

**Estimating the prevalence of chronic medical co-morbidities in the
serious mentally ill in primary care.**

A modelling framework

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Abstract

An increasing body of evidence suggests that, in comparison to the general population, patients with severe mental illnesses such as schizophrenia or bipolar disorder have worse physical health and a far shorter life expectancy, due primarily to co-morbid chronic diseases.

The standardised mortality ratio for all forms of mental disorder is at least 1.5 and varies with the type and severity of the disorder. Whilst data on the prevalence of chronic diseases in primary care is available nationally, there is a lack of health intelligence on medical co-morbidities associated with chronic mental illnesses.

The aim of this PhD was to develop and validate epidemiological models for predicting expected prevalence of two major chronic medical conditions namely, coronary heart disease (CHD) and chronic obstructive pulmonary disease (COPD), on general practice data for people with concurrent serious mental illness (SMI) group.

The study probed the national epidemiological synthetic estimation of the two physical disorders to determine their prevalence within a local primary care setting and their co-existence within the serious mentally ill (SMI) group identified through the Quality Framework dataset (QOF) within GP practices and their localities. The expected prevalence was compared with recorded cases.

Methods

The national model used multinomial regression to arrive at odds ratios based on a basket of variables including age, sex, ethnicity, rurality and smoking status. This study examines the possibility of using a similar multi-nomial logistic regression model in conjunction with other locally sensitive data to map the expected risk at very small area level (GP practice level) in order to derive the expected prevalence of the two medical conditions at local levels.

The model takes into account local variations with adjustments made to obtain a more accurate estimation. These were applied to the local SMI datasets (QOF data) to establish co-morbidity levels. Validation was carried out using external data, including population-based epidemiological data and case-finding initiatives. The co-morbidity estimation of SMI with each and both conditions was derived using Bayesian methodology.

Results

Risk factors, odds of disease and expected prevalence of CHD and COPD were consistent with external data sources and supported trends from HsFE. Higher prevalence rates were associated with population deprivation, poorer quality and supply of primary health care services and poorer access to them. For both medical conditions they were under reported at local levels. The ratio of recorded to expected prevalence were significantly different ($p < 0.001$).

Medical co-morbidity prevalence associated with SMI was 2.5 fold greater than the general population. Case findings showed strong evidence of difference between expected and actual prevalence of the two diseases in the localities ($p < 0.001$).

Conclusion

The physical health of patients with severe mental illnesses is too often neglected, thus contributing to a compounded health disparity. The reintegration of psychiatry and medicine, with the ultimate goal of providing optimal services to this vulnerable patient population, represents the most important challenge for psychiatry today, requiring urgent and comprehensive action from health care commissioners.

The model predicts more accurately individual local cases in a given area, which a national model cannot because of the low size of population. By aggregating the local units of GP practices within an area and expressing the result as the relative probability of predicting number of cases is very practical for local commissioning as it enables better planning.

Epidemiological prevalence models based with local datasets and national data sources such as NHS Comparators, data from Public Health Observatories and a number of national reports could be invaluable for health care planners. Early experience suggests that they are useful for guiding

case-finding at practice level and improving and regulating the quality of primary health care. Comparisons with external data, in particular prevalence of disease detected by general practices, suggest that model predictions may be useful tools to help Health Commissioners.

Local practice-level analyses indicate a trend of undiagnosed disease prevalence together with the unreliability of QOF datasets suggest a fundamental problem of local health intelligence and subsequently a flaw in health commissioning and planning. A more effective method of achieving more accurate prediction for co-morbidity in the SMI population and undiagnosed medical conditions at local levels is for a more collaborative approach to validate and compare modelling methods using a framework that is more sensitive to local information. National leadership is needed to further develop and implement disease models. It is likely that prevalence models will prove to be most useful for identifying undiagnosed diseases with a slow and insidious onset, such as CHD, and COPD among the mentally ill. Such early detection will contribute to addressing the health inequalities.

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Table of abbreviations

ADQ/STAR	Average Daily Quantity of statins per standardised population
AF	Atrial Fibrillation
AIC	Akaike Information Criterion
APHO	Association of Public Health Observatories
AUROC/AUC	Area Under Receiver Operating Characteristics curve
BMI	Body Mass Index
BRFSS	Behavioural Risk Factor Surveillance Survey
BRHS	British Regional Heart Study
BTS	British Thoracic Society
CCC	Concordance Correlation Coefficient
CfH	Connecting for Health
CHD	Coronary Heart Disease
CI	confidence interval
CKD	Chronic kidney Disease
COPD	chronic Obstructive pulmonary disease
CQC	Care Quality Commission
CVD	Cardiovascular Disease
DH	Department of Health
DsPH	Directors of Public Health
ECG	Electrocardiogram, Electrocardiographic
FCE	Finished Consultant Episode
FEV1	Forced expiratory Volume in 1 second
FPG	Fasting Plasma Glucose
FVC	Forced Vital Capacity
GHQ	General Health Questionnaire
GP	General Practitioner
GPES	GP Extraction Service
GPRD	General Practice Research Database
HDL	High Density Lipoprotein
HES	Hospital Episode Statistics
HRG	Healthcare Resource Group
HSfE	Health Survey for England
HUM	High impact User Model
IC	Information Centre for health and social care
ICD	International Classification of Disease
ICER	Incremental Cost Effectiveness Ratio
IGT	Impaired Glucose Tolerance
IHME	Institute for Health Metrics and Evaluation
IMD	Index of Multiple Deprivation
IRR	Incidence Rate Ratio
JBS	Joint British Societies
JSNA	Joint Strategic Needs Assessment
LA	Local Authority
LR	Likelihood Ratio

LSOA	Lower Super Output Area
MI	Mental illness
MI	Myocardial Infarction
MLM	Multiple logistic regression modelling
MONICA	Multinational MONItoring of trends and determinants in disease CARdiovascular
MSOA	Medium Super Output Area
NatCen	National Centre for Social Research
NHANES	US National Health and Nutrition Examination Survey
NHS	National Health Service
NICE	National Institute for Clinical Excellence
NS-SeC	National Statistics Socioeconomic Class
NSTS	National Strategic Tracing Service
OLS	Ordinary Least Squares
ONS	Office for National Statistics
OR	Odds Ratio
PAR	Population Attributable Risk
PARR	Patients At Risk of Readmission
PCPH	Department of Primary Care & Public Health
PCSCs	Primary care Sensitive Conditions
PCT	Primary Care Trust
QOF	Quality and Outcomes Framework
RMSE	Root Mean Squared Error
ROC	Receiver Operating Characteristics
SBP/DBP	Systolic/Diastolic Blood Pressure
SE	Synthetic Estimate(s), Estimation
SVM	Support Vector Machine
UK	United Kingdom
UNAIDS	Joint United Nations Programme on HIV/AIDS
VIF	Variance Inflation Factor
WeLReN	West London Primary Care Research Network
WHO	World Health Organization

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Declarations

The work presented in this thesis is my own and where any material could be construed as the work of others, it is fully cited and referenced and/or full acknowledgement is given.

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1. Introduction

1.1 Background

The health needs of a population are derived from knowledge gained from the prevalence of diseases, i.e. the numbers of people suffering from different types of illnesses. However, looking only at the numbers of patients currently being treated for a disease does not show the true prevalence and its impact on the population's health. At any given time there are many people who have a disease but are unaware because they have not yet been diagnosed.

A robust and well-designed disease prevalence model can help health commissioners to assess the true needs of their community, calculate the level of services needed and invest in the appropriate level of resources for prevention, early detection, treatment and care. Prevalence models provide estimates of underlying prevalence derived from population statistics and scientific research on the risk factors for specific disease.

Whilst there has been progress in determining population (large scale) prevalence trends, there has been a relative shortage of research on local area (small area estimation (SAE) prevalence estimation modeling. An intrinsic problem is the lack of population-based data on key metrics, such as risk factor and disease prevalence at primary care and local authority (LA) level which has resulted in a total reliance on national datasets such as the Health Survey for England (HSfE).

This issue is highlighted in *Securing Good Health for the Whole Population*, Wanless (2008), which noted that...” the information collected nationally is often poor and there is no regular mechanism by which a PCT or LA can gather reliable information on its own population”. It proposes that in order to “improve understanding of prevalence of disease and to enable proactive management of personal risk factors, much greater use needs to be made of primary care data systems”. Assessing the potential population benefit of a health intervention requires consideration of many elements including disease prevalence and population characteristics, effectiveness and cost (Department of Health 2007).

The challenge is already being taken up by “Informing Healthier Choices”, the Department of Health’s (DH) public health information and intelligence strategy, (Department of Health 2007), which states that prevalence models will need to be generated for the common health problems which commissioners need to address. These will allow the current situation in an area or population group to be evaluated against an expected level of need.

Local area modelling using statistical models to link national surveys outcome variables, such as disease indicators, to local area predictors, regional demographic and socioeconomic variables, are needed so that prevalence rates for small areas can be predicted. According to the Department of Health (2007) and Druss *et al* (2001) social indicator variables such as age, race/ethnicity, gender, education, income, family structure and employment status are commonly used to define high-risk sub-populations for targeting

health promotion and disease prevention. Relating health status, behaviour and disease

prevalence statistics for small areas like counties to these demographic and socioeconomic predictors provides a direct calibration of the indicators to the outcomes of interest. SAE methods are applied to cases where the number of area-specific sample observations is not large enough to produce reliable direct estimates (Druss 2001).

One area which has not been addressed to-date is the modelling of the prevalence estimate for concurrent co-morbidity and in particular for those linked with mental illness. The co-existence of more than one chronic condition (co-morbidity) is a generally recognised feature of older people. It is estimated that for those over 65 this could range between 60-90%. What is not well known is the extent of multi-morbidity among the mentally ill and in particular those with chronic problems. According to a morbidity survey by Osborne et al (2010), more than 68 percent of adults with a mental disorder had at least one medical condition. Co-morbidity is associated with elevated symptom burden, functional impairment, decreased length and quality of life and increased costs. According to Phelan *et al* (2001) the pathway causing co-morbidity is complex and bi-directional. Medical disorders may lead to mental disorders and mental conditions may place a person at risk for certain medical disorders. However, mental and medical disorders may share common risk factors (Phelan et al 2001; Harris & Barraclough 1998).

This study covers the research needed to address two main issues at the heart which face the commissioning process. The first is the need for an approach that enables a more sensitive prevalence estimation of chronic disease within small areas. The second is to develop a framework to estimate the presence of these illnesses within the serious mentally ill (SMI). The two problems are intrinsically linked. It is anticipated that this approach will enable local primary care commissioners to improve their local health intelligence, through better data management, which will enable more accurate local estimations.

The study focuses on two medical conditions, namely chronic obstructive disorders (COPD) and coronary heart disease (CHD) to make this case.

1.2 Aims and Objectives

Aims

There have been many studies that have researched the various epidemiological aspects of common conditions such as diabetes, HIV, CVD and others. To date no prevalence models have been developed for medical morbidities that co-occur with mental illnesses in small areas such as locality-based services.

Population prevalence is a method used to estimate and forecast the number of people in the population with a particular condition and monitor how that might change over time. This approach to population estimates allow us to:

- ◆ Describe the pattern of disease in a population,

- ◆ Estimate the number and pattern of undiagnosed cases,
- ◆ Plan and deliver services in a rational way,
- ◆ Monitor performance.

The effectiveness of a prevalence model lies within its validity to provide robustness, reliability over time and fitness of purpose. The initial proposal contained the two broad aims of developing methods for prevalence estimation, of both disease and risk factors and their concurrent association with chronic mental illnesses. The research aim therefore proposed to gather and assess the fitness of general practice (GP) data, for the purpose of the prevalence estimation and to develop new methodologies, to adjust primary care data for sources of bias and to factor in estimates from mental health data to support healthcare commissioning.

Research Questions

- ◆ How should the prevalence of medical co-morbidity of chronic diseases in SMI be estimated?
 - ◆ How valid are the prevalence estimate models?
- ◆ How do different chronic disease prevalence estimation methods compare in terms of their validity?
- ◆ What is the best available methodology, given the requirements for prevalence models for co-morbidity?

Objectives

The objectives of the study were to:

- ◆ Develop a methodology for local prevalence estimation and modelling of chronic disorders for people with SMI, initially by extending logistic regression modelling to examine predictors of chronic obstructive pulmonary disease (COPD), coronary heart disease (CHD), stroke and hypertension using the HSfE datasets,
- ◆ Developing an adjustment ratio to calculate the prevalence of SMI in the estimated prevalence of the chronic diseases i.e. SMI as a subset of main data sets,
- ◆ Determine new prevalence estimates and future projections at PCT and GP level and support their use by PCTs (now Clinical Commissioning Groups (CCG)), practices and other agencies,
- ◆ Explore the use of risk factor prevalence to model future disease prevalence in relation to those people with serious mental illness,
- ◆ Undertake a validation of one or more disease prevalence models, through testing case-finding strategies at practice level, involving data from a West London primary care services,
- ◆ Explore the links between registered and estimated disease prevalence and primary and secondary healthcare utilisation, with the aim of developing specific utilisation ratios which can be used to project future healthcare capacity requirements.

1.3 Outputs / benefits

In the first instance, a methodology paper will be published on the physical disease prevalence model for the mentally ill. Later publications will include validation studies and case-finding strategy results.

Subsequent publications will explore the links between disease prevalence, health determinants and healthcare factors, ideally through practice-level analyses. Current person-based ONS survey data is available via the UK Data Archive. It was not clear at the outset whether the same level of access by researchers to person-based data would be permitted for other sources e.g. HES, but this would be an ideal eventual data linkage. The data and its implications for health policy will be presented to local health care commissioners and the wider network.

1.4 Study framework

The study was conducted over a number of phases:

Phase one (Chapter 1)

A study of the literature was undertaken to review two areas namely:

- Serious Mental Illness, its definitions (clinical and operational) and how services are commissioned and managed within community,
- Explore the concept of “prevalence modelling” and its application in public health epidemiology.

Phase two (Chapter 2)

Mental health needs assessment

A mental health needs assessment was undertaken to provide local information required for the modelling exercise, both from a broader community and local mental health delivery perspective. The exercise also assessed the fitness for purpose of general practice (GP) data for the prevalence estimation.

Phase three – Chapter 3

Definition of the methodological framework for study.

It is anticipated that a more sensitive prevalence estimation that was sensitive to variations and develop a “method” for a public health-driven primary care mental health delivery service.

Phase four (Chapter 4/5)

Development and validation of the synthetic prevalence estimate model using two chronic (medical) disorders as exemplars.

Using national datasets, the prevalence estimates of two chronic diseases were validated and then applied to local settings. Local health intelligence and information were linked to broader health determinants as explored in the needs assessment.

A rough prevalence estimate of the level of serious mentally ill (SMI) health based on existing Quality Outcome Framework datasets (QOF) was used as the baseline.

Phase five – Technique for estimating co-morbidity

Development of an adjustment factor for the prevalence estimate models

The rationale was to use an extrapolation (from national prevalence estimates) to determine relative co-morbidity in the SMI groups. The approach used Bayesian methodology to extract local estimates from existing datasets for co-morbidity estimation.

Stage six (Chapter 6)

Discussion and recommendations

The study was reviewed with considerations to its merit, weaknesses, usefulness and application it has in public health practice.

Ethics

As the PhD involved secondary data analysis the study did not conform to research governance criteria requirements and therefore did not require an ethical opinion.

2. Review of the literature

2.1. Serious mental illness and medical co-morbidity

Serious Mental Illness (SMI), although widely considered a severe long term condition, has been marked over time by a difficulty to formerly define. Even in 2013 there is still no nationally recognised definition in the UK. Traditionally, definitions have comprised of three elements - a medical condition of the brain; with significant functional impairment; over a significant period of time. This definition became summarised by 'the three Ds', diagnosis, disability and duration. Goldman et al. formalised this definition in 1981, explicitly stating relevant diagnoses, the level of disability and required duration of illness. Later in the 1980s, McLean and Leibowitz (1989) continued the three Ds approach, adding emphasis of patients' continued and regular contact with health services.

The next advances in the UK came in the 1990's. In 1995, the Department of Health continued the above theme, adding two further areas to be considered when diagnosing SMI. They included the safety of the patient and/or others and a requirement for community as well as medical support (Department of Health 1995). In 1999, the UK's National Service Framework (NSF) for Mental Health reverted to a broader definition, requiring simply the diagnosis of a mental disorder with either recognised severity or significant health service use. Despite small modifications, the NSF for Mental Health definition of SMI is broadly that still used in the UK. Internationally, the situation mirrors the UK. In the United States for example, according to Drake *et al* (2007), where a diagnosis is required to access publically funded mental health service;

definitions encompassing the three Ds are used across federal health care providers.

The word 'serious' within the phrase SMI, has the power to suggest other mental disorders are 'non-serious'. The term is merely used to represent conditions commonly grouped together. The definitions of SMI above encompass a range of conditions and clinical diagnoses. They can, however, still be considered useful. Grouping conditions based on severity will be especially useful for service providers and commissioners. In other words, although heterogeneous in medical diagnoses, the group is homogeneous in terms of service use and need.

The definitions above stem from a service administration or commissioner perspective, one where the severity and burden on the health system are critical to the definition. The first stage in identifying this group will, however, require a clinical diagnosis. There is no single clinical definition of SMI; it is a suite of disorders. Khatana *et al* (2001) supported the views of Wang *et al* (2002) that frequently, operationalised definitions of SMI cover three domains of the Diagnostic and Statistical Manual of Mental Disorders classification (DSM); mood disorders, anxiety disorders and non-affective psychoses. The DSM is a set of clinical codes used to define known mental disorders and map to the International Classification of Diseases (ICD).

In the US, Schinnar (1990) suggested that a patient had a severe mental illness when he or she had the following: a diagnosis of any non-organic psychosis; a duration of treatment of two years or more; dysfunction, as

measured by the Global Assessment of Functioning (GAF) scale (American Psychiatric Association 1987; Khatana *et al.* 2011). Specifically, the two levels of dysfunction defined by cut-off points of the GAF are tested: moderate or severe dysfunction a GAF score of 70 or less, indicating mild symptoms or some difficulty in social, occupation or school functioning; or only severe dysfunction a GAF score of 50 or less, indicating severe symptoms or severe difficulty in social, occupational or school functioning. The broad definition; the 'two-dimensional definition' is based on the fulfilment of the latter two criteria only (American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders 2002).

Yet more simple definitions include only specific disease groups. Three conditions, schizophrenia, schizoaffective disorder and bipolar disorder, were considered by the National Institute for Health and Clinical Excellence (NICE) (National Institute for Health and Clinical Excellence 2009) for use as the Quality and Outcome Framework (QOF) indicators relating to SMI. Finally, recent work by the Mental Health Foundation (2007), included psychosis, bipolar disorder, schizophrenia, schizoaffective disorder and puerperal psychosis within SMI. These small numbers of conditions, especially schizophrenia, schizoaffective disorder and bipolar disorder do cover a large proportion of the total SMI cases. This group of clinical definitions is not used universally, with different variants of DSM IV domains. They are however the most common, covering the majority of conditions considered as an SMI.

Prevalence

Recent data in the UK from the Information Centre for Health and Social Care (2011), the QOF reported a national prevalence of 0.8 percent (438,000 patients). This covers patients registered with primary care who have a diagnosis of schizophrenia, bipolar disorder or other psychoses and who are considered to have serious morbidity. The prevalence varies between primary care trusts (PCTs), ranging from 0.5 to 1.5 percent as documented by the National Institute for Health and Clinical Excellence (2009).

Epidemiological data concentrating broadly on SMI are scarce. According to the Information Centre for Health and Social Care (2009), all psychotic disorders, not solely cases defined as 'serious', have estimated prevalence 5 per 1,000, with an incidence of 31.7 per 100,000 in England. Schizophrenia is the most common psychotic disorder, affecting approximately 400,000 in England. Point prevalence estimates vary from 1.1 to 2.4 percent (National Institute for Health and Clinical Excellence 2009) with an estimate of lifetime prevalence of 8.7 per 1000. Recent estimates, from systematic review, place incidence in England at approximately 15 per 100,000 person years (Halliwell *et al.* 2007). Finally, estimates of schizoaffective disorder remain scarce; indeed debates remain as to whether it is a discrete condition, or merely co-occurrence of schizophrenia and mood disorders. Lifetime prevalence, according to Halliwell *et al.* (2007), estimates range from 2 to 10 per 1,000. Raw data suggest the prevalence may match schizophrenia, however, not when accounting for its diagnostic uncertainty.

The National Institute for Health and Clinical Excellence (2009) reported that bipolar disorder affects an estimated 545,000 patients in the UK. International estimates report a lifetime prevalence of bipolar disorder of between 0.9 and 2.1 percent and a prevalence of bipolar disorder with manic episodes of approximately 1 percent (Halliwell *et al.* 2007). There is variation between populations with estimates of the prevalence without manic episodes especially varied, between 0.2 and 2.0 percent. Khatana *et al.* (2011) again supports the views of Pini *et al.* (2005) that an estimate of the incidence of bipolar disorder across three UK cities, in those aged 16 to 64 years, was 4.0 per 100,000, although this varied from 1.7 in Bristol to 6.2 in London. More generally, estimates place annual incidence at 7 per 100,000, with a lifetime prevalence of bipolar disorder with manic episodes of between 4 and 16 per 1,000 as documented by the National Institute of Excellence (2006). A recent Finnish study, using a nationally representative population-based screening study, placed lifetime prevalence slightly higher than previous estimates at 2 per 1000 (Piri *et al.* 2007).

Demographics patterns in SMI

Evidence of differences in SMI across population sub-groups is varied, but generally is believed to affect men and women equally. Lifetime prevalence of schizophrenia and bipolar disorder, according to The National Institute for Health and Clinical Excellence (2009) and Kirkbride *et al.* (2012), is equal between the sexes. A review on available literature by Abrams *et al.* (2008) showed schizoaffective disorder may have a greater impact in women, although as discussed above, the literature on this condition remains sparse.

Although the overall prevalence is equal between sexes, the patterns in incidence with age differ. Both schizophrenia and bipolar disorders have an earlier onset in men compared with women (The National Institute for Health and Clinical Excellence 2009; Information Centre for Health and Social Care 2009; Kirkbride 2012), both occur most frequently between late adolescence to early adulthood. Schizophrenia typically has first onsets between the ages of 20 and 30 years, with estimates of a mean age of 21 in men and 27 in women. Bipolar disorder presents between the ages of 17 to 29 years (Pini *et al.* 2005; Halliwell *et al.* 2007), with estimates of a mean of 30 years in men and 35 in women, typically later than schizophrenia. Schizoaffective disorder has a marginally wider range, covering the modal ages of the two conditions above. The incidence of all declines with age, however, schizophrenia can have a secondary peak in incidence in the mid to late-forties (Loranger 1984).

There are differences in many common mental illnesses across socio-economic position (SEP), with the most deprived suffering a greater prevalence. The relationship with incidence appears weaker. Social fragmentation may promote incidence in deprived communities; however, this remains under-studied, with methodological weaknesses. Most importantly, little work has used longitudinal data to establish whether low SEP is causal of or caused by SMI.

Knowledge about ethnic variations in mental illness and specifically SMI is more established. Notably, the population from black ethnic backgrounds, both of African and Caribbean origin, suffer a higher burden of SMI (Sharpley

et al. 2001). Remarkably, this is a relationship not found elsewhere in the world. According to the results of the AESOP study (Lloyd *et al.* 2005), the diagnosis of both schizophrenia and bipolar disorder is more common in the black population in the UK compared with other ethnic groups. Meta-regression suggests black African and Caribbean groups have a relative risk of 4.7 to 5.6 for all psychotic disorders, compared with baseline population group in England (Kirkbride 2012). Traditionally, the impact of SMI has been considered equal in Asian and white ethnic groups; however, evidence is emerging of an increased risk in certain south Asian populations. There is evidence of a raised risk of schizophrenia in women of Pakistani or Bangladeshi ethnic backgrounds, with some suggestion of an increased risk of bipolar disorder. Finally, urbanicity, once controlling for age, sex and ethnicity, stands as an independent risk factor for schizophrenia (Kirkbride 2012).

Recent trends in SMI

Evidence behind recent trends in SMI is highly variable in terms of quality and the outcomes studied. Overall, there appears to be no change in incidence over approximately the last half a century (Kirkbride *et al.* 2009; Kirkbride *et al.* 2012), a similar picture to that of wider mental health conditions. Incidence of schizophrenia has remained stable, although there is a suggestion of increased rates in London. These, however, stem from studies not accounting for the changes in the ethnic make-up of the population (Kessler *et al.* 2005). Some studies have shown a decline in psychotic disorders, but again there are limitations. When accounting for different diagnostic practices over time, this relationship is lost (Kirkbride *et al.* 2012). Finally, one area with significant

increase over time is drug-induced psychosis, with up to a 15 percent increase each year (Kirkbride *et al.* 2009; Kirkbride *et al.* 2012).

Economic burden of SMI

SMIs have a considerable burden on society, with substantial disability and economic impacts (Insel 2008). Indeed, the impact of SMIs far outstrips their prevalence. All diseases have indirect costs - costs not directly from the medical or social care, but from wider societal impacts. Mental illness, most notably SMI are remarkable for their burden of indirect costs (Das Gupta 2002). There are few estimates on the cost of SMI specific to the UK. Bipolar disorders were estimated to cost the UK £2 billion in 2000 (The Sainsbury Centre of Mental Health 2003). Recent experimental estimates of the cost of all mental illness in the UK placed the burden at approximately £77 billion in 2003. Only 16 percent of this was from health and social care; with £41.8 billion in human costs, for example from losses to quality of life; and £23.1 billion from losses in economic output. These figures do not contain social security payments which were not considered a cost, merely a transfer of spending power from the state to individuals (Kessler *et al.* 2008). This is, however, an opportunity cost, with spending diverted from other causes. In 2003, these social security payments amassed to an estimated £9.5 billion. Unemployment and sickness absences stand as a particular cost for SMIs, with up to 46 percent unemployment in patients with bipolar disorder (The Sainsbury Centre of Mental Health 2003).

Unemployment in patients with SMI particularly impacts patients i.e. more than the state: analysis from the United States estimated a \$16,300 (£12,600 in

2012) per year reduction in earning in patients with SMI (Centorrino *et al.* 2009).

Patients with SMI induce increased health care spending. In a Swedish cohort, for patients with bipolar disorder compared with match general population controls; prescription expenditure was 6 times greater, emergency department 5 times, inpatient cost 5 times, and outpatient cost three times more. Just comparing subsets with other morbidities, for example patients with bipolar disorder and CVD compared with CVD alone, differences dropped but were still large. In-patient costs were four times greater, out-patient two times greater and emergency department costs three times greater. This relationship held across a range of diagnosed co-morbid conditions, but was especially great for diabetes and metabolic syndrome. Even restricting further to specific aspects of prescribing within specific conditions, spending was still greater in patients with bipolar disorder. Spending on controlling glycaemia in patients with diabetes, for example, was 50 percent higher per patient with bipolar disorder (Kessler *et al.* 2001). Generally, patients with SMI have high health service use. Estimates place patients with bipolar disorder more likely to use health services than all conditions except for those with panic and psychotic disorders (Pini *et al.* 2005).

SMI and co-occurrence of physical medical diseases

Patients diagnosed with an SMI suffer generally poorer health compared with the general population. This is borne out most clearly by their life expectancy. Estimates place the life expectancy of patients with SMI at between 15 and 30 years lower than the general population (Druss 2007; Jones *et al.*2004), although possibly a ten year difference in those with schizophrenia compared with those without (Drapalski *et al.* 2008). There is further worrying evidence in recent years from the United States that this gap may be growing. In general, poor health outcomes stem from a combination of poor health behaviours, direct impacts of the SMI, impacts of medication and fragmented care (Drapalski *et al.* 2008). Patients with SMI have poor help-seeking behaviour, which particularly impacts on poor outcomes. It is, however, not the SMI directly that has greatest impact on mortality: it is secondary conditions that add significantly to morbidity and mortality (Druss 2007). These co-morbid conditions (presenting in conjunction with the index disease), contribute significantly to poor health outcomes. From a large sample of Medicaid recipients in the US, estimates suggest over 70 percent of patients with an SMI had a second diagnosed chronic condition, with 50 percent having two or more (Carney *et al.* 2006). When examining co-morbidity with SMI, authors conclude that patients are at an increased risk of diseases affecting every organ of the body (Felker *et al.* 1996). Notably, although incidence rates of co-morbid conditions are increased, there is evidence that mortality rates are increased to a greater degree (Kilbourne *et al.* 2011). This is an important finding, indicating that once diagnosed with co-morbid conditions, care outcomes are significantly worse (McIntyre *et al.* 2006). There finally is a

greater amount of undiagnosed diseases, so patients with SMI are less likely to be aware that they have a co-morbid condition even if diagnosed (Radke *et al.* 2010).

Co-morbid conditions exacerbate the impact of SMI. For example, in a sample of patients suffering from manic episodes, the presence of a physical co-morbidity increased inability to work (a two-fold increase comparing one co-morbidity with none) (Kilbourne *et al.* 2005). There is further evidence of an association between greater co-morbidity with lower income, greater benefits claims and medical consultation. There is even evidence of a dose response as the number of co-morbidities increase (Kilbourne *et al.* 2005).

Although the co-morbid conditions affect the entire body, certain co-morbid conditions are particularly common in patients with SMI. Older SMI patients, for example, suffer an especially greater burden and circulatory and pulmonary co-morbidity (Hennekens *et al.* 2005). CVD is the most frequent cause of death in patients with bipolar disorder, followed by cancer and respiratory disease. Below the evidence behind a number of co-morbid conditions is reviewed in greater detail.

Cardiovascular Diseases (CVD)

The cardiovascular diseases (CVD) are the leading cause of death in both patients with schizophrenia and bi-polar disorder, with coronary heart disease (CHD) being the single biggest cause (Osby *et al.* 2001; Miller *et al.* 2006; Goff *et al.* 2005). Estimates place patients with an SMI at between a 2 to 3-fold increased risk of a CVD event compared with the general population (

Drapalski *et al.* 2008; Osby *et al.* 2001; Goldstein *et al.* 2009; Osborn *et al.* 2007), with a similar 2 to 3-fold increased risk of death (Druss 2007). One review of data for patients with schizophrenia concluded that approximately two thirds of this population are expected to die from CVD, compared with approximately half of the general population (Osby *et al.* 2001). For patient sub-groups, women may have a greater increase in CVD risk than men. There is also an earlier onset of CVD. In one US survey, patients with CHD were on average 13 years younger if they had a SMI co-morbidity (Osborn *et al.* 2007).

The best English data comes from over 40,000 patients with SMI in the general practice research database (GPRD) - a nationally representative collection of primary care records. Under the age of 50 years, patients with SMI had a 3-fold increased risk of CHD mortality and 2.5 times increased risk of stroke; over 50 years these were both 2-fold increases (Johannessen *et al.* 2006). The increased risk of a first CVD event, did not match that for mortality, with in fact no increased risk for those aged 50 years and over.

The main cause behind this substantial co-morbidity and cause of mortality is clear. Patients with SMI have substantially poorer CVD risk factor profiles than the general population. A large review of published data from the United States clearly indicates the problem. Patients with schizophrenia have a higher smoking prevalence; poorer control of blood lipids; a marginally higher prevalence of hypertension (19 vs. 15 percent) and considerably more obesity (Osby *et al.* 2001). The increased risk of hypertension is less clear. In one Swedish sample incidence was significantly higher in patients with bipolar

disorder, but not for those with schizophrenia. Other data suggests only a slight increase in patients with SMI.

Similar data from the UK shows patients with schizophrenia have the entire suite of CVD risk factors, much worse than the general population. This includes more smoking, obesity, physical inactivity, poor lipid profiles and poor diet (McCreadie 2003). Osborn et al. reported recent data on the cardiovascular health in SMI patients in UK general practice (Osborn *et al.* 2006). Compared with the general population, SMI patients were twice as likely to have a raised global CVD risk scores, with a higher median risk score. The difference in global risk was especially apparent for younger ages. There was a higher smoking prevalence, lower HDL cholesterol, higher total cholesterol, a small increase in raised BP and a higher diabetes prevalence (diabetes being an independent risk factor for CVD) (McCreadie 2003) (see page 23). Older patients have smaller differences in CVD risk factors, probably due to a healthy survivor effect (Johannessen *et al.* 2006).

Two particular risk factors for CVD (as well as risk factors for other poor health outcomes) stand out in groups of patients with SMI. Estimates of smoking prevalence for those with SMI are between 60 and 90 percent, approximately 3 times those in the general population (Drapalski *et al.* 2008; Osby *et al.* 2001). There is also evidence of an even higher difference in the prevalence of very heavy smoking (Drapalski *et al.* 2008). Increased smoking rates are likely for many reasons, including the alleviation of symptoms and social acceptance. Adding to these problems, there is evidence from routine care,

that smoking cessation efforts can be less effective than in the general population. This critically does not have to be the case. Trials show smoking cessation can be effective in patients with SMI, especially if tailored for the patient and drug therapy is included (Osby et al 2001).

The second risk factor to particularly impact patients with SMI is obesity. An American sample suggested an approximately two-fold increase in prevalence, with an especially large increase in highly obese women (Dickerson *et al.* 2006). Women with SMI suffer a higher burden of obesity compared with men (Drapalski *et al.* 2008). Of greater concern to cardiovascular health is the increase in abdominal obesity compared with obesity per se (Drapalski *et al.* 2008) (see page 25). In addition to risk factors, deficiencies in care outlined above have an impact on mortality rates; including poor prevention and acute care. UK data show the increased risk of CVD mortality is significantly greater than the first event; secondary prevention and acute care for CVD remains suboptimal in patients with SMI (Johannessen *et al.* 2006).

Respiratory Illness and other conditions

Respiratory illness is another significant co-morbidity for patients with SMI. One US study, showed COPD was the single greatest co-morbidity (Carney *et al.* 2006). Similarly, in another study, COPD and asthma in SMI group were the second and third most prevalent (point prevalence) conditions after hypertension (Sokal *et al.* 2004). There is evidence that respiratory diseases in this group shows a greater increase in standardised mortality than the

general population, when compared with circulatory disease and diabetes. There is increased risk of many respiratory illnesses, including COPD, bronchitis and asthma (Drapalski *et al.* 2008; Himelhoch *et al.* 2004). Data suggest a three to four-fold increase in chronic bronchitis, a five-fold increase in asthma risk and approximately two-fold increase in COPD (Drapalski *et al.* 2008; Sokal *et al.* 2004). Although largely due to smoking prevalence, this may not be the only factor. Second hand smoke, as well as other as yet unknown factors may be important (Drapalski *et al.* 2008; Sokal *et al.* 2004).

As discussed above, patients with SMI are believed to be at increased risk of co-morbidities affecting the entire body. There is evidence of higher HIV prevalence in some SMI populations (Drapalski *et al.* 2008). For example in one US sample, patients with schizophrenia had a 1.5 times greater adjusted prevalence and a four-fold increase in patients with affective disorders (Bank *et al.* 2002). There is some evidence of women with SMI having an increased risk of obstetric complications (Thornton *et al.* 2010). Finally, the impact of how patients with SMI interact with health services is highlighted in paper which describes late presentation with appendicitis and how this results in poorer outcomes and more complications (Cooke *et al.* 2007).

Diabetes

Diabetes (and impaired fasting glycaemia (IFG)/ impaired glucose tolerance (IGT) has an established and significantly increased impact for patients with SMI. There is a clear increased incidence and prevalence of diabetes in patients with SMI. Older estimates from the US indicate a 1.5 to two fold higher prevalence of diabetes, although this may now be higher (Osby *et al.*

2001; Bushe 2004). More recent estimates quote this figure as a three to four fold increase compared with the general population (Drapalski *et al.* 2008; Bushe 2004; Goldstein *et al.* 2009). One significant international study, including 220,000 respondents from 52 countries showed diabetes prevalence in patients with SMI varied considerably between countries, ranging from nearly zero to eleven percent (Nuevo *et al.* 2011). Finally, there is some evidence that if diabetic, SMI patients are more likely to have complications (Felker *et al.* 1996; Nuevo *et al.* 2011). This indicates poor glycaemic control, which is likely in part to be due to poorer care and delayed diagnosis, but this together with anti-psychotic medication also potentially important (Nasrallah *et al.* 2006; Holt *et al.* 2010).

Despite being evident, there is considerable debate over the cause of this increased risk. Reasons include, poor health behaviours and lifestyle which are evident in patients with SMI (Bushe 2004; Osby *et al.* 2001); weight gain and insulin resistance caused by anti-psychotic medication (Bushe 2004; Osby *et al.* 2001); or an unknown physio-pathological cause, potentially through a direct genetic link or inflammatory mechanisms (Nuevo *et al.* 2011; Thakore 2005). Current studies can, at times, offer contradictory views. One longitudinal study in Wales found, in a sample of patients presenting with psychosis, that before contact with the health service, diabetes prevalence was equal to the general population. After the first encounter, however, incidence doubled (Le Noury *et al.* 2008). This, the authors concluded, suggested treatment for SMI was responsible for diabetes. A second study of note showed that as psychotic symptoms increased, there was linear increase

in the prevalence of diabetes. This was independent of SMI diagnosis, medication and known metabolic risk factors. This suggests a more direct link between symptoms and diabetes, although how this might occur is entirely unknown (Nuevo *et al.* 2011). Finally, there is evidence of an increased prevalence of aspects of 'the metabolic syndrome' (see below) in patients with SMI, of more severe symptoms and of more limited impacts of treatment (McIntyre *et al.* 2010). In reality, it seems likely that both lifestyle and the medications are implicit in the diabetes risk, with potential for a third, less understood pathway.

Metabolic syndrome

The metabolic syndrome is a cluster of conditions relating to metabolic abnormalities, and stemming from a small number of common causes (Grundy *et al.* 2004). These are risk factors for a number of diseases, most notably CVD and diabetes. Numerous definitions exist, but generally the cluster includes abdominal obesity, raised blood pressure, dyslipidemia, hyperglycaemia and micro-albuminuria. Although one can consider them as merely risk factors for other diseases, these tend to be grouped due to their common causes. Between 50 and 60 percent of SMI patients are considered to have the metabolic syndrome internationally, the prevalence of metabolic syndrome in bipolar disorder ranges from approximately 20 to 55 percent. The two share both lifestyle and patho-physiological risk factors. The depressive side of bipolar disorder, for example, increases eating and therefore weight gain. There are also metabolic traits shared by the conditions, for example gluco-corticoid resistance and immune system abnormalities. Recent genetic

analyses show that metabolic syndrome, and indeed CVD itself share basic genetic pathways (de Almeida *et al.* 2012).

As mentioned above, anti-psychotic drugs (neuroleptics) may be implicit in the metabolic syndrome. These drugs cause weight gain, although there is variation between agents. Two medications, Clozapine and Clanzapine, may be especially associated with metabolic syndrome, increasing total weight gain, abdominal adiposity, insulin resistance and affecting lipid metabolism (Newcomer 2007). In a randomised trial comparing anti-psychotic medication with a placebo, there was a 1.2 to 5 fold increase in impaired glucose tolerance and evidence of up to 4kg in weight in 10 weeks when taking the medication (Druss 2007). Increased risk of metabolic syndrome, therefore of CVD and diabetes, is complex in patients with SMI and multi-factorial in its causes.

Mental co-morbidity

Other mental co-morbidity, despite potentially not having the same impact on mortality as some conditions, does represent a significant burden on morbidity in patients with an SMI. The majority of patients with bipolar disorder have at least one other axis-1 disorder, with estimates of lifetime prevalence of upwards of 65 percent and a point prevalence of one third (Pini *et al.* 2005; McElroy *et al.* 2001). The greatest single mental co-morbidity is anxiety disorder, with some lifetime estimates of prevalence reaching 65 percent (Pini *et al.* 2005; McElroy *et al.* 2001). The risk is not restricted to anxiety disorder, with for example a ten-fold increased risk of panic disorder. The prevalence of

concurrent mental co-morbidities is exemplified by data from patients in one English mental health unit from the 1990s (Pini *et al.* 2005). Twenty percent of patients with an SMI reported a second mental health diagnosis from within the previous six months (Virgo *et al.* Journal of Mental Health 2011).

Substance misuse is highly prevalent in patients with SMI. Although now 20 years old and from the US, the largest general population survey of mental health co-morbidity exemplifies this. The rate of lifetime alcohol or drug use disorder in the general population was approximately 17 percent. This was compared with 47 percent for people with schizophrenia, 56 percent for people with bipolar disorder and around 30 percent for people with other mood disorders or an anxiety disorder. Overall, across SMI this was summarised as a 50 percent lifetime prevalence (Regier *et al.* 1990), with a 25 to 35 percent point prevalence. Comparing the odds of risk with the general population, there is 10 to 20 times higher odds of alcohol abuse and up to 30 to 40 times increased odds of illicit substance abuse in both patients with bi-polar disorder and schizophrenia (Felker *et al.* 1996; Nuevo *et al.* 2008).

Despite this bleak prognosis, there is evidence of successful interventions for what is called the “dual diagnosis”, SMI with substance abuse. Trials using peer-support, longer residential interventions and even intensive outpatient intervention can all reduce substance abuse. No data currently assess the cost incurred by health systems from these co-occurring mental conditions. It is, however clear, that patients with an SMI require wider care for their mental health than the index condition alone.

2.2 Commissioning for patients with SMI

There is evidence of poorer acute care, including follow-up after disease events and hospital in-patient care, in-patients with SMI (England *et al.* 2005; NHS Diabetes Commissioning Mental Health and Diabetes Services 2001). Again in a national US sample, patients with SMI were less likely to receive hospital care for CVD and diabetic complications. Similarly, despite evidence of increased need, patients with SMI are significantly less likely to receive appropriate care following a CVD event, patients with SMI were up to three times less likely to receive necessary procedures (England *et al.* 2005).

Following heart failure, there is evidence that SMI patients are less likely to receive follow-up care and more likely to face re-admission. One final piece of evidence concerning the quality of care, suggests that patients with schizophrenia have a greater rate of adverse events in hospital, compared with the general population (Mechanic 1995).

The reasons behind limitations in care and the quality of care are complex and remain understudied. Generally, it is the evidence based and more cost effective practices that are underused, whereas some areas, such as use of emergency departments, can be overused. One of the largest weaknesses in care provision is the separation of mental health services from other aspects of care. This separation can be geographic, through funding, through organisation and expertise, or the culture of providers. Although possibly the most simple difference, the geographic separation is likely to be vital. There is direct evidence that co-location of wider medical and preventative services

with mental health services can increase the use of a suite of services and result in better patient outcomes, including blood pressure control.

One thing that is clear is that integrated care is of paramount importance for patients with SMI. As such, primary care has been proposed as the best setting to improve patients' health. Primary care practitioners, have the greatest experience of holistic and integrated care. For this to happen, however, a number of barriers must be overcome. There firstly needs to be collaboration between primary care and psychiatric services. Primary care doctors can be unfamiliar with psychiatrists, which can limit access to care resulting in ? generally face poor co-ordination and collaboration (Cooke *et al.* 2007). Secondly, primary care clinicians must accept psychiatric conditions do not inhibit routine medical care. Clinicians can view patients with SMI as disruptive to their practice, cite time constraints as a barrier or simply be uncomfortable with the situation. All of these stop general practitioners managing patients with SMI. Qualitative work does, however, suggest general practitioners are willing to take a central role, not only in the diagnosis of, but also the routine care of patients with SMI (Johannessen *et al.* 2006).

The appropriate site of health care for patients with SMI is still debated. Psychiatrists often acknowledge they should provide physical care. This, however, frequently does not happen. Psychiatrists can delegate care and need not keep up to date with evidence-based practice outside their specialty. Some psychiatrists, although a minority, do not consider the physical health of their patients, which contributes to the morbidity.

These barriers are frequently caused by the SMI itself, and include forgetting required care, a lack of knowledge of how to access care and difficulties in communication.

There is little specific guidance on commissioning services for patients with SMI, and less evidence on the effectiveness of different strategies. One certainty is that both continuity of care and integrated care play particular importance for patients with SMI (England *et al.* 2005; NHS Diabetes Commissioning Mental Health and Diabetes Services 2001). They must be able to 'seamlessly' navigate between aspects of care, especially mental health care – but also physical care. Integration is particularly important because patients with SMI are amongst the most socially excluded (England *et al.* 2005); therefore have fewer opportunities for care and the condition can make it hard to negotiate care pathways. They also require care over prolonged periods, a situation in which integration is vital (England *et al.* 2005; Mechanic 1995).

Integration is especially difficult for patients with SMI. Jurisdiction for care can span many bodies, for example mental health trusts, secondary trusts, primary care and social services. Integration requires both the co-ordination and co-location of services. Shared training and learning amongst practitioners may also be important (England *et al.* 2005). Finally, a model in which patients are assigned a single, multi-disciplinary team to manage physical health need may be effective. These teams can co-ordinate, but also support those in direct contact with patients, reducing fragmentation (Mechanic 1995).

A number of considerations for commissioning services for patients with SMI emerge from the evidence above, with a particular focus on co-morbidities. Firstly, and important to consider for all commissioning decisions, given the differential increase in mortality compared with disease incidence for patients with SMI, there is a very real need to improve the routine care in those with diagnosed disease (NHS Diabetes Commissioning Mental Health and Diabetes Services 2001). Commissioning services that allow for regular physical assessment may be effective. Following a formal guide, such as one for diabetes produced by NHS diabetes may also enable the correct processes for evidence based commissioning (NHS Diabetes Commissioning Mental Health and Diabetes Services 2001).

The setting of physical care may be important. Firstly, there is evidence that distance to care can be especially inhibitive for patients with SMI. Co-location of physical care with mental health services may be effective. At the very least there has to be a clear statement of where physical healthcare is received and who is responsible. This will prevent patients with SMI from falling in between providers, thus missing out on care. Mental health nurses are considered, due to their relationship and contact with patients, to be in an excellent position to help improve the physical health of the SMI. Frequently, however, they are lacking competencies and training. Using evidence based health improvement profiles (HIPs) and training may promote this care pathway to reach its full potential (Robson *et al.* 2007).

Patients with SMI suffer poorer than average outcomes from national screening programmes, largely due to decreased uptake. An alternative approach to screening or at least extra focus on patients with SMI may be warranted. Bespoke interventions for patients with SMI to reduce CVD risk can be effective (Smith *et al.* 2007). Currently CVD risk in those without diagnosed vascular disease is the focus of the NHS Health Check programme. Incorporating patients with SMI into the programme, but like one London PCT, separate performance management to promote uptake is a possible solution (NHS Diabetes Commissioning Mental Health and Diabetes Services 2001). Bespoke interventions for 'dual diagnosis' (SMI and substance abuse) can also be effective. Finally, dental care is poorer in patients with SMI than the general population. Although there are few evidence-based interventions, focus must be placed on access to care and good oral hygiene, with particular focus on provision for in-patients.

In conducting this review, several gaps in the literature on mental health and medical co-morbidity became evident. First, most of the existing literature on co-morbidity examines the impact of particular co-morbid conditions on an index medical or mental illness (e.g., diabetes and depression). While there is value in these specific, clinically-focused approaches to understanding co-morbidity, patients with co-morbid conditions share many common features that make them valuable to examine as a distinct population of interest. They are, in many ways, analogous to racial and ethnic disparities groups who are monitored separately and often require tailored quality improvement programs. Second, nearly all of the current evidence for this population

focuses on clinical models rather than organisational or systems level approaches to implementing those models. Comparative effectiveness trials will be needed to compare organisational approaches to delivering and sustaining these evidence-based approaches to improving care for persons with co-morbid conditions. Finally, health reform will include a broad range of changes in insurance coverage and care delivery that could have a disproportionate impact on persons with co-morbid medical and mental conditions. Tracking the impact of this legislation on costs, burden and outcomes of care for this population could provide important information to inform future iterations of health legislation.

2.3 Prevalence modelling

Prevalence modelling is a technique used to estimate the number of people with a particular condition or risk factor in a population when direct evidence is not available (American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders 2002). Direct evidence may be lacking because surveys or data collection have not been undertaken, are technically impractical, or are unreliable.

Methods for generating synthetic or modelled estimates range in complexity from simple to highly sophisticated. Crude estimates of the number of cases can be generated by applying known prevalence rates to a different population, for example applying national rates measured in a large survey to a local population; or applying local rates for a recent year to a projected future population. However, many factors such as age, gender, deprivation

and ethnicity can influence the prevalence of a behaviour, risk factor or disease and more complex epidemiological modelling techniques are required in order to take such factors into account.

The need for prevalence Modelling

In many cases, routine data are not available to measure directly the frequency and distribution of diseases or behaviours in local populations (Diez-Roux 2000). Modelling is often the best alternative for quantifying prevalence in the absence of reliable direct measures. Typically, direct measures are not available at local level for lifestyle behaviours such as smoking or alcohol consumption, or for diseases that are generally managed in primary care, for example diabetes or hypertension.

Understanding the distribution of behaviours that affect health (either positively or negatively) is increasingly important in the allocation of public health resources and the delivery of interventions (Congdon 2006; Congdon *et al.* 2007). Prevalence modelling can be used to assess need and help identify those communities that will most benefit from public health initiatives. Modelled estimates of prevalence can also be helpful in explaining variations in care utilisation and outcomes (Congdon *et al.* 2007).

The quality and completeness of routine datasets, such as the Quality and Outcomes Framework (QOF) for primary care, are improving, and QOF is now a reasonable basis for prevalence estimates of many diseases. However, the measured prevalence is limited to diagnosed disease (Congdon 2008; Cooper

et al. 2002). Modelled estimates that include undiagnosed disease in the population can offer additional information that can inform case-finding initiatives and highlight areas where under-diagnosis could be an issue.

There is considerable interest in obtaining estimates of expected prevalence at various geographies and for different subgroups of the population, for example ethnic groups or age cohorts, to assist in understanding and tackling health inequalities (Congdon *et al.* 2007).

Methods

Many methods exist for creating synthetic estimates of prevalence, and in many cases methodologies are combined and adapted to make best use of the information and data available (Homer *et al.* 2006). There is often a balance to be struck between increasing the complexity of the model by incorporating more contributory factors and the availability of good quality data at local level to populate the model. These input requirements of a model are often restricted by what information is available (Gunnars-Schepers 1989). Complex models can also suffer from difficulty of interpretation, which negates the benefit of increased accuracy.

2.4 How does the model work?

All models are based on assumptions. Good models clearly state the assumptions that have been made and good interpretation of modelled estimates takes into account the limitations of the assumptions.

1) Regression models using demographic characteristics from large surveys

Multiple variables from large surveys can be used to model the risk factors for a behaviour or disease, using techniques such as multi-nomial logistic regression. However, it is important to limit the factors considered to those for which data are available in the population of interest. For example, cholesterol level or family history of disease may be important risk factors which were recorded in the source survey, but such information is not usually available at population level and therefore these are not appropriate variables to be included in a disease prevalence model.

National surveys are usually limited to people living in private households and omit populations such as the homeless, those living in institutional care, 'special populations' (armed forces and prisoners) who are the people particularly likely to decline to participate. For some disease areas, notably some types of mental illness, these omitted populations can be particularly important. Despite this limitation, national surveys are often the best source of prevalence information available, but where possible should be used in conjunction with other evidence about the likely extent to which they miss cases. Models can then be adjusted to take account of the resultant under-estimation of prevalence.

Although regression models most commonly use survey data, other data sources, for example information recorded in general practices, can also be used to create this type of prevalence model.

2) Capture-recapture methods

Capture-recapture methods are used to estimate the number of people with a disease or behaviour, for example the total number of injecting drug users, including those unknown to any services. A random sample of people is taken ('captured') from the whole population, and examined for the characteristic of interest. 'Sample 1' is the number of individuals found to have the characteristic. A second random sample of the whole population is then taken and 'Sample 2' is defined similarly as those found to have the characteristic. Some people will appear in both Sample 1 and Sample 2 and the proportion of Sample 2 that was also in Sample 1 is calculated. This proportion is assumed to be equivalent to the proportion of all the people with the characteristic in the whole population that were captured in Sample 1. Hence, by dividing Sample 1 by this proportion an estimate of the total number with the characteristic is obtained.

3) Combining multiple sources

Often there are several estimates of prevalence rates available from larger and smaller scale epidemiological studies, which need to be integrated. For example, regional prevalence rates from large national surveys can provide control totals for smaller geographies for which synthetic estimates are generated. Each source will have strengths and weaknesses: national surveys may have robust sampling and include a wide range of risk factors, but can lack local detail, whereas local studies may use more elaborate methods, for example capture-recapture techniques, but may focus on unrepresentative

areas. Combining prevalence estimates requires critical appraisal of the appropriateness of each source and development of mathematical methodology, to integrate the variance estimates from unrelated sources to produce an overall confidence interval for the synthetic estimate.

Meta-analysis techniques have been developed to combine multiple estimates of prevalence, each of which may have data quality issues, to produce one triangulated estimate with improved quality at small area level. Estimates from a wide range of sources can be combined, including prevalence estimates from surveys, data from primary care and modelled synthetic estimates.

Bayesian statistical methods can be employed to synthesise a diverse set of available data into a prevalence estimate. For example, Goubar et al (2008) combined an array of information, including routine surveillance data and anonymous surveys, to estimate HIV prevalence in various risk groups using Markov chain Monte Carlo simulation.

2.5 Validation, confidence intervals and robustness

Prevalence model validation is problematic for a number of reasons. There is no other major or definitive source of the national HSfE prevalence data to use for the models, apart from population-based prevalence research for specific diseases. The literature search before developing each model revealed what studies currently existed and this was also useful for initially validating the models. The accuracy of model outputs depends on the predictive power of the model and on the accuracy of the input data. Models should be subjected to validation checks to ascertain their robustness and general applicability.

Estimates of the accuracy of prevalence estimates based on simple models can be generated by combining the uncertainty in prevalence rates from the source study or trial with the stochastic variation expected given the size of the local population. This approach results in a range estimate for the prevalence, rather than confidence intervals. The range estimates are calculated using the same methods as those used to derive the control limits for funnel plots.

However, if there is uncertainty around both the population data and the input data, the calculation of confidence intervals can be complex. Bootstrapping methods are commonly used in such situations.

Four ways of validating models

a. Sensitivity testing

Sensitivity testing can be useful in assessing how the uncertainty in input data affects prevalence estimates. For some models, very small variations in the input data will have a large effect on the results. Other models may be relatively insensitive to variability in input data. For example, 10 different sources of practice level smoking prevalence data were input into the APHO chronic obstructive pulmonary disease (COPD) models. Estimated COPD prevalence in general practices ranges from 1% to 7%. In 92% of cases, changing the source of smoking prevalence data made an absolute difference of less than 0.3% in the COPD prevalence estimate. Estimates generated using different smoking prevalence source data were strongly correlated with

each other ($r^2 > 0.95$).

One-way sensitivity analysis such as this evaluates the impact of a change in one variable on the model results. Multi-way sensitivity analysis is more powerful and can be used to assess the impact of changing two or more variables simultaneously.

b. Internal validation

One method of assessing the performance of a model is to use it to predict the response for each subject in the source data (e.g. a large survey). These predictions are called fitted values. The differences between the fitted and the observed values are called residuals. Residual analysis can be used to check the adequacy of any assumptions used when creating the model. It can also be used to identify whether any additional factors should be included.

To check the accuracy of the model, the predicted 'classification' of each individual (i.e. whether or not they have the disease or behaviour that is being modelled) can be compared with their actual classification. This will result in a 'misclassification' (also known as a 'contingency' or 'confusion') (Table 1).

	Negative	Positive
Negative	A (True negative)	B (False negative)
Positive	C (False positive)	D (True positive)

Table: 1. Misclassification table of modelled results

c. External validation

- ◆ One method of assessing external validity is to map observed and expected prevalence and investigate associations with a low ratio of observed to expected cases, at both local authority, PCT and/or practice levels. The gold standard validation would be comparison of model predictions with a comprehensive population survey or case-finding efforts in a number of differing populations: deprived and advantaged, rural and urban, etc.

- ◆ Receiver-Operating Characteristic (ROC) Analysis

ROC analysis is a useful way of assessing the accuracy of a model by understanding the trade-off between the sensitivity (in this sense referring to the true positive rate; Table 2) and the specificity (the true negative rate). The method was developed to assess the accuracy of distinguishing signal from noise in radar systems and has since been applied in many other settings, including clinical diagnostic testing and the evaluation of regression models that classify cases into two categories, for example diseased and non-diseased. Sensitivity is plotted against 1-specificity (specificity subtracted from one) over a range of values and the area under the curve (AUC or AUROC) is used as a summary of the predictive or diagnostic accuracy. A 'perfect' model that accurately predicts every case has AUROC = 1. Typically, models have a convex ROC curve and an AUROC between 0.5 (equivalent to random chance) and 1. A model with AUROC < 0.5 is less accurate than random chance.

2.6 Projections and forecasting using models

Prevalence models can often be adapted to predict future prevalence. The sophistication of projected prevalence estimates depends on the modelling methodology adopted, and falls into three broad categories:

- a. Same risk, changing (e.g. increasing and/or ageing) population. Use the same model coefficients or risks of disease but incorporate population projections. For example, what will be the prevalence of coronary heart disease (CHD) in 2020 if we assume that the age-specific risks do not change but we take into account the aging population? This is sometimes called the 'prevalence ratio method'
- b. Same population, changing risk. Use the same demographic information but change the risk profile. For example, what will be the prevalence of CHD if smoking prevalence reduces?
- c. Modify the population and the risks to produce 'scenario models' e.g. what will be the CHD prevalence with an ageing population and reduction in smoking.

One of the characteristics of complex systems such as health is that no matter how tightly the present state of the system is specified the future state cannot be confidently predicted. Extra care should be taken in interpreting modelled estimates of projected prevalence as the assumptions inherent to the model may not hold in the future.

2.7 Using prevalence models

It is important to remember that prevalence figures generated by models are synthetic estimates of the expected prevalence of disease. They are not 'real' measures of prevalence. It must be remembered that 'all models are wrong but some are useful.' Discrepancies between modelled estimates and other sources of data (such as primary care disease registers) may be due to local variations not captured by the model and cannot be solely attributed to weaknesses in directly measured prevalence data. For local populations that differ significantly from a 'typical' population (e.g. a large black and minority ethnic (BME) population that has a very different smoking pattern to the national average) the assumptions of a model may not apply and discrepancies may occur. Local expert opinion (e.g. local GPs' knowledge of the pattern of disease) can be invaluable in interpreting and applying synthetic estimates of prevalence.

A typical use of prevalence estimates is to compare expected prevalence with recorded prevalence, for example from the QOF in England. Such an approach needs to be taken with care. Are the two populations comparable, or are you trying to compare adult prevalence with all-age prevalence? Is the definition of disease used in the modelled estimates the same as the clinical definition used for diagnosis in primary care? Does the model include an estimate of undiagnosed disease or not? An understanding of these issues and differences is vital in interpreting any comparisons made between synthetic estimates and measured prevalence.

Because modelled prevalence is an estimate of expected prevalence, generally with the assumption that the local area behaves in the same way as the population from which the source data were derived, it is not straight forward to use synthetic estimates to evaluate the impact of a local intervention. For example, low modelled prevalence of binge drinking in a local area that has invested heavily in action to decrease alcohol misuse is not proof that the investment has reduced binge drinking. It is only an indication that the area can expect a low prevalence, given its demographic characteristics. Local interventions or prioritisation of an issue may explain discrepancies between modelled and directly measured prevalence, but the discrepancy does not prove that an intervention or policy is effective. It is not advisable to use prevalence models for performance management or to evaluate the impact of a local programme.

It is also inappropriate to use modelled estimates to monitor changes over time. Changes in estimated prevalence could be due to updated local input data (e.g. demographics) or changes in the source data used to generate a new version of the model. There may also have been adjustments in the modelling methodology used if source data have been re-modelled.

2.8 Issues with Small Area Estimates (SAE)

A geographical area is regarded as "small" if the area sample is insufficient to yield direct estimates with adequate precision and reliability. In order to make estimates for small areas with adequate levels of precision, it is standard to use indirect estimates that utilise information from outside areas with similar

characteristics to the area of interest. Generally, a statistical model is used to obtain indirect estimates for geographical areas considered to be "small". The information from respondents who are outside the geographical area and other geographical characteristics are incorporated through the use of a statistical model. Small area estimates of the prevalence is important where risk factors vary widely, but are important for decision and policy makers, and their quality is a crucial concern. One example is in health promotion, when addressing area-specific health issues or lifestyle behaviours. In some deprived areas people might have more restricted access to screening programmes or preventive healthcare campaigns, or they may have a higher level of certain risk factors. Knowledge of the prevalence of risk factors in small areas is essential to make health promotion strategies more effective.

Small area estimation is conducted in two stages. In the first stage, regression analysis is performed modelling survey data (e.g. HSfE) against predictors of the condition under investigation. This analysis is conducted for the subset of areas covered by the survey. The output from this first stage is a set of parameter estimates. At the second stage each area of the population, the coefficients of the predictor variables obtained from the first stage model, are attached to the identical set of variables at the small area level to produce an estimate for the area as a whole.

Synthetic modelling for small areas uses a number of methods to generate estimates:

- a. Simple (non-modelled) methods using indirect standardisation,
- b. Models using individual level covariates only,

- c. Models combining individual and local area-level covariates,
- d. Models using area level covariates only,
- e. Other approaches for larger areas of geography,

Only two (a and c) of the methods are reviewed here as they are more applicable to the study in question.

a) Indirect standardisation

This involves applying national estimates derived from national surveys to area-level population counts to generate area estimates. An example of this approach – If we have to calculate the proportion of men smoking in a particular ward would involve (a) using national estimates of smoking patterns and (b) applying these to the local population, weighted by the proportion of persons in the sub-group in the small area.

This model has an intuitive appeal in its generally easy and inexpensive to use (Nacul *et al.* 2007). The major drawback is that it assumes that the national rates of each subgroup are applicable uniformly across all areas.

b) Using covariates from census

This is an extension of the above method. It uses the information regarding the relationship between individual health behaviour measures obtained from surveys against a set of predictor variables for the same individuals.

These models estimate the probability that a person with specific known characteristics (e.g. age, gender) currently smokes, is obese etc. The model-based probabilities are then converted into estimated proportions in each

subgroups defined by the covariates who fall into relevant health category. These proportions are then applied to the covariate counts available from the census to derive an overall estimate for the small area in much the same way for indirect standardisation. The major drawback concerns its data requirements. This form of synthetic estimation requires exact correspondence between the covariates used at national and local level.

The two methods above are all at the individual level. An alternative model is the multi-level models incorporating random effects (also known as mixed models). Their importance to small area estimation lies in the fact that a random effects specification assumes that significant systematic variation between small areas remains after the effects of covariates in the model have been accounted for. Such 'unexplained' variation is modelled through the addition of small area specific random coefficients to the fixed effects. Such multilevel models give rise to more complex ways of building a model for health behaviour measures; generating small area estimates from these models parameters and thus calculating the confidence interval for them. Using this technique, a model can be applied to survey data that simultaneously account for individual and area influences on health behaviours such as smoking.

Conceptually and methodologically, the model is more useful than simple models as it combines both individual and geographic level data. However, it also requires stringent data requirements (as above) and estimating standard errors are infinitely more complex.

2.9 Summary

So how should the “fitness for purpose” of prevalence models, including validity, be defined? In the case of disease risk scores, the technical criteria are widely agreed. The accuracy of a risk prediction score can be judged on two main components—calibration and discrimination. A well-calibrated score is one in which the predicted risk is similar to the observed risk. The more important component of accuracy is discrimination or the ability of a score to differentiate between people who will have an event from those who will not, over a defined period of time.

Similarities and differences are discussed in the sections dealing with model development. In the absence of more robust local data QOF data is useful to an extent. However, use of case-finding in practice populations is seriously considered as a good predictor. In general, it is expected that modelled prevalence estimates to exceed QOF registered prevalence for 90 per cent of practices, with previously described model limitations leading to under prediction in perhaps ten percent of practices.

3. MENTAL HEALTH NEEDS ASSESSMENT

3.1. Background

The health and social care costs of mental health in England are around 22.5 billion per year (New Economic Foundation 2011) and mental ill health accounts for more than 12% of the NHS budget. The number of people with mental health problems is likely to rise by 14.2% to 9.88 million by 2026 (The Royal College of Psychiatrists 2008; Murali *et al.* 2004).

NHS Brent has a priority for commissioning mental health services. Part of the commissioning process requires a comprehensive needs assessment of the community to make decisions about delivery of an optimum service. This needs assessment needed to be evidence-based via three strands:

1. geographical information mapping,
2. framework to improve equity of access and,
3. a process to improve mental health well-being and health promotion.

An effective needs assessment exercise has to be inclusive of normative, comparative and social needs. It must have the robustness to represent the various spectrums into the mix of understanding the mentally ill's needs (Marmot Review Team 2011; North East Public Health Observatory 2012). Felt and expressed need are usually obtained from interviews and surveys while normative and comparative needs are based on more grounded research and professional opinion (Smiley 2005).

Normative studies of needs assessments usually incorporate for instance, prevalence rates of a particular group while comparative needs focus on a comparison of services of a particular group.

Several factors affect the estimation of mental health needs of the community. The prevalence of people at high risk of admission to a mental health service will be only one of many factors which affect the need for care, as expressed as number of inpatient beds, outpatient slots and community mental health and primary care slots (and corresponding clinical resources) needed (Institute of Public Health 2011). Some of these influencing factors, among others, are likely to be:

- Number (and trend) of cases in community,
- Number of presenting cases in community per year (incidence),
- Catchment population for the facilities,

Clinical criteria and severity thresholds for those criteria, are used to make the clinical decision for referral from each part of the service to another ('discharge' threshold

As part of this PhD study, a mental health needs assessment (MHNA) was conducted to identify and appraise the current and future level of service provision for the long term mentally ill. In addition to collating current data and intelligence as part of a better commissioning process, the exercise was to further refine a tool for the more accurate estimation of the prevalence of

mental health in the community and offer some projections with regard to changes in mental health needs over the next ten years.

3.2. Aims

The specific aims of this MHNA were:

- To provide a broad health profile of the localities, with specific focus of the prevalence of mental illness in the London Borough of Brent.
- Explore patterns of access to primary services.
- Highlight areas of unmet need and gaps in provision.
- Highlight the limitations in available data and intelligence and expose gaps in understanding.
- Provide health intelligence for the development of a tool for a more accurate estimation of mental health prevalence and associated co- morbidities.

Approach

The structure of the needs assessment involved a population approach of identifying the chronic mentally ill group with a focus on areas of needs, supply, and demand. The concept of “need”, from a public health perspective, incorporates those needs felt and expressed by local people as well as those defined by professionals. It moves beyond the concept of demand and takes account of people’s capacity to benefit from health care and public health programmes. Supply refers to the number, type and distribution of services and resources available from all providers within Brent, public, private and third/voluntary sector. Demand is defined as the services that people ask for

and use, and can be difficult to measure. In this report, “activity” (i.e. numbers of people accessing and using services) can be used as a “proxy” for demand.

3.3. Methodology of the needs assessment

The needs assessment used rapid participatory appraisal (RPA) technique to complete a community health profiling (CHP). The latter has been described as an attempt to understand and describe the locality in order to prioritise need and has also been viewed as a ‘snapshot’ of the population, providing a systemic approach to assessing community health needs and resources (North East Public Health Observatory 2012).

This technique is often used in public health to gain community perspectives of local health and social needs with aims to translate these findings into action. Some researchers (Bowen 2008) considers RPA as a form of “action research” in that the researchers and participants undertake a collective, self reflective inquiry in order to understand and improve upon practices in which they participate and situations in which they find themselves. Some primary data were obtained through local services delivery units (NHS, Local Authority and the 3rd sector) – this information, as part of the PCT data collection routine, not only represented real-time service activity but also provided general information on local mental health delivery programme to enable a profiling of the local mental health community.

The RPA provides a robust framework in an adaptable structure (information triangle) that holds together data from various sources. It uses a multi-method approach and incorporates data that is immediately available from primary and secondary sources including the national census. This enables the researcher to draw inferences, conclusions or assessments in a limited period of time and is thus relevant to health service evaluation. Data collected from one source were validated or rejected by checking with data from at least two other sources or methods of collection. Informants are not selected randomly but “purposefully”—that is, asking a range of people who are in the best position to understand the issues. The approach allows only a brief time frame and uses limited resources.

The technique has limitations and statistics so produced must be interpreted cautiously as they may be based on routinely collected data, which may be of questionable accuracy, completeness, and reliability. However, the term “rapid” should not necessarily be taken to imply a “quick and dirty” method lacking in rigor. The inherent triangulation of sources of data and methods of data collection provides opportunities for cross-checking and validating findings throughout (Koelen, Vaandrager, & Colomer, 2001; Rhodes et al., 1999; Tones & Green, 2004). The cyclical process also provides the potential for members of the community to reflect on findings as they take shape, and encourages their active participation in the research process (Koelen et al., 2001).

3.3.1. Data extraction: - Exploring data sources (data mining)

The primary method was to explore local, regional and national datasets for a comparison of prevalence estimates and a basket of indicators reported and/or known about service usage, giving profiles at both Borough and locality levels.

Secondary analysis of the wider determinants of physical health and wellbeing in terms of healthcare services that supply either in primary or secondary care were considered.

Much of the epidemiological analysis in this profile has been undertaken using an anonymised patient-level dataset from GP practices in Brent (QOF registers) and some Hospital Episodes Statistics (HES). Data from the Public Health GP dataset are recorded using Read codes and the date of extraction can vary across GP practices. The data source is in the appendix 3. Discrepancies in numbers when comparing information from QOF and the Public Health GP datasets are due to the method of extraction and coding of disease conditions.

Secondary data was extracted using Dr Foster data and Local Authority. Dr Foster is a health informatics service that is used by the NHS to monitor the acute services (<http://www.drfoosterhealth.co.uk/>). The information routinely processed by Dr Foster include key information on admissions, discharges, length of hospital stay, demographics (including language and ethnicity),

behavioural and clinical risk factors, key conditions, details on the control and management of conditions, key medications and interventions.

3.3.2. Collecting data on service usage

All NHS Commissioners including Brent community Trust are required to organise meetings with stakeholders and healthcare professionals to explore and examine patients experiences of their care journeys. This enables the gathering of intelligence with regards to the usage of specific services and identified issues and performance details. The nature of information is inclusive of qualitative material from the “Quality Framework” service evaluations schemes which carried out routinely. Data for the mapping come from a variety of sources including nationally available statistics – the NHS Information Centre for Health and Social Care, Dr Foster and the local authority.

The study used this opportunity, as part of the needs assessment, to profile the mental health services delivery programme. Through a number of iterative discussions the exercise produced the mapping of existing health and social provisions in relation to model patient pathways and service uptake. Routine information was collated on service provisions, uptake, complaints, satisfaction of services and included identified gaps in current services and other provisions.

3.3.3. Analysis framework

Analysis framework

Based on material uncovered, the study provided recommendations for ways to further improve both the services and ways by which they can be managed. It must be noted that any analysis involving service provision is difficult due to the fact that there is a great deal of change in service provision at the present time. This is due to NHS re-organisation and the economic climate. Subsequent meetings of the project group confirmed that the focus of the needs assessment was a population approach of identifying at-risk groups and areas of need, including the prevalence of mental health problems. The MHNA was a complex exercise and required a critical pathway for logistic reasons and other practicalities. As such, the exercise was undertaken and built up through a number of stages.

3.3.4. Case definitions for serious mental illness

There is no clear global definition of severe and enduring mental illness (Gask et al 2000, Slade et al 2002). However, the Audit Commission (1994) defined severe mental illness as affecting those patients with a diagnosis of psychosis and compulsory admissions, or aggregate of one-year stay in hospital over a five-year period, or three or more admissions in the previous five years. The definition outlined by the DH (1995) considers diagnoses of psychosis, severe neurotic illness, personality disorder, dementia, with aspects such as a history of self-harm, self-neglect or violence.

For this study, the QOF definition (as used by the commissioners) of SMI is used. This includes the ICD 10 diagnosis groups F20-29 (schizophrenia and delusional disorders), F30-39 (affective disorders like depression), and F60-69 (personality and behavioural disorders). For the diagnosis groups F 20-29 the terms 'psychotic disorders' and 'functional psychosis' are used interchangeably. These categories are in keeping with the ONS Psychiatric Morbidity Survey. The needs assessment excluded dementia and drug and alcohol dependencies.

3.4. Profile of the London Borough of Brent

Brent district covers 43.2 square kilometres and is located in the North West of London. Officially it has a population of 270,000 (ONS 2006) although Council-commissioned research suggests that this figure could be over 15,000 higher and is growing steadily. Recent figures indicate significant numbers of people moving into the borough creating new emerging communities, as well as significant numbers of transient people within the borough.

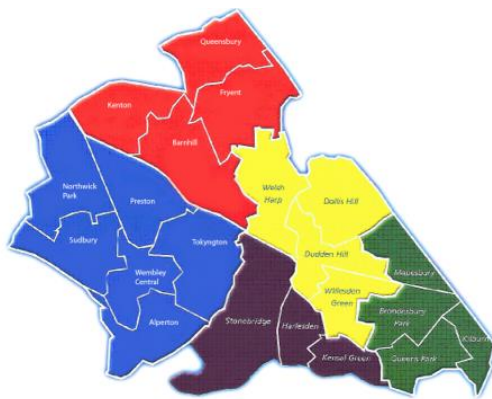


Figure 1. London Borough of Brent and its localities

The GLA predicts that Brent's population will increase by roughly 10,000 people every ten years and is predicted to be 305,575 by 2018.

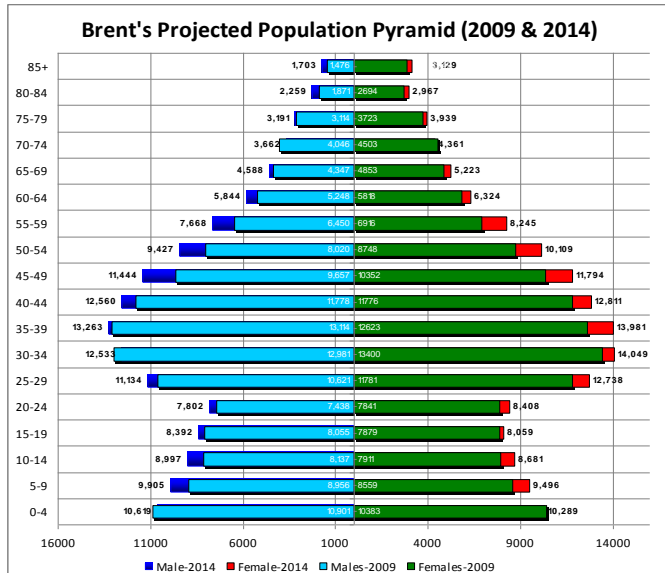


Figure 2. Brent projected population

Diversity

Dynamic population movements have resulted in Brent becoming the most ethnically heterogeneous borough in the country (Office of Statistics). It is one of only two local authorities serving a population where the majority of people are from ethnic minorities, and these groups are growing faster than any other borough (Fig 2). This means that the chances of 2 people in Brent being from different ethnic groups are higher than anywhere else in the country. Black and minority ethnic (BME) groups make up the majority of the population at 54.7% including 18.5% Indian, 10.5% Black/Black British Caribbean and 7.8% of Black (other). Approximately 130 languages are spoken in schools in Brent

and it has the highest proportion of people born outside the EU in England and Wales.

In the next 10 years the BME population is expected to increase to 60% of the population. The largest increase is expected to be in the Asian population which is expected to increase to just under a third of the population (32%) by 2014. Substantial increases are expected in the numbers of people in BME groups aged 30-65 years and smaller increases in people aged 65 years or over. This will have implications for the demand for health care as Asian groups tend to have higher rates of diabetes and heart disease and develop these diseases about 10 years earlier than white groups, whilst black groups have higher rates of diabetes, hypertension and stroke and also develop these diseases earlier

Different ethnic groups are concentrated in different parts of the borough. The highest concentrations of black residents are in Stonebridge and Harlesden wards. Asian residents tend to be located towards the west of the borough and the white population towards the east. Kilburn, Mapesbury and Dollis Hill wards have the highest numbers of white Irish residents.

Due to the fact that ethnicity was not routinely collated as part of the GP dataset (before 2011) we have an incomplete picture. However, the available data shows that, within GP practice, the ethnicity of 32.74% of people on the severe mental illness register is unknown. This may introduce a bias into any further analysis of the ethnicity data as it is not possible to say whether the

people with unknown ethnicity follow a similar prevalence to those that are unknown.

Deprivation

Brent has an IMD (Index of Multiple Deprivation) score of 29.22, which means that it ranks 53rd out of the 354 boroughs in the country i.e. it is in the 15% most deprived local authorities in the country. The indices combine information on economic, social and physical issues to produce scores for small areas across the whole of England. These indicators are used as proxies for levels of deprivation and socio-economic status. All mortality rates, admission rates and prescribing data have been linked with the Index of Multiple Deprivation by electoral ward. The higher the score, the more deprived the ward.

For the purposes of this mental health needs assessment the Index of Multiple Deprivation 2007 (IMD) has been used, primarily because it is the most comprehensive of the available indicators, as it takes into account not only employment status, but also broader determinants of health such as education, housing and geographical access to services.

However, Brent has also large sections of the community which are relatively affluent; The neighbourhoods experiencing the highest deprivation are largely located in the south of the borough. Our most deprived residents also have the lowest income levels, highest unemployment levels, poor and overcrowded housing and the worst health outcomes (Figure 3).

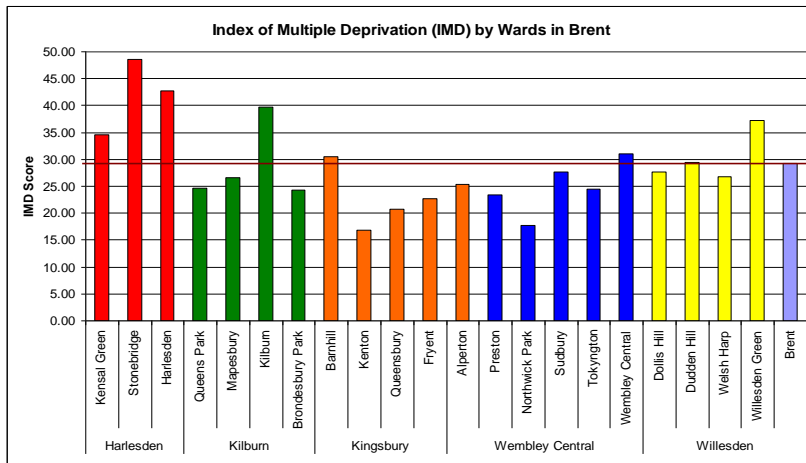


Figure 3. Deprivation scores (IMD) by localities.

Life Expectancy

There is a 9.3 year gap in life expectancy between the lowest (Harlesden-south) and highest wards (Northwick Park – north). Differences in health within Brent are dramatically illustrated by examining male life expectancy along the Bakerloo line. A journey of 3.5 miles takes you from Harlesden, which has the lowest life expectancy for men, to south Kenton, where male life expectancy is approximately 9 years higher.

The gap in life expectancy in Brent has persisted over a number of years. Recent figures show a slight reduction in the gap; however, this is because of a reduction in life expectancy in Northwick Park rather than an improvement in Harlesden. Life expectancy for women in Brent is 83.4 years; this is significantly greater than the England average of 80.9 years and London at 82.0 years (2007-2011). Life expectancy for men is 78.2 years which is

approximately equal to the England and London average of 77.4 years (2004-2006).

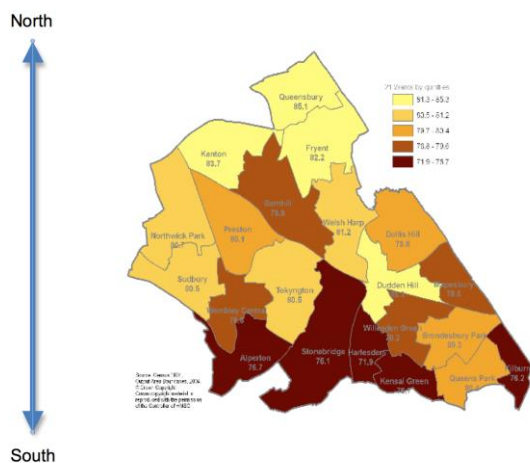


Figure 4: Life expectancy in the Borough. Shows a 7 year gap between north and south (within a distance of 3.5 miles).

Life style and health profile of Brent

Below (figure 5) is a summary of data collected annually for the locality. These reflect the overall pattern of lifestyle across the borough. It does not give an distribution across the various localities of Brent.

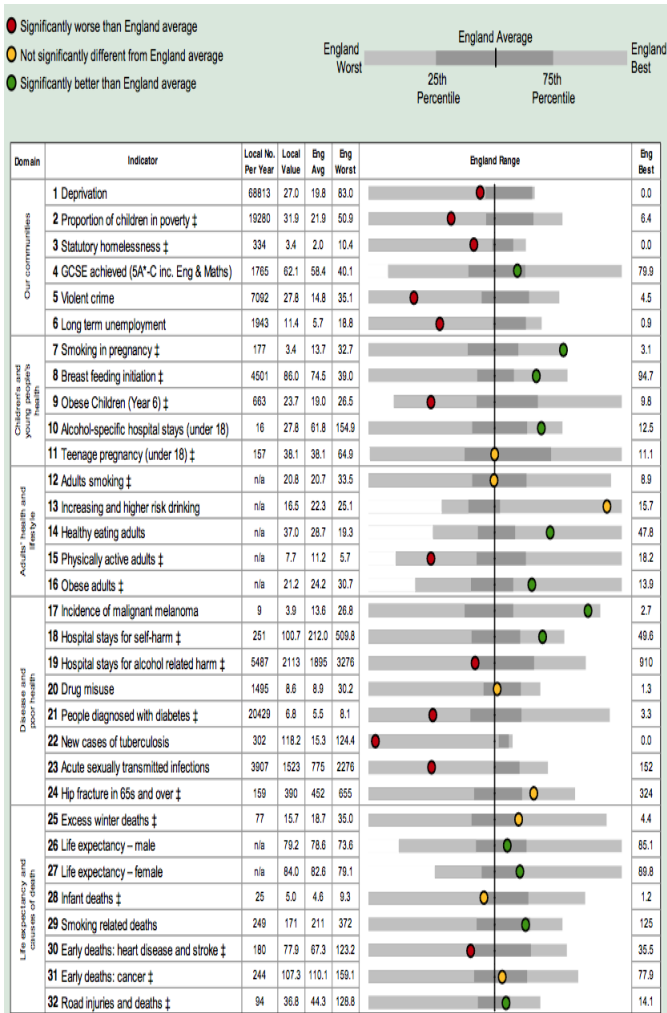


Figure 5. Summary of the Borough's health profile (2010-11)

3.5. Disease profile -

Mortality

Standardised mortality ratios (SMRs) are a measure of how more or less likely a person living in a particular ward is to die compared to that of the standard population, in this case England and Wales. This measure takes in account differences in the age and sex structure of a population. A value of 100 indicates that there is no difference in mortality compared to the rest of England, a higher value suggests that mortality is higher than England and vice versa.

SMRs for both males and females have improved considerably over the past decade. Males, aged 15-64, SMR has decreased from 146 in 1993 to 106 in 2011. The SMR for females, ages 15-64, has decreased from 131 in 1993 to 98 in 2011. There has also been a significant reduction in mortality rates from circulatory diseases and cancers. But there are pockets of relatively high social deprivation and no data exist on the local variations with regards to SMR estimation

Morbidity

QOF

Quality and Outcomes Framework (QOF) is a voluntary incentive scheme for GP practices in the UK, rewarding them for how well they care for patients. It contains groups of indicators, against which practices score points according

to their level of achievement and gives an indication of the overall achievement of a practice through a points system.

Since 2004 there has been a central collection of information from GPs about how many of their registered patients have certain conditions as part of the QOF. This provides information about the prevalence of key conditions in Brent and compares it to similar information for the rest of NHS London Strategic Health Authority and the rest of the country. There are limitations to the data. It should be remembered that not everyone with these conditions will be registered with a GP and of those that are, not all will be reported by the GPs practice. In some conditions, such as diabetes, the true prevalence will be higher than the QOF data suggests, because many people have the disease for some time before they develop symptoms and are diagnosed.

3.6. Selective profile of physical diseases common to SMI population

Key Facts (refer figure 6) :

- QOF average data revealed that during 2010-11, 63,396 patients were on Smoking Register (prevalence 18.10%). Overall cardiovascular diseases (e.g. hypertension, CHD, stroke) prevalence was about 15%. Prevalence of CVD related diseases conditions (obesity, diabetes, CKD) was 13.5%.

- Cardiovascular diseases and cancers are Brent's biggest killers, and mortality rates are up to 50% higher within the most deprived wards. Healthy lifestyles and early intervention can have a major impact on these deaths.
- Respiratory disorders are a major cause of mortality, morbidity and health inequalities in Brent.
- There are likely to be large numbers of individuals with COPD who remain undiagnosed.
- There has been a large increase in the emergency hospital admission rate for asthma over the last few years.
- Rates of admission due to pneumonia are significantly higher than the rest of London.
- Chronic disease and Long Term Conditions (LTCs) are endemic in Brent; for example, diabetes prevalence is amongst the highest in the country (and second highest in London) at 5.61% of the population diagnosed and additional undiagnosed cases of circa 2%.
- The prevalence of diabetes is expected to increase to around 8.5% of the adult population by 2014. In addition, prevalence of key diseases such as hypertension, CHD and COPD will increase over the next 5 years.
- Rates of tuberculosis (TB) in Brent are amongst the highest in the country.
- The number of people over 75 with dementia is expected to increase from 2,027 to 2,226 between 2009 and 2014.

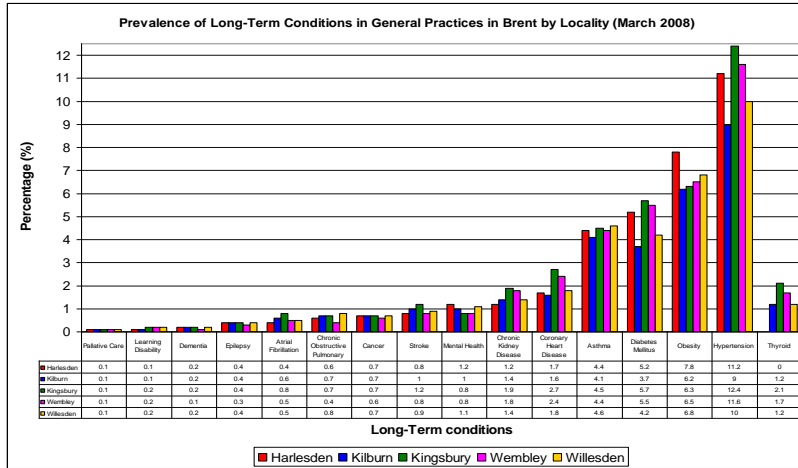


Figure 6: Reported prevalence of diseases linked with SMI population

3.7. Mental health profile

Serious mental illness (SMI)

It is estimated that severe mental illness affects around 1% of the population . Almost 0.9% of registered patients in the borough are registered as being followed up or treated for a severe mental health problem in primary care (similar to national figures). Under the mental health clinical area, practices are asked to maintain a register of those with Serious Mental Illness, defined as those suffering from schizophrenia or bipolar disorders or other psychoses. It does not include the sizeable group of individuals suffering from personality disorder or severe depression.

QOF registers are used routinely to manage the serious mentally ill. Currently they provide the following statistical evidence for each GP practice in Brent using the 6 registers namely:

- **MH9:** The percentage of patients with schizophrenia, bipolar affective disorder and other psychoses with a review recorded in the preceding 15 months. In the review there should be evidence that the patient has been offered routine health promotion and prevention advice appropriate to their age, gender and health status.
- **MH8:** Is a register of people with schizophrenia, bipolar disorder and other psychoses.
- **MH6:** The percentage of patients on the register who have a comprehensive care plan documented in the records agreed between individuals, their family and/or carers as appropriate.

- **MH7:** The percentage of patients with schizophrenia, bipolar affective disorder and other psychoses who do not attend the practice for their annual review who are identified and followed up by the practice team within 14 days of non-attendance.
- **MH4:** The percentage of patients on lithium therapy with a record of serum creatinine and TSH in the preceding 15 months.
- **MH5:** The percentage of patients on lithium therapy with a record of lithium levels in the therapeutic range within the previous 6 months.

3.8. QOF information

a. Case registers

In 2010-11, 0.98% of the Brent registered population (all ages) were on the primary care serious mental health register, a total of 3455 individuals. An apparent increase between 2008/09 and 2009-10 may reflect more specific criteria for inclusion on the SMI register. Using the 2009 reconciled population base (lower than the unadjusted 2008 population base), the rate is 0.90%. Practice rates varied from 0.4% to 1.8%. Overall the locality prevalence rate is above national and SHA levels with some practices incurring almost twice the national level (approximately 67%). As expected, there is wide differences within the geographical region with some practices showing very low level of mental illness (< 0.04%) as shown in figures 6 and 7 below. The commissioners needs t seek re-assurances on the accuracy of the data to explain the wide variations.

These indicators are the proportion of people registered to GPs who are on the QOF register with severe mental health problems schizophrenia, bipolar disorder and other psychoses in primary care. The data should not be interpreted as 'disease prevalence'. QOF data do not necessarily present an accurate picture of disease burden, as disease prevalence reported as low could be explained by under-recording or unmet need within the practice population. This information is more a measure of service use, practice recording and service quality for people with severe mental health problems managed in primary care. Figures 7 and 8 show the pattern of activity for the MH09 registers.

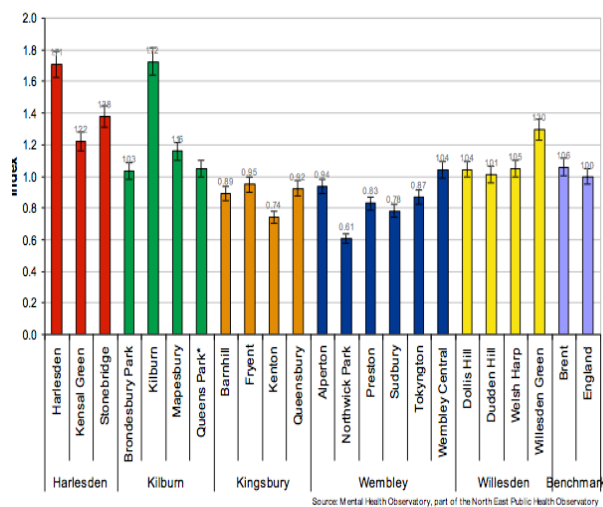


Figure 7: An overview of the SMI (QOFs) across the various localities.

Data source: OMAS database - 2008/09
 data as at end of June 2009

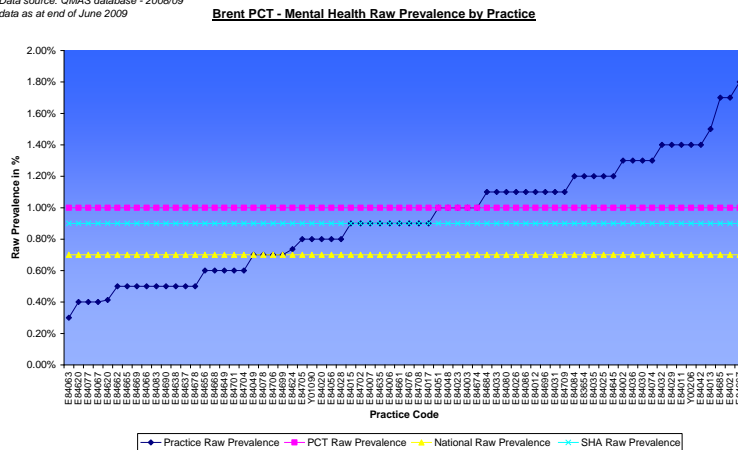


Figure 8: Mental health raw prevalence as indicated by the QOF register in Brent and compared to the UK.

b. Physical review recorded for those on SMI register

Patients with serious mental health problems are at considerably increased risk of physical ill-health than the general population and shorter life expectancy (Phelan et al. 2007). It is therefore good practice for a member of the practice team to review each patient's physical health on an annual basis. Health promotion and health prevention advice is particularly important for people with serious mental illness. However, there is good evidence that they are much less likely than other members of the general population to be offered these kinds of checks.

Overall, 91% of those who are on the SMI register and eligible for an annual review are recorded as receiving one (QOF Indicator MH09). Figure 9 shows the percentage of patients with schizophrenia, bipolar affective disorder and other psychoses who had an annual review is high and compares favourably

with the national level. The PCT scores compares favourably against national figures (figure 9). However, the Trust needs to explore the patterns of exceptional reporting on its chronic mentally ill patients. shows that there was little variation by locality. Seven practices had a percentage less than 80%.

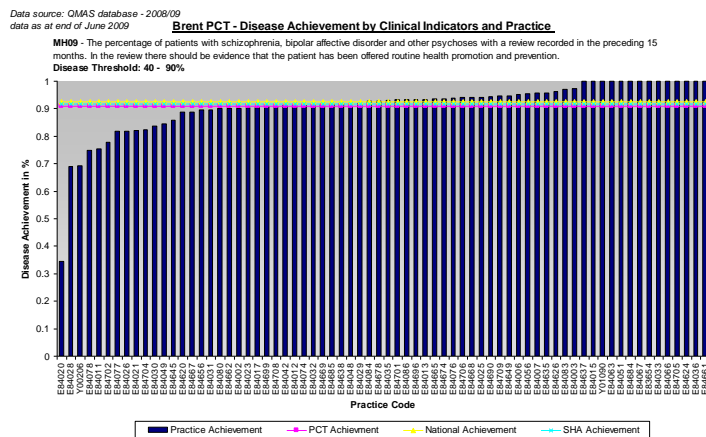


Figure 9: QOF Performance

c. Care plans for those on the SMI registers

QOF guidance states that patients on the mental health register should have a documented primary care consultation that acknowledges, especially in the event of a relapse, a plan for care. The care plan which should be reviewed annually should include the views of their relatives or carers where appropriate.

Up to one half of people who have a serious mental illness are seen only in a primary care setting. For these patients, it is important that the primary care team takes responsibility for discussing and documenting a care plan in their primary care record. However, if a patient is treated under the care

programme approach (CPA), then they should already have a documented care plan discussed with their community key worker available.

Overall 88% of those on the SMI register are recorded as having a care plan (QOF indicator MH6). This figure should be 100% as a care plan should be developed for all those on the SMI register either with primary care or for those on CPA with the patient's care-coordinator.

d. Comprehensive care plans

QOF MH06 is an index of measurement of patients who had had a comprehensive care plan documented in the records. 21% of practices did not conform to this clinical guideline with some variations along geographical settings. 13% of practices had exceptional reporting in this category (figure 10). Although it is below the national average, it is imperative to understand this variation. If patients are being exceptionally reported it may be that some are falling through the net and may eventually get compromised.

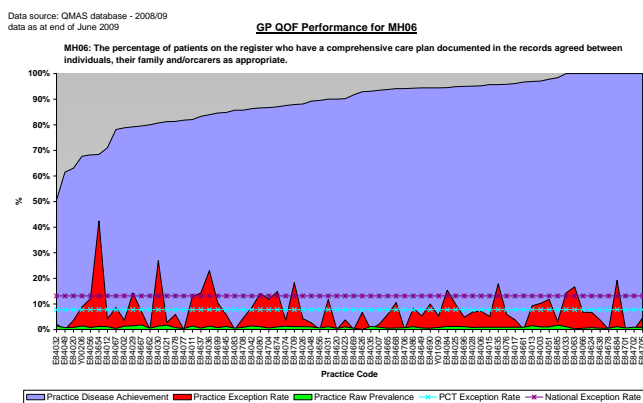


Figure 10: QOF care plans

Within the borough, the proportion of patients on the practice registers from 0.29% up to 3.9% (3.9% were registered by the Harness group in Harlesden. This practice provides services for homeless and transient individuals). Other practices with more than 1.2% on the register included practices largely in the south of the borough.

Analysis also showed that a significant number (65%) of SMI tend to be clustered around Central Middlesex Hospital (the acute mental services). This clustering nexus has been reported by Congdon (2011) for other parts of the UK. This geo-location of SMI is an interesting phenomenon which is further explored in the study as part of the COPD prevalence estimate.

Other key facts

The majority of people with severe mental disorders, as picked up in the assessment, are within the 18-64 age range, with 71% of females and 81% of males falling within this range. The male-female split is even with only 18 different between the two; however there are a higher percentage of females of an older age than males.

Psychotic disorders and bipolar disorders account for 90% of all the diagnoses (62% and 28% respectively). Local data confirmed the national rates from the Psychiatric Morbidity Survey 2010 that showed that more females than males would be expected to have a psychotic disorder within the 16-74 age range.

The data shows that, when grouped into quintiles of deprivation by Medium Super Output Area (MSOA), that the more deprived the area is the higher the prevalence of severe mental health issues. This is based on the deprivation of where the people actually live rather than on the deprivation scores of the practice as used in the QOF section. This would suggest a direct link between deprivation and severe mental illness.

Within Brent as a whole under 10% of patients who are on a GP severe mental illness register have not had a recorded review in the previous 15 months as at 31st March 2010.

The percentage of patients who are on a GP severe mental illness register and not followed up for non-attendance of a review is lower than 10%. However, there is a large variation across the locality with only 3.9% of patients not followed up in Wembley and 16.5% in the Harness locality.

The percentage of patients who are on a GP severe mental illness register and do not have a care plan is documented as being just under 11%. There is little variation across the PCT having the lowest percentage at 9.6% and the highest at approximately 13%.

3.9. Ethnicity and mental health

There is good evidence that there is an unequal distribution of mental health problems among black and various minority groups. As mentioned previously, Brent has the UK's most ethnically diverse population. Marginalised groups

such as asylum seekers and the homeless who are likely to have experienced traumatic life events are more likely to have complex mental health problems and therefore vulnerable to deliberate self-harm. Brent is committed to target its health promotion campaigns to these communities. A crucial factor is accessibility to service.

The rate of admissions for BME population is below that expected – significantly less than the London rate, and below the national rate (table 2). Similarly the white ethnic group admission ratios are significantly low compared to other parts of London (figure 11). However, this reflect the ethnic composition of Brent.

Brent Ethnicity 2010 GLA Estimates	Total Adult		Adult Inpatient			Adult Community		
					Variation			Variation
All Ethnicities	189,468	100.00%	660	100.00%	0.00%	2953	100.00%	0.00%
White	81,742	43.14%	208	31.52%	-11.60%	1138	38.54%	-4.60%
Black Caribbean	17,119	9.04%	147	22.27%	13.20%	477	16.15%	7.10%
Black African	15,066	7.95%	96	14.55%	6.60%	236	7.99%	0.00%
Black Other	5,541	2.92%	51	7.73%	4.80%	163	5.52%	2.60%
Indian	38,878	20.52%	35	5.30%	-15.20%	288	9.75%	-10.80%
Pakistani	7,898	4.17%	24	3.64%	-0.50%	125	4.23%	0.10%
Bangladeshi	653	0.34%	8	1.21%	0.90%	11	0.37%	0.00%
Chinese	2,673	1.41%	8	1.21%	-0.20%	11	0.37%	-1.00%
Other Asian	12,017	6.34%	42	6.36%	0.00%	201	6.81%	0.50%
Other	7,878	4.16%	43	6.52%	2.40%	303	10.26%	6.10%

Table 2: The percentage of community using mental health services in Brent.

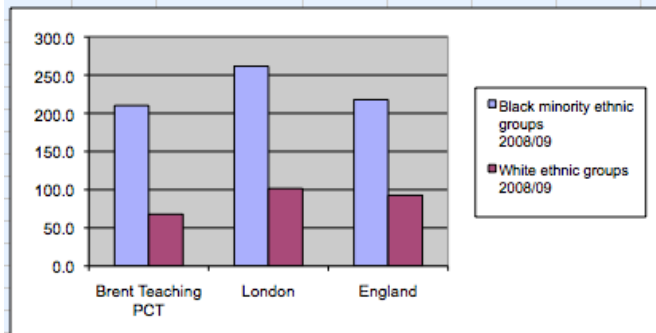


Figure 11: Use of services – A comparison with London and UK.

Co-morbidity

Co-morbidity is the presence of two disorders or illnesses occurring simultaneously in the same person. Surveys show that drug abuse and other mental illnesses are often co-morbid. Six out of ten people with a substance use disorder also suffer from another form of mental illness.

The prevalence of co-morbid alcohol, other drug, and mental disorders in the UK total community and institutional population is estimated to be around 22.5% for any non-substance abuse mental disorder, 13.5% for alcohol dependence-abuse, and 6.1% for other drug dependence-abuse. Among those with a mental disorder, the odds ratio of having some addictive disorder was 2.7, with a lifetime prevalence of about 29% (including an overlapping 22% with alcohol and 15% with another drug disorder). The highest mental-addictive disorder co-morbidity rate was found for those with drug (other than alcohol) disorders, among whom more than half (53%) were found to have a mental disorder with an odds ratio of 4.5.

Physical review recorded for those on SMI register

Patients with serious mental health problems are at considerably increased risk of physical ill-health than the general population and shorter life expectancy (Marder et al. 2004). It is therefore good practice for a member of the practice team to review each patient's physical health on an annual basis. Health promotion and health prevention advice is particularly important for people with serious mental illness. However, there is good evidence that they are much less likely than other members of the general population to be offered these kinds of checks. Overall, 91% of those who are on the SMI register and eligible for an annual review are recorded as receiving one (QOF Indicator MH09). There was little variation by locality. Seven practices had a percentage of less than 80%.

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3.10. Factors influencing need for mental health services

The community prevalence of those people at high risk of admission to a mental health service will be only one of the several factors, noted above, which affect the need for care, as expressed as number of inpatient beds, outpatient appointments and community mental health and primary care appointments (and corresponding clinical resources) needed. Influencing factors, among others, are likely to be:

- Number (and trend) of cases in community (prevalence)
- Number (and trend) of new presenting cases in community per year (incidence)
- Catchment population of the facilities
- Which clinical criteria and severity thresholds for those criteria are used to make the clinical decision to admit to services
- Availability (i.e. amount of) and development of service quality and quantity in each locality
- Which clinical criteria and severity thresholds for those criteria, are used to make the clinical decision for referral from each part of the service to another ('discharge' threshold)

3.11. Service provision in community services

Primary care services

Primary care provides a wide array of services and is diverse in terms of its organisation, the services offered and the professionals involved. The services for patients with mental ill health that are provided by primary care practitioners include health promotion; assessment and detection/diagnosis; management, advice and information, treatment including medication, psychological interventions or complementary therapies and referral; follow-up and continuing care of chronic and recurring disease; rehabilitation after illness; and co-ordination of services.

GP Services

There were 166 Whole Time Equivalent (WTE) GPs working in 72 GP practices in Brent as of 1st January 2009 included:

- 19 single handed practices
- 6 PCT salaried practices
- 12 PMS practices providing a range of services for refugees and asylum seekers, the homeless population and those who are unregistered
- There were 351,000 patients registered with a Brent GP as of the 1st January 2011. Patient turnover at approximately 20% per annum is high. The number of WTE GPs per 100,000 population weighted by age and need was 68.8 per 100,000 in 2006. This is higher than the England rate of 61.8 per 100,000 and the 15th highest in London. Analysis of primary care data within Brent shows a higher percentage of smaller practices as compared with national averages.

70% of Brent practices are one and two handed practices compared to 54% in London and 42% in England.

- GP practices list sizes are varying from just under 2,000 to about 15,000 patients. Overall highest list size is observed in Kilburn. Most of the smaller practices have about 2,000 or fewer patients. Most of the GP practices, except in Kilburn localities, have maximum between 8,000 to 11,000 patients.

Service uptake

Overview

There were a total of 769 admissions and 789 discharges to inpatient services in year 2008/9. Some patients may have been readmitted more than once in the year. Half of all patients are discharged within the three weeks and patients will usually spend less than six months on an acute inpatient ward, although problems with discharge may mean that this is not achieved in practice.

Analysis of the admission data highlights a number of issues. Males have higher admission rates for schizophrenia and delusional disorders than females, which reflects national prevalence rates for these conditions. Whilst females have nearly double the admission rates for mood affective disorders which reflects national prevalence rates for these conditions. Since 2005 there has been an increase of 20.8% in the total number of mentally ill in Brent.

A significant number of Brent patients with a mental health diagnosis are also admitted to acute hospital wards. In many cases, these are short stay for alcohol related mental health problems. Some may be admitted via acute A&E and then be transferred to specialist MH providers.

The rate for personality disorders provides an interesting local picture. Nationally men have a higher prevalence rate for these conditions, but Brent in-patient data illustrates that it is females who have higher admission rates than males.

Generally the number of admissions for schizophrenia and affective disorders is in line with predictions based on deprivation and demographics. There is however some variance in some wards. Brent standardised admission rates are generally lower than national rates, except for schizophrenia. Senility and organic mental disorders are rising over the last 4 years. There is an upward trend in number of alcohol-related disorders.

The rate of admissions for schizophrenia and related disorders was between 240 and 280 cases over the last 5 years. Apart from senility related disorders, most diagnostic groups do not showing any significant trend.

It should be noted that the arithmetic mean or 'average' is often used to compare stay lengths between different hospitals. This is not an ideal method as averages can be badly distorted by small numbers of long-stayers. The most appropriate figure to reflect overall stay length for an in-patient unit is the median – i.e. the number of days by which 50% of admissions will have finished. For Brent over the last 5 years the median (13 days) is considerably lower than the average (50 days).

Key details

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3.12. Commissioning model

The study sought to explore needs, demands, use and outcomes within mental health services using a more refined local prevalence index and how these can be framed into a framework that could enhance commissioning. The fundamental relation between these variables is complex.

Essentially, the mediating factor relating need, demand and activity (e.g. how much need is turned into demand) are the clinical decisions at each stage, including patients' decisions whether to seek services. To appreciate this dynamic, it is best to use a framework to explore the relationship. This can be summarised by this schematic diagrams (figures 12 and 13) below.

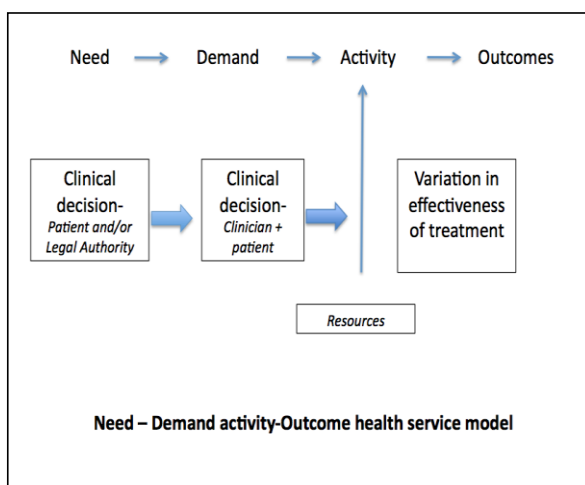


Figure 12: Schematic diagram showing the decision flow for service commissioning

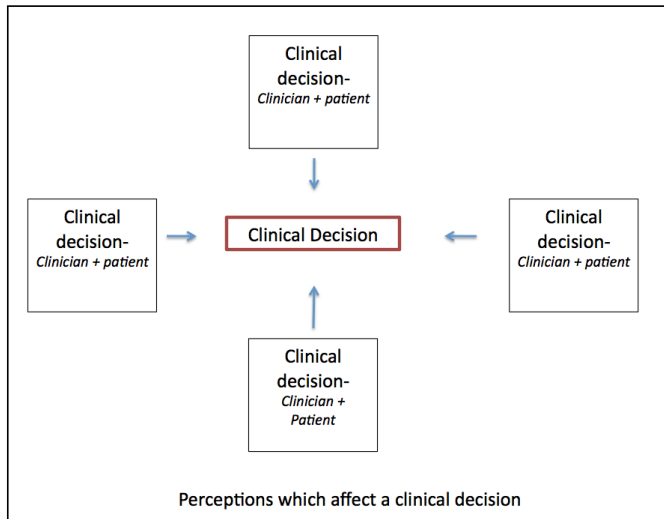


Figure 13: Schema for service commissioning using clinical decision flow.

However, the other factors are equally important in mediating the transition from need to activity, via 'demand' and together produce a feedback loop increasing (or decreasing) 'supply induced demand', which is often 'supply awareness demand.'

This makes it difficult to use epidemiological estimates of prevalence of mental disorders to estimate 'need for mental health care'. This is because the 'need' depends on the thresholds of severity at which the clinical deciders use to define a 'case' requiring treatment. In turn, this severity decision depends on the effectiveness of treatments: some treatments might only be effective on mild cases; other treatments might only be suitable for more severe cases because they have side effects which are only worth being exposed to if the severity of need (and therefore potential benefit) is large.

It is more difficult to use the epidemiological 'need for care' (even assuming that thresholds of admission to various mental health services are operationally agreed and adhered to in practice) because treatment activity is incompletely effective and suboptimal outcomes either prolong treatment or require readmission to services, thus increasing the amount and kind of mental health service provision required.

Therefore, the appropriate commissioning/planning question is not how many mental health service resources of various kinds do we need. The appropriate question is, given the reality of present patterns of resources, what kind of information do we require to have an effective commissioning outcomes?

This whole complexity of public health planning depends ultimately on the need to have good epidemiological information and good prevalence estimates are essential to this process.

This suggests that prevalence of severe mental illness, a widely used proxy for levels of long term mental health care need in local populations, can be effectively modelled using regularly updatable population data published by the Office of National Statistics area-type classification with local primary care data.

3.13. Limitations of the needs assessment

There are gaps in the needs assessment - what is presented is for areas where data is available, and what is possible within the time limits of the

project. It was not possible to carry out a full assessment of user engagement in the commissioning process. Other areas not covered that could be considered in future needs assessments include the mental health needs of people with other types of disability, including visual impairment, HIV and physical disability.

Date Quality

The reliability of data is crucial in the whole aspects of service design, planning and delivery. Quality data underpins everything from needs assessment, to pathway development from service redesign to defining positive outcomes for users.

However, there is a national consensus that data generated from this area is at best contestable. Mental health data has a reputation for being of poor quality and reliability. This is an area that needs to be addressed urgently. It is applicable across primary and secondary care. Raising awareness amongst clinicians and practitioners in primary and secondary care on the importance of good clinical coding should be seen as an important element of their work not a tedious bureaucratic exercise.

A further development that would greatly advance the relevance and accuracy of such comparisons would be to feedback information to each participating trust, so that each could decide on a knowledge-based plan to improve specified areas of practice in anticipation of a subsequent further appraisal, thus continuing and enriching the audit cycle.

3.14. Conclusion

The prevalence of enduring mental illness identified is over three times that which was expected using national prevalence data underlining the particular challenges of mental ill health in deprived localities. The overall prevalence of patients identified as suffering a long-term mental illness was three per 1000 patients registered, but rates varied widely between practices, in part due to a higher prevalence of patients with psychotic disorders in the more disadvantaged areas, which was not unexpected. The methods used here would not have identified long-term mentally ill patients in the community who had not been in touch with any health or social services for some time, such as the homeless.

In the south of the borough there were some high mean general practitioner consultation rate of 8.1 consultations per year in some areas compared with the rate of 6.5 per year found in the north sector. Though few in number, most long-term mentally ill patients are demanding of general practitioners' time. However, 29 patients (7% of the total) had had no recorded contact with their general practitioners for a year. This confirms suspicions that some disabled long-term mentally ill patients are not seen regularly by their general practitioners, although they seem to be few in number in these practices.

According to practice records, most contacts with general practitioners were for minor physical problems, repeat prescriptions and sickness certificates. It

is possible, however, that mental state review occurred more often and was not specifically recorded in the notes.

The findings presented here suggest that patients in long-term contact with specialist services cannot be taken as representative of the whole population with long-term mental illness.

This study has demonstrated that long-term mentally ill patients can be readily identified in general practice. General practitioners could perhaps use their contacts with these patients to play a greater role in monitoring their mental state and psychotropic medication.

Future direction

For effective planning and commissioning, the commissioners need information on:

- The prevalence of disorders in the present and in the future
- The prevalence of the determinants of mental illness
- Current service activity and predictions of likely future activity

The commissioners should work with acute mental health services to develop accurate, comprehensive and timely data on each of these four areas. The first two are for the most part not directly under the control of the commissioning process and developments in this should be about developing a resource / database of local and national data sources which are of benefit in planning, forecasting and commissioning services. The APHO report on

mental health Indications of Public Health in the English Regions (APHO, 2007) provides a useful starting point but not sufficient. At regional level it presents a wide range of data on the factors which can give rise to poor mental health, the mental health status of populations, provision of interventions of care for mental illness, effectiveness of partnerships, service user experience, workforce capacity and traditional outcomes such as suicide.

To develop service intelligence on activity and outcomes, there are a number of relevant initiatives that need to be considered. It is important to combine the national mental health observatory (mental health minimum data set) with a local pathfinder site minimum data set for a better prevalence estimate within small area population. Locally, links should be made to relevant agencies to understand what data they might hold, its strengths and weakness and how it might be used to support the commissioning process.

Finally, the use of MHNA as a methodology to understand and analyse risk factors at neighbourhood level cannot be emphasise too highly. In an area like Brent, the mobility of its resident is fluid. Annually, there is around 30% of the population shift, particularly in areas such as Harlesden and Kilburn. National prevalence estimates are not sufficiently sensitive to these changes. Routine MHNA can help commissioners with a useful guide for health investment in areas of needs. This is a powerful tool in shaping better local health prevalence measures.

4. **Local area estimate prevalence modelling**

4.1. A methodological framework

Health commissioners and public health departments depend on good local health intelligence to plan services. Where it is not possible to obtain local prevalence data, the only way is to estimate the number based on national prevalence and local resident population. This makes the assumption that the national prevalence estimates apply and reflect the local population setting. Chronic conditions like cardiovascular and respiratory diseases risk factors such as high blood pressure, cholesterol, smoking, diabetes and physical inactivity are linked to socio-economic determinants such as working conditions, housing, or social relationships. These parameters vary from locality to locality within and between borough and districts. As such crude estimates based on national surveys which ignore local differences can be sometime misleading and may have a serious impact on service delivery. This scenario is further complicated when dealing with a population suffering from co-morbidities.

A pragmatic model that derives information from national estimates and factors local variances into national predictions is proposed. This approach aimed to provide a more “realistic” prevalence estimates of CHD and COPD within primary care was discussed and was supported by the local commissioners and the public health department.

The study aimed to generate a model to estimate local level prevalence rate of two diseases namely CHD and COPD in the first instance and to further refine this estimation at GP locality area level. The model is then applied to determine the prevalence of a sub population group namely the SMI with concurrent medical morbidities of the two conditions (as single disease entity or a combination of both).

The definition of a “local level” refers to a borough/metropolitan area of approximately between 300,000 - 450,000 patients (size of a typical PCT). A “GP locality”, on the other hand is a small area covering between 10,000 – 25,000 patients. The approach does this specifically through an adaptation of the methodology used by the UK’s office of national statistics, known as synthetic regression estimation fitted using local level co-varieties. The choice to focus of two conditions namely CHD and COPD was based on the epidemiological differences including is a significant volume of “undiagnosed” cases in COPD (in relation to CHD) as highlighted by APHO (2008). Osborn et al (2007) also suggest that the people with SMI suffer from COPD co-morbidity due to the fact many of them are smokers and live in socially deprived and challenging areas.

The estimate modelling is done through an iterative process involving a number of phases. This approach facilitates sequentially the extraction of the data with each phase feeding the next step. In the first instance, there is a need to establish how the chosen physical medical condition prevalence is estimated at regional level. The next step is to derive a local prevalence

estimate model for each of the co-morbidities. It is important to remember that the prevalence figures generated by existing models are estimates of the expected prevalence of disease for national levels. Due to local variances for a wide range of factors, discrepancies between modelled estimates at practice level and other sources of data such as QOF disease registers may be due to local variations not captured by the model and cannot be solely attributed to weaknesses in QOF data. For practices with populations that significantly differ from a 'typical' population (e.g. large black or ethnic minority population that has very different smoking pattern to the local average) the assumptions of the model may not apply and discrepancies may occur.

In summary, this is an iterative sequential process that draws from the national prevalence estimates to predict the prevalence of a chronic physical disease within the local practices population base. This is done through the following sequence:

1. Determine the estimate prevalence model of a small population area for a stated disease condition
2. Develop the local template to adjust regional estimation for that disorder
3. Apply the local template to estimate the prevalence of that disorder within the SMI population using a probability estimate

a. Local area prevalence estimate

A multivariate modelling analysis using data from Health Survey For England (HsFe) and regression models are used to estimate of both CHD and COPD at CCG (PCT) level. The variables include demography, socio-economic

factors, smoking behaviours, QOF data and attributes associated with the named conditions. The study also used geographically weighted regression (GWR) to associate geographical patterns to disease conditions namely COPD. Geo-location habitat patterns for chronic diseases have become a concern for public health. They create “health ghettos” that makes planning and delivery of care harder.

b. Developing estimate template for smaller areas (GP practice level).

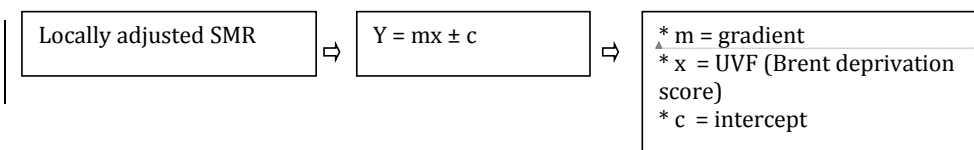
Using the regional prevalence rates, the re-adjustment of the number of patients is calculated using an approach suggested by Nacul et al (2008) and recommended by APHO. The rationale takes consideration of the local health inequalities and makes an adjustment. The assumption is that increased mortality (SMR) of a condition will reflect an equivalent increase in that disease prevalence, thus using an SMR ratio for the adjustment factor. For example, the national estimates suggest if a condition (A) in Brent has an estimated SMR of 155, then the prevalence in each GP in the locality is increased by 55%.

Since all GP practices in the borough will have different levels of deprivation (as measured by UV67 for each practice), an adjustment factor is introduced. This factor (multiplication factor) takes into account the local deprivation scores (based on UVF).

The proposed algorithm is as follows:

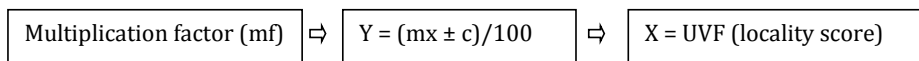
Calculating a locally adjusted prevalence estimate

Step 1

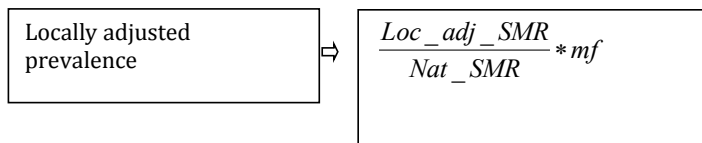


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Step 2



Step 3



Step 1

Using national estimates, a prediction of the prevalence rate is made for the practice. It takes account of local variances within Brent. In the absence of sufficiently precise published data on the relationship between deprivation and condition (x), the model makes an assumption that areas with higher mortality rates have a comparably higher prevalence of that disease. For example, assuming condition (x) in Brent is reported to have an SMR of 117, then the model increased the predicted prevalence in each practice in the locality by 17%.

Step 2 – The multiplication factor

This step establishes a multiplication factor (mf) which will be used to correct the local differences. The APHO endorses this form of statistical adjustment in modelling framework (Technical Briefings 3, 2008). This is derived from a linear regression equation of $(Y = mx \pm C)$, where **m is the slope and (+/-)C is intercept**. The mf is derived by dividing the above equation by 100 namely, $mf = ((\text{Expected local SMR} \times \text{UV scores})) / 100$. Applying this factor to a ratio of the adjusted disease SMR (local) to national SMR will yield an a more locally sensitive prevalence rate.

Thus, a GP locality with a UV67 score of 20% (level of deprivation) will yield a linear relationship like : $\text{Cond}(x) \text{ SMR} = (2.604 \times \text{UV67}) + 25.97$, where the slope is 4.39 and 16.04 is intercept. The mf is calculated for each locality (based on UVF scores) and will be factored in the adjusted prevalence estimation.

Worked example

For example, let us assume that Brent's standardised mortality ratio (SMR) for condition (x) was 140 - Increased the predicted prevalence of Brent by 40%.

Locality	Harlesden	
SMR prediction (borough)	140	* based on national estimates
UV67 score (for a practice) eg E84077	35	* based on 2001 Census Regression line: $\text{Cond}(x) = (4.389 \times \text{UV67}) + 16.04$
Adjusted SMR	169	$(4.389 \times 35) + 16.04$
Derived multiplication factor	1.69	* $((4.389 \times 35) + 16.04) / 100$

Step 3 – Prevalence (local estimate - adjusted)

Adjusted prevalence rate (AP) = (SMR (calculated on local UV67 scores)/national (SMR estimates))/multiplying factor

C. Determining the prevalence estimate of SMI with physical co-morbidity

Having established the prevalence estimate for condition (x) for the locality, the next step is to calculate the likelihood of the presence of that condition from our SMI population. Ideally, we should also have developed a local estimate of SMI using the above technique, but due to technical and clinical issues, this is not possible to date. The APHO is currently developing an approach to resolve this problem. Instead, we use the QOF data (routinely collected by GPs) to have a crude estimate of the number of SMI within the locality. The estimation of concurrent co-morbidity is carried out through probability modelling (using Bayesian statistics). A schematic illustration is presented below (figure 14).

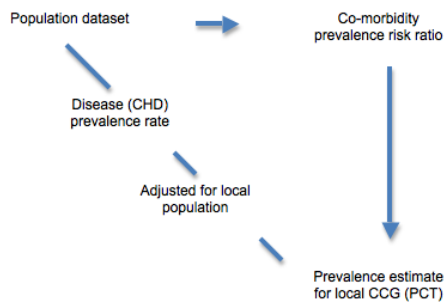


Figure 14 : Schematic diagram to describe algorithm for establishing prevalence estimation for co-morbidity among SMI at local levels

Here, the study sought to establish the probability that two conditions e.g. CHD and COPD (A, B) are prevalent in our local SMI group. This necessitated an estimation of what are the chances all, none or one of these conditions given that they have SMI. To calculate this, we must to determine:

1. The probability of having 2 disorders
2. The probability of having none of these conditions
3. The probability of having one of the conditions
 - The probability of having 2 conditions is a product of the separate prevalence: $P(A,B) = (P(a))(P(b))$
 - The probability of having one of the diseases, namely
$$P(1) = \{(Pa)(1-P(b))\} \{(Pb)(1-P(a))\}$$
 - The probability of having two (2) diseases,
$$P(2) = \{(Pa)(1-P(b))\} \{(Pb)(1-P(a))\}$$
 - probability of having none of the conditions,
$$P(0) = (1-P(a))(1-P(b))$$

These probability estimates are then applied to the local data sets of GP practices.

5. Disease prevalence model development

5.1. Introduction

The Association of Public Health Observatories (APHO) has produced estimates of the number of people in each primary care trust (co-terminous with local authorities) who they estimates have CHD. Whilst this is useful it is probably more useful to examine this at practice level. The accuracy of the estimates depends on the modelling methodology. This section considers the development of a model for coronary heart disease (CHD).

5.2. Coronary heart disease model (CHD)

Coronary heart disease (CHD) is the UK's biggest killer, with one in every four men, and one in every six women dying from the disease. Nationally, around 300,000 people have a heart attack each year, and 1 in 50 people have angina, an estimated 1.2 million people with the condition (Minnino *et al.* 2000). It is widely recognised that coronary heart disease is a major cause of health inequality and a major course of premature mortality under 75 years of age. Nationally, just over a quarter of deaths in people under 75 years of age are due to circulatory disease, and of these, over half (57%) are due to coronary heart disease, In addition, coronary heart disease alone is estimated to cost the UK more than £7 billion each year (Jordan *et al.* 1998).

The HSfE for 2006 estimated, based on respondents' self-reports of doctor-diagnosed CHD, that the prevalence is about 6.5 per cent in men and 4.0 per cent in women, and this increases markedly with age (Minnino *et al.* 2000).

This prevalence has remained static over the last ten years. However, the QOF, covering over 8,000 practices and 53 million patients, shows a GP-registered unadjusted prevalence of only 3.5 per cent (but note that unadjusted prevalence rates show these registers as a percentage of the total practice list size i.e. for all ages) (Jordan *et al.* 1998; Phelan *et al.* 2001).

The disparity between CHD prevalence estimates from large surveys, in particular the HSfE, and the number of patients diagnosed with CHD and registered in QOF led to demand for a CHD prevalence model at PCT and Local Authority level that gives an accurate estimate of true prevalence (Druss *et al.* 2001). The Association of Public Health Observatories (APHO) published a simple prevalence model to support development of 2009-10 Local Delivery Plans. However, it was acknowledged that this was a crude model and that more was needed to make sensitive model for local use.

Prevalence terminology

The term CHD can be confusing and this may often mask its true identity and this may lead to various methods of estimating CHD prevalence. Questionnaire responses such as those used in HSfE to define CHD, may be less accurate than clinical diagnosis. Conversely, reliance on a medical diagnosis may under-estimate prevalence, as patients with unrecognised angina or very mild symptoms may not attend (or be correctly identified by) their GP.

Defining prevalence

There are differences between various methods of estimating CHD

prevalence. Questionnaire responses such as those used in HSfE

to define CHD, may be less accurate than clinical diagnosis. Conversely, reliance on a medical diagnosis may underestimate prevalence, as patients with unrecognised angina or very mild symptoms may not attend (or be correctly identified by) their GP. A Belgian analysis of the records of four large Belgian epidemiological studies during the past 30 years compared clinical and electrocardiographic (ECG) findings showed that Q wave patterns, ST segment depression or elevation, T wave inversion or flattening, and left bundle branch block are often seen as indications of silent myocardial ischaemia. The occurrence of ischaemia-

like findings on the ECG was comparable between men and women (9.0% v 9.8%). The results from this and other studies consistently show that ischaemia-like ECG changes are associated with an approximately twofold increased risk of dying of CHD.

In the British Regional Heart Study (BRHS), there was considerable overlap of questionnaire and ECG evidence of CHD and high agreement between self-report and medical record for diagnosed CHD: for example, 80 per cent of men with a GP record of angina, reported their diagnosis and 70 per cent of

men who reported an angina diagnosis had confirmation of this from the record review (National Institute of Excellence 2010; Department of Health 2010). The prevalence of diagnosed angina in 1992 in these older men was 10.1 per cent according to self-reported history and 8.9 per cent according to GP record review.

However, only half of those with a definite electrocardiogram could recall a medical diagnosis of CHD (Department of Health 2010). Even in patients with severe (grade 2) angina, 40 per cent could not recall being told that they had heart disease. Overall, only one in five of those regarded as having CHD were able to recall such a diagnosis having been made by a doctor, and these were likely to be those most severely affected.

It must be noted that there was substantial agreement between self-report and GP record of angina (Information Centre for Health & Social Care 2009). The BRHS subsequently combined two questionnaire-based definitions to define prevalence: either *current angina symptoms*, which were defined as a positive response to standard World Health Organisation (Rose) questionnaires (overall prevalence 11.1 per cent); or *history of diagnosed CHD* was defined as subject recall of ever having had a doctor's diagnosis of either angina or heart attack (overall prevalence also 11.1 per cent) (Information Centre, NHS, 2008).

Age	1994		1998		2003 Unweighted		2006 Unweighted		2003 Weighted		2006 Weighted	
	M	F	M	F	M	F	M	F	M	F	M	F
16-24	-	0.2	0.1	-	-	0.2	0.2	0.1	-	0.3	0.1	0.1
25-34	0.3	0.1	0.4	0.3	-	-	0.2	0.2	-	-	0.2	0.1
35-44	0.5	0.3	0.9	0.6	0.9	0.4	0.7	0.3	1.0	0.5	0.6	0.3
45-54	3.0	2.3	4.3	1.8	3.5	2.0	3.7	1.3	3.4	1.9	3.6	1.3
55-64	10.3	5.9	13.6	6.3	11.1	5.9	10.7	3.4	11.1	5.8	10.6	3.5
65-74	21.0	10.5	20.2	12.5	21.5	9.7	20.6	10.2	21.6	9.7	20.8	10.0
75+	22.7	15.9	23.4	18.4	26.4	18.4	28.5	19.3	26.5	18.1	28.4	19.3
All	6.0	4.1	7.1	4.6	7.4	4.5	6.2	3.0	6.4	4.1	6.5	4.0

Table 3: Percentage prevalence of IHD, by survey year, age and gender.
Source: HSfE, Information Centre

While CHD mortality has greatly declined in the last four decades, the use of age-adjusted rates to describe CHD mortality obscures the fact that the decline largely represents the postponement of CHD deaths until older age. In fact, the overall burden of CHD is increasing in parallel with the increase in life expectancy. As the burden of prevalent CHD is increasing, identifying persons with CHD, measuring its incidence and outcome and how these vary over time and across populations is essential to understanding the determinants of the trends in CHD. This in turn is crucial to define the relative contributions of risk factor reduction and therapeutic improvements, which is necessary to design effective interventions to reduce CHD.

Community surveillance is a comprehensive approach designed to track disease at the community level and is less costly and more efficient than cohort studies. In the US several community surveillance studies have reported on temporal trends in CHD prevalence e.g. the Atherosclerosis Risk in Communities study, the Minnesota Heart Survey the Olmsted County

Study and the Worcester Heart Attack Study (Information Centre for Health & Social care 2009). An analysis of US NHANES data on participants aged ≥ 40 years who attended the medical examination, the age-adjusted prevalence of angina pectoris, self-reported myocardial infarction and ECG-defined myocardial infarction were 5.8% of 9255, 6.7% of 9250 and 3.0% of 8206 participants, respectively (De Bacquer 2000). The age-adjusted prevalence of coronary heart disease defined by the presence of any of these conditions was 13.9% among men and 10.1% among women. These studies suggested that in the US medical care of clinical CHD was the main contributor to the mortality decline (Walker *et al.* 1998).

Outside the USA, the World Health Organisation's (WHO) MONICA (Multinational Monitoring of trends and determinants in cardiovascular disease) Project was established in the early 1980s to monitor trends in cardiovascular diseases and to relate these to risk factor changes. Its central goal was to explain the trends in cardiovascular disease mortality observed from the 1970s. There were 32 MONICA centres in 21 countries (Walker *et al.* 1998). In these populations, the decline in coronary disease mortality is mostly related to the decline in CHD events, thereby pointing to primary prevention as the main source. However, the study populations excluded over 65s in whom most CHD occurs.

In a survey of a rural Indian population, CHD was diagnosed based on past documentation, response to WHO - Rose questionnaire, or changes in ECG. The prevalence of CHD (clinical + ECG criteria) was 3.4% in males and 3.7%

in females. According to ECG criteria only, it was 2.8% in males and 3.3% in females and according to Q-waves only, it was 1.6% in males and 0.9% in females (Shaper *et al.* 1984). In a Finnish population survey, *Ahto et al* found the prevalence of angina symptoms was 9.1% among men and 4.9% among women aged 64-97 (Shaper *et al.* 1984). Ischaemic ECG findings were common: 32.9% of men and 39.3% of women had such changes. An international systematic review and meta-analysis found that angina prevalence varied widely across populations, from 0.73% to 14.4% (population weighted mean 6.7%) in women and from 0.76% to 15.1% (population weighted mean 5.7%) in men (Lampe *et al.* 2001).

In the UK, Carroll *et al* used GP records in London and found a prevalence of 8 per cent of men and 5 per cent of women over 44 years of age - although this may be lower than the true national average (Alexander *et al.* 2003). There was a history of myocardial infarction in 30 per cent of men and 22 per cent of women with CHD. Lampe and colleagues examined trends in the prevalence of CHD in men participating in the BRHS. The authors demonstrated a decrease in the prevalence of current angina symptoms: the age adjusted annual percentage change in odds was -1.8%. However, there was no evidence of a trend in the prevalence of history of diagnosed CHD (Information centre. NHS 2008).

A study by Davies *et al* examined trends in CHD incidence prevalence, and mortality in the UK between 1996 and 2005, using the THIN GP database (a total of 5 million patients). The results indicate that, while CHD mortality

declined, CHD incidence decreased less than mortality, resulting in an increase in CHD prevalence (Shaper *et al.* 1984). From 1996 to 2005, age-standardised incidence of CHD decreased by 2.2% in men and 2.3% in women per year (average percentage change). Age-standardised, all-cause mortality among those with CHD, decreased by 4.5% in men and 3.4% in women per year (average percentage change). Age-standardised prevalence increased by 1.3% in men and 1.7% in women per year (average percentage change). The decline in incidence had some impact on limiting the increase in prevalence, but its effect was offset by the increase in prevalence occurring as a result of improved survival among people with CHD. Although patients with nitrate prescriptions were also included, this study relied mainly on CHD diagnostic codes which may underestimate actual prevalence.

5.2.1. Local area prevalence estimation framework

The APHO used regression models to estimate prevalence and used the HSfE as its main source of data. This approach does not take account of geographic context, exemplified by interactions between demographic risk factors and geographic locations, or by effects of local geographic variables e.g. cultural norms. For example, assumptions do not hold for minority ethnic populations e.g. South Indian women.

The proposed local model incorporates both national survey information (HSfE) on patient risk factors and local geographic data (Moran *et al.* 2010). This approach is applied to drive micro area prevalence estimates, specifically estimates of CHD for the locality of Brent. The model incorporates

prevalence differentials by age, gender and ethnicity from the national survey. Whilst national model uses random effect that allows for spatial correlations, local area information takes account of health inequalities relating to poverty and urbanity.

Model Construction

A regression model for prevalence includes person level attributes (age, gender, ethnicity, etc.) that are known to have significant CHD risk gradients.

Data source

a. Populations

The CHD prevalence model used ONS 2009 mid-year LA population estimates by ethnic group, age and sex. Five ethnic groups were used: white, black, Asian, mixed and other. In order to calculate estimate prevalence of CHD in the future, population projections were incorporated into the model. ONS has not published population projections by ethnic group, so the 2009 (LA) distribution of ethnic groups was used to generate population estimates to 2020.

Local population data were data to this mix. This provided a “more realistic” scenario for local estimates.

b. Ethnicity

The proportion of practice population in ethnic groups was supplied by the Care Quality Commission (CQC). HES is the only routine dataset which includes GP practice and patient ethnicity and has high levels of completion and data quality. It is assumed that the hospital admissions (excluding

maternity and mental health) reflect the true ethnic population of the practice and there is no systematic bias. The proportions by ethnic group for each practice were calculated by dividing admissions within each ethnic category by the total admissions for the practice. The same ethnic distribution is applied across all age bands as there are insufficient hospital admissions to robustly calculate the distribution of ethnic groups by age and sex for practices.

c. Deprivation

Deprivation scores are taken from IMD 2008. Deprivation scores for PCTs were calculated by taking a population weighted average of the scores for each Medium Super Output Area (MSOA) which in turn was calculated by taking a weighted average of the IMD2004 scores of each Lower Super Output Area (LSOA) within the PCT. Five deprivation categories are used in the model. Note that these categories are based on quintiles of IMD score at LSOA level (Table 5). When the cut-offs are applied to larger geographies (LA or PCT) there is not an even distribution across all categories.

Rank	IMD		Number	%
	HSfE 2006	HSfE 2007		
1	0.59 -8.34	.55 – 9.02	3,803	17.87
2	8.35 – 13.71	9,03 -14.14	3,573	16.79
3	13.72 – 21.15	14.15 – 21.17	3,788	17.80
4	21.16 – 34.20	21.18 – 33.52	4,551	21.38
5	34.21 – 86.36	33.53 – 85.69	5,571	26.17
		Total	21,286	100

Table 4 : Index of multiple deprivation banding

d. Smoking status

National (England) proportions of smokers, ex-smokers and current smokers by age and sex are taken from HSfE (2007-2009 pooled). These proportions were then adjusted for each LA/PCT using the synthetic estimates of smoking prevalence for 2006- 2009, using the following algorithm:

$$S_{asl} = S_{asn} \times \frac{S_l}{S_n}$$

Local prevalence of smokers to (age, sex, category) = national prevalence of smoking in (age, sex, category) * local overall smoking prevalence / national overall smoking prevalence . Local ex-smokers in (age, sex) category not adjusted.

$$N_{asl} = 1 - E_{asl} - S_{asl}$$

where

S= proportion of population who are smokers
E= proportion of population who are ex-smokers
N= proportion of population who have never smoked
L= local
N= national
as= by age and sex

This approach assumes that the proportion of ex-smokers in each age-sex category is fixed and the number of never-smokers increases as the number of smokers decreases. Regional analysis of the relationship between prevalence of smokers and ex-smokers in the HSfE shows no systematic relationship and therefore it was decided that the ex-smoking rate should not be locally adjusted (see figures 15).

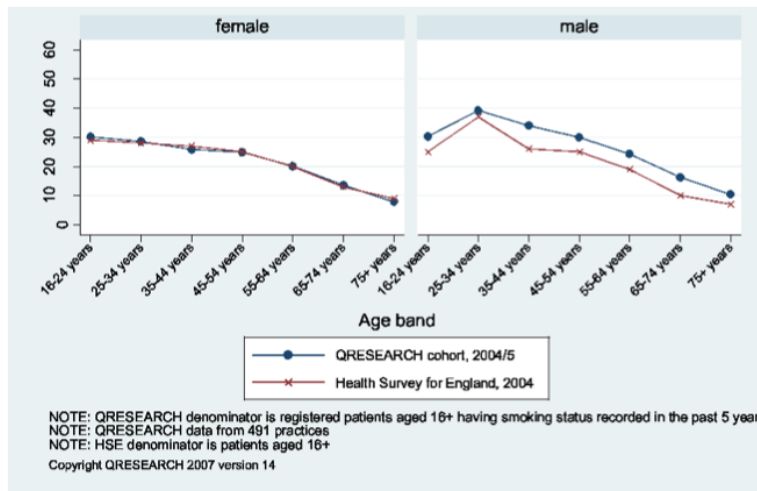


Figure 15: Prevalence of smoking in the UK (gender)

The same smoking prevalence rates are applied across all ethnic categories. This was partly due to the fact that no data (by ethnicity) existed at the time of the study. Also future changes in smoking prevalence are not taken into account in the CHD prevalence projections. This is because of the uncertainty associated with predictions of smoking prevalence and the lag time between smoking cessation and improved health. Even if there was a rapid drop in the number of smokers over the next few years, any associated decrease in CHD would not be seen for many years.

Operationally, three issues had to be considered. First, the modelling predicts the number of people with identified CHD within each population, taking account only of the demographic distribution of the population. The prevalence of patient-reported doctor-diagnosed CHD in each age/sex stratum is based on national data from the HSfE. It allows first for the differential risk of each

CHD symptom for the various ethnic groups as against the white as reference category. Data for educational attainment could not be used as this was not available. Brent has a significant number of immigrants and displaced population cohort (due to conflicts elsewhere) and this information was unavailable and probably not reliable.

The second takes account of inequalities and includes geographic effects but without any interactions between area and local attributes. This focused on the county level variables such as poverty index. Many geographic influences may be unobserved (e.g. various environmental and health behavioural influences) and these are represented in the second models by random effects. It is sensible to allow unobserved influences to be spatially correlated to reflect smoothly varying risk factors in space.

The third approach allows area-person interactions, in that random effects are taken to be ethnic specific. Differentiation of area effects by ethnicity reflects epidemiological evidence such as that noted by Casper et al (2006) that CHD mortality and prevalence disparities between ethnic groups vary by place of residence.

The modelling and estimation of the effects of interest was carried out using STATA. The initial output consisted of two tables: one with the estimated regression coefficients, corresponding p values and 95% confidence intervals, and another with the estimated odds ratios ($\exp(b)$), which in the table appear as relative risk ratios (RRRs) and 95% confidence intervals. A positive sign of

the estimated coefficient is associated with an increase in the odds of the outcome had angina or heart attack, and a negative sign is associated with a decrease in the odds. Since $\text{Prob}(A) = \text{Odds}(A) / 1 + \text{Odds}(A)$, for uncommon outcomes such as CHD, RRR can be assumed to be the same as the odds ratio (OR). (See explanation in methodology section page 99).

Assumptions

It is assumed that the:

- Proportion of smokers, ex-smokers and never-smokers is the same across ethnic groups
- Proportion of ex-smokers in each age-sex group is the same in all areas
- Smoking prevalence rates from the model-based estimates of lifestyle behaviours are reliable
- Prevalence of CHD in those aged under 40 is negligible
- Due to lack of data, it was not possible to treat ex-regular-smokers and ex-occasional smokers separately. Ex/occasional smokers are treated as non-smokers

In summary, this method is straight-forward and extremely cost-effective relative to the implementation of a population survey, but it does assume that the local prevalence of a condition or behaviour is entirely dependent upon the socio-demographic composition of the area. Models which combine individual and area level effects represent a significant advance, but it has proved

difficult to quantify the precision of small area estimates without simplifying assumptions.

5.2.2. Model construction: Validation process

The methodology supporting model-based estimation for large population has been validated and is well established, and it is not the purpose of this study to re-examine it. Rather, it aims to validate individual sets of ward-level model-based estimates of the prevalence of cardiovascular risk factors.

These estimates should be valid and accurate provided that:

- (a) The risk factor in question is strongly associated with individual level and area level covariates,
- (b) The developed model is well fitted.

If the first criterion is satisfied then it is possible to create a model that explains a large proportion of the variance in the prevalence of risk factors. If the second criterion is satisfied then the developed model accurately describes the relationship between uptake of the risk factor and the individual and area-level covariates.

The validity assessments focus were on:

- a. Internal
 - b. External
- ◆ Predictive validity –
1. Is there any association with QOF data?
 2. Does it converge with Case findings?
 3. Face validity

The extent to which the models supporting the estimates are well fitted—that is, they adequately describe the relationship between the health behaviour and individual level and area level covariates is assumed as it operates on the same principle of the regional model.

a) Internal validation

The prediction of the model was assessed in two ways:

- by deriving predicted probabilities of the CHD outcome in Stata from the models and comparing these to the observed cases
- by generating a receiver operating characteristics (ROC) curve using the predicted probabilities of the CHD outcome compared to the observed cases

One method of assessing performance is to use regression model to predict response for each subject. These predictions are called fitted values. The difference between the fitted and the observed values are called residuals. Ideally the best prediction should result from utilising the most risk factor information in the regression model. Ideally the best prediction should result from utilising the best locally available information. However only a limited range of HSfE variable data is either available or can be estimated at the primary care organisation (PCO) or local authority (LA) level, so there is no purpose in including other variables (see Table 6). The study validated the local model by comparing it, in terms of prediction, to a model including all available and significant HSfE variables. In addition, however, the amount of missing data affects the prediction of a model. The Stata10 software package was used for analysis. All variables were re-coded to drop negative values for

estimation purposes. The methodology applied was multinomial logistics regression with the “cluster” adjustment option for local variables. For analysis of two categories (where needed), the multinomial regression was reduced to binomial logistic regression. The prevalence in each age group, gender, ethnic group, area of residence and level of deprivation were derived from the odds, using the formula: $\text{prevalence} = \text{odds} / (1 + \text{odds})$.

In the complete HSfE variables the largest proportion of missing data occurred in those variables related to drug treatment for high blood pressure. Treatment with ACE inhibitors, beta blockers and calcium blockers are significant. However these may also be used to treat established CHD, so the association may be unrelated to hypertension. In addition, the HSfE relies upon patient recall for drug treatment. Not unexpectedly, much of the data for these variables is missing. Systolic and diastolic BP were added as ordinal variables to the model, but this resulted in major changes to ORs for other variables. This may simply be related to model instability because of the large numbers of variables included. The addition of a single variable for hypertension, either treated or untreated, will be explored as a further later step in model development.

The prevalence figures generated by the models are estimates of the expected prevalence of disease. Discrepancies between modelled estimates at practice level and other sources of data such as QOF disease registers may be due to local variations not captured by the model and cannot be solely attributed to weaknesses in QOF data. For practices with populations that

significantly differ from a ‘typical’ population (e.g. large black or ethnic minority) were excluded. Hence the model COMP 1 included the “complete” list of variables, including BP drugs; model COMP 2 included the “complete” list of variables, but excluded BP drugs (table 5). The model included impact of age, Index of Multiple Deprivation (IMD) and ethnicity on outcomes (table 7 and 8). The predicted prevalence by BM category is shown in table 7.

COMP 1 = “Complete” variables with BP drugs

COMP 2 = “Complete” variables without BP drugs

LOCAL = only using locally available data

* only a selection of variables is shown in table

Name	Label	COMP 1	COMP 2	LOCAL
ihdis	had IHD (angina or heart attack)	X	X	X
aceinh	ace inhibitors (blood pressure)	X		
addnum	address number	N/A	N/A	N/A
adtot30	adults: total days/4 weeks active 30 mins + moderate +	X	X	
age	age last birthday	X	X	X
area	sample point	N/A	N/A	N/A
beta	beta blockers (blood pressure)	X		
bmival	valid body mass index	X	X	X
calciumb	calcium blockers (blood pressure)	X		
cholest	total cholesterol result (blood data)	X	X	
cigst1	cigarette smoking status - never/ex-reg/ex-occ/current	X	X	X
cluster	stratification level	N/A	N/A	N/A
diabete2	doctor diagnosed diabetes (excluding pregnant)	X	X	
diur	diuretics (blood pressure)	X		
ethnici	ethnic group	X	X	
famcvd	family history of cvd	X	X	
fatvala	fat score	X	X	
fldlchol	LDL cholesterol result (fasting blood data)	X	X	
ftriegl	triglycerides result (fasting)	X	X	

Table 5: Variables included in merged dataset

Risk factor	RR	S-Error	z	P>z	95% CI
Age 25-34	1				
Age 35-44	6.68	2.98	4.41	0	2.91
Age 45-54	19.51	8.21	7.06	0	8.55
Age 55-64	65.71	27.23	11.65	0	29.16
Age 65+	154.67	65.16	11.65	0	68.87
IMD 0.59-8.35	1				
IMD 8.35-13.72	1.27	0.14	1.75	.08	0.97
IMD 13.73-21.16	1.35	0.15	2.63	.009	1.02
IMD 21.17-86.36	1.85	0.25	6.49	0	2.12
White	1				
Mixed	1.23	0.85	0.35	0.72	0.39
Black	0.76	0.16	-1.23	0.21	0.49
Asian	1.51	0.24	2.56	0.01	1.12
Other	0.16	0.17	-1.76	0.07	0.02

Table 6: Odds Ratios for LOCAL (public health datasets) , model with only locally available variables Including IMD – Index of Multiple Deprivation

	Age band						Total
	16-24	25-34	35-44	45-54	55-64	65+	
Don't know	0	0	0	0	0	3	3
Yes	2	6	43	97	262	694	1,104
No	2500	3855	4409	3379	2792	20121	35156
Total	2502	3861	4452	3476	3054	20818	36363

Table 7: Respondents reporting doctor diagnosed CHD by age band

	BMI <18.5 1	BMI >18.50 & BMI<25	BMI >25 & BMI <30	BMI >30 & BMI <40	BMI >40	Total
CHD	12	187	386	268	251	1,104
No	306	6,693	6,502	3,483	3,194	20,178
Total	318	6,880	6,888	3,751	3,445	21,282

Table 8: CHD prevalence by BMI category

Table 6 shows the frequency of the CHD outcome by age group in the merged dataset. The regression model for risk factors for CHD in the “local” prevalence model is shown in table 9. As expected, ORs increase strikingly with increasing age in all models. In the prevalence predictions, using coefficients (not shown in these tables), this results in age-related increases in prevalence which closely match the crude overall prevalence. Surprisingly the only significant comparison for smoking is for category 3 “used to smoke regularly” i.e. this group is more likely to report CHD compared to the group “never smoked cigarettes at all”. There is a significant comparison for male sex. ORs, p values and confidence intervals are generally similar to the “Complete” models. Unfortunately, however, local synthetic estimates of smoking prevalence do not include categories for occasional/regular smokers.

Risk factor	RRR	Std E	z	P>z	95% CI.
Age 25-34	1.00				
Age 35-44	7.13	6.89	2.03	0.04	1.07
Age 45-54	18.44	17.4	3.08	0.01	2.89
Age 55-64	50.09	46.7	4.2	0	8.05
Age 65-74	121.1	112	5.17	0	19.65
Female sex	1.00				
Male sex	2.25	0.32	5.74	0	1.71
Never Smoker	1.00				
Used to smoke occasionally	1.03	0.32	0.13	0.9	0.56
Used to smoke regularly	1.51	0.23	2.63	0.01	1.11
Current Smoker	1.11	0.23	0.52	0.61	0.74
White ethnic group	1.00				
Black/Black British e	0.83	0.35	0.43	0.66	0.36
Asian/Asian British ethnic	1.51	0.43	1.57	0.11	0.89
BMI <18.51	1.00				
BMI >18.50 & BMI <25	0.53	0.56	0.59	0.55	0.06
BMI >25 & BMI <30	0.89	0.93	0.11	0.91	0.11
BMI >30 & BMI <40	1.02	1.08	0.02	0.98	0.12
BMI >40	0.69	0.73	0.35	0.72	0.08
Total cholestrol:HDL ratio	0.74	0.04	4.44	0	0.65
Diabetes; yes	0.68	0.14	-1.8	0.07	0.45
Family History of CVC; no	0.62	0.11	-0.87	0.01	0.45

Table 9: Odds ratios for CHD model

Using AUROC (Area Under Receiver Operating Characteristics Curve)

Receiver- operating characteristic (ROC) analysis was originally developed during World War II to analyse classification accuracy in differentiating signal from noise in radar detection. Recently, the methodology has been adapted to several clinical areas heavily dependent on screening and diagnostic tests, in particular, laboratory testing, epidemiology, radiology, and bioinformatics.

ROC analysis is a useful tool for evaluating the performance of diagnostic tests and more generally for evaluating the accuracy of a statistical model (e.g. logistic regression, linear discriminant analysis) that classifies subjects into one of two categories, diseased or non--diseased. Its function as a simple graphical tool for displaying the accuracy of a medical diagnostic test is one of the most well- known applications of ROC curve analysis.

An ROC curve is a plot of sensitivity on the y axis against specificity on the x axis for varying values of the threshold, t . The 45° diagonal line connecting (0,0) to (1,1) is the ROC curve corresponding to random chance. The ROC curve for the gold standard is the line connecting (0,0) to (0,1) and (0,1) to (1,1). Generally, ROC curves lie between these two extremes. The area under the ROC curve is a summary measure that essentially averages diagnostic accuracy across the spectrum of test values. The area under the curve (AUC) is an overall summary of diagnostic accuracy. AUC equals 0.5 when the ROC curve corresponds to random chance and 1.0 for perfect accuracy. On rare occasions, the estimated AUC is <0.5 , indicating that the test does worse than chance (figure 16).

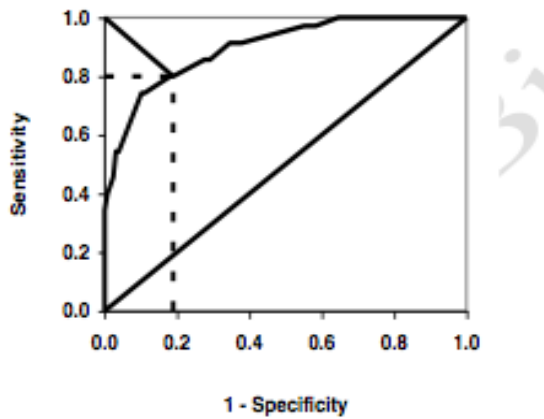


Figure 16: AUROC curve

AUROC's for the model tested above were estimated using Stata10. These are shown in the chart below. If both sensitivity and specificity area of importance in a CHD model, the optimal threshold of t would be 0.75, where sensitivity and specificity equal 0.77. The local model, with an AUROC of 0.8071 exceeds this level, although the complete model has even better performance, with an AUROC of 0.8304.

b) External validation

a) Predictive validity

Because the intended use of this model is primarily for estimation purposes, rather than testing a particular theory, the study sought to focus on the predictive accuracy of the model. This was carried out by testing number of predicted cases against observed events within GP case loads.

Testing the model

The model was applied locally to test its applicability. Based on Health Survey for England data and local demographic distribution, SMR for CHD in the London Borough of Brent is estimated at 112. The estimation increases the predicted prevalence of each Brent's practices by 112%.

Using ..

$SMR (CHD) = (2.402 \times UV67) + 25.24$, where 2.402 is the gradient, UV67 is the local community (Kilburn) deprivation index and 25.24 the intercept of the regression line.

The multiplication factor (mf) is identified by:

$$mf = ((2.402 \times 40) + 25.24)/100 \quad (**40 \text{ is local deprivation index}) \gg 1.21$$

To get the local prevalence rate for GP Practice X, the local CHD (SMR) is divided by national CHD (SMR) and multiplied by the mf ie 1.21

$$\frac{LocEst}{NatEst} \times MF$$

where LocEst (local estimate) and NatEst (national estimate)

Thus

$$\frac{121.32}{112} \times 1.21 = 1.31 ,$$

where 121.32 (local predicted SMR); 112 (national SMR (see above) and 1.21 is the mf

This shows that Practice X (in Kilburn locality) has a higher prevalence than Brent (as a borough). This was calculated for each GP practice in the Kilburn area and for the other four localities in the borough localities (table 10). A comparative prevalence of CHD for Brent localities calculated through this model giving a predicted number of cases (table 11)

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Kilburn practices	Population	Prevalence probability	Predicted numbers
E84696	1844	0.0195	36
E84667	2398	0.0150	47
E84035	2568	0.0140	50
E84705	2838	0.0127	56
E83654	2914	0.0124	57
E84056	3069	0.0117	60
E84077	3079	0.0117	60
E84702	3124	0.0115	61
E84080	7155	0.0050	140
E84042	7409	0.0049	145
E84006	13521	0.0027	265
E84025	14367	0.0025	282
E84023	5466	0.0066	107
E84674	6020	0.0060	118
E84012	6811	0.0053	134

Table 10: CHD prevalence estimation - Kilburn location

	Nos. practices	Population	Estimated prevalence	Predicted cases
Kilburn	15	82583	0.023	1618
Harness	10	32014	0.048	1537
Wembley	15	70153	0.003	2315
Kingsbury	15	56458	0.035	1491
Willesden	10	53361	0.0196	800

Table 11: Prevalence estimates for CHD cases for all the localities

To test the association between the number of registered cases and the predicted numbers by the model, a series of chi-square tests (χ^2) (table 12). Results show that there is a significant association between predicted and actual cases.

The model does not necessarily represent the actual number of people who should be diagnosed with CHD for each practice; it is only a guide. The characteristics of each practice differ and needs to be considered. Furthermore, it does not include undiagnosed cases of CHD. In Brent, it is anticipated that there could be relatively large levels of undiagnosed disease compared to more affluent areas where people are more likely to present to their GP with symptoms.

	CHD (cases)		Chi-sq (χ^2)	Sig.
	Registered	Predicted		
Kilburn	2082	1259	4.87	P<0.05
Harness	1914	1567	3.78	P<0.05
Wembley	1751	2315	3.81	P<0.05
Kingsbury	1748	1459	3.35	P<0.05
Willesden	1388	2017	4.04	P<0.05

Table 12: Chi-square tests to demonstrate the association between the various localities (within and between).

A CHD funnel plot “observed vs expected” prevalence by GP practices in Brent (2009-10) carried out using QOF datasets for Brent is estimated to be 3.1% in 2009 (n=295,678) gave further support to the findings. This is higher than the prevalence reported by Brent PCT (2.2%) (figure 17). However, this was not unexpected due to the data quality issues and the quirks of QOF. It does indicate that NHS Brent is under-reporting its CHD prevalence.

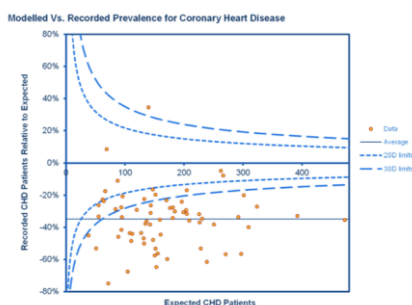


Figure 17: Funnel chart for CHD indicating an under reporting of CHD cases for 2010-11

5.3. Estimating prevalence of co-morbidities

This phase is concerned with estimating the prevalence of CHD within the SMI patient population. This is undertaken (a) calculating the prevalence of the concurrent disease and (b) using the Bayesian method to determine the probability of having this condition in the SMI group. The determination of the prevalence rate of CHD in the local population has been carried out in the precedent phase. The SMI prevalence rate is taken from the QOF's dataset at practice level and as expected the level is uneven across the borough. The

model assumes that the SMI population via QOF represents the “true level” of SMI within the locality. At the time of the study, local estimate of mental illness is not sufficiently robust to make a more accurate estimation.

The extrapolation algorithm has been explained in the methodology section. Using this probability equation, the prevalence rate of CHD is extrapolated from the SMI group using the following formulation:

$$P(CHD | SMI) = P(CHD)P(SMI) / (PSMI | CHD)$$

Where (see figure 18 for a schematic view)

$P(CHD)$ is the probability of CHD in the local population

$P(SMI)$ is the probability of SMI in the population (using QOFs)

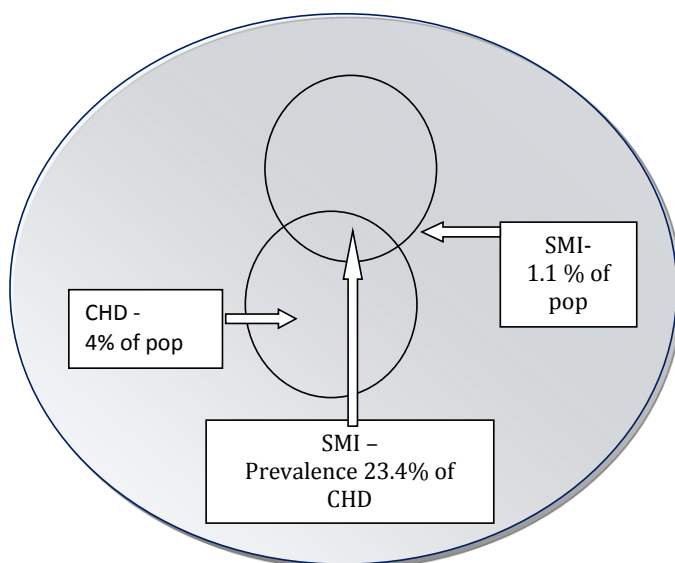


Figure 18: Schema to show the relative prevalence (%) of SMI with CHD within the population

Worked example:**Testing the model**

In order to test the predictive value of the model five localities in Brent agreed to participate in this project. Five case studies below explain how the disease prevalence models could be used by health commissioners to factor local variances.

Example**a. Calculation for the presence of CHD in the SMI population within locality**

- Establish probability of CHD prevalence (based on the estimate model methodology).
- Use SMI prevalence rate as determined by QOF.
- Use the probability estimate (based on systematic reviews) to establish the likelihood of CHD with SMI.
- Calculate the likelihood with SMI to have CHD.
- Test against case finding. In order to find this statistic, the GPs had undertaken a complex search of the system to match the two conditions.

b. Estimation of CHD in SMI population

The prevalence rate of the SMI was based on QOF data (as reported by GPs). It should be noted that due to technical difficulties, it is not possible (to-date) to make regional estimation of the mentally ill. Since 2008, QOF have been used as a proxy measure to determine prevalence. Given that QOF is an incentive scheme and that many patients do not regularly go for checks up, this source of data is at best unreliable. Using Bayesian probability, we have

$$P(CHD | SMI) = P(CHD)P(SMI) / (PSMI | CHD)$$

Worked example:

$$P(CHD | SMI) = P(CHD)P(SMI) / (PSMI | CHD)$$

$$P(CHD | SMI) = (0.04) * (0.011) / (0.23) = 0.00191$$

**** 0.04 (the probability of having CHD) ; 0.11 (the probability of having SMI) – (refer to schematic illustration above)**

Kilburn locality

The formula was applied to the GP practices within the Kilburn locality. Using Bayesian probability and the predicted prevalence of CHD (adjusted value), an estimation of patients having SMI and CHD was calculated (table 13). Figure 19 graphically demonstrate the difference between the number of cases derived from the national prediction against the local estimates numbers.

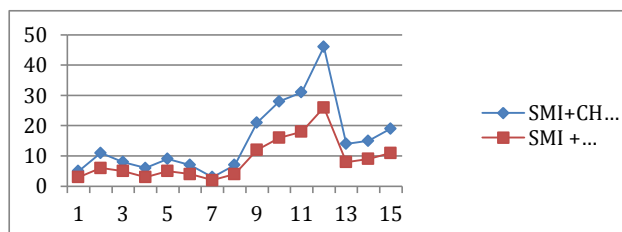


Figure 19: Graph showing SMI with CHD (expected vs recorded) within Kilburn locality.

Kilburn								
PracticesCode	Mental Health		CHD			SMI + CHD		
	QOF_MH_Prevalence	Register_Count	CHD Register_Count	CHD Observed(%)	CHD Predicted	CHD Predicted (%)	Probability estimation using Bayes method	Expected number
E84696	1844	26	30	2.2	36	4.5	0.24	7
E84667	2398	34	40	2.8	47	4	0.15	26
E84035	2568	36	42	2.9	50	5	0.12	12
E84705	2838	40	47	2.1	56	4.5	0.25	3
E83654	2914	41	48	2.2	57	4.8	0.15	13
E84056	3069	43	51	2.2	60	3.9	0.17	4
E84077	3079	43	51	3.2	60	4.9	0.24	4
E84702	3124	44	52	2.2	61	6.1	0.36	7
E84080	7155	100	118	2.4	140	6.3	0.22	6
E84042	7409	104	122	2.2	145	4.7	0.36	29
E84006	13521	189	223	2.8	265	6.4	0.3	23
E84025	14367	201	237	2.2	282	6.3	0.36	31
E84023	5466	77	90	3	107	5.5	0.22	82
E84674	6020	84	99	2.2	118	4.9	0.36	12
E84012	6811	95	112	2.5	134	6.1	0.3	24

Table 13: SMI with CHD co-morbidity – Kilburn locality (Actual vs Expected)

Modelled CHD prevalence and prevalence probability estimate of its co-morbidity with SMI are shown on table 14. Results show a significant difference between observed and case notes findings (table 15). For each locality the modelled prevalence estimates were higher than the register prevalence, which would be expected if the model reflected the level of diagnosed plus undiagnosed CHD in the community .

Locality	Population	Mental Health		CHD		SMI + CHD			
		QOF MH Prevalence	Register Count	CHD Register Count	CHD Predicted	CHD Predicted (%)	Probability estimation	Expected number	Observed numbers
Harness	77106	0.98	684	1670	2772	5.13	0.22	216	130
Wembley	87267	0.91	562	1675	2014	5.65	0.23	175	120
Willesden	44835	0.93	426	1388	2017	5.84	0.24	127	104
Kingsbury	70153	0.88	661	1748	1967	5.59	0.206	162	113

Table 14 : Number of expected cases and observed cases of SMI with CHD.

t-test	Kilburn	Harness	Wembley	Willesden	Kingsbury
Mean	116.684	106.7	84.04	104.76	76.733
Variance	1551.852	1093.66	687.89	1332.65	852.67
df	9	10	14	8	15
t-stat	2.906	-2.95	3.01	2.78	-4.89
p(T<t)2 tail	0.017	0.016	0.009	0.008	0.0004

Table 15: T-tests show significance difference between expected and recorded cases across all localities.

5.4. Summary

There are significant differences between the numbers of patients with a diagnosis of CHD as recorded by their GP (based on the Quality and Outcomes Framework (QOF) register and those reported by the modelled data. One possible explanation is that the GP registers are not always up-to-date (due to boundary movements) and patients not informing their practices. Such issues tend to inflate practice patient numbers which affect the denominator line.

However, this does not tell us total prevalence, only how many have been diagnosed. If conditions such as CHD are undiagnosed, and therefore unmanaged, outcomes are likely to be poor (for example, premature death or disability due to heart attack). However, by comparing the QOF registers with the modelled prevalence it is possible to estimate the level of unmet need, in this case, the number of patients thought to have CHD who have not been diagnosed.

6. COPD

6.1. Introduction

Chronic obstructive pulmonary disease (COPD) is a chronic condition characterised by progressive airflow obstruction, which is not completely reversible. It has been called the "silent epidemic" and is the fourth leading cause of death in the general population (Information Centre. GP Extraction Service. 2010). This disease accounts for nearly 30,000 deaths each year in the United Kingdom (UK), corresponding to 5.7 percent of adult male and 4 percent of adult female deaths, including a significant number of premature deaths (Billings *et al.* 2006). A meta-analysis of studies of the general population published between 1990 and 2004 revealed geographical disparities in the pattern of the disease due to socio-economic variables (Dr Foster Intelligence 2010). The prevalence of COPD was estimated to be 7.6% (95% CI 6–9.2%) independent of the defined diagnostic criteria. On the basis of 38 studies, the prevalence of chronic bronchitis was estimated to be 6.4% (95% CI 5.3–7.7%). The prevalence of emphysema (via chest radiograph) was estimated to be 1.8% (95% CI 1.3–2.6%) on the basis of eight studies.

In addition, 1.4% of the population consults their general practitioners (GPs) for COPD each year. It accounts for 2% of hospital admissions and over 3 percent of bed-days in adults, costing the NHS £800 million, and leading to 24 million working days lost each year (Billings *et al.* 2006; Dr Foster Intelligence 2010).

Risks factors of public health importance include; air pollution, socio-economic deprivation, occupational exposures and possibly ethnicity. Stopping smoking prevents the development of COPD, or slows its progress and reduces the risk of hospital admissions. Not surprisingly, smoking is the strongest independent predictor of COPD, with smokers having over eight times the odds of having COPD than the non-smokers (Soljak and Flowers 2008; Dr Foster Intelligence 2010).

This "epidemic" seems to be even more silent among individuals with serious mental illness who are at particular risk of developing this condition from smoking, which is a modifiable risk factor. A team at Queen Mary university, London (Congdon 2001) reported that patients with serious mental illness had over three times the odds of having chronic bronchitis and over five times the odds of having emphysema than a matched group of national comparison subjects (Goddard 2005). In their study, they reported prevalence of COPD among those with serious mental illness in the order of 22.6% (Congdon 2006). Consistent with previous research, they found the prevalence of current smoking to be 60.5%, which is more than twice the average (27.4%) and nationally (22.6%).

COPD is measured by degree of airflow obstruction to the lungs. It is measured by spirometric testing, in which the patient performs a forced expiration into an airflow measurement device called a spirometer, and the volume of the air they exhale is measured over time until they can exhale no more. According to the current Global initiative for Chronic Obstructive Lung

Disease (GOLD) guidelines, COPD is diagnosed when the ratio of the air exhaled in one second (FEV1) to the total exhaled volume of air (FVC) is less than 0.7, indicating the presence of obstruction (Horgan *et al.* 2010). The severity of the disease is determined by the variance between the FEV1 measured and that predicted by age. Mild COPD is diagnosed when FEV1 is greater than or equal to 80% of the predicted value. Very severe COPD is diagnosed when FEV1 is less than 30% of the predicted value. Conversely, a person's lung age is defined as the age of the average healthy individual performing a similar spirometric test; someone with a low FEV1 compared with what is predicted for his or her actual age would have a high lung age.

6.2. National prevalence model

Developing the model

The national prevalence model used the HSfE as a representative population-based annual survey, which in 2001 included the assessment of respiratory function using spirometry, as well as comprehensive data on risk factors. The variables included in the model, based on their association with COPD in logistic regression analysis, were age group, gender, ethnicity, smoking prevalence, area of residence (rural, suburban or urban) and area-based deprivation score (McFadden *et al.* 2009). The 2001 data refers to 5269 men (98%) and 6133 women (95%) over the age of 15 years tested using spirometry (Morris *et al.* 2005). Additional data for multivariate analysis were available for 94.3% of the sample. COPD was defined using the British Thoracic Society (BTS) criteria: forced expiratory volume in 1s (FEV1) divided by forced vital capacity (FVC) under 0.70, and FEV1 <80% of predicted using

reference values from the HSfE. The approach used a complex synthetic estimation technique using logistic regression.

The baseline odds of COPD in non-smokers was obtained directly from the data set (Graubard *et al.* 2007). The strength of association between each explanatory variable and COPD caseness was used to calculate the relative odds, which were applied to the baseline odds to derive the prevalence estimates for subgroups of risk factors. The main results were expressed as expected/predicted prevalence of COPD for population subgroups. The model was applied to obtain the total COPD prevalence for 354 local authorities in England. The prevalence in each age group, ethnic group, area of residence and level of deprivation and smoking status category were derived from the odds, using the formula:

$$\text{prevalence} = \text{odds}/(1+\text{odds}).$$

The APHO accepts that this current model is rather crude and that prevalence model based on a more comprehensive regression model using more sensitive local data would be more discriminative.

Model Validity

The model validity was tested by comparing COPD expected prevalence results to an alternative model, based on a survey of prevalence studies. An additional test involved testing the population of Belfast, Northern Ireland population and compared the results with those from a population survey of

the same population. The results were slightly lower but within the 95% CI of those estimated from the survey (4.9% total prevalence in the 40-69 year olds compared with 6.1% (95% CIs = 4.5-7.7) in the survey. The prevalence estimates for the whole of England were similar to the Health Needs Assessment Report and to other studies that used the BTS definition of COPD. The significant correlation between expected prevalence and diagnosed COPD and COPD mortality gives reassurance of validity.

Findings

Table 16 shows the prevalence of COPD by age and gender in England. The overall prevalence in the population over 15 years of age was 3.1% (3.9% in men and 2.4% in women). For those over 45 years old, the estimated prevalence was 5.3% (6.8% and 3.9% in men and women respectively). This corresponds to over 1.3 million people in England with COPD, of whom nearly 800,000 or 60% are men. The odds ratio calculated for gender among current and ex-smokers show a wide difference between men and women smoking history (table 17). The assumption that ethnicity is not associated with being a case of COPD, i.e. that all population has the same risk as the white population did not change the total national prevalence estimates considerably (1,297 thousand in 15 year-olds and over and 1,065 thousand over 45s under this assumption). When we considered the risk of COPD in under 45s as equal to the average baseline risk in this age group (in non-smokers), the total number of cases estimated was reduced by 60,800, resulting in an overall prevalence of 1.25 million or 3% (3.8% in men and 2.3% in women) (table 18).

Age group	Men number (%)	Women number (%)	Both sexes number (%)
15 -44	137,530 (1.30)	93,450 (0.89)	230,980 (1.10)
45-54	75,720 (2.38)	64,840 (2.00)	140,560 (2.19)
55-64	198,400 (6.90)	122,440 (4.11)	320,840 (5.48)
65-74	199,840 (10.03)	105,704 (4.81)	305,580 (7.29)
75+	172,700 (11.65)	132,400 (5.55)	305,100 (7.89)
Total	784,190 (3.89)	518,870 (2.41)	1,303,060 (3.15)

Table 16: Number and proportion of people estimated to have COPD by age group and gender in England (estimates for 2009).
Values in brackets correspond to mean values in extreme quintiles of deprivation score or approximately the 10th and 90th percentiles of the prevalence distribution

Variables	Odds ratio (95%)	
	Men	Women
Smoking Status		
Never	1	1
Former	3.63 (2.54 – 5.21)	1.70 (1.07 – 2.64)
Current	3.81 (2.64- 5.52)	2.3 (2.23 – 5.14)

Table 17: Risk factors for COPD and selection of variables for COPD model

Age	(p<0.01)	
<35	0.56 (0.13 – 1.65)	0.32 (0.11 – 1.2)
35-54	2.05 (1.33 – 4.50)	1.65 (1.3 – 3.6)
55-64	6.91 (4.02-11.89)	1.9 (3.2 – 7.9)
65+	10.40 (6.08 – 17.80)	8.68 (5.2 – 14.88)
Regional locality		
Urban	1	1
Suburban	0.70 (0.50 – 0.97)	0.45 (0.3 – 0.87)
Rural	0.58 (0.39 – 0.86)	0.34 (0.12 – 0.86)

Table 18: Risk factor COPD – Age and locality

The latter estimates assume that all cases of airflow obstruction in the younger age groups are due to other diagnoses than COPD, such as asthma. Table 18 shows the estimated prevalence of COPD in urban, suburban and rural England, based on the national population distribution and smoking prevalence. The values in brackets show the estimated average prevalence for areas in the lower and highest quintiles of deprivation. The average prevalence in over 35s varies 4-fold, with the highest values in men in deprived urban areas, and the lowest in women in wealthy rural areas. When the effect of ethnicity is also considered, the variation in prevalence reaches 7-fold, from 1.7% in Asian women from rural areas in the lower

quintile of deprivation to 12.5% in black men from urban areas in the upper quintile of deprivation.

Model validation

a. Predictive Validity

The model validity was tested by comparing COPD expected prevalence results to an alternative model, based on a survey of prevalence studies. An additional test involved testing the population of another West London locality population and compared the results with those from a population survey of a similar locality from another borough of London. Using a desktop assessment approach, the results were slightly lower but within the 95% CI of those estimated from the survey (4.9% total prevalence in the 40-69 year olds compared with 6.1% (95% CIs = 4.5-7.7) in the survey. The prevalence estimates for the sector were similar to the Health Needs Assessment report and to other studies that used the BTS definition of COPD. The significant correlation between expected prevalence and diagnosed COPD and COPD mortality gives reassurance of validity. The significant correlations between expected prevalence and both diagnosed COPD and COPD mortality, gives us further reassurance of validity.

b. Density location

Demand for health care provision in the community is assumed to be evenly distributed according to needs and demand. The location of health care services depends largely on the local health care system mediated by needs to have rapid access services, historical factors and so on. However, access

to primary care is a significant part of that process and where there is a high density population of a particular condition in a given location this brings enormous pressure on the system.

The possibility that there may be a high expected risk in some small areas and as part of the validity of the model proposed, the study looked at the density issue for COPD. This is equally applicable to any long term condition.

The use of multinomial logistic regression which uses odd ratios based on age, sex, ethnicity, rurality, smoking and deprivation scores logistic use of prevalence measures for prevalence estimates is generally accepted and recommended by the Association of Public Health Observatories (APHO).

From the results in this study suggested that the ratio of recorded to expected prevalence was not synchronous. Whilst this was not unexpected and considering that has a problem of health inequality within the borough, this may suggest that there is a north-south problem. The south (with its high deprived areas) will have a higher recorded prevalence and there may be a problem of under-diagnosis. This may be also helped by the fact that there is a high mobile population in this area.

Using the same parameters for COPD prevalence estimates e.g. age, gender and post-code, the odds ratios for each person was calculated. As odds ratio are multiplicative, an overall odds ratio for each person is derived as the

product of the individual odds ratios. In general, if a patient has a set of n odds ratios for a particular condition, the patient's odds ratio is expressed as

$$O_p = \prod_{i=1}^n O_i$$

where O_p = Odds ratio of population n

The overall prevalence rate P was calculated as the expected total number of COPD patients (from the model) derived by the number of residents in the age range.

The calculated risk population for an area $C(a)$ was defined as:

$$C_a = P * O_a$$

The expected number of COPD patients $[E(a)]$ in an area $C(a)$ was calculated as:

$$E_a = C_a * P$$

The risk per registered person in an area $[R(a)]$ was calculated as:

$$R_a = \frac{C_a}{m}$$

where m is the total number of residents

The results were mapped at postcode level in the GIS System (software used in public health) using boundary files to plot COPD risk density across the borough.

The map (figure 20) shows the risk density at post code level (the sum of the odds ratios for all registered patients living in the post code boundary). The shading can be interpreted that the chance of finding a COPD case in a post code with the darkest colour is more than 20 times that of finding a COPD

patient in a postcode with the lightest shading, which may suggest an efficient strategy for a targeted approach by the health authority.

Discrepancies between modelled estimates at practice level and other sources of data such as QOF disease registers may be due to local variations not captured by the model and cannot be solely attributed to weaknesses in QOF data. For practices with populations that significantly differ from a 'typical' population (e.g. large black or ethnic minority population that has a very different smoking pattern to the local average) the assumptions of the model may not apply and discrepancies may occur.

While some quite wide areas have low risk density and others are overall high, in many areas postcodes having a high risk of COPD are very close to post codes of low risk. Also, the pattern of risk does not always reflect the overall deprivation in the borough. For example, patients resident in parts of the north of the borough, which is very affluent, have a relatively high risk of COPD, whilst the reverse is also true of the south.

The local area model predicts individual numbers of cases in an area, a method that becomes less reliable as the size of the population decreases. It would be inappropriate to use it on a population as small as one post code. By aggregating the individual risks within an area and expressing the result as the relative probability of finding a case in the area, rather than predicting actual number of cases, case findings strategies based on looking where the risks are the highest, can be formulated.

The model described here is open to investigation and question and the results can be aggregated to any defined geographical area. Output as post code level enables GPs to identify where in their catchment areas they have the greatest likelihood of finding a previously undiagnosed patient with COPD, especially where their catchments have highly varied COPD densities.

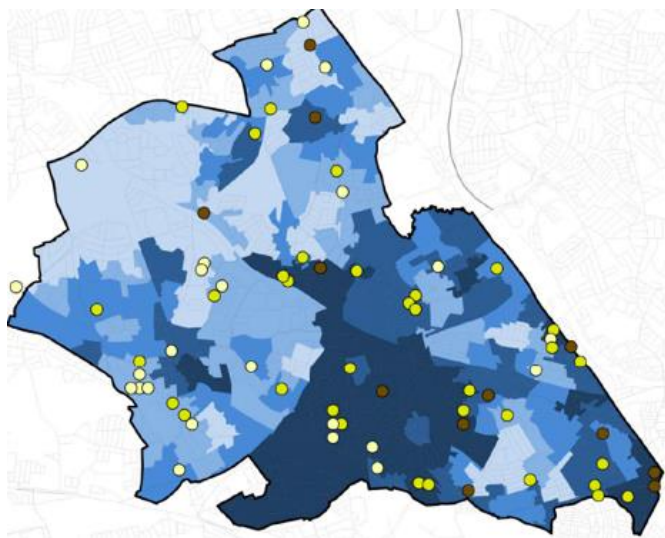


Figure (20) – Map showing the density location of COPD cases in Brent.

c. Expected versus Observed

The model was evaluated by comparing COPD expected prevalence results to an alternative model, based on a survey of prevalence studies.

For the predictive validity, GP-diagnosed and registered prevalence of COPD, obtained from the QOF were used. QOF COPD prevalence estimates are based on populations registered with GPs. The study derived residence-based

registered prevalence estimates for LAs using a look-up table—a pooled extract of England GP registers—from the National Strategic Tracing Service (NSTS). These were applied to the GP registered population in NHS Brent. HES (hospital admission data) from one calendar year was used, patients were counted once in each year they were admitted rather than once over the three year period.

The overall prevalence in the population over 15 years of age was estimated at 3.1% (3.9% in men and 2.4% in women). For those over 45 years old, the estimated prevalence was 5.3% (6.8% and 3.9% in men and women respectively). The gender difference may be related to their longer history and intensity of smoking, as compared to women. The effects of ethnicity and area of residence are more evident in women, among whom deprivation score is not apparently relevant, after other variables are considered. Urban environment increases the risk of COPD, possibly through higher air pollution levels.

Social deprivation may increase the risk of COPD through complex mechanisms in addition to the higher prevalence of smoking. This may include different smoking habits (the model does not take into account duration and intensity of smoking as such information is not readily available) and a higher likelihood of exposure to other risk factors, which are not easily measured, such as passive exposure to tobacco smoking, history of respiratory infections and less access to health services and information. Ethnic differences in

susceptibility are less clear and less well understood, but might involve a combination of behavioural, environmental and possibly genetic factors.

The assumption that ethnicity is not associated with being a case of COPD, i.e. that all population has the same risk as the white population did not change the total national prevalence estimates considerably (1,297 thousand in 15 year-olds and over and 1,065 thousand over 45s under this assumption).

A key advantage of the COPD model is that it is based on high quality data from a large representative sample of the population and uses standard and specific diagnostic criteria for COPD, which is based on lung function rather than symptoms. Response rates were high in the survey with the achieved samples matching the target populations closely. Prevalence estimates are based on the strength of association between key risk factors for COPD, including the effects of ethnicity, area of residence and deprivation, which were shown to be independent risk factors for COPD in the HSfE survey. This represents a significant advantage in relation to previous COPD prevalence models, (Rushton 2005; Barnes 1999) which were based only on smoking status, age and gender (also used in the COPD model) of mostly white populations outside the United Kingdom. The distribution patterns of COPD cases is shown below (figure 21). It would be noted that there is a significant number of under reporting of COPD.

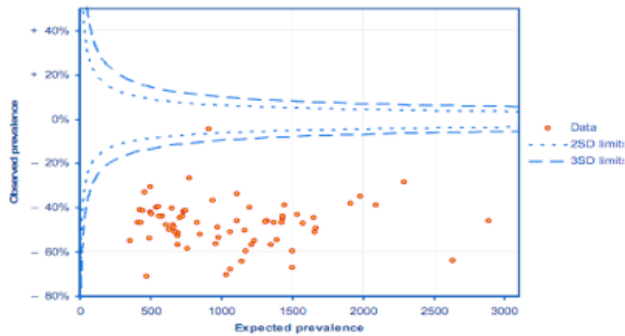


Figure 21: Funnel plot showing under reporting of COPD prevalence in Brent.

6.3. Developing the local model

Phase I

As discussed, the national model becomes less reliable as the size of the population decreases. An effective local model (at GP level) has to consider the variances and nature of local data.

The three-stage process as described previously for CHD (page 100) is applied to the prevalence estimate of COPD. The predictions made concern the five (5) localities of the borough focussing on GP practice populations. As with CHD modelling framework, the approach used the national datasets to make a crude estimate of Brent COPD SMR. The model assumes that SMR is correlated with prevalence rate.

Taking into consideration the various risk factors within and between local authorities, SMR for Brent was estimated to be 120. So the model would predict that GP practices will have an increased predicted SMR by 20%. The

model makes the assumption that areas with higher COPD mortality rates have comparably higher prevalence of COPD.

Using a linear regression model in the form of $Y = mx \pm c$, the prevalence estimate for locality (a) is calculated sequentially. First, the local SMR (adjusted) is determined by **COPD SMR = (4.389 × UV67) – 26.04** where 4.389 is the gradient, UV67 the deprivation index (for Brent) and 26.04 is the intercept. This is fully explained in the CHD section.

The second stage gives the multiplication factor using an UVF score for a named locality. To get the adjusted prevalence measure, the adjusted local SMR is divided by the national SMR estimation for Brent.

Model assumptions

The model assumptions include:

- ◆ The real prevalence of COPD in non-smokers under 35 years of age (baseline prevalence) is the same as the prevalence in non-smokers of the same age group and gender in the 2001 HSfE population.
- ◆ The ratio of odds and prevalence of COPD in the various age groups compared to the baseline group is the same as in the HSfE for each gender, smoking status and other risk factors. The risks in those falling within each of the risk categories are uniform.
- ◆ The model outputs are the prevalence of COPD for the relevant geographic area as defined by the user. In this instance, we are focusing on the Borough of Brent through its 5 localities based on the estimated population (2009-10).

6.4. APPLYING THE MODEL

Applying the prevalence estimate model to 1 local GP practice datasets (from Kilburn) we have:

Worked example		
COPD SMR Based on linear regression (fitted factors)		120
Estimated prevalence	All GP practices are expected to have an excess of 20% above baseline	
UV67 for practice X	* derived from 2001 Census	35
Adjusted COPD SMR _{local}	$Y = mx \pm c$ $= (4.389 \times UV67) - 26.04$	= 127.6
Multiplication factor	$= ((4.389 \times 127.6) - 26.04) / 100$	= 1.3
Adjusted prevalence rate	$= \frac{Loc_adj_SMR}{Nat_est_SMR} \times mf$ $= (127.6/120) \times 1.3$	= 1.4

This shows that GP practice X within the Kilburn locality had a higher prevalence than borough estimated by national formula. The formula was applied to each GP practice in Kilburn (table 19) and extended to the localities in the borough. A summary of all localities is summarised below (table 20). Analysis of the data show that the difference between the two sets showing a deficit of 83 cases for Kilburn and 313 for the whole borough. As the NHS is based on programme-based budgeting this would lead to significant reduction in fundings for this case-mix.

Practice	Population	Prev prob	Adj_est	Nat_est
1	1844	0.013	24	22
2	2398	0.017	41	38
3	2568	0.016	41	38
4	2838	0.01	28	26
5	2914	0.013	38	35
6	3069	0.011	34	31
7	3079	0.013	40	37
8	3124	0.013	41	37
9	7155	0.017	72	62
10	7409	0.013	96	89
11	13521	0.013	126	112
12	14367	0.03	188	154
13	5466	0.023	126	116
14	6020	0.013	78	72
15	6811	0.015	102	94
			1074	991

Under estimation of cases 83

Table 19 : Adjusted COPD prevalence estimation using the local modelling framework against national estimates for Kilburn GP practices.

Locality	Population	Adj_est	Nat_est
Harness	32014	416	384
Wembley	72171	938	866
Kingsbury	70153	912	842
Willesden	55824	726	670
Kilburn	82583	1074	991
		4066	3753

Under estimation of cases 313

Table 20 : Adjusted prevalence estimation for COPD in all localities (local versus national estimates)

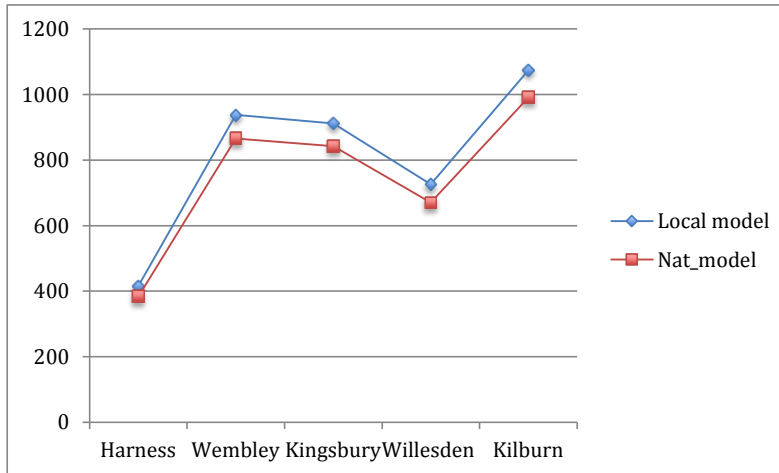
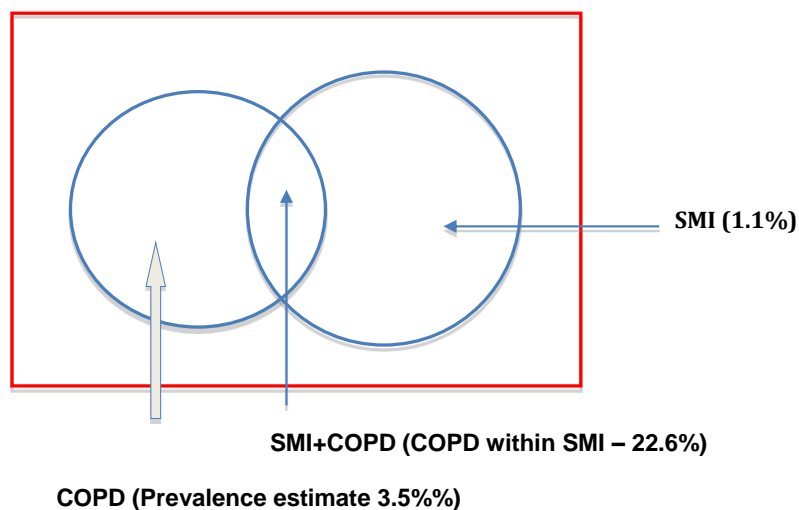


Figure 22: COPD predicted prevalence of local adjusted estimates against national data within the localities.

6.5. Prevalence estimate of SMI population with COPD at local levels

There is a good body of evidence that there is a high risk of COPD among the SMI population (Osborne et al 2007). However, as with CHD, there is no national template for estimating this co-morbidity within the SMI population. Using the Bayesian probability estimation rationale, it is possible to derive this estimate (see chapter 4). Applying it to the Kilburn locality as an example, a step-wise calculation is shown on how this derivation is determined (figure 23).

Figure 23. A schema to illustrate the relationship between COPD and SMI in a given population.



Applying to local setting

Calculation for the presence of COPD in the SMI population within the locality was undertaken by the following process:

1. Establish probability of COPD prevalence (based on the estimate model methodology).
2. Use SMI prevalence rate as determined by QOF.
3. Use the probability estimate (based on systematic reviews) to establish the likelihood of COPD with SMI.
4. Calculate the likelihood with SMI to have COPD.
5. Test against case finding. In order to find this statistic, the GPs had undertaken a complex search of the system to match the two conditions.

Estimation of COPD in SMI population

The prevalence rate of the SMI was based on QOF data (as reported by GPs)

$$P(COPD | SMI) = \frac{P(COPD)(PSMI)}{P(SMI | COPD)}$$

Where

- $P(COPD | SMI)$ is the probability of having COPD given that one has SMI
- $P(COPD)$ is the probability of COPD in the local population
- $P(SMI)$ is the probability of SMI in the population (using QOFs)

Worked example:

$$P(COPD | SMI) = \frac{P(COPD)(PSMI)}{P(SMI | COPD)}$$

$$P(COPD | SMI) = (0.02) * (0.11) / (0.23) = 0.00973$$

Applying the formula to the Kilburn practices, the number of cases of SMI with COPD were calculated (table 21). Case findings showed the actual numbers were less than the predicted numbers. It is possible that (a) the number of actual cases in practices are under reported (b) prediction is “inaccurate” or a combination of both. Figure 24 (a-e) show the expected versus predicted numbers in the district.

Kilburn Population	Prevalence by locality		SMI + COPD	
	Mean=0.23	Predicted	Actual	
1844	0.023	3	2	
2398	0.023	4	3	
2568	0.023	3	3	
2838	0.023	4	3	
2914	0.023	4	3	
3069	0.023	3	3	
3079	0.023	3	2	
3124	0.023	4	3	
7155	0.023	3	2	
7409	0.023	3	2	
13521	0.023	3	2	
14367	0.023	3	2	
5466	0.023	6	4	
6020	0.023	4	3	
6811	0.023	3	2	

Table 21: SMI with COPD – Expected vs Registered

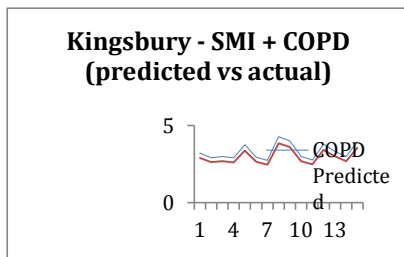


Fig. 24 (a)

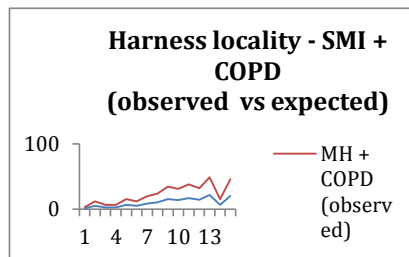


Fig. 24 (b)

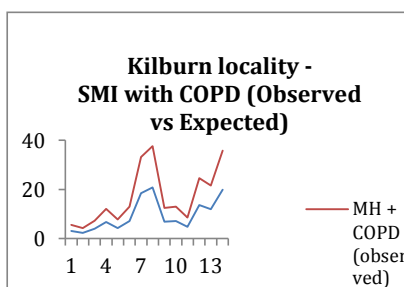


Fig. 24 (c)

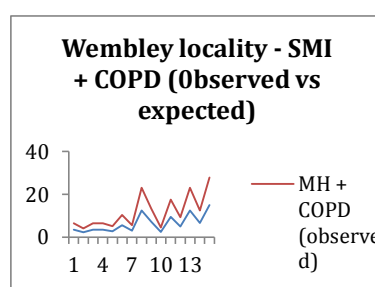


Fig. 24 (d)

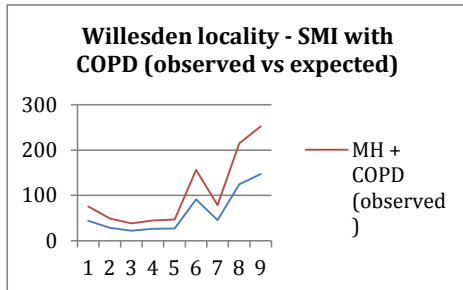


Fig. 24 (e)

Figure 24 (a-e): Graphical representation of SMI with COPD in five localities

Table 22 T-tests showed difference between the case findings and the predicted prevalence ($p < 0.0013$, $df = 14$) for the Kilburn locality. Tests for the other localities are summarised in tables 23 and 24 .

Kilburn locality		
t-test: Paired 2 sample for means		
	Register count	Predicted
Mean	89.73	128
Variance	1153.35	2355.71
observations	15	15
df	14	
t (2 tailed)	7.61E-08	
t critical value	2.14	

Table 22: t-test for SMI and COPD (Kilburn)

Locality	Prevalence by locality		SMI + COPD	
	SMI	Mean	Predicted	Actual
Harness	3214	0.048	31	24
Wembley	511	0.076	95	82
Willesden	113	0.084	147	106
Kingsbury	647	0.079	155	125

Table 23: COPD predicted prevalence against observed cases within the localities.

	Harness	Wembley	Willesden	Kingsbury
Mean	104.47	93.14	112.33	102.44
Variance	2692.84	881.65	3183.77	987.78
Observations	15	10	10	13
df	14	9	9	12
t stat	7.785	9.86	6.3	5.67
<i>P (2 tailed)</i>	<i>1.8E-06</i>	<i>4.3E-06</i>	<i>0.00004</i>	<i>0.003</i>

Table 24 T-tests – Showing the difference within and between localities. For all localities (t-test, df 9-12; p< 0.01)

6.6. Multiple co-morbidity

Multiple co-morbidity is the co-occurrence of a number of diseases within the existence of another disorder. This study sought to estimate the prevalence of two physical disorders namely CHD and COPD within the SMI population at a local level. The prevalence estimation was based on a one-year incidence based on data available for the last full year. After the estimated prevalence of the physical disorders was calculated, the model was then adjusted to calculate the expected number of SMI within that estimated prevalence. In order to estimate the prevalence of SMI with condition (X), Bayesian statistics were applied in the form:

Estimation of population size = ((P COPD local Population) X P SMI (SMI in local Population)), where P is the probability.

The generic formulation was used to estimate prevalence values for any population for which appropriate demographic information is available. As such, the model could be adapted to calculate any co morbid chronic disorders provided we had the relative risk ratios.

Pathway for determining prevalence estimate for SMI.

The CHD prevalence in Brent is estimated to be 7.8% in 2009 (n=295,678). This is higher than the prevalence reported by Brent PCT (2.2%). However, this was not unexpected due to the data quality issues and the quiriness of QOF.

Having established the prevalence estimate for the two conditions CHD and COPD within the SMI group for the locality, the next step was to calculate the likelihood of the prevalence of these conditions simultaneously. The estimation of concurrent co-morbidity was carried out through probability modelling (using bayesian statistics).

A. The probability of having two conditions is a product of the separate prevalence, namely

$$P(A,B) = (P(a))(P(b))$$

$$P(\text{CHD,COPD}) = \{ (\text{Probability (CHD)}) \} \{ \text{Probability (COPD)} \}$$

Alternatively,

B. The probability of having none of these conditions

$$P(0) = (1-P(a))(1-P(b))$$

C. To calculate the probability of having one of the diseases, namely

- Having A and not B
- Having B but not A

$$P(1) = \{ (P(a)(1-P(b))) \} \{ (P(b)(1-P(a))) \}$$

The probability of having two (2) diseases,

$$P(2) = \{ (P(a)(1-P(b))) \} \{ (P(b)(1-P(a))) \}$$

Locality - Kilburn

Using the above formula, the predicted number of cases of the SMI population that suffer from CHD and COPD for Kilburn is 52. (table 25).

cluster	Population	SMI_Prev	CHD_prev	COPD_Prev	SMI+CHD+COPD	Predicted number
Kilburn	1844	0.5	0.023	0.0274	0.0006302	1
Kilburn	2398	1.4	0.023	0.0274	0.0006302	2
Kilburn	2568	0.7	0.023	0.0274	0.0006302	2
Kilburn	2838	0.6	0.023	0.0274	0.0006302	2
Kilburn	2914	1.2	0.023	0.0274	0.0006302	2
Kilburn	3069	0.9	0.023	0.0274	0.0006302	2
Kilburn	3079	0.8	0.023	0.0274	0.0006302	2
Kilburn	3124	0.9	0.023	0.0274	0.0006302	2
Kilburn	7155	1.3	0.023	0.0274	0.0006302	5
Kilburn	7409	1.1	0.023	0.0274	0.0006302	5
Kilburn	13521	1.3	0.023	0.0274	0.0006302	9
Kilburn	14367	1.1	0.023	0.0274	0.0006302	9
Kilburn	5466	1.5	0.023	0.0274	0.0006302	3
Kilburn	6020	0.4	0.023	0.0274	0.0006302	4
Kilburn	6811	1.1	0.023	0.0274	0.0006302	4
TOTAL						52

Table 25: Estimated prevalence probability (and numbers) for SMI with CHD and COPD for the Kilburn locality.

The calculations were extended to the rest of the localities (table 26). The total number expected to have these multi co-morbidities is 206.

	SMI Prev	CHD Prev	COPD Prev	SMI + CHD + COPD	Predicted number
Harness	1.06	0.0048	0.0274	0.00132	42
Wembley	0.84	0.02	0.02	0.003	35
Kingsbury	1	0.028	0.027	0.00143	43
Willesden	0.92	0.03	0.04	0.001	34

Table 26 – Estimation of SMI with CHD and COPD in the other localities.

7. Discussion

The aim of the study was to develop a model for estimating the prevalence of multiple physical co-morbidity within the SMI group at local area level. As direct estimation is not possible, this was carried out in stages. The first stage was to estimate the prevalence of the physical disorders within the geographical locality followed by a synthetic estimation of these conditions within the local SMI population (based on QOF registers). The estimation was made using Bayesian methodology. This discussion focuses on the extent to which the research undertaken can answer the research questions originally posed in 2009 These are:

- i. How do estimations of chronic disease for local areas compare in terms of their validity?
- ii. What is the best methodology for estimating the prevalence of multiple co-morbidity within the SMI population?
- iii. How useful are these prevalence models compared with case findings? Can they be used instead of case registers?
- iv. What are the implications of the findings for public mental health planning policy?
- v. What are the potential impacts of this study?

These questions are obviously closely related. The answer depends on the criteria which should be used to compare prevalence modelling methodologies, their “fitness for purpose” in providing reasonably robust local, small population/area estimates which can be used by PCTs/ GP practices

and in future GP commissioning groups and LA public health departments. It is important to distinguish between decision-analysis “cost-effectiveness” models and population-based “surveillance” models. A surveillance or prevalence model differs from decision-analysis models in that rather than representing a hypothetical cohort, it models the population, that is, a collection of birth cohorts, over a specified period of time.

7.1. Validity of prevalence estimation of chronic disorders at local level

In order to consider this question, we need to reflect on the purpose of a methodology for prevalence modelling at local levels. National prevalence estimates are designed to provide rough but reliable estimates at a macro level and estimates for a particular geographical area described as more "direct" are more preferable for local commissioning. National prevalence surveys are not designed to produce "direct" estimates for counties as the sample sizes are too small, and hence, the estimates are not reliable or stable (Department of Health 2001).

A geographical area is regarded as "small" if the area sample is insufficient to yield direct estimates with adequate precision and reliability. In order to make estimates for small areas with adequate levels of precision, it is standard to use indirect estimates that utilize information from outside areas with similar characteristics to the area of interest. To that effect, this study used a statistical model to obtain indirect estimates for geographical areas considered to be "small". The use of such a model decreases the variability of the small

area estimate, but if some of the characteristics which the model relies upon e.g. QOF, are not stable, it may introduce bias into the estimates. For example, the movement of patients within and between localities (which could be as much as 30%) could be significant in prevalence calculations.

The statistical methodology proposed could be viewed as a proof of concept. This model-based estimate is intended to be an improvement on national estimates, if the models used are appropriate. However, that may not mean that the model-based estimates are close to the true values for every area.

The International Society for Pharmaco-economics and Outcomes Research (ISPOR) has published principles of good practice for decision analytic modelling in health-care evaluation (Anselin 2006). This is a comprehensive framework for validating population-based chronic disease simulation models and has been used in a review of published model validation guidelines (Leung *et al.* 2000). Based on the review, a set of recommendations were formulated for gathering evidence of model credibility. Evidence of model credibility derives from examining:

- a. Model development process
 - i. Is it conceptually robust? Are the theories and assumptions underlying the conceptual model correct and are the model's structure, logic and mathematical and causal relationships reasonable for the intended purpose of the exercise?
 - ii. Are the parameters of variables justified? Are relationships between the variables specified correctly and do they come from both theory and empirical

data? Evidence generally comes from examining the process used to derive a value for the parameter (primary source, method of derivation) and comparisons with data from other sources.

b. Evidence from examining model performance

1. Has it face validity? Is it plausible? Do we have evidence involving comparisons of output with expectations?
2. Internal consistency: assessed by considering functional and logical relationships between different output variables.
3. Between-model comparisons: “modellers should co-operate in comparing results and articulating the reasons for discrepancies”. This could be achieved by comparing the APHO prevalence models with, for example, Congdon’s prevalence models (SB P 2004).
4. Can the data be used in comparison with external data? Available data should be used in model development and data should not be withheld for the purpose of external validation.

Using this approach as a broad framework for our validity assessment, the study has to satisfy the major components. We can start by stating that the use of simulation models to provide local estimates is a new trend. Such approaches have until now focused on model construction - the iterative process of scope selection, hypothesis generation, causal diagramming, quantification, and reliability testing in large populations, rather than combining simulation models with small area co-variates. However, it is feasible to do this and it will certainly be done in the future.

It is likely, but by no means certain, that more sophisticated methods of model development provide better predictions, but a notable feature of the literature is that very little comparative validation has been carried out and most have compared similar methods.

The use of Discrete Event Stimulation (DES) technique as opposed to Markov chains may be more popular with decision makers as it gives superior face validity. Furthermore, this model automatically provided a probabilistic sensitivity analysis, which is cumbersome to perform with a Markov model. DES models also allow inclusion of more variables without aggregation, which may improve model precision. However, the differences were not significant in terms of the actual predictions. Moreover, these comparisons are only one aspect of model validation. The use of the more recent technique of GWR has yet to be fully considered.

For example, two or more modelling methodologies could be compared to a validation gold standard of local prevalence. This data is difficult to obtain; one option would be using data from populations in which extensive case-finding has apparently detected nearly all cases of disease. NHS Health Checks data might serve this purpose, once a large enough proportion of the population has been screened. Unfortunately, although the present national policy excludes cases currently on QOF registers, it does not specify that questions about patient-reported doctor-diagnosed disease be asked to ensure that cases not on practice registers are found.

An important step of the validation framework involves determining the minimum sample size needed to achieve sufficient correspondence against a gold standard. The gold standard serves as a benchmark judged to be the best available direct estimate for the small area domain (Audit Commission 2009). The IHME gold standard is to use sufficiently large sample sizes, which can be obtained by choosing small domains with large sample sizes in a single survey year, pooling multiple survey years, or increasing domain size. They used as their gold standard the direct, age-standardized, sex-specific estimates for counties that had more than 900 observations in both periods 1996-2004 and 2000-2008 (the validation sets).

Such a validation environment allows the selection of a modelling strategy that optimally mixes the three approaches of pooling data across time, harnessing spatial patterns in the distribution of the outcome of interest and adjusting for estimates for local area characteristics. This approach is analogous to that used in the development of risk predictions models, where derivation and validation cohorts are often initially derived from the same population: for example, in the case of QRISK2 derivation, random sampling was used to assign practices to the derivation or validation cohort and then their population data was used. However, validation should still be carried out in other populations if possible.

Finally, two comments on the statistical analyses used in the study. The first concerns the procedures used in the study. These were drawn from extensive

work, mostly outside the mental health field. However, this work remains experimental and vulnerable to several methodological limitations (Kisely *et al.* 2009). The associated inferential techniques have not been fully validated and not incorporated into available statistical packages. Secondly, is the use of Bayesian methodology. This is at the root of local estimation paradigm. The advantage of using the Bayesian iterative calculations is that it gives a practical usefulness for commissioners compared to classical analysis (Nasrallah *et al.* 2006). Bayesian modelling provides an ease of obtaining predictions and the possible extension to incorporate other relevant information or beliefs. However, it does have some disadvantages in that it requires specialist software and the difficulties that are sometimes encountered in achieving Markov chain Monte Carlo convergence (Burns and Cohen 1998).

In summary, although the model has a working validity, the use of small-area analysis techniques for sub-population (multiple co-morbidity within a specific group of illnesses e.g. SMI) has not been fully validated and is an area for future research. The model developed in this study needs to be tested with data from parallel yet independent research. There are gaps in establishing model credibility e.g. credible interval estimates; the majority of the above criteria have been satisfied.

7.2. What is the best methodology for estimating the prevalence of co-morbidity within the SMI population?

The study focus was on establishing the prevalence of specific disorders within the SMI group and in that context looked at individual conditions sequentially e.g. co-existence of CHD, followed by COPD etc. The rationale for this approach was determined by availability of data and other practical constraints such as the extraction tool that was available for GP systems i.e. EMIS (Information centre for Health and Social care 2011). The methodology used was both appropriate and justified.

However, patients with SMI have multiple pathology and as such some methodological choices and different analytical strategies have to be considered (Wang *et al.* 2002). Of importance is to consider if co-morbidity or multi-morbidity should be used as the dependent variable or an independent variable. Hudson (2009) points out, for example, the proportion of subjects in the over 65 suffering from two or more disorders from a list of four (hypertension, emphysema, psoriasis and osteoporosis) was 2.8%. When glaucoma, diabetes and gout were added to this selection of diseases, the prevalence of multi-morbidity in the same population increased to 8.9%. The same rationale is applicable to the SMI populations.

A key decision to be made before the start of future studies is whether or not to account for known patho-physiological relationships between diseases. For example, do diabetes mellitus, retinopathy and a diabetic foot in one person count as one or as three diseases? The research question should be the main

guide in decision making. For example, in a study on the complications of diabetes, one is probably interested in the whole spectrum of co-occurring diseases, whereas in other studies it might be more interesting to gain insight into various disease entities. An obvious difficulty when taking pathophysiological relations into account is that present knowledge of those relations is still limited.

Another important point to consider when analysing co-morbidity is the influence of effect modifying or confounding variables. For example, as age is a strong determinant of many diseases, it is generally important to take this variable into consideration when analysing the co-occurrence of diseases. The model did not take this into consideration. If these influences are not taken into account or at least described, this can lead to unrealistic or irrelevant outcomes. An important conceptual consideration in this context is whether the co-variable is an element of the causal chain to be or just a confounder without relevance to the causal chain of primary interest (when adjustment is useful). Evaluation of effect modification may be helpful to identify different co-occurrence patterns in the sub-groups.

A suggestion is that when analysing combinations of three or more co-occurring diseases, stratified analyses are a good option as long as the study population is sufficiently large, giving the opportunity to account for the main co-variables. Another option is to carry out a stepwise multiple logistic regression analysis, evaluating the determinants of the presence of a disease additional to a specific disease or combination of diseases.

7.3. How useful are these prevalence models compared to case findings? Can they be used instead of case registers?

The regionally observed prevalence data, to which the national estimates are compared, may not be representative of the national situation (Halliwell *et al.* 2007). Prevalence variations show distinct geographic 'contextual' effects that are differentiated between ethnic and other demographic categories (Barnard *et al.* 1999). This study identified similar findings to that of other studies which found that major geographic variations do not seem to be explained by area demography alone.

As an example of how local estimates can be generated and used, a local Canadian public health agency (population 250,000) recently published a "textbook" for local disease incidence modelling. Age standardized incidence ratios for cancer and the prevalence of Census co-variables were calculated for each of 331 dissemination areas. The standardized incidence ratios (SIRs) for cancer varied dramatically across these areas. Employing Bayesian hierarchical models, areas in the urban core were found to have significantly higher SIRs for male lung cancer than the remainder; and neighbourhoods in some urban and surrounding rural areas exhibited significantly higher SIRs for prostate cancer (Druss 2007). After adjustment for age and spatial dependence, average household income attenuated much of the spatial pattern of lung cancer, but not for prostate cancer (Goldman *et al.* 1981).

Because both models are based on the assumption that incidence and prevalence are in a steady state, the occurrence of trends in incidence or

mortality would lead to discrepancies between the model estimates and the observations. Prevalence is a stock variable, comprising all past incident cases that are still alive. It is therefore dependent on incidence and case-fatality from the past as well as the present.

In the SMI group, where multiple medical problems are frequent and pathological examinations are performed relatively infrequently, misclassification of some disorders like COPD may lead to the over registration for the more frequent types of COPD (National Institute for Health and Clinical Excellence 2009). This incompleteness seems to be concentrated in geographical areas often proximal to acute mental health institutions. This would cause distorted prevalence estimates and could contribute to the wrong conclusion of our prevalence estimates.

The under registration of incidence may also help to explain the impossible negative prevalence calculated for physical illnesses. Under registration cannot, however, explain the finding that the prevalence estimates are generally higher than the observations for the other age groups.

However, this present study demonstrates the feasibility and utility of local datasets to obtain more accurate estimations. This is what the Informing Healthier Choices project aimed to do with the APHO/ICL prevalence models. Further work is needed to evaluate the impact of using case registers routinely at local level.

7.4. Impact of the study

The findings from this study provide some useful context for health care planning. They support the general view that data surrounding co-morbidity in general and specifically among the serious mentally ill is not readily being addressed by mainstream public health departments in the UK. Information available at local levels are of limited use to the commissioners.

Results show that prevalence estimates of multi-morbidity vary widely among the localities. The largest difference was observed in geographical areas with high deprivation. It could be argued that differences of this magnitude are unlikely to reflect real differences between populations and more likely to be due to biases in methods. In addition to their differing geographic settings, prevalence studies differed in recruitment method and sample size, data collection and operational definition of multi-morbidity, including the number of conditions and the conditions selected. All of these factors may affect prevalence estimates.

Some of the implications are serious in that we are aware that people with serious mental illness die, on average, 25 years earlier than the general population. This has been demonstrated in a number of recent studies (Information Centre for Health and Social Care 2009). While suicide accounts for about 30% of excess mortality, about 60% of premature deaths are due to “natural causes”, such as cardiovascular and pulmonary disease. Cardiovascular mortality in schizophrenia has increased from 1976 to 1995, with the greatest increase in Standardized Mortality Ratios in men from 1991

to 1995 (Information Centre for Health and social Care 2009). This is a serious public health problem that is poorly recognised and rarely addressed. Many of the risk factors for these “natural causes” of death, such as smoking, obesity and inadequate medical care, are modifiable. Increased attention from policy makers as well as persons served, family members and the mental health and general health care system is needed (Virgo *et al.* Journal of Mental Health 2010, volume 14).

The estimation of medical co-morbidities within the SMI group is a real challenge. As indicated, the presence of these co-morbidities is a variable constant as it depends on various socio-economic factors. The application of a simple adjustment tool has a potential pragmatic value. It allows us to have a broad measure of the underlying epidemiological trends.

7.5. What this study adds

Work by Phelan *et al* (2000) has shown that the chronically mentally ill have serious risks of developing physical health problems. The current commissioning model is based on a single disease framework and does not address issues of co_morbidity. Part of the problem is that there is lack of relevant information needed for policy planning. Within public health, not much work has been conducted in prevalence estimates for multiple comorbidities .

The present study sought to address the complexity of estimating the prevalence of concurrent medical disorders (using CHD and COPD as exemplars) within the serious mentally ill population in small population

samples. To-date very few studies have investigated estimation of prevalence rates linking these diseases together for commissioning intentions.

The part of the problems lie in that national prevalence models in the UK use a wide number of population characteristics such as socio-economic parameters which are derived outdated information sources e.g. from 10-yearly population censuses. More pertinent, is that these national data are out of date and very often not relevant to local settings. Allowance are not made for the effect of deprivation, but to do this quantitatively requires a numerical estimate of the likely extent of its effect in any area.

As such, the present prevalence models do not reflect the local variations and do not produce satisfactory predictive power as they do not take into consideration geographical patterns. Thornicroft (1991) and Carr-Hill et al. (1994) argued that prevalence modelling work should entail defining specially tailored combinations of individual census items, rather than taking the simpler approach of using national estimates.

The study showed that the prevalence estimate for the borough was not in synchrony with that indicated by Public Health (England) and used by the local commissioning group. The wide variability within the locality points for the adoption of a focussed risk adjustment for mental health service planning policy for the borough. The finding that over 60% of SMI group live within 1 mile radius of the local acute mental health services needs further investigation.

The findings also suggest that the projected costs for the physical health care treatment of adults with SMI should be adjusted for risk in the same way as costs for mental illness treatment. Under-use of medical services by the seriously mentally ill is a growing concern and if projected costs for adequate health care are underestimated because severity and prevalence of physical health were not taken into consideration, medical services may not be available to everyone who needs them. This would be a gross health inequality.

Within that context local commissioners face are two folds (a) develop an approach to undertake local estimates methodology for diseases (b) determining a methodology for estimating co-morbidity prevalence within serious mental illness.

The model proposed by this study suggests that in order to obtain prevalence estimates that are more sensitive to local variations, a set of sequential procedures should be initiated:

1. Use annual needs assessment to ensure veracity of local indicators
2. Revise national regional estimates considering local variances to estimate prevalence of CHD/COPD within GP localities
3. Test prevalence estimates using case findings. This can be done routinely via annual audits e.g. QOF. Bi- annual mental health needs assessment

should be part of the commissioners requirements as they provide a unique opportunity to explore local variances.

4. Use a simple algorithm(as developed by the study) and some basic statistical techniques, to develop a better estimate forecast of co-morbidity within SMI population.

The model described here is open to investigation and question, and the results can be applied to any defined geographical area. Commissioners can have a better understanding of high pockets of needs and risk densities.

7.6. Limitations

Possible confounding variables

An important point to consider when analysing co-morbidity is the influence of effect modifying or confounding variables. For example, as age is a strong determinant of many diseases, it is generally important to take this variable into consideration when analysing the co-occurrence of diseases. Part of the co-occurrence of diseases can be explained by known influences of age (e.g., benign prostate hypertrophy and osteoarthritis). Of course other variables such as socio-economic status, environmental factors and psychological features can also be very influential (Majeed et al 2000). If these influences are not taken into account or at least described, this can lead to unrealistic or irrelevant outcomes. An important consideration in this context is whether the co-variable is an element of the causal chain to be evaluated or just a confounder without relevance to the causal chain of primary interest (when adjustment is useful - Diez-Roux 2000).

Evaluation of effect modification may be helpful to identify different co-occurrence patterns in various subgroups. When analysing combinations of two diseases, known co-variables can be adjusted by using a multiple regression analysis (using one of the diseases as the dependent variable) or a stratified analysis according to when analysing combinations of two or more co-occurring diseases, stratified analyses are a good option as long as the study population is sufficiently large, giving the opportunity to account for the main co-variables. Another option is to carry out a stepwise multiple logistic regression analysis, evaluating the determinants of the presence of a disease additional to a specific disease or combination of diseases.

Because of the influence of various factors on the occurrence of diseases in general, it is also important to pay attention to confounders and effect modifiers when analysing multi-morbidity. Again, age is an obvious determinant. In multiple regression analysis with the presence or absence of multi-morbidity as the dependent variable, it is fairly simple to adjust for age or any other co-variable by including it as an independent variable. Also in this context, the consideration of the conceptual framework of the possible relationship between the evaluated associations of primary interest, possible confounding and effect modification is important.

Weaknesses

The study has a number of weaknesses including technical and systemic difficulties. The baseline prevalence for SMI was taken from QOFs data, which are generally recognised as weak. From this baseline, the levels of multi-comorbidity were established. The framework was designed to deliver evidence-based interventions into general practice. Prior to its introduction, GPs worked reactively and in an uncoordinated way by dealing with the problems patients brought to the surgery. Although QOF data are routinely collected for patients with schizophrenia, bipolar disorders and other psychoses, elements like follow up are not completed because the Gps do not chase their patients. The reported rates is close to, but usually above, the reported rate of psychiatric admissions provided by the National Centre for Health Outcomes Development (NCHOD). It is important to recognise that since QOF datasets include patients who are managed in the community without admissions, there is a disparity between NCHOD and QOFs rates. A degree of under reporting will always appear in QOFs.

There are some problems regarding the fullness and timeliness of the data. For example, the SMI population is known to have a tendency to engage in smoking behaviour and are also likely to be very heavy smokers. This has not been factored into the regression equation. The local smoking cessation teams do not routinely gather data for that group for the time being (Nijhuis et al 2006). As such, the data used for the prevalence estimates do not discriminate within the population. This is likely to increase the odds ratio and

will further increase the prevalence estimates. Further studies in this area will need to address this issue.

The risk factors were derived from data that may not be current. Ethnicity assumptions derived from census data almost ten years old may well be questionable, in the light of changing patterns of immigration in the period.

Modelling provides estimates, not answers. Any model is strictly “wrong”, but may be useful within certain limits. The methodology by means of which the models were created does not easily permit the calculation of confidence intervals on the expected numbers of patients even though 95% confidence intervals were presented for the individual factors’ odds ratios, so the models’ outputs do not incorporate an indication of the strength of belief we can have in their results. The statistical procedure used is rather basic and needs to be modified and refined. However, it nevertheless provides a new way of undertaking prevalence estimates at local levels.

7.7. Personal account

The idea of prevalence modelling was germinated in the early 1990s and was further developed by Nita Farouli and the team at Brent PCT when the diabetes prevalence model was tested. The use of prevalence modelling as a public health tool for planning, has gained momentum since then. However, availability of good health intelligence for local health planning remains a challenge, especially where the outcome of interest has multiple causes or influences. In the course of this study, I have developed a greater appreciation

for the possibilities and limitations of public health epidemiological research methodology. Public health units within primary care within the UK are struggling to juggle the conflicting roles of delivering a public health strategy which reflects national trends and meeting local variances. This is a particularly big concern for mental health.

While there may be mileage from using national data which bring a baseline of information from which we can plan services, there are some serious problems within this approach when dealing with mental health data. Unfortunately, the doctrine of evidence based medicine and the hierarchy of evidence means operational management data is often under-valued. This may lead to the under-utilisation of routinely collected data.

During the 1970s, a number of approaches were developed to use time series data to study the effect of interventions. I found the textbook by Cook and Campbell provided an excellent overview of these approaches. However, these methods are not commonly seen in current public health literature, even though many important public health questions cannot be answered by RCTs.

In my study I have considered a number of such issues and their impact on service delivery, such as ineffective primary care interventions in mental health, lack of good routine data collection, rigid (poor) health commissioning principles leading to a “silo” approach to health care planning.

In the course of this work, it has become apparent to me that to understand

the good public health research approach to deal with such complex issues such as mental health co-morbidity requires data from multiple sources to triangulate the findings. This is especially important for health outcomes which are influenced by multiple factors.

From an epidemiological perspective, I felt that the biggest limitation of my study was the inability to define the population at risk. This was particularly problematic when considering the chronic mental health population. Without knowing the mortality risk among those with physical co-morbidity, it is difficult to interpret the drivers behind changes in trend. Finally, I have become more cautious when interpreting short time series or year-on-year trends. Both of these are frequently presented in public health documents or publications and it is easy to over-interpret annual fluctuations or short-term changes in trend

7.8. Conclusions

The methods described offer a potentially useful way forward for identifying undiagnosed cases. They are applicable to other long term conditions for which similar models are available on the APHO website. Ultimately, the validity or otherwise of the approach will depend on empirical results of its use.

The future

a. Linkages with risk factor prevalence data from GP systems

The case has been made on how the current models can be linked, both with each other and with other modelled data such as smoking prevalence (eg

Chronic Obstructive Pulmonary Disease Model). As noted above, local estimates of prevalence or population means (for continuous variables) of disease risk factors and lifestyle behaviours e.g. current smoking, obesity, blood pressure and physical activity, are of great interest to policy-makers, and increasingly to local organisations for planning and performance monitoring purposes e.g. in Local Area Agreements. They are also of interest to researchers examining the relationships between health determinants, such as income and education, and health outcomes such as disease prevalence, SRHS and death, because risk factor prevalence and means provide a linkage between determinants and outcomes and may be used to predict the latter.

Until now, risk factor prevalence estimates have come mainly from the HSfE, from local surveys either run independently from the HSfE, or from synthetic estimates. The traditional ("direct") approach to estimation used for the HSfE uses classical design-based survey sampling. However, sample sizes are typically small within small areas, so the direct estimators have large sampling errors (and hence large confidence intervals) even when pooling years of survey data (which prevents trend estimates). When there are no sample observations in the small areas of interest, direct estimations cannot even be calculated.

Potential use of primary care data for risk factor prevalence/mean estimation

The quality and quantity of population-based data collected by primary healthcare teams has improved dramatically in recent years, as a result of

overall quality improvement, specific data quality initiatives and the impact of pay for performance through the QOF (Cox *et al.* 2006; Mc Fadden *et al.* 2009; Fotheringham *et al.* 2010). The data is also becoming more accessible through national GP databases, local IT projects and, eventually, aggregation through the Secondary Uses Service via GPES, which could cover over 50% of English practices. This would allow near to real-time monitoring of risk factor prevalence data.

There is a large volume of risk factor and lifestyle data in GP systems. For example, our analysis of the 2005-6 IMS national GP database (about 1million active patients) shows that overall about 30% of patients have their smoking status recorded in the last 12 months and 17% had their BMI recorded in that period. There is therefore the potential to use this data, either alone or in combination with other data, to improve local prevalence estimation but data is incomplete. It is also easier to measure risk factor prevalence through population-based programmes such as NHS Health Checks than it is to ensure QOF registers reflect actual disease prevalence. However, there are a number of methodological problems to resolve. For example, the data is non-random - sicker and older people and females are more likely to be sampled and these may introduce a bias.

Case for the development of a simulation model

A major disadvantage of the current prevalence models is that they are static, with a cross-sectional structure. In the US, the Centres for Diseases Control are already well advanced in the development of dynamic models (Cortes and

Vapnik 1995). In San Francisco, Archimedes is continuing to expand both in its scope and functionality. The UK should be developing equally dynamic epidemiological models. This requires at the very least a multi-disciplinary team with on-going support, not a lone researcher and a single injection of funding.

In the UK, in addition to the prevalence models described here, there is already a great deal of academic expertise in mathematical modelling, particularly for mental health. Examples include work by Gyles Glover for the quantitative modelling on the Mental Health Minimum Data Set (MHMDS) (Glover 2003). After very promising initial work in the late 1990s and early 2000s there has been very little support for work in this area.

There is a need for a UK modelling research for physical disease in mental health. As indicated above, this is probably because of a lack of public health interest in this area of work. This concern was voiced at the Faculty of Public Health Annual conference in 2011. A possible way forward would be to make this a public health epidemiological research priority.

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Annex 1

Mental health needs assessment

1. A section of the data compiled as part of the MHNA exercise for Brent GP practices. Colour coding reflects levels of concerns for various metrics used to monitor performance.

Practice Name	Cluster	QOF Return Analysis				Inpatient Analysis			Outpatient Analysis			Community Analysis		A&E attendances		Demographic			
		OP_Listr Population	QOF_MH Prevalence	DEP_reported_perk	DEP_expected_perk	Dementia Reported perk	Dementia expected perk	Adult Admissions perk	Expected adult admissions perk	rate per 1000 admissions self harm	First OP attend perk	Op Psych attends total perk	Community Adult Payments perk	Community Appts in Year perk	Community Elderly Patients perk		A&E MH Adult attends O&OP perk	A&E Elderly MH attendance rate perk	A&E Adult MH attendance rate perk
KENTON MEDICAL CENTRE	Wemb	3024	0.6	31.20	88.33	3.4	70.5	0.68	2.04	0.11	6.80	37.03704	4.7661197	68.428	10.07	1.7	0.0	1.0	15.47
SUDBURY COURT SURGERY	Wemb	4012	1.7	58.11	84.00	56.3	77.2	1.21	2.41	0.25	7.39	26.17149	17.6813341	207.354	32.15	2.4	4.0	5.2	19.76
LANFRANC MEDICAL CENTRE	Wemb	6995	0.5	26.87	86.65	34.9	64.0	1.33	2.23	0.81	2.52	21.01501	4.7419191	111.326	11.90	0.9	1.5	1.7	19.79
SUDBURY & ALPERTON MEDIC	Wemb	8728	0.9	43.16	86.18	11.0	63.1	1.31	2.30	0.27	6.91	19.94041	13.1386622	159.963	18.02	2.3	1.0	3.6	22.40
BEECHROFT MEDICAL CENTRE	Wemb	5583	1.2	16.84	86.55	16.8	65.7	2.33	2.33	0.36	18.86	34.03183	26.6507645	1462.663	58.92	4.1	1.7	3.6	23.85
PRESTON MEDICAL CENTRE	Wemb	3332	0.5	9.77	85.05	8.6	69.1	0.00	2.42	0.30	3.40	7.803121	3.22806155	41.171	6.47	2.0	0.0	2.4	24.03
EAGLE EYE	Wemb	2459	0.5	23.40	88.00	5.1	55.8	0.00	2.09	0.14	1.89	5.286702	3.13136058	30.792	5.08	1.6	0.0	3.7	26.25
ALPERTON MEDICAL CENTRE	Wemb	5543	0.5	5.91	86.82	2.0	64.1	0.72	2.17	0.24	5.17	18.22118	6.7642877	77.791	14.03	1.9	0.0	1.9	26.27
STANLEY CORNER MEDICAL CE	Wemb	5231	1	62.38	86.71	25.2	60.3	1.50	2.25	0.76	9.83	26.38119	14.2510688	181.264	31.45	4.3	2.1	2.3	26.58
HAZELDENE MEDICAL CENTRE	Wemb	3264	0.5	15.31	84.81	2.5	63.3	0.88	2.20	0.51	12.37	20.52696	5.72239471	94.639	10.13	3.5	2.5	3.5	26.71
PREMIER MEDICAL CENTRE	Wemb	4016	1	43.41	85.89	19.8	61.3	0.70	2.45	0.33	15.76	34.86056	14.0281967	260.924	23.72	4.0	0.0	4.2	26.94
THE SURGERY (WCHC)	Wemb	2990	0.6	26.13	85.86	15.6	65.8	1.72	2.15	0.11	6.80	22.40803	6.0132291	85.041	31.25	3.0	0.0	3.0	27.07
SMS MEDICAL PRACTICE	Wemb	2372	0.8	72.46	84.81	3.7	58.6	0.00	2.51	0.56	3.23	17.28499	6.30477271	83.853	10.99	2.5	0.0	1.9	27.71
SUNFLOWER MEDICAL CENTRE	Wemb	3323	0.9	30.55	88.39	6.8	67.6	3.71	2.17	0.90	15.51	41.52874	25.0729659	246.541	21.65	5.6	3.4	6.5	28.23
LANCELOT MEDICAL CENTRE	Wemb	4314	0.3	9.05	86.02	12.6	58.6	0.29	2.31	0.08	3.24	9.27237	5.77617329	47.653	16.74	2.3	0.0	4.0	29.33

2. Based on the model developed by the study, prevalence estimates of the locality by the public health unit. Example 2011-12

Ward	Expected Schizo	Actual Schizo	Estimated Affective	Actual Affective	Estimated other	Actual other	Estimated total	Actual Total	Variance to estimate
Alperton	8	14	7	3	7	11	22	28	27%
Barnhill	9	15	8	8	9	20	26	43	66%
Brondesbury Park	10	10	8	9	9	15	27	34	27%
Dollis Hill	10	7	8	6	9	9	26	22	-15%
Dudden Hill	10	12	8	10	9	13	26	35	33%
Fryent	8	4	7	2	7	5	22	11	-50%
Harlesden	17	14	12	17	16	8	45	39	-14%
Kensal Green	12	10	9	12	11	17	32	39	20%
Kenton	6	5	5	4	5	6	16	15	-9%
Kilburn	19	24	13	11	18	12	50	47	-7%
Mapesbury	14	9	10	12	12	13	37	34	-7%
Northwick Park	5	7	5	4	5	7	14	18	28%
Preston	7	6	6	8	6	10	19	24	29%
Queens Park	12	17	9	6	11	12	32	35	10%
Queensbury	8	2	7	4	7	4	21	10	-53%
Stonebridge	17	11	12	9	15	22	44	42	-4%
Sudbury	8	8	6	7	7	9	21	24	14%
Tokington	8	10	6	7	7	6	22	23	6%
Welsh Harp	9	9	7	6	8	7	24	22	-7%
Wembley Central	9	14	7	9	8	7	24	30	25%
Willesden Green	14	17	10	4	12	9	36	30	-17%
TOTALs	219	225	169	158	198	222	587	605	

Annex 2

Routine data collected by GPs regarding chronic diseases. Please note that exceptional reporting routine can distort figures.

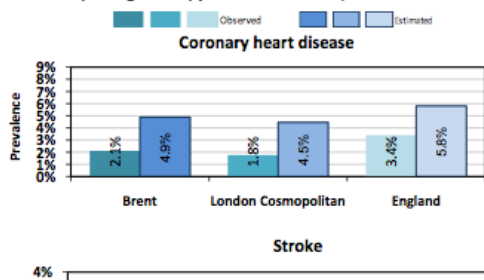
Quality and Outcomes Framework - exceptions	
PCT	2010/11 EER
Brent	5.0%
London Cosmopolitan	4.9%
England	6.4%

GPs can exclude patients from the calculation of measures in the Quality and Outcomes Framework, to allow practices to pursue the quality improvement agenda and not be penalised, where, for example, patients do not attend for review, or where a medication cannot be prescribed due to a contraindication or side-effect. However, the number of such exceptions varies substantially between practices. In 2010/11, the exception rate in Brent was 5.0%. Within England, the exception rate varied between 2.2% to 7.5% for individual PCTs.

Number and percentage of practices with high exception reporting rates

	Atrial fibrillation	Coronary heart disease				Stroke & TIA	Practices with any high exception rates	Total number of practices
		heart disease	Heart failure	Hyper-tension				
Brent	2	6	1	2	3	9	9	
Brent	22.2%	66.7%	11.1%	22.2%	33.3%	100.0%	9	
London Cosmopolitan	36.7%	51.9%	15.2%	12.7%	31.6%	100.0%	79	
England %	21.3%	47.1%	19.8%	15.0%	36.9%	85.7%	848	

Observed (GP registered) prevalence in 2010/11 versus estimated prevalence in 2011

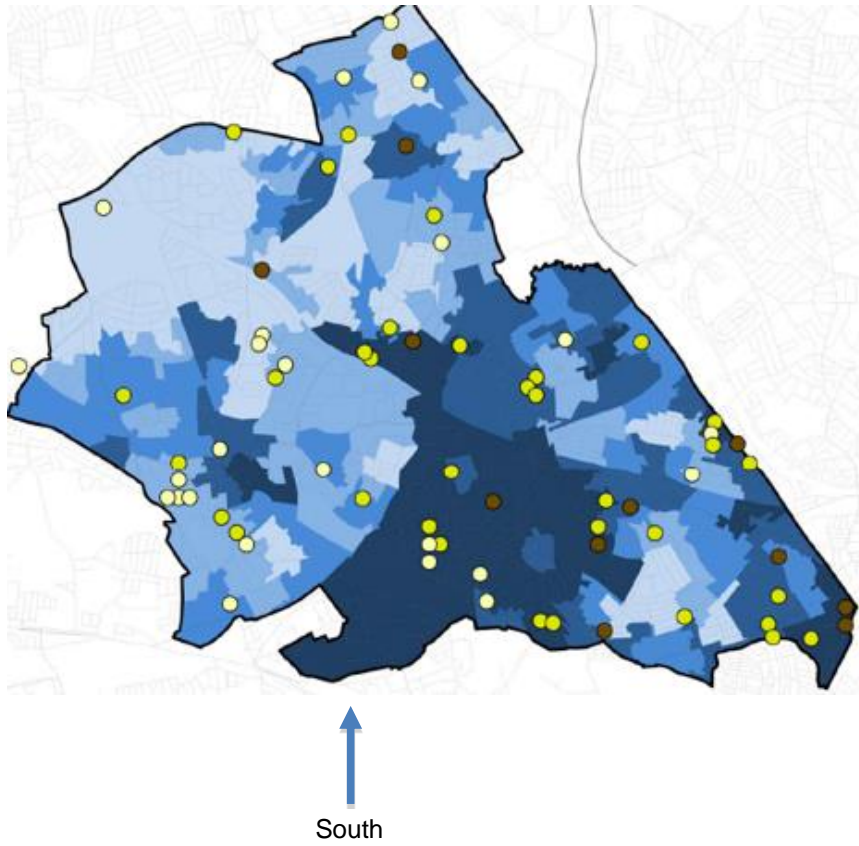


GPs record information on whether their patients have CHD or have a stroke. This information is crude and does not consider population structure. The estimated prevalence is population structure adjusted, but is for the 16+ population, so does not match the all age population of GP registers.

The observed prevalence for CHD in Brent is 43.1% of the estimated prevalence. This compares to 58.7% for England and 39.5% for London Cosmopolitan.

Annex 3

Map showing density of population with COPD (based on mapping based on the sum of the odds ratios for all registered patients living in the post code boundary within Brent). The south (high deprivation locality) is a high risk area.



Annex 4

DATA SOURCE FOR MENTAL HEALTH NEEDS ASSESSMENT

These included:

Nationally available statistics – NHS Information Centre for Health and Social Care.

- ◆ Data at GP practice level, PCT level, Borough level, provider level covering
 - i. Hospital Episode Statistics and Mental Health Minimum Datasets
 - ii. Prescribing data
 - iii. QOF returns
- ◆ Public Health Information service (Health observatories) statistics
- ◆ Data Submissions to national collection services by Providers
- ◆ Data Submissions to commissioners by Providers
- ◆ Research Studies at Local, Regional and National level

Qualitative, Policy and Guidance sources included:

- ◆ NICE
- ◆ Department of Health
- ◆ Sainsbury Centre
- ◆ Kings Fund

Local Information and reports has been provided by:

- ◆ Brent Public Health
- ◆ Brent Mind

- ◆ Central and Northwest London Mental Health Trust (CNWL)
- ◆ Brent Finance Department
- ◆ Brent Carers

A coarse estimated prevalence from mental health profile was based on various sources of information (national and local) was carried out. Information sources were derived from:

- ◆ Adult Psychiatric Morbidity Study 2007 - APMS Reports
- ◆ Dr Foster (HES/SUS data) 2003-10 – Dr Foster data is based on HES data from secondary care, profiled nationally against practice list sizes. Specifically, data was filtered for the London Borough of Brent. These included data on:
 - GP records - QOF information
 - Mental health register (recording adults who have a serious mental health diagnosis)
 - Prescribing evidence (number of prescriptions and rate of prescribing per head of population)

Others included:

- ◆ Indexes of deprivation: IMD (Index of multiple deprivation) – available at ward level
- ◆ Mental Health Needs Index: MINI/Mini2000 and National Psychiatric Morbidity Survey 1993 (NPMS) – available at ward level
- ◆ Local Index of Need (LIN): Borough level

Annex 5

A presentation of the MHNA to the commissioners as part of the study