Public understanding of science

## **Chapter 1. Introduction**

The prevalence of daily smoking in Australia has declined substantially in recent decades from 35% in 1980 to 12.8% in 2013 (Australian Institute of Health and Welfare, 2014). However, smoking remains a major public health problem that is the largest avoidable contributor to the burden of death and disease in the country (Begg et al., 2007). The reduction in the prevalence of smoking has occurred alongside, and been influenced by, a significant cultural shift in public understandings of smoking.

### **1. Changing public portrayals of smoking**

Prior to the recognition of the health risks of smoking and the addictiveness of nicotine, smoking was commonly portrayed as a personal choice and a bad habit. Quitting was often described as a simple matter of exercising one's willpower (White, Oliffe, & Bottorff, 2013). More recent conceptualisations of smoking frame it as "nicotine addiction": a chronic health condition that entails a loss of control over smoking that requires treatment by health professionals (Fiore et al., 2008).

This is part of a more general shift towards the biomedicalisation of addiction (Campbell, 2007; Midanik, 2006). A key driver of the biomedicalisation of smoking has been the growth in neuroscientific research on addiction. Findings from neuroscience research have demonstrated differences in the neurochemistry and neuroanatomy of addicted individuals, which are theorised to account for addicted persons' impaired control over their drug use. This has led to the claim that addiction is a "chronic, relapsing brain disease" (National Institute on Drug Abuse, 2007).

Proponents of this "brain disease model of addiction" (BDMA) hope that it will lead to the development of novel pharmacotherapies for treatment and a reduction in the stigma associated with drug addiction (Volkow & Koob, 2015). Because nicotine addiction is often excluded from discussions about the BDMA, the social implications of this discourse for nicotine addiction have not been adequately explored. This is problematic as public portrayals of smoking increasingly refer to the role that the brain plays in tobacco dependence, and describe new brain-based therapies for smoking, such as transcranial magnetic stimulation, that are being developed (Rose et al., 2011).

### **1.1. Public perceptions of smoking and quitting**

Lay understandings of smoking have the potential to influence the behaviour and cessation strategies of smokers. It is therefore important to investigate the extent to which members of the public, including smokers, see smoking as a medical problem in need of treatment. In the tobacco control field, there has been some debate about the benefits of the biomedicalisation of smoking. In Australia, the use of pharmacotherapy for smoking cessation is increasing and healthcare practitioners are encouraged to identify smokers and provide them with pharmacological assistance to quit (Zwar et al., 2014). While an increase in treatment seeking is generally seen as a positive development (Aveyard & Raw, 2012; Britton, 2009; Zwar et al., 2014), some have speculated that emphasising the biological basis of addiction and medical treatment for tobacco dependence may make smokers think that quitting smoking is harder than it actually is (Chapman, 2011; Zhu, Lee, Zhuang, Gamst, & Wolfson, 2012). There is concern that this could reduce self-efficacy, which is central to successful quit attempts (DiClemente, Prochaska, & Gibertini, 1985; Schnoll et al., 2011). Others hypothesise that the availability of medical treatments for smoking cessation may lead to a belief that there are “magic bullets” to treat tobacco dependence, which is especially concerning for young people at risk of tobacco dependence (Dingel, Karkazis, & Koenig, 2011). There is little empirical evidence with which to test these competing claims about the social impact of biomedicalised views of smoking.

### **1.2. Overview of the thesis**

The original aim of this thesis was to explore whether smokers endorse the idea that smoking is a brain disease, and the potential of beliefs about smoking and the brain to influence smoking behaviours and treatment choices. As the literature review progressed, and data collection began, it became clear that a wider point of view was necessary in order to examine this question. Firstly, the brain disease model of addiction was one example of a more general shift towards the medicalisation of smoking. This was a contentious matter in the tobacco control field, with topical debates about the efficacy and cost-effectiveness of quit attempts using pharmacotherapy and support from health professionals, compared to that of unassisted quitting; and theorised potential adverse consequences that could arise from promoting a medical model of smoking cessation. Also, it was impossible to examine ideas and beliefs around the brain disease model of addiction without reference to alternative models of smoking that were held by the Australian public. Thus, the aim of the thesis shifted to an exploration of the extent to which biomedical understandings of tobacco dependence have been incorporated

into public understandings of smoking and attitudes towards smoking cessation in Australia. The brain disease model became one aspect of this analysis.

In order to more fully explain the context of the study, Chapter 2 outlines the debates surrounding the biomedicalisation of cigarette smoking. This chapter includes a brief outline on the theory of medicalisation, a discussion on the extent to which smoking has become medicalised, and a critical review of debates in tobacco control about the benefits of a medical approach to smoking cessation. The BDMA, as an influential medical model of addiction, is presented, and its implications for tobacco control discussed.

Chapter 3 investigates the extent to which medical understandings of smoking are present in public discourse on tobacco dependence and smoking cessation. The chapter first explains the importance of investigating lay understandings in public health research. It then presents the limited empirical research on lay conceptualisations of addiction and smoking, including the role that biology is perceived to play in smoking behaviour. Examples of advertisements and health promotion materials from Australia are provided to illustrate how the Australian public is being exposed to a biomedical discourse on smoking and smoking cessation.

Chapter 4 describes the methods that were used in this project. It explains the mixed methods approach that was employed and the rationale and design of the research project. Three separate studies were conducted as part of the thesis:

*Study 1* – A secondary analysis of data from interviews with 55 members of the Australian public (including never smokers, ex-smokers and current smokers) on their views about addiction and its treatment. The analysis reported here focused on the extent to which the participants believed a medical approach was suitable for smoking cessation.

*Study 2* – A qualitative study that involved semi-structured interviews with 29 Australians who smoked daily. The interviews explored their understandings of addiction, their attitudes towards various quitting strategies, their awareness of the role of the brain in smoking, and their attitudes towards the idea that smoking is a brain disease.

*Study 3* – A quantitative study that involved a web-based survey with 1538 participants. The survey items drew on results from the qualitative studies and examined: attitudes

towards, and the use of, medical treatments for smoking cessation; endorsement of brain-based explanations of smoking; and beliefs about the consequences of the brain disease model of nicotine addiction.

Chapter 5 presents the results of Study 1. In order to begin to assess the extent of the medicalisation of smoking in Australia, this analysis focused on the attitudes of members of the Australian public towards medical strategies for smoking cessation and their views on quitting unassisted. These views form part of the social environment in which smokers make choices about the cessation strategies to use when they make a quit attempt. The results reveal the extent to which a medical discourse of nicotine addiction has infiltrated public understandings of smoking in Australia.

Chapter 6 reports findings from study 2 in which smokers were asked their views and experiences on various quitting strategies. This chapter explores participant attitudes towards a range of quitting methods, including smoking cessation medications, quitting unassisted and quitting using counselling or self-help resources. It also outlines the factors that smokers report considering when making decisions about how to make a quit attempt. The findings in this chapter demonstrate the complexity of the relationship between smokers' beliefs about smoking and their treatment choices.

Chapters 7 and 8 also draw on results from Study 2, reporting qualitative findings on smokers' understanding of the role of their brain in tobacco dependence. Chapter 7 examines the beliefs that smokers hold about the role of their brain in smoking and addiction. It explores how these beliefs might relate to treatment choices and participants' sense of smoking self-efficacy. Chapter 8 considers the attitudes of the same participants toward labelling nicotine addiction as a "brain disease." While use of this label is not widespread in Australia, it is increasingly being disseminated by leading research bodies in the USA. It is important to examine the possible implications of this terminology for smokers' self-understanding.

Chapter 9 reports on findings from the quantitative survey of Australian smokers about their attitudes towards biomedical explanations of smoking (Study 3). The analysis investigated: the proportion of Australian smokers who endorsed neurobiological explanations of tobacco dependence; the relationship between endorsement of these neurobiological explanations and attitudes towards smoking cessation medications; and the relationship between endorsement of

neurobiological explanations of smoking and quitting self-efficacy amongst Australian smokers.

Chapter 10 draws together findings from the empirical components of this thesis. It discusses the extent to which participants perceived smoking as a biomedical issue and a brain disease. It outlines the alternative discourses about smoking that became evident during the course of this research. The likelihood that biomedical discourses of smoking will increase the use of pharmacotherapies for cessation attempts or reduce quitting self-efficacy is evaluated. It is clear that there is an increasing emphasis on the neurobiological aspects of addiction to nicotine in public discourse about smoking. The chapter concludes with recommendations about how brain-based information about smoking can be presented in clinical interactions and public health messages in ways that maximize its impact and minimize negative consequences.

### **1.3. Novel contribution of the thesis**

Current debates in tobacco control about the value of biomedical approaches to smoking cessation have neglected the views of the general public, and of those most likely to be affected by shifting conceptualisations of smoking and the changing nature of treatment for tobacco dependence: smokers themselves. This thesis addresses this gap in the literature by exploring attitudes in Australia towards medical approaches to smoking cessation amongst the general public and daily smokers. It is the first study to examine how smokers understand the role of the brain in their smoking, and whether they agree with the controversial description of tobacco dependence as a “brain disease.”

In order to explore this topic the interdisciplinary thesis draws on, and contributes to, three sets of literature: sociological writings on biomedicalisation, public health research on lay understandings of health and illness, and policy debates in the tobacco control field about the treatment of tobacco dependence. The limitations of medical approaches to smoking cessation are outlined, and it is argued that biological understandings of tobacco dependence cannot be disentangled from wider discussions about the meaning of addiction, and the nature of smoking in general. Asking participants about their ideas about smoking and the brain elicits alternative ideas around smoking that are based on deeply held cultural ideas about the centrality of willpower and individual choice. In doing so, this thesis contributes to an emerging body of evidence which suggests that increasing public knowledge about neuroscience is unlikely to



revolutionise public understandings of addiction, or human nature more generally. These findings have practical implications for public health practitioners working in tobacco control and clinicians who interact with smokers. Portrayals of smoking as a medical problem with a medical solution may be helpful for some smokers but also has the potential to alienate others if everyday experiential understandings of smoking are downplayed or ignored.

## **Chapter 2. The bio/medicalisation of tobacco dependence: Evidence and Arguments**

There is an ongoing debate in the tobacco control literature about the value of a medical model in reducing smoking related harm. The variety of medical treatments available for smoking cessation are increasing and health professionals are increasingly encouraged to provide these medical interventions to aid smoking cessation. However, some question whether medical understandings of smoking will undermine the responsibility of the tobacco industry for the harms of smoking. Others worry that it will lead individual smokers to deny their personal responsibility for smoking cessation. Will it create beliefs in “magic bullets” for smoking cessation, or will medical understandings erode smokers’ confidence in their ability to quit? This chapter examines the extent to which cigarette smoking has become medicalised and outlines why medicalisation is contested in the tobacco control field. It also introduces the “brain disease model” of addiction and examines the implications of this model for tobacco control.

### **2.1. Medicalisation: A brief background**

In all societies and historical epochs, sickness has been a problem in need of an explanation. Anthropologists have outlined religious models of sickness, where “diagnosis and healing are both undertaken in a sacred context” (Turner, 2000, p.5), and where individuals are assigned moral responsibility for disease and ill health. In the context of the growth of empirical rationalism, the rise of capitalism, and discoveries in experimental medicine, religious explanations of health and illness began to be questioned from the 17<sup>th</sup> century onwards (Turner, 2000). In the 19<sup>th</sup> century, great improvements in the health of populations resulted from improved diet and nutrition, living conditions, sanitation, water infrastructure, and the communication of public health messages regarding hygiene. The “contagion” model, where diseases were attributed to parasites that were spread between individuals, resulted in an understanding of how diseases are spread via everyday practices and poor sanitary infrastructure, and led in the 20<sup>th</sup> century to the development of vaccines against common infectious diseases (Awofeso, 2004).

The era was followed by the “preventive medicine era”, where efforts began to be directed towards groups identified as being at high risk of disease or ill health (Awofeso, 2004). This was associated with “physicians’ enhanced ability to shape political and public perceptions of health policy issues” (Awofeso, 2004, p705). The enhanced influence and status of the medical profession, as well as the growth of pharmaceutical treatments for various disorders, influenced a process that has been labelled “medicalisation.”

Medicalisation has been defined as “a process by which nonmedical problems become defined and treated as medical problems, usually in terms of illnesses or disorders.” (Conrad, 1992, p. 209). The first discussion about the medicalisation of smoking occurred with the advent of nicotine replacement therapy (NRT), in the form of gum (Blum, 1984). Since then, new forms of NRT and prescription medications for smoking cessation have become available to help smokers quit.

Conrad and Schneider’s seminal work on medicalisation described the key feature of medicalisation as the definition or labelling of a social issue as a medical condition (Conrad & Schneider, 1992). They wrote that “medicalization occurs when a medical frame or definition has been applied to understand or manage a problem” (Conrad & Schneider, 1992, p. 211). Many of the early writings on medicalisation focused on psychiatric diagnoses and depicted the use of pharmacological treatment regimes as exercising social control over those perceived to be deviant (e.g., Szasz, 1974). Medicalisation was typically seen as a negative process that increased the power of the medical profession and disempowered socially marginalised individuals (Ballard & Elston, 2005).

Most writings on medicalisation assumed that the negative consequences of medicalisation outweighed the positive. Conrad and Schneider (1992) have outlined the “brighter and darker” sides of medicalisation. One potential benefit of medicalisation is that defining a problem as a medical issue may reduce stigma due to the implication that afflicted individuals do not have full control over it. This is a particularly pertinent issue for drug addiction, and increasingly relevant to smoking tobacco, where the stigmatisation of smokers is a controversial strategy employed to reduce smoking rates (Amonini, Pettigrew, & Clayforth, 2015; Bell, Salmon, Bowers, Bell, & McCullough, 2010; Brown-Johnson & Prochaska, 2015). The adoption of a “sick role” might reduce feelings of self-blame by lessening an individual’s sense of responsibility, and could increase treatment seeking. In relation to the “darker side”, the

fundamental criticism of medicalisation is that it “decontextualizes social problems” by transforming them into individual problems (Conrad, 1992, p. 223). It pathologises individual differences, leading to a “dislocation of individual responsibility”, and reduces efforts to improve social environments (Conrad & Schneider, 1992, p. 152). Critiques of medicalisation have been accused of underestimating the benefits of implementing medical approaches for previously unmedicalised problems (Ballard & Elston, 2005).

Power differentials between groups are central to medicalization theory. Early work on medicalisation emphasised the power of the medical profession (i.e., doctors/physicians) in transforming social deviance into medical illness (Zola, 1972). Medicalisation now appears to be accelerating through the influence of institutions such as pharmaceutical and health insurance companies (Clarke, Shim, Mamo, Fosket, & Fishman, 2003). Clarke and colleagues (2003) believe that “technoscientific innovations” such as genomics and biotechnology have led to a fundamental transformation in the medical arena. They have labelled this biomedicalisation. A key characteristic of biomedicalisation is that it encompasses health *and* illness. Attaining and maintaining good health has come to be seen as a moral responsibility of each citizen (Crawford, 1980) and many “surveillance technologies” are now available to monitor one’s health (Lupton, 2012). There is some debate about whether biomedicalisation fundamentally departs from medicalisation, or simply represents a shift of influence and power (Conrad, 2005). It is clear, however, that pharmaceutical companies, biotechnology firms, and health insurance companies now play a key role in defining disorders and deciding which warrant treatment, issues previously primarily within the purview of organised medicine (e.g., Moynihan, 2003).

Another change has been a move towards a more active role for consumers in healthcare. The focus on power relations in the sociological literature has often positioned the individual as a passive subject of those wielding institutional authority (Ballard & Elston, 2005). More recent work acknowledges that patients are not simply submissive recipients of medical labels and care. Research has documented consumer resistance, as well as advocacy for medicalised definitions in those affected by various disorders (Singh, 2003; Valentine, 2010). The considerable literature on deliberate lack of adherence to medications and treatment recommendations also signifies the active role of patients (Horne, 1997; Osterberg & Blaschke, 2005). It is now generally acknowledged that patients are stakeholders who frequently take an active and interested role in decisions about their own healthcare (Ballard & Elston, 2005).

Conrad (1992) described three levels at which medicalisation can occur: the conceptual, institutional and interactional. The conceptual level refers to the application of a medical definition or label. An example is the labeling of low libido in women as “female sexual dysfunction” (Moynihan, 2003). Medicalisation at the institutional level involves the uptake of medical models by key institutions, such as the creation of medical treatment guidelines for national health services. The interactional level involves communication between a healthcare provider and their patients. Examples include diagnosing a patient with a disorder, or prescribing medication for a problem. Medicalisation is rarely complete, and may occur at one level but have little impact at another (Conrad, 1992). Conrad has therefore recommended assessing the *degree* of medicalisation, rather than classifying an issue as medicalised or not in a dichotomous fashion. He has listed the following factors that can influence the degree to which a condition is medicalised: “the support of the medical profession, availability of interventions or treatments, existence of competing definitions, coverage by medical insurance, and the presence of groups challenging the medical definition.” (Conrad, 1992, p. 220).

The remainder of this chapter will consider the degree to which tobacco dependence has been medicalised. The following sections will focus on countries that are in the later stages of the tobacco epidemic, that is countries such as Australia and England where smoking prevalence is declining in both men and women (Lopez, Collishaw, & Piha, 1994). These countries have typically adopted strong tobacco control policies, such as increased taxes on cigarettes and smoking bans in public places.

## **2.2. Conceptions of tobacco dependence: From “bad habit” to “nicotine addiction”**

*‘If [cigarettes] are behaviorally addictive or habit forming, they are much more like ... Gummi Bears, and I eat Gummi Bears, and I don't like it when I don't eat my Gummi Bears, but I'm certainly not addicted to them’ J.Morgan , President and CEO of Philip Morris, 12 May 1997 US District Court for the District of Columbia. (Cited in Robertson & Hurt, 2010, p. 449)*

The first step in exploring the extent to which cigarette smoking has been medicalised is to look at how it is defined. A number of historians have documented the changing conceptualisations of cigarette smoking throughout the 20<sup>th</sup> century (Berridge, 1997; Brandt, 2007; Hilton, 2000). Prior to the 1950s cigarettes were seen as more like a food stuff than an addicting drug (Berridge, 1997). While some had observed features of cigarette smoking that seemed to signal addiction (Tyrrell, 1998), the discourse of addiction was not widespread and

addiction was not recognised in official government documents on smoking. In the 1950s, the health risks of smoking began to be documented and publicly disseminated. This led to an increase in quit attempts and it became clear that many people had difficulty in giving up their smoking “habit.”

Nonetheless, it was not until 1988 that the US Surgeon General conducted an extensive review of the scientific evidence and concluded that tobacco smoking was addictive, and that it was the nicotine in tobacco which produced its reinforcing effects. Cigarettes were thus redefined as a “delivery device” for nicotine, an addictive drug “in the same sense as ... heroin and cocaine.” (US Surgeon General, 1988, p. vi). The change from describing tobacco use as “habituation” in the 1964 report to “nicotine addiction” in 1988 was influenced by neuroscience insights about how nicotine worked in the brain and evidence that nicotine replacement therapy reduced cravings for cigarettes (Elam, 2015b).

Some commentators have also noted changes in the social context that made the labelling of smoking as an addiction more publicly acceptable. Firstly, by 1988 the prevalence of smoking was declining and the makeup of the smoking population was changing. Those of lower socioeconomic status continued smoking in greater numbers than the economically privileged who were more likely to quit. Brandt writes that “[I]n a culture prone to stigmatize its poor and disfavoured, changing perceptions about the ‘average smoker’ eased the growing attribution of addiction.” (Brandt, 2004, p. 391). Also, perceptions of “the addict” were changing, with increasing advocacy for reducing the stigma of addiction and a recognition that it could “happen to anybody” (Mars & Ling, 2008).

Unsurprisingly, the tobacco industry resisted accepting that cigarettes were addictive. Even after the U.S. Surgeon General’s report of 1988, they continued to deny publicly that smoking was addictive until the late 1990s, while admitting in internal documents that people only smoked due to the rewarding properties of nicotine (Robertson & Hurt, 2010). The idea that smoking was solely an individual choice was one they could not easily give up because it was central to their defence against litigation (Mars & Ling, 2008).

Preceding and intertwining with these debates about the role of nicotine in tobacco dependence, were debates about whether tobacco dependence should be included in the American Psychiatric Association’s Diagnostic and Statistical Manual III (DSM-III). Despite the tobacco

industry claim that it would be “ridiculous” to label an everyday behaviour of so many as a mental disorder (Hirshbein, 2014), the DSM-III included “tobacco dependence” as a diagnostic category. In the 1987 revision of the DSM-III, tobacco dependence was renamed “nicotine dependence”, a label that remained in the DSM-IV (American Psychiatric Association, 2000). The diagnostic criteria for nicotine dependence were based on those for other drugs. They included tolerance, withdrawal symptoms, compulsive use, and use despite negative consequences. The World Health Organisation’s International Classification of Disease (ICD) developed similar diagnostic criteria, but employed the label tobacco dependence (World Health Organisation, 2011).

There was renewed debate about the value of the DSM diagnostic criteria for nicotine dependence in the lead up to the release of the DSM-5. Critics noted that the DSM criteria for diagnosing tobacco dependence were not widely used in research or in clinical practice (T. B. Baker, Breslau, Covey, & Shiffman, 2012; Hughes, 2006). Other measures of dependence were more often used, such as the Fagerstrom Test for Nicotine Dependence (FTND), and its shortened form, the Heaviness of Smoking Index (HSI). These measures were developed specifically for tobacco dependence and were more predictive of relapse than the DSM criteria (T. B. Baker et al., 2012). This led to a dialogue about whether the generic criteria developed for drug addiction were applicable to tobacco smoking. Baker and colleagues (2012) concluded that nicotine dependence was different enough from other addictions to warrant separate diagnostic criteria.

One of the key differences between nicotine and other drug addictions is that nicotine produces only mild psychoactive effects. Smokers do not become intoxicated, and most dependent smokers do not suffer from the types of “behavioural harm” experienced by persons addicted to other drugs (Hughes, 2006). The idea of nicotine “abuse” (as opposed to dependence) is also not applicable to smoking, as individuals do not smoke to become intoxicated. The DSM-5 has addressed the last criticism by removing the distinction between substance abuse and substance dependence. Nicotine dependence has been renamed “tobacco use disorder.” Very similar diagnostic criteria apply but dependence is diagnosed along a continuum of severity, with mild, moderate or severe dependence, depending on the number of diagnostic criteria an individual meets.

Heirshbein (2014) has criticised the DSM-5 for being more “expansive” than previous versions. She believes that anyone who uses tobacco can now be diagnosed as having tobacco use disorder and notes the influence of the pharmaceutical company in defining smoking as a psychiatric disorder requiring treatment by medical specialists. In her words, “the psychiatric diagnosis of tobacco use disorder is more about the current social, political, and economic context of US medical and public health approaches to cigarettes than a valid description of a disease state.” (Hirshbein, 2014, p. 2082).

This brief outline illustrates how smoking has increasingly come to be defined as a medical problem through labelling smoking as an addiction, and applying diagnostic criteria. The key point of such a medical diagnosis or definition, according to Robert West, “is to establish whether and what kind of medical treatment or care may be appropriate.” (West & Miller, 2011, p. 863). It is to the treatment of “tobacco use disorder” that we now turn.

### **2.3. Pharmacotherapy for smoking cessation and the role of the health professional**

One driver of the medicalisation of tobacco use is the increased availability and use of pharmacotherapies for smoking cessation. Nicotine replacement therapy was first developed in the 1960s in the form of nicotine gum. It provides the brain with nicotine, reducing withdrawal symptoms. It typically uses slower acting methods of administration and produces lower blood nicotine levels than are obtained by smoking cigarettes. It was developed to mitigate the health harms of smoking by reducing cravings for cigarettes and thus preventing relapse. Initially there was some debate over whether NRT would be marketed as an alternative and safer way of consuming nicotine (a harm reduction approach) or solely as a therapeutic device for smoking cessation (a medical approach) (Elam, 2015b). The therapeutic approach came to dominate and clinical trials have since demonstrated that NRT increases the chances of a successful quit attempt by 50-70% (Hartmann-Boyce, Stead, Cahill, & Lancaster, 2013; Stead, Perera, Bullen, Mant, & Lancaster, 2008).

There are now many forms of NRT available that vary in strength and speed of nicotine delivery. These include: gum, patches, inhalators, lozenges, mouthspray, nasal spray, dissolvable oral strips and pouches that resemble a Swedish tobacco product known as portion snus. While initially available only via prescription, most forms of NRT are now available over the counter in pharmacies and at general retailers in Australia and many other countries. In



Australia, nicotine patches were listed on the Pharmaceutical Benefits Scheme (PBS) in 2011, such that the cost is publicly subsidised when NRT is prescribed by a registered medical practitioner. At a global level, the World Health Organisation has added NRT to the Model List of Essential Medicines (World Health Organisation, 2015), which encourages governments to provide essential medicines to their population at a low cost.

Two non-nicotine prescription-only medications for smoking cessation are available in Australia. Bupropion (sold in Australia as Zyban and Prexaton) is an antidepressant that was marketed as a cessation aid after it was noticed that patients taking it for depression reported reduced urges to smoke. Clinical trials show bupropion to be as effective as NRT in increasing quit rates (Hughes, Stead, & Lancaster, 2010). The most recent prescription-only pharmacotherapy for smoking cessation is varenicline (marketed in Australia as Champix). Varenicline is a partial agonist, meaning it stimulates nicotinic receptors, but not to the same extent as smoking. This reduces withdrawal symptoms and the satisfaction of smoking. Varenicline increases the success of quit attempts up to threefold compared to a placebo (Cahill, Stead, & Lancaster, 2012). Bupropion and varenicline are listed on the PBS in Australia. Other pharmacotherapies that have been shown to be effective, but are not used widely in Australia, are cytisine and nortriptyline.

In Australia, the use of smoking cessation pharmacotherapies has increased steadily since their introduction. In 2002, 32% of those who had made a quit attempt in the previous year had used pharmacotherapy (NRT, Champix or Zyban). This increased to 52% in 2008 (Cooper, Borland, & Yong, 2011). The listing of varenicline on the PBS in 2008 increased use from 5% to 24% between 2007 and 2008 (Cooper et al., 2011). Despite high use of pharmacotherapies, there is evidence of poor adherence, with many users prematurely discontinuing use (Balmford, Borland, Hammond, & Cummings, 2011; Fucito, Toll, Salovey, & O'Malley, 2009; Shelley et al., 2015). Also, despite previous studies showing NRT purchased over the counter is as effective as NRT bought on prescription (Hughes, Shiffman, Callas, & Zhang, 2003), more recent evidence suggests that NRT purchased over the counter may not increase quitting (Hughes, Peters, & Naud, 2011).

The widespread use of pharmacotherapy contrasts with low uptake of behavioural support for smoking cessation, despite behavioural support being required under the PBS prescribing guidelines for smoking cessation pharmacotherapies. Research shows that only 15% of those

who had made a quit attempt in the year 2008-2009 had used behavioural support, such as the *Quitline* or private counselling (Cooper et al., 2011). Findings from the UK are similar. A 2011 report found that 54% of smokers who had made a quit attempt used pharmacotherapy, but only a small proportion attended smoking cessation clinics, where more intensive assistance was provided (West & Fidler, 2011). The low use of behavioural support is disappointing given that a combination of pharmacotherapy and behavioural intervention is the most effective way to quit smoking (Hartmann-Boyce et al., 2013).

Tobacco treatment guidelines in the USA, Australia, and the UK all recommend that health professionals identify smokers and intervene by providing brief advice about smoking cessation and a prescription for a pharmacotherapy (Fiore et al., 2008; National Institute for Health and Clinical Excellence, 2008; Zwar et al., 2014). The “5 A’s” were developed by the U.S. Surgeon General for health practitioners. They are shown in Table 2.1.

Table 2.1 - The “5 A’s” model for treating tobacco use and dependence

<b>Ask about tobacco use</b>	Identify and document tobacco use status for every patient at every visit.
<b>Advise to quit</b>	In a clear, strong, and personalized manner, urge every tobacco user to quit.
<b>Assess willingness to make a quit attempt</b>	Is the tobacco user willing to make a quit attempt at this time?
<b>Assist in quit attempt</b>	For the patient willing to make a quit attempt, offer medication and provide or refer for counselling or additional treatment to help the patient quit.  For patients unwilling to quit at the time, provide interventions designed to increase future quit attempts.
<b>Arrange follow up</b>	For the patient willing to make a quit attempt, arrange for follow up contacts, beginning within the first week after the quit date.  For patients unwilling to make a quit attempt at the time, address tobacco dependence and willingness to quit at next clinic visit.

From Fiore et al. (2008)

In Australia, the Royal Australian College of General Practitioners (RACGP) recommend that pharmacotherapy be offered to all smokers who are “nicotine dependent.” The RACGP does not provide definitive criteria for diagnosing nicotine dependence but lists the following “indications of nicotine dependence”: smoking within 30 minutes of waking, smoking more than ten cigarettes per day, or experiencing withdrawal symptoms on previous quit attempts (Zwar et al., 2014). Brief smoking cessation advice by physicians is recommended for all smokers, and referral to Australia’s free smoking cessation telephone helpline (called *Quitline*) is also recommended. While the use of telephone smoking cessation counselling has been shown to be an effective intervention (Stead, Hartmann-Boyce, Perera, & Lancaster, 2013), the proportion of smokers who use this service is very low. In Australia, where the *Quitline* is free, easily accessible, advertised on television and on all cigarette packets, less than 5% of smokers had used the service within the last year (Miller, Wakefield, & Roberts, 2003). Moreover, of those who had used it, many reported that it was not helpful in their quit attempts (Hung, Dunlop, Perez, & Cotter, 2011).

#### **2.4. Smoking as a chronic disease**

A number of authors have recently recommended that all smokers who come in contact with health professionals should receive treatment for their smoking, regardless of their desire or “readiness” to quit (Aveyard & Raw, 2012; Richter & Ellerbeck, 2014; Zwar et al., 2014). They argue that smoking should be treated as a “chronic disease” because of the propensity to relapse, and they compare the treatment of smoking to hypertension, diabetes, or high cholesterol (Steinberg, Schmelzer, Richardson, & Foulds, 2008). For example, Aveyard and Raw advocate for a new paradigm where health professionals support “(nearly) all smokers most of the time”, rather than offering support only to those who have expressed an intention to quit (Aveyard & Raw, 2012, p. 255). They argued that this means treating “smoking and nicotine addiction like a chronic disease.” (Aveyard & Raw, 2012, p. 255). Their choice of words is instructive: they state that nicotine addiction should be treated *like* a chronic disease, not that it *is* a chronic disease. Others are more direct in the language used. The US Public Health Services Guidelines states that “Tobacco dependence is a chronic disease that often requires repeated intervention and multiple attempts to quit.” (Fiore et al., 2008, p. vi). Tobacco smoking is thus portrayed not only as a risk factor for disease, but as a disease in itself.

The use of the disease label for smoking has been influenced neurobiological research on addiction to cocaine and heroin that described the neurochemical changes in the brain related to prolonged use of these psychoactive drugs (Leshner, 1997; Volkow & Li, 2004). The same approach was later used to study nicotine dependence. Before outlining what has come to be known as the “brain disease model of addiction”, a brief summary of the neuroscience of nicotine addiction is now provided.

## **2.5. The neuroscience of nicotine addiction**

Nicotine is a natural component of tobacco that can be highly toxic to the body and cause death in high doses (Mayer, 2014). In small amounts, typical of that absorbed from a cigarette, it causes mild psychoactive and cognitive effects. Nicotine is the primary addictive agent in tobacco, but it is not the only chemical constituent of cigarette smoke associated with the clinical signs of dependence. For example,  $\beta$ -Carboline has been demonstrated to inhibit the release of monoamine oxidase, possibly contributing to the negative affect experienced by smokers during a period of abstinence (Bruijnzeel, 2012). However, scientific evidence demonstrates that nicotine is the primary psychoactive and addictive component of tobacco (US Surgeon General, 1988). It is also the most well-known to the public, and therefore it is the main focus in this thesis.

### ***Effects of smoking on cognition and mood in humans***

While smoking often produces aversive symptoms in new users, such as nausea and dizziness, these effects typically dissipate with continued smoking and the development of tolerance. In dependent individuals, smoking creates a sense of pleasure, feelings of relaxation, and enhanced alertness and attention. On cessation of tobacco use, withdrawal symptoms that are commonly reported include irritability, cravings, anxiety, sleep disturbance, and increased appetite (Hughes, 2007). Individuals may smoke to achieve desirable cognitive or emotional effects (positive reinforcement) and to relieve withdrawal symptoms (negative reinforcement).

### ***Acute effects of nicotine use on the brain***

When tobacco is smoked, nicotine is suspended in particles of tar that are inhaled into the lungs, where the nicotine is absorbed into the bloodstream. Nicotine travels quickly from the lungs to the brain, crossing the blood-brain barrier, where it binds to receptors in the brain on which the neurotransmitter acetylcholine usually binds, known as nicotinic acetylcholine receptors

(nAChRs). Nicotine binding to nAChR receptors opens ion channels in the neuron membrane allowing positively charged ions to enter the neuron that influences the chemical signals it transmits (Lavolette & van der Kooy, 2004; Stoker & Markou, 2013).

While nAChRs are widely distributed throughout the brain, the effects of nicotine in the mesocorticolimbic reward circuit of the brain are thought to be critical to the development of addiction (Lavolette & van der Kooy, 2004). Specific anatomical structures of the brain that have been implicated are the ventral tegmental area, nucleus accumbens, corpus striatum, amygdala, hippocampus and the prefrontal cortex (Figure 2-1).

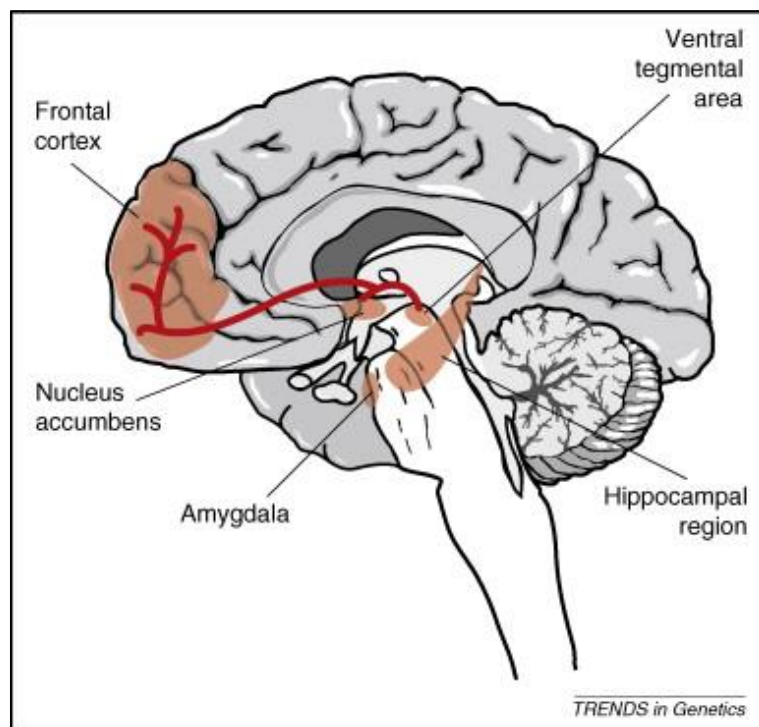


Figure 2-1 The reward pathway of the brain<sup>1</sup>

The ventral tegmental area contains a high concentration of nAChRs. Nicotinic activation of nAChRs in this area causes the release of the neurotransmitter dopamine in the nucleus accumbens shell (Benowitz, 2010). These dopaminergic neurons project to structures in the limbic and cortical systems, including those that play a key role in memory (hippocampus), emotion (amygdala), and decision-making (prefrontal cortex) (De Biasi & Dani, 2011).

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<sup>1</sup> (Kalsi, Prescott, Kendler, & Riley, 2009, p. 50). Reprinted with permission from Elsevier.

### *The neural basis of nicotine dependence*

Converging lines of evidence suggest that dopamine release in the mesolimbic area plays a major role in the reinforcing properties of nicotine (Pistillo, Clementi, Zoli, & Gotti, 2015). Dopamine release in the mesolimbic circuit is believed to signal the salience or importance of stimuli. Nicotine self-administration in rats decreases when the release of dopamine is inhibited by chemically induced lesions of the mesolimbic dopaminergic pathway or the administration of dopamine antagonists (Corrigall & Coen, 1991; Corrigall, Franklin, Coen, & Clarke, 1992; DeNoble & Mele, 2006).

The brain's stress system is also affected by chronic nicotine administration in ways that may contribute to the withdrawal symptoms of anxiety and irritability that many smokers experience on cessation. Nicotine administration has been associated with the activation of the hypothalamic-pituitary-adrenal axis, and the subsequent release of the hormones corticotropin and cortisol, hormones which are usually produced in response to stress (Bruijnzeel, 2012). Cessation of smoking is associated with reduced cortisol levels and smokers with lower levels of cortisol are more likely to experience withdrawal symptoms, although causation has not been established (Bruijnzeel, 2012). A review by Adrie Bruijnzeel (2012) concluded that nicotine exposure leads to dysregulation of the brain's stress system, producing feelings of stress on withdrawal that can potentially cause relapse.

Changes in the brain associated with nicotine addiction occur at numerous levels. At the cellular level, plastic changes in the brain, known as neuroadaptations, occur with repeated nicotine use. These are associated with the development of the clinical signs of addiction: craving, withdrawal, and tolerance. Repeated exposure to nicotine desensitizes nAChRs and increases the length of time that receptors are in an inactive state (Govind, Vezina, & Green, 2009). When abstinent from nicotine, the receptors again become active and available for binding.

Upregulation is another plastic change in the brain that is thought to result from chronic nicotine use. It involves an increase in the number of nAChRs that is thought to occur in response to the desensitization of nAChRs described above (Peng, Gerzanich, Anand, Whiting, & Lindstrom, 1994). Upregulation is a complex process and its role in the development of the signs of nicotine dependence has not been fully elucidated. It is also thought to play a role in

nicotine desensitisation and withdrawal symptoms (De Biasi & Dani, 2011; Govind, Walsh, & Green, 2012).

Imaging studies have shown a number of structural differences in smokers compared to non-smokers, including: increased grey matter in the insula; decreased grey matter volume in the prefrontal cortex; and thinning of the orbitofrontal cortex (Jasinka, Zorick, Brody, & Stein, 2013). A review of human and animal brain imaging studies of the effects of nicotine on the brain found a *general decrease* in total brain activity, but a *specific increase* in brain activity in the prefrontal cortex, the visual system and the thalamus. These changes may explain the improved visual attention and increased reaction time in cognitive studies of the effects of nicotine (Brody, 2006).

## **2.6. The brain disease model of addiction**

The belief that addiction is a disease is not new, but the idea that it has a specific neurobiological basis that can be pinpointed in the brain of affected individuals is a more recent development. Older models of addiction framed drug dependence as a disease of the will (White, 1998). The brain disease model locates addiction in the brain of the individual, and proponents argue that this focus on biological factors will reduce moral judgements about addicted individuals, and enable more humane public policy responses to addiction (Dackis & O'Brien, 2005).

According to Campbell (2010), the BDMA was enthusiastically adopted because the field of addiction studies was in “conceptual disarray” due to the wide variety of drugs that seemed to work in different ways and had varied “social histories” (Campbell, 2010, p.92). A “unifying framework” of addiction attracted scientists to the field, aided by the prestige of neuroscience and its associated funding (Campbell, 2010, p.92). Similarly, Vrecko (2010) has described the social context in which the BDMA emerged in America. He has argued that the field of addiction neuroscience was made possible due to large amounts of funding from US government during Richard Nixon’s “War on Drugs” of the 1970s, leading to a “neurobiological problem space” where “the brain and the scientist’s laboratory have become obligatory points of passage for those who wish to produce truths about addiction” (Vrecko, 2010, p. 61).

The National Institute of Drug Abuse (NIDA) in the US defines addiction as a “chronic, relapsing brain disease” (National Institute on Drug Abuse, 2007) and its two recent Directors have actively promoted this explanation of addiction to policymakers, research funders and the public. They employ the metaphor of drugs “hijacking” the circuits of the brain that respond to natural rewards such as food and sex, overriding rational thought (Dackis & O'Brien, 2005; Volkow & Li, 2005). This language has been echoed by the American Association for Addiction Medicine, who recently redefined addiction as “a primary, chronic disease of brain reward, memory and related circuitry” (American Society for Addiction Medicine, 2015). The central tenets of the brain disease model are that:

- chronic drug use leads to long-lasting changes in brain structure and function
- these brain changes explain the behaviours that accompany addiction, specifically a loss of control over drug use and a risk of relapse, and
- addiction is chronic and relapsing, similar to diabetes or hypertension, and should be treated as a chronic health problem (Dackis & O'Brien, 2005; Leshner, 1997; Volkow & Li, 2004).

One of the key arguments for the BDMA is its potential to improve treatments for drug addiction. A number of novel treatments for smoking cessation informed by neuroscience research have been proposed. For example, transcranial magnetic stimulation is a non-invasive procedure in which a magnetic device placed near the surface of the skull stimulates neurons in a localised area of the brain. A small number of studies have used this procedure on smokers and findings suggest that it may reduce cravings for cigarettes in the short-term (Amiaz, Levy, Vainiger, Grunhaus, & Zangen, 2009; Rose et al., 2011) but there is little evidence that it increases cessation success in the long-term. Results from other neuroscience studies have led to the suggestion that brain scans could be used in the future to identify those at risk of relapse of smoking and to match individuals to interventions that are most likely to be effective (Addicott, Sweitzer, Froeliger, Rose, & McClernon, 2015; Loughead et al., 2015).

### **2.7. Criticisms of the biomedicalisation of tobacco smoking**

While some are optimistic about the potential for biomedical research to produce new treatments for smoking cessation, there are concerns about potential negative impacts of viewing and treating tobacco dependence as a disease in need of medical treatment.



Despite the effectiveness of pharmacotherapy for smoking cessation in clinical trials, there have been criticisms of the extent to which pharmaceutical aids for quitting have been promoted. While these medications increase the success rate of quit attempts, most smokers who use NRT or prescription medication still fail to quit (Chapman & MacKenzie, 2010). Moreover, many of the findings about the effectiveness of pharmaceutical treatments for smoking are based on clinical trials sponsored by the pharmaceutical manufacturer (Chapman & MacKenzie, 2010; Etter, Burri, & Stapleton, 2007).

Community surveys to assess the effectiveness of cessation medications when used without medical supervision have produced more mixed findings on their efficacy in everyday use. This failure to replicate the results in clinical studies raises questions about the generalisability of findings from clinical trials to “real world” settings (Alpert, Connolly, & Biener, 2013; Walsh, 2008). However, a recent study that included data from Australia, Canada, the USA and the UK found that use of pharmacological cessation aids significantly increased the chance of a successful quit attempt (Kasza et al., 2013). The authors argue that a lack of positive findings in previous studies was due to a failure to control for important sources of bias in who uses these aids, and due to smokers’ poor adherence to guidelines on the effective use of these aids.

Advocates of a medical model of smoking cessation argue that even if interventions are successful for only a minority of smokers, small improvements in quit success make these interventions cost effective compared to other health interventions (Parrott & Godfrey, 2004). Some have argued that more smokers should be encouraged to use them: “If there is a major failing in the UK approach, it is not that it has medicalised smoking, but that it has not done so enough.” (Britton, 2009).

Concerns about the promotion of pharmacotherapy for smoking cessation do not solely centre on efficacy. There are also concerns that a biomedical model of smoking will distort public perceptions about how easy it is to quit smoking. For example, Chapman and McKenzie (2010) believe that a focus on “treatment” for smoking neglects the fact that most of those who have quit have done so unaided. An emphasis on the need to use medications for smoking cessation may increase perceptions that quitting smoking is difficult, thereby eroding self-efficacy and agency (Chapman & MacKenzie, 2010; Zhu et al., 2012). Chapman and McKenzie (2010) argue that more research should be conducted into why unassisted quitting is so effective.

Chapman and McKenzie (2010) attribute what they consider an overemphasis on a medical approach to smoking cessation to a number of factors. The first is that most people working on smoking cessation interventions have a positivist approach to knowledge, meaning they prioritise individual level variables and experimental approaches. The alternative public health model takes into account more distal variables and their complex interactions. Another factor is the commodification of smoking cessation under the influence of the corporate interests of pharmaceutical companies. Lastly, there is a belief, unsupported by evidence in their view, that those smokers who have not already quit are “hard core” smokers in need of intensive assistance (Chapman & MacKenzie, 2010).

Other commentators are also worried that a medical model of smoking that emphasises the addicted brain will have negative consequences. Caron and colleagues (2005) outlined the “fundamental changes” in “popular, clinical, and public health views of smoking” that may result from a “neurogenomic” understanding of addiction (Caron et al., 2005, p. 181). These include: a diminution of the role of choice in public portrayals and understandings of smoking; a neglect of structural and social aspects of smoking; and the potential for discrimination based on the neuromolecular status of individuals. Also, a shift in emphasis from the cigarette to the smoker may deflect attention from the tobacco industry and their dangerous products. In fact, there is evidence that the tobacco industry have supported genetic research on addiction as a strategy to deflect blame for the health consequences of smoking from the cigarette to the “genetic constitution” of the smoker (Gundle, Dingel, & Koenig, 2010, p. 974).

The BDMA has faced similar criticisms as those levelled at the more general medical model of addiction. Critics argue that it is overly essentialist, emphasising biological factors at the expense of social ones (Dingel et al., 2011; Kalant, 2010). A key criticism of the BDMA is that it may marginalize other perspectives and treatment modalities by prioritizing basic science research on addiction at the expense of funding for social and behavioural interventions (Dingel et al., 2011). A focus on the treatment of individuals may reduce the emphasis on broader, effective population based measures. In tobacco control these include increasing tobacco taxes and banning smoking in public spaces (Gartner, Carter, & Partridge, 2012).

There is also some evidence that biological understandings of addiction may actually *increase* the stigma directed towards addicted individuals (Kvaale, Gottdiener, & Haslam, 2013;

Pescosolido et al., 2010). Elam (2015a) argues that the BDMA is responsible for “remoralizing” addiction. Drug users are believed to have lost control of their drug use and are portrayed as “brain hostages”, but at the same time they are encouraged to take responsibility for protecting their brains and working to return them to a more healthy state. Constant vigilance is required in order to “avoid being terrorized by our own neural circuitry gone awry” (Elam, 2015a, p. 59).

These potential benefits and pitfalls of the medicalisation of addiction are summarised in Table 2.2. The pitfalls listed closely align with the “darker sides” of medicalisation described earlier in this chapter, namely, the individualisation of social problems and the dislocation of individual responsibility.

Table 2.2 - Potential social effects of biomedical explanations of addiction

<b>Benefits</b>	<b>Pitfalls</b>
Improved treatments	Perception that quitting is difficult without medication
Reduced stigma	Increased stigma
Reduced sense of self-blame	Decreased sense of personal responsibility
Greater treatment seeking and adherence	Reduced self-efficacy and increase in fatalism
Improved quit rates	Decrease in quit attempts
Increase in funding of addiction science	Decrease in funding on population level approaches

## **2.8. Limits to the biomedicalisation of smoking**

There are a number of factors that have limited the extent of the biomedicalisation of smoking. Firstly, the tobacco control field grew primarily from epidemiology and public health (Berridge, 1997). A medical approach that has more recently developed has not displaced the population based measures that remain central in the field’s research and policy. The WHO Framework Convention on Tobacco Control has a strong emphasis on population-based measures, with only one item discussing the role of treatment for smoking cessation (World Health Organisation, 2005). Because many of the countries where smoking prevalence is increasing have limited resources, an emphasis on cost-effective population level interventions

is vital. It has been argued that for countries in early stages of the tobacco epidemic, the widespread treatment of individual smokers with pharmacotherapy is not financially viable (Chapman & MacKenzie, 2010), but others have advocated for NRT as a cost-effective medication even in developing countries (Kishore, Bitton, Cravioto, & Yach, 2010).

Second, there has been a growing acknowledgement of socioeconomic disparities in smoking, which increasingly concentrated amongst the poor and marginalized, as the greatest decline in smoking has occurred amongst the wealthiest and most educated groups (Australian Institute of Health and Welfare, 2014). The social embeddedness of smoking has been demonstrated by qualitative studies which illuminate the roles and meanings that disadvantaged smokers ascribe to their tobacco consumption. For example, smoking is described as a means of coping with the stresses associated with poverty, as a means for forging social connections and identity, and as a form of resistance to dominant middle-class values (Pateman et al., 2016; Thompson, Pearce & Barnett, 2007; Wiltshire, Bancroft, Parry & Amos, 2003). This “uneven social geography” of smoking signifies that smoking is not simply an individual health behaviour, and that social context must be considered by tobacco control researchers (Poland et al., 2006).

A third factor limiting the biomedicalisation of smoking is nicotine’s ambiguous place amongst other drugs (Keane, 2002). As previously mentioned, nicotine has only mild psychoactive effects, and regular smokers do not become intoxicated. Smoking does not usually adversely affect an individual’s ability to function in everyday life or cause the social problems associated with other substance use, and the adverse health effects of smoking are often delayed by decades. These differences are why nicotine is often considered separately from alcohol and illicit drugs, or even ignored in discussions of drug addiction. Hughes (2013) found that 75% of articles that referred to drug/substance abuse, dependence or addiction did not include nicotine as a focus, and few papers provided any reason for excluding it. The intensity of medical treatment for addiction to alcohol or illicit drugs differs from that of nicotine. Treatment for other drugs often involves supervised detoxification, extended rehabilitation programs, or long-term substitution programs (e.g., methadone maintenance therapy). It is very rare for smokers to receive inpatient care solely for their smoking, although there are exceptions (Mayo Clinic, 2015).

Lastly, many healthcare practitioners are unenthusiastic about a medical approach to smoking cessation. Despite the clinical recommendations described above for identifying and treating

smokers, Australian research reveals that only half of smokers who had been to a doctor in the last year were advised to quit smoking (Cooper et al., 2011). Similar findings have been reported in the USA (Centers for Disease Control and Prevention, 2011), and the UK (West & Fidler, 2011). Barriers to identifying and treating smokers in general practice include doctors' concern about patients taking offence, a lack of time, and a lack of confidence in addressing smoking (Zwar & Richmond, 2006). While the rate of pharmacotherapy use amongst smokers is increasing, there also seems to be resistance amongst smokers to seeking professional assistance with cessation. As previously described, the vast majority of smokers do not use free smoking cessation services such as cessation clinics in the UK and the *Quitline* in Australia.

### **2.9. The role of nicotine addiction in the tobacco “endgame”**

Virginia Berridge has written that the public health approach to smoking symbolized “an absolutist approach to smoking” (Berridge, 1997, p. 50) according to which nicotine addiction is an evil to be eradicated. The medical approach, by contrast, has been more open to employing nicotine substitution for harm reduction. This includes the use of nicotine in NRT, as well as other products such as snus, and more recently, e-cigarettes. Kozłowski (2013) describes three perspectives on the role of nicotine addiction in tobacco control. The first is that nicotine addiction is a disease in itself that needs to be eradicated. The second is that nicotine addiction should be eliminated, but that this is not a priority. The third perspective is that nicotine addiction can be employed as a tool to reduce the harms of cigarette smoking.

The contradictory role of nicotine as an agent of addiction and as a treatment for addiction has been described by Keane in relation to NRT:

“Thus NRT actually performs two contrasting forms of nicotine dependence: a disorder which is the opposite of freedom and autonomy and a component of the treatment which re-establishes freedom and autonomy.” (Keane, 2013, p. 191)

Keane also notes that when NRT became available over-the-counter, there was a shift from it being a medicalised option for smoking cessation, to a consumer product that did not require the smoker to become a patient (Keane, 2013). In this sense, increasing smokers' access to NRT may have played a role in the partial de-medicalisation of nicotine addiction as well as contributing to the medicalisation of smoking cessation.

Debates around the role of nicotine addiction are becoming more explicit with the increasing availability and use of electronic cigarettes, or e-cigarettes. E-cigarettes have been highly controversial in the tobacco control field because of their potential to normalise nicotine addiction, as well as their resemblance to smoking in appearance, hand-to-mouth action and visual similarity of the exhaled 'vapour' to smoke. Criticisms of e-cigarettes are often couched in terms of their unknown health risks and their potential to be a gateway into cigarette smoking for young people. Bell and Keane (2012) argue, however, that opposition to e-cigarettes is primarily based on the “ideological challenge” that they pose to those in tobacco control. Nicotine has been dichotomised into the “bad” form that is sold by tobacco companies and the “good” form that is sold by pharmaceutical companies. E-cigarettes cannot be neatly categorised into either of these categories. Moreover, they represent a potential for nicotine to be consumed for pleasure, rather than for therapeutic purposes, without the harms caused by smoking tobacco.

In summary, while it is obvious that smoking has been medicalised to some degree, this has remained “partial and fragmented” (Rooke, 2012). Tobacco smoking has been defined as a chronic (brain) disease by some influential organisations, but this conception of tobacco dependence has not gone unchallenged and various factors may limit its reach. One thing that is clearly missing from the debate about the merits or otherwise of the medicalisation of tobacco dependence and nicotine addiction are the voices of those who smoke.

#### **2.10. Lay understandings of smoking and nicotine addiction: the missing link?**

Much of the literature outlined above speculates about the impact of the biomedicalisation of smoking on public perceptions of smoking and cessation. For example, Chapman & McKenzie state that disempowered smokers might feel that “it would be foolish to attempt to stop unaided when unassisted cessation is dismissed in pharmaceutical industry-supported demonstrably misleading propaganda” (Chapman & MacKenzie, 2010, p. 4). Caron et al write that medicalising smoking “could affect smokers’ understandings of the health risks associated with their behaviour, their perception of the nature of their addiction, and their perception of the need for medical assistance in quitting” (Caron et al., 2005, p. 188).

There is a dearth of research investigating whether smokers do in fact hold these fatalistic views and, if so, whether they are linked to beliefs about the nature of their tobacco dependence. The

next chapter will describe and evaluate the current evidence on lay understandings of nicotine addiction, attitudes towards smoking cessation treatment, and the role played by neurobiology in addiction to smoking.

## **Chapter 3. Lay understandings of smoking and the role of the brain in addiction.**

The previous chapter concluded that lay perceptions of smoking need to be examined to assess the extent to which smokers endorse biomedical approaches to smoking cessation. This chapter outlines research on lay understandings of addiction and smoking. It then describes a specific example of biomedicalisation: the brain disease model of addiction; outlines evidence on lay understandings of the brain; and describes the need for further research on how smokers understand the role of the brain in addiction and smoking.

### **3.1. Lay understandings of health and illness: A brief review of the literature**

The importance of conducting research on lay understandings of health and illness has been acknowledged in psychology (Furnham, 1988; Molden & Dweck, 2006), public health (Popay & Williams, 1996) and addiction research (Quintero & Nichter, 1996). During the 1970s, there was a growing emphasis on the importance of lay beliefs about health and illness, particularly within medical sociology. This arose from concerns that dominant biomedical and mechanistic models of the body excluded important experiential aspects of health and illness, and did not take account of socio-environmental influences on health and disease. There was a tendency on the part of the medical profession to characterise lay or “folk” knowledge as inaccurate, with public and patient education recommended to correct common “misperceptions” of health and illness and encourage healthier lifestyle choices. This has come to be known as the *deficit model* of lay beliefs (Popay & Williams, 1996). An alternative way of understanding lay beliefs is exemplified by Popay and Williams (1996), who argue that “lay expertise” should be seen as a valid form of knowledge with important consequences for public health:

“...through a more or less systematic process whereby experience is checked against life events, circumstances and history, lay people acquire an 'expert' body of knowledge, different from but equal to that of professionals in the public health field.”

(Popay & Williams, 1996, p. 760).

The term “lay epidemiology” was introduced by Davison, Smith and Frankel in 1991 to describe the sophisticated “daily cultural practice” by which people account for the development (or otherwise) of health problems (Davison, Smith, & Frankel, 1991). They conducted ethnographic studies that explored how members of the public in Wales understood the risk of developing heart disease. While they observed overlap between aetiological beliefs



of laypeople and medical professionals, they also described how aetiological lay beliefs were “personalised modifications” of those disseminated throughout society by professional organisations and the media. These modifications were employed to account for the “anomalous deaths and unwarranted survivals” that were not consistent with public health campaigns about the lifestyle factors that cause heart disease (Davison et al., 1991). The authors argued that public health professionals were guilty of “worthy dishonesty” in exaggerating the risks of certain behaviours to the individual, and overstating the benefits of their adopting behaviour change. They noted the potential for this strategy to backfire, undermining public trust of public health information.

In another seminal work on lay understandings of illness, Blaxter (1983) conducted interviews with low-income women from Wales about their beliefs about the causes of disease and illness. Like Davison and colleagues (1991), Blaxter emphasised the sophisticated nature of lay beliefs about the causes of disease. Even when wrong, these beliefs were “painstakingly” developed, based on social factors and an individual’s personal history. The important role of these beliefs in clinical interactions was highlighted. Blaxter wrote “In the surgery, the doctor’s views of the disease process must be reconciled with the patient’s. The diagnosis must make sense in terms of the patient’s models or it will not be accepted.” (Blaxter, 1983, p. 69). Much research since has shown that patient beliefs about illness, health, or medication can affect their health behaviours, treatment preferences and treatment adherence (Fucito et al., 2009; Horne & Weinman, 1999; S. Munro et al., 2007; Petrie, Jago, & Devcich, 2007).

The field of Public Understanding of Science (PUS) is another interdisciplinary field that has developed in parallel with lay understandings of health and illness. Like research on lay understandings of health, PUS began from a deficit model in which “the public” was often portrayed as lacking in scientific literacy. This lack of scientific knowledge was seen as obstructing the acceptance of new scientific technologies. “Science literacy” was proposed as the solution and the role of the scientist was to educate the lay populace about science on the assumption that this would increase positive attitudes towards the technology (Bauer, Allum, & Miller, 2007). The field has since shifted to a “science and society” paradigm that acknowledges lay expertise (Wynne, 1992), questions key assumptions about science (e.g., that it is value free), and advocates for engagement between scientists and the public (Bauer et al., 2007). It is now acknowledged that there are multiple publics made up of various stakeholder groups rather than a homogenous whole that includes everyone except scientists.

While there has been some debate about the extent to which lay knowledge should be considered as a form of “expert” knowledge (Prior, 2003), there is a general agreement that lay beliefs about disease or biotechnology can influence healthcare decision-making. Studying lay beliefs about health and illness is therefore an important endeavour. The following section will outline empirical research on lay understandings of addiction in general and then consider lay concepts of addiction to cigarettes specifically.

### **3.2. Lay understandings of addiction**

Furnham and Thomson define a lay person in relation to addiction as “a non-medical/psychological professional who has no expert knowledge on addiction.” (Furnham & Thomson, 1996, p. 29). Research on lay understandings of addiction is important because it can elucidate public attitudes towards addicted individuals and the attitudes of addicted individuals towards their own addiction. Beliefs held by the public and politicians about addiction can influence policy regarding the most appropriate forms of treatment for addiction and the allocation of resources to such treatments (Broadus & Evans, 2015; Kuppin & Carpiano, 2006; Luty & Grewal, 2002).

Scientific and clinical definitions of addiction are complex and contested (Campbell, 2007; Edwards, 2010; Howard, 2010; Sussman & Sussman, 2011; White, 1998). In the scientific and clinical arena, there is little agreement on whether the terms addiction or dependence should be used to describe the constellation of behaviour and experiences of those who struggle with their drug use. Diagnostic criteria and labels for addiction have changed over time. As we have seen, since nicotine addiction came under the purview of the DSM, it has been named “tobacco dependence” (DSM-III), “nicotine dependence” (DSM-IV), and “tobacco use disorder” (DSM-5). Many researchers now use the words interchangeably, but each has differing implications (O'Brien, 2011). Given the debate and lack of clarity amongst experts, it is not surprising that lay understandings of addiction/dependence are also sometimes contradictory and varied.

As described in the previous chapter (Section 2.2), the history of addiction to nicotine has differed to that of other drugs. This is reflected by differences in public perceptions of smoking compared to alcohol dependence and illicit drug use (Cunningham, Sobell, Freedman, & Sobell, 1994; Hughes, 2009). It is beyond the scope of this thesis to comprehensively review the literature on professional and lay understandings of all forms of drug addiction. However,

a number of key findings can be drawn from the literature on lay understandings of addiction more generally.

First, clinicians, addicted individuals, and the general public hold multidimensional, and often contradictory, understandings of addiction. Their responses do not fit neatly into pre-defined theoretical models of addiction, such as the “moral model” or the “medical model” that have been proposed in attribution theory (Brickman et al., 1982). Even those who work in the medical arena do not solely explain addiction as an individual, medical problem. For example, Palm (2004) found that drug and alcohol clinicians supported a mixture of moral, disease *and* sociological models of addiction. Most agreed that addicted individuals had responsibility for the development of their addiction, and for resolving it, but many also agreed that addiction was a disease, particularly addiction to alcohol. Importantly, Palm (2004) notes that a major limitation of their research is that it is not clear how the respondents interpret the word “disease” in this context. While the AA (Alcoholics Anonymous) model of addiction as a disease does not necessarily involve a medical or biological portrayal of addiction, more recent descriptions of addiction as a “brain disease” highlight the biological and medical aspects of the disorder. It is unknown to what extent members of the general public, or addicted individuals themselves, locate the disease concept of addiction within this medical frame.

West and Power (1995) also found that the oft-cited “helping-coping” model of addiction, where beliefs about responsibility for addiction are related to beliefs about the need for treatment, was too simplistic to account for their research findings with addicted individuals. The alcohol dependent patients they surveyed held contradictory beliefs about the cause of their addiction. For example, they thought that their addiction was their own responsibility but also believed that it was caused by external factors. Moreover, they often agreed that they needed to take control of their own recovery, but help and support from others would still be required. The authors found no association between beliefs about responsibility for addiction and beliefs about whether treatment or assistance was required. Similarly, an empirical study with addiction clinicians and neuroscientists found that most believed that addiction impaired control over drug consumption, but that individuals remained responsible for their behaviour (Carter, Mathews, Bell, Lucke, & Hall, 2013).

The research on lay understandings of addiction has focused on survey studies, where items are often developed in a top-down manner based on existing scientific theories of addiction,

rather than arising from exploratory qualitative research with members of the public or addicted individuals themselves (Broadus & Evans, 2015). Where exploratory research is conducted with addicted individuals, they are mostly clinical populations, and thus do not represent the many who do not seek formal treatment, though there are exceptions (Broadus & Evans, 2015). Survey studies are limited because they may ask whether people think addiction is a disease, but it is not clear what conceptualisation of disease individuals think of when they respond to this question. Similarly, survey research enquires about responsibility for addiction, but it is unknown how responsibility is conceptualised by participants: are participants thinking about their moral responsibility for the choices they have made in leading to addiction, or responsibility for future decisions in order to overcome their addiction? To overcome the lack of empirical evidence on how lay people conceptualise addiction, Weinberg has recommended an empirical approach to addiction, with an emphasis on the “local practice” of addiction as an embodied experience (Weinberg, 2002, 2013).

A number of qualitative studies have taken this approach. One such study asked a small sample of addicted males from a prison treatment program, as well as addiction experts (psychologists), to define addiction in their own words (Walters & Gilbert, 2000). They found similarities and differences between lay beliefs about addiction and “expert” definitions. For example the authors found that few of the addicted individuals referred to addiction as a biological problem, physical dependence, or a disease. They were more likely to describe addiction in terms of craving, pre-occupation, self-destruction, abuse and diminished control. The expert group were more likely to describe addiction as physical dependence, compulsion, and as a biological problem. The authors argue that the client group held a broader view of addiction that could encompass behavioural addictions, while the expert group focused more on the physical aspects of addiction to a substance. Nonetheless, both groups agreed that preoccupation with drug use, and diminished control were key aspects of addiction.

A more recent qualitative study asked members of the public (not addicted individuals specifically) open-ended questions about what they thought addiction was. They found that references to character were most common in explaining addiction (Meurk, Carter, Hall, & Lucke, 2013). Participants described the role of choice, willpower and personality as central to addiction. They often referred to the emotional aspects of addiction, for example that people smoke to feel pleasure, or to escape boredom. Societal factors, such as family exposure and peers, were also cited frequently as influencing the development of addiction. Biological

factors were discussed frequently, with 71% talking about the role of the body in addiction, and 51% spontaneously talking about the role of the brain in addiction. Most participants ascribed addiction to more than one of these causal categories.

Another important finding from the literature on lay understandings of addiction is that beliefs about addiction vary with socio-demographic characteristics such as age, gender and political affiliation. For example, Broadus and Evans (2015) found that in the US, females were more likely to endorse a psychological explanation of addiction, and males more likely to believe that addiction had a moral component (Broadus & Evans, 2015). Another study found that women were more likely to attribute addiction to biology than men, and also more likely to see value in treatment (Kauffman, Silver, & Poulin, 1997). Those who had received treatment for addiction were less likely to believe that addiction was a moral weakness and more likely to believe it was a disease. This could be because exposure to a disease model via treatment centres affected their beliefs (Broadus & Evans, 2015), or because those who have a pre-existing belief that addiction is a disease are more likely to seek medical treatment. Politically conservative Americans were more likely than liberals to believe that addiction was a sign of moral weakness (Broadus & Evans, 2015; Furnham & Thomson, 1996). Together, these findings show that conceptions of addiction are not uniform across social groups. Therefore important to examine group differences in research on lay understandings of addiction.

Beliefs about addiction also vary over time and between cultures. Pescosolido and colleagues (2010) examined changes in attribution of the causes of addiction in the US general public between 1996 and 2006. They found that the belief that alcohol dependence was caused by biology (genetics or chemical imbalance in the brain) increased from 38% in 1996 to 47% in 2006. At the same time, attributions of “bad character” for alcohol dependence increased from 49% of participants to 65%. This suggests that a moral and a biological conception of addiction are not necessarily mutually exclusive in lay understandings of addiction. They also found that accepting a neurobiological conception of alcohol dependence did not reduce the stigma of addiction, as has been predicted by proponents of the BDMA. Indeed, it was associated with increased levels of stigma. Compared to other mental illnesses and physical illnesses such as cancer and myocardial infarction, those addicted to alcohol are seen as more responsible for their illness, and the public express a desire for social distance from alcohol-dependent individuals (Pescosolido et al., 2010; Schomerus, Matschinger, & Angermeyer, 2006). This

suggests that addiction to drugs or alcohol is not seen as a “disease like any other” by the public.

### **3.3. Smokers’ understandings of nicotine addiction**

The literature on lay understandings of addiction to smoking spans numerous disciplines and encompasses varied methodological approaches. Most tobacco control research uses validated measures of tobacco dependence that are based on smoking behaviours. For example, the Heaviness of Smoking Index (HSI) measures level of nicotine dependence using two questions: the time to first cigarette of the day after waking and the number of cigarettes smoked per day. These items provide a more objective measure of dependence than subjective ratings of self-reported addiction (Heatherton, Kozlowski, Frecker, Rickert, & Robinson, 1989). A smokers’ subjective assessment of their own level of addiction to cigarettes is often neglected, even though it may have implications for treatment choices. This is particularly pertinent in the case of pharmacotherapy for smoking cessation. Nicotine replacement therapy is predicated on the assumption that the individual smoker has a physiological addiction to nicotine, and that replacement of nicotine via NRT will reduce their withdrawal symptoms and craving and increasing their chance of quitting. However, if an individual smoker does not believe that they are addicted to nicotine, it is unlikely that they will find NRT an appealing option, or comply with recommended ways of using it, if it is prescribed.

Surprisingly few quantitative studies have examined smokers’ subjective assessment of their addiction to smoking. Those that have assessed subjective views on smoking have used inconsistent terminology to enquire about addiction. Studies have asked about addiction to tobacco (Carpenter et al., 2009; Hughes, 2009; Okoli, Richardson, Ratner, & Johnson, 2009), to smoking (Levinson et al., 2007; Martin, 1990), and to cigarettes (Stippe Kohl et al., 2012; Torchalla, Okoli, Malchy, & Johnson, 2011; Weinstein, Slovic, & Gibson, 2004). Interestingly, only one quantitative study found in the literature asked about subjective assessments of *nicotine* addiction (Zinser, Pampel, & Flores, 2011). Studies also differ in whether they ask participants if they are addicted to smoking, or the more general statement that smoking is addictive. Despite these methodological variations, the results of these studies show that most daily smokers report that they are addicted to smoking, and that smoking is addictive. In the majority of these studies, 70% or more daily smokers report that they are addicted to smoking. The proportion of people who agree that they are addicted to smoking is lower in younger smokers (Arnett, 2000; Weinstein et al., 2004), in those who don’t smoke daily (Edwards,

Bondy, Kowgier, McDonald, & Cohen, 2010) and in Latino smokers in American studies (Zinser et al., 2011).

Despite the fact that many smokers report that they are addicted to smoking the fact that pharmacotherapies for smoking cessation are widely available, approximately 50% of quit attempts are made without assistance. When pharmacotherapy is used, it is often discontinued early or used in lesser amounts than recommended (Balmford et al., 2011; Shiffman, Ferguson, Rohay, & Gitchell, 2008). A number of studies looking at smokers attitudes towards NRT have provided some insight into why this may be.

Three main themes have emerged from this research. Firstly, many smokers do not believe that NRT is effective for smoking cessation (Hammond, McDonald, Fong, & Borland, 2004; Shiffman et al., 2008; Willems, Willemsen, Nagelhout, & de Vries, 2013). This is partly attributable to a belief that NRT does not help with cravings (Vogt, Hall, & Marteau, 2008). While some clinicians see this as a misperception that requires correction (Shiffman et al., 2008; Vogt et al., 2008), others believe it to be a realistic assessment based on the past experiences of many smokers who have failed to quit using pharmacotherapy.

Smith, and colleagues (2015) conducted qualitative interviews with 21 Australian ex-smokers who had quit without assistance about their experiences. They identified a number of reasons why smokers had rejected a medical approach to cessation, and chosen to quit without medical assistance. Participants often evaluated the efficacy of pharmacotherapy based on their personal experiences, rather than on information from health professionals or pharmaceutical companies. As previously noted, although pharmacotherapy does marginally improve one's chances of quitting, it is not successful for the majority of those who use it, especially as many do not adhere to the recommended dosage or treatment duration. This means that smokers can easily draw to mind instances where pharmacotherapy for smoking cessation has not been successful. Also, the authors describe the way participants engaged in a cost-benefit analysis in relation to quitting methods. Because unassisted quitting is free, immediate, and safe, it was often preferred over methods that required more preparation, financial cost and had risks of side effects. Cultural beliefs about personal responsibility, strength, and self-control were also influential in decisions to quit unassisted. However, it must be noted that this study interviewed only those who had quit unassisted, so was likely to recruit those with negative views or

experiences of assisted quitting. Those who had successfully quit with assistance were excluded from the study.

Second, many smokers express concerns about the health risks of pharmacotherapy for smoking cessation. A number of studies have found that smokers mistakenly believe that nicotine is harmful to the body and causes cancer (Bansal, Cummings, Hyland, & Giovino, 2004; Cummings et al., 2004; Mooney, Leventhal, & Hatsukami, 2006; Shiffman et al., 2008). This is likely to have occurred because of the close association between nicotine, cigarettes and the health harms of smoking (Shiffman et al., 2008). It may be difficult for people to reconcile the idea that the harmful effects of nicotine addiction in the form of cigarette smoking are much reduced when they obtain their nicotine in the form of NRT (Keane, 2013; Smith et al., 2015). Concerns about health risks are even more pronounced in relation to prescription pharmacotherapies for smoking cessation. Vogt, Hall and Marteau (2008) found that many participants believed that NRT had mild side effects while bupropion had serious and life-threatening side effects. The stringent requirements for labelling of side effects of pharmacotherapies have been suggested as one reason for concern about the health risks of pharmacotherapies (Shiffman et al., 2008). Unfortunately, there is little research on smokers' attitudes towards varenicline, where potential health harms (e.g., increased suicide risk and suicidal ideation, depression and aggression) have been prominently reported in the media. While recent pharmacoepidemiological evidence suggests that varenicline is safe (Kotz et al., 2015), the FDA in the US have maintained a "black box" warning on medication packaging alerting consumers to potential neuropsychiatric side effects. No such warning is required in Australia. Interestingly, though prescription medication for smoking cessation has relatively low use in Australia, a sample of recent quitters from Australia found that those who had used it rated it as having high helpfulness (Hung et al., 2011).

Efficacy and safety are not the only reasons why smokers do not use cessation medications. A number of qualitative studies report that willpower and personal responsibility are key terms in smokers' discourses about their smoking and cessation (Katainen, 2006; Kayser & Semenic, 2013; Smith et al., 2015; Vogt et al., 2008). The use of NRT may be discordant with smokers' conceptualisations of quitting that centre on the key role of internal strength and willpower. In addition, some smokers reject the idea that they have a physiological dependence on nicotine (Vogt et al., 2008), while others believe that this is only a part of their addiction (Wiltshire et al., 2003). Other aspects of smoking that are central in smokers' accounts of their smoking are



the pleasure that they experience from smoking, the use of smoking as a tool for emotional regulation (e.g., to deal with stress or boredom), and the embeddedness of smoking in their everyday life and social relationships (Amos, Wiltshire, Haw, & McNeill, 2006; Bancroft, Wiltshire, Parry, & Amos, 2003; Laurier, McKie, & Goodwin, 2000; Parry, Thomson, & Fowkes, 2001; Thompson, Thompson, Thompson, Fredickson, & Bishop, 2003). Smoking is often described by smokers as having a powerful behavioural or psychological aspect that cessation medications do not address (Uppal, Shahab, Britton, & Ratschen, 2013; Vogt et al., 2008; Wiltshire et al., 2003). The key common finding in these studies is that smokers believe that the physiological aspect of their addiction only partly explains why they continue to smoke.

A small number of studies have examined smokers' attitudes towards a biomedical model of smoking. A very early study by Eiser and Van Der Pligt (1986), presented participants with 20 items about their perceptions of smoking. Based on the results the authors suggested that there were two key ways that smokers understood their smoking. The first was as a "sickness". The participants who scored highly on this factor were more concerned about the health consequences of smoking and more likely to seek treatment from health professionals. The second factor was being "hooked". These smokers had low quitting self-efficacy and resented efforts by others to convince them to quit. Eiser and Van Der Pligt (1986) hypothesised that these beliefs could influence behaviour change and suggested that health professionals could tailor their message to the beliefs that their patients held.

A more recent study with British smokers identified four "smoking identities" (Farrimond, Joffe, & Stenner, 2010). The authors related the first two identities, "addicted smoker" and "in control smoker" to the biomedical model of smoking. Like Eiser and Van Der Pligt (1986) these authors found that "addicted smokers" were concerned about the health risks of smoking, agreed that smoking was a medical problem, and felt they had low levels of control over their smoking. Smoking was perceived as a negative behaviour that was nonetheless functional, in that it helped them to deal with emotions such as stress and boredom. The "in control" smokers also believed that smoking was addictive and detrimentally impacted health, but were more likely to describe smoking as a "habit" or as a social tool, and believed that they were in control of their smoking. Another smoking identity was labelled the "no big deal" smoker. These smokers were more likely to question the health harms and the addictiveness of smoking. Smoking was seen as a luxury, a means of escape and as a way to deal with boredom. Smoking

was not a central aspect of identity but a behaviour that could be controlled. Lastly, they described the “proud” smoker, who did not hold strong views about the health or addictive aspects of smoking, and endorsed positive aspects of a smoking identity, such as “smoking is sexy” and “rebellious.” Smoking was seen as a choice and as a “right.” Those who fell into this category were more likely to say they enjoyed smoking. The authors suggest that different smoking interventions may be more suitable for different types of smoker identities (Farrimond et al., 2010).

Another study that interviewed ex-smokers who had quit unassisted, found that one of the common reasons for rejecting pharmacotherapy and medical treatment was that they did not see smoking as a medical issue (Smith et al., 2015). Instead they described smoking as a matter of willpower and gave primacy to individual responsibility and autonomy.

Lastly, a study by Hughes (2009) looked at current smokers’ preparedness to endorse biomedical explanations of smoking. The participants were asked why they thought people continued to smoke. The results showed very high levels of agreement with the ideas that an inability to stop smoking was caused by addiction (88%) and was due to habit (also 88%). Interestingly, despite high agreement that smoking was addictive, there was low agreement that inability to quit smoking was caused by biological factors (21%), or that smoking was a “disease” (10%). Hughes concludes that:

“In terms of significance, although scientists may argue that certain concepts are anti-*thetical* (e.g., addiction vs. habit), many, if not most, smokers appear to not see contradictions in simultaneously endorsing what appear to be very different causes: i.e., addiction, habit, willpower and motivation, as causes of the inability to stop smoking. Thus, one implication of our results is that scientists cannot assume that smokers have the same network of concepts or the same denotative or connotative meanings of terms that scientists do”. (Hughes, 2009, p. 1008).

Hughes recommended conducting more qualitative research to explore the meanings that smokers attach to terms such a “habit” and “addiction.”

Together, these findings show that for many smokers addiction is a very broad concept that encompasses more than a physiological dependence on nicotine. Smokers do not necessarily equate addiction to smoking with addiction to nicotine, or believe that the addiction is

biologically-based. When reflecting on their smoking, they give prominence to factors such as personal choice, the social aspects of smoking, pleasure, and the roles that cigarettes play in their everyday lives.

### **3.4. Lay beliefs about the brain and behaviour**

As described in the previous chapter, there is an increasing emphasis on the role of the brain in the development and maintenance of all forms of addiction. Some have claimed that addiction is a brain disease, and that describing it in this way will lead to widespread changes in the way that the public, and addicted individuals themselves, perceive addiction. More specifically, it is hypothesised that it will lead addicted individuals to see their addiction as a medical problem requiring medical treatment. An increasing number of studies have investigated stakeholder knowledge and attitudes towards neurobiological technologies and their implications for various facets of human existence, including adolescence (Choudhury, McKinney, & Merten, 2012), mental illness (Borgelt, Buchman, & Illes, 2011; Bröer & Heerings, 2012; Pescosolido et al., 2010) and addiction (Meurk, Carter, et al., 2013; Meurk et al., 2016; Netherland, 2011).

Neuroscience findings are increasingly being reported in mainstream media (O'Connor, Rees, & Joffe, 2012). Public interest has often focused on brain imaging studies using functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) (Racine, Bar-Ilan, & Illes, 2006; Racine, Waldman, Rosenberg, & Illes, 2010). While in-depth anthropological studies have outlined the complex decisions and judgements involved in producing these images (Dumit, 2003; Joyce, 2005), they are often mistakenly portrayed in the media as objective “snapshots” of an individual’s brain function (Beck, 2010; Fine, 2010). They have been used to demonstrate the “realness of a disorder” (Racine, Bar-Ilan, & Illes, 2005) and to compare the characteristics of “normal” and “disordered” brains (see Figure 3-1).

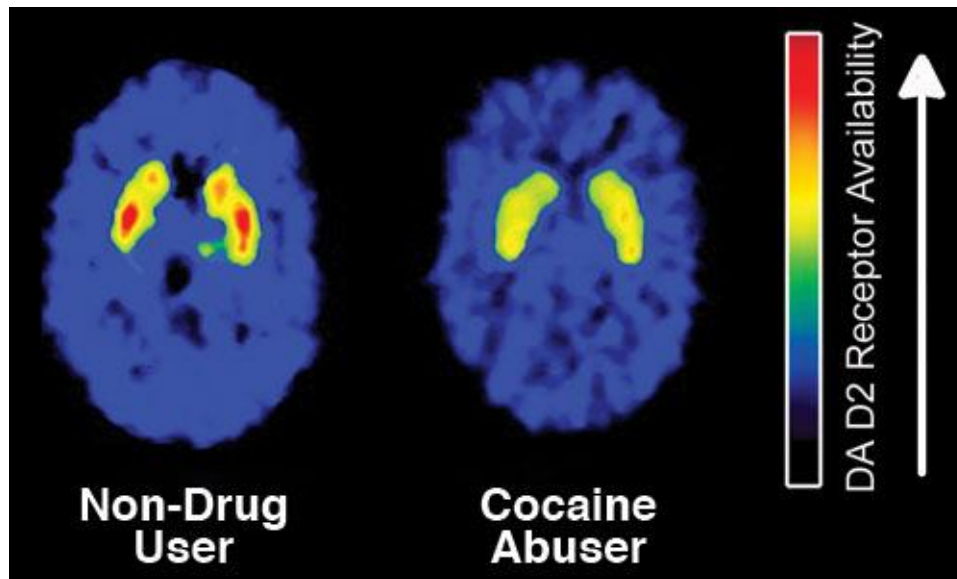


Figure 3-1 Image comparing non-drug user and cocaine user brain scans.

From NIDA webpage Cocaine: Abuse and Addiction

(<http://www.drugabuse.gov/publications/research-reports/cocaine-abuse-addiction/what-are-long-term-effects-cocaine-use>).

Although neuroimaging does not currently have the power to diagnose individuals with an addiction or a psychiatric disease, there is hope that it will one day provide reliable biomarkers to diagnose individuals or to identify those at greatest risk of becoming addicted (Agarwal, Port, Bazzocchi, & Renshaw, 2010). To explore patient knowledge and attitudes towards neuroimaging, Buchman, Whiteley and Iles (2013) conducted interviews with those diagnosed with major depressive disorder or bipolar disorder. Participants typically believed that neuroimaging would reduce the stigma of their mental illness and make it more “real” in the eyes of others. Explanations of mental illness involving a “chemical imbalance” in the brain were central to many of these participants, who were optimistic that their illness might be better treated if their brain scans could show this in an objective way (Buchman et al., 2013). Ethnographic research has similarly shown that those with mental illnesses often see brain scans as legitimizing their disorder, by providing evidence that it has a physical basis (Cohn, 2010; Dumit, 2003). Simon Cohn (2010), who conducted such research at scanning sites, describes informants who carried their brain scans in their handbag, presented their brain scans to their family or friends, or framed them and put them on display.

The “persuasive power” of brain images has been demonstrated experimentally. McCabe and Castel (2008) provided a sample of undergraduate students with a number of invented news articles about cognitive neuroscience findings in which the conclusions in the articles were

inconsistent with the data. Each article was accompanied by a brain image, a bar graph, or no image. Participants were asked to provide ratings of the validity of the scientific reasoning in each article. Ratings were highest when the article was accompanied by a brain image. A more recent study however, did not support this conclusion, finding no significant difference in participants' judgments of articles accompanied by no image, a brain image or alternative images (e.g., an artistic representation of a head) (Gruber & Dickerson, 2012). A related experiment showed that satisfaction with an explanation increased when adding textual neuroscientific information to explanations of psychological phenomena, even when the neuroscientific information was irrelevant. This was true even when the original explanation was a circular restatement of the original report and thus did not add any explanatory improvement (Weisberg, Keil, Goodstein, Rawson, & Gray, 2007). These results indicate the potential for neurobiological information to influence reasoning about human behaviour, but the conflicting findings indicate that more research is required on how it may do so (Baker, Ware, Schweitzer, & Risko, 2015).

Other studies have revealed more ambivalence among the public about neuroscientific explanations of human behaviour. Choudhary, McKinney and Merten (2012) conducted focus group and surveys with young people about the “neurological adolescent”, the idea that the brains of teenagers are different from those of adults in ways that can account for adolescents' greater risk taking and reduced attention to the consequences of their behaviour. While some young people thought that neuroscience might help adults to better understand their behaviour, they were also concerned that it would reinforce negative stereotypes. It was common for these young people to widen the explanatory frame by providing alternative explanations that did not rely on brain science. The authors conclude that:

“...neuro-identity formations are more fractured, resisted and incomplete than some of the current social science literature on neuro-subjectivities seem to suggest and that the effects of public policy and popular education initiatives in this domain will be more uneven and complex than currently imagined.”(Choudhury et al., 2012, p. 565)

This finding is supported by other research demonstrating that persons with ADHD combine explanatory frameworks in understanding their disorder. Neurobiological explanations of ADHD are common but do not dominate; psychological and social explanations remain central (Bröer & Heerings, 2012).

Pickersgill, Cunningham-Burley and Martin (2011) interviewed various stakeholder groups about their ideas regarding neuroscience. These included: patients with a brain-based disorder such as epilepsy or dementia; professionals expected to come into contact with neuroscience ideas in their work; and neuroscientists themselves. They found that many of those affected by a brain-based disorder expressed interest in neuroscience and in understanding how their brain worked. However, they often resisted accounts that ascribed dominance to their brain in their identity or actions. Pickersgill and colleagues conclude that the brain is “an object of mundane significance”, and that is only one factor in the complex process of identity formation (Pickersgill et al., 2011, p. 361).

Pickersgill (2009) has found that even amongst scientists involved in neurobiological research, their ideas about the aetiology of mental disorders are complex, often acknowledging the importance of social factors such as family upbringing, socioeconomic status and the social environment. He conjectures that:

“Long-standing critiques of biological reductionism are rendered problematic by the complex linkages scientists make between psyche, soma and society” (Pickersgill, 2009, p. 58).

A systematic review by O'Connor & Joffe (2013) investigated the impact of neuroscience on various aspects of personhood. They identified three primary claims made about the impact of brain-based understandings of human behaviour: (1) that biology will become central to individual identities; (2) that people will become more fatalistic about their behaviour; and (3) that there will be a reduction in stigma associated with certain groups and behaviours. They conclude that empirical evidence to date does not support any of these claims. Rather, they suggest that people incorporate beliefs about neuroscience into pre-existing worldviews in creative and often unpredictable ways. Cliodhna O'Connor (2013) also conducted interviews with members of the public about their ideas about neuroscience. She found that neuroscience was not considered to have much relevance in most participants' lives. When they did deliberate on brain research, those interviewed often described the brain as “a tool over which individuals could exert control, and as a source of human variation invoked to articulate and explain social differences” (O'Connor, 2013, p. 249).

### **3.5. The place of neurobiology in public understandings of addiction**

Most research conducted thus far on lay understandings of the brain has focused on mental illness. There is little comparable research on how addicted individuals make sense of addiction in light of brain-based models, although such research has been recommended (Caron et al., 2005; Fry & Buchman, 2012; Weinberg, 2013). One recent study has explored the potential of brain-based explanations to influence the ways in which addicted individuals understand their drug use. In interviews conducted with 37 opiate addicted participants treated with buprenorphine (a synthetic opiate used for replacement therapy of opioid addiction), most participants did not express a scientific account of the effect of drugs on their brain, despite their heavy exposure to a medical model of addiction via their opiate substitution treatment regime (Netherland, 2011). Rather, they discussed ideas such as pleasure, normality and rationality/irrationality, and provided explanations of their addiction that focused on external factors that influenced their drug use and lifestyle (Netherland, 2011).

Another study examined how US addiction counsellors attributed responsibility for the development of addiction and recovery in light of their exposure to addiction neuroscience (Steenbergh, Runyan, Daugherty, & Winger, 2012). The study found that addiction counsellors were more likely to agree that biological factors caused addiction than social factors, spiritual factors and individual choices. They also found that exposure to neuroscience was associated with lower ratings of personal responsibility for developing addiction, but this was not true for personal responsibility for resolving addiction. This may be due to the personal experiences of counsellors who assist addicted individuals to make the choice to reduce or cease drug use, and for whom it is necessary to believe that their profession can influence addicted persons to become abstinent. The authors conclude that “A view of addictions that balances biological factors with personal agency, like the one espoused by our sample of counsellors, seems necessary for successful intervention.” (Steenbergh et al., 2012, p. 427). Other research with addiction professionals, including clinicians and researchers in Australia, has similarly found that the professionals did not uncritically support the brain disease model of addiction. They were also cognisant of the potential adverse impacts of the model such as an increase in stigma and a reduction in quitting self-efficacy (Barnett & Fry, 2015; Bell et al., 2013).

### **3.6. Smoking and the brain: Public portrayals**

As described above, brain-based explanations of smoking are increasingly being presented in the popular media. Media reporting on neuroscience has been criticised for portraying brain

scans as a direct representation of reality, and for speculating on unlikely or infeasible policy implications of neuroscience research (O'Connor et al., 2012; Racine et al., 2010). In Australia, the mainstream media have reported on neuroscience findings about smoking. Some excerpts are provided in Figure 3-2.



**Smokers' addiction all in the brain (Courier-Mail, 23 March 2002, Ronald Kotulak)**

Working to unravel a long-standing puzzle of cigarette addiction, University of Chicago researchers have discovered why smoking is uniquely pleasurable and why nicotine has such ferociously addictive powers. Published in the scientific journal *Neuron*, the research shows nicotine stimulates pleasure in the brain's reward centre and also has the unique ability to "turn off" the "switch" that subdues good feelings.

The new evidence helps explain how one cigarette quickly teaches the brain cells of a first-time smoker to crave nicotine. And for the thousands who try to quit smoking each year and fail, the findings show why breaking the habit is so hard. Drug companies' efforts to develop anti-addiction medicines have been hampered because they didn't know how the brain became addicted, says John Dani, a Baylor College of Medicine neuroscientist and one of the first to show nicotine's effect on dopamine. "McGehee's work will allow both academic and pharmaceutical researchers to focus on the mechanisms of addiction with a greater understanding of how they work."

**Science pinpoints smoking addiction (The Advertiser, 27 January 2007, Radowitz & Kleinig)**

Smokers have been warned not to rely on medical research to deliver them from addiction after U.S. scientists pinpointed the brain part responsive to the drug. Quit SA manager David Edwards said the discovery, by U.S. scientists, could help to develop a quit-smoking drug. "But the reality for smokers in the medium to short-term is still dealing with the challenge of breaking the link between nicotine and their bodies and brains," he said yesterday. "Getting advice and coaching from places like Quit and nicotine therapies are still the best combination."

The journal *Science* yesterday reported the discovery of a 2.5cm wide pleasure centre deep within the brain. The insula, in the cerebral cortex, has been described as a "platform for feelings and emotion". Researchers found smokers who suffered damage to this part of the brain were able to quit easily. They suspect the insula may also be involved in other forms of addictive behaviour that keep people hooked on drugs or excessive eating. Understanding the link could lead to new strategies and treatments for addiction.

**Quitting is a brain game (The Gold Coast Bulletin, 19 March 2011, Author unattributed)**

Researchers are finding evidence that parts of the brain are involved in gaining control over smoking and nicotine addiction, and that different areas may be important for each individual. Two recent reports look at the neural systems from various angles. One study found that people who had a stronger brain response in certain brain regions when getting tailored smoking-cessation messages were more likely to quit four months later.

The second study also used brain imaging to watch three specific brain regions known to affect inhibition of unwanted or habitual behaviour. The brain scans showed how well each person activated their "response inhibition" regions. "The more you activate those three brain regions when you are engaging successfully in stopping, the more likely you are to successfully deal with your cravings in real life," said Elliott Berkman, a researcher and co-author of the study.

Figure 3-2. Examples of media reporting on the neuroscience of smoking in Australia.

These examples attribute addiction to smoking to brain mechanisms that have been altered by nicotine consumption. They then link these brain changes to difficulty quitting and individual differences in quit success. The example from *The Advertiser* (27/1/2007) is of particular interest, as the Cancer Council spokesperson counsels smokers to quit using traditional means rather than waiting for brain-based therapies to become available, anticipating that unrealistic expectations may be raised by media reporting on neuroscience.

It is not only the media who disseminate brain-based explanations of smoking. A number of scientific journals have published papers suggesting that clinicians inform smokers of the neurobiological basis of nicotine addiction to increase their use of medications for smoking cessation. For example, Leone and Evers-Casey recommend turning the personal and “visceral” experience of addiction into a “tangible story that can be discussed and deconstructed” (Leone & Evers-Casey, 2012, p. 56). They specifically suggest that “a short conversation about the biological basis of motivation helps to minimize the exaggerated sense of self-blame and responsibility that smokers often feel, validates the patient’s position, and provides a viable framework for working through inevitable obstacles during the quit process.” (Leone & Evers-Casey, 2012, p. 56).

Raupach et al (2010) describe an intervention in which smokers enrolled in a cessation program were presented with information about the neural mechanisms of addiction using the metaphor of a pizza delivery service. Those exposed to this physician-run education session were more likely to use NRT than those who did not receive the education session. Unfortunately, the study was not sufficiently powered to detect a difference in cessation rates. Also, as the authors note, it was possible that physician presence in the “neural education” group, rather than the information itself, was responsible for the difference in NRT use. Lastly, Finnell (2000) has recommended that addicted individuals be provided with information on the neurobiological basis of addiction in order to “empower patients, ease their defences, and reduce the stigma they experience” (Finnell, 2000, p. 157). Only anecdotal evidence was presented that doing so would produce these positive outcomes.

Despite a lack of empirical evidence on how smokers incorporate neuroscience information into their understandings of addiction, Australian health promotion materials have begun to include information about the brain’s role in smoking. For example, a project aimed at Indigenous Australians called “Brain Stories” has been developed by Menzies School of Health

Research. The resources developed as part of the Brain Stories project emphasise the role of the brain in addiction to smoking, with the aim of overcoming the stigma of addiction and “empowering people with knowledge” (see Figure 3-3)

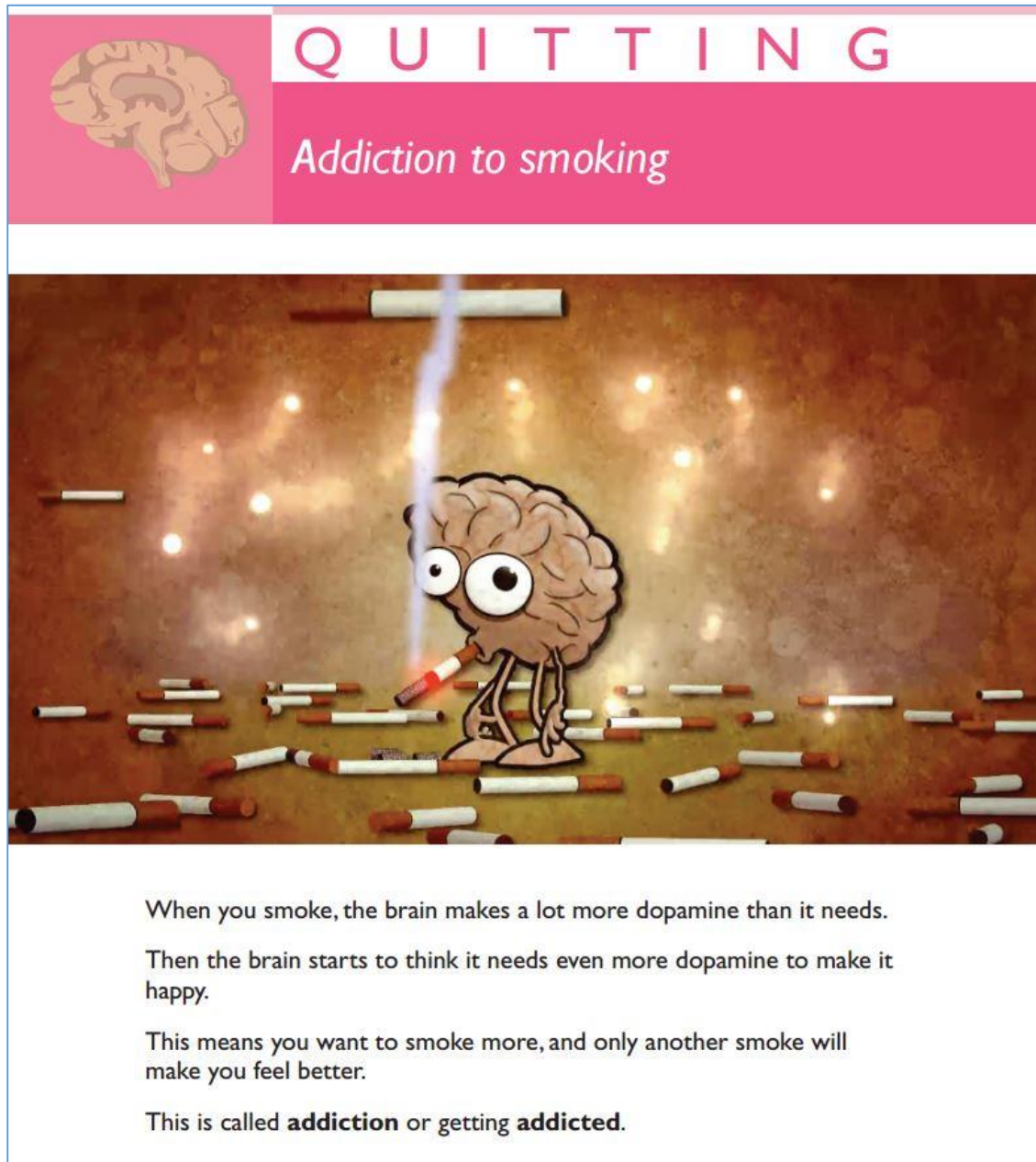


Figure 3-3. Image from the “Tobacco Stories” resource by Menzies School of Health  
From: [http://www.menzies.edu.au/page/Resources/The\\_Tobacco\\_Story/](http://www.menzies.edu.au/page/Resources/The_Tobacco_Story/)

Pharmaceutical companies are also using brain-related imagery and information to promote their smoking cessation products. On Pfizer’s “Nail Quitting” website visitors are first

presented with the text: “Some things are too hard to do on your own.” The website contains an animated video of a brain smoking a cigarette (See Figure 3-4) accompanied by the words:

“Cigarettes contain nicotine. When you smoke, the nicotine releases chemicals in the brain, producing feelings of pleasure. If the levels of these chemicals drop, you start to crave yet another cigarette. It’s called nicotine addiction. That’s why it’s so hard to quit smoking, even when the enjoyment is gone. Quitting isn’t easy. Your doctor has a range of quitting options, with and without nicotine.” (<http://www.nailquitting.com.au/quit-now/>)

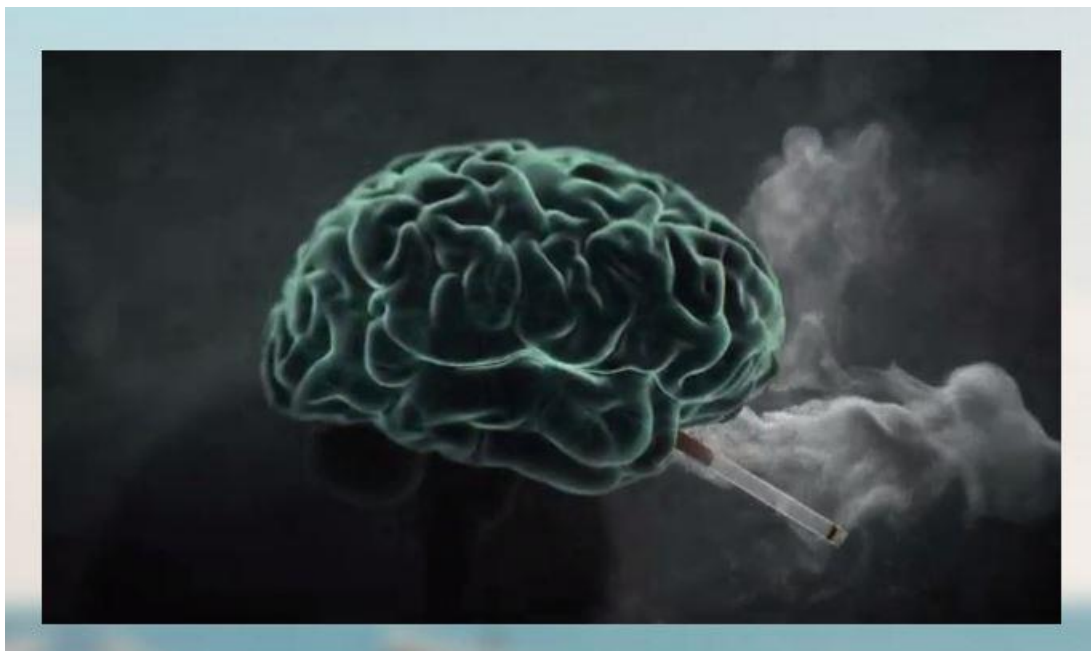


Figure 3-4. Image of a smoking brain from Pfizer’s “Nail Quitting” website

How might such public portrayals influence lay understandings of smoking? A number of theories articulate potential links between media representations and lay understandings of illness. For example, social representations theory states that representations are images or understandings that allow classification of people or phenomena (Jodelet, 1991). Various competing representations exist in relation to a particular phenomenon such as addiction, and people can confirm such representations, reject them or re-articulate them based on existing representations (Howarth, 2006). Smoking as a brain disease could be conceptualised as a novel social representation that becomes “anchored” to existing understandings of addiction. Anchor points are typically “core meanings” that underpin societies’ “overarching systems of ideologies, beliefs, maxims and categories.” (O’Connor, 2013).

In a less constructivist vein, many health behaviour theories conceptualise the way that people respond to health promotion messages that aim to change behaviour. For example, the health belief model proposes that people make health-related decisions by balancing ideas about the severity of the illness, their susceptibility, the effectiveness of taking action, and the social consequences of taking (and not taking) action. Media or health promotion messages may influence perceptions of severity or reduce perceptions of the cost of action in order to prompt a desired health behaviour (Becker, 1974). While such theories differ in terms of terminology and epistemology, they concur that new information about health and illness is not passively accepted, but is incorporated in complex ways into existing understandings, which are themselves drawn from a combination of cultural representations and everyday experiences.

### **3.7. The place of neurobiology in lay conceptions of smoking**

There is currently insufficient evidence to evaluate the impact of neurobiological explanations of addiction on smoking cessation. Only one study identified in this literature review directly examined smokers' ideas about the role of the brain in their smoking. This study described the experiences of people living with alcohol or nicotine dependence, with particular emphasis on how neurobiological and genetic explanations of addiction fitted into their personal narratives (Hammer, Dingel, Ostergren, Nowakowski, & Koenig, 2012). They found that over half of their 63 participants provided an explanation of addiction that included a biological component. Importantly, they noted that those recruited from treatment centres that emphasised the biological basis of addiction in patient education material were more likely to explain addiction in terms of the brain or genetics (Hammer et al., 2012). Given that most smokers do not seek formal assistance for quitting, this sample is unlikely to be representative of the general population of smokers.

Results from studies that provide smokers with information about the genetic basis of their nicotine dependence have found no clear evidence that this leads to either a significant increase in quit rates or an increase in fatalism, although it is conceivable that such information may reduce the perceived efficacy of quitting using willpower alone (Cappella, Lerman, Romantan, & Baruh, 2005; Wright et al., 2007; Wright, Weinman, & Marteau, 2003). More recently, a number of studies have explored the impact of providing "personalised" pharmacotherapy for smoking cessation based on genetic test results. Results suggest that such treatment does not result in fatalism or a reduction in sense of control over smoking (Marteau et al., 2012; McClure et al., 2013).

There are important differences between genetic and neuroscience explanations of addiction (Green 2006) and this thesis will examine whether providing neurobiological information about nicotine addiction has similar effects on smokers' perceptions of control over their smoking given. The research conducted thus far on how individuals respond to neurobiological explanations of behaviour has mostly been based on clinical populations. It is important that research also elicits the views of non-treatment seeking individuals, who may differ from treatment seekers in many important ways. The current research will address this gap in the literature by recruiting a community-based sample of smokers to explore their knowledge and understandings of smoking in light of brain-based explanations of addiction. It will address questions such as: Do smokers view their nicotine addiction as a "chemical imbalance" or a "brain disease"? In what ways do they incorporate neurobiological understandings into their everyday explanations for their smoking? And what implications might these brain-based understandings of smoking have for a smokers' sense of quitting self-efficacy, and treatment preferences?

### **3.8. Original contribution of literature review chapters**

This chapter, and Chapter 2, have outlined the literature relevant to understanding how smokers conceptualise the role of nicotine in their smoking, and the extent to which they accept biomedical treatments and explanations of their smoking behaviours.

Chapter 2 focused on the literature around biomedicalisation and applied this to cigarette smoking. Critics of medicalisation are concerned about the portrayal of smoking as a chronic disease that requires medical treatment. They believe that this approach will negate free will and neglect alternative non-medical approaches to quitting. The brain disease model of addiction is an example of a strong biomedical approach which has been widely critiqued. I argued in Chapter 2 that these concerns are likely to be overstated as there are a number of factors that limit the extent of the medicalisation of smoking cessation. These include the historical emphasis in tobacco control on population based measures (and their success in reducing smoking prevalence); the clear social inequalities that influence smoking rates in different groups; the ambiguous place of nicotine amongst other drugs; and a resistance among healthcare practitioners to adopting a medical approach to smoking cessation. Moreover, many

of the claims about the negative impacts of biomedical models of addiction have not been empirically investigated. The aim of this thesis was to contribute to the emerging literature on lay understandings of addiction neuroscience by investigating how addicted individuals understand the role of the brain and biology in their addiction. Further, this issue has not been explored in relation to nicotine addiction and smoking, and one aim of this study was to address this neglect.

To inform the methodological approach of this study, the literature on lay understandings of health and illness, and the related field of PUS, were explored in Chapter 3. This chapter outlined the importance and impact of everyday understandings of health and illness. There was a focus on studies that investigated the meanings that patients ascribe to their health issues, their treatment, and their interactions with health care practitioners. Interrogating the existing literature on lay understandings of smoking, it became clear that medical understandings did not dominate. While most smokers acknowledged that they were addicted, the meaning of addiction varied widely, and was not synonymous with nicotine dependence. Across numerous studies, the themes of willpower, personal choice and motivation were present. However, this chapter also outlined recent examples where the role of the brain and biology was brought to the fore in media and health promotion materials that are being presented to smokers. It was therefore important to investigate the impact that these materials may have, without assuming in advance that they will be incorporated into lay understandings in predictable ways. Because there is very little empirical research that has detailed smokers' understandings of nicotine, or their acceptance of neurobiological explanations of nicotine addiction, an exploratory approach was employed that is outlined in the following chapter.

## Chapter 4. Method

As outlined in Chapters 2 and 3, little research exists about the extent to which smokers are prepared to endorse biomedical models of smoking, or the implications that accepting these beliefs has for quitting attitudes and treatment preferences. The research presented in this thesis aims to explore the extent to which biomedical understandings of nicotine addiction have infiltrated lay discourse on smoking. The research questions I aimed to answer were:

- 1) To what extent do smokers and non-smokers accept biomedical understandings of smoking and smoking cessation?
- 2) How do smokers understand the role of the neurobiology in tobacco dependence?
- 3) What proportion of Australian smokers are prepared to endorse the BDMA, and how does this affect their self-efficacy or treatment choices?

A mixed methods research design was used to examine the extent to which Australians endorse a biomedical model of tobacco dependence. This chapter will provide a brief outline of mixed methods research and provide my justification for choosing one particular form of mixed methods research: the exploratory sequential design (Creswell & Plano Clarke, 2011). Because further details about the methods are described within papers embedded in the following chapters, this methods section will provide a more detailed description of the method that was not possible in published papers because of space constraints. There is a particular focus in this chapter on the use of mixed methods, thematic analysis, and the use of online panels for conducting quantitative surveys.

### 4.1. Mixed methods research: Introduction and justification

Mixed methods research involves the use of both qualitative and quantitative methods of data collection to study a research question, as well as the integration of the qualitative and quantitative results. The rationale for using a mixed methods approach is that the combination of qualitative and quantitative methods can to some extent overcome the limitations of each individual method, producing richer and more rigorous understandings of the topic being studied.

“Its central premise is that the use of quantitative and qualitative approaches, in combination, provides a better understanding of research problems than either approach alone.” (Creswell & Plano Clarke, 2011, p. 5)



Because quantitative data typically uses large samples that are systematically selected, we can be reasonably confident that the results from the sample can be generalised at a population level. However, because of the large sample sizes and the use of standardised questions in survey collection instruments, quantitative data often does not capture the detailed voices and stories of individual participants, or illuminate the assumptions and motivations behind their answers. Qualitative research, by contrast, allows the voices of individual participants to be heard, but the findings most often cannot be generalised to a population because of small sample sizes and the contextual embeddedness of narratives. Combining quantitative data with personal narratives allows a research question to be viewed from “multiple standpoints” (Greene, 2007, p. 20).

The combination of various methods to study one research question has often been referred to as “triangulation.” Triangulation has been defined as “the combination of methodologies in the study of the same phenomenon” (Denzin, 1978, p.291). There has been some debate in the mixed methods literature about the use of the word triangulation, as it is seen by some as a catch-all word that has been “overused to the point where it means nothing” (Teddlie & Tashakorri, 2009, p.32). While earlier work on triangulation focused on justifications for the use of multiple methods, more recent work emphasises the practical aspects of *how* to combine different methods in meaningful ways. A common misperception of triangulation is that its purpose is to validate the findings of one method by searching for similar findings using a different method. Patton takes issue with this view, stating instead that finding inconsistencies across methods “ought not be viewed as weakening the credibility of results, but rather as offering opportunities for deeper insight into the relationship between inquiry approach and the phenomenon under study” (Patton, 2002, p.248).

Greene, Caracelli and Graham (1989) have outlined more specific purposes for conducting mixed methods research, with triangulation being just one of them (see Table 4.1). The purposes most relevant to this project are “development” and “complementarity”. Complementarity refers to the enhancement of one set of results by reference to results from another method that overcomes some of the limitations of the other. Development means that one method is used to inform the construction of measures in another method. A key outcome from this project is the development of a survey to assess smokers’ endorsement of biological explanations of smoking. As outlined in Chapters 2 and 3, my review of the literature

demonstrated that very little research existed to inform the development of such a survey. Qualitative research was therefore required to explore how smokers understand addiction to cigarettes, and the language that they use to describe the role of biology and brain in their smoking.

Table 4.1 - The purposes of mixed methods research

<b>Purpose</b>	<b>Rationale</b>
Triangulation	To increase the validity of constructs and inquiry results by counteracting or maximising the heterogeneity of irrelevant sources of variance attributable to method bias.
Complementarity	To increase the interpretability, meaningfulness, and validity of constructs and inquiry results by both capitalizing on inherent method strengths and counteracting inherent biases in other methods and other sources
Development	To increase the validity of constructs and inquiry results by capitalizing on inherent method strengths.
Initiation	To increase the breadth and depth of inquiry results and interpretations by analyzing them from the different perspectives of different methods and paradigms.
Expansion	To increase the scope of inquiry by selecting the methods most appropriate for multiple inquiry components.

Adapted from Greene, Caracelli & Graham (1989)

#### **4.2. Methodological orientation**

Whether acknowledged or not, all researchers have a methodological orientation that includes assumptions about what can be studied, and how it should be studied. Quantitative research typically aligns with a positivist paradigm, where there is belief in an external reality that can be “discovered” through the use of rigorous experimentation. Qualitative research often takes a more constructivist approach, believing that reality is created by individuals, including researchers. Most researchers position themselves somewhere between these two extremes. While some believe that these two research paradigms are incompatible and should not be combined, it is increasingly common to take a pragmatic approach that accommodates both methodologies within a single research project.

Tashakkori and Teddlie define pragmatism as a “deconstructive paradigm that debunks concepts such as ‘truth’ and ‘reality’ and focuses instead on ‘what works’ as the truth regarding the research questions under investigation.” (Tashakkori & Teddlie, 2003, p. 713). The shift towards a pragmatic approach has been particularly marked in the field of public health, due to an emphasis on applied research and the desire for practical outcomes, a desire to apprehend the multi-causality of health and illness, and acknowledgement of the need to incorporate patient perspectives in healthcare (Baum, 1995; Forthofer, 2003). Fran Baum has written that researchers in public health are “faced with a smorgasbord of methods from which they can select those methods likely to produce the most comprehensive and valid answers” (Baum, 1995, p. 463). A pragmatic approach moves towards *understanding* and *improving*, rather than just *describing*, the health needs of communities (Baum, 1995). For a fuller explanation of the pragmatic perspective on questions of epistemology and ontology, interested readers can refer to Morgan (2007). For this project, I have adopted a pragmatic approach in my exploration of smokers’ understandings of the biomedical model of tobacco dependence. My rationale for the study design chosen is provided below.

### **4.3. Overview of study design**

Methodologists have outlined various mixed methods research designs. They vary in the emphasis placed on: 1) whether the researcher prioritises the qualitative or quantitative stage of the project; 2) the order in which the research is conducted (concurrent, sequential or more complex designs); and 3) how the two methods interact (Creswell & Plano Clarke, 2011).

The exploratory sequential design was selected for this research project. This method involves exploratory qualitative research as the first stage. The results from this qualitative research guide the development of a quantitative stage of study, typically informing the construction of a survey. This method is suitable for research questions that are relatively unexplored and where quantitative measures do not currently exist. Creswell and Plano Clarke (2011) outline the conditions under which this method is appropriate:

- There is not an existing quantitative instrument that measures the variables of interest.
- There is little existing research to guide the variables you select to measure.
- There is no clear theory or framework by which to guide the development of a survey.

This was certainly the case for the research questions examined in this thesis, where there was very little quantitative or qualitative research, and the interdisciplinary nature of the topic meant there was no single theory that could be used to guide the development of a survey.

A summary of the study design for this project is shown in Figure 4-1. This research project began with an analysis of pre-existing data from 55 face-to-face interviews conducted with members of the Australian public, including smokers and non-smokers. I analysed interview data relating to their views on the best treatment method for smoking cessation. Study 2 involved 29 face-to-face interviews with Australian smokers about their views on various strategies for quitting smoking, and their beliefs about the role of the brain in their smoking. The last phase of the project (Study 3) was the development of a survey examining treatment behaviours and beliefs around smoking and their relationship to neurobiological beliefs about smoking. This survey was completed online by 1,538 Australians who smoked daily. Each phase of the project will be described in more detail in the following section.

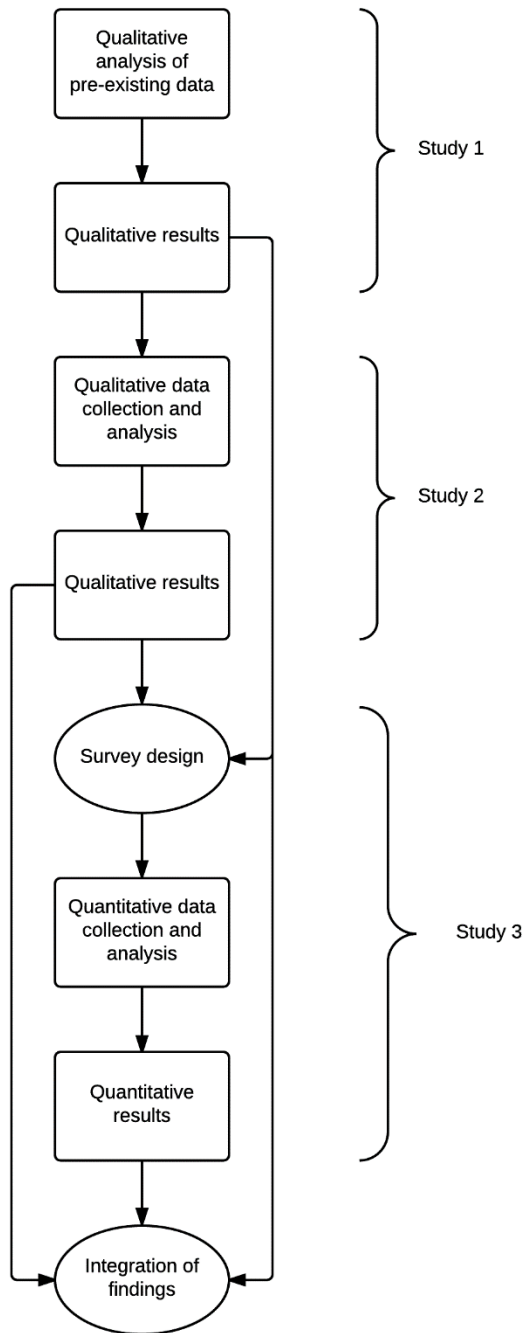


Figure 4-1. Research design

#### 4.4. Study 1: Public attitudes towards smoking cessation treatments

##### *Rationale*

The first stage of the research reported in this thesis involved an analysis of a subset of previously collected qualitative data. This data was collected via 55 semi-structured interviews

with members of the Australian public on their understandings of addiction to heroin, alcohol and nicotine, and their beliefs about the role of the brain in addiction. The primary analysis of this data is reported elsewhere (Meurk, Hall, Morphett, Carter, & Lucke, 2013). Based on debates about the medicalisation of smoking as outlined in Chapter 1, I explored the extent to which members of the Australian public perceive medical treatment as suitable and effective for smoking cessation. The aim was to assess the claim that as smoking cessation becomes more medicalised, there will be a reduced emphasis on quitting unassisted, and pharmacotherapy will come to be seen as the best, if not the only, way to quit.

### *Design and recruitment*

The analysis was conducted on pre-existing data that had been collected in 2011. Face-to-face, semi-structured interviews were conducted with 55 members of the public from the Greater Brisbane area. A market research company was contracted to recruit participants, carry out the interviews, and provide verbatim transcripts of the interviews.

The market research company recruited the participants from their existing nationally representative database of Australian households. Households from the Greater Brisbane area were randomly selected from this database and contacted by phone. One participant from each household was asked to participate. In order to correct for the underrepresentation of young people in such research, preference was given to the youngest male aged over 14, followed by the youngest female aged over 14. In order to obtain a cross-section of Australian smokers, quotas were set in relation to age and gender.

Participants were asked their ideas about addiction to heroin, alcohol and nicotine, as well as their thoughts about the best treatments for these addictions. My analysis looked at participant responses to questions about what participants thought was the best treatment for “nicotine addiction”.

Interviews ranged in length from 20 minutes to one hour and were conducted where convenient for participants. Participants were provided with a gift voucher to reimburse them for their time. All participants provided informed consent and the study received ethics approval from the University of Queensland Human Research Ethics Committee (Approval number: 2009001022). Demographic information about participants can be found in Chapter 5.

### *The secondary analysis of qualitative data*

As described above, the data for this study were drawn from already completed interviews that were part of a larger project about neuroscience and addiction. The aim of the original study was to examine public understandings and knowledge around the role of the brain in addiction. Nicotine was just one of the drugs that participants were asked about, and the questions around treatment for smokers were included to elicit participants' endorsement of a biomedical approach to treating addiction. While not originally intended to be analysed independently from the other data, it became apparent that nicotine addiction was sufficiently different from other drug addictions to warrant a separate, more detailed analysis. In addition, the topic was very relevant to the subject matter of this thesis, and it was not being utilised by other researchers. The researchers involved in data collection provided permission to access and analyse the data.

Advantages of secondary data analysis include fuller utilisation of data that is time and resource intensive to collect, the opportunity for validation of research findings, and allowing researchers more time for dedicated and comprehensive data analysis (Ziebland & Hunt, 2014). While the secondary analysis of quantitative data is common and various data archives facilitate such sharing of data, the secondary analysis of qualitative data is more controversial.

There are a number of reasons for this. One is the epistemological position that qualitative research is a "joint construction" between a researcher and a participant. An "outsider" analysing the data will not have access to the experiences of the researcher who undertook the initial fieldwork, and thus the contextual nature of the interview (or other form of fieldwork) may be neglected. Relatedly, there is also the possibility that focusing on only one component or stage of an interview will decontextualize the data, which has been shaped by surrounding topics and questions.

There are also issues around consent, confidentiality and ownership of data when data is accessed by researchers without the permission or knowledge of the research participant (Parry & Mauthner, 2004). Consent forms which allow for data archiving and the future use of data for other purposes have been deemed problematic, as participants are unable to foresee the purposes to which the information will be put to use. In the case of the research reported here, these ethical concerns were mitigated by the fact that the data was only used by the university

and the research group to which the participants originally consented. Confidentiality was maintained as only de-identified interview transcripts were provided.

While there is the potential for secondary analysis of qualitative data to result in validation of the original findings, there is also the potential for disparate findings to result when researchers hold differing theoretical perspectives and frame their analysis in diverse ways. While this was not a significant issue for this study, as the data analysed here had not been reported elsewhere, it is becoming very topical in qualitative research with the increasing expectation that data will be made publicly available.

Many of these issues around secondary analysis of qualitative data are unresolved. Nonetheless, it has been argued that a “pragmatic approach” is employed that balances the potential for harm against the potential for informed decision making in health (Ziebland & Hunt, 2014).

### *Thematic analysis*

Thematic analysis was used to analyse results from all qualitative data in this thesis. Thematic analysis is widely employed in the health sciences. Like other forms of qualitative analysis, it involves identifying and interpreting patterns in data (Braun & Clarke, 2006). While some describe thematic analysis as a process to be used within a broader method, Braun and Clarke argue that it is a method in its own right, and should be seen as a “a foundational method for qualitative analysis.” (Braun & Clarke, 2006, p. 78).

Braun and Clarke’s seminal 2006 paper outlines six steps for conducting a thematic analysis, which are reproduced in Table 4.2. Although they are presented as linear steps, it must be acknowledged that qualitative research is a recursive process and during the course of the analysis, and I moved backwards and forwards between these steps, as recommended by Braun and Clarke (2006).

The two main products of thematic analysis are codes and themes. Coding in thematic analysis refers to chunking the text into “repeated patterns of meaning” (Braun & Clarke, 2006, p. 86). These codes are then collated into broader themes. While there is no clear definition about what a theme is, or how to decide whether something should be labelled as a theme or not, Braun and Clarke state that the key characteristics of a theme are that it “...captures something



important about the data in relation to the research question, and represents some level of patterned response or meaning within the data set.” (Braun & Clarke, 2006, p. 82). Including a pattern as a theme is not determined by its prevalence within the dataset but rather by a researcher’s judgements about its salience to the research question (Joffe, 2012). For all qualitative analysis, the computer program NVivo 9 or NVivo10 was employed to aid the coding of data and to collate the codes into themes.

Table 4.2 - Braun and Clarke’s steps for thematic analysis

Phase	Description of the process
<i>1. Familiarizing yourself with the data</i>	Transcribing data (if necessary), reading and rereading the data, noting down initial ideas.
<i>2. Generating initial codes</i>	Coding interesting features of the data in a systematic fashion across the entire data set, collating data relevant to each code.
<i>3. Searching for themes</i>	Collating codes into potential theme, gathering all data relevant to each potential theme.
<i>4. Reviewing themes</i>	Checking if the themes work in relation to the coded extracts (Level 1) and the entire data set (Level 2), generating a thematic ‘map’ of the analysis.
<i>5. Defining and naming themes</i>	Ongoing analysis to refine the specifics of each theme, and the overall story the analysis tells, generating clear definitions and names for each theme.
<i>6. Producing the report</i>	The final opportunity for analysis. Selection of vivid, compelling extract examples, final analysis of selected extracts, relating back of the analysis to the research question and literature, producing a scholarly report of the analysis.

Reproduced from Braun and Clarke (2006)

While I conducted the majority of the analysis in all studies presented, another author was always involved to ensure the reliability of the coding and thematic extraction. A subset of transcripts was examined by this second investigator, who independently coded the data and identified themes. Results were systematically compared and any discrepancies or major differences were discussed and negotiated until a consensus about themes was reached (Hansen, 2006). Further details about the process of analysis for each qualitative study that comprised this thesis are provided in Chapters 5, 6 and 7, and 8.

#### **4.5. Study 2: Smokers attitudes towards quitting strategies and neurobiological explanations of smoking**

##### ***Rationale***

The aim of Study 2 was to explore how smokers understand the concept of nicotine addiction, their views about assisted and unassisted quitting, and to assess how they interpreted information about the neurobiology of addiction.

##### ***Design and recruitment***

Study 2 involved semi-structured, face-to-face interviews with 29 current smokers. Eligibility criteria were: currently smoking daily and aged 18 years or older. Purposive sampling (Patton, 1990) was used to recruit a wide variety of smokers in relation to age, gender, heaviness of smoking and socioeconomic status. Recruitment was limited to the greater Brisbane area. A number of recruitment methods were employed in order to obtain a diverse sample, including: handing out and posting flyers on community noticeboards and at neighbourhood centres; advertisement on the online classified site *Gumtree*; UQ staff newsletter; and a university registry of older adults. The demographics of participants are presented in Chapter 6. Participants were given a \$20 Coles Myer voucher in appreciation for their time.

##### ***Ethical considerations***

The Human Research Ethics Committee of the University of Queensland granted ethics approval for this study (Approval number: 2009001022). The main ethical considerations were around informed consent, the maintenance of confidentiality and anonymity, and the potential for distress during the interviews.

In relation to informed consent, participants were provided with verbal information about the project over the phone or via email. Prior to the interview, an information sheet was emailed to all eligible participants who had an email address. The information sheet is included as Appendix B. It contained information about the purpose of the research, what participant would involve, discussion about their right to withdraw at any time, and details about the maintenance of confidentiality and anonymity.

The interviews were audio recorded and sent to an external company for transcription. This company was not provided with the names or details of participants, and were asked to remove names from the transcripts where they had arisen during the interviews. Participant names and details were stored separately from the data, and all records were password protected for electronic files or stored in a locked filing cabinet for paper copies. No identifying information about participants was included in published papers or other reporting on the data.

Only minimal discomfort was expected to be associated with this study (e.g. anxiety due to being interviewed). The study did not aim to collect any personal information that would be expected to cause discomfort. Participants were made aware of the nature of the interview prior to consenting and were informed that they were able to stop the interview at any time, leave the study and ask for their responses to be deleted, if they wished. Also, it was made clear that participants were free to refuse to answer any question they were not comfortable with.

It was deemed unlikely that participants would become distressed during the interview. In the event that they did become distressed, they were to be provided with pamphlets with the number of the Quitline and encouraged to see their general practitioner. If participants requested information on quitting smoking they were provided with a pamphlet for the Quitline.

The consent form contained a space where participants could leave an email or postal address if they wanted to receive a summary of the results of the research.

### ***Semi-structured interviews and study materials***

Semi-structured interviews are the most common method of data collection in qualitative research in the public health field. According to Patton, qualitative interviewing assumes that “the perspective of others is meaningful, knowable, and able to be made explicit” (Patton, 1990,

p. 341). All interviewing techniques lie on a continuum from structured interviews, which are akin to a quantitative approach, to unstructured approaches that allow participants to entirely construct their own narrative about the research topic. As Patton notes, different types of interviewing can be used in different stages of the interview (Patton, 1990, p.347). In semi-structured interviews, the researcher asks the participant interview questions that are typically pre-determined and structured in the same way for all participants. While the questions and the basic structure are predetermined, semi-structured interviews provide flexibility to delve deeper when unforeseen topics arise, to alter the order of questions dependent on how the interview progresses, and to skip questions that have already been answered. Semi-structured interviews have been described as “guided conversations” that provide a space for the co-creation of meaning (DiCocco-Bloom & Crabtree, 2006).

Semi-structured interviews were deemed the best way to collect data for this project as they allowed an initial exploratory stage of the interview, where participants were asked their general thoughts about smoking and addiction, before moving to more direct questions designed to explore specific issues that have been raised in the literature. This strategy allows participants’ understandings of addiction to smoking to emerge, before “scientific” understandings of addiction were presented, as these had the potential to bias results. Towards the end of the interview participants were shown a short PowerPoint presentation outlining current knowledge about the effects of nicotine on the brain and its role in addiction. Participants were asked to give their thoughts about whether they were previously aware of this information and whether it influenced their understanding of their smoking or their ideas about treatments for smoking.

Interviews took place primarily in coffee shops at locations convenient to the participant, although a small number occurred in university meetings rooms. The coffee shop as an interview site was particularly conducive to the conversational nature of the interview. Coffee shops are typically viewed as “casual” places where people gather to talk with friends. The initial meeting and ordering of drinks also allowed the building of rapport prior to the beginning of the interview.

The PowerPoint presentation (see Appendix A) was based on resources developed by the National Institute of Drug Abuse (NIDA) in the USA, one of the most influential disseminators of information about drugs and the brain. As the study aimed to recruit a wide variety of

smokers with varying demographic and educational backgrounds, it was important that the information presented to participants was understandable to those who do not have extensive education or scientific knowledge. NIDA's online resource "Mind Over Matter: Tobacco Addiction" brochure designed for high school students, and the associated resource for teachers (National Institute on Drug Abuse, 2006) were used as the basis for the presentation. The presentation was piloted with people without a scientific background, drawn from the researchers' social networks, and refined to maximise clarity and comprehensibility. Audio narration was added to cater for participants with poor reading comprehension.

The interview schedule (see Appendix C) was developed from themes identified in the literature review, as well as those identified from prior studies (Meurk, Hall, et al., 2013; Meurk et al., 2014). It was piloted with five volunteers and minor refinements were made to question wording.

The main topics covered in the interview schedule were:

- Smoking history and thoughts about their own smoking
- Understandings of addiction, including issues of control and responsibility
- Questions about the treatment and prevention of smoking, including public health measures and individual treatment options
- Participant responses to the aforementioned presentation on the neurobiology of nicotine addiction
- Attitudes toward the idea that nicotine addiction is a "brain disease"

In addition, a short demographic and smoking history survey was completed by participants prior to completing the interview (See Appendix C).

### ***Data analysis***

All interviews were audio recorded and transcribed verbatim. Data was imported into NVivo 9 or 10 and analysed using thematic analysis, as described in Section 4.4.

#### **4.6. Study 3 – Survey of Australian smokers’ endorsement of brain-based explanations of smoking and relationship to treatment intentions and self-efficacy**

A survey was undertaken with a sample of Australian smokers in order to assess whether the results of the qualitative studies generalised to a larger population, and to investigate the relationship between biomedical understandings of smoking, demographic variables and smoking-related behaviours and preferences

##### ***Eligibility criteria***

Current smokers who were aged 18 or over, smoked daily, and had smoked at least 100 cigarettes in their lifetime were recruited to the study. Participants must also have been Australian citizens or residents to ensure data were reflective of the Australian population.

##### ***Survey development and design***

Survey items were developed from a review of the literature and from themes identified in the qualitative phase of the research project. Survey development was the key phase of the study where integration of the qualitative and quantitative components occurred. The key themes and observations from the qualitative studies that informed the development of the survey are described in Chapter 9. The relevant items from each section of the survey are described below. The full survey is included as Appendix D.

##### ***Ethical considerations***

The Human Research Ethics Committee of the University of Queensland granted ethics approval for this study (Approval number: 2009001022). No personal information of a sensitive nature was collected as part of this study and the major ethical issues raised in doing this survey were: 1) ensuring that participants provided free and informed consent to participate in this study; and 2) ensuring the confidentiality of any information provided by participants.

In accordance with standard practice for online questionnaires, participants were presented with a screen that contained information about what the study involved prior to commencing. This included information that:

- Participants were free to withdraw from the study at any time by exiting the survey webpage.

- The University of Queensland would not receive any personal information about participants, including name, address or email.
- Once the survey was completed, the University would be unable to delete responses, as we could not link the respondent's name with the survey data.
- That participation will involve completing one 20 minute questionnaire about smoking, and thoughts about quitting methods and addiction.

Respondents needed to agree to proceed to the survey.

In relation to confidentiality and anonymity, the market research panel was bound by the National Privacy Principles (on Market and Social Research Privacy Principles), approved by the Federal Privacy Commissioner. Further detail on the privacy policy is included as Appendix E. Only de-identified data was provided by the market research company.

#### Eligibility criteria and basic demographics

This included age, gender, number of cigarettes per day, and citizenship status. This data was collected in order to determine eligibility.

#### Section 1 – Smoking variables

This section asked standard questions about smoking history and behaviour. The two items of the HSI were included to assess nicotine dependence (Heatherton et al., 1989). Quitting history was assessed by number of previous quit attempts in the last year, as well as longest quit period. Other questions included plans to quit (Australian Institute of Health and Welfare, 2011) desire to quit, and enjoyment of smoking (West & Fidler, 2011). Self-efficacy was measured using a 5 point item that has been used in other national surveys (Bonevski et al., 2015; Hyland et al., 2006). A number of novel items measuring beliefs about the brain and smoking were also asked in this section, including endorsement of the idea that nicotine addiction is a brain disease. These items were drawn primarily from common statements made in the interviews, and are described in more detail in Chapter 9.

#### Section 2 – Attitudes towards quitting methods

A list of common methods of quitting were adapted from the National Drug Strategy Household Survey (Australian Institute of Health and Welfare, 2011). Questions measured the perceived effectiveness of these methods, prior use of the method, and intention to use each.

Participants were given the opportunity to name other quitting strategies they had used but which were not listed.

### Section 3 – Reasons for smoking

An existing scale was adapted to assess participants reported reasons for smoking (Fidler & West, 2009). Further questions were then included about the BDMA. A statement from a recent book by Robert West, a leading smoking researcher, about the role of the brain in smoking was presented (West, 2013). This quote emphasised the neurobiological basis of tobacco dependence. Items measured the extent to which this participants thought this explanation explained the smoking of themselves, and of other smokers. A set of items were developed to assess beliefs about the consequences of accepting a neurobiological understanding of smoking in relation to stigma, self-efficacy and treatment seeking. Lastly, participants were again asked if nicotine addiction was a brain disease, to assess whether providing more detail about the claim, in the form of the Robert West excerpt, changed the participant’s level of endorsement. A number of additional items explored opinions about the use of pharmaceutical nicotine as a harm reduction strategy. These items will not be explored in this thesis.

For most questions, each item was presented on a separate page in order to reduce response bias and satisficing. Pilot testing was conducted to refine the presentation of the questions.

### ***Sampling and recruitment***

The sample for the quantitative component of the project was drawn from an opt-in online panel of Australians. The use of online panels for social research has become common in the social and health sciences, but remains controversial. This section will outline the advantages and disadvantages of using online research panels and the steps that were taken in this research project to ensure the highest quality data for the survey.

### ***Benefits and drawbacks of online research panels***

Online panels have been defined as “a pool of registered persons who have agreed to take part in online studies on a regular basis.” (Goritz, Reinhold, & Batinic, 2002, p. 27). The primary advantage of using an online panel is time and cost efficiency. A large “ready-made” sample of participants is available, with the bulk of the recruitment process undertaken by the market research company. Whilst this saves the researcher time, it also means ceding control of



recruitment to an external body. Most online panels do not randomly select potential panel members. Rather, panel members self-select to join the panel, usually after exposure to advertising. Thus, an online panel cannot be said to be representative of the general population, but is rather a form of “convenience” sampling.

Because of this, there are certain biases that may result from using online panels. The most obvious is that only those with internet access can join an online research panel, leading to coverage error. In Australia, internet penetrance is high, with 83% of Australian households having internet at home (Australian Bureau of Statistics, 2014). However, internet access does vary according to certain sociodemographic variables. For example, households with children, and higher income households, are more likely to have an internet connection at home (Australian Bureau of Statistics, 2014). In relation to personal internet use, younger people are more likely to use the internet than older adults (97% of 15-17 year olds compared to 46% of people aged 65 or over (Australian Bureau of Statistics, 2014). In the USA, those who join online panels have higher education levels, more frequent internet use, and are less likely to belong to an ethnic minority (American Association for Public Opinion Research, 2010).

Whilst these sampling issues cannot be overcome entirely, the use of online panels can still provide meaningful data, and their use is becoming common in addiction research (Buykx, Gilligan, Ward, Kippen, & Chapman, 2015; Hughes, 2009; Quaak, Smerecnik, van Schooten, de Vries, & van Schayck, 2012; Vogt, Hall, & Marteau, 2010; Willems et al., 2013). Firstly, the issues raised above are not unique to studies that employ market research companies. There are also problems with alternative means of obtaining samples for social research. Response rates in surveys using population-based sampling are typically low, and hence the recruited sample may no longer be representative of the whole population (Fogliani, 1999). Random digit dialling has been the traditional way to obtain a probability sample. However, the use of landlines is declining rapidly in many countries because of increased use of mobile phones. The Australia Institute reported that samples from online surveys were very similar in representativeness to samples produced by a random telephone sample (Bambrick, Fear, & Denniss, 2009). The Australian Communications Media Authority reported that 25% of Australians no longer have a landline, and that those without a landline are younger (Australian Communications and Media Authority, 2014). In addition, there are benefits to this type of online research which have led to rapid growth in its use (American Association for Public Opinion Research, 2010). These include reduced response rates for random digit dialling,

flexibility of web-based questionnaire design, and quick turnaround times for online data compared to traditional probability samples. Also, the expense of conducting random mail-out surveys or random digit dialling makes it unfeasible to use these methods for many smaller projects with limited budgets. The American Association for Public Opinion Research recognise that nonprobability online surveys are suitable for certain types of research:

“There are times when a nonprobability online panel is an appropriate choice. Not all research is intended to produce precise estimates of population values and so there may be survey purposes and topics where the generally lower cost and unique properties of Web data collection is an acceptable alternative to traditional probability-based methods.” (American Association for Public Opinion Research, 2010, p. 5).

While the limitations of online panels need to be recognised and acknowledged, there are many steps that can be taken in order to ensure that the best quality data is collected. One recommendation from a report of the American Association for Public Opinion Research on online panels was that potential panels are researched and assessed rigorously (American Association for Public Opinion Research, 2010).

### *Selection of online panels*

There are many commercial operators that maintain and advertise online panels for social research. These vary in terms of the size of their panels, their recruitment methods, and the panel makeup. Reputable companies can provide potential clients with an Esomar 28 document, which answers 28 questions relating to the quality of their panel. The Esomar 28 document for the panel that was selected for this project, The Online Research Unit (ORU), is available upon request. Taverner Research was the company that liaised and administered the survey via the ORU. The key factors that influenced the selection of the online panel are described below.

#### Size of panel

It is important to ensure that the company selected can provide the required number of participants. Many companies use “panel partners”, where they make commercial arrangements to supplement their own panel members with that of another panel in order to obtain the desired sample. This practice can be problematic, as an individual may be a member of both panels, raising the potential for duplication. Whilst there are generally processes in place for identifying duplicates, it is better practice to source all participants from a single

panel. For this project, companies that could not provide all participants from a single panel were excluded from consideration.

### Recruitment

While it is self-evident that all participants in an online panel must have internet access, it is preferable for recruitment purposes for the panel to be advertised via both online and offline channels in order to increase exposure by harder to reach groups. The ORU uses a combination of online and offline advertising measures, with 53% of their advertising using offline methods such as telephone, mail, and print advertising.

In relation to joining procedures, most online panels now use a double opt-in process, whereby participants initially express an interest in joining the panel by clicking on a link, responding to an email, or calling the membership department of the company. They are then sent an email asking them to confirm that they wish to join the panel. At this point, they are usually directed to a profile page, where they can answer questions about themselves that allow the panel to direct surveys to certain types of people. The use of a double opt-in process was a requirement for this study. The selected panel also conducts periodic “panel maintenance” where inactive members, duplicate members, or members identified as fraudulent, are removed.

### Quality standards for online research

There are two standards that online opt-in panels can adopt. These have been developed by the International Organization for Standardization and are labelled ISO20252 and ISO26363. Many Australian panels conform to requirements for ISO20252. The panel selected for this survey meets standards for ISO20252 and ISO20363. ISO20363 is a more recent standard (2009) with higher quality standards (e.g., the use of a double opt-in recruitment process). In addition, the ORU is endorsed by the Association of Market and Social Research Organisations, which place stringent privacy and ethics requirements on members.

### Incentives

Online research panels typically provide participants with incentives for completion of surveys. The incentives are often in the form of “panel points” which can be converted to cash or gift cards. For this research, participants were provided with panel points.

### Sampling strategies

For this research, we required a sample of smokers that was similar to that of the population of smokers in Australia. In order to meet this requirement, a target population structure was set based on age and gender data about smokers obtained from the National Drug Strategy Household Survey (Australian Institute of Health and Welfare, 2014). The distribution of participants was checked daily, and invites and reminders managed in order to keep the sample screened as close as possible to the population targets. The only group that was slightly under-represented was younger males, which are recognised as being a difficult group to recruit. Demographic details about survey participants are provided in Chapter 9.

### **4.7. Summary**

This chapter has outlined the rationale and design of the research project. Mixed methods were chosen as the most appropriate means to assess the extent to which a biomedical model of tobacco dependence has infiltrated lay conceptualisations of smoking and quitting among a sample of Australians. Two qualitative studies employed semi-structured interviews to explore attitudes about the use of medications for smoking cessation. This qualitative research also investigated the ways that lay discourse incorporated the role of the brain into explanations of smoking. Based on findings from these qualitative studies, a quantitative measure was designed in order to investigate the prevalence of neurobiological beliefs about smoking amongst a large sample of Australian smokers, and their impact on treatment intentions and other relevant smoking variables. The following chapter will expand on the rationale for interviewing a random sample of the Australian public, and present the results of these interviews.

## **Chapter 5. Public attitudes toward the treatment of nicotine addiction**

As discussed in Chapter 1, the key components of medicalisation are: 1) the labelling of individuals as having a medical problem with subsequent surveillance by health practitioners; and 2) the provision of medical treatment for a condition.

Chapter 1 outlined the current debate in the tobacco control field about the value of biomedical definitions and treatments for smoking cessation. Some believe that an emphasis on medical treatment is misguided, as it reduces the emphasis on unassisted quitting. They are concerned that if a biomedical approach to cessation comes to dominate, people will come to see unassisted quitting as an ineffective and inappropriate means of quitting smoking (Chapman & MacKenzie, 2010). Meanwhile, smoking cessation pharmacotherapies have only modest effectiveness, and their promotion, particularly by pharmaceutical companies, may provide the public with unrealistic expectations regarding their efficacy (Wakefield & Durrant, 2006). The alternative interpretation is that smokers should be encouraged to quit with pharmacotherapy and counselling, as current scientific evidence suggests that this is the most effective cessation strategy (Hartmann-Boyce et al., 2013). Proponents of a biomedical approach argue that even small increases in the chance of a successful quit attempt are worthwhile, and that the provision of medical treatment for cessation is a cost-effective intervention at the population level (Britton, 2009).

The aim of the study described in this chapter was to explore the extent of medicalisation of smoking cessation in Australia by examining how the public believes nicotine addiction should be treated. Have medical definitions and treatments for tobacco dependence filtered into lay explanations of smoking cessation? Does the Australian public see medical treatment as the best way to quit smoking? It is often assumed that a medical view of smoking will be passively accepted by the Australian public and smokers themselves, however this has not been empirically investigated. The evidence outlined in Chapter 2 suggests that alternative discourses of smoking are common in lay discourse.

This chapter presents an analysis of qualitative data drawn from a pre-existing dataset on public understandings of addiction. It aims to explore the views of the general public about what is

the best method to use to quit smoking and why. The remainder of this chapter is primarily comprised of a paper that was published in *Nicotine and Tobacco Control* in 2013, followed by a summary of its implications for this thesis.

Citation: **Morphett, K.**, Lucke, J., Gartner, C. E., Carter, A., Meurk, C., & Hall, W. (2013). Public attitudes toward the treatment of nicotine addiction. *Nicotine and Tobacco Research*, 15(9), 1617-1622. Published by permission of Oxford University Press.

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## **Abstract**

**Introduction:** The increasing use of medications for smoking cessation has concerned some commentators, who believe that emphasising medications for smoking cessation may lead to a belief that there are “magic bullets” for nicotine dependence, or alternatively that quitting smoking is very difficult, thereby discouraging unassisted quit attempts. There is little evidence on which to test these speculations. This article aims to address this gap by examining public understandings of nicotine addiction in order to assess the extent to which medical explanations of smoking have permeated public beliefs about treatments for smoking cessation. **Methods:** Interviews were conducted with a representative sample of 55 members of the Australian public that included smokers, ex-smokers and non-smokers. The data were analyzed using a standard content analytic method to identify recurrent themes. **Results:** The results revealed that although pharmacological cessation aids were the most commonly mentioned method for quitting, they were often recommended alongside methods such as behavioural strategies or counselling. Unassisted quitting was mentioned frequently, but there were mixed views on its effectiveness. Seeing a doctor was rarely recommended. Two common themes were that smokers had to “really want to quit”, and that the best treatment method would depend on the individual. **Conclusions:** Medical discourse of smoking cessation does not dominate public understandings of smoking cessation. Rather, ideas about individual choice, motivation and willpower are emphasised.

### **5.1. Introduction**

Smoking is increasingly described and treated as a medical issue. One indicator of this is the growing number of smokers who use pharmacological treatments such as nicotine replacement

therapy (NRT), varenicline and bupropion for smoking cessation. In 2003, 32% of Australians who had made a quit attempt in the previous year reported using a pharmacological aid. This proportion rose to 52% in 2009 (Cooper et al., 2011). The use of medications for smoking cessation has also risen in the USA and the UK and is associated with increased over-the-counter availability of NRT, the marketing of pharmacological cessation aids by pharmaceutical companies and government subsidies for cessation medications (West, DiMarino, Gitchell, & McNeill, 2005; Zhu et al., 2012). Associated with the growing use of medical cessation aids is the increasing role of health professionals in the identification and treatment of smokers (Fiore et al., 2008; Zwar et al., 2014).

Despite evidence for the effectiveness of pharmacotherapy for smoking cessation (Cahill et al., 2012; Eisenberg et al., 2008; Hughes et al., 2010; Stead et al., 2008), some public health researchers have criticized the prominence given to pharmaceutical aids over unassisted cessation (Chapman & MacKenzie, 2010). These critics argue that although such medications increase the success rate of quit attempts in clinical trials, overall success rates are lower, with the majority of smokers who use NRT failing in their quit attempt (Chapman & MacKenzie, 2010). Moreover, evidence on the effectiveness of pharmaceutical treatments for smoking are often based on clinical trials sponsored by pharmaceutical companies (Chapman & MacKenzie, 2010; Etter et al., 2007). Studies on the use of NRT in community samples have shown more mixed results. One population-based study of smokers in the US, Canada, UK and Australia found that smokers who had used pharmacological cessation aids were more likely to remain abstinent than those who did not use medications (Kasza et al., 2013). Other studies however, have failed to show such benefits (Alpert et al., 2013; Walsh, 2008), raising questions about the generalizability of findings from clinical trials to “real world” settings.

Chapman and MacKenzie (2010) contend that the emphasis on cessation aids such as NRT has overshadowed the fact that most smokers quit unaided. They argue that the pharmaceutical industry has a vested interest in promoting the view that quitting is difficult without medical help, thereby making smokers think that quitting is harder than it is. These concerns are shared by researchers in the USA (Pierce, Cummins, White, Humphrey, & Messer, 2012). An opposite concern has been expressed about the unintended consequences of increased availability and promotion of medicines for smoking cessation, namely that it may lead smokers to believe that there are ‘magic bullets’ for nicotine addiction that will enable them to quit easily should they choose to do so (Dingel et al., 2011). This may be of particular concern for young people, with

one study suggesting that young people at risk of becoming smokers thought that quitting smoking would be easier after viewing ads for NRT and bupropion compared with ads promoting the Quitline (Wakefield & Durrant, 2006).

A number of commentators have framed these issues as reflecting the risks of increased medicalisation of smoking (Blum, 1984; Caron et al., 2005; Chapman & MacKenzie, 2010; Dingel et al., 2011). Medicalisation refers to the process by which issues not previously under the purview of medicine come to be conceptualised in terms of illness and health. Concerns about medicalisation have typically centred on issues of power, such as the power of medical professionals to diagnose an individual as “healthy” or “ill” thereby creating deviant identities (Conrad, 1992). More recently concern has centred on the power of “technoscientific” industries, particularly pharmaceutical corporations and biotechnology companies (Clarke et al., 2003), to redefine behaviour as illness in order to increase profits.

Finally, these critics argue that medicalising smoking may overemphasise the treatment of smokers and overlook the well-documented social influences on smoking uptake and maintenance. For example, developments in pharmacogenetics, which involve tailoring treatments for smoking cessation based on the results of individual genetic tests, may reduce support and funding for population wide tobacco control strategies that have proven effective in reducing smoking prevalence (Caron et al., 2005; Gartner et al., 2012).

These claims about the potential adverse impact of medicalisation on individual beliefs and smoking behaviour are not typically based on empirical research. There is little research examining the extent to which medical views of smoking have penetrated the understandings of the public in general and smokers in particular. Survey data suggest that there is considerable public ambivalence about the idea that smoking is a medical problem. For example, both the general public and smokers tend not to support the view that smoking is caused by biological factors, and are more likely to endorse the idea that smoking is a habit (Cunningham et al., 1994; Hughes, 2009). Qualitative and quantitative research has demonstrated some resistance to treating smoking as a medical issue, with many smokers emphasising the importance of willpower and expressing a belief that using medication to quit smoking is sign of weakness (Balmford & Borland, 2008; S. Carter, Borland, & Chapman, 2001).



Public health researchers have acknowledged the importance of understanding how lay concepts of health, illness, and treatment may affect health behaviour (Kuppin & Carpiano, 2006; Popay & Williams, 1996). In light of the increasing use and promotion of medications for smoking cessation, there is a need for research exploring lay attitudes toward smoking cessation and the role that drug treatments play in it. This article outlines findings from qualitative research undertaken with a cross section of the Australian population that included smokers, ex-smokers and non-smokers and explored participants' views about treatments for nicotine addiction. Although the views of non-smokers are rarely solicited in smoking research, they are important to explore because research shows that the attitudes of non-smoking family and friends towards smoking can influence the motivation of smokers to quit (Australian Institute of Health and Welfare, 2011; Fiore et al., 2008; Patten et al., 2011). This study aims to explore the opinions that the public hold on the role of treatments in smoking cessation, examine the extent to which medical explanations of smoking have affected the understandings of the public, and describe alternative conceptualisations of smoking that may influence attitudes in relation to treatment options for nicotine addiction.

## **5.2. Method**

The data analysed in this article were drawn from a study exploring public understandings of addiction. Findings about the ways in which the public describe the causes of addiction and their ideas about the brain disease model of addiction have been published elsewhere (Meurk, Carter, et al., 2013). The study involved face-to-face semi-structured interviews with 55 participants, exploring their ideas about addiction to heroin, alcohol, and nicotine. A market research company was contracted to recruit the participants and conduct the interviews using an interview schedule designed by the researchers.

Potential participants were selected from a representative database of Australian households developed by Roy Morgan Research. Households from the database located in the Greater Brisbane area were randomly selected. Once households were selected, the youngest male aged more than 14 years (or if not available, the youngest female aged more than 14 years) was asked to participate. This approach increased the representativeness of the sample of young people, particularly young males who are the hardest to recruit. Quota sampling was used to ensure that the sample was age and gender representative (Table 5.1). Semi-structured qualitative interviews, followed by a brief quantitative survey, were conducted with willing participants at a location of their choice. All participants received a gift voucher in appreciation

for their time. The findings in this article were drawn from participants' responses to the question "What do you think is the best way to treat someone who is addicted to nicotine, and why?" Of the 55 participants, 14 reported being current smokers (10 were daily smokers, 2 weekly smokers, and 2 less than weekly smokers) and 40 were non-smokers at the time of the interview. One participant did not answer this question. Of the 40 non-smokers, 11 reported being ex-smokers.

Table 5.1 - Sample characteristics for Study 1

Gender	Male		Female	
		47% (n=26)		53% (n=29)
Age	Under 50 years of age		50 years and older	
	51% (n=28)		49% (n=27)	
Highest level of education	No formal qualification	Secondary school	Post-secondary qualifications (e.g. trade training)	University/college degree
	4% (n=2)	40% (n=22)	30% (n=17)	26% (n=14)

### 5.3. Data analysis

Interviews were transcribed and imported into qualitative research software NVivo 9 (QSR International Pty Ltd., 2010). Each treatment method mentioned by a participant was coded as a category. In addition, thematic data analysis was used in order to identify recurrent themes across treatment methods. Categories were verified by an iterative process that involved reading each interview in light of the categories developed, and amending or adding categories as required. Each participant quoted is referred to by their age, sex, and whether they are a current smoker, an ex-smoker or have never smoked.

### 5.4. Results

The most commonly mentioned treatment for smoking cessation was the use of pharmacological cessation aids ( $n=30$ ). Although not strictly a "treatment", unassisted quitting was the next most frequently described strategy for quitting ( $n=24$ ). The use of behavioural strategies ( $n=10$ ) and counselling ( $n=7$ ) were also discussed relatively frequently, although not as often as pharmacological cessation aids or unassisted quitting. An important observation was that participants often recommended a number of treatment strategies, as outlined below.

### *Pharmacological cessation aids*

When asked what they thought was the best treatment for nicotine addiction, participants most often mentioned pharmacological cessation aids, mainly in the form of NRT patches. This, no doubt, reflects the fact that NRT is the most widely used pharmacological cessation aid in Australia (Cooper et al., 2011) and the only pharmacological cessation aid that can be advertised directly to consumers. It was less common for participants to mention prescription medications and those that did often referred to “tablets.” Very few participants mentioned a specific prescription medication. This suggests that most Australians have only a vague knowledge of cessation medications, which is not surprising given that they are not allowed to be advertised directly to consumers in this country.

Although a few participants mentioned pharmacological methods as a sole treatment for nicotine addiction, it was more common for pharmacological cessation aids to be discussed alongside behavioural strategies, counselling, and hypnotherapy. The following quote is typical:

*Well the nicotine patches are quite good. There’s a couple of medications that are used that are quite good. Having a plan; having a friend doing it with you helps. And often you can make a plan of when you’re going to stop, whether you’re going to use patches. (male, 50-59, never smoked)*

The belief that medications were useful did not necessarily displace the importance of alternative methods, such as counselling:

*Well obviously these alternatives - nicotine patches and all that are successful to a great degree. But I think there’s a need for counselling in that regard too. Why are you smoking? Let’s get to the bottom of it, et cetera. (male, 70 or over, never smoked)*

Patches were seen as effective, but insufficient because they did not address the underlying reasons why someone smoked. Another common theme was that different methods would work for different people. Again, pharmacological cessation aids were situated as one method among others:

*I think that depends on the person a lot of times. With the things that I've seen, sometimes hypnosis works, you know, sometimes those tablets that they take work. That book by that guy Carr, or whatever, that I gave my brother because he couldn't give up, that worked for him. Yeah, I think it depends on the person. (female, 40-49, never smoked)*

Both smoking and non-smoking participants commented that “really” wanting to quit was a prerequisite for successful quitting, regardless of whether pharmacological cessation aids were used or not:

*I think there are some medicines, some type of tablets or something, people have – somebody taking that tablets you don't feel is – you want to smoke or something, but the rest is on the people. Like me, if I want to quit I should quit, should say to myself that's okay, it's enough, I want to quit. So that's up to the people, if they want really want to quit, they would quit. (male, 25-29, current smoker)*

This approach portrayed individual decision making, motivation, and willpower as the key factors in successful quitting, not medical assistance. For example, one smoker, after an unsuccessful attempt using patches, developed a side effect from prescription medication. However, she blamed her lack of success on a deficiency in willpower, rather than any limitations of the medication:

*Yeah, no that didn't help – and I had some pills I got from the doctor and they just - I just came out in boils everywhere – they were no good. It's just willpower, I got no willpower. (female, 50-59, current smoker)*

Another smoker who had been given prescription medication by their doctor was reluctant to use it for fear of side effects. She expressed a preference for quitting unaided:

*Yes well there's quick – there's a lot of different things you can do. And as I said I've got tablets but I'm reluctant to take them. But hopefully I'll be able to do what I did before and just stop. But I can't answer that. I hope I do for my own sake. (female, 60-69, current smoker)*

This ambivalence toward cessation aids was not expressed by all smokers. One long-time smoker expressed a fear of quitting, but seemed to gain encouragement from the availability of cessation aids:

*Well for me I'm sort of – I want to give it up but I'm scared to because I've smoked all my life. So it's also a fear factor there – I know I couldn't give it up cold turkey but with those Nicorette's and all this sort of stuff- no I'm going to give it up. (male, 49-49, current smoker)*

### ***Unassisted quitting***

In asking participants about the best “treatment” for nicotine addiction, there was the potential to bias responses toward medical or psychological interventions. Nonetheless, participants still referred to unassisted quitting, which was often positioned as the ideal method for smoking cessation. The use of cessation aids was sometimes described as a secondary option, to be used only where unassisted quitting had been unsuccessful:

*I mean maybe you try what my husband did, the cold turkey, and if that doesn't work you try the patches or whatever. (female, 60-69, never smoked)*

*After you've made that decision, be consistent, follow through with it, persist. And – well of course some people might need medical additional help, patches but personally I never had that problem so I can't empathise with – put myself in their place but I believe it's best to just drop it instead of just slowly trying to. (male, 60-69, never smoked)*

Participants often talked about how difficult or easy it would be to quit unassisted. Respondents were divided on this point. Some believed that quitting unassisted would be very difficult and that only strong-willed people would be able to stop smoking this way:

*My parents just stopped like that and I always find that incredibly impressive when people do that. So I can appreciate it can be done but at the same time I do appreciate that it is very hard and some people will try over and over again before they finally make it. (female, 40-49, never smoked)*

This fitted with the idea that different methods work for different people. Quitting cold turkey was often described as a method suitable only for some people, typically those with willpower or “strong minds”:

*Yeah, there's a lot of help out there and I have seen people with strong minds just stop, even though it's hard. But there's other people that need more help. (female, 40-49, never smoked)*

Others expressed the belief that unassisted quitting would be the easiest way to quit. These views were often expressed by those who saw smoking as a habit that smokers can and should “just stop”:

*I just think nicotine is probably a mental thing, not so much as your body probably needs it. I just think it's more like a habit. You just walk downstairs; have a coffee and a cigarette. Whereas walk downstairs, have a coffee and have a chocolate bar instead or something and then do that for five days in a row and you'll probably be cured from wanting to smoke anymore. (female, 25-29, never smoked)*

The above quote illustrates the way in which an individual's explanatory model of nicotine addiction influenced a participant's views about the most appropriate way to quit. Understanding smoking as a habit, rather than a physiological dependency, led the participant to see quitting as a matter of replacing one habit with another. A small number of those who saw smoking as a habit rejected the idea that smokers needed any treatment for nicotine addiction because they did not believe that nicotine was as addictive as claimed:

*I actually don't think they're really addicted to tell you the truth. I don't think nicotine is as an addictive drug as everybody makes out. (male, 50-59, ex-smoker)*

The concept of motivation and the importance of personal decision making were again emphasized in discussions around unassisted quitting:

*He has to help himself. He wants to do it. Throw it away, don't just go through the stages, just throw it away, make a decision, publicise it, tell everybody that counts. (male, 60-69, never smoked)*

*Best way to treat them is you can't treat them. It's up to them whether they want to give up or not. You're not going to force anyone to do anything that they don't want to. (male, 40-49, ex-smoker)*

### ***Behavioural strategies***

The use of behavioural strategies was also mentioned often as a treatment option for nicotine addiction. The strategies described under this heading included reducing the intake of cigarettes, replacing smoking with another behaviour, recruiting social support for abstinence,

and avoiding environments where smokers were exposed to tobacco smoke or other smoking-related cues:

*You just need to take them out of the environment, so that they - well, take them out of the environment and even into a neutral environment where there's no need for temptation and something that won't - teach them not to be drawn to it kind of thing. (female, 18-24, never smoked)*

*I don't know, hey. I reckon to – because my dad was addicted. The way- how he got off smoking was to keep himself busy and not just sit around. So he just keeps himself busy every day, every day. And chew a bubble-gum. [Laughs]. Chew a bubble-gum, 'cause that will keep your mind off smoking. (female, 18-24, current smoker)*

### **Counselling**

Counselling was one of the most commonly discussed methods for treating nicotine addiction. Generally, a less intensive form of counselling was thought to be required for smokers than for those addicted to alcohol or heroin.

*Well nicotine is not really – I mean, someone who takes nicotine's not completely – just – they can be a functioning member of society. So you don't need a rehab, but possibly a counselling or somebody to help like a motivational coach, or something like that.” (male, 18-24, current smoker)*

### **Seeing a doctor**

Seeing a doctor was rarely mentioned by participants, with only one participant describing doctors as having a supervisory role in relation to smoking. Several participants described the cost of seeing a doctor and the cost of medications for smoking cessation as a barrier to seeking medical assistance for smoking.

## **5.5. Discussion**

These results suggest that although the medicalisation of smoking cessation has occurred to a degree, the process is far from complete. Pharmacological cessation aids were the most frequently mentioned treatment for smoking cessation, but unassisted quitting was often described as the ideal method. Participants often recommended that medications only be used when unassisted quitting had been unsuccessful. Even then they often articulated that behavioural methods that dealt with the social and emotional aspects of smoking, for example

counselling, were also desirable to reduce relapse. The finding that the public ascribe prominence to unassisted quitting is strengthened by the fact that the wording of the question, in which respondents were asked what they thought was the best “treatment” for nicotine addiction might have biased their answers towards medical options.

There was also little evidence that medications for smoking cessation were perceived as “magic bullets”. Doctors were rarely mentioned in relation to smoking cessation, perhaps reflecting the market dominance of NRT, which is available over the counter and so does not require a doctor’s visit. It may also partially reflect the commonly held belief that smoking is a habit, or at the least a much less serious addiction than other drug addictions, and hence that smokers are not in need of medical assistance.

It is clear that the Australian public has not uncritically accepted the idea that smoking requires medical treatment. Early theorising about medicalisation described it as a one-way process, with the public portrayed as passive instruments of institutional power (Ballard & Elston, 2005). However, more recent research has emphasised the active ways in which people respond to this process, including the ways in which the public resists medical discourse (Ballard & Elston, 2005). The results of this study are more supportive of the latter view in revealing some resistance to the medicalisation of smoking. Only a small number of participants believed that nicotine was not as addictive as commonly thought and that smokers should “just stop.” But the idea that smoking is a habit remained a common theme in public discourse about smoking that has not been purged from the lexicon, as suggested in the first issue of the journal *Tobacco Control* (Davis, 1992).

The tobacco industry has promoted the idea that “habit” and “addiction” are mutually exclusive terms (Davis, 1992) but smokers appear willing to accept both of these explanations of smoking (Hughes, 2009). The results suggest that ideas about the biological bases of smoking have not displaced the idea that smoking is a habit. This may explain why participants in this study often recommended cessation medications *and* behavioural strategies or counselling.

The belief that smokers have to “really want to quit” to be successful was a commonly expressed belief in this study as it has been in others (Balmford & Borland, 2008; Carter et al., 2001). This belief was expressed by smokers, non-smokers and ex-smokers. Balmford and Borland (2008) note that this belief may discourage quit attempts if individuals believe that it



will be fruitless unless they feel no ambivalence about their smoking and are “ready” to give up, a point that many smokers may never reach. Another important idea about smoking cessation expressed by participants was that the best treatment “depends on the individual”. What works for one smoker will not necessarily work for another. This view of nicotine addiction implies that smokers may need to try a number of methods in order to find one that suits them. Pharmacological cessation aids are positioned within this discourse as one method among others. Given that these two discourses circulate widely in public discourse, it is likely that smokers are frequently exposed to them. Further research should be conducted exploring the potential behavioural consequences of the commonly held beliefs that smokers must “really want” to quit to be successful and that the best method for quitting depends on the individual. In addition, attention should be given to the implications for how clinicians should deal with smokers who express these views.

This study examined the views of the general public about treatments for smoking cessation. This is important given that smokers do not make decisions in isolation, but are influenced by their social environment, including family, friends, and messages in the media. Although some smokers were part of this sample, their recruitment was incidental. Further qualitative research specifically targeting smokers and exploring their attitudes toward medications for smoking cessation would complement this research. It is important that qualitative research about smoking continues to be conducted in order to capture the complexity of public beliefs around smoking and quitting. It is also important that this research be conducted at multiple timepoints in order to explore changes in public discourse about smoking in the face of new treatment developments and continued promotion and increased availability of medical aids for smoking cessation.

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## **5.6. Concluding remarks on Chapter 5**

This paper revealed that medications for smoking cessation are widely acknowledged as an appropriate treatment for smoking, but are not seen as magic bullets for quitting. Medications for smoking cessation are seen as one method among many, and are seen as particularly suitable for those who find it difficult to quit unassisted. Unassisted quitting was typically viewed positively, and associated with traits such as willpower and personal strength.

One of the features of biomedicalisation is that it places a moral responsibility on each individual to constantly assess and address their own risk factors and health status (Clarke, et al, 2003). The individual is judged morally responsible for illnesses that are deemed preventable (Lupton, 1993). As smoking is a key risk factor for many serious diseases, there is significant moralising around cigarette smoking, to the point where advertisements shaming smokers have been recommended as a strategy to encourage quit attempts (Amonini et al, 2015). For those interviewed for this study, this focus on individual responsibility was central to narratives about smoking cessation. The individual was deemed responsible for making a decision to stop, and this was associated with cultural beliefs about self-sufficiency, personal strength and willpower. Less emphasis was placed on *how* someone stopped and it was thought that the best method would “depend on the person.”

While a medical approach was rarely rejected outright, medications were generally seen as insufficient on their own for dealing with nicotine addiction. The role of motivation and desire to quit were central to public talk about smoking cessation. It was felt that if an individual was not “ready” to quit, then any attempt would be unsuccessful, regardless of the method used. This suggests that the individualisation described in works on the social science of biomedicine can be dissociated with the process of medicalisation. In relation to smoking at least, an emphasis on taking personal responsibility for one’s health does not necessarily mean engaging with a medical model of treatment. It is perceived as more about motivation than means.

The results from this study suggest that, contrary to concerns outlined in Chapter 1, a medical view of smoking cessation does not predominate in discussions about smoking cessation with the Australian public. A more complex discourse exists in public talk about smoking cessation. The medicalisation of smoking has occurred to some extent, but older discourses of habit, readiness to quit, and willpower remain dominant in lay understandings of smoking and quitting. This may limit the uptake of smoking cessation pharmacotherapies in Australia. However, as medical technologies develop, and neuroscience knowledge about how nicotine works in the brain is more widely disseminated, it is possible that increasing expectations will be placed on smokers to use existing or emerging medical treatments. This possibility will be discussed further in forthcoming chapters.

### **5.7. Original contribution of Chapter 5**

There is little qualitative research conducted in Australia that explores public attitudes towards treatments for smoking cessation, and the research reported in this chapter begins to address this gap. As new medical treatments for smoking cessation become available, and public portrayals of smoking change over time, it is important to monitor the shifting attitudes towards smoking and cessation. Lay attitudes towards smoking have changed drastically over decades, leading to reduced smoking prevalence and an increased stigmatization of smoking. Chapter 1 described a shift from smoking being portrayed as a “bad habit” that can be overcome with willpower, to a “nicotine addiction” that should be treated by medical professionals. This research reveals that even when smoking is described as nicotine addiction, the former view of smoking still holds much sway in public discourse. Lay understandings of smoking and quitting amongst the general public draw strongly on cultural beliefs about self-sufficiency, personal strength and willpower. Medications are seen as suitable for those lacking in these typically admired traits. Rather than being dismissed or wholeheartedly accepted, medications for smoking cessation are incorporated into already circulating ideas about smoking and nicotine addiction. This is consistent with Nancy Campbell’s assessment that the impact of medicalisation on addiction has been limited because addiction is a “hybrid cultural construct that conveys sociocultural meanings that persist in ways that work against full bio/medicalization.” (Campbell, 2012, p. 6).

Exploring public attitudes towards addiction is important because such attitudes are part of the social climate in which smokers make decisions about whether and how to quit. Examining public attitudes can indicate the uptake of messages from the media and policy statements. While some smokers participated in this study, their recruitment was incidental. Chapter 6 will outline the results of qualitative research conducted with a sample of Australians who smoke daily about their views on a variety of quitting methods, including medications and unassisted quitting.

## **Chapter 6. Why don't smokers want help to quit? A qualitative study of smokers' attitudes towards assisted versus unassisted quitting**

The previous chapter reported on qualitative data from interviews with members of the general public. While some current smokers were included in this sample, their recruitment was incidental. This chapter reports data from Study 2, a qualitative study that involved semi-structured interviews with current daily smokers. The data reported here looks at the attitudes of these current smokers towards assisted versus unassisted quitting. Assessing views about medications for smoking cessation amongst smokers provides further insight into the degree that smoking has become medicalised in Australia. There is little qualitative research on Australian smokers' views on specific quitting methods. Some studies have examined attitudes towards assisted and unassisted quitting in Australia (Balmford & Borland, 2008; Hung et al., 2011) but these are quantitative studies that do not reveal the factors that smokers consider when making judgments about quitting methods. In addition, the majority of these survey studies on smoking cessation do not gather data on attitudes towards unassisted quitting (Smith, Chapman, & Dunlop, 2013). As demonstrated in the previous chapter, attitudes towards quitting with medication are often constructed in light of existing ideas about quitting "cold turkey". Therefore neglecting to ask about this quitting method is a serious shortcoming of many studies on smoking cessation.

A recent exception is a study by Smith and colleagues (2015)<sup>2</sup>. They conducted qualitative interviews with Australian former smokers who had quit without assistance on their views of assisted and unassisted quitting. Not surprisingly, those who had successfully quit unassisted had positive views of this method. They also found that people chose to quit unassisted held a strong belief that quitting was a personal responsibility, thought that quitting unassisted more closely aligned with their identity as a smoker, and stated that quitting without assistance was a rational choice based on an assessment of the costs of benefits associated with different methods. As only smokers who had quit without assistance were included in their sample, we cannot apply these findings to all smokers and former smokers. There may also be a retrospective bias, whereby the difficulty of quitting unassisted is minimised by those who have

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<sup>2</sup> Data collection for the Smith et al (2015) study began in December 2012 and was completed in December 2013. The data that makes up this thesis was collected between October 2012 and July 2013.

been successful. Another possibility is that those who are successful at quitting unassisted may be those who had less difficulty quitting due to lower nicotine dependence.

The remainder of this chapter contains a paper published in 2015 in the *International Journal of Environmental Research and Public Health*. The paper reports results from in-depth interviews with Australian daily smokers, exploring their views and experiences of a variety of quitting strategies, including nicotine replacement therapy (NRT), prescription medications, counselling, self-help materials and unassisted quitting. The aim was to evaluate claims that an increasing emphasis on assisted cessation will lead to a concomitant decrease in positive attitudes towards unassisted quitting. Have smokers come to see unassisted quitting as too difficult? Do they view quitting with medications as the best way to quit? This study also provides insight into the way that smokers' views about cessation strategies are formed. Attention is given to the justifications and examples provided by participants to explain their inclination or disinclination to use certain cessation methods.

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**Abstract:** The development of prescription medication for smoking cessation and the introduction of evidence-based guidelines for health professionals has increasingly medicalised smoking cessation. There are debates about whether medicalisation is a positive development, or whether it has devalued unassisted quitting. In this debate the views of smokers have been neglected. This study explored the attitudes of smokers towards a range of quitting methods, and their considerations when judging their value. We conducted semi-structured interviews with 29 smokers and analysed data using thematic analysis. The results show that the perceived nature of an individual smoker's addiction was central to judgments about the value of pharmacological cessation aids, as was personal experience with a method, and how well it was judged to align with an individual's situation and personality. Unassisted quitting was often described as the best method. Negative views of pharmacological cessation aids were frequently expressed, particularly concerns about side effects from prescription medications.

Smokers' views about the value of different methods were not independent: attitudes about cessation aids were shaped by positive attitudes towards unassisted quitting. Examining smokers' attitudes towards either assisted or unassisted quitting in isolation provides incomplete information on quitting preferences.

### **6.1. Introduction**

Smoking cessation has become increasingly medicalised since the introduction of nicotine replacement therapy (NRT) in the 1970s. More recently, increased knowledge about the physiological mechanisms of nicotine dependence have led to the development of new medications, such as varenicline, that increase the chances of successful cessation (Cahill, Stevens, Perera, & Lancaster, 2013; Hartmann-Boyce et al., 2013). Clinical guidelines now encourage health professionals to identify smokers and facilitate quit attempts by prescribing pharmacological cessation aids and/or referring smokers to counselling services (Fiore et al., 2008; National Institute for Health and Clinical Excellence, 2008; Zwar et al., 2014). A recent commentary has called for treatment to be provided to all smokers who attend a health provider, not just those who express readiness, or an interest in quitting (Richter & Ellerbeck, 2014).

A number of commentators have been critical of this medicalised approach to smoking cessation. Some have concluded, based on population-based observational studies, that pharmacological cessation aids are no more effective than no treatment (Alpert et al., 2013). Others have argued that while pharmacological treatments have demonstrated efficacy in clinical trials, these individually focused treatments have not significantly reduced smoking prevalence (Pierce et al., 2012; Smith et al., 2013). It has also been suggested that the promotion of cessation aids by pharmaceutical companies may imply that quitting without formal assistance is more difficult than it is, thereby undermining smokers' willingness to try to quit and their belief in their ability to stop (Chapman & MacKenzie, 2010; Pierce et al., 2012; Zhu et al., 2012) Chapman and McKenzie argue that unassisted quitting, or "cold turkey", has the greatest impact on reducing smoking prevalence and accordingly should receive greater clinical and research attention (Chapman & MacKenzie, 2010).

This debate has implications for public health messages about how smokers should quit, and how smoking cessation is discussed in clinical interactions. Should people who smoke be told, as has been recently suggested, that they have a "chronic, relapsing disease"? (Wolff, Hughes, & Woods, 2013). Should they be informed that it is difficult to quit and relapse is likely? Do

we want them to believe that smoking cessation requires treatment by a health professional? Or should they be informed that the majority of people quit unaided; that quitting is often easier than anticipated; and that with motivation and willpower, they can quit on their own?

The aim of this paper is not to conclusively answer these questions, but to explore how smokers themselves evaluate, and deliberate on, different methods for quitting smoking. Public health researchers have long recognised and examined the influence of lay beliefs about health and illness on health-related behaviours such as treatment choice and adherence (Lawlor, Frankel, Shaw, Ebrahim, & Smith, 2003; Milburn, 1996; Popay & Williams, 1996). For example, Horne and Weinman and Hankins (1999) found that treatment adherence was predicted by the difference between beliefs about the necessity of treatment and concerns about side effects. Research conducted with smokers on their beliefs about smoking cessation has typically surveyed their attitudes towards specific quitting methods, usually with the aim of identifying barriers to the use of pharmacological cessation aids. These studies have shown that smokers often display negative attitudes towards pharmacologically assisted cessation and express concern about their safety and efficacy (Ferguson et al., 2011; Gross et al., 2008; Mooney et al., 2006). Not surprisingly, these negative perceptions predict a lower intention to use pharmacotherapies and poorer adherence in those smokers who do use them (Bansal et al., 2004; Etter & Perneger, 2001; Fucito et al., 2009; Shiffman et al., 2008).

Quantitative research on perceptions of safety and efficacy may provide an incomplete view of the factors that smokers consider when making choices about how to quit. In addition, few of these studies have examined views on the most common method that people use to quit: “cold turkey” or quitting unassisted. A systematic review of the Australian literature on how smokers quit found that only 19 of 185 studies included data on unassisted quitting (Smith et al., 2013). A recent study that did include data on unassisted quitting found that NRT and prescription medications were rated as helpful by those who had used them. However, unassisted quitting was used substantially more often than either, and also rated as helpful (Hung et al., 2011).

Qualitative research may provide a more nuanced account of smokers’ attitudes towards treatment for smoking. For example, smokers have reported that NRT did not reduce their cravings and that they were concerned about becoming addicted to it (Bott, Cobb, Scheibmeir, & O’Connell, 1997; Vogt et al., 2008). Also, NRT was not seen to address the critical role of willpower in quitting smoking, or the ways in which cigarette smoking was intertwined with

routine and social aspects of everyday life (Wiltshire et al., 2003). Many young smokers felt they did not require NRT because they did not see themselves as physiologically addicted (Amos et al., 2006).

While the existing qualitative research has provided insight into smokers' thoughts about quitting in general, there is little that has compared smokers' views about multiple different quitting methods. This paper addressed this gap by providing a nuanced view of (1) smokers' attitudes towards assisted *and* unassisted quitting, and (2) the factors that smokers take into account when evaluating and comparing different methods of quitting.

## **6.2. Method**

Semi-structured interviews were conducted with 29 daily smokers aged 18 years or over from a large metropolitan Australian city. We employed purposive sampling in order to document the breadth of ideas about quitting methods. Prior to the interview commencing, a short survey with questions about demographics and smoking history was completed by participants (see Appendix C). The recruitment strategy was periodically adjusted as required to obtain a "maximum diversity" sample in relation to age, sex, education, and socioeconomic status (Patton, 1990). For example, flyers were distributed to neighbourhood community centres in order to recruit socially disadvantaged smokers. A university mailing list was employed to recruit university educated smokers, and a seniors database used to recruit older participants. Other methods included handing out flyers in person, advertising via an online classified site, and placing the adverts on community noticeboards. We judged that thematic saturation had been reached at 29 interviews, when a sufficiently diverse sample had been obtained and no new themes were emerging from ongoing analysis. Participants were provided with a gift voucher in appreciation for their time. All recruiting and interviewing was conducted by KM between October 2012 and July 2013.

The interview questions reported here are a subset from a larger project about neurobiological understandings of nicotine addiction. Participants were asked about their attitudes toward various methods for quitting smoking, specifically: "*What is your view on the following methods for people trying to quit?*" This initial exploratory question was designed to elicit unprompted views about the methods, in order to ensure a space for emergent themes. Prompts were then provided where appropriate. Example prompts were: "*Do you have any experience*



using [insert method]? Do you think it is safe? Do you think it is effective? Participants were asked what method they would choose if they were to make a quit attempt and why; and were asked to describe any previous quit attempts.

All participants were asked about each of the following methods for quitting: 1) no treatment (prompt: cold turkey); 2) nicotine replacement therapy (prompt: gum, patches); 3) prescription medication (prompt: Champix, Zyban); 4) counselling, including the *Quitline*; and 5) self-help materials (prompt: books, information pamphlets). We also asked whether participants had thoughts on any other methods not mentioned.

Interviews were recorded and transcribed verbatim. They ranged in length from 25 minutes to one hour and 20 minutes. The confidentiality and anonymity of participants was maintained at all times through adherence to standard ethical procedures (National Health and Medical Research Council, 2007). All participants provided informed consent and the study was approved by the Human Research Ethics Committee of The University of Queensland.

We employed thematic analysis, as described by Braun and Clarke, to analyse the data (Braun & Clarke, 2006). Thematic analysis has been described as the most useful method for “capturing the complexities of meaning within a textual data set.” (Guest, MacQueen, & Namey, 2012) and it is well suited to exploratory studies using interview data. An inductive approach was utilized, whereby KM developed descriptive codes based on patterns observed in the data and conducted a critical analysis of these codes in order to collate them into major themes. Data coding was conducted using NVivo 10 software (QSR International Pty Ltd., 2010). Another author (BP) read the transcripts and developed themes independently. There was good agreement about the themes and any discrepancies were discussed until a consensus was reached. In addition, another member of the research team conducted double-coding of a subset of data in NVivo in order to ensure that the final coding scheme had adequate reliability.

### **6.3. Results**

#### ***Participants***

Participant demographics are presented in Table 6.1.

Table 6.1 - Participant demographics for Study 2

<b>Demographic</b>	<b>Number</b>
Gender	
Male	15
Female	14
Age (yrs)	
18-25	9
26-40	11
41-54	4
55+	5
Highest level of education	
No formal qualification	4
Secondary school	4
Post-secondary qualifications (e.g., trade training)	10
University degree	11
Employment status*	
Employed	15
Unemployed	5
Student	7
Retired/Pensioner	3
Cigarettes per day**	
1-10	10
11-20	11
21-30	3
31+	4

\*Multiple selections permitted. \*\*Missing data = 1. N= 29.

In order to gain an overview of participant experience with quitting methods, basic data was collected in the pre-interview survey about quitting history. Participants were presented with a list of methods adapted from the Australian National Drug Strategy Household Survey (Australian Institute of Health and Welfare, 2014) and asked to select the methods that they had used. Multiple selections were permitted. Four participants reported that they had not previously made a quit attempt. The quitting strategies that the remaining participants reported having used are listed in Table 6.2. Though NRT was the method that the most participants reported having used, a significant proportion of the participants had no direct experience with pharmacological cessation aids.

Table 6.2 - Strategies used on previous quit attempts

<b>Method</b>	<b>Number of participants</b>
Discussed smoking and health at home	10
Contacted the “QUIT” line	3
Asked your doctor to help you stop smoking	4
Used nicotine gum, nicotine patch, or nicotine inhaler	11
Used a smoking cessation pill (e.g., Zyban, Champix)	3
Bought a product other than nicotine patch, gum or pill	2
Read “How to Quit” literature	9
Used the internet to help you quit	5
Done something else to help you quit?	7
None of the above	8

### ***Unassisted quitting***

Unassisted quitting was frequently described as the best way to quit smoking and it was participants’ overwhelmingly preferred method for their next quit attempt. A number of justifications were provided for this preference. First, there was a belief that if someone had a strong desire to quit and was “ready” for it, then assistance would not be required. Desire and motivation was seen as the foundation of quitting success:

*You’ve really got to want to do it and have that courage, strength, determination to do it. You’ve really got to have that thinking in your mind, this is what I want. I personally believe that the mind has a most powerful part in this whole process. (female 55+, 21-30 cigarettes per day (CPD))*

This was tied to the belief that cold turkey would only be effective at a time when someone had reached a point where they “really wanted” to quit. Many of our participants said that willpower, or a strong desire to quit, was a necessary condition for a successful quit attempt. However this seemed to imply that that if a person failed in their attempt to quit cold turkey, then this meant that they hadn’t really wanted to quit; that they weren’t strong enough; or that they didn’t have the right “mindset”. For example, the participant quoted below had stopped smoking and relapsed a number of weeks later. She attributes this relapse to not “wanting” it enough.

*Because I always imagined if you'd stopped for a few weeks, how would you go about having that first cigarette? You would be just like no it's not worth it, but I did. I don't even know when it was. It was probably I was out with my friend and I didn't even*

*realise I did it. You know I just - yes. I think if you want, I think that's the main way. If you don't want to do it, you're not going to do it.(female, 18-25, 1-10 CPD)*

Another participant implied that his failed quit attempts were due to a lack of internal strength or desire.

*It depends how strong you are and how much you want to. If you are strong, if you really want it, you know. I couldn't do it. Simple as that, I'm still smoking. (male, 41-54, 31+ CPD)*

High value was placed on the sense of achievement that was anticipated as a result of quitting unassisted. This was more common amongst younger male participants, who saw quitting smoking as a challenge or a competition with oneself. Seeking assistance in the form of other cessation aids was seen as a “crutch” or a form of “cheating” that would mean you hadn’t won against smoking:

*I think it would be more of a trial for myself. Like a goal setting thing. I'm a very goal-orientated person. If I can go cold turkey that would be like a big achievement for me. (male, 18-25, 1-10 CPD)*

The role of personal experience was a salient consideration when participants spoke about unassisted quitting. Most participants had tried to quit cold turkey, so could reflect on their own experience with this strategy of smoking cessation. As described above, some attributed past failures to personal weakness or a lack of desire to quit. There were other participants who considered past unassisted quit attempts successful, despite the fact that they had relapsed and were still smoking.

*Interviewee: I'll just determine that I want to quit and I can.*

*Facilitator: Why would you use that method now?*

*Interviewee: Because I've tried it before and it's working for me. Yeah, so I think that's the easiest one. (male, 26-40, 1-10 CPD)*

The experiences of friends and family were sometimes used to justify an inclination or disinclination to use unassisted quitting.

*Terrified. I know people who've done it but they've usually gone back to smoking again. They've often really struggled. I've seen people be very stressed and distressed during the cold turkey. So clearly some are able to do it but it looks pretty difficult. (female, 55+, 11-20 CPD)*

Despite many stating a preference for quitting cold turkey, it was common to acknowledge the difficulties associated with it. Indeed, cold turkey was sometimes seen as both the hardest and best way to quit. The major difficulties were attributed to withdrawal symptoms and cravings.

Some smokers, particularly those with a history of failed cold turkey quit attempts, thought that the method was better suited to those who were stronger or who had more willpower than themselves.

*It depends on the person. I mean some people can do that and some people have the willpower or the determination to do it. They don't need aids but yeah most people would. (female, 26-40, 31+ CPD)*

The perceived level of addiction was another factor participants saw as important for unassisted quitting. Unassisted quitting was seen as most suitable for those who were not heavily addicted to cigarettes. The participant below equates heavy smoking with dependence.

*I have friends who quit like that, cold turkey, and it worked out pretty well. But then again, they're not those really heavy ones so I guess it works for people like us who aren't that hooked on that shit yet. (male, 18-24, 1-10 CPD).*

### ***Assisted cessation***

NRT is the most commonly used pharmacotherapy for smoking cessation. In Australia, the cost of NRT is heavily subsidised if participants attend their doctor and receive a prescription. Despite this, only four of 29 participants intended to use NRT on their next quit attempt. Cost was mentioned as a barrier to the use of NRT by some participants, which could indicate a lack of awareness about government subsidisation. However, a more common consideration was the individual's assumption about the nature of a smoker's addiction. NRT was thought to be most appropriate for those with a "physical" or "physiological" addiction. Many described themselves as addicted to the act of smoking and saw their addiction as "psychological", or as a habit built into their daily routine. These participants did not necessarily have negative views of NRT; but thought it was more suitable for smokers who had a "real" physiological addiction.

*[NRT] might be extremely effective on people who are very physically addicted. If they're psychologically addicted I don't see how it's going to have any effect. (female, 41-54, 11-20 CPD)*

NRT was seen by some of these smokers as failing to deal with the psychological or routine aspects of smoking that they considered central to their dependence.

*I think it could help some people, but still it's because it's such a habit to smoke it's not just the nicotine. ... Each cigarette we smoke is the fact of doing it, is having the pack in your bag, it's like all those things should be replaced and so probably replacing it could help the craving for those people who are very hooked up. But I don't think it*

*would completely solve the issue and it wouldn't definitely help 100% to quit smoking, there are a lot of other things involved. (female, 18-25, 1-10 CPD)*

A small number of participants who had experience using NRT acknowledged the role of physiological dependence in their smoking and thought that NRT had been effective for them *because* it dealt with the physiological aspect of smoking. It allowed “breathing space” to deal with the more habitual, routine aspects of smoking.

*Yeah, I think that does help because it does take away that initial physical withdrawal feeling so that you can concentrate on trying to manage the habit part of it. That, for me as I said, it only took a couple of weeks for me to get that clearing out of my system and then it was just a matter of trying to manage the ritual habit part of it. So that definitely made it a lot easier. (female, 26-40, 31+ CPD)*

As with unassisted cessation, an individuals' experience with NRT played a key role in their attitudes towards it. Participants rarely reported using NRT as directed. Rather, participants were more likely to use NRT short-term during long-haul flights or short-term stays in hospital. It was also used by a small number of participants as a one-off “experiment” to see what would happen:

*The patches - we've got the patches on and we've just - we'll see if that works. We're not trying to give up smoking. I've just left them on there and thought, right I'll have a cigarette when I want a cigarette. (male, 41-54, 31+ CPD)*

Personal experience was particularly salient in relation to side effects. Those who had used NRT and experienced unpleasant side effects reported that they would not use it again. Even hearing about someone else's experience of side effects was enough to dissuade people from using NRT:

*I'm kind of skeptical on all the other stuff - the products on the market to stop it, patches and stuff like that. I'm kind of - I don't know. Because I had a friend who used the patches and he used to have nightmares and - yeah, stuff like that. So I'm not too keen on it. (male, 18-25, 1-10 CPD)*

A small number of participants were concerned about developing dependence on NRT. They saw dependence on nicotine as a negative state, with there being no essential difference between whether they consumed nicotine via smoking cigarettes or via NRT.

*At some point you do need to just stop. You can't just keep feeding your body this drug that you're addicted to, you might as well be smoking. (female, 26-40, 11-20 CPD)*

The prescription medications bupropion and varenicline are publicly subsidised forms of pharmacotherapy for smoking cessation in Australia. Approximately one third of participants

were unaware of the existence of these prescription medications for smoking cessation. Because direct to consumer advertising of prescription medications is not permitted in Australia, this is perhaps not surprising. Amongst our participants, those who were older and heavier smokers were more likely to be aware of these medications. Those who did know of these medications frequently expressed concern about their safety. Cost was mentioned less often, perhaps because the fear of side effects dominated considerations of costs. While only a few had tried the prescription medications for smoking cessation, many had heard reports about adverse side effects from their friends, family or acquaintances. The most commonly mentioned were mental health issues and nightmares. These side effects were cited as the main reason why most would not consider using prescription medication.

*Then they try and tell me that these medications will stop me smoking although I'll have nightmares, I'll have all the other side effects. I heard about one, I can't remember the name of it, and my dad had it - reckoned he nearly died. Made him really sick. I've heard about people having the nightmares and things, so that really makes me question what they're giving you other than nicotine. You might stop smoking, but you're just as irritable from not sleeping. So, I don't know. I just don't agree with the pharmaceuticals. If you're going to quit, quit. (male, 26-40, 11-20 CPD)*

This dislike of prescription medications for smoking cessation was sometimes an expression of a more general dislike of “relying” on any sort of medication. The participant quoted below positioned “taking pills” as an extreme measure for smoking cessation, especially when quitting without assistance was a realistic alternative. It may be that a “reliance” on medication conflicts with the value of self-reliance that many participants identified with.

*I'm really against, not against, but I think like taking pills and taking things like that should be done only if it's really needed and as long as I feel like I could do it without, it would always be better option than relying on medicine. (female, 18-25, 1-10 CPD)*

As with NRT, some participants thought that prescription medication would be more suitable for “other” smokers with a more serious addiction; they were not inclined to use these medicines themselves. Medications were associated with “illness” and “sickness” that heavier and older smokers might develop.

*I personally just can't get my head around doing something like a pharmaceutical pill or something like that ... It seems over the top but I understand that some people who are really ill and continue smoking will probably need that. (male, 26-40, 11-20 CPD)*

As with NRT, perceptions of efficacy were also closely tied to the experiences of family and friends who had used these medications.

*...some of my friends have tried both of those and I still find that they're smoking so I've seriously questioned that. Maybe their commitment wasn't strong enough or whatever. But yeah I'm just still hoping for the wonder drug to be out there or something. (female, 55+, 11-20 CPD)*

As the quote demonstrates, having sufficient willpower was still perceived as important, even when medication was taken. The participant expressed a hope that a “wonder drug” would be developed that, presumably, would overcome this need for willpower or sustained effort. However, medication was not generally seen as replacing willpower and mindset, which were seen as essential ingredients of a successful quit attempt:

*I'd probably have to go to the doctor and ask to go for the Champix or something because as I said I'm on patches at the moment, that's not effective. But I do know that I have to change my personal situation so that's helpful and my mindset changes too. (female, 55+, 11-20 CPD)*

Interestingly, the few participants who had used prescription medication found it effective and reported positive attitudes towards it, despite a subsequent relapse.

*Yeah, I reckon that Champix, like that helped me. I slowed down so much in the first two weeks like from going to 20 a day I might have like one in the morning, one at night sort of thing and then maybe, and then a bit further on maybe just one at lunch, that's it. Then I stopped taking it, like I sort of messed up, muddled up and yeah then I just started smoking more and smoking more and you go oh, I'm smoking again. But I think if I had have continued with it I probably - I want to give it another go, so. (male, 26-40, 11-20 CPD)*

As already described, guidelines for treating tobacco dependence recommend that counselling is combined with pharmacotherapy. Few participants in this study reported any personal experiences with counselling for smoking cessation. This is despite the widespread promotion of Australia's *Quitline*: a free, government-funded telephone counselling service that can be accessed by any smoker. The number for the *Quitline* is displayed prominently on all Australian cigarette packs, and health professionals are encouraged to refer smoking patients to the *Quitline*. In addition, referral to a counselling service, which is typically the *Quitline*, is a necessary condition for doctors to prescribe subsidised NRT or prescription medications for patients. Participants reported a number of negative perceptions of the *Quitline*, including that it was “preaching”, that there was nothing *Quitline* counsellors could tell smokers that they didn't already know, and that it was scripted and impersonal.



*Again I think that is completely dependent on the person. I'm far too stubborn to ever listen to anything like that. I think it would just make it worse if someone was preaching to me, which is the way I would see it, whether it was actually like that or not. (female, 26-40, 1-10 CPD)*

It was common for participants to state a preference for “personal” support from family and friends.

A less commonly discussed theme was a lack of interest in counselling. A number of participants said that they “weren’t talkers” and therefore were not inclined to use counselling to quit smoking. Even those who expressed positive views of counselling were reluctant to use it for smoking cessation. Only one person intended to use the *Quitline* on their next quit attempt, and two said that they would use generic counselling.

Participants were also questioned about their views on self-help material such as books, pamphlets, and online information. While participants had moderately positive views about self-help materials, they did not hold strong views about them. A handful of participants described specific materials that they had found useful. The framing of the message was described as being important, with some complaining about “scare-mongering” in self-help materials. Self-help materials were perceived by a few as insufficient for quitting smoking. Others were not interested in them because they did not enjoy reading.

*Could be good, yeah. Depends on individual - if someone is having reading as a hobby, could be helpful. People like me who is not really into reading, yeah, could be waste of time for me. (male, 26-40, 1-10 CPD)*

#### **6.4. Discussion**

We found that smokers’ attitudes towards cessation options were shaped by several factors, some of which were consistent across different methods for smoking cessation. Many participants believed that the best method for quitting would “depend on the person”. Dispositional or character-based factors were often cited when evaluating the potential of a quitting method. Unassisted quitting was seen as suitable for those with willpower, strong motivation or internal strength. The nature of an individual’s addiction to smoking was also seen as important when smokers deliberated about cessation options, as was perceived efficacy, which was typically assessed on the basis of their own experience, or that of family and friends.

Negative experiences of friends and family were frequently reported, perhaps because such experiences are more salient than positive ones.

Practical factors such as cost or side effects were regarded as significant for some quitting methods. Cost was mainly mentioned as a barrier to using NRT, less often for prescription medication. Side effects were discussed frequently in relation to NRT and prescription medication. The number of side effects mentioned by study participants, particularly in relation to prescription medication, was higher than what would be expected from epidemiological evidence (Gibbons & Mann, 2013; Svanström, Pasternak, & Hviid, 2012). This may be because smokers misinterpret nicotine withdrawal symptoms as side effects of smoking cessation medications; or because people are more likely to discuss experiences of medication use where they have experienced side effects than those in which they haven't.

Our finding that some participants did not use NRT in accordance with clinical recommendations is consistent with evidence from quantitative surveys. The latter have found that most smokers do not use NRT as directed and few use a full course of NRT as recommended (Balmford et al., 2011). This lack of adherence increases the likelihood that withdrawal symptoms will be experienced and perhaps mistaken for side effects.

The fact that not all participants were aware of the existence of prescription medications is not surprising given the lack of direct to consumer advertising of prescription medications in Australia. Additionally, since one of the indications for the prescription of smoking cessation medications in Australia is smoking more than ten cigarettes per day, it should be expected that lighter smokers will have less awareness of prescription medications for smoking cessation (Zwar et al., 2014). Research in the UK has shown that young and healthy smokers who attend their doctors are less likely to be prescribed pharmacotherapy for smoking cessation than older smokers with existing health problems (Huang, Britton, Hubbard, & Lewis, 2013). While our research was not able to assess this possibility, it may be that different types of smokers are being provided with different information about their quitting options by health practitioners.

It is interesting that smokers' concerns about side effects usually trumped efficacy, especially in the case of prescription medications. Even if smokers perceived prescription medication to be helpful, this was weighed against the risk of side effects that many decided made the potential benefits not worth the risk. The literature on risk perception in smoking shows that

the distal nature of the health risks are a deterrent to quitting, particularly for young people, as they often hold optimistic beliefs about their ability to quit smoking prior to developing any smoking related health problems (Weinstein et al., 2004). Any side effects from using pharmacological cessation aids for smoking cessation are more immediate, and may therefore take precedence over the longer-term health risks of smoking. This is consistent with evidence on the use of medications more generally, where the difference between the perceived necessity of a medication and concerns about its use predict poor medication adherence (Horne et al., 1999). It is important that future research assessing smokers' attitudes towards quitting methods take their more general views on the use of medications into account in the study design.

Beliefs about addiction were influential in our smokers discourse on smoking cessation. For example, those who believed that they were not physiologically addicted to nicotine, or who did not consider themselves to be "heavy" smokers, did not see pharmacological cessation aids as appropriate for them. These participants were more likely to hold positive views about cold turkey quitting. This is a complex topic for health practitioners to negotiate. One potential implication of this finding might be that smokers need to be educated about nicotine addiction in order to convince them that they have a physiological dependence that can be treated using medications. However, as Chapman and McKenzie (2010) argue, such an approach may unintentionally devalue unassisted quitting, and produce a counterproductive effect in which smokers who are told how difficult it will be for them to quit smoking, are less inclined to try to quit. Indeed, a recent paper suggests that health practitioners "emphasize the difficulty of quitting without assistance" in order to promote uptake of medications for smoking cessation (Myers, Strong, Linke, Hofstetter, & Al-Delaimy, 2015). We suggest that a more sensitive, tailored approach is employed by healthcare practitioners. Where patients are very averse to medications, it would be counterproductive to emphasize the difficulty of quitting unassisted. Probing patients about their views on nicotine addiction and their attitudes towards medications may aid doctors in designing individualised treatment plans for patients who have tried and failed to quit cold turkey on a number of occasions.

Our study shows that smokers evaluate a given method for quitting in light of a range of alternatives and contingencies. For example, when thinking about varenicline, smokers might think that it will be effective, but believe that the side effects are not worth it. Unassisted quitting is seen as a particularly salient alternative to pharmacological cessation aids because

it is free, safe and perceived by many smokers to be the most effective way to quit. This point has been neglected in the smoking cessation literature, where smokers' views on unassisted quitting have not often been sought (Smith et al., 2013). Males in particular preferred cold turkey quitting because they anticipated a strong sense of achievement from quitting without help. The value placed on this sense of achievement from quitting unassisted has been observed in another study (Vogt et al., 2008). It may be helpful to take this into account when designing interventions aimed specifically at men.

Our research suggests that it was common for smokers to believe that quitting cold turkey will only be effective if the smoker is “ready” to quit, and has the right “mindset”. This idea that smokers need to be ready to quit has also been prominent in smoking cessation literature and programs, thanks to the influence of the transtheoretical model of behaviour change. The transtheoretical model posits that individuals pass through a set of ordered stages in their journey to behaviour change, and that different interventions are suitable for different stages of change. For example, those in the “pre-contemplation” stages are not yet psychologically ready to change their behaviour. Interventions aimed at people in this stage are primarily informational and aim to increase desire and motivation to move smokers into the next stage – contemplating a quit attempt- rather than promoting an immediate attempt to quit. The transtheoretical model has been strongly criticized on the grounds that behaviour change is more dynamic and complex than the model assumes (West, 2005) and that unplanned, spontaneous quit attempts may be more successful than planned ones (Ferguson et al., 2011; Medbo, Melbye, & Rudebeck, 2011; West & Sohal, 2006). Moreover, the belief that an unqualified desire to quit is required prior to a successful quit attempt has been identified as a barrier to making quit attempt (Balmford & Borland, 2008; Richter & Ellerbeck, 2014). Our research is consistent with this view, with many smokers stating that they would quit at some precise point in the future when they were “ready”. It may be effective for stop smoking campaigns to challenge the idea that you need to achieve a “readiness to quit.” Along these lines, a recent commentary has recommended that all smokers presenting to their primary health physician should be provided with treatment, regardless of their expressed readiness to quit (Richter & Ellerbeck, 2014).

The results of this study are also consistent with previous qualitative research in showing that smokers consider willpower, strength, and motivation as central to successful quitting (Kishchuk, Tremblay, Lapierre, Heneman, & O'Loughlin, 2004; Vogt et al., 2008; Wiltshire et

al., 2003). Cessation aids were not perceived as “magic bullets” for cessation. Rather smokers emphasised that willpower and personal choice were necessary, even when cessation aids were used. This discourse of “willpower” has long been central to lay accounts of smoking. It aligns with Western cultural values of free choice and individual strength. It is a view that has been heavily promoted by the tobacco industry to argue for fewer government interventions to prevent or discourage smoking (White et al., 2013).

Even with the increasing biomedicalisation of smoking cessation, it seems highly unlikely that the discourse of willpower will disappear from public discourse on smoking. Therefore incorporating beliefs about willpower into smoking cessation campaigns and clinical interactions may be of value. For example, messages that tell people who are using pharmacological cessation that willpower is still required may allow successful quitters to attain the sense of achievement that was valued by some in our study. It also provides more realistic expectations about the efficacy of current pharmacological options. Relatedly, only a small minority of participants believed that cessation aids would be necessary and sufficient to quit smoking. This finding should allay the concerns of those who fear that the medicalisation of smoking cessation will create a sense of fatalism and decrease smokers’ sense of control over their smoking.

The negative views of the *Quitline* expressed by participants are consistent with evidence of low uptake of counselling in general (Kaufman, Augustson, Davis, & Finney Rutten, 2010; Miller et al., 2003). This may be of concern, given that counselling is required in conjunction with the prescription of pharmacological cessation aids in clinical practice guidelines. Despite many acknowledging the psychological and behavioural aspects of smoking, few participants expressed an interest in counselling and only one participant intended to use the recommended combination of pharmacotherapy and counselling for their next quit attempt.

One limitation of this study was that participants were asked about their views of unassisted quitting using the prompt “cold turkey.” Cold turkey is generally taken to mean quitting suddenly, rather than gradually cutting down on the number of cigarettes. Cutting down is a method of quitting that is commonly used by smokers but we did not directly ask about it. Future research in this area should ask about cutting down separately from quitting “cold turkey”, or more clearly describe methods of unassisted quitting before questioning participants.

Lastly, although nicotine replacement products are widely advertised in Australia, there is no direct to consumer advertising of prescription medications. It would be useful to examine attitudes in countries where direct to consumer advertising for prescription stop-smoking medications is permitted (e.g., New Zealand, USA) to see if advertising influences smokers' attitudes towards prescription cessation aids or unassisted quitting.

It should be noted that this was qualitative research and no inferences about the prevalence of these beliefs in the larger population of smokers can be drawn. However, these findings provide an insight into the range of factors that smokers consider when evaluating quitting methods. This information is useful to inform future work in this area. Specifically, smokers' judgments about which methods to use for smoking cessation are not simply based on perceived safety and efficacy. They reflect their ideas about the nature of their addiction, how well a given method suits their perceived situation and personality, and their own and others' experiences with the method. Their views about different methods are often not independent. For example, views about NRT are shaped by very positive attitudes towards quitting cold turkey. Looking at attitudes towards assisted or unassisted quitting in isolation may provide incomplete information on quitting preferences. It is therefore important that the above-mentioned factors are considered when conducting research into treatment preferences for smoking cessation. Smokers' views should be compared across different quitting methods and at the very least, include quitting unassisted as a comparator.

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### **6.5. Concluding remarks on Chapter 6**

This chapter examined Australian smokers' views on assisted and unassisted cessation. Because these participants were daily smokers, this sample is more likely to have been exposed to medical models of smoking cessation than those interviewed in Chapter 5. Current smokers are likely to pay more attention to advertisements for smoking cessation medications and to have interacted with health professionals where possible strategies for quitting were discussed. Despite this, their attitudes largely echoed those of the general public. Unassisted quitting was often portrayed as the best method for quitting and it was the method against which other methods were judged. Unassisted quitting was frequently seen as convenient, safe, and free of

cost. In addition, it seemed to align more closely with many participants' understandings of their smoking as a personal choice that they were responsible for dealing with.

Concerns that the increasing biomedicalisation of smoking will lead smokers to perceive unassisted quitting as extremely difficult, and too hard to attempt, were not supported by this data. While many acknowledged that quitting cold turkey is difficult, this was usually based on past personal experience. Moreover, perceptions of difficulty did not seem to lessen the appeal of unassisted quitting for many smokers. In fact, for some, particularly males, the difficulty of quitting unassisted seemed to contribute to the appeal of unassisted quitting, because they anticipated a sense of achievement from using one's own resources of willpower and strength to quit without help.

This is consistent with other qualitative research on lay understandings of health and illness that show a disinclination to take or medications or adhere to recommended dosage regimens. Pound et al (2005) conducted a synthesis of qualitative studies on "lay experiences of medicine taking." They found that people often "tested" medicines in order to be able to conduct a cost/benefit analysis. Patients evaluated a medicine based on side effects, how the treatment regime fit into their everyday life, and how well it dealt with any symptoms. There were also concerns about becoming dependent on medications. In relation to smoking, this is often seen by smokers as replacing one addiction with another (Vogt, Hall & Marteau, 2007).

The fact that the few participants who had used prescription pharmacotherapies in the past had positive perceptions of them, despite having relapsed to smoking, could have implications for smoking cessation. It may mean that attitudes towards particular medications are not just based on long-term smoking abstinence, but may be influenced by other factors such as a reduction in the number or strength of cravings, a reduction in the number of cigarettes smoked, or the ability to remain abstinent for some period of time. Further research is warranted on how smokers evaluate the efficacy of pharmacotherapies as they are using them, and what they judge as a successful quit attempt is warranted.

A lack of clarity in the literature of what constitutes assisted versus unassisted quitting presents a significant problem. According to Chapman, assisted cessation encompasses pharmacotherapy or "any individual or group behavioural or cognitive intervention" (Chapman & MacKenzie, 2010, p. 1). However, in another paper speaking to the *Quitline* on a one-off

basis is classified as unassisted quitting, as is the use of stop smoking internet-based applications for quitting (Smith et al., 2013). It can be unclear whether a particular quit attempt should be defined as assisted or unassisted, and there is variation in studies as to how quit attempts are classified (Smith et al., 2013). In the interviews reported here, smokers were asked their views on “cold turkey, or unassisted quitting.” However, they were not asked what they thought these terms meant. Further research could explore what laypeople define as assisted quitting, unassisted quitting, and cold turkey.

For example, the ambiguity about whether use of the *Quitline* is a form of assisted or unassisted quitting was reflected in participant opinions about the *Quitline*. Participants generally held unfavourable opinions about the *Quitline*. *Quitline* counselling was viewed as someone telling them what they “already knew” or “lecturing” them about their smoking, which they disliked. This is likely to be associated with the self-reliance that participants valued, but could also be associated with their ideas about authority. *Quitline* staff are labelled on Quit websites as “quit specialists” (<http://www.quit.org.au/preparing-to-quit/choosing-best-way-to-quit/quitline>), and smokers may be uncertain about the background or qualifications of those who staff phone lines. By stating that *Quitline* staff would not be able to tell them anything they didn’t know, many smokers were positioning themselves as the authority on their own smoking, and questioned the ability of others to offer additional insight or assistance.

Lastly, the presence or absence of free will in drug dependent individuals has long been debated in the addiction field (White, 1998). The increasing interest in the neurobiological basis of addiction has increased interest in this question. The head of NIDA has stated that drug addiction should be understood as a “disease of free will,” (Volkow, 2015), while others argue that negating free will is inaccurate and will lead to fatalism amongst addicted individuals (Lewis, 2015). In addiction, disease models based on brain science are often associated with abstinence-based approaches, while harm reduction approaches acknowledge the existence of free will and give drug dependent individuals information about managing their addiction (Szott, 2015). Szott (2015) has shown that health care practitioners who work with injecting drug users often combine a disease model that abrogates responsibility from the individual, with a harm reduction model that grants them autonomy to make choices about safe drug use. The research presented in this chapter with smokers shows that concepts of free will and autonomy are the central concepts underlying smokers’ understandings of their own smoking.



How these beliefs influence attitudes towards a disease model of nicotine addiction will be elucidated in later chapters.

### **6.6. Original contribution of Chapter 6**

As outlined by Smith and colleagues (2013) there are few studies in tobacco control that explicitly examine smokers' views on unassisted quitting. Furthermore, there is very little qualitative research on their views on prescription medications for cessation. This study begins to address these gaps in the current literature on smoking and cessation. In relation to unassisted quitting, evidence has been presented in this chapter that some smokers do not see themselves as having a physiological addiction to nicotine, or see this as only a small part of the reason for their continued smoking. These smokers reject a medical approach to smoking cessation and state a preference for unassisted quitting, which is seen as convenient, cheap and appropriate for their "type" of addiction. Attitudes towards prescription medication tended to be negative. There were strong concerns about the safety of these medications, and most participants thought they should be used as a last resort. Interestingly, the few participants who had used prescription medications found them helpful, despite the fact they had not led to long-term cessation.

After eliciting their views on quitting strategies and experiences, the same participants were questioned more deeply about their understandings of addiction; in particular, their views about the idea that their smoking is due to physiological changes in the brain from smoking that represent nicotine addiction. The remainder of this thesis explores smokers' ideas about the role of their brain in their addiction to smoking, and the potential implications for cessation strategies, particularly the use of medications for cessation.

## **Chapter 7. A qualitative study of smokers' views of brain-based explanations of tobacco dependence.**

Chapter 2 described a recent development in the biomedicalisation of smoking cessation: the development and promotion of the brain disease model of addiction (BDMA). Up until recently, the BDMA has not been applied to smoking in the same way as it has been to alcohol and dependence on illicit drugs. But as was demonstrated in Chapter 3, there is an increasing emphasis on the role of the brain in health promotion materials about smoking, pharmaceutical company advertisements for cessation pharmacotherapy, and media reporting on smoking. Academic and institutional definitions of smoking are beginning to incorporate the idea that it is a “chronic disease”, and that the primary feature is the brain’s reliance on nicotine. While previous qualitative literature outlined in Chapter 3 has found some evidence that physiological explanations of smoking as a nicotine addiction do not dominate lay understandings of smoking, more recent evidence is required as scientific understandings of smoking move towards a biological conception that focuses on addiction to nicotine, and the neurological mechanisms by which this occurs.

While some have been enthusiastic about the potential for brain-based explanations of behaviour to alter societal and individual attitudes towards drug use, empirical evidence thus far does not support these claims (O’Connor & Joffe, 2013). It is important to assess how lay understandings may be influenced by neurobiological ideas in specific fields. This chapter explores how the sample of smokers described in Chapter 6 view the role of their brain in their smoking, and evaluates the potential for brain-based explanations of addiction to lead to increased feelings of fatalism, decreased stigma, and more positive attitudes towards medications in smokers, as has been hypothesised in the academic literature outlined in Chapter 3.

The majority of this chapter is comprised of a paper that was published in *The International Journal of Drug Policy* in 2016, with a conclusion summarising the implications for this thesis.

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## **Abstract**

**Background:** The role the brain plays in the creation and maintenance of tobacco dependence has become increasingly prominent in explanations of smoking that are presented to the public. The potential for brain-based explanations of smoking to influence smokers' understandings of their addiction, their sense of self-efficacy, and perhaps even their treatment preferences, has been raised by some working in the addiction field. However, little empirical evidence exists in this area. **Methods:** This paper reports on semi-structured interviews with 29 daily smokers. Participants were shown a brief presentation about the neuroscience of nicotine dependence. They were then queried about their awareness of the role of the brain in smoking, and the consequences of this knowledge for their understandings of smoking and their treatment preferences. **Results:** Our results indicated that many participants displayed some awareness of the link between the brain and addiction. While there was a diversity of ideas about the potential impacts of neuroscience knowledge about smoking, there was an overall tendency to maintain pre-existing treatment preferences, and to assert individual responsibility for smoking. Emergent themes that arose were the brain as a special organ, the discourse of the "other" smoker, and the distinction between physical and psychological facets of addiction. **Conclusion:** While brain-based explanations of smoking are unlikely to revolutionise lay understandings of smoking, neuroscience information should be presented in a way that does not negate people's sense of agency and self-efficacy in relation to quitting smoking.

## **7.1. Introduction**

Public portrayals and perceptions of cigarette smoking have changed dramatically in recent decades. There has been a shift from the idea that smoking is a habit to the idea that it is an addiction, and more recently, an addiction located in the brain. This paper examines the attitudes of smokers toward brain-based understanding of addiction to smoking, and the ways they interpret its relevance for their everyday practice of smoking.

Neuroscience research has provided strong evidence demonstrating the addictive nature of tobacco smoking. This research has focused mainly on the role of nicotine, and has revealed that nicotine produces behavioural reinforcement by binding to nicotinic acetylcholine receptors in the brain, thereby influencing dopamine release in the brain's mesocorticolimbic reward circuit (Benowitz, 2010; De Biasi & Dani, 2011). The brain's stress system is also affected by chronic nicotine administration in ways that may contribute to the withdrawal symptoms such as anxiety and irritability that many smokers experience on cessation (Bruijnzeel, 2012). At the molecular and cellular levels, plastic changes in the brain, such as changes in synaptic connectivity and the regulation of gene expression, occur with repeated nicotine use and are associated with the development of the clinical signs of addiction: craving, withdrawal, and tolerance (Govind et al., 2009; Govind et al., 2012; Peng et al., 1994).

The general news media regularly report on neuroscientific research, often uncritically, but it is difficult to evaluate the extent to which the public have been exposed to or accept these findings. Articles have appeared in the mainstream media with titles such as "Nicotine takes control of brain" (Fewster, 2002, June 11) and "Quitting is a brain game" (Author not attributed). The public have also been exposed to brain-based explanations of smoking via advertisements for smoking cessation medications. An Australian campaign by Pfizer is headed with the phrase "Break the hold nicotine has over your brain" (Pfizer, 2015). Additionally, influential institutions in the USA have begun to define addiction as a "brain disease" or "brain disorder." For example the National Institute of Drug Abuse (NIDA) describe addiction as a "chronic, relapsing brain disorder" (National Institute on Drug Abuse, 2007) and the American Association for Addiction Medicine define it as "a primary, chronic disease of brain reward, motivation, memory and related circuitry" (American Society for Addiction Medicine, 2015). An emphasis on how drugs affect the brain is evident in much of NIDA's public education material, including that on smoking (National Institute on Drug Abuse, 2006).

The veracity of the "brain disease model" has been critiqued at length in the academic literature (Courtwright, 2010; Hall, Carter, & Forlini, 2015; Russell & Davies, 2009). Our goal here is not to address the "reality" of the claim, but to anticipate its potential social implications, specifically its effects on smokers' understandings of their own smoking behaviour. A number of claims have been made about how an emphasis on the role of the brain could influence the way addicted individuals understand their addiction and the best ways to quit. Proponents believe that it will reduce the stigma associated with addiction, thereby increasing treatment

seeking, and also that it will lead to the development of more efficacious and technological treatments (Dackis & O'Brien, 2005; Gardner, Tapper, King, DiFranza, & Ziedonis, 2009; Leshner, 1997). Others however, express concern that the BDMA could reduce feelings of individual responsibility for tobacco smoking or other substance use and undermine addicted individuals' beliefs in their ability to stop using or their willingness to try. In the case of smoking, critics are concerned that quitting self-efficacy will be reduced if smokers are told that they require medical treatment due to a biological "need" to smoke (Caron et al., 2005) and hence that medicalisation of smoking may reduce unassisted quit attempts (Chapman & MacKenzie, 2010). Conversely, some believe that new smoking cessation treatments will be viewed as "magic bullets", with smokers becoming overly optimistic about the potential for medical treatments to "cure" their addiction to smoking (Dingel et al., 2011).

These can be seen as examples of a wider concern about the influence of "brain talk" on subjectivity and identity. Nikolas Rose believes that neuroscientific discourses of human behaviour are creating "neurochemical selves" (Rose, 2003). That is, individuals are coming to understand their identity and behaviour as mediated by chemical occurrences in their brain. Sociological accounts of addiction have problematised such an emphasis on biology. They have noted the power relations inherent in reductive biomedical accounts of addiction, and prefer to describe drug use as a rational response by social actors (Weinberg, 2011). But as Weinberg has noted, in doing so, some sociological work on addiction has downplayed the sense of "viscerally felt compulsion" that is evident in the accounts of those who describe themselves as addicted to drugs (Weinberg, 2002). Weinberg recommends a post-humanist, empirical approach to addiction that acknowledges the "local practice" of addiction as an embodied experience (Weinberg, 2002, p. 2013).

There is little empirical research examining the psychological or behavioural impact of neurobiological understandings of nicotine addiction on smokers. A survey study by Hughes (2009) found that many smokers believed that an inability to quit smoking was due to addiction, but only a small proportion believed that biological factors were to blame. Hughes also found that smokers' causal beliefs were not strongly related to treatment preferences. He recommended that qualitative research be conducted in order to explore in more depth smokers' understanding of the causal determinants of addiction and their treatment preferences. Research from the genetics field has looked at the impact of genetic understandings of tobacco addiction on smokers' sense of control and treatment preferences (Cappella et al., 2005; Park

et al., 2011; Wright et al., 2003), however mixed findings and variations in study design mean that no clear conclusions can yet be drawn from this data. Moreover, it remains to be seen if people will respond to genetic and neuroscience information in similar ways, given important differences between the two (Green 2006).

This paper will report on qualitative research examining how smokers interpret and apply information about the brain and addiction to their own lived experiences as smokers. The aims are to:

- Explore the extent to which smokers believe their brain is involved in their smoking behaviour;
- Document the ways that smokers incorporate neurobiological explanations of addiction into their mental models of smoking; and
- Assess the ways in which brain-based understandings of addiction might influence smokers' sense of self-efficacy and their treatment preferences.

## **7.2. Method**

The data reported here are drawn from the initial qualitative component of a mixed-methods study examining neurobiological understandings of smoking and addiction. For this qualitative stage of the research, semi-structured interviews were conducted with 29 participants who smoked daily. Participants were recruited from a large metropolitan city in Australia. Because this was an exploratory study, purposive sampling was used in order to recruit a diverse range of participants in relation to age, gender, and education. The technique of maximum variation sampling was employed, with periodic reviews of the sample in order to ensure diversity (Patton, 1990). Means of recruitment included handing out flyers, advertising on mailing lists, and placing flyers at community centres and on noticeboards.

Participants were interviewed at a location that suited them and provided with a gift voucher in appreciation of their time. Interviews were conducted individually, except in one case where two relatives attended together and were interviewed concurrently. In order to introduce the type of research that exists on smoking and the brain, we prepared a short audiovisual presentation outlining findings on how tobacco works in the brain and its relationship to nicotine dependence. This was shown to participants on a tablet device during the interview. As NIDA are a major proponent of the brain disease model of addiction, the information we included in the slideshow was adapted from their publication aimed at teenagers titled "Mind

Over Matter: The brain's response to nicotine" (National Institute on Drug Abuse, 2006). This is a colourfully illustrated pamphlet which provides information about how tobacco works in the brain to produce addiction in easy to understand language. The fact that we provided information framed as "scientific" to stimulate discussion about the brain and smoking inevitably influenced the responses of participants to the questions that followed. In some cases it provoked discussions about the trustworthiness or otherwise of science and scientists. Also, we presented a "strong" version of the neurobiology of addiction, where the complexity of the interactions of neurobiology with other factors was not discussed. This method of providing what might be labelled a "short science explainer" for participants has been used in other studies to introduce a potentially unfamiliar topic to participants and to provide a stimulus for discussion (Buchman et al., 2013; Choudhury et al., 2012; Horstkötter, Berghmans, Feron, & De Wert, 2014). This method of eliciting participant responses to the same information can reveal the idiosyncratic ways that such information is interpreted and applied.

After viewing the presentation, participants were asked a series of questions about the role of the brain in smoking behaviour. While the interview guide allowed flexibility in questioning and sometimes varied according to the trajectory of individual interviews, typically the following questions were asked:

- Were you aware of the ways in which nicotine acts on the brain? If so, where did you hear about it?
- To what extent do you think that smoking affects your brain?
- How does this information influence your understanding of your smoking?
- Does it make stopping smoking seem easier or harder? How does it affect your belief in your ability to stop smoking?
- Does it affect your view of your personal responsibility for smoking? How?
- Does this information affect your view on the best method for quitting?

These interview questions were based on themes found in the literature on the effect of biological explanations on lay understandings of behaviour, as well as data from a previous study that looked at how members of the general public responded to information about the brain's role in addiction (Meurk, Hall, et al., 2013). The University of Queensland Human Research Ethics Committee granted ethics approval for this study.

The framework of thematic analysis was used to analyse the interview data (Braun & Clarke, 2006). Patterns observed in the data were labelled as codes using the NVivo 10 software (QSR International Pty Ltd., 2010) by the first author (KM). Another author (AC) independently developed codes after reading a selection of transcripts. A consensus approach was utilised, where differences in codes and themes were discussed and negotiated until a consensus was reached about the final coding scheme. KM then conducted a critical analysis of the coding scheme in order to collate the codes into the major themes that are presented here.

### **7.3. Results**

#### ***Participants***

We recruited 29 participants who were evenly distributed in relation to gender (M=15, F=14). Most participants were in the middle age ranges, with fifteen participants aged 26-54, nine younger participants (18-24 years olds) and five participants older than 55. Eleven had completed a university degree, ten had completed a trade certificate or diploma, four had completed secondary school and four had not finished secondary school. With regard to heaviness of smoking: seven participants were heavy smokers, smoking more than 20 cigarettes a day (CPD); 11 smoked 11-20 CPD; and ten smoked 1-10 CPD (missing  $n=1$ ). The Heaviness of Smoking Index (HSI) was calculated to measure the tobacco dependence of participants (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991). Each participant quoted below is categorised as having low, medium or high dependence based on the following cut-offs: 0-1: low dependence, 2-3: medium dependence, 4-6: high dependence (Balmford et al., 2011). Based on these categories, 10 participants were highly dependent on nicotine, nine were moderately dependent, and 9 had low dependence. We were unable to calculate the HSI for one participant due to missing data.

#### ***Awareness of neurobiological aspects of nicotine addiction***

Much of the speculation about the psychological impact of brain-based explanations of addiction assumes that people are currently unaware that their brain is involved in their addiction. They posit that neurobiological understandings of addiction will constitute an important *change* in the way that addiction is understood. Contrary to this claim, most participants reported that they were already aware that smoking influenced their brain. For them, the information presented about the neurobiological basis of addiction to smoking was not surprising, shocking or revolutionary.



*“I knew it goes to the brain and it activates something, but I just wasn’t aware of the terminology and things like that.” [Male, aged 26-40, low dependence]*

Only a small minority of participants reported having no knowledge that smoking affected the brain.

*“I did not realise that – I thought it was sort of more your lungs that it was related to, not your brain, if that makes sense.” (female, 26-40, medium dependence)*

Similarly, another young woman stated that: *“because I always assume drugs messes your head, whereas smoking was purely like physical, you know like lungs and heart and throat and stuff”* (female, 18-25, low dependence). This participant separates nicotine from other drugs by emphasising the physicality of smoking and the involvement of other (non-brain) parts of the body, in contrast to the mind-altering nature of illicit drugs. Because the psychoactive effects of nicotine are mild, and regular smokers do not experience any obvious intoxication, the brain does not become central in accounts of smoking.

For the majority of participants their knowledge of this link between nicotine addiction and the brain was often “fuzzy” or “vague” and most could not cite a specific source from which they had acquired this information. Their discussions about the role of the brain in smoking were characterised by qualifiers and filler words such “something” and “things like that”. Terms such as “chemicals” and “receptors” were used in imprecise ways. The role of nicotine was not well understood. Some smokers thought that nicotine was a depressant because they felt relaxed after smoking. Others thought that nicotine was responsible for the negative health effects that have been attributed to tobacco smoke. Only a handful of participants, mainly those who had studied biological science at university level, demonstrated a more sophisticated knowledge about how nicotine works in the brain. So while many participants knew the brain was somehow involved in addiction, few understood how it was involved.

*Like say with caffeine you get a headache and whatever so you sort of know all that. I wouldn't go into the technical bit with the receptors and all that lot but pretty much you've got to keep your levels. (male, 26-40, medium dependence)*

It was common for participants to state that the details about the way nicotine worked in the brain to produce dependence were “interesting”, or that this information offered an “insight” into their smoking.

*I guess it clarifies exactly what processes are going on inside your body in terms of why it gets a hold of you. Why it has that addictive properties. Why the withdrawal then happens and it's similar to things that I've heard of before. It's just massive over-stimulation of receptors then leads to them being so deadened that nothing affects them anymore. So you need to just keep going and keep going and keep going. It's basically understanding that physiological process makes it a bit clearer, but I still think that it is a choice to start and then once one is aware that they're addicted, it's not really something you can turn around and say well it's not my fault my brain told me to do it. (female, 26-40, high dependence)*

As the above quote shows, insight into the biological basis of nicotine addiction does not necessarily influence ideas about the fundamental nature of smoking. In this participant's case, and that of many others, smoking is framed in terms of personal choice and autonomy. Also, not all participants found the link between smoking and the brain interesting or noteworthy. One questioned the significance of the brain's role by pointing out that *"anything - absolutely anything that you do and you do repetitively and for a long duration of time affects your brain, anything."* (male, 26-40, medium dependence)

The focus of the remainder of this paper is on whether neuroscience findings about addiction might result in anything other than curiosity value amongst smokers.

### ***The brain and quitting methods***

The claim that biological explanations of addiction will increase the use of, and adherence to, medication assumes that people's explanatory models of addiction correspond in predictable ways to the treatments they choose: it is assumed that people who have biological understandings of their addiction will be more likely to seek assistance from health professionals and use medications, Conversely, those who believe their addiction is a social or psychological problem may see less value in using medications and therefore be more likely to quit unassisted or to favour psychological or behavioural approaches.

Smokers interviewed for this study often stated that neuroscientific accounts of nicotine addiction might inform the cessation strategies that they, or other smokers, would employ.

However, the ways in which they thought it would inform quitting tactics varied widely, as illustrated by the contrasting quotes below:

*“It’s just - like, the neurotransmitter - it’s like playing sport. When you smoke a lot you get really good at it, and then so what you’re trying to do, what I’m trying to do when I quit is to get bad at it, try and reverse it. So I’m just trying to maybe go cold turkey, slow it down, try and push myself.” (male, 18-25, low dependence)*

*“I suppose if it’s so entrenched within your system and sometimes we do need a helping hand. Yes we want to go cold turkey but maybe there’s a point where we can’t and it pretty much consumes us. So we may need to go to a doctor and get prescribed medication to kick the habit.” (female, 55+, high dependence)*

These two individuals come to very different conclusions about the implications of addiction neuroscience for quitting methods. The first participant acknowledges that smoking causes changes to the brain, but believes the brain can also be returned to its original form without medical assistance. All that is needed is to “get bad” at smoking by weaning off or stopping smoking. This is consistent with the idea of brain plasticity, where individuals can “work on” or “improve” their brain by way of brain exercises or other behavioural alterations. The concept of brain plasticity has become a culturally salient concept that is widely reported in the media and popular science (Pickersgill, Martin, & Cunningham-Burley, 2015; Pitts-Taylor, 2010), so it is not surprising to find it used by smokers to understanding quitting. However, for the second participant, the role of the brain implies an “entrenchment” of addiction that can only be overcome using medical intervention.

Attitudes toward the use of the cessation medication varenicline reveal the diversity in lay representations of neuroscience knowledge. Participants were provided with a brief description of how varenicline (trademarked as Champix in Australia) works in the brain. More than one third of participants were unaware of the existence of prescription medications for smoking cessation prior to the interview, however even the few participants who had used varenicline seemed unaware of how it worked in the brain. Most of those who discussed varenicline reported that knowing how it worked in the brain increased their positive perceptions of it:

*“I think the information about Champix filling the receptors, like that's good information. I think it's just the side effects I don't like about Champix.” (female, 18-25, medium dependence)*

However, having positive perceptions of cessation medications, or believing that they “could” be a good idea, did not mean that they were a preferred quitting method. A fear of side effects was common. Also, many participants, particularly those from younger age groups, expressed a preference for unassisted quitting.

*“Well yeah, I still think the best method is habit change but if they perfected Champix to not do everything else that it does side effect wise and was just filling receptors to help with withdrawal then I think that would be great.” (female, 18-25, medium dependence)*

For a smaller number of smokers the idea that Champix worked in the brain made it *less* likely they would use it. They were concerned that Champix would interfere with the normal function of their brain and described this possibility as “scary”. For example, one participant stated that *“I knew Champix did something scary, chemical stuff going into your brain, don't like it”* (female, 26-40, low dependence). Another thought that if Champix reduced feelings of pleasure for smoking, then it may do the same for other pleasurable experiences:

*“I didn't realise actually what Champix did either. It actually stops the receptor - I don't like that. I don't like that at all...it's playing around with your brain, if that makes sense and I don't know, I know this might sound silly but if you – I don't know, because sometimes you eat something and it feels good. Is that going to stop also that pleasure as well?” (female, 26-40, medium dependence)*

The association between cessation medications and the brain was not the only reason for disliking medication for cessation. Negative attitudes towards pharmacological cessation aids reflected a general aversion to taking medication. There was a belief, again expressed mainly by younger adults, that medication should be taken only when absolutely necessary, and that quitting smoking was not serious enough to warrant it.

*“I'm always against medical treatment. Even if I have a small headache, I never go and take the medicine. If I have a bit of cold, I never take any tablet or anything. I just try and let it go with the time.” (male, 26-40, low dependence)*

The contradiction inherent in being frightened by the effects of pharmaceutical medications whilst consuming a harmful product (tobacco) that contains a psychoactive drug (nicotine) was not lost on all participants. The participant quoted below noted it, but it remained unresolved:

*“I knew Champix did something scary, chemical stuff going into your brain, don't like it. I mean I'm fully aware that nicotine's going into your brain but I don't know, scary stuff.” (female, 26-40, low dependence)*

Even among those positively disposed to using pharmacological treatment for smoking cessation, the importance of psychological factors was often emphasised. The use of terms such as willpower, mindset, and “readiness” to quit were very common throughout many interviews. For example, smokers who had tried many times to quit, including attempts using pharmacological cessation aids, attributed their failure to a lack of willpower, or a belief that they weren't yet “ready” to quit. The smoker quoted below acknowledges the availability of medications but does not believe that this negates the need for the individual to exert “effort” in their quit attempt.

*“There's medications available. What do you think personally is going to work for you? We can try a range of things here. But the effort has to come from the individual regardless.” (female, 55+, high dependence)*

### ***Autonomy and feelings of fatalism***

Participants in this study were asked whether knowledge about how nicotine worked in the brain to produce addiction affected their beliefs about their ability to quit. The most common response was that it made quitting smoking seem easier. Some thought that emphasising the role of the brain in addiction made smoking seem more serious, thus increasing motivation for quitting. One participant, who had not known that smoking affected the brain, stated that:

*“I guess it hasn't made it harder for me to stop smoking, I guess it's made it easier in the respect that I guess I'll think twice before I reach for a cigarette.” (female, 26-40, medium dependence)*

A number of participants thought that knowing how varenicline worked made it seem more effective, and consequently made quitting seem easier.

*“I suppose understandings how the, you know Champix and things like that are supposed to work, makes it seem more likely that it would work. Yeah, so I guess easier.” (female, 41-54, medium dependence)*

A few participants thought that knowing exactly how nicotine worked in the brain was empowering. They equated a greater amount of understanding about smoking to a greater sense of control over their smoking.

*“The more understanding I have of it the more I have control of it. That’s how I feel. I think I have more control over the smoking now that I know what I’m doing.” (male, 18-25, low dependence)*

Not all believed that an awareness of the neural basis of addiction would lead to a greater sense of control over smoking. More than a third said that emphasising the role of the brain in tobacco dependence made it seem more difficult to quit smoking. Some commented that it made their addiction to tobacco seem more “real.” This coincided with a belief, expressed by several participants, that nicotine addiction was less serious and easier to cure than addiction to other drugs. They expressed shock at comparisons between the similar effects of nicotine and illicit drugs on the brain.

*“You know it shocks me to watch it and I’m like oh gosh, you’d think that would make you want to stop, but it almost makes me think, no I am addicted. You know whereas before I just feel like, no I could stop.” (female, 18-25, low dependence)*

As can be seen above, emphasising the role of the brain in nicotine addiction did lead to fatalistic responses in some participants.

*“So it’s something that I can’t control, it’s something that the brain’s just automatically doing. So that’s going to make it even harder, not am I just fighting this, I’m actually fighting something that’s chemically happening.” (female, 26-40, high dependence)*

This individual speaks of “the brain” as if it is separate from her, and believes that it operates “automatically”, independently of her own desires and motivation. For this smoker, an awareness that “chemicals” are involved implies that there is more to smoking than just individual choice.

It is important to note that there were also smokers who were dismissive of the idea that neuroscience information on addiction would have any effect on the way they perceived their smoking. These people often stated that knowing how nicotine worked in the brain provided some level of insight into their smoking, but it was mainly a “matter of interest” that would not affect their smoking behaviour or attitudes.

*“I'm very sort of intellectual in my thinking, so it's sort of oh that's nice to know. I'm glad I know it. It's not so much that that's going to affect my smoking.”*  
(female, 26-40, high dependence)

Participants were also asked whether knowing how addiction occurred in the brain affected their sense of personal responsibility. Almost all participants in this study strongly rejected the idea that an awareness of the neurobiological basis of tobacco dependence would influence their sense of personal responsibility for their smoking.

*“No, because I'm not going to say that - for want of a better term - I'm fucked in the head now, so I'm not responsible for my actions.”* (male, 26-40, high dependence)

Only a single participant thought that knowing about the brain’s role in addiction would reduce self-blame, and even here, it was framed around this discourse being useful for other smokers, rather than for herself.

*It's not necessarily your fault that you're finding it difficult to quit smoking when you have changed your physiology through what you've done.* (female, 26-40, high dependence)

Predicting the effects of raising awareness in smokers of the neural mechanisms of nicotine addiction will be difficult, and there may be unintended consequence such as increasing feelings of fatalism. One participant discerned the contradictory effects that may arise from brain-based understandings of smoking, saying that it make quitting seem:

*“A little bit harder. It makes it seem more technical. Then at the same time you're just like okay well, if that's what this does, then you can do this and that and formulate something to replace it, which is like nicotine or Nicorette. Both ways, I guess.”* (female, 18-25, low dependence)

## **Emergent themes**

Three emergent themes recurred throughout the interviews. These were 1) the brain as a special organ and 2) the construction of the “other” smoker, and 3) nicotine addiction versus smoking as a habit.

***The brain as a special organ.*** Many participants were concerned at the idea that drugs would interfere with the “normal” functioning of the brain. This concern applied to both recreational drugs such as nicotine in the form of cigarettes, and to pharmacological cessation aids that influenced the neurochemistry of the brain to reduce cravings and relapse. Outside influences on the brain were perceived as being harmful, with several participants describing drugs as “messing with” or “screwing with” the brain. This was of particular concern for the participant quoted below, who explicitly linked their brain with their identity. The brain is described as a special organ, because unlike other organs, it contains the “essence” of an individual.

*“It's a lot more terrifying than having lung cancer and I know that sounds terrible but it just is. It's my brain and we all sort of associate it with it's our essence, it's us, you know, yeah.” (female, 26-40, low dependence)*

Others thought the brain was special because it is so vital to the functions of everyday living. These participants were not concerned with addiction, but were concerned that brain “impairment” that would lead to a loss of function.

*“Well I guess from an intellectual point of view I don't want my brain any further impaired than it already is. I'm fairly reliant on it. Not a good scenario.”(female, 55+, high dependence)*

Physical illnesses were contrasted with illnesses of the brain, because the latter afflicted the mind – *“I don't know, and it shouldn't be because physical illnesses are bad as well, but you always worry about your brain don't you?” (female, 18-25, low dependence).*

***The construction of the “other” smoker.*** Participants very frequently contrasted their own smoking with that of other smokers by distinguishing themselves from other smokers on a number of dimensions. For example, smokers were described as differing by personality traits. Many commented that the impact of brain-based understandings of smokers would vary depending on factors such as a smokers’ sense of personal responsibility, or the amount of willpower and motivation they had:



*“But it does very much depend on personality type. There's defeatist people out there that just accept and don't say well okay that's critical information. That just means that whatever I choose in order to give up once I'm committed to it then these things have to be more strongly in place because it's going to be a tough battle.” (female, 55+, high dependence)*

There was a tendency for some participants to position themselves as better educated and more knowledgeable smokers, who would interpret neuroscience information in the “correct” way. Other uneducated, or “unintelligent” smokers would misinterpret material emphasising the role of the brain.

*“If you're not intelligent enough to understand it for what it is, then you might go, oh, well, then my brain is rewired now. I can't. I can't quit. There's a lot of people out there that are not thinking on the same level that we are, so too much information in their hands - when they don't understand that information properly - is useless to them.” (male, 26-40, low dependence)*

Several smokers made the distinction between those who were physically addicted to nicotine and those who were psychologically addicted to smoking. When asked whether educating smokers about the neural basis of addiction would be helpful, one responded:

*“...there are different kinds of smokers and each one has a different kind of addiction, you know so I mean this would be really helpful to those who are addicted to cigarettes just because of the nicotine, but you wouldn't help the vast majority.” (male, 18-25, medium dependence)*

For those smokers who did not believe themselves to have a physical addiction to nicotine, information about the neurobiological mechanisms of addiction was not seen as personally relevant.

*Yeah, I mean I don't think I've gotten stupider or anything but, and that's the other thing I don't, I smoke really light milligram cigarettes and I don't smoke a lot of them so yes I am addicted to the habit and I'm fully aware that I am, I'm not in any kind of denial there, I don't know how much nicotine, you know, like they're talking about unpleasant withdrawal symptoms and that stuff. I don't really get any withdrawal symptoms if I don't smoke. (female, 26-40, low dependence)*

It was seen as interesting information, but more applicable to heavier, or more physiologically addicted smokers.

#### **7.4. Discussion**

The primary aim of this research was to explore how smokers engaged with ideas about the neural basis of nicotine addiction. Many of our participants had some awareness that smoking tobacco affected their brain. However, few were aware of the detailed neurobiological mechanisms associated with addiction to smoking, and some were misinformed. This is consistent with other literature on NRT that has found that smokers do not have a good understanding of how nicotine works in the body, and often hold erroneous views about its safety due to misunderstandings about the mechanisms of its actions (Ferguson et al., 2011; Mooney et al., 2006). This inexactness of smokers' knowledge about smoking, nicotine and the brain is not surprising. We live in a time with a rapidly expanding corpus of scientific findings relevant to health, and laypeople cannot be expected to keep up to date with all scientific developments, even those that are personally relevant to their health. It is likely that exposure to ideas about the neuroscience of nicotine addiction will add detail to smokers' existing understanding of smoking, rather than revolutionise the way that people conceptualise their smoking.

Research in other fields indicates people are more likely to incorporate neuroscience information into their existing models of human behaviour, than to radically alter their mental models to account for biological research findings (Choudhury et al., 2012; O'Connor & Joffe, 2013; Pickersgill, 2013). Our findings are consistent with this, in that most participants did not think that an emphasis on the neurobiological basis of nicotine dependence would change their own treatment preferences or their smoking behaviour. Also, the claim that educating smokers about the biological basis of addiction will reduce their feelings of self-blame and lead to them absolving themselves of responsibility for their smoking by blaming their brain is strongly disputed based on the findings from this study. The participants were adamant that they remained personally responsible for their smoking. They believed that knowing the role the brain plays in creating and maintaining addiction should not negate the need for smokers to expend effort in their quit attempts and take responsibility for their actions. Many thought that an awareness of the neurobiological basis of addiction *increased* an individual's responsibility to stop smoking. Partly this was due to the perceived "special" nature of the brain. The brain

was seen as a vital organ, and just knowing that it was being affected by smoking should motivate people to quit. This may somewhat be explained by the fact that the discourse of neuroplasticity infiltrated the accounts of some participant around smoking and the brain. The idea of brain plasticity was used by these participants to counter messages about the long-term “chronic” changes in the brain that lead to addiction being labelled a “relapsing disease.”

A common concern regarding the BDMA is that if addicted individuals believe that their addiction is “hard-wired” into their brain, they may develop a sense of fatalism. Whilst some smokers mentioned this possibility in our interviews, they mostly thought that this would occur in other smokers, rather than themselves. Smokers in this study maintained their sense of autonomy in the face of brain-based understandings of addiction by setting themselves apart from “other” smokers. They imagined that other smokers were more heavily addicted or had a physiological addiction to smoking. Also, they sometimes positioned themselves as more educated and informed about smoking than others. It was a common theme that pharmacological treatments for nicotine addiction were suitable for “other” smokers, those who had a strong physiological addiction. A number of the smokers in this study, particularly those who were younger, stated that they did not have a physiological addiction to cigarette smoking, or that their addiction was mild compared to others. Therefore they did not see their own smoking as serious enough to warrant the use of medications. While they identified as being addicted, they conceptualised addiction differently, seeing it as a “behavioural” addiction or a “habit.” This supports the finding of Hughes (2009), who found that it was common for smokers to agree that an inability to quit was due to both addiction and to habit, but not to biology. Others have found that young people in particular believe themselves to be less addicted than other smokers (Weinstein et al., 2004).

The BDMA highlights the addictiveness of smoking, which is portrayed as a “chronic” condition that requires treatment, much like alcoholism (Wolff et al., 2013). There is very limited evidence that explores whether belief that one is addicted to smoking impacts in any way on quit attempts or treatment choices (West, 2011). Our research demonstrates that explaining how prescription medications worked in the dependent brain increased positive perceptions of cessation aids for some smokers, but there were suggestions that this would not necessarily equate to increased uptake of pharmacological cessation aids. This is a very complex area to negotiate because it is possible that this emphasis on addiction will conflict with public health campaigns that aim to increase quitting self-efficacy in smokers, and also

with lay understandings of smoking and cessation. For some of our participants, highlighting the role of the brain did make tobacco addiction seem more real and serious, so this possibility cannot be entirely discounted. Overall though, our research suggests that emphasising the biological basis of addiction will not lead to radical changes in smokers' confidence in their ability to quit smoking.

Because this is qualitative research, the results are not generalisable to the entire smoking population. Large quantitative studies are needed to assess the generalisability of our findings and to look at differences in relation to age, gender, level of education and heaviness of smoking. Also, it should not be assumed that users of illicit drugs or alcohol would respond in the same way as smokers because tobacco is a drug with a unique history (Berridge, 1997) and users of other drugs may have very different ideas about the role of the brain in their drug-taking.

A potential limitation of this study is that participants were presented with a specific interpretation of neuroscience research on smoking. While the goal was to stimulate discussion, there is the potential for such information to constrain the expression of ideas about smoking and the brain if participants simply agree with the view of nicotine addiction presented to them. This might occur due to a deference to scientific authority, or because participants feel they do not have time to process and interpret the information. Moreover, participants may feel an expectation to respond to these ideas as if they were novel and new. Martyn Pickersgill (2013) has noted a tendency amongst social scientists to overplay the novelty or significance of neuroscience while members of the public are more likely to treat it as an object of "mundane significance." That is, they may ignore it, acknowledge it, or find it "entertaining, rather than profound" (Pickersgill, 2013, p. 330). While our results were generally consistent with participants treating this information as having "mundane significance", a minority of smokers expressed shock at the "new" information about their smoking that had been presented to them, and believed that it might influence their ongoing behaviour.

It is important to anticipate the way in which those who experience addiction as an everyday reality will interpret neuroscience knowledge about addiction (Fry & Buchman, 2012). Our analysis reveals that providing smokers with information about the neurobiology of nicotine addiction can lead to complex and contradictory reactions. For many of our participants, the provision of information about the role of the brain in tobacco dependence provided insight

and interest, but they did not think it would affect their pre-existing beliefs or intentions. Our data suggest that neuroscience information is more likely to influence people's beliefs about *other* smokers than it is to modify explanations of their own behaviour. However, the results also pointed to the possibility that such information may make quitting smoking appear more difficult for a minority of smokers.

Because of the wide variety of ways that neuroscience knowledge about nicotine addiction is interpreted by smokers, it may be better to present this information in a clinical setting rather than in widely disseminated health promotion materials. Health professionals in clinical settings can gauge the attitudes of the individual smoker in response to more “medicalised” explanations of smoking and correct any misperceptions they may have. Where it is included in educative material for smokers, neuroscience information should be presented in a way that does not negate people's sense of agency and self-efficacy in relation to quitting smoking.

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### **7.5. Concluding remarks on Chapter 7**

When presented with information about how smoking influences the brain in ways that make it difficult to quit, smokers respond in a variety of ways. Firstly, some negate the significance of this neuroscientific information. They report already being aware of it, or observe that “everything affects the brain”, so why should smoking be any different? Some believe this information is relevant to other smokers, but not to themselves as they were not addicted to nicotine; or were less addicted than other, heavier smokers. Separating oneself from the “heavy smoker” identity was frequent, particularly amongst young people in the sample.

As with Chapters 5 and 6, individual responsibility, willpower and motivation were again prominent themes and describing the neuroscientific basis of nicotine addiction did not negate their importance. It is clear that smokers are very averse to the idea of absolving themselves (or others) of personal responsibility for smoking by taking up a neuroscientific discourse about smoking. Steenburgh and colleagues (2012) reported similar findings in drug addicted individuals and recommended that neuroscientists acknowledge that a sense of personal responsibility is an important factor in ceasing drug use. As has been noted by others, drug addiction may lessen self-control, but does not eliminate it (Carter & Hall, 2007; Hyman, 2007).

The impact that neurochemical explanations of smoking and cessation will have on attitudes towards cessation medications remains unclear. These findings reveal a tendency for information about the neuroscientific mechanisms by which medications work to increase positive attitudes towards them. Whether this will influence the actual use of medications is unknown. Even where positive attitudes towards medications seemed to increase when the mechanisms of their action in the brain were explained, the participants mainly maintained a preference for unassisted quitting methods. Moreover, a small number of participants rejected the use of varenicline precisely because it worked on the brain. The brain is often described as the location of one's identity, and the idea that something may be "messing" with it was disturbing for some participants.

The general disinclination to take medications expressed by numerous participants is relevant to sociological discussions around pharmaceuticalisation. Pharmaceuticalisation has been defined as "the process by which social, behavioral, or bodily conditions are treated, or deemed to be in need of treatment/intervention, with pharmaceuticals by doctors, patients, or both" (Abraham, 2010a, p.290). There is some debate about whether pharmaceuticalisation is one aspect of medicalisation or a separate but linked process. Abraham posits that pharmaceuticalisation is a separate process but that increasing medicalisation is "a significant factor in explaining increased pharmaceuticalization" (2010b, p.608). He argues that increased use of pharmaceuticals is not simply a result of improving access to effective medications, but has been facilitated by pharmaceutical industry interests influencing the development of diagnostic criteria and treatment guidelines, as well as exaggerating the benefits of various drugs.

Pharmaceuticalisation is strongly linked to medicalisation in relation to smoking cessation pharmacotherapies. Most smokers who are eligible to use cessation pharmacotherapies are at risk of diseases from smoking, rather than suffering from such diseases. This means they may dismiss the need for medications, which are seen as too serious for their smoking "habit." (See Chapter 6). This is particularly the case where the potential for negative and serious side effects have been publicised, as they have been for varenicline. While "tobacco use disorder" is listed in the DSM as a diagnosable condition, the uptake of this term and the use of diagnostic criteria for smoking has been limited in clinical practice. This may have influenced the shift amongst

some organisations to begin to describe nicotine addiction as a health condition in its own right by emphasising its biological basis in the brain. This explanation of addiction justifies the use of medications that work in the brain to alter the neurochemical pathways related to drug dependence. Whether this strategy is successful remains to be seen. The research presented here suggests that understanding the biological basis of prescription medications for smoking cessation could improve attitudes towards varenicline. But there is also the potential for this strategy to backfire amongst those who do not want medications “messing” with their brain. Moreover, the strength of alternative explanations of smoking centred around willpower and motivation are likely to attenuate the effects of describing nicotine addiction as a brain-based disorder.

As described in Chapter 3, much work on lay knowledge of medicine or illness, as well as findings from the field of public understanding of science, show that scientific findings are not uncritically accepted, but are subjected to various forms of critical appraisal. One means of appraisal is to compare the information provided by experts to everyday experiences, or what is dismissively referred to by some as “anecdotal data.” This is what Davison, Smith & Frankel (1991) described as “lay epidemiology”, and this process has been demonstrated in this study and in others in relation to lay understandings of smoking and cessation (Smith et al, 2015).

Another way that such information is judged is according to the credibility of the sender. In PUS literature this has centred around the topic of trust. How do laypeople judge the credibility of those who are attempting to influence the ideas and opinions of various public stakeholder groups? And how does scientific information and its presentation influence lay attitudes towards certain scientific claims? Based on qualitative research with Cumbrian sheep farmers and their engagement with scientists following the fallout from Chernobyl, Wynne (1992) outlined a number of “lay criteria” which people used to judge the credibility of scientific claims. These include whether the scientific knowledge works in practice, whether scientists have taken account of all available knowledge, the extent of engagement with “lay experts”, the affiliations of the scientists, and whether the format of the information is accessible and recognisable to laypeople.

Because the information provided to participants in this study about nicotine dependence and the brain was presented as “scientific fact,” it is likely participants were making judgements about the credibility of the information and the motives behind its presentation. However, the

information in the Powerpoint was portrayed as disembodied knowledge, rather than being attributed to a particular scientists or interest group. (E.g., “When you smoke tobacco, nicotine enters your bloodstream” , “Research shows...”). This may have made it difficult for participants to critique the validity or trustworthiness of the information. Most participants did not question the accuracy of the information presented, although some thought it did not portray the “whole picture” of smoking. However, in the next chapter the role of scientists are brought to the foreground, resulting in explicit discussion about their credibility and motives.

Overall, these findings reveal the complex way that information about the neuroscientific mechanisms of addiction is negotiated in relation to smokers’ views on smoking and quitting. Assumptions that it will induce fatalism, increase treatment seeking, and reduce a smoker's sense of personal responsibility, are not supported by these findings. This is consistent with research by O’Connor (2013) who found that while the brain was often associated with a person’s essence or soul, this did not negate the importance that people attached to free will in explaining human behaviour. The evidence presented here begins to outline the complex and contradictory ways that neuroscientific information is incorporated into everyday understandings of addiction and smoking.

#### **7.6. Original contribution of Chapter 7**

Speculation about the impact that neuroscientific research findings might have on lay understandings of various disorders and behaviours is rife. However, as has been noted by others, much of this work is “over theorized” (Pickersgill et al., 2011, p. 362) and not based on empirical evidence. In relation to addiction studies, Steenburgh (2012) has written that “[a]s addiction studies benefit from what can be learned from neuroscience, the neuroscientific study of addictions might also benefit from considering what can be learned from the practical experience of addiction counsellors and individuals struggling with addiction.” (Steenbergh et al., 2012, p. 427).

This is the first research project to examine how Australian smokers understand the role of their brain in their smoking. It is important to document how smokers understand their smoking in light of new treatment options and advances in scientific knowledge. For most participants in this study the idea that smoking influences the brain was not controversial, novel, or influential. Perhaps more controversial and more novel is the description of tobacco



dependence as a “chronic disease” (Fiore et al., 2008) or a “chronic brain disorder” (Prochaska & Benowitz, 2016). The next chapter examines the reactions of the same participants to the statement that smoking is a “brain disease”.

## **Chapter 8. “I’m already bad enough” – A qualitative exploration of smokers reactions to labelling nicotine addiction as a brain disease.**

As described in Chapter 2, the controversial definition of addiction as a “chronic, relapsing brain disease” is beginning to be applied to smoking. In the interviews reported in Chapter 7, smokers' knowledge and attitudes regarding the role of the brain in smoking behavior was explored prior to introducing the term "brain disease". This allowed elicitation of their views about the role of the brain in smoking, rather than their views on the *label* of “brain disease”. The results described in Chapter 7 revealed that most participants accepted that smoking affected their brain but this did not typically have a large impact on the way that they understood their smoking or thought about stopping smoking. Subsequently, participants were questioned on the extent to which they agreed that smoking is a brain disease. The reasons they agreed or disagreed were analysed, and are reported in this chapter.

### **8.1. Introduction**

The main tenets of the BDMA have been described in Section 2.6. Proponents of the BDMA argue that it will reduce feelings of self-blame in addicted individuals, reduce the stigma associated with drug dependence, lead to the development of more efficacious treatments, and promote more sympathetic policy responses to drug addiction (Dackis & O'Brien, 2005; Gardner et al., 2009; Leshner, 1997). The BDMA portrays addiction as more than simply a poor choice or a moral failing (Leshner, 1997) but as a loss of control over behaviour that is driven by persistent neurobiological changes produced by chronic drug use that drives continued drug use despite the harm that it causes.

The BDMA has been criticised as being overly essentialist, downplaying the complex interplay between biology, psychology, social environment (e.g., drug availability), and individual upbringing (Caan, 2012; Kalant, 2010; Midanik, 2006). In the case of smoking, for example, the tobacco industry has promoted the idea that smokers are genetically predisposed to be harmed by smoking as a way of deflecting blame from their unsafe product (Gundle et al., 2010). Other critics argue that the BDMA undermines free will and agency (Heyman, 2009; Lewis, 2015) in ways that may make stopping smoking seem more difficult than it is. This may

discourage smokers from attempting to quit without using smoking cessation medicines (Caron et al., 2005; Chapman & MacKenzie, 2010; Pierce et al., 2012).

The claim that the BDMA will reduce stigma has been questioned (Buchman, Illes, & Reiner, 2011; Trujols). Some research suggests that labelling those who experience mental illness as having different and problematic types of brains, can lead to an increase in stigma (Angermeyer, Holzinger, Carta, & Schomerus, 2011; Kvaale et al., 2013). In the case of smoking, the issue of stigma is complex. The stigma associated with alcohol and illicit drug addiction is generally seen as negatively impacting upon treatment-seeking, drug use and self-esteem. In contrast, the stigmatisation of smoking is often viewed positively, under the banner of “denormalisation,” which encourages smokers to quit and reduces uptake by young people. An explicit example is the use of shame messages in anti-smoking advertising (Amonini et al., 2015).

Scholarly debates about the accuracy and value of the BDMA have rarely been informed by the voices of the individuals who are most directly affected by this framing: those who are substance dependent. Commentators from various fields have postulated that advances in knowledge about the neural correlates of behaviour will change the way that laypeople understand their own actions. This chapter explores how smokers respond to the claim that smoking is a brain disease and how useful they find the brain disease label in understanding their own smoking. Is this definition of addiction acceptable to them, and how applicable is it to their own smoking? Do they believe that this way of defining addiction will reduce stigma, or change the way that smoking is understood in helpful ways? We also look at how smokers make judgements about the credibility of this neuroscientific claim.

## **8.2. Method**

The data presented in this paper are drawn from the same interviews described in Chapter 6 and 7 and their demographic data can be found in Section 6.3. As described in Chapter 7, during the interview, participants were presented with a short slideshow outlining the ways that nicotine acts in the brain to produce dependence. Participants’ general understanding of the role of the brain in their smoking and its relevance to treatment choices were reported in Chapter 7. The interviewer then made the following statement: “Based on the information presented earlier, some scientists have claimed that addiction is a brain disease.” Respondents were asked what they thought about the idea that smoking was a brain disease, and whether

such a label would increase or decrease the stigma associated with smoking, whether it was good or bad way to describe smoking, and whether they had heard this definition of addiction before. It is important to note that this research was not designed to examine existing attitudes towards the idea that smoking is a brain disease. Because application of this term to smoking has only begun recently, it was assumed that most participants would not have been exposed to it and therefore they would not have pre-existing attitudes that could be prompted. Rather, our aim was to elicit smokers' initial reactions when presented with this view of the scientific evidence.

Again, thematic analysis was performed to analyse the results (2006). After close reading, initial codes which represented patterns observed within and across interviews were generated by the author. These codes were then combined into themes when they were deemed to represent a broader topic. The approach taken combined inductive and deductive processes. Some themes were included in the interview schedule as they were pre-identified from an exploration of the literature, other themes were identified as the data was being analysed. Adrian Carter conducted independent coding and labelling of themes from a subset of transcripts in order to ensure the validity of the themes. Any discrepancies were discussed until a consensus was reached about the final themes. All data was stored and managed using NVivo 10 software (QSR International Pty Ltd., 2010).

### **8.3. Results**

Our analysis revealed three primary considerations that participants referred to when reflecting on the claim that smoking is brain disease: 1) the accuracy of the "brain disease" terminology; 2) the potential social consequences of labelling smokers with a brain disease, and 3) the likely social utility of the description. While some participants focused primarily on one of these, it was more common to use a mixture of these considerations in forming an overall judgement on the acceptability of using the BDMA to understand tobacco smoking.

The idea that smoking is a brain disease was new to most participants. Some had heard this terminology in relation to other drugs, but had not heard it applied to nicotine addiction. Because this was a question they had not previously considered, participants' initial responses were often uncertain and questioning:

*A brain disease? A brain disease? Interesting. People seeing addiction as a brain disease. I don't know what to say to that. I mean, define disease, you know what I mean? (male, 18-25, low dependence)*

Most were, nonetheless, able to reflect on the claim and make some judgements about its value. Their views often evolved in the course of the interview as they talked through the implications of this terminology. Some people who initially agreed that smoking could be a brain disease later disendorsed the idea and vice versa.

### ***Evaluating the accuracy of the “brain disease” explanation of smoking***

Many participants disagreed with the suggestion that cigarette smoking was a brain disease. Participants had diverse views of what they thought the term “disease” meant. One referred to a disease as something that is “degenerative and harmful.” (male, 26-40, moderate dependence). He did not think that nicotine addiction would fit this description unless it was “destroying” brain cells. Another thought that diseases were “viral” (male, 18-25, low dependence). A disease was also seen as “something that changes in your blood cells and deforms something.” (female, 26-40, high dependence). Others associated the term brain disease with physical damage to the brain. Some participants rejected the idea that their brains were damaged, citing as evidence that they were fully functioning members of society.

Some did not like the term but struggled to articulate why. They said that it didn't “sound right”, or that it was a “weird” label to apply to smokers. These participants reacted most strongly against the word “disease”. The fact that nicotine may change the brain of smokers, however, was not disputed by most participants.

*Yeah, I agree that okay I'm addicted to nicotine and yeah, it affects my brain, I agree with all that but no, I don't think, not as a disease, no. It's just something I guess you've got to work on every day. (female, 26-40, moderate dependence)*

A number of those interviewed suggested using different language to describe nicotine addiction. One thought that “disorder” was a more appropriate term because “disease” referred to something “viral”, whereas smoking was more like a mental disorder such as bipolar disorder, “but a very minor grade of the same league.” (male, 18-25, low dependence). Significantly, another suggested the term “illness” as an alternative, because an illness is

something that you can recover from, whereas a disease suggests chronicity and the need for long-term management, which was not consistent with his understanding of his smoking.

There were a number of common beliefs that affected participants' preparedness to accept or reject the idea that smoking was a brain disease. The most common was that a disease was something that an individual had no control over – it was perceived as something that “happened” to people. As one participant put it, it is “something that’s caused by an outside influence.” (female, 26-40, high dependence). This was seen as being inconsistent with smoking, which was conceptualised as the result of a choice. The brain disease explanation of smoking was rejected because it was perceived as downplaying or ignoring the central role of choice in smokers' understandings of their smoking.

*See, the thing is I could stop smoking right now. I could throw my ciggies in the bin and decide, no, never ever ever having one again. I mean, yeah, it's going to be a hard decision to make, but I can make it. I can do it. It's just I'm choosing not to. Anyone who disagrees is either an idiot or they're irresponsible (male, 26-40, low dependence)*

A number of people thought that the term “disease” was too serious to apply to cigarette smoking. One participant thought that it would “make it look probably worse than what it is” (female, 18-25, low dependence). The role of nicotine addiction in sustaining smoking was downplayed by comparing smoking to other “habits”:

*I think disease is a little harsh, I would think. I would say it's a - what was that word? It's not disease, it's - I lost the word. But I wouldn't say it's a disease. It's something that your brain is accustomed to, just like how you're feeling - you know, like when you're driving how you check the mirrors, when you wake up how you do your hair, you brush your teeth. It's something that your brain picks up and learns. So what you can learn, you can unlearn. But I wouldn't go so far as to call it a disease. It's just harsh. (male, 18-25, low dependence)*

This quote emphasises that smoking is something that you can “unlearn” or overcome. Diseases are perceived as afflictions that individuals have no control over – you cannot stop having a disease, but you can decide not to smoke your next cigarette. This view was supported by the observation that people could quit smoking without assistance.

*But then again, how is it then that some people can still stop if it's affecting - the heavy smokers - there have been heavy smokers that have stopped, so how is it that they've been able to do that if their brain - or is it like a lot of diseases, some people smoke, smoke, smoke and never get lung cancer, other people never smoke and get it. Yeah, it's probably a too general a hypothesis. (female, 55+, high dependence)*

A minority of those interviewed agreed that smoking was a brain disease. One reason for accepting this view was the invocation of a predisposition toward addiction in some individuals. For these participants, the fact that addiction ran in families, and individuals differed in their ability to quit, suggested that smoking was “more than a habit”. Rather, it was more a behaviour with a physiological and possibly genetic basis. These considerations made the brain disease concept more believable to them.

*I've been in a few rehabs, psych hospitals. The people that have problems, the people that don't, that people that keep coming back over and over again, the people that get treatment and go on to be okay and then the people that just never have an interest in using drugs at all to start with. It really seems to vary from person to person. For someone like me I cannot comprehend how someone can be a social smoker and just on the weekend I have a couple of puffs - you what? I don't understand how that's possible. I don't have that kind of physiology. I don't have that make-up. (female, 26-40, high dependence)*

Some participants thought that nicotine addiction might partially be understood as a brain disease, but it was not seen as capturing the complexity of smoking in people's everyday lives:

*Maybe partially, but I don't think fully. I don't think it's fully your brain. I think it's your daily life, your routine, your social group, it's everything else, I think, and then maybe partially a brain disease or whatever. It sounds really terrible saying it's a brain disease, but maybe partially. (female, 18-25, low dependence)*

A handful of participants thought that the brain disease explanation may apply to some, but not all, smokers. For example, one participant stated that “normal” smoking would not be accurately classified as a brain disease, but once people were addicted enough to experience withdrawal symptoms, then this label might apply (female, 18-25, moderate dependence).

Agreement with the BDMA was often not enthusiastic or wholehearted even among those who accepted it. It was more common for participants to offer qualified agreement. Some who thought that the brain disease label was accurate did not think it was a good idea to describe it that way.

*I don't think it's helpful if scientists are putting that view forward even though it may have a hell of a lot of merits. (female, 55+, high dependence)*

They listed potentially negative social consequences of doing so, the topic to which we next turn.

### ***Concern about the consequences of the “brain disease” explanation of smoking***

Participants were questioned about the effect they thought the brain disease terminology would have on the stigma associated with smoking. Many reported feeling the stigma of smoking acutely, and reacted strongly to anything that they thought might increase this.

*I mean, personally for me I do feel that as a smoker I'm already bad enough, and then I mean if you just keep pushing a person further you reach a point then when the person thinks that okay I'm screwed up I might as well not bother and continue being screwed up. (male, 18-25, moderate dependence)*

Most thought that the brain disease label would increase stigma because the word “disease” had negative connotations. Examples of words that participants associated with the concept of disease were: “dirty”, “disability”, “mental disorder”, “leper”, and “unstable.” Associations with the concept of “brain damage” were seen as especially negative. One participant was concerned that he would become seen as the “local simpleton” if this terminology was introduced (male, 26-40, high dependence). Some participants were aware of the stigma and discrimination associated with mental illness, and were concerned that the brain disease label would equate smoking with a mental illness.

*...to what extent is brain condition conflated to mental illness and the assumptions are therefore maybe not being able to do their jobs or behave appropriately which mightn't actually be consciously examined assumptions but I think nevertheless we know they exist. (female, 55+, moderate dependence)*



Fewer participants thought that the brain disease explanation of addiction would reduce stigma. A minority thought that describing nicotine addiction as a brain disease might reduce negative judgements of smokers because it implied they did not have full control over their behaviour.

*People might be less likely to judge people. Sort of, oh well, poor things can't help it, sort of anymore that you can help having asthma or something. (female, 41-54, moderate dependence)*

Not all saw increased sympathy from non-smokers as a positive though. Some thought it might come in the form of pity.

*Yeah, there's always going to be a certain section of the community that goes, oh, those poor people, they've got a disease. I mean, yeah, but with education, we could reduce that. (male, 26-40, low dependence)*

Another frequent concern participants expressed was that other smokers would use the brain disease label to avoid personal responsibility for their smoking. It was often described as an “excuse” and a “cop out” that would discourage people from using their willpower to quit smoking.

*You don't want people going around making excuses, I've got a brain disease, I smoke. They're never ever going to get out of the rut of maybe I want quit. (female, 55+, high dependence)*

*No, I see it more as an excuse. They're going to go to their doctor and say I have a medical thing that I can't probably function anymore and I can't change it. Because a lot of people will say no, it's deep in my brain, that's a serious problem as well, that's something that you might - I think it just opens a door to people who can't do the mind over matter or whatever. (male, 26-40, high dependence)*

Participants thought that *other* smokers would readily accept the implied reduction in personal responsibility but rarely saw their *own* smoking in this way. In contrast to the dominant view, one participant reported that a biological understanding of smoking reduced her feelings of self-blame. She described the BDMA as “comforting” because it provided a medical explanation for her struggle to control her cigarette intake.

*It's kind of comforting. It does take away that sense of - well I guess it's - yeah, the sense of not being able to control the smoking is also a self-esteem issue. It's like oh, here I am, I should know better and I smoke. (female, 55+, moderate dependence)*

Participants who discussed the implications of labelling smoking as a “brain disease” for quitting and treatment-seeking differed in their views. One thought that it could increase treatment-seeking by helping smokers to recognise that they have a “real problem” (female, 26-40, high dependence). A few thought that this language might encourage quit attempts because brains are “precious” organs associated with one’s identity and spirituality. Others thought the word “disease” was problematic because it made stopping smoking seem more difficult.

*It would make it look probably worse than what it is, and like I said before it might overwhelm a lot of people and they might think that there's nothing to do because they've got a disease, and I think disease is a big word. Probably for the medicine world it would be fine, but for the public, it's a big word to put in their minds. (female, 18-25, low dependence)*

A number of male smokers had quite emotional responses to the claim that smoking was a brain disease. They expressed anger or became irritated that someone might apply this label to them. A handful stated that they would engage in retaliatory smoking if someone labelled them as having a brain disease.

*I would be like - you think it's a disease, I'll show you disease, man. I'll smoke even more or something. (male, 18-25, low dependence)*

Some participants said that they would be deterred from seeking treatment if health practitioners labelled nicotine addiction as a brain disease.

*I just think it would scare smokers away from treatment programs. They're just saying - well my reaction would be look, just leave me alone please, stop talking, we've all got problems and throwing the word disease around is very dangerous, I think. (male, 18-25, low dependence)*

These participants expressed suspicion about public health practitioners and scientists. Anti-smoking education campaigns were described as “scaremongering.” One participant described them as “hippies”, and another thought the brain disease label was a “nanny state” campaign

to tell people that “we are not allowed to do what we want to do.” (male, 41-54, high dependence). These participants thought that the information presented to them was exaggerated or not entirely truthful, and asked to be presented with just “the facts.”

While explicit distrust of science in general was only expressed by a few participants, many disputed the “scientific claim” that was presented to them. Some gave a brief “apology” for disagreeing, or a disclaimer that they were not scientists and were simply expressing their personal opinion.

*I wouldn't say it's a brain disease. I don't want to prove the scientists wrong, but it's just my opinion. I don't think it's a disease. (male, 26-40, low dependence)*

### **Utility of the “brain disease” model of addiction**

It was common for participants to question the feasibility of introducing the brain disease label into public discourse about smoking. This was partly because they thought that it would not be accepted by most smokers, regardless of its scientific accuracy. One participant phrased this as “buy in”. He did not “buy into” the medical explanation of smoking as a brain disease and he thought that this would be true for most smokers. Others thought smokers would find it quite amusing to think of smoking in this way because it was inconsistent with their conceptions of smoking.

*Smokers would probably laugh at it for trying to make it even worse...people would see right through it. (male, 26-40, moderate dependence)*

Other participants thought that the language of smoking as a brain disease may have different consequences for different groups in the community. For example, it was common to distinguish between the effects of this discourse on smokers and non-smokers. Some believed that thinking of smoking in terms of a brain disease might “scare” smokers into making a quit attempt so that they could regain a “healthy brain.” However, they were also concerned that the brain disease label would increase the judgemental attitudes of non-smokers.

*A good thing for some people, a bad thing for others. You know what I mean? Like, if you're a smoker now it may shock you into stop smoking for a - so that's a good thing. The bad thing would be a non-smoker prejudicing towards a smoker and saying that they're a - they have a disability. (male, 18-25, low dependence)*

Some distinguished the utility of the brain disease concept for young people and for older, established smokers. They thought it might deter young people from starting to smoke while discouraging older and more established smokers from attempting to quit.

*...If I wasn't a smoker and someone said that people who smoke have brain disease, it would probably deter me from starting. If I was a smoker and people were calling me someone with a brain disorder, I would probably smoke more just in spite of them. (female, 18-25, low dependence)*

Others thought it unnecessary to introduce the label because current models of smoking were adequate. Introducing this term would only confuse discourses about smoking in society.

*I just think it's a waste of time. I think it's something that doesn't need to be - it's like don't re-invent the wheel. We know it's a drug, we know it's addictive. Leave it at that. (male, 26-40, moderate dependence)*

#### **8.4. Discussion**

While most smokers accepted that nicotine acts on the brain to influence their smoking, the majority rejected the label of smoking as a brain disease. The main reasons for this were: doubts about the scientific accuracy of the claim; concerns that such terminology would increase stigma and prejudice against smokers; and a belief that it would lead smokers to absolve themselves of personal responsibility for their smoking. Participants also believed that most smokers would not endorse such a label, even if it was scientifically accurate.

While a few participants were prepared to defer to the authority of science and accept that nicotine addiction was a brain disease, most pointed to what they perceived as inaccuracies and deficiencies in the term. This is consistent with the broader empirical literature on public understanding of science (PUS) which shows that publics are not passive in their reception of scientific information; they more often resist or re-work scientific ideas based on their own values or experiences (Choudhury et al., 2012; G. Munro, 2010; O'Connor & Joffe, 2013; Pickersgill et al., 2015; Wynne, 1992).

While some in this study made apologies about disagreeing with scientists, a minority questioned the motives of the public health approach to smoking. The latter group believed that they were being provided with biased information in an attempt to manipulate their behaviour, and felt irritated at being told what to do by a “nanny state.” This response highlights the

importance of public trust in judgements about science and technology, which has long been recognised as an important influence on lay acceptance of science in PUS (Wynne, 1992). The tobacco industry has fostered a distrust of public health by criticising tobacco control strategies as forms of “nanny state” interference in personal choice and “freedom” (Daube, Stafford, & Bond, 2008). The tobacco industry has always attempted to position smoking as a free choice that is made by responsible adults who can control their smoking through the use of their willpower (White et al., 2013). The gendered nature of beliefs about the role of willpower and individual responsibility have been noted by White, Oliffe and Bottorff (2013). They argue that the “masculine ideal” of an autonomous and self-contained individual, with internal resources of self-control and willpower, reduced the responsibility attributed to the tobacco industry. These views about the value of individual responsibility and inner strength persist in societal discourses, and in this study, it was largely men who engaged in “nanny state” discourse.

This individualisation of illness has been identified as a feature of contemporary public health and of biomedicalisation (Clarke et al, 2003; Lupton, 1993; Pearce, 1996). This entails a moral responsibility for individuals to identify the disease risk factors relevant to them and to work towards addressing them. If they do not, then they may be subjected to stigma or public shaming (Amonini et al, 2015; Lupton, 2014). The findings reported in this chapter show that “the individual” is the central locus in lay understandings of smoking amongst smokers. Participants emphasised that they took full responsibility for their own smoking, and were concerned that other smokers would use a brain disease explanation of smoking to absolve themselves of personal responsibility. This also echoes findings from Chapter 5, where the general public thought that the best method would “depend on the individual” and their willpower and motivation. Thus, both the BDMA, and lay understandings focused on willpower, embrace the emphasis on the individual smoker. However, as described in Chapter 5, smokers have a moral responsibility to try to stop smoking, not to stop smoking in a particular way. Taking responsibility for your smoking does not entail a medical approach to cessation.

Suspicion of science and public health was not the most common reasons for rejecting the brain disease label. Personal experience seemed more influential. Smoking is a topic about which smokers have considerable experience and they drew on their own smoking careers, which often involved decades of smoking and many failed quit attempts, as well as observations of the smoking trajectories of their friends and family. These personal experiences were used to

construct a schema of smoking and for many of our participants, the brain disease label did not fit with their understandings of smoking. Many described smoking as a habit, or a matter of willpower. The idea of smoking as a choice, while co-opted by the tobacco industry, was also a part of public understandings of smoking because many people have simply quit smoking and apparently succeeded without difficulty (Chapman & MacKenzie, 2010; West & Sohal, 2006). The denial of this element of choice, as implied by the BDMA, was a common reason participants gave for rejecting it.

The fact that participants did not generally reject the scientific information presented in the Powerpoint about nicotine addiction (Chapter 7), but often rejected scientists labelling smoking as a “brain disease” hints at the role that laypeople believe scientists should have in disseminating ideas about addiction. Specifically, speaking factually about the biological aspects of addiction was acceptable, but moving beyond this by labelling smokers as a certain type of person was not. This was especially so, because of the negative connotations that many associated with being labelled as having a “brain disease.” These included a refutation of personal autonomy and will, the application of a “sick” role, and an association with “brain damage” and the associated loss of full personhood. It was only when scientists were deemed to be going “too far” that questions about their credibility began to emerge in the discussions.

Much of the sociological critique of medicalisation describes the adverse social consequences of using medical labels for human behaviour. Our research showed that smokers were also aware of these potential consequences of particular understandings of smoking. Conrad (1992) criticised medicalisation because it can lead to a “dislocation of responsibility”, creating a group of people deemed to be in control, and another group of people who are deemed not to be in control of their behaviour. Many of the participants interviewed for this study recognised this possibility and thought that other smokers would use the brain disease label to diminish their personal responsibility.

Conrad has also argued that labelling a behaviour as a disease is “to deem it undesirable.” (Conrad, 1992, p. 249). Smoking is already undesirable in many countries, and those interviewed reported experiencing significant smoking-related stigma in their everyday lives. They recognised that the disease label was value-laden and had negative connotations that could increase stigma. Most smokers in this study showed no interest in taking on a “sick role” in regards to their smoking. The potential for stigma may partly explain this, but also it may be

because, as Keane (2002) has described, cigarettes have a “unique cultural location” in Western society. Nicotine is acknowledged as an addiction, however it is an addiction that does not often interfere with the activities of everyday life. Also, the harms of smoking are “abstract possibilities” (Keane, 2002), when compared to the more immediate harms associated with alcohol dependence and addiction to most illicit drugs. This makes more intensive interventions such as drug rehabilitation or prescription medications for smoking cessation seem “too serious” for many smokers.

The word “disease” was also problematic because smokers attached many and varied meanings to it. This is not surprising because the term lacks definitional clarity even among academics (DeVito, 2000; Hofmann, 2010; Rääkkä, 1996; Scully, 2004). This makes it easy to use narrow definitions of the term to dismiss it. Using the term “brain disease” may be counterproductive if potentially useful (and more acceptable) messages about the role of the brain in smoking are disregarded. Because the term brain disease creates confusion, distrust and disbelief among smokers, it is not likely to be helpful in health promotion material or clinical interactions with smokers. However, rejection of the brain disease model does not rule out a more nuanced view of the role of the brain in smoking. This could play a useful role in providing smokers with insight into their own smoking. In fact, many participants who rejected the brain disease label found the information about the role of the brain in their smoking interesting and thought it provided insights into their smoking, as described in Chapter 7.

Some may argue that smoking is not often described as a brain disease in the tobacco control or smoking cessation literatures. However, the use of this type of language is implicit in the rationale offered for medical approaches to smoking cessation, e.g., drug treatments such as Champix that are claimed to act on neurotransmitters, and experimental treatments such as transcranial magnetic stimulation that acts directly on brain function (Li et al., 2013). This conceptualisation of addiction is also promoted in health education materials disseminated by NIDA that are designed for use by the general public and schools (National Institute on Drug Abuse, 2006). Moreover, the results reported here demonstrate that it is the label of smoking as a chronic disease that many people rejected, rather than the emphasis on the brain. The chronic disease label is increasingly being applied to tobacco dependence. The US Public Service Guidelines on treating tobacco dependence define it as “a chronic disease that often requires repeated intervention and multiple attempts to quit” (Fiore et al., 2008, p. vi). A number of authors have called for tobacco dependence to be treated as a chronic disease, similar

to diabetes or hypertension (Aveyard & Raw, 2012; Leone & Evers-Casey, 2012; Prochaska & Benowitz, 2016; Wolff et al., 2013). The results of this research suggest that this definition is at odds with the way that many smokers understand their own smoking.

### **Original contribution of Chapter 8**

In the USA, addictions are increasingly being labelled as “brain diseases”. The implications of this terminology for the tobacco control field have not been widely examined, despite nicotine addiction being the most prevalent form of drug addiction globally. This research is the first to look at how smokers respond to being labelled as having a “brain disease”. Our findings suggest the need for caution in applying the NIDA “brain disease” label of addiction to nicotine addiction. The concerns of the smokers interviewed for this research echoed academic critiques of the BDMA. They felt that it did not capture the complexity of their smoking, that it could increase (rather than decrease) stigma, and that it may diminish the role of autonomy and choice in smoking cessation, something that was believed to be key to a successful quit attempt. Disseminating the idea that smoking is a brain disease may increase some smokers’ distrust of health professionals, deterring them from seeking assistance or using cessation aids. The fact that the description of nicotine addiction as a brain disease is discordant with the lived experience of smokers should be thoughtfully considered in debates about the social benefits of this approach to addiction. The next chapter will outline the results of a quantitative survey that sought to elicit smokers’ views on the role of their brain in their smoking, and the potential impact of these views on quitting self-efficacy and treatment intentions.



## **Chapter 9. Do neurobiological understandings of smoking influence quitting self-efficacy or treatment intentions?**

### **9.1. Introduction**

The qualitative findings reported in Chapters 5, 6, 7, and 8 suggested that medicalised explanations of nicotine addiction do not predominate in lay discourse on smoking. When asked about the role of the brain, the smokers that were interviewed acknowledged that tobacco use affected their brain, but they did not believe this played the primary role in their continued smoking. They attached greater importance to willpower, motivation and personal responsibility. The new language of smoking as a brain disease that has begun to infiltrate scientific and public explanatory models of addiction was rejected by most participants, who questioned its accuracy and believed that it could have negative social consequences for smokers.

These findings from the qualitative component of this research inform our understandings of how people interpret information about the neurobiology of nicotine addiction however it is unclear whether these results will apply in a larger and more representative sample of Australian smokers. Qualitative research is by its nature local and situated. The qualitative stage of this research project reported in preceding chapters provided unique insights into smokers' understanding of their smoking that informed the development of questions included in a large quantitative survey. The aims of the quantitative survey were to: (1) determine the prevalence of smokers' attitudes identified in the qualitative stage in a large sample of Australian smokers; and (2) explore relationships between demographic and smoking-related variables and smokers' beliefs about the role of the brain in smoking.

The quantitative study described in this chapter examines the extent to which smokers endorse neurobiological explanations of smoking, and assesses whether their endorsement of brain-based explanations of smoking influences quitting self-efficacy or their preferences for using particular smoking cessation methods. The specific research questions addressed were:

- What proportion of Australian smokers endorse neurobiological explanations of tobacco dependence?

- What sociodemographic variables predict the endorsement of neurobiological explanations of smoking?
- Is there a relationship between endorsement of neurobiological explanations of tobacco dependence and intentions to use pharmacotherapy for future smoking cessation attempts?
- What is the relationship between endorsement of neurobiological explanations of smoking and feelings of quitting self-efficacy amongst Australian smokers?

## **9.2. Method**

### ***Design and Sampling***

The data reported in this chapter come from an online survey of 1,538 smokers in Australia. All participants were smokers recruited from a commercial online research panel accredited to quality assurance standards ISO20252 and ISO26363. Participants on this panel were recruited from both online and offline sources and those who completed the survey received points for participation that could be converted to gift vouchers. The median length of time it took to complete the survey was 18 minutes.

Invites were sent to panel members who had previously reported that they were a smoker and the invitation strategy was adjusted daily with quotas to obtain a sample representative of the demographic profile of the population of Australian smokers in terms of age and gender (Australian Institute of Health and Welfare, 2014). To be eligible, participants were required to be aged 18 years old or older, an Australian citizen or resident, smoked daily, and had smoked more than 100 cigarettes in their lifetime. The recruitment process is outlined in Figure 9-1. Of the 6,520 invited participants who clicked on the link to the survey, 4,273 did not smoke daily, 49 had not smoked at least 100 cigarettes and 16 exited the survey prior to completing the eligibility questions, and were therefore excluded. Of those who met the eligibility criteria (N=2,182), 625 dropped out prior to completion. Seven were identified as duplicate cases caused by a computer error, and were removed from the dataset. Despite reporting daily smoking on the screening questions to assess eligibility, 12 participants stated that they smoked zero cigarettes per day on a subsequent question and were excluded from the dataset.

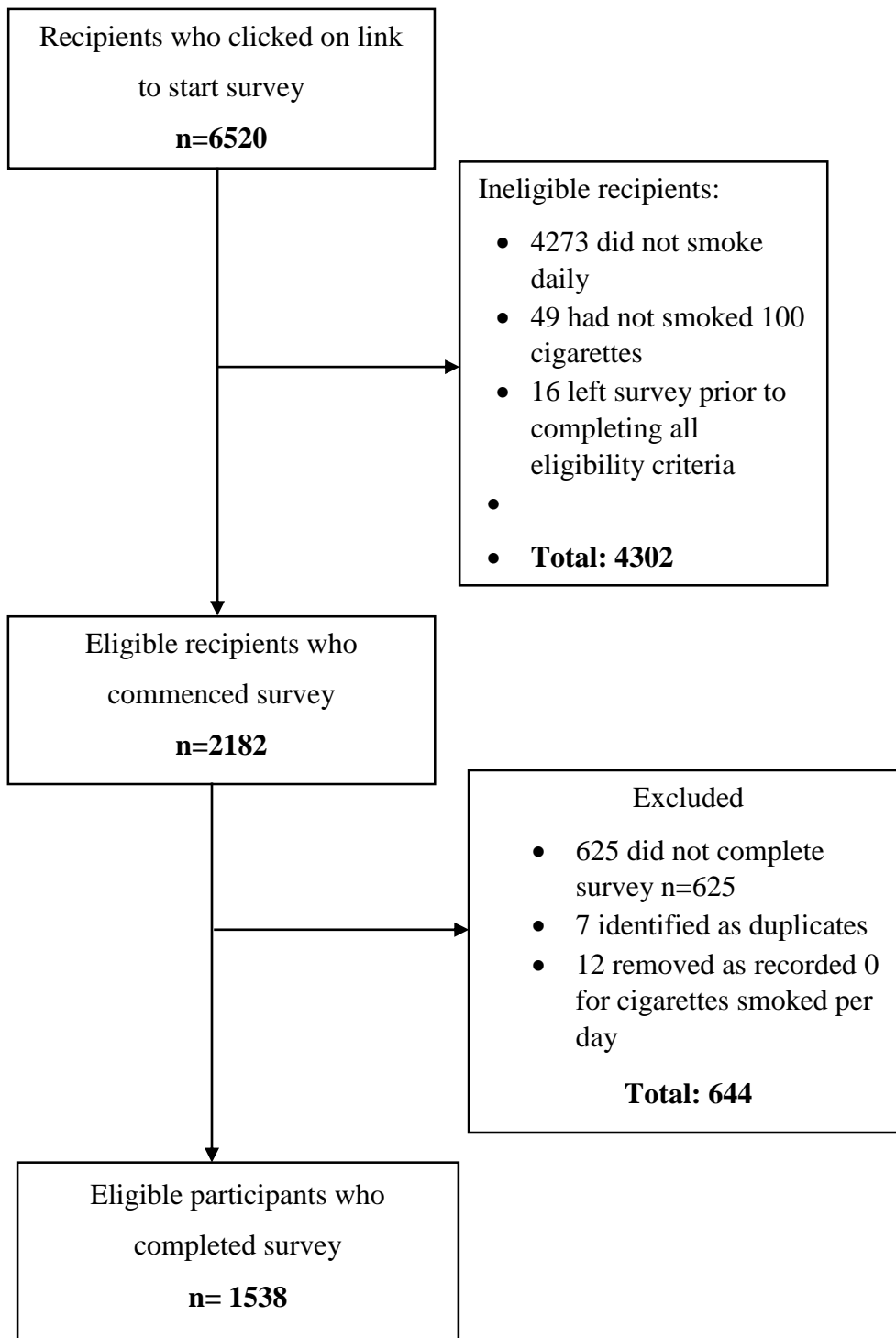


Figure 9-1 - Recruitment process for survey

### 9.3. Survey development

The survey was informed by an extensive literature review (Chapters 2 and 3) and the results of the qualitative component of the project (Chapters 5, 6, 7, 8). Given the paucity of research in this area, the survey design was also informed by research in related areas, such as studies

of the attitudes of persons experiencing other drug addictions towards the role of the brain in their addictions (Meurk et al., 2014; Netherland, 2011; Pescosolido et al., 2010) as well as on public perceptions and knowledge of genetics (Docherty et al., 2011; Houfek et al., 2008; Park et al., 2011; Phelan, Cruz-rojas, & Reiff, 2002; Sanderson, O'Neill, Bastian, Bepler, & McBride, 2010; Smerecnik, Grispen, & Quaak, 2012; Wright et al., 2007).

A number of important themes that emerged from the qualitative component of the research described in earlier chapters were incorporated into the survey instrument. For example, during the interviews, it became clear that participants often described themselves as being different from other smokers. When responding to questions about the nature of smoking, participants often referred to “other smokers”, rather than themselves as smokers. Therefore, we decided to include questions examining participants’ views on their own smoking and their views on other smokers

#### **9.4. Measures**

##### ***Demographic and smoking related variables***

Sociodemographic variables included: age, gender, education (less than year 12, completed secondary school, post school qualifications, bachelor degree or higher), whether the participant was born in Australia, employment status (employed or self-employed, unemployed, a student, home duties or carer, volunteer or charity work, retired or on a pension, unable to work, other) and postcode.

Smoking-related variables included: age of smoking initiation, how many times they had tried to quit in the last year, future quitting intentions, and their enjoyment of smoking. Level of nicotine dependence was measured using the HSI, which contains two items: time to first cigarette of the day, and number of cigarettes smoked per day (Heatherton et al., 1989).

Quitting self-efficacy was measured using a single item asking “*If you decided to give up smoking completely in the next six months, how sure are you that you would succeed?*” This item has been used extensively in the International Tobacco Control Policy Evaluation Project (ITC) (Borland et al., 2010; Hyland et al., 2006; Siahpush, Yong, Borland, Reid, & Hammond, 2009) and in other national surveys (Bonevski et al., 2015). Response options were: not at all sure, slightly sure, moderately sure, very sure, or extremely sure.

The Reasons for Smoking Scale used in the UK Smoking Toolkit Study (Fidler & West, 2009) was adapted for the purposes of this survey. Those items not prominent in the qualitative results reported in earlier chapters were replaced with those that featured frequently in qualitative data from this project. For example, smoking for pain relief was not mentioned by any participants in the interviews, so was replaced with “psychological addiction”, which was frequently given as a reason for smoking.

### ***Beliefs about the neurobiology of smoking***

Four items were developed to assess strength of endorsement of beliefs about the role of neurobiology in smoking. Participants were asked to rate the extent to which they agreed with the following statements on a four-point scale (strongly disagree, disagree, agree, strongly agree) and the option of “don’t know” as a response:

- “Smoking is a brain disease”;
- “Smoking changes the chemistry of the brain”;
- “Smoking damages the brain”
- “Smoking is a brain disorder”; and
- “Don’t know.”

To investigate the *extent* to which they believed that the brain was implicated in smoking, they were given the statement “Smoking causes...” and were asked to complete the sentence with one of the following options: “*Long lasting changes to the way the brain works*”; “*Temporary but substantial changes to the way the brain works*”; “*Minor changes to the way the brain works*”; “*No changes to the way the brain works*”; or “*I have no idea what effect smoking has on the brain.*”

Based on the interview data, we expected participants to have limited knowledge of how the brain was involved in nicotine dependence. Therefore, later in the survey, participants were shown an excerpt from a book written for the general public by Professor Robert West, a leading UK tobacco researcher. The book aims to present scientific evidence about quitting to smokers to help them develop evidence-based strategies for quitting. The excerpt (quoted below) gives more detail about how the brain is implicated in tobacco dependence, to allow participants to respond to a more detailed, nuanced description of the role of the brain than that presented in previous items. Professor West describes the neurobiological basis of smoking as follows:

*“You smoke because the nicotine you have been inhaling all those years has changed your brain chemistry to create powerful urges to smoke. The urges come about because every puff on a cigarette sends a rapid nicotine “hit” to the part of your brain that makes you do things...These urges are triggered because nicotine has trained the part of your brain that gets you to do things to light up a cigarette whenever you find yourself in a situation where you would normally smoke.” (West, 2013, p. 34)*

Participants were asked how well this description explained their own smoking, and how well it explained the smoking of others. Response options were: not a lot, a little, a lot, completely, or don't know. They were then asked again if they believed that smoking was a brain disease, in order to assess whether providing details about the neurobiological basis of tobacco dependence from an authoritative source influenced their responses to this question.

A set of questions also explored participants' views on possible social consequences of the BDMA. They were asked whether they thought that accepting a brain-based explanation for smoking would influence: smokers' desire to stop smoking, smokers' confidence in their ability to quit; negative social attitudes towards smokers; personal responsibility for smoking; sympathy for smokers; treatment-seeking for smoking cessation; and smokers' feelings of guilt. Options were: decrease, have no effect, increase or don't know. The full survey is included as Appendix D.

### ***Use of pharmacotherapy for smoking cessation***

Participants were provided with a list of smoking cessation strategies and asked to check all of those that they had previously used. Those who reported having used nicotine replacement therapy (NRT) or prescription medications (Champix or Zyban) were coded as having used a medication for smoking cessation. In addition, intention to use pharmacotherapy in future quit attempts was assessed with the question *“If you decided to make a quit attempt, how likely is it that would use the following method.”* Response options were: definitely wouldn't use, probably wouldn't use, probably would use, definitely would use, and don't know. Those who selected probably or definitely would use for NRT or prescription medications were categorised as intending to use medications. All other responses were classed as not intending to use medication.

Further questions explored general attitudes toward various quitting strategies. These are not reported here. A first round of pilot testing of the survey with a convenience sample of smokers

from the research team's networks (n=5) was conducted to assess the comprehensibility of the questions and the length of time it took to complete the survey. In order to improve comprehensibility, minor changes to wording were made based on suggestions from those who piloted the survey. A second round of pilot testing with 50 participants recruited by the market research company ensured that there were no problems with the online administration. As no problems arose in this second round of pilot testing, data from these 50 pilot surveys are included in the final dataset.

### **9.5. Statistical data analysis**

After descriptive analyses (counts and percentages) were conducted, two key items measuring the strength of endorsement of brain-based explanations of smoking were selected for further analysis: 1) "*Smoking changes the chemistry of the brain*"; and 2) "*Smoking is a brain disease*." The latter item was chosen to represent NIDA's BDMA. The former measures a weaker representation of the role of the brain in smoking and does not include the controversial term "disease." For each of these items, the five point Likert scale was converted into a three level categorical variable that comprised of: (1) disagree (disagree combined with strongly disagree), (2) agree (agree combined with strongly agree), and (3) don't know. While similar analyses in the literature have excluded "don't know" responses, this was not appropriate in this study because a high proportion of respondents selected "don't know" for these items.

For each of these two key items, contingency tables and the Pearson's chi-squared statistic were used to examine which variables were associated with scores on these two items. Categorical independent variables were gender, education, level of nicotine addiction (measured by the HSI), self-efficacy, and intention to use medication. Age was analysed as a continuous variable using ANOVA.

A binary logistic regression analysis then explored the relationship between endorsement of each of the two key neurobiological explanations of smoking and intention to use medication in a quit attempt. In the first binary logistic regression, intention to use medication was entered as the outcome variable (0=no intention and don't know, 1=intend to use). Predictor variables were entered in two blocks using the Enter method in SPSS v22. The first block contained demographic variables and smoking characteristics: gender (male=0/female=1), age (continuous), level of education, HSI score (low=0-1, moderate=2-3, high=4-6), self-efficacy

(low=not all sure and slightly sure, moderate = moderately sure, high = very sure and extremely sure), past use of cessation medications (0 = no, 1= yes). In the second block endorsement of brain-based explanations were added, specifically “*Smoking is a brain disease*” and “*Smoking changes the chemistry of the brain*”. A correlation analysis including the last two items showed that all items were sufficiently distinct from each other to treat them as separate variables (see Appendix F).

Another binary logistic regression analysis investigated the relationship between endorsements of neurobiological explanations of smoking and self-efficacy. The outcome variable was a dichotomised version of self-efficacy (0 = low, 1 = moderate/high). Again, predictor variables were entered in two blocks using the Enter method in SPSS v22. The first block contained demographic variables and smoking characteristics: gender (male=0/female=1), age (continuous), level of education, HSI score (low=0-1, moderate=2-3, high=4-6), past use of medications (0 = no, 1 = yes). In the second block endorsement of brain-based explanations “*Smoking is a brain disease*” and “*Smoking changes the chemistry of the brain*” were added.

## **9.6. Results**

### ***Participant demographics***

The age of participants ranged from 18-88 years old with a mean age of 43 years (SD 16.1). Soft quotas were set for recruitment so that the sample would align with the proportion of the Australian population who smoked by gender and age, based on data from the National Drug Strategy Household Survey (2014). All quotas were met except for a very slight under-representation of males in the 18-24 and 25-29 age groups.

The proportion of the sample born in Australia aligned closely with the population data [72.8% born in Australia] (ABS, 2013). In relation to education, 26.4% had no post-secondary qualification, 32.2% had completed some post-secondary education at less than bachelor degree level, and 31.3% had completed a bachelor degree or higher. See Table 9.1.



Table 9.1 - Participant demographics for survey

Age, mean (SD)	43.0 (16.1)
Gender %	
Male	54
Female	46
Educational attainment %	
Did not complete high school	18.6
High School	17.8
Post-secondary qualifications (e.g., trade training)	32.2
Bachelor Degree or higher	31.3
Born in Australia %	72.3
Employment status	
Employed or self-employed	54.9
Unemployed	6.3
Student	6.6
Home duties/carer	9.1
Retired/Pension	16.7
Other	6.4

Employment status – other = volunteer work, unable to work, something else.

### ***Smoking related variables***

Table 9.2 reports on the variables relating to participants' smoking. The mean number of cigarettes smoked per day was 15 (SD 9.6). In line with previous studies, a score of 0-1 on the HSI was categorised as low dependence, 2-4 as moderate dependence, and 5-6 as high dependence (Borland et al., 2010; Cooper et al., 2010). Approximately 75% of participants reported moderate or high nicotine dependence.

Most participants (80%) had tried to quit in the past, and a similar proportion intended to quit in the future. Almost half reported low levels of self-efficacy in relation to quitting, and approximately half had used cessation medication for a past quit attempt (either NRT or prescription medication). Approximately two thirds of participants said that they would use NRT or prescription medications if they were to make a quit attempt. This is consistent with data showing that around 60% of smokers in Australia had used help to quit (Cooper et al.,

2011), but discordant with qualitative findings reported in preceding chapters, which showed negative attitudes towards cessation aids.

Table 9.2 - Smoking-related variables

Cigarettes per day, mean (SD)	15.11 (9.6)
Nicotine dependence %	
Low	25.8
Moderate	45.8
High	28.4
Ever tried to quit (% Yes)	79.9
Intend to quit %	
Yes, within 30 days	14.4
Yes, after 30 days, but within 3 months	31.4
Yes, but not within the next 3 months	34.9
No, not planning on giving up	19.2
Self-efficacy %	
Low	47.0
Moderate	32.4
High	20.5
Ever tried medication %	
Nicotine replacement therapy	41.7
Prescription medication	24.0
Total used medication	49.6
Intend to use medication %	
Nicotine replacement therapy	54.1
Prescription medication	45.4
Total intend to use medication	67.2

Nicotine dependence measure HSI: 0-1 = low dependence, 2-3 = moderate dependence, 4-6 = high dependence.

### ***Reasons for smoking***

There was a tendency to endorse most of the reasons for smoking that were listed (see Table 3). Almost 80% of participants agreed or strongly agreed that they smoked because they were addicted to smoking. Most also agreed or strongly agreed that they were addicted to nicotine (72%), and that they smoked because they experienced cravings (78% agree or strongly agree).

The idea that smoking was a habit was strongly endorsed, with 87.4% of participants agreeing or strongly agreeing that they smoked because their smoking had become a habit. Approximately 80% agreed or strongly agreed that they smoked because it helped them to deal with stressful situations. More than half agreed or strongly agreed that they smoked because of a psychological (61%) or physical addiction to smoking (64.1%). Less commonly endorsed reasons for smoking were that they had an addictive personality and that smoking helping them to socialise.

Table 9.3 - Reported reasons for smoking

	Strongly disagree	Disagree	Agree	Strongly agree	Undecided
I am addicted to cigarettes	3.1%	10.5%	50.1%	28.5%	7.7%
I have a psychological addiction to smoking	5.5%	15.3%	42.5%	18.5%	18.1%
I experience cravings for cigarettes	2.7%	11.8%	50.7%	27.3%	7.5%
Smoking is an important part of my everyday routine	4.9%	17.8%	51.4%	16.7%	9.2%
It helps me to socialise	16.5%	33.2%	32.1%	7.5%	10.7%
I want to avoid withdrawal symptoms	6.8%	19.8%	43.2%	18.8%	11.3%
I am addicted to nicotine	3.8%	12.8%	45.5%	26.5%	11.3%
I have an addictive personality	10.9%	26.8%	34.3%	12.9%	15.0%
It helps me deal with stressful situations	2.9%	10.3%	53.2%	26.3%	7.3%
It has become a habit	1.9%	6.0%	47.1%	40.3%	4.7%
I have a physical addiction to smoking	5.4%	15.5%	44.8%	19.3%	14.9%

### ***Endorsement of brain-based explanations of smoking***

Table 9.4 shows the percentage of participants who agreed or disagreed with a number of specific statements about the role of the brain in smoking. The majority (57.9%) agreed or strongly agreed that smoking changed brain chemistry. The findings were similar for the

statement that smoking damages the brain (63.9% agree or strongly agree). Fewer participants agreed that smoking was a brain disease or a brain disorder. Nevertheless, around one third agreed or strongly agreed that smoking was a brain disease. There were high proportions of “don’t know” responses for each item, suggesting that many were unfamiliar with the role of the brain in smoking, or did not feel confident enough to make a judgement. Because a neutral option was not included in the scale, it is also possible that those who have a neutral position selected the Don’t Know option.

Table 9.4 - Endorsement of brain-based explanations of smoking

	Strongly disagree	Disagree	Agree	Strongly agree	Don't know
Smoking is a brain disease	13.1%	25.2%	24.8%	9.6%	27.2%
Smoking changes the chemistry of the brain	3.6%	9.3%	42.2%	15.7%	29.3%
Smoking damages the brain	4.0%	12.3%	38.8%	15.8%	29.2%
Smoking is a brain disorder	14.5%	26.1%	24.1%	8.5%	26.7%
Some smokers have an increased chance of becoming addicted to cigarettes because of their genes	7.7%	17.8%	35.4%	12.9%	26.1%

An additional item examining the *extent* to which participants thought that their brain was affected by smoking revealed that 27.4% thought that smoking caused long-lasting changes in brain function, 18.7% thought it caused temporary but substantial changes, 15.1% thought smoking caused only minor changes to the way the brain works, and 8.6% thought smoking did not change the brain. Again, a high proportion of smokers expressed uncertainty about the relationship between smoking and the brain, with 30.2% selecting the response “I have no idea what effect smoking has on the brain.” Correlations between these items were moderate, ranging from 0.50 to 0.68, meaning that the items were distinct enough to be treated separately in analyses. The correlation table can be found in Appendix F.

After being presented with the quote from Robert West, participants were asked to what extent this description described their own and others’ smoking. Responses are shown in Table 9.5.

Table 9.5 - Acceptance of Robert West’s explanation of smoking

	Not at all	A little	A lot	Completely	Don't know
Accept explanation - self	5.2%	34.2%	35.3%	15.5%	9.8%
Accept explanation others	2.0%	26.7%	40.9%	13.7%	16.8%

Most participants thought that this description of smoking explained their own smoking to some extent. Only a very small proportion believed it did not explain their smoking at all. There were some small differences between whether participants accepted this explanation in regards to their own smoking versus that of others. Participants were more likely to express uncertainty about whether this description applied to other smokers than themselves.

Participants were again asked if they thought smoking was a brain disease, to assess whether providing a description of how nicotine worked in the brain would increase their acceptance of their explanation. A cross-tabulation showing movement between categories is provided in Table 9.6. Table 9.6 shows that there was some movement between categories, but the majority of participants who agreed or strongly agreed remained in one of these categories after being presented with Robert West’s explanation of the role of the brain in smoking. Similarly, the majority who disagreed or strongly disagreed selected one of these options on the second occasion they were asked. There were few who initially selected “strongly agree” or “strongly disagree” who later switched to an opposing category. Notably, almost half of the participants who initially selected the “don’t know” option changed their response after reading the Robert West quote. Of the 45.1% who changed from “don’t know”, 25.1% selected “agree” or “strongly agree”, while 20% switched to “disagree” or “strongly disagree”. This suggests that providing more detail about the neurobiological basis of tobacco dependence may reduce uncertainty about the claim that smoking is a brain disease, but does not sway people to change their opinions in one direction over the other.

Table 9.6 - Change in agreement that smoking is a brain disease after being presented with Robert West explanation

		Smoking is a brain disease (Time 2)					Total
		Strongly agree	Agree	Disagree	Strongly disagree	Don't know	
Smoking is a brain disease (Time 1)	Strongly agree	45 30.4%	70 47.3%	6 4.1%	6 4.1%	21 14.2%	148 100.0%
	Agree	31 8.2%	230 60.5%	54 14.2%	15 3.9%	50 13.2%	380 100.0%
	Disagree	11 2.8%	85 21.9%	179 46.1%	42 10.8%	71 18.3%	388 100.0%
	Strongly disagree	8 4.0%	21 10.4%	67 33.2%	81 40.1%	25 12.4%	202 100.0%
	Don't know	7 1.7%	98 23.4%	70 16.7%	14 3.3%	230 54.9%	419 100.0%
Total		102 6.6%	504 32.8%	376 24.5%	158 10.3%	397 25.8%	1537 100.0%

Missing data n = 1

Participants were also questioned about the potential consequences of emphasising the role of the brain in smoking dependence (see Table 9.7). The most common response for most items was that describing smoking in the way outlined by Robert West would have no effect but the “don’t know” response was again very common. Forty per cent of participants thought that emphasising the role of the brain would increase their desire to quit smoking, with only 5.1% reporting that they thought it would decrease their desire to quit. Almost half of those surveyed thought that the number of people willing to seek medical treatment would increase if smoking was explained in neurobiological terms. Most did not think it would increase negative attitudes towards people who smoke (stigma), or decrease smokers’ confidence in their ability to quit.

Table 9.7 - Expectations of consequences of brain-based explanations of smoking

	Decrease	No effect	Increase	Don't know
The desire to stop smoking	5.1%	34.0%	40.1%	20.8%
Smokers confidence in their ability to quit	11.6%	32.6%	32.4%	23.4%
Negative attitudes towards people who smoke	11.1%	44.6%	23.0%	21.3%
The sense of personal responsibility that people feel for their smoking	9.3%	34.7%	31.0%	25.0%
Sympathy towards people who smoke	5.6%	45.7%	27.4%	21.3%
The number of people willing to seek medical treatment for their smoking	2.9%	26.5%	46.2%	24.4%
Feelings of guilt in those who smoke	10.9%	40.4%	25.9%	22.8%

### ***Who endorses brain-based explanations of smoking?***

The results of the bivariate analyses are presented in Table 9.8. Endorsement of the belief that “smoking changed brain chemistry” was not significantly associated with gender or level of nicotine dependence but it was strongly associated with age. The mean age of those who agreed that smoking changed the chemistry of the brain was ten years lower (39.55, SD = 14.55) than those who did not know (49.27, SD = 16.64), and approximately 4 years lower than those who disagreed (44.12, SD = 17.64).

It was also associated with education. Those who had a lower level of education were more likely to say that they did not know whether smoking changed the chemistry of the brain (37.4%) compared to those with a bachelor degree (19.7%). Those with a bachelor degree or higher qualification were more likely to agree that smoking changed brain chemistry (68.5%) than those who had not completed high school (47.6%).

Those with low self-efficacy were less likely to agree that smoking changed the chemistry of the brain than those with high self-efficacy (50.5% versus 67.4%). Those who agreed that smoking changed brain chemistry were more likely than those who disagreed to intend to use medication on their next quit attempt.

The findings were similar for endorsement of the statement that “smoking is a brain disease”. Again, level of nicotine dependence was not related to endorsement while age was strongly related. Younger participants were more likely to agree that smoking was a brain disease, and less likely to say that they did not know. Females were more likely to disagree that smoking was a brain disease than males (41.3% versus 35.9%). Again, those with a university degree were more likely to agree that smoking was a brain disease (45.6%) than those with the lowest level of education (26.2%) and less likely to say that they didn’t know. Those who agreed that smoking was a brain disease had higher self-efficacy and were more likely to express an intention to use medication.



Table 9.8 - Proportion of smokers who endorsed brain-based explanations of smoking stratified by demographics and smoking characteristics

Strata	Smoking changes the chemistry of the brain			<i>p</i>	Smoking is a brain disease			<i>p</i>
	<i>Agree</i>	<i>Disagree</i>	<i>Don't know</i>		<i>Agree</i>	<i>Disagree</i>	<i>Don't know</i>	
<b>Gender</b>				0.94				0.04
<b>Male</b>	484 (58.2)	107 (12.9)	240 (28.9)		308 (37.1)	298 (35.9)	225 (27.1)	
<b>Female</b>	406 (57.4)	91 (12.9)	210 (29.7)		221 (31.3)	292 (41.3)	194 (27.4)	
<b>Age (M, SD)</b>	39.55 (14.55)	44.12 (17.64)	49.27 (16.64)	<0.001	38.18 (14.26)	43.66 (16.63)	48.09 (16.12)	<0.001
<b>Highest education</b>				<0.001				<0.001
<b>Did not complete high school</b>	136 (47.6)	43 (15)	107 (37.4)		75 (26.2)	110 (38.5)	101 (35.3)	
<b>Completed high school</b>	151 (55.1)	36 (13.1)	87 (31.8)		83 (30.3)	102 (37.2)	89 (32.5)	
<b>Post-school qualification</b>	273 (55%)	62 (12.5)	161 (32.5)		151 (30.4)	200 (40.3)	145 (29.2)	
<b>Bachelor degree or higher</b>	330 (68.5)	57 (11.8)	95 (19.7)		220 (45.6)	178 (36.9)	84 (17.4)	
<b>Level of dependence</b>				0.80				0.57
<b>Low</b>	232 (58.6)	51 (12.9)	113 (28.5)		135 (34.1)	160 (40.4)	101 (25.5)	
<b>Moderate</b>	414 (58.9)	85 (21.1)	204 (29)		251 (35.7)	364 (37.6)	188 (26.7)	
<b>High</b>	242 (55.5)	61 (14)	133 (30.5)		142 (32.6)	164 (37.6)	130 (29.8)	

<b>Self-efficacy</b>				<0.001				<0.001
<b>Low</b>	365 (50.5)	92 (12.7)	266 (36.8)		186 (25.7)	301 (41.6)	236 (32.6)	
<b>Moderate</b>	312 (62.5)	59 (11.8)	128 (25.7)		188 (37.7)	186 (37.3)	125 (25.1)	
<b>High</b>	213 (67.4)	47 (14.9)	56 (17.7)		155 (49.1)	103 (32.6)	58 (18.4)	
<b>Intention to use medication</b>				<0.001				<0.001
<b>Intend to use medication</b>	661 (64)	117 (11.3)	255 (24.7)		401 (38.8)	377 (36.5)	255 (24.7)	
<b>No intention to use medication</b>	229 (45.3)	81 (16)	195 (38.6)		128 (25.3)	213 (42.2)	164 (32.5)	
<b>Total</b>	57.9	12.9	29.3	-	34.4	38.4	27.2	-

Figure is presented as n (% within strata), except for age (mean, SD).  $X^2$  used to test for statistical significance except age where one-way ANOVA conducted (Welch). Agree = agree plus strongly agree, disagree = disagree plus strongly disagree. Medication = prescription medication or NRT. Level of dependence (HSI): 0-1 low, 2-3 moderate, 4-6 high.

*Is intention to use medication influenced by beliefs about the neurobiological basis of smoking?*

As described in Section 9.5 above, forward stepwise binary logistic regression analysis assessed whether brain-based understandings of smoking were related to intention to use medication. The regression was conducted in two blocks to assess whether the two key brain-related items improved on a model including only demographic and relevant smoking related variables. The Enter method in SPSS V22 was used for each block. The first block included the demographic and smoking-related variables of age, gender and level of education, nicotine dependence (HSI), smoking cessation self-efficacy, and past use of medication. This model was statistically significant ( $p < 0.001$ ) and together these variables correctly predicted intention to use medication in 67.7% of participants. When agreement with the statements that smoking changes brain chemistry and smoking is a brain disease was added in Step 2 of the model, the model remained statistically significant but the predictive power of the model only increased very slightly to 68.6%. Though this increase was statistically significant, the small difference is unlikely to have any practical significance. The final model with all variables included is presented in Table 9.9.

Intention to use medication for smoking cessation was not related to age (OR 1.00, 95% CI 0.99-1.00) or gender (OR 0.87, 95% CI 0.69-1.10), once other factors were controlled for. Education was significantly related to intention to use cessation medications: those who had a university degree were more likely than those with no high school education to intend to use medications (OR 1.75, 95% CI 1.23-2.48). Those with a moderate level of nicotine dependence (scored 2-3 on the HSI) were less likely to intend to use medications than those categorized as having low nicotine dependence (OR 1.34, 95% CI 1.02-1.78). There were no statistically significant differences in intention to use medication between those with low and higher levels of self-efficacy. Participants who agreed that smoking changed the chemistry of the brain were more likely to report an intention to use medication (OR 1.62, 95% CI 1.13-2.31) as were those who agreed that smoking was a brain disease (OR 1.5, 95% CI 1.11-2.01). These effect sizes were statistically significant but only of moderate size. The biggest predictor of intention to use medications was past use: those who had used cessation medications in the past were approximately three times more likely to intend to do so in the future (OR 3.14, 95% CI 2.46-4.02).

Table 9.9 - Binary logistic regression model regressing intention to use cessation medications (yes versus no) on socio-demographic and smoking characteristics

Factor	Odds Ratio	95% (CI) confidence interval around OR		P-value
		Lower	Upper	
Female sex (reference = males)	0.87	0.69	1.10	0.216
Age	1.00	0.99	1.00	0.266
<b>Education</b>				
<b>Did not complete high school (ref)</b>				
<b>Completed high school</b>	1.07	0.74	1.54	0.727
<b>Post-school qualification</b>	1.30	0.94	1.80	0.117
<b>Bachelor degree or higher</b>	1.75	1.23	2.48	0.002
<b>Nicotine dependence (HSI)</b>				
<b>Low dependence (ref)</b>				
<b>Moderate dependence</b>	1.34	1.02	1.78	0.038
<b>High dependence</b>	1.35	0.97	1.87	0.072
<b>Self-efficacy</b>				
<b>Low self-efficacy (ref)</b>				
<b>Moderate self-efficacy</b>	1.30	0.99	1.71	0.061
<b>High self-efficacy</b>	0.85	0.62	1.16	0.301
<b>Prior use of medication</b>				
<b>No (ref)</b>				
<b>Yes</b>	3.14	2.46	4.02	<0.001
<b>Agreement that smoking changes brain chemistry</b>				
<b>Disagree (ref)</b>				
<b>Agree</b>	1.62	1.13	2.31	0.008
<b>Don't know</b>	0.87	0.59	1.29	0.496
<b>Agreement that smoking is a brain disease</b>				
<b>Disagree (ref)</b>				
<b>Agree</b>	1.50	1.11	2.01	0.007
<b>Don't know</b>	1.06	0.79	1.44	0.690

Missing cases =7. Sex: Male = 0, Female =1. Nicotine dependence (HSI): 0-1 = low, 2-3 = moderate, 4-6 = high. Prior use of medication = ever use of NRT or prescription medications for smoking cessation. Agreement that smoking changes the chemistry of the brain = agree and strongly agree. Disagree that smoking changes the chemistry of the brain = disagree and strongly disagree. Agreement that smoking is a brain disease = agree and strongly agree. Disagree that smoking is a brain disease = disagree and strongly disagree.

### ***Is smoking cessation self-efficacy influenced by beliefs about the neurobiological basis of smoking?***

Another binary logistic regression analysis tested the relationship between self-efficacy and beliefs about the role of the brain in smoking. While some studies have suggested that believing that addiction to smoking is based in the brain could decrease self-efficacy, the qualitative research reported in Chapters 7 and 8 suggested that it could also increase some smokers' self-efficacy. The self-efficacy item was measured on a five point scale, but for the purposes of this analysis it was dichotomised so that those with low self-efficacy (those who had responded that they were not at all or slightly sure they could quit within the next six months) were compared to those who had selected one of the remaining responses (moderately sure, very sure, extremely sure). This dichotomisation is consistent with a previous study (Fathelrahman et al., 2009), and its suitability was assessed by cross-tabulations of self-efficacy responses with other important variables. These showed that those who reported being not at all sure or a little sure that they could quit had similar responses on other questions. Conversely, those who reported moderate-high levels of cessation self-efficacy responded quite differently to those with low self-efficacy. These cross-tabulations are presented in Appendix F. The results of the binary logistic regression are presented in Table 9.10.

Predictor variables entered in the first block were age (continuous), gender (male=0, female=1), level of education, level of nicotine dependence (HSI) (low, moderate/high), and past use of medication (No=0, Yes=1). This model was statistically significant ( $p<0.001$ ) and correctly assigned 63.4% of participants on self-efficacy. Block 2, where agreement that smoking changed the chemistry of the brain (no, yes and don't know) and that it was a brain disease (no, yes and don't know) were entered, and was also statistically significant ( $p<0.001$ ). The addition of two brain-endorsement items marginally increased the predictive power of the model, correctly assigning 66% of participants.

Table 9.10 - Binary logistic regression model regressing level of self-efficacy on socio-demographic and smoking characteristics

Factor	Odds Ratio	95% (CI) confidence interval around OR		P-value
		Lower	Upper	
Female sex (reference = males)	1.03	0.83	1.29	0.78
Age	0.98	0.98	0.99	<0.001
Education				
Did not complete high school (ref)				
Completed high school	1.40	0.98	1.99	0.065
Post-school qualification (eg diploma)	1.43	1.04	1.95	0.026
Bachelors degree or higher	1.60	1.16	2.22	0.005
Nicotine dependence (HSI)				
Low dependence (ref)				
Moderate dependence	0.77	0.59	1.01	0.06
High dependence	0.40	0.29	0.54	<0.001
Prior use of medication				
No (ref)				
Yes	0.80	0.64	1.00	0.05
Agreement that smoking changes brain chemistry				
Disagree (ref)				
Agree	0.92	0.65	1.30	0.62
Don't know	0.62	0.42	0.91	0.013
Agreement that smoking is a brain disease				
Disagree (ref)				
Agree	1.73	1.32	2.26	<0.001
Don't know	1.08	0.81	1.45	0.59

Missing cases =7. Sex: Male = 0, Female =1. Self-efficacy: low = 0, moderate and high = 1. Nicotine dependence (HSI): 0-1 = low, 2-3 = moderate, 4-6 = high. Prior use of medication = ever used NRT or prescription medications for smoking cessation. Agreement that smoking changes the chemistry of the brain = agree and strongly agree. Disagree that smoking changes the chemistry of the brain = disagree and strongly disagree. Agreement that smoking is a brain disease = agree and strongly agree. Disagree that smoking is a brain disease = disagree and strongly disagree.

Gender and past use of medication did not make a statistically significant contribution to the final model (OR gender 1.03, 95% CI 0.83-1.29; OR use of medication = 0.80, 95% CI 0.64-1.00). Those who were younger were less likely to report low self-efficacy (OR 0.98, 95% CI 0.98-0.99), although this effect was small. Higher levels of education were significantly associated with high cessation self-efficacy. The odds of high self-efficacy were 1.6 times higher in those with a university degree than in those who did not complete high school (OR 1.6, 95% CI 1.16-2.22). As might be expected, participants with high levels of dependence had lower levels of self-efficacy than those with low-levels of nicotine dependence (OR = 0.40, 95% CI 0.29-0.54). Those who agreed and disagreed that smoking changed the chemistry of the brain did not differ in terms of their self-efficacy but those who said that they “don’t know” were more likely to belong in the low self-efficacy group (OR 0.62, 95% CI 0.42-0.91). For the brain disease item however, there was a statistically significant difference between those who agreed and disagreed: those who agreed that smoking was a brain disease were more likely to have high self-efficacy than those who disagreed (OR 1.73, 95% CI 1.32-2.26).

## **9.7. Discussion**

One aim of this quantitative study was to investigate the proportion of Australian smokers who endorse brain-based explanations of smoking. Such research has not been conducted in Australia, but is worthwhile as it can indicate of the extent of the medicalisation of smoking, and the uptake of neuroscientific explanations of addiction. The results demonstrate that around one third of our sample of Australian smokers agreed that smoking was a brain disease, and a similar proportion did not know whether this explanation of smoking was true. Given the generally very negative sentiment toward this terminology in the qualitative interviews, we expected higher levels of disagreement with this statement. As expected, a higher proportion of participants (57.9%) agreed with the statement that smoking changes the chemistry of the brain, but a substantial proportion of participants were uncertain. Further analyses revealed that those who were less educated were more likely to say that they did not know. This is consistent with other research on PUS (Bauer, 1996). It was unclear whether this is because they have less knowledge around the topic of smoking and the brain, or because they are more generally less likely to express opinions on unfamiliar topics. The complexities of the “don’t know” response have been outlined by researchers in PUS. In particular they have noted that this response does not always signify ignorance, but can be due to “the absence of representation,

to a sense that the question is irrelevant to the respondent and/or it may relate to the defensive needs of the individual” (Bauer & Joffe, 1996, p. 11).

Another aim was to examine whether socio-demographic variables predicted endorsement of brain-based understandings of smoking. Results revealed that endorsements of brain-based beliefs about smoking were not uniform across social groups. Those who endorsed the stronger form of the “brain disease” explanation of smoking were more likely to be: male, younger, have a higher level of self-efficacy, have more education, and intended to use medication on their next quit attempt. Those who agreed with the less controversial language that smoking changes the chemistry of the brain were similarly younger, more highly educated, more likely to intend to use medication, and had higher self-efficacy than those who disagreed. These findings are discordant with predictions that the BDMA will reduce self-efficacy in addicted individuals but supports speculation that it could increase the use of cessation medications (Chapman & MacKenzie, 2010).

A third aim was to assess whether endorsement of brain-based explanations of smoking were associated with intention to use medication. A binary logistic regression analysis with intention to use medication as the outcome variable showed that when other factors were controlled for, endorsement of smoking as a brain disease and agreement that it changed the chemistry of brain were both positively associated with intention to use medication. This effect, although statistically significant, was only of moderate effect size. A greater effect was seen for past use of medication, with those who had used medication in the past having around three times higher odds of intending to do so in a future quit attempt. This is consistent with research showing that those who have used NRT or prescription medication often report finding them helpful (Hung et al., 2011) and that current smokers who had used NRT or bupropion in the past were more likely to perceive them as helpful compared to those who had not previously tried them (Hammond et al., 2004).

The last aim was to investigate whether believing smoking was a brain disease was associated with low self-efficacy. A binary logistic regression with level of self-efficacy as the outcome showed that after other factors were controlled for, those who agreed that smoking was a brain disease had greater odds of having high self-efficacy. This conflicts with the speculation of those such as Chapman and McKenzie (2010), who believe that the biomedical understandings of addiction will increase feelings of fatalism in relation to smoking.



It must be acknowledged that the size of many of these statistically significant differences were small, reflecting the large sample size in this study. Statistical significance does not necessarily mean that the predictor variable will have a large (or any) significant impact at a clinical or population level. The findings from this study suggest that endorsement of brain-based explanations of smoking may have a small effect on treatment preferences and self-efficacy. While these effects may be small, the belief that smoking changes brain chemistry or is a brain disease in some cases had a larger effect than other factors such as age, gender and level of nicotine dependence.

A number of limitations must be acknowledged. Firstly, participants were not recruited via random sampling. Participants were existing members of an online market research panel so there may be differences between smokers who are members of this panel, and the general populations of smokers. While resourcing requirements ruled out the possibility of other sampling methods, such as random digit dialling for recruitment, the changing nature of survey recruitment means that online panels are increasingly being used as a cost-effective and valid means of collecting survey data (American Association for Public Opinion Research, 2010). In addition, the validity of older methods of random sampling such as random digit dialling is being challenged by the increasing use of mobile phones and reductions in response rates (Australian Communications and Media Authority, 2014; Fogliani, 1999). Also, our sample closely matched the Australian population of smokers in relation to age, gender, and being born overseas. Our cross-sectional data also preclude drawing conclusions about the direction of the relationships between the outcome and predictor variables.

The high proportion of “don’t know” responses was of interest, and further research could investigate the reasons why respondents selected “don’t know.” It could be due to a low level of knowledge among smokers about how smoking affects the brain, or more simply could be the result of not including a neutral response category. There is debate in the survey development literature about whether to include a neutral option and/or don’t know option in attitudinal surveys. However, no consensus has been reached and context must be considered when making decisions for each item. For items relating to endorsement of brain-based understandings, a “don’t know” option was provided because it was expected that some participants would be unfamiliar with the topic. A neutral response was not included, as it has been shown that people may select this option to reduce the cognitive load of choosing a

positional response. It is possible that some of the people who selected “don’t know” in this survey would have selected a “neutral” or “midpoint” option if it were presented.

As described in Chapter 7, it has been claimed that advancements in neuroscience will lead to a transformation in the way that “personhood” is understood (O’Connor & Joffe, 2013). More specifically, it is thought that emphasising the role of the brain in behaviour across various domains will mean that people come to view themselves as biological beings who are controlled by their brain, thereby reducing perceptions of free will (Caron et al., 2005; O’Connor & Joffe, 2013; Rose, 2003). This quantitative study contributes to the literature on neuroscience and society by exploring empirically whether brain-based understandings of smoking are associated with attitudes towards medical treatment, or feelings of self-efficacy. The findings presented here counter the claim that biological understandings reduce self-efficacy by showing that the belief that smoking is a brain disease was associated with *higher* quitting self-efficacy. This is consistent with qualitative research that describes the complex way that neuroscience information is incorporated into beliefs about human nature and behaviour in ways that maintain a sense of autonomy and agency (Pickersgill, 2011; O’Connor & Joffe, 2013). It is also consistent with the qualitative component of this research (Chapters 7 and 8), in which participants acknowledged the potential for neurobiological beliefs to induce fatalism in *other* smokers, but strongly felt that it would not lead to an exculpation of personal responsibility or a reduction in quitting self-efficacy in relation to themselves.

In relation to understanding oneself as a “neurochemical self”, this research shows that neurochemical explanations of smoking were accepted by many smokers. The “brain disease” label, however, was not endorsed by the majority. This demonstrates that neurobiology, when presented as scientific fact, rather than a claim or label applied to an individual or group, was acceptable to the majority of smokers. When this scientific information was used to label smokers as a certain type of neurologically-determined person, it was often resisted and questioned. Thus, the way neurobiological information about addiction is presented is likely to have a great impact on the way it is interpreted and incorporated into public understandings. This aligns with the qualitative research presented in Chapter 8 that demonstrates that the participants in our sample resisted being labelled as a sufferer of a “brain disease”, and that for some smokers, this labelling led to questions being raised about the credibility and motives of those who described smoking in this way. The label of smoking as a “brain disease” was not

accepted as a logical or valid extension of the information about the role of the brain in maintaining smoking behaviour.

Those concerned about the biomedicalisation of smoking predict that it will lead to an emphasis of medical treatments at the expense of unassisted quitting (Chapman & McKenzie, 2010). The research presented here is consistent with other Australian studies (Cooper et al., 2011) in showing that around half of smokers had used pharmacotherapy in the past. This was the first Australian study to examine the extent to which causal beliefs about smoking were associated with intention to use medications for quitting. The findings show that agreement with neurobiological explanations of smoking were associated with greater intention to use pharmacotherapy for smoking cessation, but it must be remembered that the brain-related variables only accounted for a small amount of variance in the intention to use medications. The only other study that has examined beliefs about the causes of smoking and their relationships towards treatment attitudes similarly found weak relationships (Hughes, 2009). There are likely to be many other factors that influence an individual's decision on whether to use pharmacotherapy for smoking cessation.

Overall, these results suggest that a neurobiological view of smoking does not dominate public understandings of nicotine addiction among smokers in Australia. Where people do endorse brain-based explanations of smoking, this does not have the negative consequence of reducing cessation self-efficacy as has been suggested by some. On these results, it is possible that emphasising the role of the brain could increase intention to use cessation pharmacotherapies but any such effect is likely to be small. Many factors influence a smoker's preference regarding cessation methods and their sense of self-efficacy. Combining all these factors into a model still accounts for only a small amount of the variance in these outcome variables. This is not surprising given the complexity of smokers' conceptions of addiction, the brain, and agency demonstrated in the interview component of this study.

## **Chapter 10. Discussion**

Cigarette smoking remains a major public health problem in Australia and worldwide. As with other types of addiction, there is an increasing emphasis on the use of medications to assist smokers to quit smoking and a growing interest in the brain mechanisms that underlie nicotine addiction. The “biomedicalisation” of smoking cessation is seen by some as a positive development that will lead to increased availability of effective medical treatments; a reduction in the stigma associated with smoking; and greater uptake of, and adherence, to biomedical treatments (Britton, 2009; Finnell, 2000; Wolff et al., 2013). Others think it could reduce attempts at unassisted quitting, increase feelings of fatalism in smokers, and exacerbate the stigma that smokers already experience (Caron et al., 2005; Chapman & MacKenzie, 2010; Elam, 2015a; Gillies & Willig, 1997). There is very little empirical evidence available to test these competing predictions about biomedicalisation or to highlight how biomedical explanations of smoking have been incorporated into lay understandings of smoking. No work of this sort has previously been done in Australia. This research project drew on the literatures of lay understandings of health and illness, (bio)medicalisation, and tobacco control in order to explore how the changing nature of tobacco treatment is reflected in the public understandings of smoking and cessation.

### **10.1. Original contribution of the current research**

This research project assessed the extent to which smoking has become medicalised in Australia by exploring lay discourse in Australia around smoking cessation, as well as the attitudes of daily smokers towards assisted and unassisted quitting. Following this more general examination, specific attention was given to the impact of the BDMA, and the effects that brain-based explanations of addiction may have had on smokers understanding of their smoking. Because this is an under-researched area, this study used mixed methods research, with an initial exploratory qualitative stage that informed the development of a large quantitative survey of Australian smokers. This discussion draws together the findings from the three studies reported in this thesis in order to answer the following questions, each of which will be elaborated on below:

- To what extent has a medical discourse of tobacco dependence been incorporated into lay understandings of smoking and cessation in Australia?

- Do smokers believe tobacco dependence is based in their brain, and what are the implications for treatment choices and quitting self-efficacy?

In addition to contributing to the tobacco control literature by exploring the meanings associated with addiction to smoking, the research also contributes to the literature on lay engagement with neuroscience. Most previous research in this area has looked at the impact of neuroscience research on those who have been diagnosed with a disorder where a change in neurobiology is implicated (Bröer & Heerings, 2012; Buchman et al., 2013; Pickersgill et al., 2011), or members of the general public who find neuroscience research mostly “innocuous” (O'Connor, 2013, p. 257). The participants in this project were somewhere in between, a non-clinical sample whose behaviour has begun to be attributed to their neurobiology by experts and the popular media.

### **10.2. To what extent has a medical discourse of tobacco dependence been incorporated into lay understandings of smoking and cessation in Australia?**

As outlined in Chapter 2, the process of biomedicalisation involves the application of medical definitions to previously nonmedical problems, the development and promotion of biotechnologies, and an increasing emphasis on risk and self-surveillance (Clarke et al., 2003). It has been argued that there is an increasing biomedicalisation of smoking in Australia and in other high income countries. This is reflected in the increased use of pharmacotherapies for smoking cessation (Cooper et al., 2011), the emphasis on smoking cessation interventions by health care professionals (Zwar et al., 2014; West, McNeill & Raw, 1998), and the labelling of tobacco dependence as a “chronic disease” (Fiore et al., 2008) or “chronic brain disorder” (Prochaska & Benowitz, 2016). In Chapter 2, it was argued that certain factors unique to tobacco control are likely to limit the reach of biomedical approaches to smoking cessation. However, there was little empirical research identified that explored the extent to which a biomedical model of smoking and cessation is present in lay understandings of smoking and nicotine addiction.

The interviews that were conducted in Study 1 and Study 2 aimed to redress this neglected topic, and revealed the limited impact of the biomedical model on lay understandings of smoking. While more smokers are using medications in Australia in quit attempts, many are not using them for a sufficient periods of time or are using less than the recommended dose

(Balmford et al., 2011; Cooper et al., 2011). The research reported in this thesis provides some potential explanations for this:

- 1) Smoking was often perceived as a matter of willpower and individual choice. It was seen as the personal responsibility of the individual to quit smoking and there was a common belief that quitting unassisted was the best method. However, this did not mean that a medical approach was completely rejected. Rather, it was frequently portrayed as suitable for “other” smokers, particularly those who were “heavy”, long-term or addicted smokers. The difficulty of quitting unassisted was often acknowledged, but the convenience, safety, and anticipated sense of achievement if successful made this method very appealing to many smokers.
- 2) There was a common belief that a smoker must feel “ready” to quit, and reach a tipping point where their desires and circumstances are aligned in order to successfully quit. Balmford and Borland (2008) note that in those who have an addiction, there will always be ambivalence about quitting. If smokers wait for a time when they do not feel any ambivalence or uncertainty about their ability to quit, then they may never attempt to stop smoking. Our findings show that the belief that someone must be “ready” to quit before they can succeed was very common and a potential barrier to making quit attempts using medications. This leads to the Catch 22 that if someone is ready to quit, they are seen as not needing the assistance of pharmacotherapy. However, if they are ready to quit, then the use of medications is not seen as necessary or sufficient.
- 3) There was some ambivalence amongst daily smokers about the nature of their addiction to smoking. While most agreed they were addicted to smoking in some sense, their conceptualisations of addiction were complex and multidimensional. They did not see their addiction to smoking as a purely physiological entity or biologically based. That is, they did not equate addiction to smoking with nicotine addiction. Rather, they used a broad concept of addiction that incorporated behavioural habituation, psychological urges and societal influences. Smokers who see nicotine dependence as only explaining a small part of their addiction, or reject nicotine addiction completely, see limited value in the use of nicotine replacement therapy.

As we saw in Chapter 2, Conrad has proposed a list of factors that facilitate or impede medicalisation, including: “the support of the medical profession, availability of interventions or treatments, existence of competing definitions, coverage by medical insurance, and the

presence of groups challenging the medical definition.” (Conrad, 1992, p. 220). The interplay of these competing factors in Australia has contributed to the incomplete medicalisation of smoking cessation observed in the studies included in this thesis, as discussed below.

In Australia, the national approach to reducing tobacco smoking has emphasized population level strategies to motivate smokers to attempt to quit (e.g. public smoking bans, mass media campaigns, retail display bans, high tobacco taxation, graphic health warnings and plain packaging) with only limited investment in interventions to assist smokers to quit (Australian Intergovernmental Committee on Drugs, 2012). Other countries, most notably the UK, have invested more heavily into treatments for smokers, with a wider range of cessation medications available at no cost, and individual face-to-face counselling and group support sessions provided at stop smoking clinics (McNeill, Raw, Whybrow, & Bailey, 2005). In Australia, NRT in the form of patches and two prescription medications (varenicline and bupropion) are subsidised by the government when prescribed by a doctor, which both increases access to these treatments by reducing their cost, and also places cessation in a medical context. However, while the medical profession supports treating smoking as a medical issue in their professional guidelines but there is some resistance amongst clinicians to routinely implementing these guidelines in doctor-patient consultations (Zwar & Richmond, 2006).

It should be noted that while NRT and prescription medications have demonstrated efficacy in clinical trials, there remains a debate within the tobacco control community about the extent to which clinical trial results translate into real world effectiveness (Alpert et al., 2013; Kasza et al., 2013). Even the most intensive treatment programs that involve the provision of pharmacological cessation aids as well as group or individual counselling lead to quitting in only about one quarter of smokers (Hartmann-Boyce et al., 2013). This means that 75% of smokers continue to smoke after using these cessation aids when combined with behavioural support. Because of this, there are also public health advocates in Australia that publicly oppose the promotion of smoking cessation medications and instead promote unassisted cessation as the best way for smokers to quit smoking (MacKenzie & Rogers, 2015). While a small increase in the success of quit attempts through increased use of cessation medicines may be cost effective and worthwhile at a population level, the research reported in this thesis shows that at an individual level, smokers are cognisant that use of cessation medications often does not result in long-term abstinence.

Competing definitions of smoking cessation are evident in public discourse. These include that it is a matter of: willpower; individual responsibility; and a personal choice. These have been promoted by the tobacco industry to deflect attention from the industry's role in promoting and sustaining the market for an addictive product (Daube et al., 2008; White et al., 2013). Quitting unassisted has a low success rate, but in this research failures were attributed by participants to a lack of willpower, or the belief that unsuccessful quitters had not yet reached the point where they “really” want to quit. Also, the findings indicate that personal experience was often the foremost consideration when smokers explained the reasons for their preferences for different cessation methods. These findings align with the research of Smith et al. (2015) who found that those who had quit unassisted prioritised “lay knowledge directly from personal experience and indirectly from others over professional or theoretical knowledge” (Smith et al., 2015, p. 1).

### **10.3. Do smokers believe tobacco dependence is based in their brain, and what are the implications for treatment choices and quitting self-efficacy?**

The growth of addiction neuroscience emerged in the sociopolitical context of moral panic about drugs, increased funding for treatment of drug dependence, and the growing prestige of neuroscience (Vrecko, 2010). Tobacco was often excluded from discussions around the role of the brain in drug addiction because of its widespread use, the influence of the tobacco industry, the long delay between initiation of smoking and adverse health outcomes such as lung cancer, and the fact that nicotine addiction typically does not impair societal functioning. While there is a long history of alcohol and illicit drug dependence being described as biologically-based “disease”, this model has been applied far less frequently to nicotine dependence. This provides an opportunity to study how the unique nature of tobacco dependence, and its history, influence the acceptability of biomedical labels and treatments.

In relation to the neurobiological basis of nicotine addiction, Chapters 7 and 8 revealed that most of the smokers that were interviewed for this research reported awareness that smoking influenced their brains. Many saw this as common sense information and could not list a specific source for this information. The all-encompassing nature of the brain in human behaviour and identity meant that everything was seen to affect the brain, including smoking tobacco. This suggests that those who speculate about the transformative impact that neuroscience information could have on lay understandings of addiction are probably



overestimating its likely effect. Neuroscience information is more likely to add detail to smokers' understandings, rather than fundamentally altering the way they perceive the nature of their dependence on cigarettes, and is likely to have even less impact on their smoking behaviour. This is consistent with research that shows that unless the role of the brain is brought to the fore via a brain injury or disorder, it is seen as operating in the background, as an object of "mundane significance" (Pickersgill et al., 2011).

While the neurobiological mechanisms of addiction were seen as interesting by many participants, most believed that this information would have a minimal impact on their smoking. During the interviews, some smokers stated that knowing how pharmacological treatments for smoking cessation worked in the brain made them more appealing. However, this rarely translated into a stated intention to use them in future quit attempts. The brain was sometimes seen as a "special organ", and a minority of participants were disturbed by the idea that medications worked in the brain. Concerns about side effects, particularly in relation to varenicline, seemed to outweigh any potential benefits of quitting using these medications. While the quantitative component of this project showed that endorsement of brain-based explanations of smoking were associated with increased intention to use medications for smoking cessation, this effect was of moderate size, and accounted for only a small proportion of the variation in intention to use medications in future quit attempts.

The findings presented here do not support the claim that emphasising the neurobiological basis of addiction leads to increased fatalism in smokers. While some participants did see the potential for neurobiological explanations of nicotine addiction to reduce feelings of control and self-efficacy, this was predicted to occur in "other" smokers, rather than themselves. The majority of smokers interviewed in Study 1 and Study 2 maintained that they had control over their smoking and would be able to stop when they really wanted to, even where the difficulty of this task was acknowledged. As already discussed, this emphasis on self-control and responsibility in relation to smoking has been linked to wider societal discourses that value autonomy, personal responsibility and inner strength (Gillies & Willig, 1997; Smith et al., 2015; White et al., 2013). In addition, the quantitative research presented here suggests that endorsement of the brain-based explanations of smoking may be associated with higher levels of self-efficacy. Together these findings should dispel concerns that educating smokers about the role of the brain in tobacco dependence will lead to feelings of fatalism about smoking and to a reduction in quitting self-efficacy.

Philosophical debates about the relationship between the brain and free will have primarily occurred in two areas: legal responsibility and addiction. In relation to legal responsibility, it has been argued that neuroscience will have a “transformative effect” on legal institutions by “transforming people’s moral intuitions about free will and responsibility.” (Greene & Cohen, 2004). This research on addiction showed the opposite, that existing beliefs about responsibility and choice are strongly maintained by addicted individuals.

The results presented here suggest that one of the key factors limiting the biomedicalisation of smoking cessation are strong lay beliefs about the role of willpower in stopping smoking. As noted by many others, lay beliefs are often grounded in the everyday experience of individuals, as well as the public discourses that they are exposed to. Theories such as lay representations theory have outlined how new representations become grounded, or anchored, in core cultural beliefs (Moscovici, 1988). In the case of addiction, new beliefs about the neurobiological basis of addiction are related back to the core cultural beliefs of free will and individual responsibility.

#### **10.4. Strengths and limitations of this research**

##### ***Representativeness of samples***

Due to time and resource constraints, it was not possible to recruit random samples for the studies described here. For Study 1, participants were drawn from a national market research database, to which they had already been recruited via random digit dialling. Households selected for the study were limited to those who resided in the Brisbane area. Quotas were set for age and gender in order to recruit a diversity of participants. However, those who were willing to respond to a market research company, and to participate in a one hour face-to-face interview, may differ from those who refused to join the panel, or who were on the panel but refused to be interviewed.

For Study 2, participants were again limited to the Greater Brisbane area to allow for face to face interviewing. While an effort was made to recruit using a range of methods in order to ensure participant diversity, the majority of participants came from a single online classified site, which limits their representativeness. Indeed, it was more difficult to recruit older adults

for Study 2, which may be the result of reduced internet access in this bracket (Australian Bureau of Statistics, 2014). A senior's database was also used to compensate for this limitation.

For Study 3, an online market research panel was contracted to recruit participants. While every effort was made to select a company that was able to provide the most robust sample of smokers, and quotas were set in relation to age and gender, the sample recruited may not be representative of the whole population of smokers. While the survey was completed by a large, national sample, it is unclear how different those who make up the online panel are from the general population of Australian smokers on the variables of interest to this study. Despite this limitation, the use of market research companies is becoming common in addiction research and Section 4.6 lists a number of ways that researchers can ensure they are getting the best sample possible using this recruitment method.

### ***Cross-sectional design***

Because this research was cross-sectional, inferences about directionality and causation cannot be drawn. The qualitative research reported here shows that many factors may influence smokers' preferences for different quitting methods. These factors include: one's own past experience with a particular method; the vicarious experiences of family, friends, and acquaintances; beliefs about the strength of one's addiction to smoking; beliefs about the nature of addiction to smoking; beliefs about the side effects of the cessation method; the cost and convenience of using the method; general attitudes towards taking medications for health problems; and trust in those promoting the method. Of these considerations, personal experience and concerns about side effects were particularly salient for many of the smokers who were interviewed. The role of personal experience in decision-making about smoking cessation was reflected in the quantitative survey that found previous use of cessation aids was the strongest predictor of intent to use in a future quit attempt. Providing neuroscience information about the mechanisms of addiction to smoking is unlikely to have a large impact on smoking behaviour or quitting intentions given the complexity with which people develop treatment preferences, with differential weightings for each factor, and a comparison of the costs and benefits along each of these dimensions.

Qualitative research informed the development of the quantitative survey but it is possible that there were other influential variables that were not measured. These might include the views of smokers' regular treating general practitioner about smoking and the role of cessation

medications, or exposure to pharmaceutical company advertising (although this is less likely in Australia, where direct to consumer advertisements of prescription medications are not permitted). It would be of interest to conduct similar research in the USA or New Zealand, where direct to consumer advertising of prescription medications is legal. Furthermore, replicating this study in a country that has adopted a greater emphasis on assisted cessation in their national approach to reducing smoking (such as the UK) would allow exploration of the impact of emphasising access to professionally assisted smoking cessation at the national level on smokers' cessation method preferences and attitudes toward the BDMA.

### *Eliciting stakeholder attitudes towards novel biotechnology and scientific findings*

Eliciting views about specific scientific developments or novel medical technologies can be difficult where prior knowledge is expected to be low, or where people have not yet had the time, reason or opportunity to form explicit attitudes. Providing participants with insufficient background on the topic may lead to confusion or feelings of inadequacy. The method used here, of providing participants with a brief summary of the science in everyday language, is a method has been used in other studies on PUS (Buchman et al., 2013; Choudhury et al., 2012; Horstkötter et al., 2014). Providing some background gives participants a stimulus to respond to and allows researchers to observe differences between participants in the interpretation of the same material.

Of course, providing such a stimulus inevitably shapes the responses of participants. Therefore, it is important that researchers make it clear when participants are responding to a specific stimulus, and provide sufficient detail about it. In some research in this field it is unclear what stimulus or interview question participants are responding to. For example, in some empirical studies on the BDMA, it is unclear whether participants were asked if they thought that addiction was “a disease”, or whether they were asked about the more recently terminology of addiction as a “brain disease” (Morphett & Meurk, 2013). Also, there has been some conflation of neuroscience and genetics, with the term “neurogenetics” being used by some social scientists (Dingel et al., 2011). While there are important linkages between genetics and neuroscience, it is important to develop at least a rudimentary knowledge of how stakeholders interpret neuroscience so that similarities and differences between lay conceptualizations of genetic information and neuroscience information can be assessed (Green 2006).

## **10.5. Clinical and public policy implications of the findings**

### ***The utility of the brain disease model of smoking***

Existing smoking cessation aids are only moderately effective and there are no signs that this situation will change in the near future. While it is unclear to what extent the optimism about biomedical treatment for nicotine addiction will be realised in the longer term, Ronald Green (2006) has warned scientists against exaggerating the therapeutic potential of neuroscience research. He writes that there is a “therapeutic gap” that is associated with translating basic science findings into clinical therapeutics, and that giving false hope about cures can have detrimental effects.

"There is a powerful tendency in all biomedical areas to offer enticing therapeutic visions as a way to elicit public support or increase governmental funding for research. Yet the same public enthusiasm that leads to increased funding can turn into budget cuts, resentment, and even research restrictions, when, instead of cures, the actual payoff is burdensome knowledge and a host of new ethical quandaries produced by poor therapeutic options." (Green 2006, p. 110)

The research presented in this thesis suggests that labelling smoking as a “brain disease” will have limited utility, and it may be that smokers themselves will not be strongly accepting of biomedical approaches to smoking until they can see evidence of the benefits of treating tobacco dependence in this way. The strong form of the BDMA was not acceptable to the majority of the smokers interviewed or surveyed. It was described by many as a stigmatizing and inaccurate description of smoking. Most agreed that smoking did influence their brains; it was the label of “disease” that was rejected.

The reasons that smokers rejected the brain disease label echoed critiques that have been made by academics, namely: that the BDMA reduces the complexity of smoking; does not account for individual choice and personal preferences for unassisted quitting; and could increase the stigma associated with smoking. As described in Chapter 2, Farrimond, Joffe and Stenner (2010) describe four different smoking identities. Importantly, they note that these identities are “produced relationally.” That is, smokers often describe their own smoking in opposition to alternative ways of understanding smoking. They write that “appealing to one ‘identity’ through health promotion might inadvertently alienate those with a different smoker identity. They recommend “highlighting the multiplicity of smoker identities” in interventions

(Farrimond et al., 2010). The findings presented here suggest that portraying smoking as a purely biomedical issue risks angering and antagonising some smokers. Because the “brain disease” description is incompatible with the way that most smokers view their own smoking, there seems to be little utility in promoting its use in the public domain.

Given the complex and contradictory ways that neuroscience information about nicotine addiction is interpreted by smokers, caution should be applied when including such information in health promotion materials. Those developing messages on smoking and the brain should be aware that such information is unlikely to have much, if any, impact at a population level. In addition, there are risks to emphasising this information, such as the possibility of stigmatizing smokers by portraying their brains as different and abnormal; and antagonizing those who reject, and are suspicious of, a biomedical approach to smoking.

Neuroscience information about nicotine addiction could still be presented to smokers in clinical interactions or in public health materials to explain how smoking cessation medicines work. It is clear that neuroscience information about nicotine and smoking is already in the public sphere and it is information that is of interest to many smokers. Based on the data from the studies included in this thesis, the following recommendations are provided on how to incorporate neuroscience information about addiction into health professional materials for use in clinical interactions.

Where information about the neuroscience of nicotine addiction is presented, health promotion messages should also acknowledge the other factors that contribute to smoking. The role of willpower and autonomy should not be downplayed. Describing pharmacological cessation as an additional support to their willpower may better align with most smokers’ views about their reasons for smoking, may contribute to a more realistic assessment of the role of medications in quit attempts and may help to retain the sense of achievement in quitting smoking that many smokers value. Medication should not be presented to smokers as a magic bullet for smoking cessation because for many smokers this is unlikely to align with their previous experience of these medications or the testimony of personal contacts. Rather, medications should be presented as only one way of assisting a quit attempt and should be recommended as part of a holistic approach to quitting, including behavioural counselling, which has been shown to increase the effectiveness of cessation medications.

A balance is needed when discussing the difficulty of quitting smoking in health promotion materials. Biomedical explanations that describe smoking as a physiological addiction to nicotine that is difficult to quit may over-emphasize the difficulty of quitting and deter smokers from making a quit attempt. As Chapman and MacKenzie (2010) have argued, most former smokers have quit unassisted and many current smokers have achieved some period of abstinence. However, disregarding the difficulty that some smokers experience when quitting may lower low self-esteem and increase self-blame and fatalism in those who repeatedly fail in their quit attempts. In health promotion materials, where it is difficult to present the complex nuances of the BDMA, any acknowledgement of the difficulty that some smokers may have in quitting because of changes that smoking has made to their brain, should be immediately followed by positive information on smokers' ability to quit despite these changes.

A recent paper has suggested a contrary view, namely, that smokers' belief in their ability to quit is an "obstacle to treatment utilization." They suggest that healthcare practitioners "normalize the need for assistance" and "emphasize the difficulty of quitting without assistance" (Myers et al., 2015, p. 220). The research presented in this thesis suggests that this message is likely to be counter-productive to smoking cessation efforts. Unassisted quitting should not be presented as an inferior method for smoking cessation, and the difficulty of quitting unassisted for some smokers should be acknowledged, but not over-emphasised. Given the diverse accounts of smoking and addiction provided by participants in this project, as well as the different smoking identities outlined by Farrimond, Joffe and Stenner (2010), it is important for clinicians to elicit patient views about the nature of their smoking, and use these to tailor treatment plans and messages about cessation to the smoker. A biomedical approach that includes medications may suit some patients, but is likely to be rejected by others.

### ***Lay engagement with neuroscience information***

The increasing emphasis on the state of the brain in accounts of human behaviour has led to the development of fields such as Neuroethics (Levy, 2008) and Critical Neuroscience (Suparna, Saskia Kathi, & Jan, 2009). Those involved in these endeavours aim to anticipate the social consequences of brain-based accounts of behaviour for legal systems, health systems, education systems and other arenas. However, these fields have a heavy focus on ethics and there is a dearth of empirical evidence on the ways in which neuroscience information is interpreted and used by stakeholder groups. The research reported in this thesis shows that one

stakeholder group, those who smoke cigarettes, largely rejected reductionist accounts of smoking as an addiction to nicotine caused by changes in neurochemistry. While the brain was typically acknowledged to play a role in smoking behaviour, this did not negate the role ascribed to personal choice, motivation and willpower. While a minority of those interviewed for this research acknowledged the potential for neuroscientific understandings of addiction to lead to feelings of fatalism, this was not often perceived as personally relevant information, but rather as a way that the “other smoker” might interpret neuroscience information.

These findings support other empirical research on public understandings of neuroscience which has found that neuroscience information is interpreted through the lens of already-existing cultural worldviews and explanations of behaviour (Bröer & Heerings, 2012; Meurk, Carter, et al., 2013; Meurk et al., 2016; O’Connor & Joffe, 2013; Pickersgill et al., 2011). An issue deserving further research attention is the way that people conceptualise the link between their brain and behaviour. Work has examined how people understand the brain in relation to clinical disorders but there is little empirical research with more general populations. There are some exceptions, such as research on adolescents’ understandings of the role that their brains play in behaviour said to be typical of teenagers, e.g., risk taking (Choudhury et al., 2012). This research found that adolescents considered neurological explanations of adolescent behaviour to be inadequate and preferred to discuss psychological and social influences on behaviour. Another recent exception is work by O’Connor (2013), who conducted interviews with people living in London about their views on neuroscience research and its import. Neuroscience research did not have great relevance for most participants, although the brain was often equated with a person’s self or essence. This however, did not result in a form of determinism, where the brain is perceived as a disembodied actor that is causing the individual to feel or act a certain way. Instead, the role of free will was emphasised. This leads to questions about whether people think of psychological and neurological states differently, or if they consider them to be the same thing? Experimental research shows that describing an action in neurological terms does not reduce attributions of personal responsibility when compared to a psychological account (De Brigard, Mandelbaum, & Ripley, 2009). It is clear that people’s ideas about neuroscience and the role of the brain in behaviour are complex and can shift according to context. Therefore it is important that empirical research is conducted with various groups in diverse settings before general claims about the transformative impact of neuroscience on public understandings are made.



## **10.6. Directions for future research**

This project has focused on lay understandings of smoking, smoking cessation, and the role of the brain in tobacco dependence. Research examining the same topics in health professionals would complement this research. Such research could examine the extent to which doctors and other health professionals see smoking as a medical problem that requires medical treatment or as a social problem that should be addressed by public policy. It could also examine their attitudes towards unassisted quitting and how health professionals accommodate the preferences of smokers when discussing quitting options. It could evaluate clinicians' knowledge of the neurobiological mechanisms of nicotine addiction, and explore how they integrate this information into their clinical interactions with smokers.

Despite the increasing use of prescription medications for smoking cessation, very little research has been done on smokers' beliefs and attitudes toward the use of medications for smoking cessation and their understanding of how these medications work. An exception is a study by Vogt, Hall and Marteau (2008) who found that bupropion was perceived as an "unnatural" substance that could cause damage to the body. This aligns with general research on medication use, which has found that many patients are reluctant to use medication. Medicines are sometimes perceived as foreign, unnatural substances that can produce dependence and serious adverse side effects (Horne & Weinman, 1999; Horne et al., 1999). It has been suggested that labelling requirements for smoking cessation medications may make smokers wary about taking these medications, even when some of these risks are trivially small in comparison to the well-known health harms caused by cigarette smoking (Shiffman et al., 2008). This may be because of the immediacy of medication side effects, compared to the long-term potential for health harms associated with smoking. It could also be the case that smokers are mistaking the symptoms of nicotine withdrawal for medication side effects, although there is insufficient evidence from current studies to draw this conclusion. It has been suggested that explaining the biophysical mechanisms by which smoking cessations work could be an effective intervention to increase use of such aids (Finnell, 2000; Raupach et al., 2010; Vogt et al., 2008). The qualitative research reported here suggests the effects of such an intervention are likely to be complicated. Some participants stated that knowing the mechanisms by which prescription medication varenicline worked increased their positive perceptions of it. But many of those still reported no intention to use it. In addition, a minority were very concerned about

the fact that varenicline entered, and influenced, their brain. Providing detail about how medications work in the brain may dissuade some people from using it, despite the contradiction inherent in the fact that they are dependent on a psychoactive substance that causes lung cancer, heart disease and has many other negative health effects. While this research begins to address the lack of research on smokers' attitudes towards using medications for smoking cessation, more research is required in this area, especially in relation to the use of prescription medications other than NRT.

It is clear that public understandings of smoking have altered dramatically in recent decades. However, there is very little empirical evidence on the effects that incremental changes in public perceptions of smoking may have had on the behaviour of smokers, and especially on their interest in attempting to quit and their confidence in their ability to do so. There are repeated time-series studies, and longitudinal studies, that map smoking behaviours and attitudes in various countries. Examples include the International Tobacco Control 4-Country Survey (Kasza et al., 2013), the National Drug Strategy Household Survey in Australia (Australian Institute of Health and Welfare, 2014), and the Smoking Toolkit Study in the UK (Fidler et al., 2011). Longitudinal studies could map various beliefs about smoking over time and evaluate their relationships with the quitting strategies that smokers have used and their quit successes. These beliefs could include the belief that medication is required for a successful quit attempt, that continued smoking is due to a nicotine addiction, and that addiction is based on changes in brain chemistry. Another line of exploration that would complement the self-report data presented here are behavioural choice experiments to examine the impact of neuroinformation on actual behaviour. Such experiments have been conducted to explore the impact of genetic information. For example, one study found that participants told they had an increased genetic susceptibility to alcoholism were more likely to enroll in a responsible drinking workshop (Dar-Nimrod, Zuckerman, & Duberstein, 2013). Another study found that informing light smokers that they had a higher risk of nicotine dependence due to their genetics increased quit rates at 30 days (Lipkus, Schwartz-Bloom, Kelley, & Pan, 2015).

Another important area of future research is the link between beliefs about nicotine addiction and attitudes towards e-cigarettes. The increasing use of e-cigarettes around the world has caused sometimes acrimonious debates in the field of tobacco control (Gartner & Malone, 2014). The regulatory landscape in relation to e-cigarettes is changing rapidly, and it is unknown whether e-cigarettes will be banned; regulated and incorporated into a medical

approach (much like NRT was); or whether they will become widely accepted as a recreational consumer product. One argument for making e-cigarettes widely available for smokers is that it is difficult for many smokers to quit due to a chemical dependence on nicotine. The use of e-cigarettes has been portrayed as a harm-reduction strategy that radically reduces the risks of tobacco-related harm by allowing those who are nicotine dependent to obtain nicotine in a form that satisfies smokers' addiction without the harms of smoking tobacco. The research reported here shows that smokers do not always equate smoking with nicotine addiction, potentially limiting the perceived utility of e-cigarettes for some smokers. However, a substantial minority of people who use e-cigarettes use non-nicotine liquid, and the hand-to-mouth action is posited to be another factor that may help smokers quit smoking by providing a behavioural replacement. Studies exploring smokers' attitudes towards e-cigarettes should further explore smokers' understandings of the role of nicotine in smoking and tobacco dependence.

In summary, this research demonstrates that biomedical explanations of smoking do not encompass the complexity of addiction as an everyday, lived experience. This study points to the importance of documenting the ways in which those who experience addiction as an everyday reality interpret and respond to changing conceptualisations of addiction.

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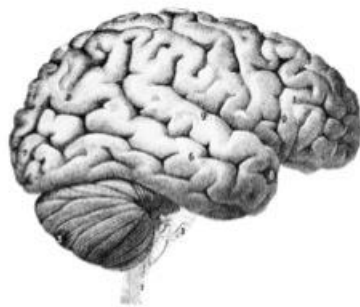
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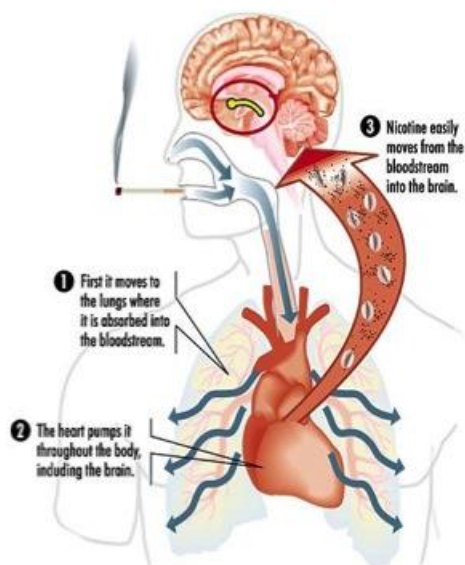
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## Smoking and the brain

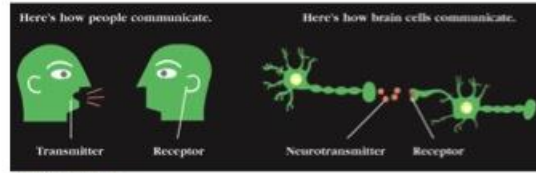


When you smoke tobacco, nicotine enters your bloodstream from your lungs.



The nicotine then spreads throughout your body and brain where it stimulates specific regions of the brain.



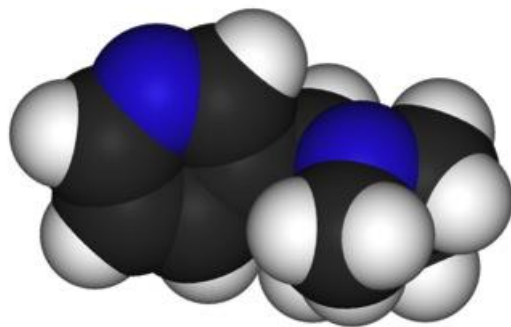


Concept courtesy: E.K. Rubin

Nicotine binds to sites in the brain called receptors. These receptors are normally activated by the chemical acetylcholine, which is produced naturally in the body.

Acetylcholine and its receptors are involved in many activities, including breathing, heartbeat, memory, alertness, and movement.

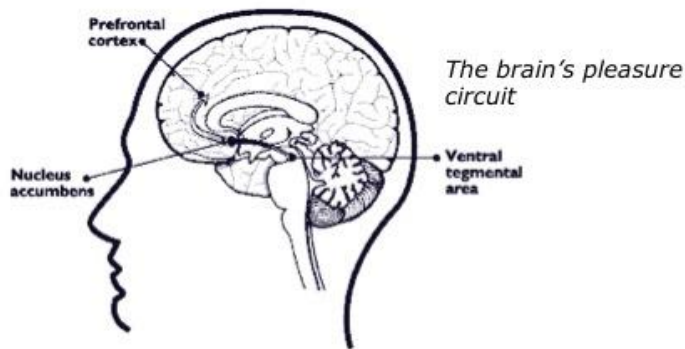
Because the chemical structure of nicotine is like that of acetylcholine, it can also activate these receptors.



*Nicotine Molecule*

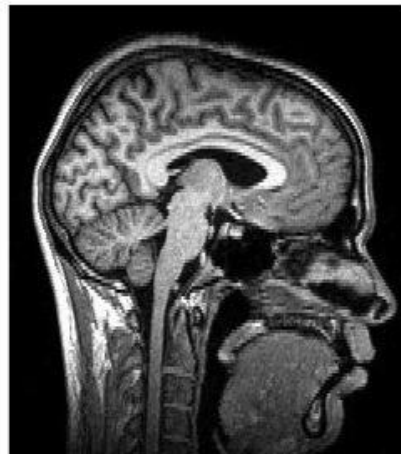
But unlike acetylcholine, when nicotine enters the brain it can disrupt its normal functioning.

Research shows that nicotine also leads to the release of the chemical dopamine in the parts of the brain involved with pleasure.



This release of dopamine is similar to that of drugs like heroin and cocaine. Dopamine produces the feelings of pleasure many smokers experience.

Regular nicotine use increases the number of these receptors and makes them less sensitive. These changes lead to the development of tolerance to nicotine, which is when more drug is needed to achieve the same effects.



Once tolerance has developed, a nicotine user must regularly supply the brain with nicotine in order to maintain normal brain functioning. If nicotine levels drop, unpleasant withdrawal symptoms may occur.

A number of medications that are used to help smokers quit, work by acting on the brain.

Nicotine replacement therapy, in the form of patches, gum or lozenges, provides the brain with nicotine, but in smaller amounts than cigarettes, reducing withdrawal symptoms.

Champix is a prescription medication that activates nicotine receptors in the brain. It also stops nicotine from attaching to the receptors. This helps to satisfy cravings and reduce withdrawal symptoms.



## Appendix B – Information sheet and informed consent for Study 2

**Project Title: Smokers’ understanding of nicotine addiction**

### Investigators

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### About the project

This project aims to examine the understandings of Australian smokers in relation to smoking and nicotine addiction. We would like to know your views about why people smoke and how to prevent people starting to smoke. In particular, we are interested in:

- how you understand your smoking
- your thoughts about addiction in light of recent biomedical research
- your ideas on treatments for nicotine addiction.

### Why this is important

Recent biomedical research on smoking could change the way that smoking is understood and lead to new technologies for the prevention and treatment of smoking. We want to know what smokers think because the impact of this information will depend on how well it is understood and accepted by smokers. This research will help to inform public policies responding to new technologies for the treatment and prevention of smoking, and existing policies to manage smoking.

### Eligibility

You are eligible to participate in the study if you are aged 18 years or over and smoke tobacco every day.

### What you will be asked to do

If you decide to be involved in this study you will be asked to participate in an interview lasting approximately one hour. Interviews will be conducted by researchers at a private location at The University of Queensland or another location that is convenient for you.

In the interview you will be asked a series of questions about your smoking behaviour, your ideas about why people smoke, and your attitudes and opinions about different forms of prevention and treatment for smoking.

The interview will be audio recorded, transcribed verbatim, and then analysed and presented in a way that does not identify you.

### **Reimbursement**

All participants will be provided with a \$20 Coles Myer voucher in recognition of their time.

### **Your rights**

If you consent to participate you are still free to withdraw from the study at any time without stating a reason. Upon withdrawal from the study all of your responses will be destroyed if you request.

Your confidentiality and privacy will be maintained at all times. Your name or other personal information will not be linked to your interview data and will be stored in a safe and secure location at the University of Queensland.

### **Need further information?**

The research staff will be able to discuss any questions you may have and will provide guidance on where to get further information or assistance. A copy of the research findings will be made available to you by post or email if you wish.

This study has been cleared by the human ethics committees of The University of Queensland in accordance with the National Health and Medical Research Council's guidelines. If you have any question about your participation in this study please contact Kylie Morphett at the UQ Centre for Clinical Research on (07) 3346 5473 or [k.morphett@uq.edu.au](mailto:k.morphett@uq.edu.au).

If you would like to speak to an officer of the University not involved in the study, you may contact the Ethics Officer on 3365 3924.

**We greatly appreciate your help and cooperation in this important study.  
Thank you very much.**

## Appendix C – Interview schedule and demographic survey for Study 2

### 1. Thoughts about own smoking

- a. How old were you when you started smoking?
- b. Can you tell me about how you started smoking? What was the main reason you started smoking?
- c. What are the main reasons that you smoke now?
- d. How much control do you feel you have over your smoking? Could you give up if you wanted to? Why/why not?
- e. How difficult do you think it would be to quit smoking? What would make it difficult/not difficult?
- f. How much personal responsibility do you feel for starting to smoke?
- g. How much personal responsibility do you feel for continuing to smoke?
- h. Do you feel others have any responsibility for your smoking? Do you feel your peers have any responsibility? Your family? The tobacco industry?
- i. Do you feel that smokers are stigmatised? What makes you feel this way? Can you give any examples?

### 2. Addiction

- a. Do you consider yourself addicted to nicotine? What makes you think you're addicted/not addicted?
- b. How do you know when someone is addicted to nicotine?
- c. Why do you think you became addicted to nicotine?
- d. Why do other people become addicted to nicotine? (prompt if necessary: social influences, genetics, personality factors, substance itself). Which factor is most important and why?
- e. Why can some people quit smoking more easily than others?
- f. Smoking is frequently described as a habit. What do you think about the idea that smoking is a habit?

### 3. Treatment and Prevention

- a) What sort of things should be done to prevent cigarette smoking by young people?
- b) What do you think the government should do about smoking?
- c) Should the government pay for stop smoking medications? Why/why not?
- d) Should the government provide free medical treatment to smokers for smoking related health problems? Why/why not?
- e) Do you think smokers should pay more than non-smokers for health insurance?
- f) Do you think a smoker should have an equal opportunity for a lung cancer transplant as a non-smoker? Why/Why not?
- g) What is your view on the following methods for people trying to quit smoking? [prompt if required: are they effective, safe, appropriate, what do you know about it?]
  - (a) No treatment – cold turkey
  - (b) Nicotine replacement therapy
  - (c) Prescription medication (eg Champix, Zyban)
  - (d) Counselling, including quitline
  - (e) Self-help materials e.g., books or information pamphlets
  - (f) Any other methods that you have thoughts on.

- h) Can you tell me a bit about any quit attempts that you have made? (how long did you quit for? did you find it easy or difficult? what method/s did you use?)
- i) If you were going to quit now, which method would you use? Why?
- j) Do you think you will quit smoking? When?
- k) What is your understanding of the effects of nicotine?

Nicotine has been shown to produce long-term changes in the brain of smokers.

[A short powerpoint presentation will be presented to participants on the neurobiology of nicotine addiction at this point in order to give them some background to the remaining questions].

#### **4. Impact of information about neurobiology of nicotine addiction**

- a. Were you aware of the ways in which nicotine acts on the brain? If so, where did you hear about it?
- b. To what extent do you think that smoking affects your brain?
- c. How does this information influence your understanding of your smoking? In what ways? How does this information make you feel about your smoking?
- d. Does it make stopping smoking seem easier or harder? How does it affect your belief in your ability to stop smoking?
- e. Does it affect your view of your personal responsibility for smoking? How?
- f. Does this information affect your view on the best method for quitting?  
[prompt: those discussed above eg cold turkey, medication, NRT, self-help materials].

#### **5. Brain disease model**

- Based on the information presented earlier, some scientists have claimed that “addiction is a brain disease.”
  - a. What do you think about the idea that nicotine addiction is a brain disease?
  - b. Do you think that describing nicotine addiction as a brain disease will increase or decrease the stigma of smoking? Why?
  - c. Do you think that describing nicotine addiction as a brain disease is a good or bad thing? Why?
  - d. How might the view that nicotine addiction is a brain disease influence your smoking?
  - e. Do you think that medical research will lead to more effective treatment for nicotine addiction?
  - f. Have you heard nicotine addiction described as a brain disease before today? If so, where did you hear it?
- 6. Is there anything else that you would like to say about this topic that we did not cover that you think is important?

**CAN YOU PLEASE FILL OUT THIS SHORT SURVEY?**

## Smokers' understanding of nicotine addiction – Survey

ID Number:

The answers to these questions will be used to make sure that we have spoken with a range of different people with different experiences. Please do not write your name on the form.

1. Are you? (tick one)

Male  Female

2. What is your age? ..... (years)

3. What is your employment status? (tick as many as apply)

- Working full time
- Working part-time
- Studying full-time
- Studying part-time
- Doing home duties
- Unemployed
- Retired

4. What is your highest level of education? (tick one)

- Did not complete senior secondary school
- Completed senior secondary school (e.g. Higher School Certificate)
- Trade/Apprenticeship (e.g. hairdresser, chef)
- Certificate (Level III or higher) or diploma (e.g. child care, technician)
- University degree (e.g. Bachelor)
- Postgraduate university degree (e.g. Graduate Diploma, Masters, PhD)

5. What is the postcode of your usual residence? .....

6. What is your occupation? (if applicable) .....

7. How long after waking do you wait before having your first cigarette (in mins?)

- 0-5 minutes
- 6-30 minutes
- 31-60 minutes
- 61 minutes or more

8. How many cigarettes do you smoke per day at present?

- 1-10
- 11-20
- 21-30
- 31 or more



9. How often do you get strong urges to smoke?

- Never
- Less than daily
- Daily
- Several times a day
- Hourly or more often
- Not applicable
- Don't know

10. Quit attempts

a. Have you ever tried to quit smoking?

- Yes
- No

b. What is the longest you have gone without smoking in the last 5 years?

- Have not stopped
- Less than a day
- A day
- Up to a week
- Up to 4 weeks
- Up to 3 months
- Up to 6 months
- Up to 12 months
- Longer than 12 months

c. Have you used any of the following to help you to quit smoking?

- Discussed smoking and health at home
- Rung the "QUIT" line
- Asked your doctor to help you stop smoking
- Used nicotine gum, nicotine patch, or nicotine inhaler
- Used a smoking cessation pill (e.g. Zyban, Champix)
- Brought a product other than nicotine patch, gum or pill to help you quit
- Read "How to Quit" literature
- Used the internet to help you quit
- Done something else to help you quit? Please specify.....
- None of the above

11. Are you planning to give up smoking?

- Yes, within 30 days
- Yes, after 30 days, but within the next 3 months
- Yes, but not within the next 3 months
- No, I am not planning to give up

***Thank you for your participation in this study.***

## Appendix D - Quantitative survey questions

First, we need to ask you a few questions to determine your eligibility for this study.

Do you smoke cigarettes every day?

1. Yes
2. No [If no, not eligible]

Have you smoked at least 100 cigarettes (manufactured or roll-your-own), or the equivalent amount of tobacco in your life? [If no, not eligible]

What is your age in years? (text box, 2 digit numeric) [If less than 18 terminate, too young].

Are you ...

1. Male
2. Female

Are you ...

1. An Australian citizen
2. An Australian resident
3. A resident in some other country [If yes, not eligible].

Were you born in Australia?

1. Yes
2. No

### **Introduction [Presented if eligible based on screening questions].**

The purpose of this study is to explore smokers' understandings of addiction to cigarettes and their attitudes towards different methods of quitting. This study is being undertaken by the University of Queensland. Participation will involve completing an online questionnaire that

will take approximately 25 minutes. This study has been approved by the Human Research Ethics Committee of the University of Queensland (Approval Number 2009001022).

You are free to withdraw from the study at any time by simply exiting the survey webpage. The University of Queensland will not receive any personal information about you, including your name, address or email. Once your survey has been completed, the University will be unable to delete your data, as we will not be able to link your personal details with the survey data.

If you have any questions about your participation in this research project, please contact Kylie Morphett at the University of Queensland Centre for Clinical Research on 07 3346 5473 or [k.morphett@uq.edu.au](mailto:k.morphett@uq.edu.au). If you would like to speak to an officer of the University not involved in the study, you may contact the Ethics Officer on 07 3365 3924.

Do you consent to participate in this research project? In doing so, you agree to Complete one 25 minute questionnaire about your smoking, your thoughts about methods of quitting and addiction to cigarettes.

1. Yes, I consent to taking part under the stated conditions
2. No, I prefer to not take part

### **Section 1. Smoking variables**

**Q1. On average, how many cigarettes do you smoke each day, including both factory-made and roll-your-own cigarettes?**

Numeric text field. Allow 3 digits.

**Q2. How soon after waking up do you smoke your first cigarette?**

1. Within 5 minutes
2. 6-30 minutes
3. 31-60 minutes
4. After 60 minutes

**Q3. How old were you when you started to smoke cigarettes regularly?** (Please try to estimate if you cannot remember exactly).

Numeric text field. Allow two digits.

**Q4. Have you ever tried to quit smoking?**

1. Yes
2. No (SKIP TO Q7)

**Q5. What is the longest time period you have ever quit smoking for?**

1. Less than a day
2. 1-2 days
3. 3-7 days
4. More than 1 week and up to a month
5. More than 1 month and up to 3 months
6. More than 3 months and up to 6 months
7. More than 6 months
8. Don't know/can't remember

**Q6. How many quit attempts, that lasted at least 24 hours, have you made in the last year?**

(Please try to estimate if you cannot remember exactly)

1. None
2. 1-2
3. 3-4
4. 5 or more

**Q7. How much do you want to give up smoking?**

1. Not at all
2. A little bit
3. Quite a bit
4. Very much

**Q8. How much do you enjoy smoking?**

1. Not at all
2. Not particularly

3. Quite a bit
4. Very much.

**Q9. Are you planning on giving up smoking?**

1. Yes, within 30 days
2. Yes, after 30 days but within 3 months
3. Yes, but not within the next 3 months
4. No, I am not planning on giving up.

**Q10. If you decided to give up smoking completely in the next six months, how sure are you that you would succeed?**

1. Not at all sure
2. Slightly sure
3. Moderately sure
4. Very sure
5. Extremely sure

**Q10a. Is there any additional information you would like to provide about your smoking and/or quit attempts?**

1. Yes [Open text field]
2. No

**Q10b Please select the one statement that comes closest to what you believe. Smoking causes :**

1. Long lasting changes to the way the brain works
2. Temporary but substantial changes to the way the brain works
3. Minor changes to the way the brain works
4. No changes to the way the brain works
5. I have no idea what effect smoking has on the brain

**Q10c To what extent do you agree with the following statement? (Strongly agree, agree, disagree, strongly disagree, don't know).**

1. Smoking is a brain disease
2. Smoking changes the chemistry of the brain

3. Smoking damages the brain
4. Smoking is a brain disorder
5. Some smokers have an increased chance of becoming addicted to cigarettes because of their genes

## **Section 2 – Attitudes towards quitting methods**

**Q11. How effective do you think the following method is for quitting smoking? (Not effective, somewhat effective, very effective, don't know).**

1. Cold turkey (quitting completely without professional or medical help, and without nicotine replacement therapy)
2. Cutting down or weaning off cigarettes (without professional or medical help, and without nicotine replacement therapy)
3. Nicotine replacement therapy (e.g., patches, gum, lozenge, inhaler)
4. Prescription medication from a doctor (e.g., Champix, Zyban)
5. Counselling from a private counsellor
6. Calling the Quitline
7. Reading books or brochures about quitting smoking
8. Online self-help materials about quitting smoking
9. Electronic cigarettes (e-cigarettes)
10. Hypnotherapy
11. Acupuncture

**Q12. Have you ever tried to quit using the following methods? Please select all that apply.**

1. Cold turkey (quitting completely without professional or medical help and without nicotine replacement therapy)
2. Cutting down or weaning off cigarettes (without professional or medical help, and without nicotine replacement therapy)
3. Nicotine replacement therapy (e.g., patches, gum, lozenge, inhaler)
4. Prescription medication from a doctor (e.g., Champix, Zyban)
5. Counselling from a private counsellor
6. Calling the Quitline
7. Reading books or brochures about quitting smoking
8. Online self-help materials about quitting smoking

9. Electronic cigarettes (e-cigarettes)
10. Hypnotherapy
11. Acupuncture
12. I have used a method not listed above to quit
13. I have not tried to quit

**Q12b. Please describe each other method you have used, one method in each box (10 additional methods allowed)**

**Q13. How helpful did you find the following method? [List all methods tried from 12a and 12b].**

1. Not at all helpful
2. A little bit helpful
3. Somewhat helpful
4. Quite helpful
5. Very helpful

**Q14. Have you ever thought about using a “clean” nicotine product (e.g., nicotine gum, electronic cigarettes etc) as a long-term substitute for smoking cigarettes? That is, using it every day as an alternative to smoking?**

1. No
2. Yes, but not seriously
3. Yes, seriously

**Q15. If you decided to make a quit attempt, how likely is it you would use the following quitting method? (Definitely wouldn't use, Probably wouldn't use, Probably would use, Definitely would use, Don't know).**

1. Cold turkey (quitting completely without professional or medical help, and without nicotine replacement therapy)
2. Cutting down or weaning off cigarettes (without professional or medical help, including nicotine replacement therapy)
3. Nicotine replacement therapy (e.g., patches, gum, lozenge)
4. Prescription medication from a doctor (e.g., Champix, Zyban)

5. Counselling from a private counsellor
6. Call the Quitline
7. Read books or brochures about quitting smoking
8. Online self-help materials about quitting smoking
9. Electronic cigarettes (e-cigarettes)
10. Hypnotherapy
11. Acupuncture

**Q16. If you were deciding which method to use to quit smoking, how important would the following be to your decision ? (Of no importance, A little important, Somewhat important, Important, Very important).**

1. The convenience of the method
2. The likelihood of side effects
3. The seriousness of side effects
4. How well it works
5. How much it costs
6. How addicted you are to cigarettes
7. Whether you have a physical addiction to cigarettes. (That is, experience unpleasant symptoms if you go without a cigarette.)
8. Your family or friends' experience of using it
9. Your own experience with it

**Q17. Is there any additional information you would like to provide on your view about quitting methods? Please type in your thoughts in the box below. If you have nothing to add, just type in No. [Open text box].**

**Q18. To what extent do you agree or disagree with the following statement?**

**Please note that by the term “medication” we mean prescription OR over the counter medications for smoking cessation e.g., Champix, Zyban, nicotine patches, nicotine gum, nicotine lozenge or nicotine inhaler. (Strongly agree, Agree, Disagree, Strongly disagree, Don't know)**

1. Quitting cold turkey (without medications) is the best way to quit



2. Quitting cold turkey is very difficult
3. Medications do not deal with important psychological or behavioural aspects of smoking
4. Knowing that medications are available makes stopping smoking seem easier
5. Smokers should try to quit without medication before using quit smoking medications
6. Medications are only helpful for heavy smokers
7. Willpower plays the biggest role in quitting success
8. If you don't have a strong desire to stop smoking, medications won't be helpful
9. Only those who are strong can quit smoking without any medication
10. Most people will need medications to stop smoking
11. Medications are only helpful for those who have a physical addiction to smoking.
12. Medications should be used in combination with counselling

**Q19. How much control do you feel you have over your smoking?**

1. No control
2. A little control
3. A moderate amount of control
4. A lot of control
5. Complete control

**Q20. How much help do you think you would need to quit smoking?**

1. No help
2. Some help
3. A lot of help

**Q21. How much control do you feel most other smokers have over their smoking?**

1. No control
2. A little control
3. A moderate amount of control
4. A lot of control
5. Complete control

**Q22. How much help do you think most other smokers would need to quit smoking?**

1. No help
2. Some help

3. A lot of help

**Q23. How likely is it that medical research will improve treatments for addiction to cigarettes?**

1. Very unlikely
2. Unlikely
3. Likely
4. Very likely
5. Don't know

**Q24. How likely is it that medical research will lead to a cure for addiction to cigarettes?**

1. Very unlikely
2. Unlikely
3. Likely
4. Very likely
5. Don't know

### **Section 3: Ideas about smoking**

**Q25. Below are some statements about reasons for smoking. Please indicate the extent to which you agree or disagree with these statements (Strongly agree, Agree, Disagree, Strongly disagree, Undecided)**

**I smoke because:**

1. I am addicted to cigarettes
2. I have a psychological addiction to smoking
3. I experience cravings for cigarettes
4. Smoking is an important part of my everyday routine
5. It helps me to socialise
6. I want to avoid withdrawal symptoms
7. I am addicted to nicotine
8. I have an addictive personality
9. It helps me deal with stressful situations
10. It has become a habit

11. I have a physical addiction to smoking

**Q26** Smoking researcher Robert West has written the following about smoking:

“You smoke because the nicotine you have been inhaling for all those years has changed your brain chemistry to create powerful urges to smoke. The urges come about because every puff on a cigarette sends a rapid nicotine "hit" to the part of your brain that makes you do things...These urges are triggered because nicotine has trained the part of your brain that gets you to do things to light up a cigarette whenever you find yourself in a situation where you would normally smoke.”

**How much do you think the statement above explains your own smoking?**

1. Not at all
2. A little
3. A lot
4. Completely
5. Don't know

**Q27. How much do you think the statement above explains why others smoke?**

1. Not at all
2. A little
3. A lot
4. Completely
5. Don't know

**Q28. If the above explanation for smoking was widely accepted, do you think it would increase or decrease the following?** (Decrease, No effect, Increase, Don't know)

1. The desire to stop smoking
2. Smokers' confidence in their ability to quit
3. Negative attitudes towards people who smoke?
4. The sense of personal responsibility that people feel for their smoking?
5. Sympathy towards people who smoke?
6. The number of people willing to seek medical treatment for their smoking

7. Feelings of guilt in those who smoke

**Q29. Some researchers in the USA have described addiction as a “brain disease.” To what extent do you agree that smoking is a brain disease?**

1. Strongly agree
2. Agree
3. Disagree
4. Strongly disagree
5. Don't know

**Q30. REASONS FOR RATING**

**Please type in the box below your reasons for saying you [DISPLAY RATING FORM Q28] that smoking is a brain disease. If you can't say just type in DK (for Don't know)**

Open text field.

**Q31. Imagine that a new "clean" nicotine product has been developed. This new product is as satisfying to use as smoking cigarettes. It is also as addictive as cigarettes, but it is far less harmful than cigarettes. It also costs slightly less than cigarettes. How interested would you be in using this product ?** (Not interested, Somewhat interested, Very interested, Don't know/unsure)

1. As a short-term aid for quitting smoking and nicotine (i.e. use for 6 months or less)?
2. As a long-term substitute for cigarettes (i.e. use for the foreseeable future)?
3. As a partial replacement for cigarettes to help you cut down the number of cigarettes you smoke?

**Q32. Please indicate how much you agree/disagree with the following statements. By “clean nicotine product”, we mean nicotine gum, nicotine lozenges, e-cigarettes or other nicotine vaporisers.** (Strongly agree, Agree, Disagree, Strongly disagree, Don't know)

1. Heavily addicted smokers should be encouraged to switch to less harmful clean nicotine products as long-term substitutes for cigarettes
2. All smokers should be encouraged to switch to less harmful clean nicotine products as long-term substitutes for cigarettes

3. Long-term use of clean nicotine products should be discouraged
4. Using clean nicotine products long-term is bad because it maintains addiction
5. Smokers should just quit smoking rather than switching to other products, like clean nicotine products
7. There is no point switching from smoking cigarettes to using clean nicotine products long-term because this is just swapping one addiction for another

**\*\*\*\*Section 4 – Some basic information about you**

**To finish we need your answers to a few questions about you that will help us to group answers together and make sense of what everyone has told us. Remember we will have no way of identifying you as an individual.**

**Q33. EMPLOYMENT STATUS**

**First, which of the following describes your main employment status** (select one only)

1. Employed or self-employed
2. Unemployed
3. A student
4. Home duties and/or carer
5. Volunteer or charity work
6. Retired or on a pension
7. Unable to work
8. Something else [SPECIFY]

**Q34. HIGHEST EDUCATION**

**What is your highest educational qualification?**

4. Bachelor degree or higher
3. Post school qualifications (e.g., certificate, diploma, apprenticeship)
2. Completed secondary school (Year 12)
1. Less than year 12

**Q35. POSTCODE**

**What is the postcode of your current address? (4 digit numeric box)**

## **Appendix E - Taverner Research Privacy Policy**

Taverner Research adheres to the Australian Market and Social Research (AMSRS) Code of Professional Behaviour, the Market and Social Research Privacy Principles, and all requirements of the Privacy Act.

In accordance with the AMSRS Code of Professional Behaviour, Taverner Research seeks to protect the privacy of individuals through protection of personal information. Market and social research is based on the willing co-operation of the public and the business community. Such co-operation depends on public and business confidence that market research is carried out honestly and objectively using processes that protect the identity and rights of individuals, and without any unwelcome intrusion.

As researchers, our responsibilities towards respondents include (taken from the text of the Code of Professional Behaviour for illustration purposes)

Respondents' identities must not be revealed without their consent to anyone not directly involved in the market research project (including the client who commissioned the work) or used for any non-research purpose.

Nobody shall be adversely affected or harmed as a direct result of participating in a market research study. Respondents must be able to check without difficulty the identity and bona fides of researchers.

Respondents' co-operation in a market research project is entirely voluntary at all stages; they must not be misled when being asked for their co-operation.

No child under 14 years shall be interviewed without parent's/ guardian's/responsible adult's consent.

All indications of the identity of respondents should be physically separated from the records of the information they have provided as soon as possible after the completion of any necessary fieldwork quality checks. The researchers must ensure that any information which might identify respondents is stored securely and separately from the other information they have provided, and that access to such material is restricted to authorised research personnel within the researcher's own organisation for specific research purposes (e.g. field administration, DP, panel or longitudinal studies or other forms of research involving recall interviews). To preserve respondents' anonymity not only their names and addresses but also any other

information provided by or about them which could in practice identify them (e.g. their company and job title) must be safe-guarded.

These anonymity requirements may be relaxed only under the following safeguards:

where the respondent has given explicit permission for this under the conditions of 'informed consent' summarised in Rule 4a and 4b

where disclosure of names to a third party (e.g. a sub-contractor) is essential for any research purpose such as data processing or further interview (e.g. an independent fieldwork quality check) or for further follow-up research. The original researcher is responsible for ensuring that any third party agrees to observe the requirements of this Code - in writing, if the third party has not already formally subscribed to the Code.

## Appendix F – Additional statistical analyses

### 1. Correlations between items on smoking and the brain (n=1538)

		Smoking is a brain disease	Smoking changes the chemistry of the brain	Smoking damages the brain	Smoking is a brain disorder
Smoking is a brain disease	Pearson Correlation Sig. (2-tailed)	1	.497** .000	.513** .000	.683** .000
Smoking changes the chemistry of the brain	Pearson Correlation Sig. (2-tailed)	.497** .000	1	.633** .000	.515** .000
Smoking damages the brain	Pearson Correlation Sig. (2-tailed)	.513** .000	.633** .000	1	.541** .000
Smoking is a brain disorder	Pearson Correlation Sig. (2-tailed)	.683** .000	.515** .000	.541** .000	1



## 2. Cross-tabulations for self-efficacy categories

Cross-tabs were conducted in order to examine differences between self-efficacy categories in relation to level of dependence, highest level of education, agreement that smoking changes the chemistry of the brain, and endorsement of the idea that it is a brain disease. Those who reported moderate and high self-efficacy were more similar to each other in relation to level of nicotine dependence and the proportion of those who had a low level of education than those who reported low self-efficacy. The former categories were combined in order to carry out bivariate analyses.

### 2.1. Self-efficacy by level of dependence cross-tabulation

			HSI Categories			Total
			Low dependence	Moderate dependence	High dependence	
Self-efficacy	Low self-efficacy	Count	138	307	276	721
		% within Self-efficacy	19.1%	42.6%	38.3%	100.0%
		% of Total	9.0%	20.0%	18.0%	47.0%
	Moderate self-efficacy	Count	152	243	103	498
		% within Self-efficacy	30.5%	48.8%	20.7%	100.0%
		% of Total	9.9%	15.8%	6.7%	32.4%
	High self-efficacy	Count	106	153	57	316
		% within Self-efficacy	33.5%	48.4%	18.0%	100.0%
		% of Total	6.9%	10.0%	3.7%	20.6%
Total		Count	396	703	436	1535
		% of Total	25.8%	45.8%	28.4%	100.0%

2.2. Self-efficacy by highest level of education cross-tabulation

			Highest education				Total
			Less than year 12	Completed secondary school	Post school qualifications	Bachelor degree or higher	
Self-efficacy	Low self-efficacy	Count	174	131	237	181	723
		% within Self-efficacy	24.1%	18.1%	32.8%	25.0%	100.0%
		% of Total	11.3%	8.5%	15.4%	11.8%	47.0%
Self-efficacy	Moderate self-efficacy	Count	77	91	177	154	499
		% within Self-efficacy	15.4%	18.2%	35.5%	30.9%	100.0%
		% of Total	5.0%	5.9%	11.5%	10.0%	32.4%
Self-efficacy	High self-efficacy	Count	35	52	82	147	316
		% within Self-efficacy	11.1%	16.5%	25.9%	46.5%	100.0%
		% of Total	2.3%	3.4%	5.3%	9.6%	20.5%
Total		Count	286	274	496	482	1538
		% of Total	18.6%	17.8%	32.2%	31.3%	100.0%