CHD Prevalence Modelling Briefing Document

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This briefing document explains how the Coronary Heart Disease (CHD) prevalence model has been developed and applied by erpho on behalf of the Public Health Observatories in England. It accompanies the release of updated CHD prevalence estimates in December 2011.

For an overview of prevalence modelling techniques, see APHO Technical Briefing 8: Prevalence Modelling http://www.apho.org.uk/resource/item.aspx?RID=100181

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1 Background

The disparity between coronary heart disease (CHD) prevalence estimates from large surveys, in particular the Health Survey for England (HSfE), and the number of patients diagnosed with CHD and reported in the Quality and Outcomes Framework (QOF) led to demand for a CHD prevalence model at GP practice, Primary Care Trust (PCT) and Local Authority (LA) level that gives an accurate estimate of true prevalence. The Association of Public Health Observatories (APHO) published a simple prevalence model to support development of 2007-08 Local Delivery Plans (1). However, it was acknowledged that this was a crude model and APHO subsequently commissioned Michael Soljak of the Department of Primary Care and Social Medicine, Imperial College, London to develop a more robust model.

This release of CHD prevalence estimates on behalf of the Public Health Observatories of England (the PHO Network) is an update to those published by APHO in September 2008 (PCT/LA) and October 2009 (Practice level).

2 Model Development

Coronary heart disease (CHD) is a relatively common condition, for which there are a number of evidence-based treatments which are known to reduce mortality. The HSfE for 2006 estimates that, based on respondents' self-reports of doctor-diagnosed CHD, the prevalence is about 6.5% in males aged 16+ and 4.0% in females aged 16+ (2), and this increases markedly with age. This prevalence has remained static over the last ten years. However the Quality and Outcomes Framework (QOF) of the GP Contract, covering over 8,000 practices and 53 million patients, shows a GP-registered unadjusted prevalence of only 3.5% (3) (but note that unadjusted prevalence rates show these registers as a percentage of the total practice list size i.e. all ages).

2.1 Background

2.1.1 Defining prevalence

There are differences between various methods of estimating CHD prevalence. The positive predictive value of questionnaire responses such as those used in HSfE to define CHD may be sub-optimal in comparison with 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 (see Table 1) (4). 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.

Table 1: Prevalence of CHD and ECG findings in 25-74 year old men and women (4)

	Sex	25-34 years	35-44 years	45-54 years	55-64 years	65-74 years	25-74 years	Odds ratios (men v women) (95% CI)
Angina pectoris	M	2.5%	3.1%	4.9%	7.9%	13.1%	5.0%	0.51 (0.46 to 0.56) for age < 55 years, 1.00 (0.85 to 1.18) for age>55 years
	F	4.0%	6.7%	8.5%	8.4%	11.9%	6.0%	
History of acute MI	M	0.0%	0.6%	2.4%	6.3%	12.8%	3.6%	2.66 (2.20 to 3.22)
	F	0.1%	0.2%	0.6%	3.0%	5.6%	1.5%	
Minnesota codes I	M	0.4%	0.9%	1.7%	3.0%	5.0%	1.8%	2.02 (1.63 to 2.48)
	F	0.0%	0.4%	0.8%	1.6%	3.3%	0.9%	
Coronary heart disease	M	2.9%	4.3%	7.8%	13.6%	22.7%	8.3%	0.70 (0.64 to 0.77) for age < 55 years 1.22 (1.06 to 1.39) for age > 55 years
	F	4.0%	7.1%	9.6%	11.4%	17.6%	7.6%	
Major ECG findings	M	1.6%	2.8%	4.8%	9.3%	19.0%	6.0%	1.42 (1.27 to 1.57)
	F	0.8%	2.0%	3.4%	6.7%	14.6%	4.3%	
Minor ECG findings	M	3.4%	5.5%	9.2%	16.6%	29.4%	10.4%	1.13 (1.05 to 1.22)
	F	3.5%	3.9%	8.3%	16.1%	29.5%	9.5%	

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% of men with a GP record of angina reported their diagnosis, and 70% of men who reported an angina diagnosis had confirmation of this from the record review (5-6). The prevalence of diagnosed angina in 1992 in these older men was 10.1% according to self-reported history and 8.9% according to GP record review.

Nevertheless, more than half of those in the BRHS with possible myocardial infarction (MI) combined with angina had no resting electrocardiographic evidence of CHD, and half of those with definite myocardial infarction on electrocardiogram had no history of chest pain at any time (7;8). Only half of those with a definite MI on an electrocardiogram could recall a medical diagnosis of CHD (8). Even in severe (grade 2) angina 40% could not recall being told that they had heart disease. Overall, only one in five of those regarded as having CHD was able to recall such a diagnosis having been made by a doctor, and these were likely to be those most severely affected.

However there was substantial agreement between self-report and GP record of angina. 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 Organization (WHO) Rose questionnaires (overall prevalence 11.1%); 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%) (9).

On the other hand the HSfE uses only the latter questionnaire definition in its prevalence estimates. Trends in prevalence are shown in Table 2. These are not dissimilar to the BRHS given the latter only included males aged 40-59.

Table 2: Percentage prevalence of IHD, by HSfE survey year, age and sex (10)

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

2.1.2 Other prevalence surveys

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 understand 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.

A literature search for recent (post-1996) CHD prevalence surveys has been undertaken. The search included only articles in English, which covered groups represented in the UK population in significant numbers. It also excluded surveys which covered CHD risk factor prevalences, or which sampled only sub-populations e.g. those with diabetes.

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 USA, several community surveillance studies have reported on temporal trends in CHD prevalence e.g. the Atherosclerosis Risk in Communities (ARIC) study, the Minnesota Heart Survey, the Olmsted County Study, and the Worcester Heart Attack Study (11). In an analysis of US National Health & Nutrition Survey 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 (12). 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 (13).

Outside the USA, the 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. 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 (14). However the study populations excluded over-65s in whom most CHD occurs.

In a survey of a rural Indian population, CHD was diagnosed on basis of 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 (15). 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 (16). 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 (17).

In the UK Carroll *et al.* used GP records in London and found a prevalence of 8% of men and 5% of women over 44 years of age (18). There was a history of myocardial infarction in 30% of men and 22% of women. Lampe and colleagues examined trends in the prevalence of CHD in men participating in the BRHS (9). 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.

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 (19). 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). Although the decline in incidence had some impact on limiting the increase in prevalence, 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.

2.1.3 Previous CHD prevalence modelling

An epidemiological CHD prevalence model was first developed in the UK to assist case-finding in Sheffield PCTs. Subsequently, English PCTs were required to set targets for CHD case-finding in their 2007-8 Local Delivery Plans negotiated with strategic health authorities. To assist them a simple PCT-based prevalence model was developed rapidly by the Public Health Observatories. Stage 1 of 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. Stage 2 takes account of deprivation levels in different PCTs in England. In the absence of sufficiently precise published data on the relationship between deprivation and CHD prevalence, the model makes the assumption that areas with higher CHD mortality rates have comparably higher prevalence of CHD. Using data for all local authorities in England, a linear relationship was calculated between 2002-04

standardised mortality ratios (SMRs) for CHD and a deprivation score (UV67) derived from the 2001 Census Classification of Deprivation:

$$\text{CHD SMR} = (2.604 \times \text{UV67}) + 25.97$$

Using UV67 scores calculated for each PCT, the above formula gives a multiplying factor for each PCT. For example, a PCT with a UV67 score of 40% (very deprived) has a multiplying factor of 1.3.

APHO accepted that this model is rather crude, and that a prevalence model based on a comprehensive regression model using HSfE data would be more robust. This model was commissioned by APHO from the Department of Primary Care & Social Medicine at Imperial College London.

Congdon has also produced a CHD prevalence model using earlier HSfE data (20). Data from the 1999 and 2003 HSfEs were used to provide model-based rates of CHD prevalence by age, sex, ethnic group, region, and area deprivation category. To take into account the effect of socio-economic factors, the HSfE model gradient over deprivation quintiles was applied to scale area prevalence estimates specific for age, sex and ethnicity. The final stage of the prevalence estimation procedure incorporated proxy information from mortality. To adjust the HSfE-based prevalence rates to take account of interdependence of area mortality and prevalence, prevalence and mortality were taken as joint (i.e. correlated) outcomes in a Bayesian model allowing also for the spatial patterning of both outcomes, using WinBUGs software.

2.2 Methods

2.2.1 Data sources

The HSfE was used as the data source, and the outcome of interest was patient-reported doctor-diagnosed CHD (called IHD in HSfE), which is the variable used for CHD prevalence in HSfE. It was also chosen as on the basis of previous research it appears to be the best **single** proxy for true prevalence. (An alternative would be to combine patient-reported doctor-diagnosed CHD with a positive Rose angina questionnaire as in BRHS.)

Because ethnicity is a known CHD risk factor, it was necessary to use a sample containing data from a large number of ethnic minority respondents. The HSfE 2004 was the last survey to include an ethnic minority boost, and the boost sample was used for the modelling. However there were relatively small numbers of whites in the HSfE 2004 sample, and they were not asked to respond to many questions, including those on CHD, presumably to save resources for the boost itself. The HSfE 2004 boost sample was therefore merged with the HSfE 2003, which was the year with the largest number of identical variables.

Table 3 shows the numbers of respondents in the original HSfE 2004 ethnic boost, and the number in the merged 2003-2004 dataset used for the model where a response for the CHD outcome variable was obtained. Note that it was necessary to collapse two of the HSfE 2003 ethnic group variables in order to use the same classification as HSfE 2004.

Table 3: Ethnic Group Breakdown of HSfE 2004 Dataset & Merged 2003-2004 Dataset

	HSfE 2004			Merged 2003-4 Dataset		
Ethnic Group	**Freq.**	**Percent**	**Cum.**	**Freq.**	**Percent**	**Cum.**
No answer/refused	21	0.21	0.21	16	0.08	0.08
Don't know	3	0.03	0.24	2	0.01	0.08
White	1,597	15.79	16.03	14,575	68.58	68.67
Mixed ethnic group	623	6.16	22.19	308	1.45	70.12
Black or Black British	2,468	24.40	46.59	1,991	9.37	79.48
Asian or Asian British	4,764	47.10	93.69	3,725	17.53	97.01
Any other group	638	6.31	100.00	635	2.99	100.00
Total	10,114	100.00		21,252	100.00	

(Note that there were 34 missing values.)

2.2.2 Model construction: data issues

The choice of variables for original inclusion in the merged dataset included all those known to be CHD risk factors. The variable names and labels are shown in Table 5. The HSfE dataset has a nested or hierarchical structure so three variables related to the sampling strata were included: area (sample point), cluster (stratification level), and hserial (serial number of household). These were used in the model to adjust for clustering of respondents. In the analysis variables cholest and hdlchol were combined to give a lipid ratio.

The bandings of Index of Multiple Deprivation (IMD) 2004 scores were slightly different between the two years, but raw scores were not provided in the dataset so it was necessary to assume identity (see Table 4 below).

The Stata10 software package was used for analysis. All variables were re-coded to drop negative values for estimation purposes (in HSfE various non-response categories are assigned negative values). The methodology applied was multinomial logistic regression with the “cluster” adjustment option for households (see above). For analysis of two categories as here, multinomial logistic regression is reduced to binomial logistic regression. However the reason for not using other logistic regression routines that take into account nested structures is that the other options available in Stata produced an estimation error, probably because of the small percentage of disease-positive respondents in the sample.

Table 4: Index of Multiple Deprivation Banding

	IMD	IMD 2004				
Rank	**Band**	**HSfE 2003**	**HSfE 2004**	**Number**	**%**	**Cum %**
least	1	0.59-8.35	0.55-9.02	3,803	17.87	17.87
	2	8.35-13.72	9.03-14.14	3,573	16.79	34.65
	3	13.72-21.16	14.15-21.17	3,788	17.8	52.45
	4	21.16-34.21	21.18-33.52	4,551	21.38	73.83
most	5	34.21-86.36	33.53-85.69	5,571	26.17	100
			Total	**21,286**	**100**	

The modelling and estimation of the effects of interest was carried out using the mlogit command. 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 Prob (A) = Odds (A) / 1+ Odds (A), for uncommon outcomes such as CHD, RRR can be assumed to be the same as the odds ratio (OR).

For categorical variables the effects are estimated relative to the reference category. Stata uses the first category as reference (baseline OR). Separate baseline odds were estimated for each gender, and also according to ethnicity, age band, area-based deprivation score etc. The model was then used to derive the prevalence ratios for CHD for subjects with various combinations of risk factors in relation to baseline, using stepwise addition of variables. The prevalence in each age group, gender, ethnic group, area of residence and level of deprivation, and smoking status category were derived from the odds, using the formula: prevalence = odds/(1 + odds).

2.2.3 Model construction: interactions between variables

Effect modification or interaction occurs if the effect of one exposure or risk factor on the outcome varies according to the level of another risk factor. This can be tested using a χ^2 test of heterogeneity e.g. Mantel-Haenszel odds ratios, Wald or likelihood tests, or by introducing interaction terms or parameters into the regression model. These allow the effect of one variable to be different in different categories of other variables. In Stata the xi command expands terms containing categorical variables into indicator (also called dummy) variable sets by creating new variables and estimates interactions and main effects.

An initial examination for interactions was carried out using Mantel-Haenszel odds ratios, Wald or likelihood ratio tests. Where these reached significance interaction terms were created as indicator variables in a regression model. None of the interaction terms tested in this way showed consistent statistical significance. The inclusion of interaction terms in the model was therefore rejected.

2.2.4 Model construction: internal validation

Ideally the best prediction should result from utilising the most risk factor information in the regression model. However only a limited range of HSfE variable data is either available or can be estimated at the PCT or LA level, so there is no purpose in including other variables (see Table 5). It was decided to validate 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. In the complete HSfE variables the largest proportion of missing data occurred in those variables related to drug treatment for high blood pressure. Therefore, these variables were excluded from a second version of the model. 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.

LOCAL only used locally available data. Smoking and BMI variables were included on the basis that local synthetic estimates are now available. Local GHQ-12 or Limiting Longstanding Illness score data will be available locally from 2009 and could be included later.

Table 5: Variables Included in Merged Dataset

COMP 1 = "Complete" variables with BP drugs
COMP 2 = "Complete" variables without BP drugs
LOCAL= only using locally available data

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 cardiovascular disease	X	X	
fatvala	fat score	X	X	
fldlchol	LDL cholesterol result (fasting blood data)	X	X	
ftrigl	triglycerides result (fasting)	X	X	
ghq12scr	General Health Questionnaire score- 12 point scale	X	X	
glucval	glucose result (fasting)	X	X	
hdlchol	HDL cholesterol result (blood data)	X	X	
hserial	serial number of household	N/A	N/A	N/A
imd2004	index of multiple deprivation (SOA level)	X	X	X
limitill	limiting longstanding illness	X	X	
nssec8	National Statistics Socioeconomic Class (8 variable)	X	X	
obpdrug	other drugs affecting BP	X		
omdiaval	omron valid mean diastolic BP	X	X	
omsysval	omron valid mean systolic BP	X	X	
porftvg	grouped portions of fruit (inc. orange juice) & veg yesterday	X	X	
roseanmi	angina or MI (Rose angina questionnaire)	N/A	N/A	N/A
sex	sex	X	X	X
topqual3	highest educational level	X	X	

The prediction of the three models was assessed in two ways:

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

2.2.5 Model construction: external validation

An early external validation will be carried out by examining the association between PCT/LA level CHD prevalence estimates and QOF registered prevalence. The regression-based model will also be validated against a prevalence model obtained by Bayesian strategies using WinBUGS. Finally, funding will be sought to undertake a validation of practice-based prevalence estimates against registered prevalence supplemented by active case finding.

2.3 The Model

Table 6 shows the frequency of the CHD outcome by age group in the merged dataset.

Table 6: Respondents Reporting Doctor Diagnosed CHD by Age Band

	Age Band							
	16-24	**25-34**	**35-44**	**45-54**	**55-64**	**65-74**	**75+**	**Total**
Don't know	0	0	0	0	0	1	2	3
Yes	2	6	43	97	262	350	344	1,104
No	2,500	3,855	4,409	3,379	2,792	1,928	1,316	20,179
Total	**2,502**	**3,861**	**4,452**	**3,476**	**3,054**	**2,279**	**1,662**	**21,286**

2.3.1 Local model

The regression model for risk factors for CHD in the "local" prevalence model is shown in Table 7. 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 prevalences in Table 6. 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.

Table 7: Odds Ratios for LOCAL, Model With Only Locally Available Variables

Risk factor	RRR	Std Error	z	P>z	95% Confidence Interval	
Age 25-34	1.000					
Age 35-44	6.867	2.998	4.41	0	2.919	16.157
Age 45-54	19.514	8.217	7.06	0	8.549	44.541
Age 55-64	65.698	27.217	10.1	0	29.169	147.972
Age 65-74	122.864	50.719	11.65	0	54.707	275.936
Age 75+	191.252	79.128	12.7	0	85.003	430.307
Female sex	1.000					
Male sex	1.849	0.139	8.16	0	1.595	2.143
Never smoker	1.000					
Used to smoke occasionally	0.757	0.131	-1.61	0.107	0.539	1.062
Used to smoke regularly	1.484	0.119	4.91	0	1.267	1.737
Current smoker	1.072	0.109	0.68	0.495	0.878	1.308
IMD2004 0.59-8.35	1.000					
IMD2004 8.35-13.72	1.226	0.143	1.75	0.08	0.976	1.541
IMD2004 13.73-21.16	1.350	0.154	2.63	0.009	1.079	1.689

IMD2004 21.17-34.21	1.645	0.183	4.49	0	1.323	2.044
IMD2004 34.22-86.36	2.420	0.256	8.36	0	1.967	2.978
White	1.000					
Mixed	1.264	0.851	0.35	0.727	0.338	4.726
Black/BB	0.763	0.168	-1.23	0.218	0.496	1.173
Asian/AO	1.511	0.243	2.56	0.01	1.102	2.071
Other	0.168	0.170	-1.76	0.079	0.023	1.227

The data was examined for interactions between variables e.g. if there is an interaction between sex and ethnicity, there will be two separate effects of ethnicity on CHD: one for males and another for females. Interactions between local variables were tested initially using Mantel-Haenszel odds ratios, and likelihood ratio and Wald tests. These indicated a possible interaction between sex and ethnicity. However a regression analysis showed significance for only one sex-ethnicity indicator level. Therefore, it was decided not to include an interaction variable in the model.

2.3.2 Complete model excluding treatment for hypertension

The regression model for risk factors for CHD in the "Complete" model excluding hypertension (either systolic or diastolic BPs or treatment for hypertension) is shown in Table 9. This shows well the impact of additional variables on ORs for "local" variables. Body mass index (BMI) is now recognised as an independent risk factor for CVD (although its effect is mediated largely through changes in "physiological" risk factors such as cholesterol:HDL ratio (23-25). Table 8). Although an expected higher OR is found in underweight patients, this is not the case for overweight/obese categories. This may be due to the fact that this is cross-sectional rather than longitudinal data and that the prevalence of obesity in older age groups, where CHD is more common, is very low.

Table 8: CHD prevalence by BMI category

	BMI <18.51	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 CHD	306	6,693	6,502	3,483	3,194	20,178
Total	318	6,880	6,888	3,751	3,445	21,282

Table 9: Odds ratios for complete model excluding treatment for hypertension

Risk factor	RRR	Std Error	z	P>z	95% Confidence Interval	
Age 25-34	1.000					
Age 35-44	7.131	6.891	2.03	0.042	1.073	47.396
Age 45-54	18.447	17.436	3.08	0.002	2.893	117.624
Age 55-64	50.099	46.710	4.2	0	8.058	311.490
Age 65-74	121.219	112.515	5.17	0	19.655	747.578
Age 75+	216.341	200.711	5.8	0	35.111	1333.034
Female sex	1.000					
Male sex	2.258	0.320	5.74	0	1.710	2.981
Never smoker	1.000					
Used to smoke occasionally	1.039	0.320	0.13	0.9	0.569	1.900

Used to smoke regularly	1.512	0.238	2.63	0.009	1.111	2.057
Current smoker	1.114	0.232	0.52	0.604	0.740	1.676
IMD2004 0.59->8.35	1.000					
IMD2004 8.35-13.72	1.215	0.241	0.98	0.325	0.824	1.791
IMD2004 13.73-21.16	1.025	0.217	0.12	0.907	0.677	1.551
IMD2004 21.17-34.21	1.101	0.230	0.46	0.646	0.730	1.659
IMD2004 34.22-86.36	1.424	0.323	1.56	0.119	0.913	2.219
White ethnic group	1.000					
Mixed ethnic group	0.208	0.144	-2.26	0.024	0.054	0.810
Black or Black British ethnic group	0.831	0.355	-0.43	0.666	0.360	1.922
Asian or Asian British ethnic group	1.553	0.436	1.57	0.117	0.896	2.693
Any other ethnic group	1.049	0.665	0.08	0.939	0.303	3.631
BMI <18.51	1.000					
BMI >18.50 & <25	0.538	0.568	-0.59	0.557	0.068	4.268
BMI >25 & BMI <30	0.890	0.936	-0.11	0.912	0.113	6.984
BMI >30 & BMI <40	1.026	1.085	0.02	0.98	0.129	8.143
BMI >40	0.693	0.734	-0.35	0.729	0.087	5.515
Total cholesterol:HDL ratio	0.748	0.049	-4.44	0	0.658	0.850
Diabetes; no	1.000					
Diabetes; yes	0.686	0.144	-1.8	0.072	0.455	1.035
Family History of CVD; yes	1.000					
Family History of CVD; no	0.622	0.103	-2.87	0.004	0.450	0.860
Limiting longstanding illness	1.000					
Non limiting longstanding illness	0.625	0.102	-2.89	0.004	0.454	0.859
No limiting longstanding illness	0.318	0.058	-6.24	0	0.222	0.456
Rose questionnaire: angina and MI	1.000					
Rose questionnaire: neither angina MI	0.007	0.004	-9.06	0	0.002	0.021
Rose questionnaire: angina, but not MI	0.074	0.044	-4.36	0	0.023	0.239
Rose questionnaire: MI, but not angina	0.174	0.096	-3.16	0.002	0.059	0.514
Top qualification nvq4/nvq5/degree	1.000					
Top qualification higher ed below degree	1.479	0.428	1.35	0.176	0.839	2.607
Top qualification nvq3/gce a level equiv	1.122	0.407	0.32	0.751	0.551	2.283
Top qualification nvq2/gce o level equiv	1.227	0.334	0.75	0.453	0.719	2.092
Top qualification nvq1/cse other grade equiv	1.443	0.504	1.05	0.294	0.728	2.861
Top qualification foreign/other	0.875	0.305	-0.38	0.701	0.442	1.732
Top qualification no qualification	1.458	0.355	1.55	0.121	0.905	2.349

2.3.3 Complete model including treatment for hypertension

The regression model for risk factors for CHD in the "complete" model with BP drugs is shown in Table 10. Hypertension is a well-established risk factor, so it is desirable to include variables related to it in a "complete" model. Treatment with ACE inhibitors, beta blockers and calcium blockers are highly 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.

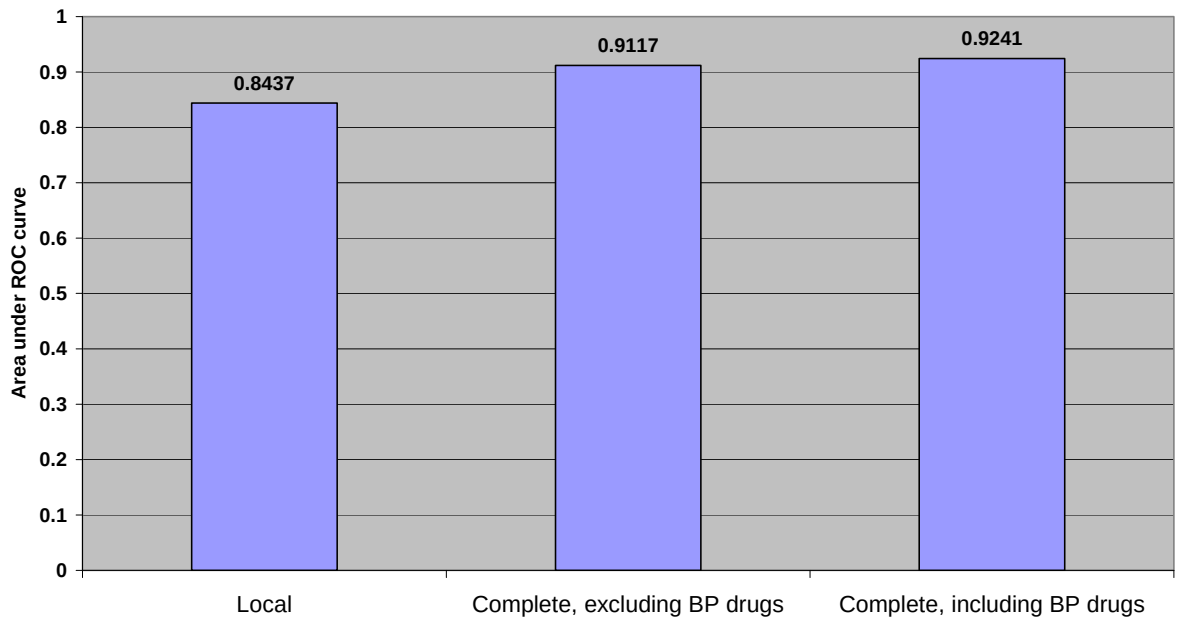
Table 10: Odds ratios for complete model including treatment for hypertension

Risk factor	RRR	Std Error	z	P>z	95% Confidence Interval	
Age 25-34	1.000					
Age 35-44	3.975	3.522	1.56	0.119	0.700	22.566
Age 45-54	8.606	7.259	2.55	0.011	1.647	44.959
Age 55-64	18.559	15.529	3.49	0	3.600	95.676
Age 65-74	36.219	30.104	4.32	0	7.103	184.684
Age 75+	61.082	50.587	4.97	0	12.050	309.643
Female sex	1.000					
Male sex	2.273	0.360	5.18	0	1.666	3.101
Never smoker	1.000					
Used to smoke occasionally	0.989	0.342	-0.03	0.975	0.502	1.948
Used to smoke regularly	1.450	0.246	2.18	0.029	1.039	2.023
Current smoker	1.201	0.278	0.79	0.43	0.762	1.892
IMD2004 0.59->8.35	1.000					
IMD2004 8.35-13.72	1.214	0.273	0.86	0.39	0.781	1.887
IMD2004 13.73-21.16	1.033	0.248	0.13	0.893	0.645	1.653
IMD2004 21.17-34.21	1.174	0.270	0.7	0.485	0.748	1.843
IMD2004 34.22-86.36	1.394	0.366	1.26	0.206	0.833	2.331
White ethnic group						
Mixed ethnic group	0.353	0.240	-1.53	0.125	0.093	1.336
Black or Black British ethnic group	0.304	0.169	-2.14	0.032	0.102	0.902
Asian or Asian British ethnic group	1.316	0.391	0.92	0.356	0.734	2.357
Any other ethnic group	0.894	0.816	-0.12	0.902	0.149	5.345
BMI <18.51	1.000					
BMI >18.50 & <25	0.435	0.441	-0.82	0.411	0.060	3.171
BMI >25 & BMI <30	0.588	0.591	-0.53	0.597	0.082	4.218
BMI >30 & BMI <40	0.680	0.689	-0.38	0.704	0.093	4.954
BMI >40	0.519	0.525	-0.65	0.517	0.072	3.763
Total cholesterol:HDL ratio	0.742	0.053	-4.14	0	0.644	0.854
Diabetes; no	1.000					
Diabetes; yes	1.034	0.250	0.14	0.888	0.645	1.660
Family History of CVD; yes	1.000					
Family History of CVD; no	0.630	0.115	-2.54	0.011	0.441	0.900
Limiting longstanding illness	1.000					
Non limiting longstanding illness	0.592	0.102	-3.05	0.002	0.422	0.829
No limiting longstanding illness	0.578	0.128	-2.48	0.013	0.375	0.892
Rose questionnaire: angina and MI	1.000					
Rose questionnaire: neither angina MI	0.007	0.004	-8.49	0	0.002	0.022
Rose questionnaire: angina, but not MI	0.063	0.039	-4.4	0	0.018	0.215
Rose questionnaire: MI, but not angina	0.182	0.107	-2.91	0.004	0.058	0.574
Top qualification nvq4/nvq5/degree	1.000					
Top qualification higher ed below degree	1.655	0.549	1.52	0.129	0.864	3.173
Top qualification nvq3/gce a level equiv	1.396	0.593	0.79	0.432	0.607	3.211
Top qualification nvq2/gce o level equiv	1.510	0.457	1.36	0.173	0.834	2.733
Top qualification nvq1/cse other grade equiv	1.768	0.665	1.52	0.129	0.847	3.694
Top qualification foreign/other	0.922	0.371	-0.2	0.84	0.419	2.031
Top qualification no qualification	1.496	0.408	1.48	0.14	0.876	2.553
ACE inhibitor: no	1.000					

ACE inhibitor: yes	1.875	0.335	3.52	0	1.321	2.661
Beta blocker: no	1.000					
Beta blocker: yes	3.467	0.570	7.57	0	2.512	4.785
Calcium blocker: no	1.000					
Calcium blocker: yes	2.735	0.468	5.88	0	1.956	3.825
Diuretic: no	1.000					
Diuretic: yes	0.832	0.140	-1.09	0.276	0.598	1.158
Other BP drug: no	1.000					
Other BP drug: yes	0.869	0.279	-0.44	0.662	0.463	1.631

2.3.4 Validation: area under Receiver Operating Characteristics curve (AUROC)

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 (24;25). 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, as in this model (26). 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 (1-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.

AUROCs for the three models tested above were estimated using Stata10. These are shown in the chart below. If both sensitivity and specificity are 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 exceeds this level, although the two more complete models have even better performance.

2.3.5 Validation: model prediction

Another method of assessing performance is to use the regression model to predict the response for each subject. These predictions are called fitted values. The difference between the fitted and the observed values are called residuals. These can then be tabulated against the observed presence of CHD to assess "misclassification" by each model. The results for the three models are shown in table 11.

Table 11: Comparison of predictions by model

Model	Observed	Predicted		Total
		No CHD	CHD	
Local	No CHD	17,614	3	17,617
	CHD	1,098	1	1,099
	Total	**18,712**	**4**	**18,716**
Complete, no BP drugs	No CHD	3,879	64	3,943
	CHD	1	230	219
	Total	**4,109**	**283**	**4,392**
Complete	No CHD	3,883	60	3,943
	CHD	203	246	449
	Total	**4,086**	**306**	**4,392**

3 Application of the Model

In this release, the local model (which includes only those variables that are available at population level i.e. age, sex, ethnicity, smoking status and deprivation score) has been applied to Local Authorities, PCTs and GP Practices to create prevalence estimates of CHD.

Only LAs, PCTs and GP practices with a complete input dataset can be included in the modelling:

- Smoking status is not available for the City of London (00AA) nor the Isles of Scilly (00HF), therefore these LAs, and any GP practices located within them, have been excluded from the CHD model
- All PCTs have complete input data for the CHD model – the Isles of Scilly LA has been assumed to have the same smoking/ex-smoking prevalence as Cornwall UA for the purposes of determining the smoking/ex-smoking prevalence for Cornwall and Isles of Scilly PCT
- 8134 practices have valid data for the CHD model.

Please note that this release does not include an update to the projected prevalence estimates published in November 2008 (27).

3.1 Input data

3.1.1 Populations

3.1.1.1 Local Authorities

The CHD prevalence model uses ONS 2009 mid-year population estimates by ethnic group, age and sex. ONS publishes the data by broad age band (28), but supplied full single year of age data to the PHO Network for the prevalence modelling project. Five ethnic groups were used: white, black, Asian, mixed and other.

3.1.1.2 Primary Care Trusts

The CHD prevalence model uses ONS 2009 mid-year population estimates by ethnic group, age and sex. ONS publishes the data by broad age band (28), but supplied full single year of age data to the PHO Network for the prevalence modelling project. Five ethnic groups were used: white, black, Asian, mixed and other.

3.1.1.3 GP Practices

The CHD prevalence model uses the Attribution dataset (ADS) GP registered populations 2011 by quinary age and sex (29).

The proportion of practice population in ethnic groups was derived from Hospital Episode Statistics (HES) admissions 2008/9-2010/11, excluding admissions with unknown ethnicity (30). The proportions by each ethnic group for each practice were calculated by dividing the count of patients admitted within each ethnic category by the total count of patients admitted for the practice. Due to the nature in which HES data is made available, it was not possible to properly pool the three years of data; subsequently, patients will be counted once in each year they are admitted rather than once over the three year period. 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.

Practices with less than 50 patients admitted and with a valid ethnicity code recorded were excluded.

3.1.2 Smoking status

England regional proportions of smokers and ex-smokers by age and sex are taken from the Jan-Dec 2010 Integrated Household Survey (IHS) accessed via the UK Data Archive (31). These proportions were then adjusted for each LA/PCT using the proportions of smokers from the IHS Apr 2010-Mar 2011 (32), using the following algorithm.

> Local proportion of smokers in age-sex category = regional prevalence of smoking in age-sex category * local overall smoking prevalence / regional overall smoking prevalence

$$S_{asl} = S_{asr} \times \frac{S_l}{S_r}$$

> Local proportion of ex-smokers in age-sex category = regional prevalence of ex-smoking in age-sex category * local overall ex-smoking prevalence / regional overall ex-smoking prevalence

$$E_{asl} = E_{asr} \times \frac{E_l}{E_r}$$

Local proportion of never-smokers in age-sex category = 1 – proportion of ex-smokers in age-sex category – local proportion of smokers in age-sex category

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

Where:

S = proportion of population who are smokers
E = proportion of population who are ex-regular-smokers
N = proportion of population who have never smoked
l = local
r = regional
as = by age and sex

The algorithm requires that PCTs be associated with a single region, however, in some areas, PCTs are split across regional boundaries. These PCTs have been allocated to a unique region (the region containing the majority of the PCT's population) according to Table 12.

Table 12: Allocation of non-coterminous PCTs to regions

PCT Code	PCT	Region Code	Region
5K3	Swindon PCT	K	South West
5LH	Tameside and Glossop PCT	B	North West
5N9	Lincolnshire Teaching PCT	E	East Midlands
5QG	Berkshire East PCT	J	South East

The proportion of smokers for PCTs, based on IHS 2010/11, was calculated by the London Health Observatory using LA population weighting.

The proportion of smokers in City of London (00AA) has been removed because of the low sample size in the IHS – this means that neither City of London, nor any GP practices in the borough, has modelled estimates for CHD prevalence. However, the small sample size was combined with Hackney to estimate smoking prevalence for City and Hackney PCT.

Table 13: Manual corrections to invalid practice postcodes

Practice Code	Corrected Postcode
K81051	PG5 4JA
N81022	SK10 5JH

Practices are assumed to have the smoking status of the LA in which they are located based on the published postcode of the practice (33). Two practices had invalid postcodes; these have been corrected manually (Table 13).

A further three practices had postcodes that were not in the latest file available for mapping postcodes to LAs. The Middle-layer Super Output Area (MSOA) corresponding to the postcode was determined using Neighbourhood Statistics (34), the MSOA then being mapped to LA (Table 14).

Table 14: Manual mapping of postcodes to LAs using Neighbourhood Statistics

Practice Code	LA code
C82020	00BN
M82015	00FN
P84022	00GG

The same smoking prevalence rates are applied across all ethnic categories.

3.1.3 Deprivation

Deprivation scores for LAs, PCTs and GP Practices are taken from IMD 2010 (35).

Deprivation scores for LAs and PCTs are the population weighted average of the combined scores for the Lower-layer Super Output Areas (LSOAs) within those LAs and PCTs respectively. The GP Practice deprivation scores are estimated by taking a weighted average of the IMD scores for each LSOA in which a given practice has registrations (based on the Attribution Data Set 2011) – these scores were provided to erpho by the Department of Health (29).

Five deprivation categories are used in the model. Note that these categories are based on quintiles of IMD score at LSOA level. When the cut-offs are applied to larger geographies, i.e. LA, PCT or Practice, there is not an even distribution across the categories.

3.2 Assumptions of the modelled estimates

It is assumed that:

- the proportion of smokers, ex-smokers and never-smokers is the same across ethnic groups
- for GP practices the distribution of ethnic groups within each age-sex group is the same as the overall distribution of ethnicity for the practice
- the smoking and ex-smoking prevalence in each GP practice is assumed to be equal to the smoking/ex-smoking prevalence of the LA in which the practice is located, based on the postcode of the practice
- the prevalence of CHD in those aged under 25 is zero
- hospital admissions reflect the true ethnic population of GP practices and that there is no systematic bias.

3.3 Using Practice Level Models

It is important to remember that 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

population that has very different smoking pattern to local average) the assumptions of the model may not apply and discrepancies may occur.

Modelled estimates of disease prevalence at practice level are provided for the adult population (aged 16+ years). By assuming that the prevalence of disease is 0% in the under-16s, prevalence estimates for all ages have all been calculated. These all-age prevalences are more closely equivalent to QOF prevalences.

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