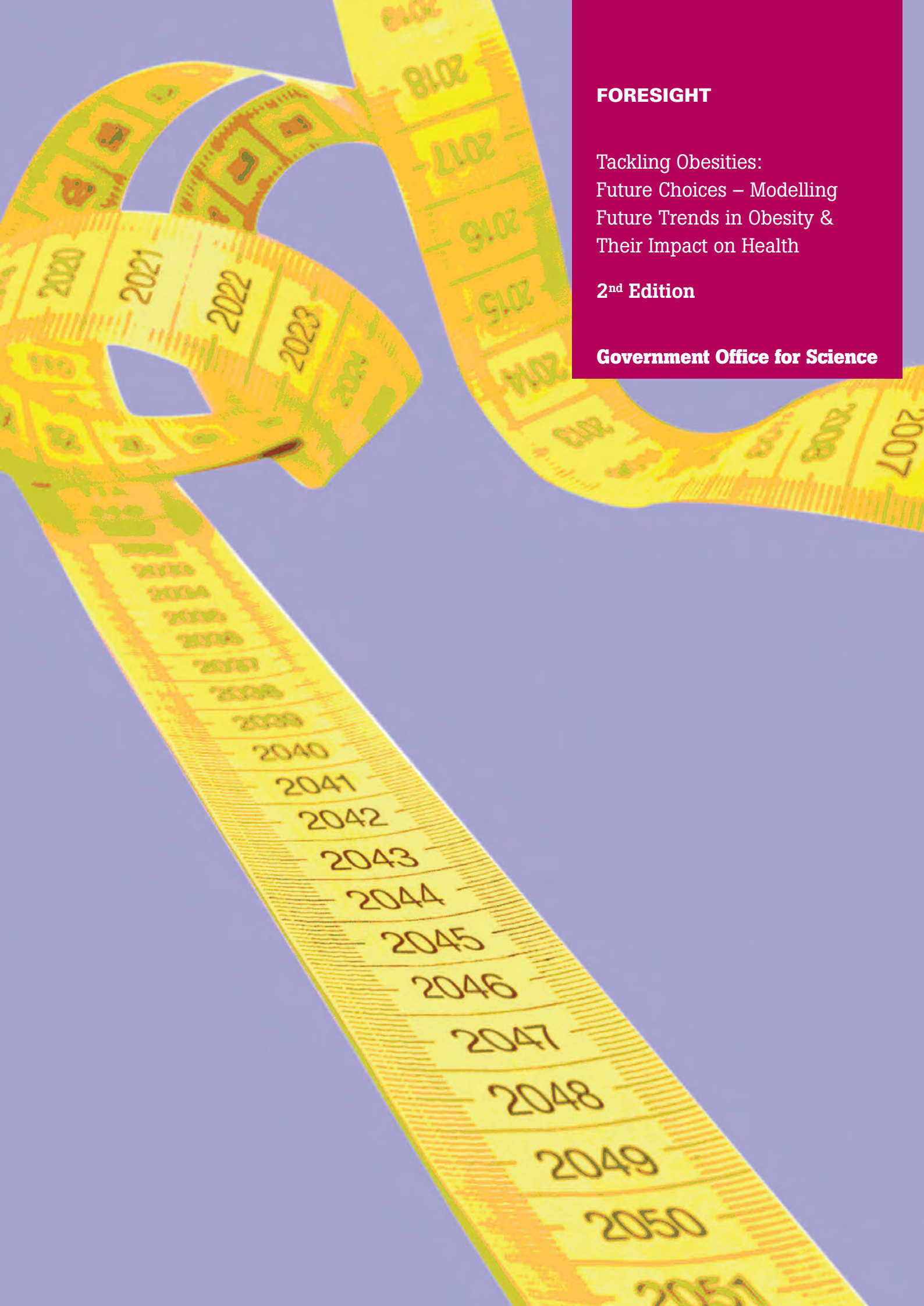

FORESIGHT

Tackling Obesities: Future Choices – Modelling Future Trends in Obesity & Their Impact on Health

2nd Edition

Government Office for Science

Fore*sight*

Tackling Obesities: Future Choices – Modelling Future Trends in Obesity and the Impact on Health

2nd Edition

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This report has been produced by the UK Government's Foresight Programme. Foresight is run by the Government Office for Science under the direction of the Chief Scientific Adviser to HM Government. Foresight creates challenging visions of the future to ensure effective strategies now.

Details of all the reports and papers produced within this Foresight project can be obtained from the Foresight website (www.foresight.gov.uk). Any queries may also be directed through this website.

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Authors's note: Definitions

Body Mass Index is a continuum and the population's BMI distribution is moving inexorably upwards. Risks of disease and incapacity increase with weight gain through the overweight range (BMI 25-29.9) and increase further with obesity (BMI 30+). The quantitative modelling captures increased risk across the spectrum of raised BMI. In the research literature the word 'obesity' is taken to refer either generally to a raised BMI or specifically to a level of BMI greater than or equal to 30. In this report 'obesity' refers to the latter unless the context unambiguously implies the more general and commonly used meaning.

1 Introduction

Rates of obesity and overweight have increased sharply in the UK since the mid-1980s and are projected to continue to rise until 2010[1]. The purpose of this modelling exercise, commissioned by the Foresight programme as part of the Tackling Obesities: Future Choices project, is to project the growth, or otherwise, of obesity rates through to 2050 and to predict the consequences for health, health costs and life expectancy.

A cell-based simulation was planned in order to test the effects of changing the main determinants of obesity on obesity rates. Two developments changed this approach. First, although attempts have been made to model the impacts of policy interventions by a team in Australia[2], the early Foresight systems mapping work[3] clearly demonstrated that the determinants of obesity were too complex for such a modelling process to be reliable. Secondly, close inspection of 12 years of data from the Health Survey for England[1] demonstrated extraordinary order and consistency in obesity trends. The case for making reliable projections from these data, entirely independent of identifying possible determinants, was irresistible, and this is the course that has been pursued. This does not exclude the possibility of incorporating epidemiological impact analysis into any future iterations of this model.

This report considers the following questions in turn:

- What will be the likely distribution of overweight and obesity across the population over the next 40 years?
- What will be the likely health and cost consequences of these extrapolated overweight and obesity trends?
- How much might these consequences be altered by effective interventions to reduce body mass index (BMI) across the population or in targeted subgroups?

Part 1 of the report addresses the likely obesity levels that will be seen in 2050 by supposing that the trends observed between 1993 and 2004 continue until 2050. Part 2 allows wide-ranging changes in the predicted trajectories of BMI rates among any specified subgroups of the population and calculates the consequences in terms of the rates of related diseases, health service costs and life expectancy (other, BMI-unrelated, determinants of these indices remaining constant).

The results reported in Part 2 have been produced using a microsimulation commissioned specifically by the Foresight Tackling Obesities: Future Choices project for this purpose. The microsimulation models the population of England from the mid-1990s to the end of the 21st century. It grows the population from its current age, gender and disease distributions. Its predictions of the future are based on current birth and death rates. Obesity-related disease and death rates are allowed to change, consequent on changing BMI.

2 Background

The Turner Commission on a new pensions settlement[4] for the 21st century noted: 'Poor lifestyle trends such as increasing obesity among young adults and children may in the long-term reduce the increase in life expectancy, but over the next 30 years they could make the burden on the working population worse, since they may reduce the number of healthy working-age people more than they reduce the number of elderly pensioners.'[4] The timescale of the report anticipates pension policy until 2050, when today's youth will be nearing pension entitlement. Its predictions rely on trends in current death rates. It is too early for the current rise in obesity to have had a major impact on these trends and it is certainly too early for rising childhood obesity and its known consequences later in life to have had any impact on them.

Therefore two significant determinants of pension policy are not addressed by the Commission: morbidity in the medium term and life expectancy in the longer term, consequent on currently changing obesity levels. These could change current trends in mortality once predicted obesity trends affect people reaching an age that brings a greater risk of dying. The effect this might have on life expectancy, as well as health service costs is of considerable interest. Rising obesity levels will almost certainly quite dramatically affect rates of disease caused or influenced by obesity.

The most recent Health Survey for England[5] shows that:

One in four adults is now obese.

For men, this figure has nearly doubled since 1993 (13%, rising to 24% in 2004).

For women, the increase is slightly lower (16% rising to 24% from 1993 to 2004).

Using Health Survey for England data and applying International Obesity Task Force (IOTF) definitions[6], around 5% of 11–15-year-old boys and 11% of 11–15-year-old girls are considered to be obese. The more commonly used definitions in the UK from 1990 UK Growth Charts (85th and 95th percentiles) show one in four 11-15-year-old boys as being obese.

Obesity prevalence for the period 1995–2004 increased from 14% to 24% for boys and from 15% to 26% for girls (UK Growth Chart definitions).

Obesity prevalence in boys aged 2–10 increased from 10% in 1995 to 16% in 2004 and in girls from 10% in 1995 to 11% in 2004 (UK Growth Chart definitions). Around 10% of 6–10-year-old boys and girls were shown as obese in 2004 (using IOTF definitions[6]).

These trends are broadly mirrored throughout western Europe, while in the USA similar rises were observed some 6–10 years earlier. In 1986, 1 in 200 adult Americans had a BMI >40 and this is now 1 in 50. The rate of increase in BMI >40 is twice as rapid as for BMI >30. Nonetheless, currently 1 in 5 Americans is now obese (BMI >30).

Obesity is related pathologically to a number of common morbid conditions (see 10 Appendix 1). Most of these conditions are uncommon while young, but become prevalent in middle and later life. Current trends suggest that around 8% of obese 1–2-year-old children will be obese when they become adults, while 80% of children who are obese at age 10–14 will become obese adults, particularly if one their parents is also obese.[7] Adjusting for parental obesity, the odds ratio of an obese 1–2-year-old being obese as an adult is 1:3, i.e. 30% more likely than a non-obese child. While for a child obese at age 15–17 years, the odds ratio is 17 fold. Among very obese children aged 10–14, the unadjusted odds ratio is 44 fold. Clearly, the increasing prevalence of obesity in childhood[8], is very likely to translate into greatly increased levels of obesity among adults, rendering them more susceptible to chronic, life-threatening illness.

In adults, obesity increases the likelihood of type 2 diabetes dramatically – by up to 80 times that of the non-obese. Diabetes is a predisposition for hypertension and coronary heart disease as well other morbidity. Obesity increases the risk of coronary heart disease by 2–3 times and, although BMI may not be a strong independent risk factor, other measures of obesity, such as waist:hip ratio, certainly are. Mortality from cancer among non-smoking obese people is elevated by around 40% compared to non-obese people. Among post-menopausal women, obesity is a significant risk factor for breast cancer. Of course, obesity is associated with many less serious but debilitating conditions such as shortness of breath, back pain, reduced mobility and poor quality of life, as well as an increased psychological and social burden.[9]

Modelling the current effects by evidence-based extrapolation and incorporating and attributing the epidemiology of related diseases allows straightforward estimates to be made for the time development of incidence and death rates over the next 50 years – at least for those conditions most closely associated with obesity. This is done by making basic assumptions about plausible rates of change in childhood obesity rates and tracking individuals into adulthood, using established likelihoods from current trends. These, in turn, can be used to compare predicted illness and mortality rates, under various assumptions, with those that arise from demographic extrapolation from existing current mortality rates (as yet unaffected by rapid, and unprecedented, changes in childhood obesity) during the coming half-century. These figures could be used to revise estimates of the healthy working population, for example, by removing the dead and accounting for the sick in a manner that incorporates known and current changes in obesity.

A great deal of effective public health policy depends on reliable information on what the future might hold, without policy change and with it. This, in turn, depends on our understanding of what health policies are feasible with what consequences, given an understanding of the contemporary causes of obesity, particularly environmental ones. This report contributes to the scientific understanding of the predictable effects of changing obesity. For public health, reliable long-term predictions are vital.

3 Methods and procedures

To predict future levels of obesity in the English population to 2050 and beyond, a two-part modelling process was undertaken. Different, but complementary, methods were used for each part, with each method having its own computer program. The first program, Obesity 1 (obesity_distribution.exe[10]), implements a cross-sectional and regression analysis; the second program, Obesity 2 (obesity.exe[11]), implements a longitudinal analysis using a microsimulation.

Using standard epidemiological methods, the implications (disease incidence and deaths) of these BMI distributions for the future health of the UK population can be estimated. The microsimulation allows for constraints on future BMi distributions to be applied, simulating the effects of successful obesity policy interventions. The consequent changes in obesity-related diseases are predicted. By utilising a basic disease-cost model, the implications for NHS expenditure in the long term can be estimated (see Section 5.4).

The microsimulation model can also be used to estimate – in principle, to the end of this century – period life expectancy for any year and cohort and by gender. This can be done under assumptions of either no change in BMI distribution or predicted changes in the distribution, and with or without specified interventions. Examples of possible implications for life expectancy are described in Section 5.4.

3.1 Cross-sectional analysis

Using the annual datasets of the Health Survey for England 1993–2004, we estimated the distribution of obesity, at all ages, for both genders as well as by ethnicity, social class group and geographical region.

The dataset is large (typically 10,000–20,000 records per year) and, especially for BMI, represents good-quality data. The Obesity 1 program is capable of sorting the dataset and implements non-linear regression analysis methods to derive BMI distributions for the projected English population in future years. The distributions are provided in either graphical format (Figures 1 and 3–8 are examples) or in spreadsheet format and are used as the basis for the longitudinal modelling of the Obesity 2 program.

From an inspection of the data, it is apparent that the growth over time of the percentage of the population belonging to any particular BMI group is approximately linear. Fitting straight lines to this data, however, presents a problem as these would inevitably show some groups exceeding 100% or falling below 0% before 2050, which of course could not occur in real life. A non-linear regression was chosen. The mathematical model that was fitted to the data allows for non-linear extrapolation of existing trends so that:

The observed approximate linearity of trends between 1993 and 2004 is maintained among all groups of age, gender, class etc. (There are very few observed exceptions to this apparent linearity and, if they do exist, are not statistically significant.)

At all times, the total prevalence of all BMI groups forming a part (see below) of the population adds up to 100% (implemented by Equation 2 and Equation 3).

Any approach to 0% or 100% by any BMI group is asymptotic and cannot exceed these limits (implemented by Equation 1).

These three constraints represent the limits of available knowledge about the future, and any further constraints would not therefore be legitimate. A simple and convenient set of slowly varying, monotonic functions that are asymptotic to 0 and 1 (the simulation uses probabilities rather than percentages) are provided by the set:

$$p(t) = \tfrac{1}{2}\left(1 + \tanh(a + bt)\right)$$

Equation 1

for different values of the coefficients {**a**,**b**} and all times, **t**. These functions have a maximum slope of $\tfrac{1}{2}b$ when $\mathbf{a} + \mathbf{b} = \mathbf{0}$; their slope smoothly tends to zero at times far removed from this point.

We used these functions as the basis for the regression analysis as follows: At any time, t, the complete population of interest (for example: 1–5-year-old males, 6–10-year-old females, over-75-year-old Social Class II males, Social Class III females in London) is partitioned according to its BMI into a number, N, of exhaustive, mutually exclusive groups: {$BMI_1(t)$, $BMI_2(t)$,..., $BMI_N(t)$}. At the time, t, the probability that a person chosen at random from the population will have a BMI that falls in group K is calculated[12] from the data as:

$$\bar{p}_K(t) = \frac{\left\|BMI_K(t)\right\|}{\left\|BMI_1(t)\right\| + .. + \left\|BMI_N(t)\right\|}$$

Equation 2

and modelled as:

$$p_K(t) = \frac{\left(1 + \tanh(a_K + b_K t)\right)}{\left(1 + \tanh(a_1 + b_1 t)\right) + .. + \left(1 + \tanh(a_N + b_N t)\right)}$$

Equation 3

The coefficients $\{a_1, b_1, a_2, b_2, \ldots, a_N, b_N\}$ are determined as those that minimise the sum of squares cost function:

$$\chi \equiv \sum_{M=1993}^{M=2004} \left(p_1(t_M) - \bar{p}_1(t_M)\right)^2 + .. + \left(p_N(t_M) - \bar{p}_N(t_M)\right)^2$$

Equation 4

This is achieved by standard non-linear regression techniques.[13]

The two computer programs make use of different numbers of BMI groups – above denoted by N – depending on the context; 3, 5 and 6 are the most common. The UK population's BMI can be sorted according to age, gender, class, ethnicity and geographical region.

A review was undertaken of the epidemiological literature (see Appendix 1), and datasets of risk factors for obesity-related diseases were collated. These datasets take the form of tables of risks and relative risks of factors for acquiring and surviving various obesity-related diseases – type 2 diabetes, coronary heart disease, stroke, arthritis and obesity-related cancer – as functions of age and BMI group. With the derived BMI-distributional data, they form another input to the microsimulation program and are reproduced in 10 Appendix 1. For some diseases, the well-understood mortality experience of incident cases was employed to predict subsequent mortality. For coronary heart disease, for instance, the mortality is often immediate, but for cancer it is usually delayed.

3.2 Microsimulation of obesity growth

In parallel with the more basic cross-sectional calculations, we developed a demonstration computer microsimulation – Obesity 2 (obesity.exe), capable of statistically quantifying both recent history and future changes in obesity levels for the population of England, by age and gender and potentially also by class and ethnicity. The model relies on a stochastic (chance) simulation of contemporary cohorts, ageing till 2050 or beyond, with estimated probabilities of transfer from one BMI level to another with age. The result provides a longitudinal growth model of the UK population with predicted continuous BMI levels across the age range and by gender for each year from 2005 to 2050 (see Section 3.3).
The component disease data sets allow modelled individuals to contract, survive or die from the set of obesity-related diseases. The Obesity 2 program has a wide variety of graphical and tabulated outputs. In addition to incidence, survival and mortality statistics, it calculates predicted attributable illness and death rates consequent on these levels of BMI. All of the figures in this report are examples of the specific outputs available on demand, but there are potentially many others that have not been presented in this report.

The microsimulation operates by generating, according to the best available current statistics, individual people and their children etc., who are born and give birth. They and their BMi scores grow in size to replicate the cross-sectional BMI levels by age and gender predicted by the cross-sectional analysis, Obesity 1. In principle, the model will also allow differential growth according to ethnicity, geographical region or social class, since each individual will have these characteristics according to current distributions. All of the modelled diseases are individually specified with respect to baseline characteristics as well as the relationship they have with different BMI levels. These can be altered and updated by the user.

The microsimulation is capable of modelling the effects of suitable, user-specifiable, constraints on future BMI growth. These simulated outcomes of effective interventions are implemented by anticipating the consequences of interventions on BMI prevalence, and transfers, by age, gender and year. It is therefore possible to explore how levels (incidence, prevalence and mortality) of chronic diseases and associated conditions would develop following such interventions.

It is also possible to calculate the costs of current disease trends to the NHS, and what the long-term impacts – on life expectancy, illness rates and costs – would be if it proved possible to modify current BMI trends in the future.

3.3 Longitudinal BMI model

The Obesity 1 program (obesity_distribution) is used to process the Health Survey for England data and produces a set of probability density distributions $p_{bmi}(\beta|A,S,t)$. These give the probability that a person belonging to age group A, and gender group S, at time t, has a BMI value β. In the program's standard mode of operation, there are 16 age groups, each of five years, covering the age range 0–75+. (This is not essential, simply a convenient and sufficiently discriminating choice.) They are labelled $\{A_0, A_1, \ldots A_{15}\}$. So, for any time cross-section, t, the population's BMI distribution is known. The problem in constructing a longitudinal model is to specify how each individual's BMI changes as that person grows older. It must be specified so that the set of all such individuals has the correct cross-sectional distribution at all times. The solution to this problem, implemented in the microsimulation, works as follows.

It makes use of the cumulative probability distribution functions that are defined in the usual way:

$$F_{bmi}(b|A,S;t) = \int_0^b d\beta \; p_{bmi}(\beta|A,S;t)$$

Equation 5

F represents the probability that the person has a BMI of at least b. The implemented rule for BMI growth is most easily stated in five-year steps. In this time, a person originally in some age group A_k at time t will move to the next age group up A_{k+1} at time t+5. The rule is that the person will then have a BMI b′, which is the solution to the equation

$$F_{bmi}\left(b' \middle| A_{k+1}, S; t+5\right) = F_{bmi}\left(b \middle| A_k, S; t\right)$$

Equation 6

Equation 6 guarantees that the population will have the correct predicted cross-sectional distribution at time t+5, provided that it has the predicted distribution at time t; it is solved for b′ in terms of b by constructing the inverse function, denoted F^{-1}, of the cumulative distribution function F:

$$b' = F_{bmi}^{-1}\left(F_{bmi}\left(b \middle| A_k, S; t\right) \middle| A_{k+1}, S; t+5\right)$$

Equation 7

The BMI growth equation, Equation 7, is integral to the simulation – it is needed for every year of a simulated person's life. In a typical Monte Carlo run (a simulation technique that uses random numbers to model some sort of a process and works particularly well when the process is one where the underlying probabilities are known but the results are more difficult to determine), Equation 7 will need to be solved ~100 million times. For this reason, the solution is implemented in RAM as a large matrix.

3.4 Disease cost model

Many of the results of this report are for total NHS costs. These are all based on the simple cost model described below.

In any year, the total NHS cost for the disease D is denoted C_D(year). If the prevalence of the disease is denoted P_D(year), we assume a simple relationship between the two of them form:

$$C_D\left(\text{year}\right) = \kappa P_D(\text{year})$$

Equation 8

for some constant κ.

For each of the trial years, the microsimulation records the prevalence of each disease – call it P_D(year|trial) for the disease D – and the trial population size for

that year, $N_{pop}(year|trial)$. It is assumed that the prevalence in the whole population, $N_{pop}(year)$, is a simple scaling of the trial prevalence, i.e.:

$$C_D(year) = \kappa P_D(year) = \lambda \frac{N_{pop}(year) P_D(year|trial)}{N_{pop}(year|trial)}$$

Equation 9

for some other constant λ. By comparing any trial year to some initial year, year0, the total disease cost in any year is given as:

$$\frac{C_D(year)}{C_D(year0)} = \frac{N_{pop}(year)}{N_{pop}(year0)} \frac{N_{pop}(year0|trial)}{N_{pop}(year|trial)} \frac{P_D(year|trial)}{P_D(year0|trial)}$$

Equation 10

The total NHS costs in year0 (in reality, the year 2004) are input to the simulation from Government statistics (see Table 12).

3.5 Software

In order that the project should have a life cycle greater than ten years, it was essential to build its computing base from tested and enduring components.

The program's source code was written in C++ (Compiler: C++ Builder v.6.0 or later, Borland Software Corporation, 1903–2007) and compiled to run on IBM-compatible PCs under recent versions (Me, NT, XP, Vista) of the Microsoft Windows operating system.

The program's necessary use of Monte Carlo analysis places a substantial burden on the host computer. The minimum computing requirements are for a single 2 GHz processor with access to more than 500 M bytes RAM. The length of time taken to run the program is largely dictated by the host processor's speed; a faster machine will shorten run-times. A typical run of 10 million individuals takes around 15 minutes on a current laptop with average specification.

3.6 Statistical error analysis

There are two significant sources of statistical error deriving, independently, on the one hand, from the regression analysis implemented in the Obesity 1 (obesity _distribution.exe) program, and, on the other, from the Monte Carlo analysis implemented in the Obesity 2 (obesity.exe) program. Outputs from Obesity 2 will suffer from both types of error.

The program Obesity 1 (obesity_distribution) fits appropriate, non-linear asymptotic, regression curves to the BMI data for the period 1993–2004 taken from the Health Survey for England. On this basis, it extrapolates possible future proportions of the population with BMIs usually in the following five categories:

BMI_5: >40 (morbidly obese)

BMI_4: 30–40 (obese)

BMI_3: 25–30 (overweight)

BMI_2: 20–25 (appropriate)

BMI_1: <20 (underweight)

Similar extrapolations can also be performed for categories of childhood obesity defined by the IOTF[6]), and, although the data are less complete, they can also be undertaken for waist:hip ratio measurements.

The Obesity 1 (obesity_distribution.exe) program predicts BMI in any of the above classifications from the Health Survey for England dataset for essentially any categorisation of age, gender, ethnicity, social class and geographical region. It can do this for any user-specified level of confidence and represent the output either graphically or in spreadsheet format.

The microsimulation Obesity 2 program inputs Obesity 1 obesity_distribution outputs having known, quantified errors. Obesity 2 produces its output by performing a user-specified number of Monte Carlo trials. Output from these trials will suffer a variance, having a characteristic behaviour that will vary as the inverse of the number of trials. Most of the results shown in this report are derived from runs of the program having 1 million trials. This appears to be sufficient to achieve satisfactory convergence of the output for most purposes.

Both sources of error are tabulated by Obesity 2 in its error analysis methods.

3.7 Using the programs

Both the cross-sectional and longitudinal analyses are readily accomplished using the programs written for this project. Cross-sectional analyses of the Health Survey for England, including data from 2005 onwards and yet to be published, can be performed for any subset of age, gender, ethnicity, class and geographical region, with generous options for the specification of time periods for prediction and varying categories of BMI classification. Levels of confidence intervals can be pre-specified and categories of BMI can be plotted individually, categorised by class and gender, for example. This enables the exploration of the trends in obesity as each year of data is added to the data set or as specific hypotheses emerge. Therefore the possibility of monitoring changing trends is both eminently feasible and efficient.

The longitudinal analyses using microsimulation are similarly flexible and easy to use. Runs are specified at the start, with user input parameters on start and finish calendar times and the specification of years for cross-sectional analyses. The number of runs and the range of simulations (with detailed input parameters including age, class ethnicity etc.) to be investigated are specified by the user. Inputs specifying baseline disease incidence (by age and gender) and risks associated with obesity can be readily altered.

The output is either shown on the screen or transferred to files in various user-specified ways. These can be in the form of images to be copied and pasted or Excel files for further analyses of incidence, mortality, costs, by diseases as well as obesity levels, life expectancy etc. of the simulated populations. These data are typically saved by age, calendar year and disease for more detailed further analysis.

Part One: Using the Obesity 1 Program

4 Obesity trends: findings

Introduction

Data from the Health Survey for England were divided by various age (and other) groups. Firstly, they were categorised into three age groups: those under 20 (children and adolescents), adults aged 21–60 and those over 60. These broad age groups were then subdivided into ten-year categories.

The Health of the Nation[14] national strategy for public health in England, published in 1992, set a target to reduce the proportion of obese men aged 16–64 from 7% in 1986/87 to 6% in 2005. The target set for women was from 12% in 1986/87 to 8% in 2005. A review of this target by the National Audit Office in 1996[15] showed that the proportion of obese people in the population had continued to rise. Childhood obesity was also singled out as an increasing concern. A target for childhood obesity was introduced to 'halt the year-on-year rise in obesity among children aged under 11 by 2010, in the context of a broader strategy to tackle obesity in the population as a whole'.[15] There is, as yet, almost no evidence that these policies have changed the trajectory of obesity growth.

4.1 Gender

Our extrapolations indicate that on current trends, by 2015, 36% of males and 28% of females will be obese. By 2025, 47% and 36% respectively are estimated to be obese, and by 2050 the proportion of the population that is obese will be 60% of males and 50% of females. The 95% confidence limits on these latter estimates are 55–65% for males and 45–55% for females. By 2021, the proportion of overweight adult men will be equal to the proportion of obese men – around 43% in each group. At this time, the proportion of men with BMI <25 will be 13% (see Figure 1). For women, Figure 2 suggests the proportion of overweight and obese adults will be equal in 2024 (both 35%). At the same time, only a quarter of adult women will have a 'healthy' BMI.

Figure 1: Probability of males aged 21–60 belonging to a specific BMI group in a given year [95% confidence limits]

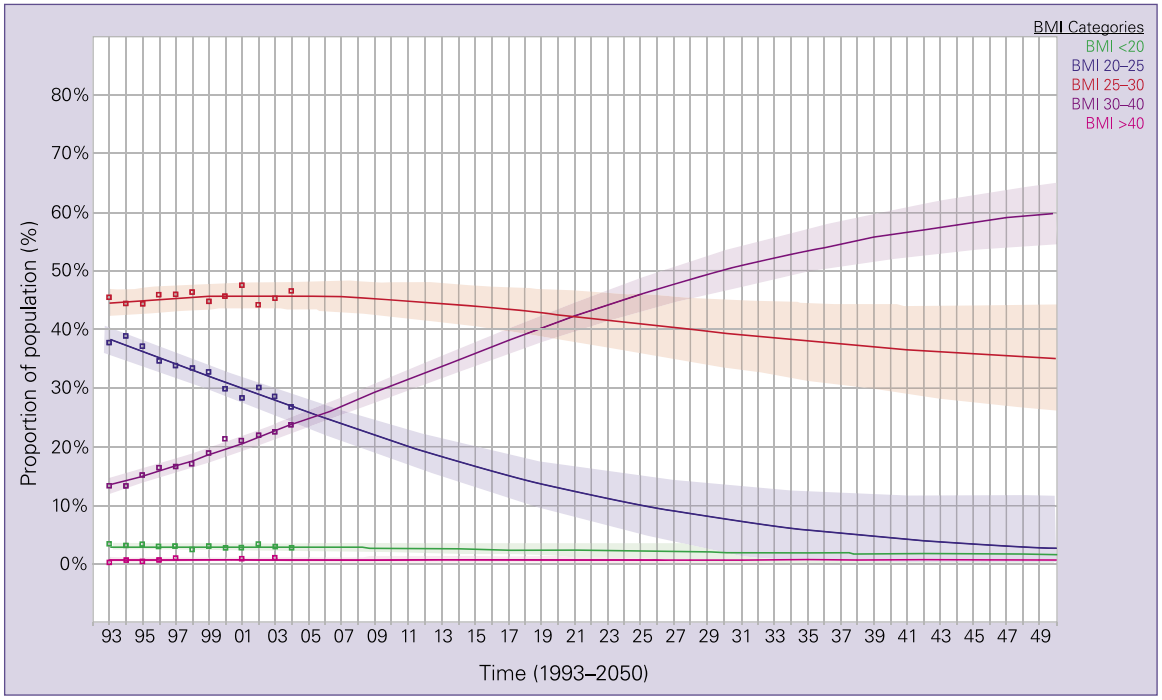
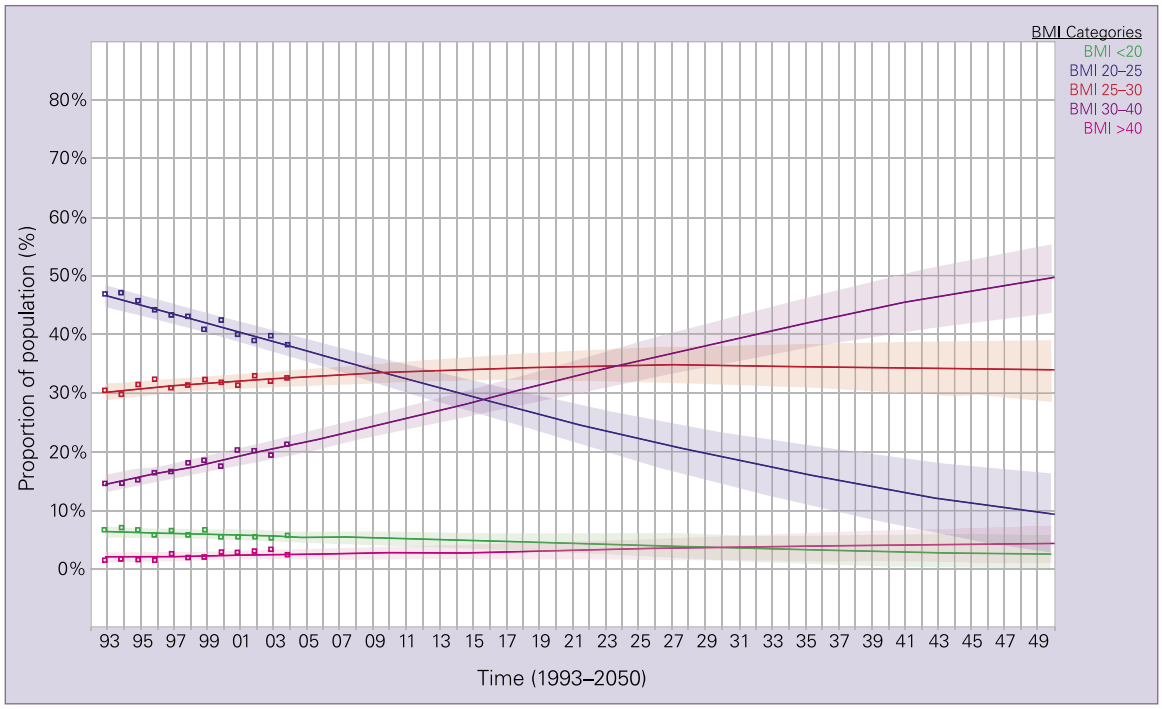


Figure 2: Probability of females aged 21–60 belonging to a specific BMI group in a given year [95% confidence limits]



4.2 Morbid obesity

While the main group of interest are those classified as obese (BMI 30–40), the morbidly obese (those with BMI >40) are also of considerable concern. Currently, about 4% of US males and nearly 6% of females are classified as morbidly obese, while comparable figures for England are 1% and 3% respectively. Predicting rates of morbid obesity from the Health Survey for England is problematic, but some have made extrapolations[16]. These show the proportion of English males who are morbidly obese reaching nearly 3% by 2030; this being 6% for females. Our figures, by contrast, suggest 1% for males and 4% for females by 2050. These estimates are somewhat model-dependent, however, since straight-line extrapolation of the proportion of adult men with BMI >40 predicts a level of around 3% in 2050. But, clearly, that makes no allowance for complementary rates among other obesity groups (similar straight-line extrapolations predict –16% among the BMI 20–25 group in 2050, for example). It is certainly too early to see a strong trend among men in this BMI group, although there is evidence of a slight trend among women. These analyses precariously suggest that the population of England may not ever achieve even current US levels of morbid obesity. The analyses may, on the other hand, represent the consequence of insufficient time to reliably document sufficient growth in the prevalence of morbid obesity. In the USA, there is clear evidence for a continuing upward linear trend, with 5% of males and 10% of females predicted to be morbidly obese by 2030.

4.3 Age

There is some controversy concerning the measurement of obesity among children because of the difficulties of predicting growth rates. The microsimulation is designed to utilise both the official Department of Health measure of childhood obesity and the measure developed on behalf of the IOTF[6]. Utilising the IOTF definition, the proportion of those who are obese in the under-20 age group, will rise to approximately 10% by 2015. By 2025, around 14% of the under-20s (with a slightly higher percentage in females than males) will be obese, and by 2050 this will be around 25%. There is evidence that, among children aged 6–10 years, boys will be more obese than girls, with an estimate of 50% (95% confidence interval range 35–61%) being obese by 2050, compared with 20% (95%confidence interval range 5–40%) of girls. Among children aged 11–15, the prediction is different: 23% (95% confidence interval range 17–29%) for boys and 37% (95% confidence interval range 25–47%) for girls. See figures 3 and 4.

Figure 3: Probability of males aged 6–10 belonging to a specific BMI group in a given year (IOTF definition[6]) [95% confidence limits]

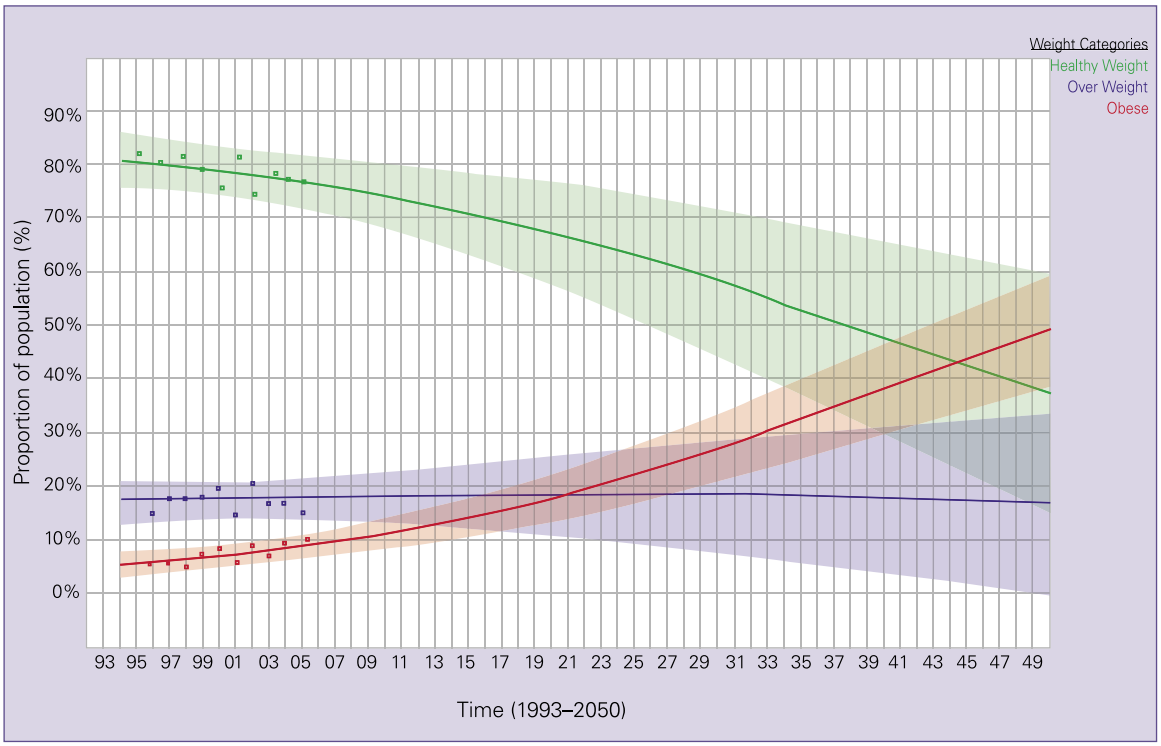
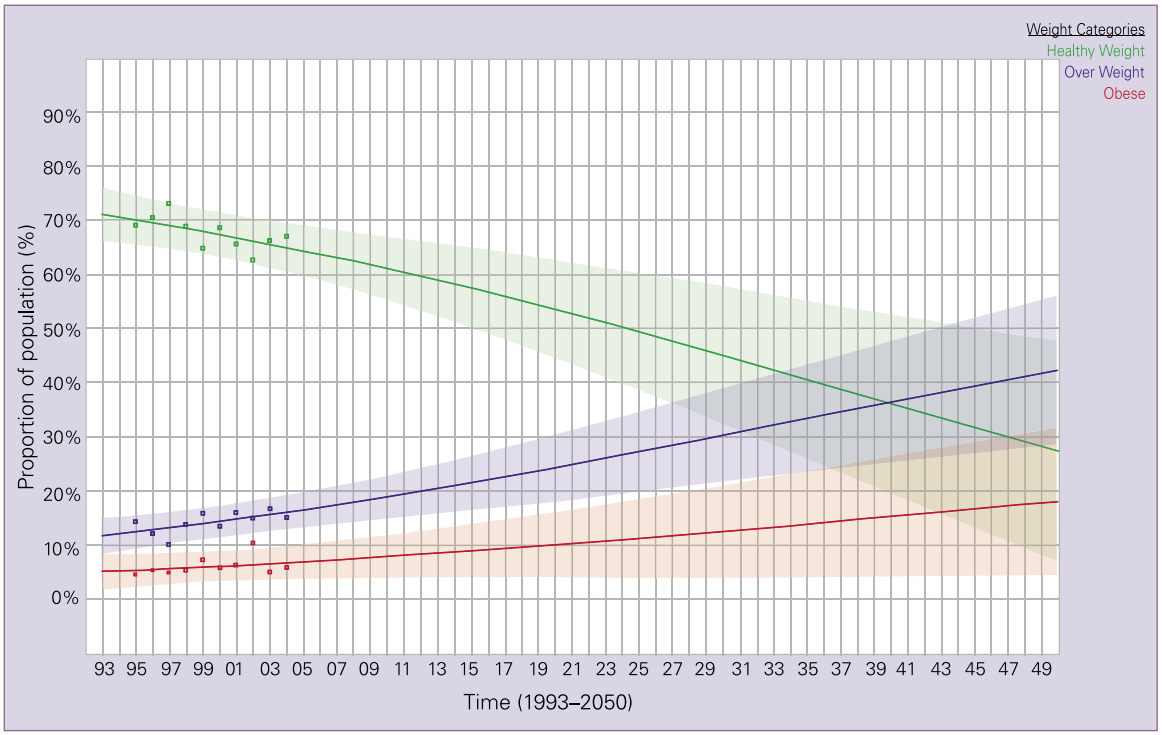


Figure 4: Probability of females aged 6–10 belonging to a specific BMI group in a given year (IOTF definition[6]) [95% confidence limits]



For adults, the predicted rates of change in obesity levels over time are not strongly influenced by ten-year age groupings, which have specific prevailing rates in 2007, but the growth is apparently relatively homogeneous, and higher than for the under-20s. For simplicity, the point estimate of prevalence of obesity predicted at 2050 is shown in Table 1.

Table 1: Percentage of age-specific population obese at 2007 and 2050

Age	Males (%)		Females (%)	
	2007	2050	2007	2050
1–20(IOTF)	7	26	10	26
21–30	15	42	13	30
31–40	28	65	22	47
41–50	26	55	23	52
51–60	32	65	27	49
61–70	31	64	32	59
71–80	28	63	27	44

4.4 Social class

There is similar homogeneity when we examine the impact of social class (figures 5 and 6). Social Class I females appear to be the only major exception for adults (aged 21–60), where the prediction from the Health Survey for England data suggests a 15% level of obesity in 2050, compared to Social Class V women, for whom the prediction is 62%. Clearly, in these subgroups, the 95% confidence limits are wider (e.g. +/– 10%). Apart from the Social Class I effect among women, there is no evidence for a widening of social class difference, and the gap between the remaining social classes is predicted to remain static, as it is among men. The proportion of Social Class I men who are obese is also predicted to be lower than that of Social Class V but not by the same extent as Social Class I women (52% by 2050, compared to 60% by 2050 in Social Class V). It can be readily seen that the stability in obesity among Social Class I women is contingent on the data over relatively few years, which emphasises the insecurity of the data among subgroups.

Figure 5: Proportion of adult males with BMI 30–40 in a given year, by social class

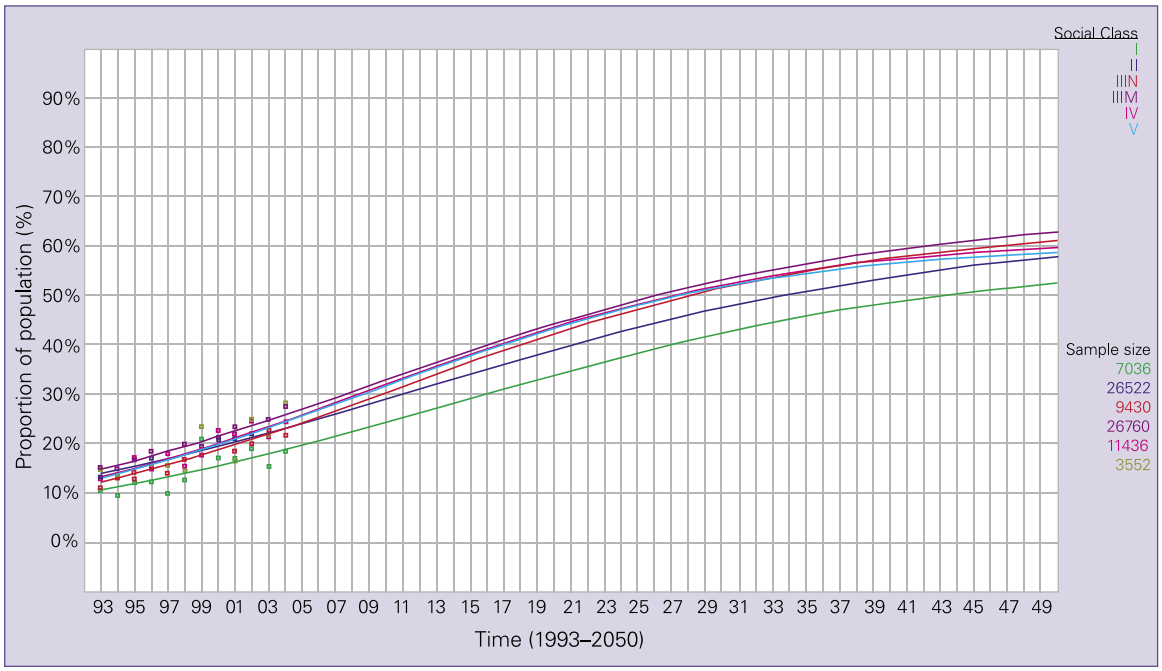
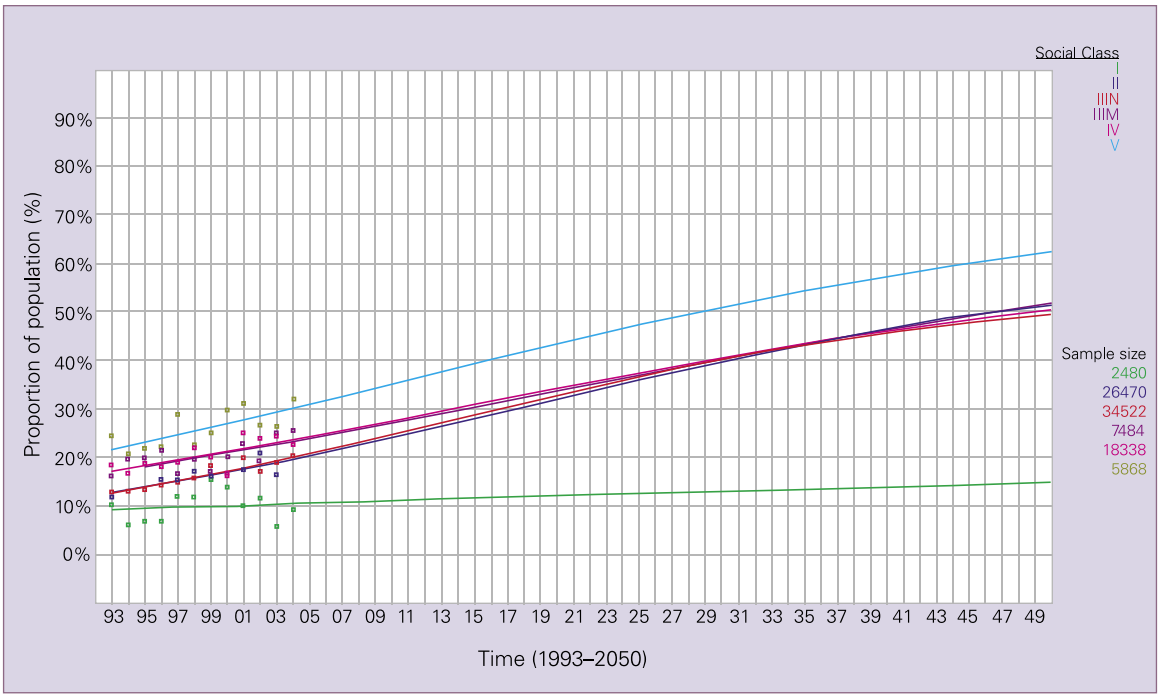


Figure 6: Proportion of adult females with BMI 30–40 in a given year, by social class



4.5 Ethnicity

Ethnicity appears to be a major determinant in obesity trends, though it must be noted that data sets for some ethnic groups in the survey are relatively small (Table 2). Black Caribbean and Chinese groups appear to be becoming less obese, with trends suggesting a proportion of just 3% being obese by 2050. Bangladeshi men are also becoming less obese, but this is not the case with Bangladeshi women, although the increase is modest here. Indian men and women demonstrate very slight increases, while black African women and Pakistani men and women appear to share the trend (though slightly attenuated) of the white population.

Table 2: Predicted percentage of population obese at 2006 and 2050, by ethnic group

Ethnic group	Males (%)		Females (%)		Number of Health Survey for England records, 1993–2004
	2006	2050	2006	2050	
White	26	63	23	57	139,914
Black Caribbean	18	3	14	1	1,458
Black African	17	37	30	50	1,036
Indian	12	23	16	18	2,848
Pakistani	16	50	22	50	2,236
Bangladeshi	26	17	24	30	836
Chinese	3	1	3	1	182

4.6 Regional variations

Looking at regional differences in obesity among adults aged 21–60 (Figures 7 and 8), the trend among women in Yorkshire and Humberside appears particularly steep, with obesity levels reaching 70% (95% confidence limits: 58–82%) by 2050, compared with the other extreme, the south-west of England, where the predicted level is 7%; a reduction from 20% currently. Among men, again, those in Yorkshire and Humberside, along with men in the West Midlands and the north-east of England, have a high predicted growth rate which rises to around 70%. There are no predicted declines in rates of obesity among men, but in the London region, the predicted rise is only to 38% for men, with the same increase for women.

Figure 7: Proportion of adult males with BMI 30–40 in a given year, by geographical region. Guide to abbreviations: (NE) North-east England, (NW) North-west England, (YHum) Yorkshire and Humberside, (EMid) East Midlands, (WMid) West Midlands, (EoE) East of England, (Lon) London, (SE) South-east England, (SW) South-west England.

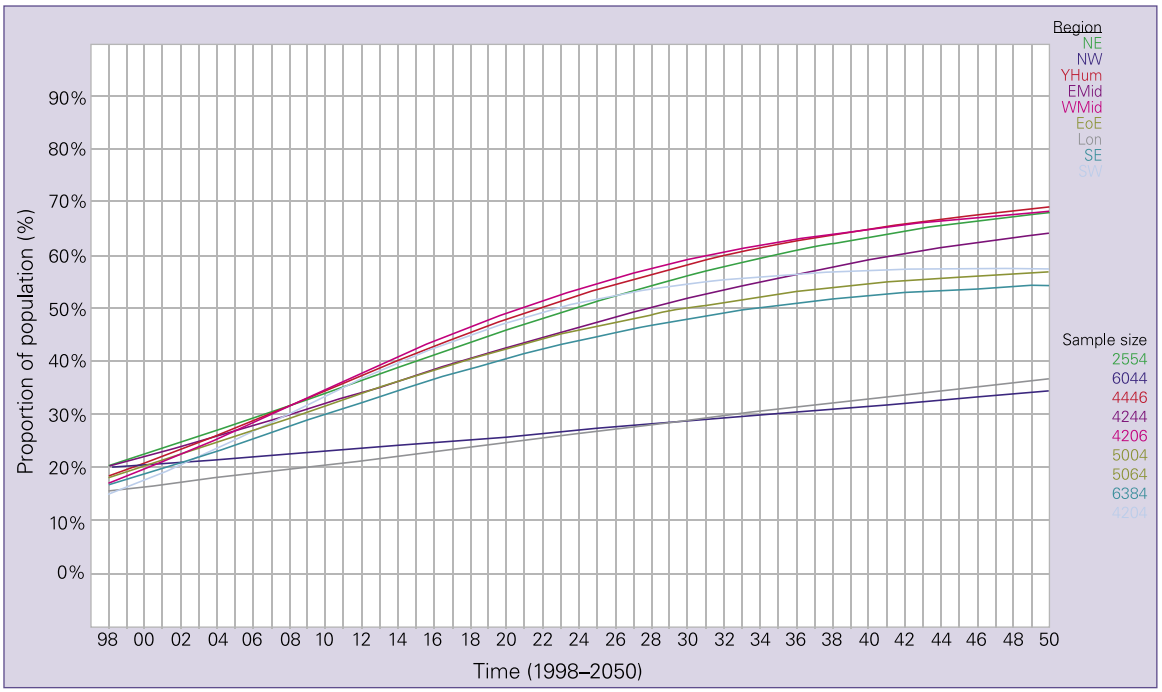
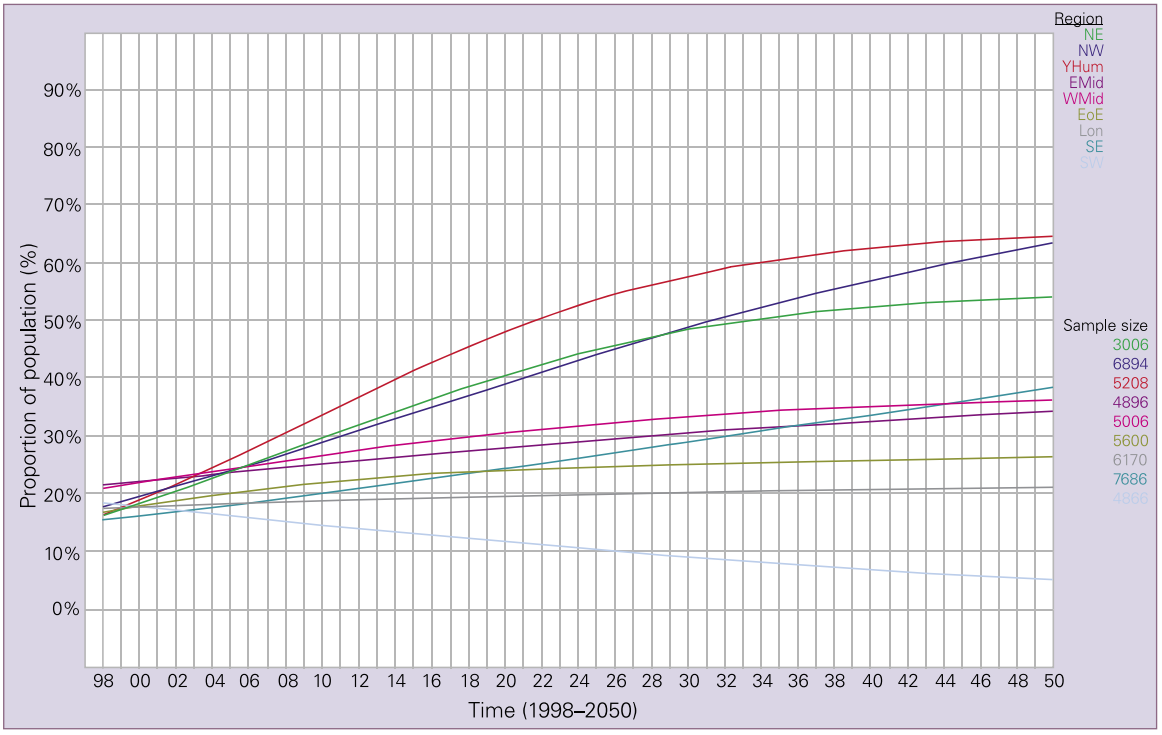


Figure 8: Proportion of adult females with BMI 30–40 in a given year, by geographical region. Guide to abbreviations: (NE) North-east England, (NW) North-west England, (YHum) Yorkshire and Humberside, (EMid) East Midlands, (WMid) West Midlands, (EoE) East of England, (Lon) London, (SE) South-east England, (SW) South-west England.

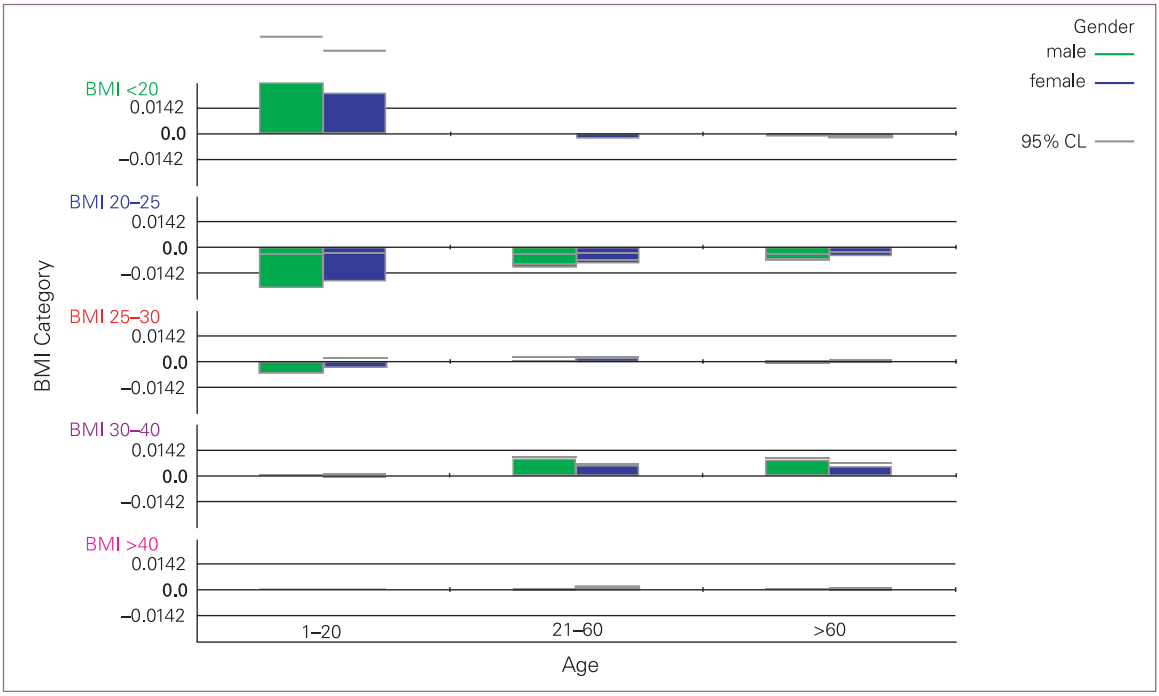


4.7 Interactions

To what extent is the rise in obesity in one region, which is apparently different from the rise in another, attributable to social class or age effects? Examining policy-relevant interactions relating to the causes of increases in levels of obesity in the Health Survey for England data is problematic because, in spite of the large size of the survey, inevitably the number of subjects in subgroups defined by age, class, ethnicity and/or geographical region will be small and therefore vulnerable to sampling error. We have addressed this problem by routinely plotting the slope (a summary measure of the trend with time) of a fitted line with its confidence limits by defined subgroup so that any important interaction could be identified. Several of these are illustrated in Figure 9.

The scale in these graphs is set by the size of the slopes and enables ready comparison across groups, but not between these graphs. Usually, only one confidence limit is displayed, while a missing confidence limit usually implies very narrow limits that can't be differentiated from the estimate of slope. Occasionally, one wide limit is omitted when it cannot be displayed within the scale and is unlikely to have any precision in the estimate of slope. Clearly, where needed, the sampling error of all estimates is available from each run by downloading the outputs. In Figure 9, there is evidence of a higher rate of increase in obesity in adults compared to children of both genders. Here, the rise among females is less than that among males.

Figure 9: The slope of trends with time in BMI groups, by age and gender



In Figure 10, the apparent lower growth of obesity with time in Social Class I women is probably an artefact of small numbers in this group, since the confidence limits are wide and can't be differentiated statistically from the slope in other groups. There is weak evidence of a social class gradient in the slope for men that is of interest.

Figure 11 shows that the evidence for systematic differences by gender and geographical region is weak since no region shows any significant difference from a common slope across both genders. There is, for example, evidence of a significant difference in slope among overweight women living in the south-west of England when compared to other regions.

Figure 10: The slopes of trends with time in BMI groups, by social class and gender

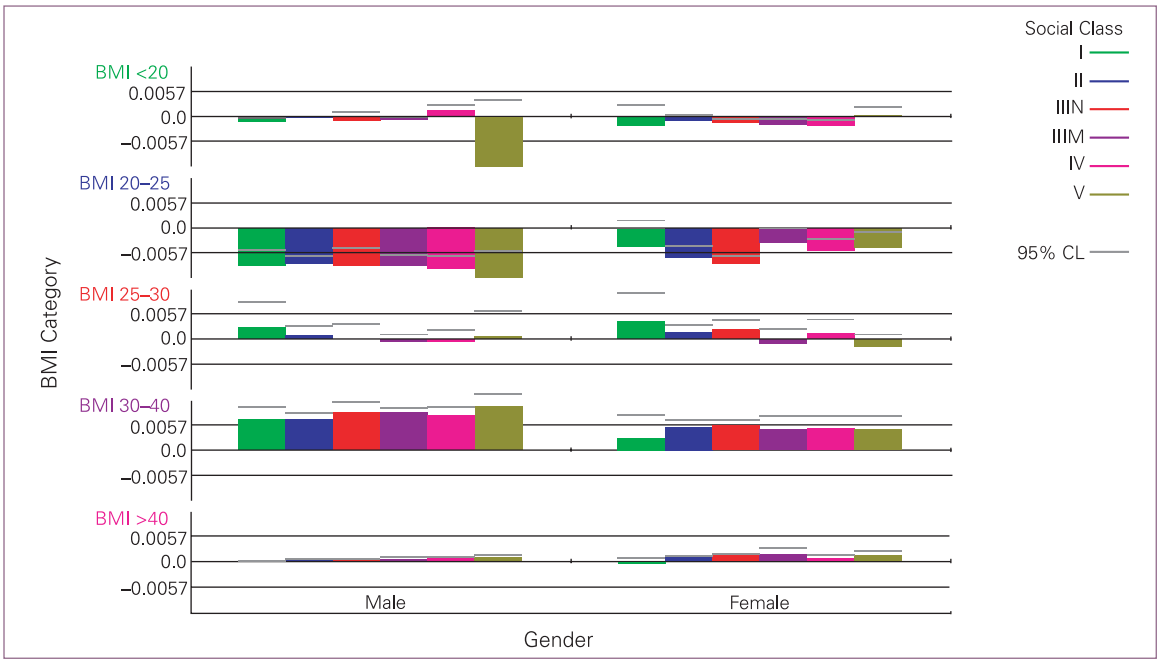
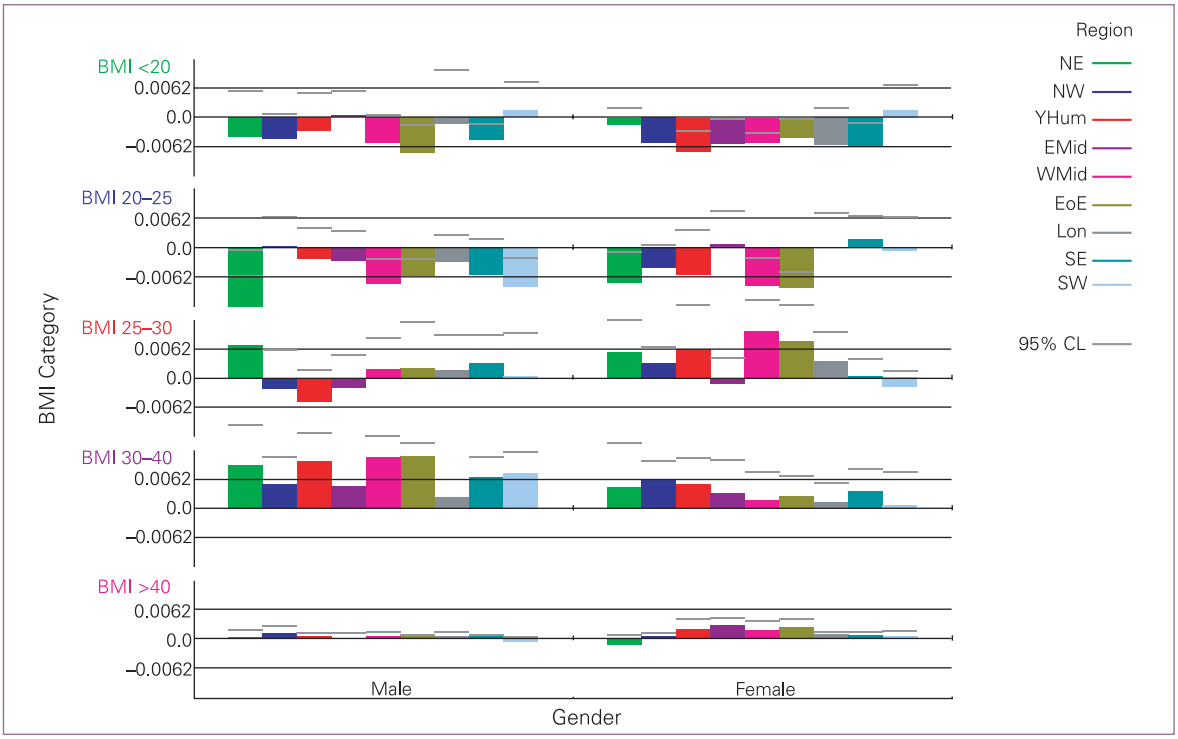


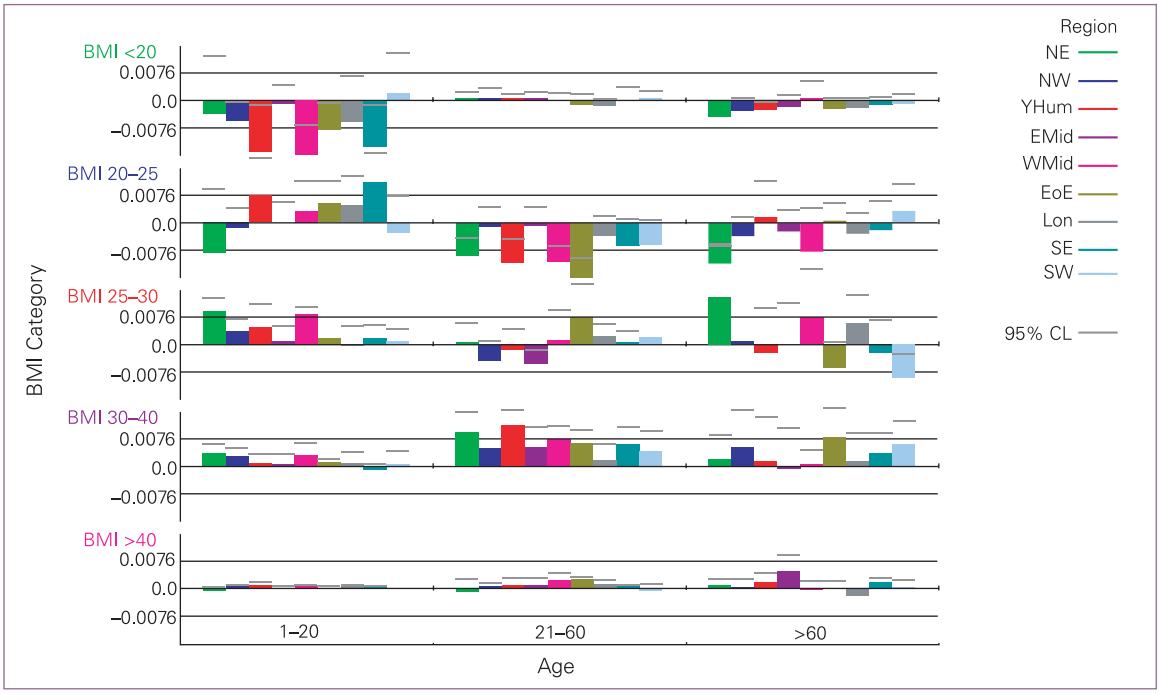
Figure 11: The slopes of trends with time in BMI groups, by geographical region and gender. Guide to abbreviations: (NE) North-east England, (NW) North-west England, (YHum) Yorkshire and Humberside, (EMid) East Midlands, (WMid) West Midlands, (EoE) East of England, (Lon) London, (SE) South-east England, (SW) South-west England.



Again, the numbers in each subgroup are too few to establish any difference in obesity trends for any age group in particular regions. While Figure 9 demonstrates greater growth in the proportion of those over 21 having BMI >30 than in younger people (in whom BMI may not be the most sensitive measure), Figure 12 does not demonstrate any significant regional effect.

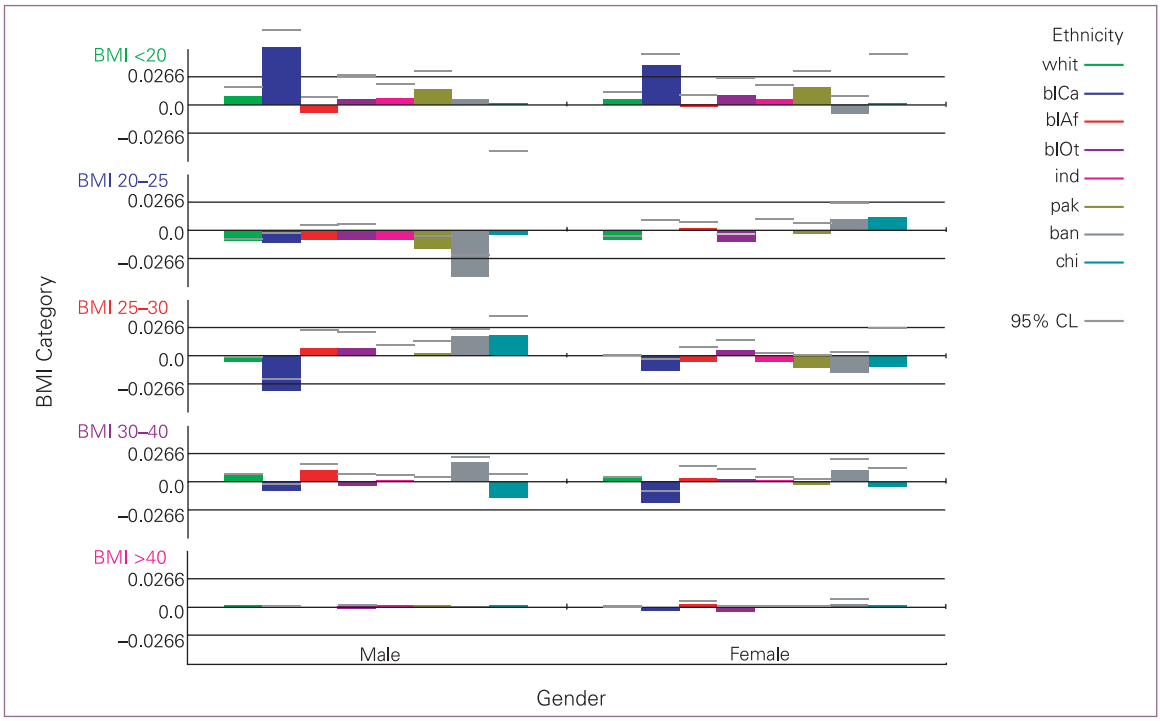
Black Caribbeans of both genders demonstrate an interesting reduction in the prevalence of obesity (Figure 13).

Figure 12: The slopes of trends with time in BMI groups, by geographical region and age. Guide to abbreviations: (NE) North-east England, (NW) North-west England, (YHum) Yorkshire and Humberside, (EMid) East Midlands, (WMid) West Midlands, (EoE) East of England, (Lon) London, (SE) South-east England, (SW) South-west England.



These analyses lack the power to detect subgroup effects – in Figure 14, between the possible confounding of regional effects by social class or visa versa. The Health Survey for England was not designed to enable such effects to be detected and it is therefore not surprising that they can't be shown. To better understand the increase in obesity, a linear model could be fitted to these slopes on the aggregated model to test for the more important interactions. The main finding, however, is of fairly uniform and linear growth with weak evidence (given the data paucity) of important interactions.

Figure 13: The slopes of trends with time in BMI groups, by ethnicity and gender. Guide to abbreviations: (blCa) Black Caribbean, (blAf)Black African, (blOt)Black other, (ind)Indian, (pak)Pakistani, (ban)Bangladeshi, (chi)Chinese



All of the slope graphs demonstrate insufficient samples among subgroups to enable us to infer anything other than plausible hypotheses about obesity growth in England. Wherever an apparent interaction seems possible, shown by estimated differences in the slopes, the confidence limits, almost invariably, cannot exclude sampling error as the most cogent explanation.

Figure 14: The slopes of trends with time in BMI groups, by social class and geographical region. Guide to abbreviations: (NE) North-east England, (NW) North-west England, (YHum) Yorkshire and Humberside, (EMid) East Midlands, (WMid) West Midlands, (EoE) East of England, (Lon) London, (SE) South-east England, (SW) South-west England.

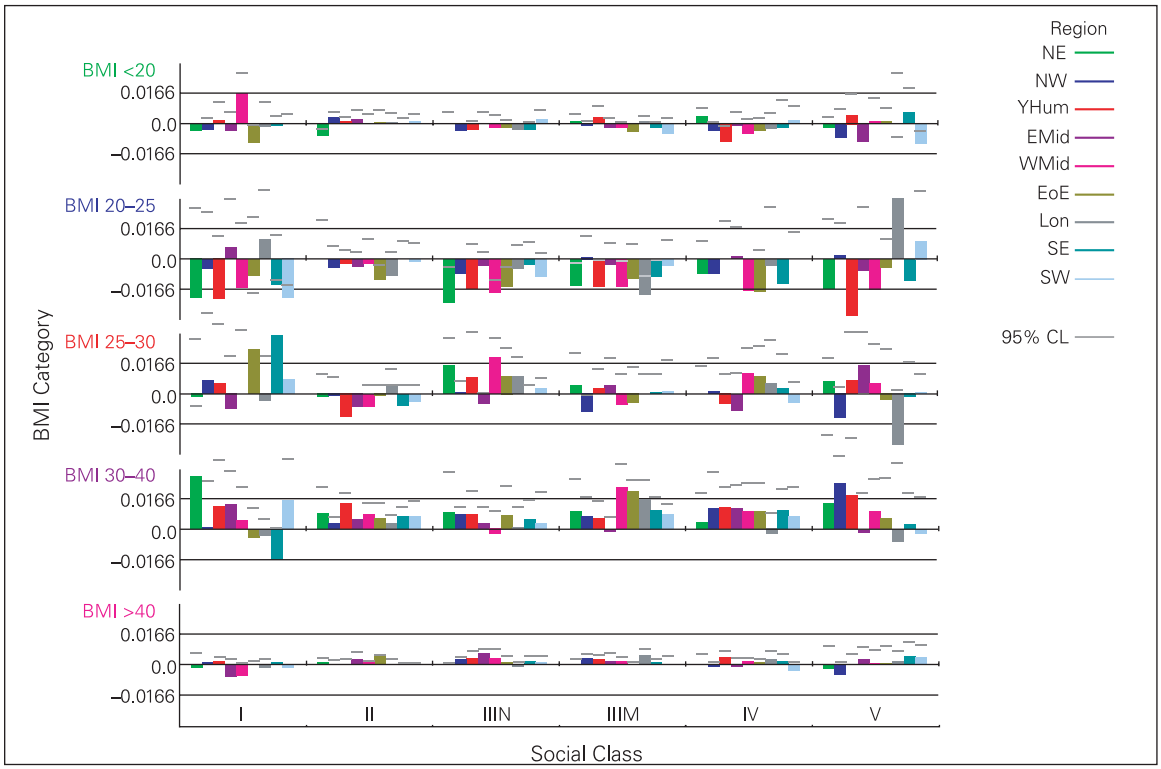
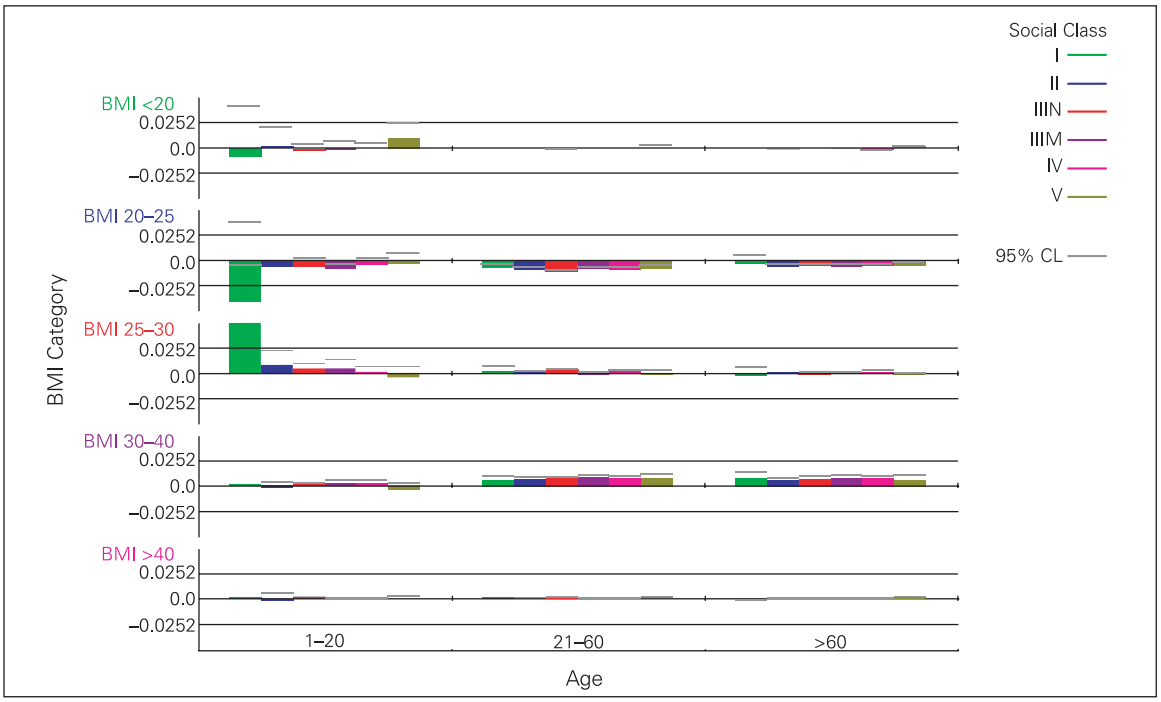


Figure 15: The slopes of trends with time in BMI groups, by social class and age



Part 2: Using the microsimulation program, Obesity 2

5 Disease attributable to elevated BMI and costs to the NHS

The microsimulation calculates, for each year, the risks of disease, given the age, gender, current morbidity and BMI level of each simulated person. Therefore the random generation of events by the microsimulation, given these determinants of risk, follows the epidemiology. This, of course, means that the duration and extent of overweight or obesity are determinants of disease, since simulated persons will continue to have these risks each year. Likewise, survival is determined by the survival experience, as far as it is reliably known, of individuals with a given disease moderated by age and gender. In this way, death is a consequence of diseases related to obesity, as well as other causes as recorded by national statistics.

5.1 The predicted changes in the distribution of obesity among adults until 2050

The predicted distribution of BMI among adults at selected years is given in Figure 16, with the mean level shown by the vertical black line. The manner in which the mean BMI is seen to increase with time is startling. The low growth in morbid obesity among males is of particular note (see Section 4.2), especially as the risk of disease is known to be increased in men. If, in future years, this prevalence is seen to be under-representative of the true situation in males, the predictions from the later microsimulations will be shown as having been conservative.

5.2 Projected incidence of related diseases

Assuming only the predicted changes in BMI and no other changes in external factors occur, the age- and gender-standardised incidence rates, after 2005, of the three commonest obesity-related diseases are shown in Figure 17. Diabetes is predicted to increase the most.

Figure 16: BMI distribution (%) for Males and Females in 2005,2010, 2015, 2025 and 2050. Mean BMI is indicated by the vertical black line.

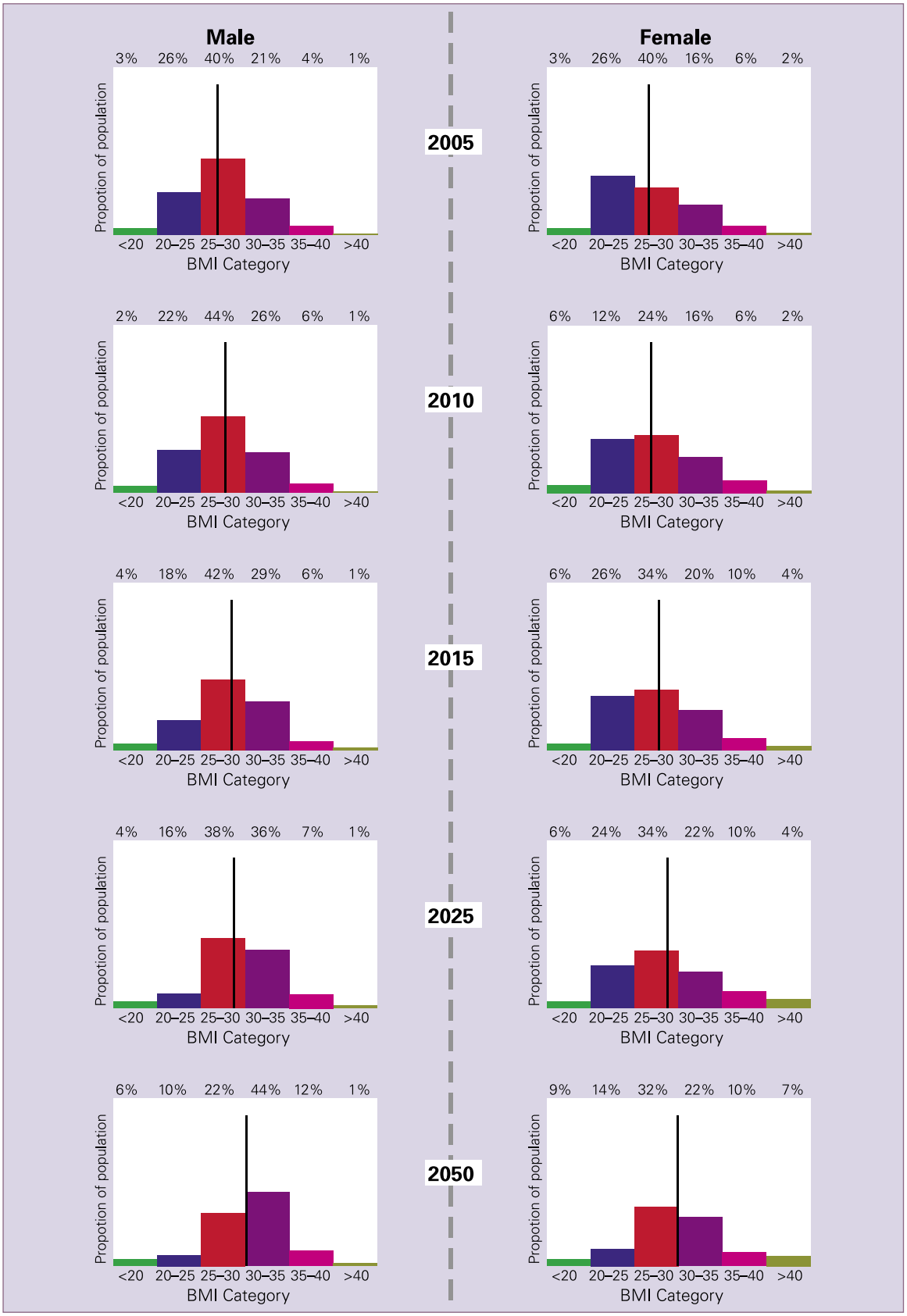
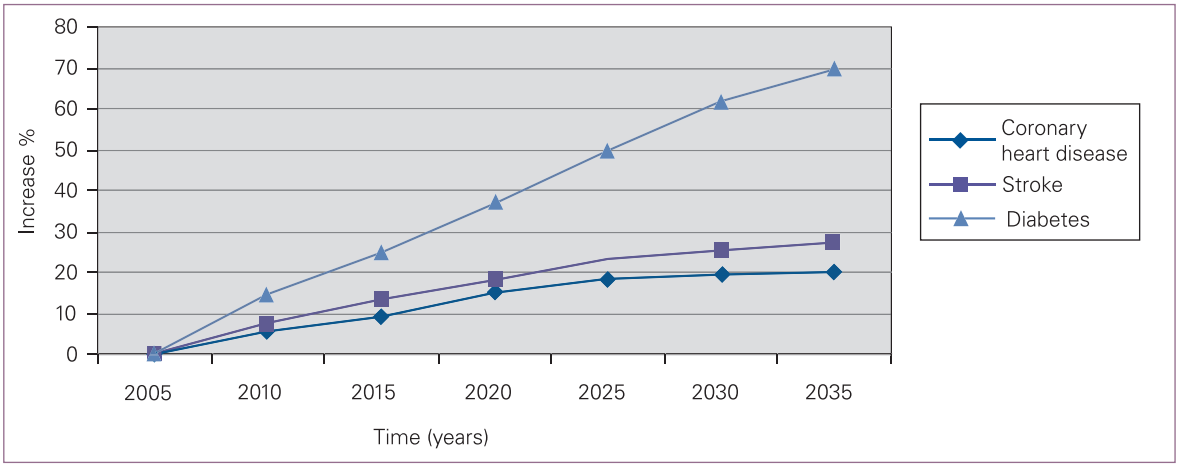


Figure 17: Increase in age- and gender-standardised incidence of diseases attributable to rising BMI levels, from 2005



5.3 Projected costs

This microsimulation utilises the best available evidence to calculate the prevalence of disease attributable to the predicted growth in overweight and obesity. Costs represent a sensitive summary measure of the consequence of overweight and obesity – where detailed knowledge of cost pertaining to particular diseases are available – so summary costs will be the major illustration of consequences here. This is not to imply any lack of importance of other consequences of an increasing incidence of disease. More detailed examination of factors beyond summary costs can be undertaken at the end of each simulation run.

The risk of developing type 2 diabetes, for instance, is some 20–80 times more likely for people who are obese compared with lean people (Appendix 1). Coronary heart disease (which itself is slightly more common among obese people) is 2–3 times more common among diabetic men and five times more common among diabetic women. Stroke is also more common among obese people (and also among those with diabetes) than in the general population, as are many cancers, particularly endometrial and kidney cancer, as well as osteoarthritis. The effect of this microsimulation is to allow individuals to accumulate health risks as they would normally do throughout their lives.

There are several different costs that should be distinguished. Of major importance is the cost to the NHS of increased levels of obesity and overweight over and above the current NHS costs. To keep matters simple, no inflation costs, either of prices generally or of healthcare costs in particular, are incorporated within the costs discussed – this allows a direct comparison to current prices to be made. These future BMI growth-related costs can be approximated either (a) by subtracting estimates of current NHS costs of obesity, as ascertained by other

authors, from projected costs derived from the model, or (b) by comparing the costs estimated by the model, that account for predicted BMI growth with those estimated assuming no change in BMI. Method (a) was preferred.

To enable estimates of these increasing NHS costs, summary costs are derived from disease-specific estimates for all diseases related to elevated BMI. Clearly, not all the NHS costs of such diseases are BMI-related. Therefore these costs are referred to as disease-related costs. Subtracting current disease-related costs from projected disease-related costs produces estimates of future BMI-related costs – since all that changes in the model with time are the population age and gender distribution, and increasing prevalence of overweight and obesity.

NHS costs are not the only impact of obesity and overweight. The costs of obesity and overweight to society include absence from work, morbidity not treated in the health service and reduction in quality of life. These have been estimated by others and we made the assumption that the ratio of total costs of overweight and obesity to NHS costs of obesity alone is likely to remain approximately constant.

Table 3 shows that on current trends, holding everything apart from BMI constant, the potential future annual health service costs of diabetes, coronary heart disease, stroke and two common cancers are predicted by the simulation to rise. The apparent precision of these estimated costs is illusory. Differences of two or three points in the first decimal place occur between runs of 10 million simulations.

Table 3: Estimated future NHS costs of diseases related to BMI, 2007–2050 (£billion per year)

	Cost/year (£ billion)			
	2007	2015	2025	2050
Diabetes	2.00	2.20	2.60	3.50
Coronary heart disease	3.90	4.70	5.50	6.10
Stroke	4.70	5.20	5.60	5.50
Colorectal cancer	0.45	0.50	0.53	0.50
Breast cancer	0.27	0.29	0.32	0.31
NHS cost (all related diseases)	17.4	19.5	21.5	22.9

The costs in Table 3 should be seen in the context of an approximation of total costs (including NHS costs) in 2001 attributable to overweight and obesity of around £7 billion per year[17], of which £1 billion was the estimated NHS costs of obesity alone.[18]

Table 4 shows the estimated costs of elevated BMI from 2001 to 2050. The cost increases denoted as 'extra' costs are attributable solely to changing BMI levels from 2001, and the complex effects this will have on, for example, longevity and therefore numbers of people at risk. The increasing levels of BMI in the future are predicted to add £7.7 billion to annual costs by 2050. If the ratio of the total wider costs of overweight and obesity to the NHS costs of obesity alone remains similar, we would be anticipating a total cost per annum of £49.9 billion attributable to increasing BMI by 2050. The figures in this table are based on the assumption that everything else, including the value of money, remained the same.

Table 4: Estimated costs of elevated BMI, 2001– 2050 (£billion per year)

	2001	2007	2015	2025	2050
Extra NHS costs of elevated BMI predicted by the micro-simulation model	0	2.2	4.1	6.3	7.7
% of all overweight who are obese	33%	40%	48%	52%	66%
Predicted extra NHS costs of obesity alone[1]	0	1.3	2.9	4.3	6.1
NHS costs of obesity alone	1.0	2.3	3.9	5.3	7.1
NHS costs of elevated BMI	2.0	4.2	6.4	8.3	9.7
Total wider costs of elevated BMI[2]	7.0	15.8	27.0	37.2	49.9

Table 5, which shows projected percentage of NHS costs arising from elevated BMI from 2001 to 2050, is predicated on the assumption that NHS unit costs stay constant, although clearly they would not do so in practice.

Table 5: Projected percentage of NHS costs of elevated BMI 2001–2050

	2001	2007	2015	2025	2050
Projected % of NHS costs @ £70 billion	2.9	6.0	9.1	11.9	13.9

5.4 Life expectancy changes attributable to obesity trends

Period estimates of life expectancy are calculated in five-year intervals over the course of the simulation from estimated death rates in these years. Such 'life tables' are normally projected into the future by extrapolating death rates from current trends. The microsimulation holds current death rates constant, apart from the estimated changes attributed to alterations in BMI levels. In order to assess the future impact on life expectancy of increasing BMI, we used the microsimulation to compare two simulations: Simulation 0 (Sim 0) and Simulation 7 (Sim 7):

Sim 0 No intervention to the increase in obesity prevalence anticipated in Section 4
Sim 7 Current BMI levels held stochastically constant

[1] Assumes NHS costs per person of being overweight are half the costs per person of being obese
[2] Some of these figures are subject to rounding effects

Figure 18 plots the expected difference in life expectancy for males and females by year. These results were produced using 10 million Monte Carlo trials for each simulation to ensure sufficient convergence of the estimates (these take approximately half an hour each on a ~2Ghz computer). It can be seen that, if obesity rises as predicted, females will lose around a fifth of a year and males nearly a third by the middle of the 21st century.

The Government Actuary's Department[19] currently predicts life expectancy to rise by around eight years for men and seven for women in this period, to 84 and 87.5 years respectively. Therefore the increase in obesity will have surprisingly little impact (less than a year, as shown in Figure 18) on period life expectancy of the population.[20] This is because current downward trends in the mortality of major obesity-related chronic disease, which are independent of obesity, remain dominant, even taking account of the large increase in obesity levels that are predicted. Figures 19 and 20 show the total aggregated incidence of related diseases for two time periods to 2010 and 2040 respectively.

It can be seen that, while the aggregated incidence of disease changes (and of course these can be derived on an age- and gender-specific basis) as a consequence of anticipated growing obesity, when compared with prevailing levels under no obesity growth (Sim 7), the effects remain merely relative, but nonetheless important.

Figure 18: Expected net loss of life expectancy (in years) for men and women, by year, attributable only to growing BMI levels compared with no change from current levels

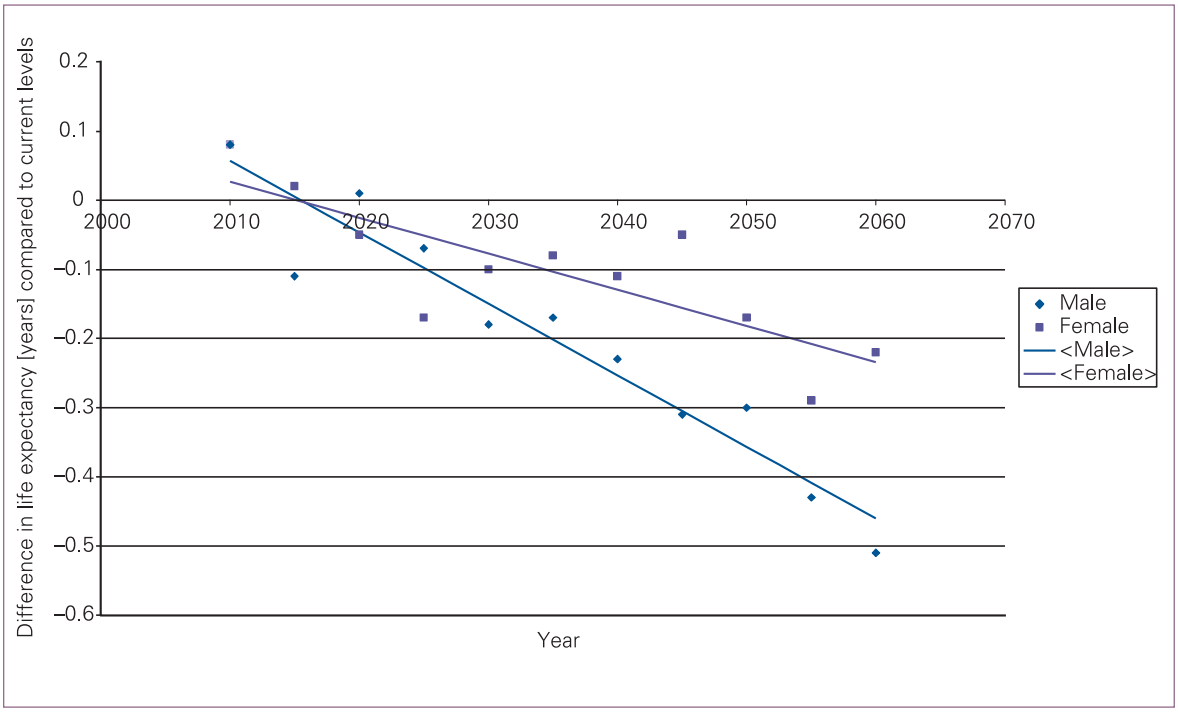


Figure 19: Aggregate annual incidence of disease predicted in 2010 comparing Sim 7 (current BMI levels held constant, light blue) with Sim 0 (rise in BMI as predicted, dark blue)

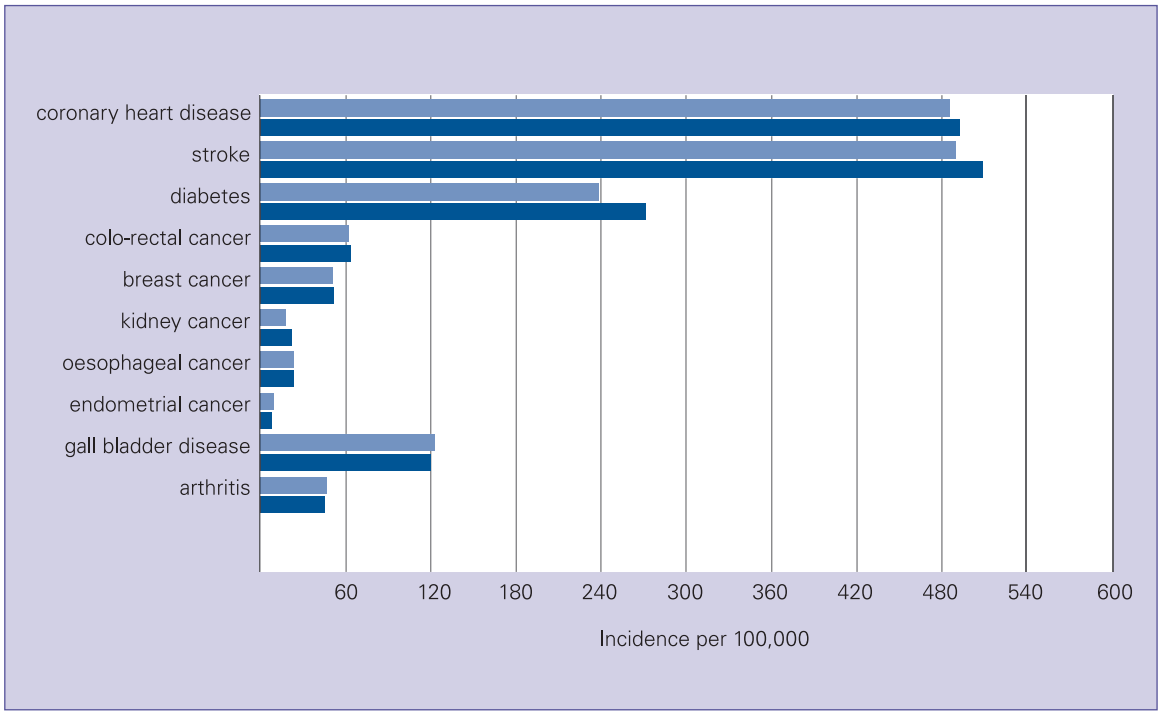
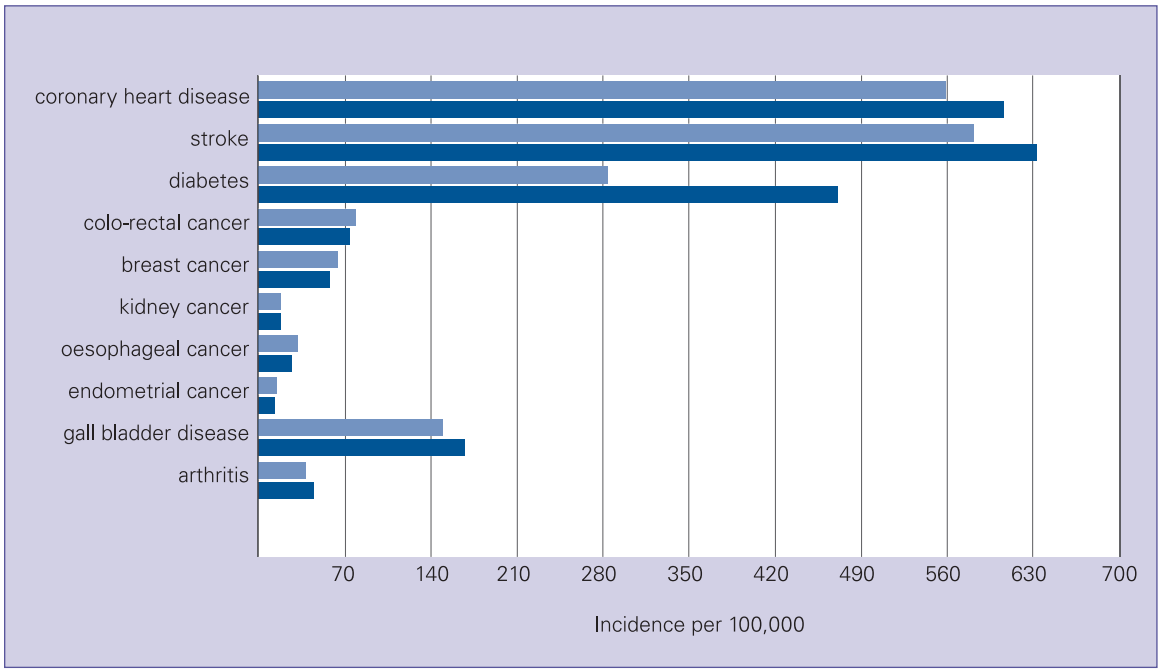


Figure 20: Aggregate annual incidence of disease predicted in 2040 comparing Sim 7 (current BMI levels held constant, light blue) with Sim 0 (rise in BMI as predicted, dark blue)



6 Simulating the effects of different BMI interventions

The microsimulation program, Obesity 2, makes it possible to test various hypothetical reductions in BMI, either across all groups by imposing a BMI cap or, in specific age ranges, by imposing no growth in obesity in those age groups. Various BMI effects are implemented by the program in different simulations. The simulations can model the effect of interventions to prevent obesity in specific high-risk groups of a particular age with specified degrees of success. Another simulation option simply reduces predicted BMI growth by a fixed average amount across the population. One simulation acts as a control in that it allows no growth in BMI from the current time.

All of these simulations can be applied to particular social classes or ethnic groups when specifying each simulation run. The program enables a rich list of plots (available on request), together with the opportunity to specify Excel tables of data in incidence, prevalence, costs and attributable disease (for a discussion of attributable disease, see Section 0). The microsimulation program also allows an exploration of the impact on chronic disease levels and the reduction in health service costs that would result if successful interventions were made that altered predicted trends in BMI.

For comparison purposes, the microsimulation program allows eight types of simulation, each of which can have different user specifications. Simulations can be batch-processed and the output collated for simultaneous presentation. Figure 21 is a characteristic plot.

As part of the validation process, many trial interventions were tested. These included reducing BMI levels by specified amounts in the whole population and interventions that targeted people with a high BMI. All of these simulations can be targeted at particular age groups and for specifiable periods of time.

Two examples of simulated interventions are described in this report:

Batch 1 A universal strategy of reducing average BMI across the population (Section 6.1)

Batch 2 A targeted strategy among potentially overweight or obese people (Section 6.2).

For each example, possible different interventions were implemented as a batch of six simulations. All interventions were initiated in 2008, four years after the selected 2004 start of the simulation, and terminated in 2060, the selected end date of the simulation. The microsimulation processes a simulation batch by performing the required number of Monte Carlo trials – in this case, 1,000,000 –

for each component simulation before storing the results and moving to the next. All components are processed collectively on completion of the batch. Therefore each of the following examples shows results for 6 million Monte Carlo trials.

6.1 Simulation Batch 1: average BMI reduction

For each batch of simulations processed, the microsimulation stores a file of the key parameters. For Batch 1, the files are given in Table 6:

Table 6: Batch 1 simulation parameters

Sim 0	Sim 0: 2008–2060: No interventions
Sim 1	Sim 1: 2008–2060: 15 ≤ Ages ≤ 50; BMI shift –2.0
Sim 2	Sim 2: 2008–2060; 15 ≤ Ages ≤ 50; BMI shift –4.0
Sim 3	Sim 3: 2008–2060; 15 ≤ Ages ≤ 50; BMI shift –6.0
Sim 4	Sim 4: 2008–2060; 15 ≤ Ages ≤ 50; BMI shift –8.0
Sim 5	Sim 5: 2008–2060; 15 ≤ Ages ≤ 100; BMI shift –8.0

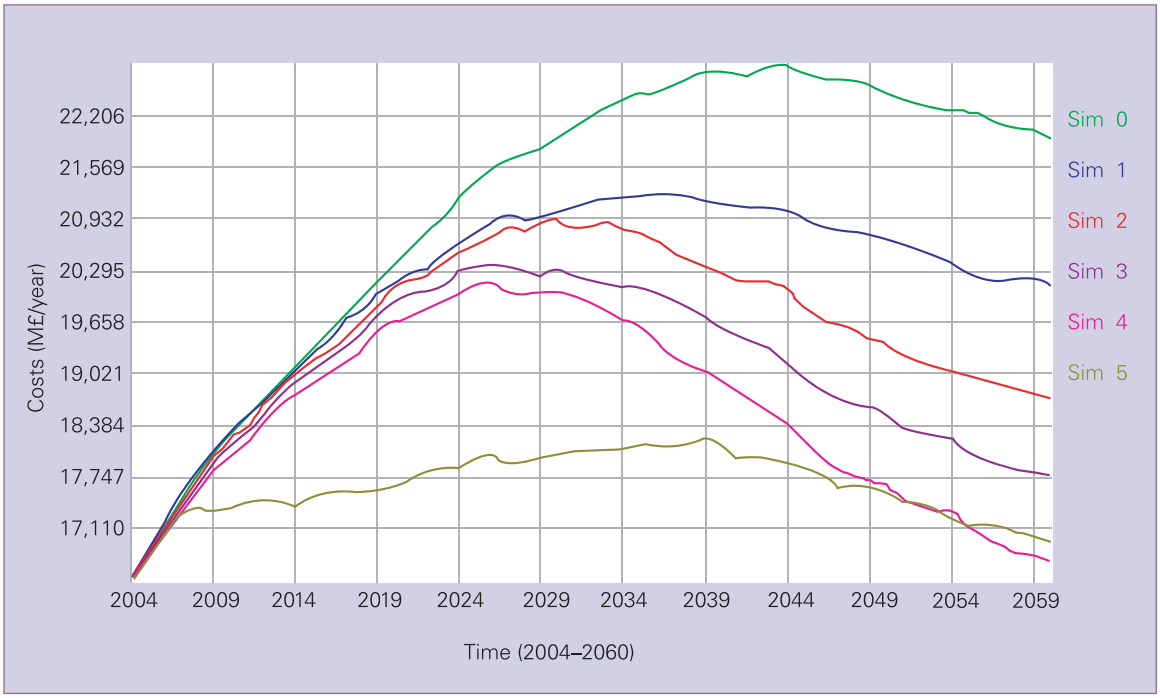
The total NHS costs by year are shown in Figure 21 and sample intermediate values are listed in Table 7.

Table 7: Batch 1: Total NHS costs for BMI related conditions (£ billion per year)

BMI reduction:	0 units	2 units	4 units	6 units	8 units	8 units
Target age:	15–50	15–50	15–50	15–50	15–50	15–100
Simulation:	Sim 0	Sim 1	Sim 2	Sim 3	Sim 4	Sim 5
2007	17.5	17.5	17.4	17.4	17.4	17.4
2015	19.3	19.2	19.2	19.1	18.7	17.5
2025	21.4	20.7	20.6	20.3	20.1	18.0
2050	22.5	20.7	19.4	18.5	17.6	17.6

In Figure 21, it can be seen that reducing BMI by an average of eight units across the entire life span (Sim 5) will result in hardly any increase in NHS expenditure and will have an immediate effect in 2008 at the start of the simulation. Reducing the BMI by a similar amount – eight units – but only for people aged 15–50 (Sim 4) would mean that the same cost would eventually be reached at around 2050, but the simulation clearly shows that such an intervention would not have an immediate effect.

Figure 21: Batch 1: NHS costs of obesity related disease predicted from average BMI reductions (0, 2, 4, 6 and 8 units) as listed in table 7



The greatest impact occurs in cases where the disease burden is greatest – usually among people over 50 (the one exception is arthritis – see Figure 22). Sim 5 (reduction by eight units over the whole population, i.e. reducing the mean BMI from say 30 to 22) starts reducing the disease burden for those aged 50+ from the outset of the simulation in 2008, whereas in Sim 4, where the reduction is restricted to the under-50s, those aged 50+ at the outset are initially at an increased risk of developing obesity-related diseases, but as younger people with lower BMIs age, the disease burden falls. Note that, in this simulation, the risk of a person developing a given disease at any age is associated with their BMI value at that age. The intermediate simulations (Sims 1, 2 and 3) show a gradual change from the outcome predicted in Sim 0 to that predicted by Sim 4. Again, the lag in the cost reduction is brought about by the time taken for the younger people with lower BMIs to grow old and have less obesity-related disease as a result of their lower BMIs.

Figure 21 and Table 7 show the total costs aggregated over all BMI-related diseases. The microsimulation is capable of displaying cost graphs for any of the individual BMI-related diseases. Those for arthritis and coronary heart disease are shown (Figures 22 and 23). Notice the graph sets are quite different from each other in character as a result of the earlier onset and greater BMI-dependence of arthritis as compared to coronary heart disease.

Figure 22: Batch 1: Predicted NHS arthritis costs, by year from average BMI reductions as listed in table 7

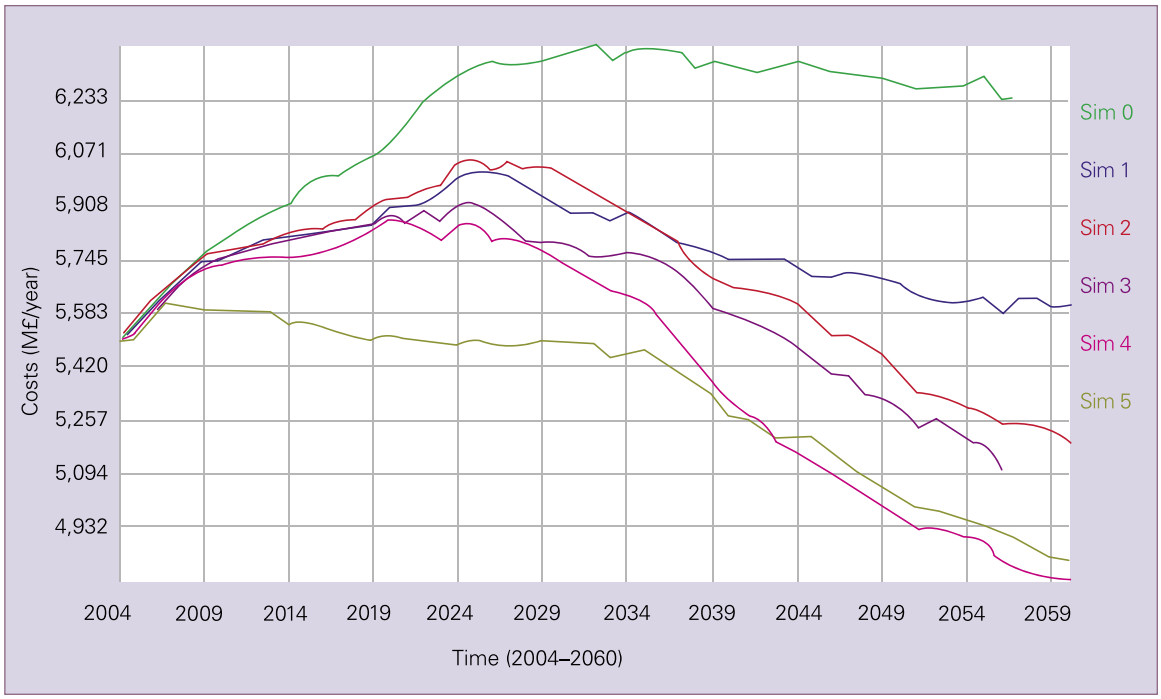
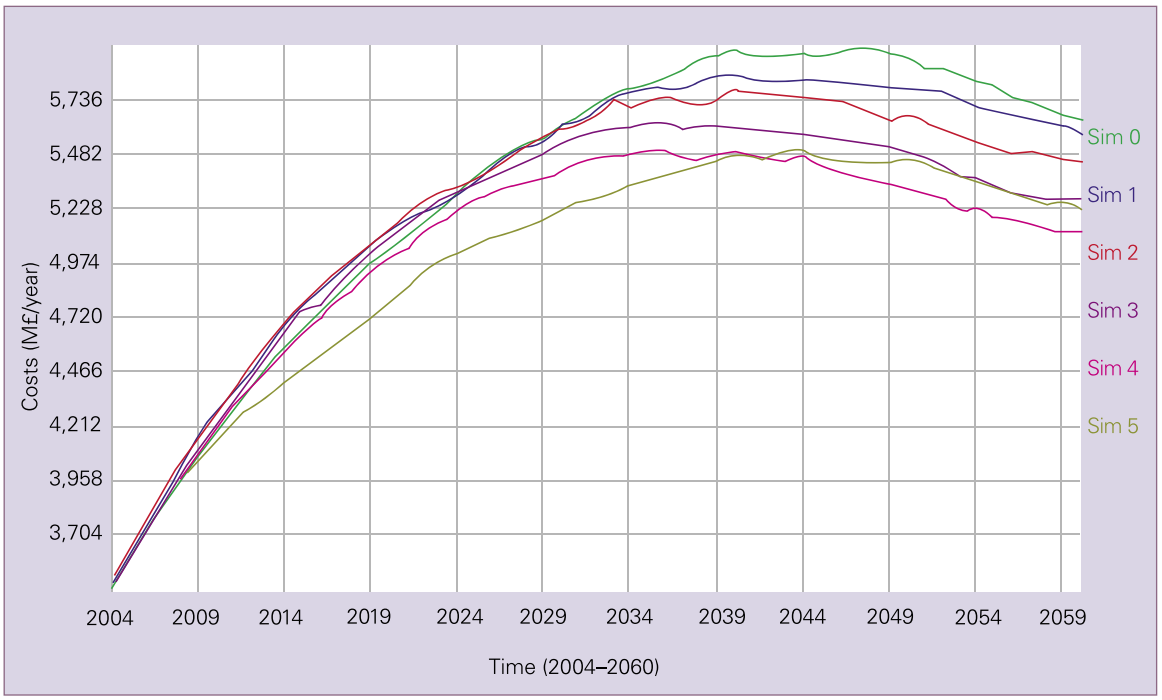


Figure 23: Batch 1: Predicted NHS coronary heart disease costs, by year from average BMI reductions as listed in table 7



6.2 Simulation batch 2: Targeted BMI reduction

For Batch 2, the simulation parameters are listed in Table 8

Table 8: Batch 2 simulation parameters

Sim 0	Sim 0: 2008–2060; No interventions
Sim 1	2008–2060; All ages; BMI cap 30; BMI caps 25%
Sim 2	2008–2060; All ages; BMI cap 25; BMI caps 25%
Sim 3	2008–2060; All ages; BMI cap 30; BMI caps 50%
Sim 4	2008–2060; All ages; BMI cap 25; BMI caps 50%
Sim 5*	2008–2060; 15 ≤ Ages ≤ 100; BMI shift –8.0
*Sim 5 from Batch 1 has been included for ease of comparison.	

The total NHS costs by year are shown in Figure 24 and sample intermediate values are listed in Table 9.

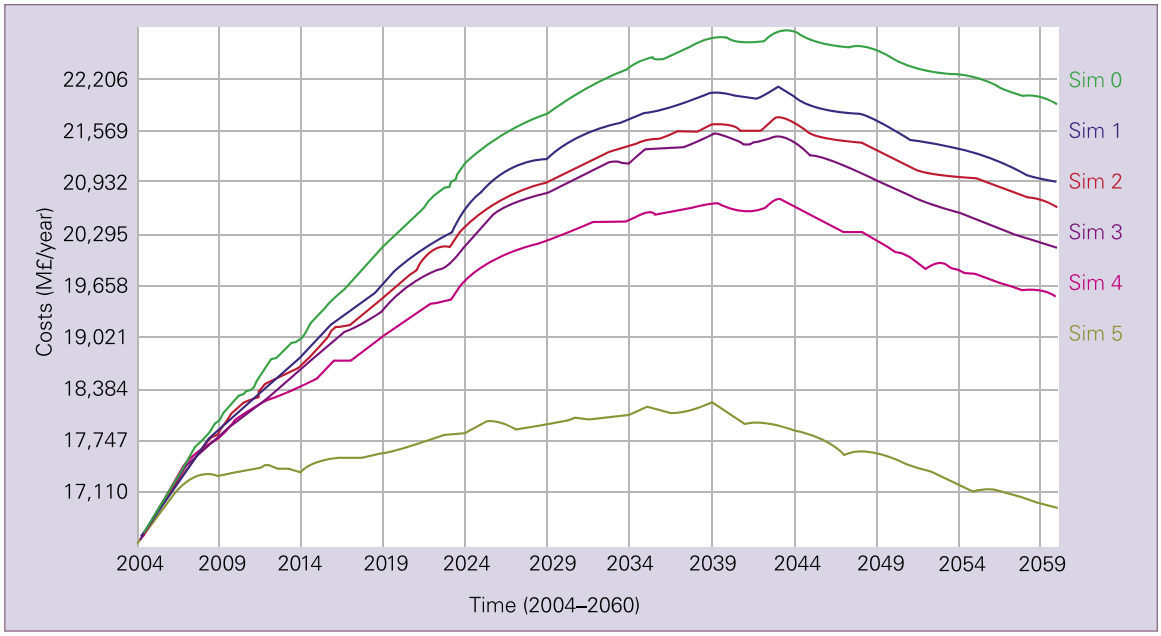
Table 9: Batch 2, NHS costs for BMI related conditions (£ billion per year)

Successfully target:		**25% of population**		**50% of population**	
Preventing BMI above:		**30 units**		**25 units**	
Simulation number	**Sim 0**	**Sim 1**	**Sim 2**	**Sim 3**	**Sim 4**
2007	17.5	17.4	17.4	17.4	17.4
2015	19.3	20.0	18.9	18.8	18.5
2025	21.4	20.9	20.6	20.5	19.9
2050	22.5	21.6	21.3	20.9	20.1

In Figure 24, we can see that the expected increase in expenditure is clearly a function of the size of the BMI cap and the success of the intervention – as one might expect. All targeted BMI reduction simulations are bracketed, year by year, by Sim 0 and Sim 5. Whether targeted interventions achieve better or worse results than average BMI reductions is not a straightforward matter – the results depend on the details. The interventions do, however, achieve a more immediate effect than age-specific interventions.

The costs for arthritis and coronary heart disease in Batch 2 simulations are similar to those in Batch 1. Of greater interest are the costs for stroke and diabetes – shown in Figures 25 and 26. The stroke costs are a little 'noisier' than any of the other diseases shown. This is due to a combination of effects. Firstly, in 1,000,000 Monte Carlo trials, there are relatively fewer stroke events (~10,000) compared to

Figure 24: Batch 2: NHS costs predicted from targeted BMI reductions as listed in table 8



those for coronary heart disease (20,000) and diabetes (~20,000). The fewer the number of events, the more Monte Carlo trials are required to achieve convergence. Secondly, the scale, set automatically to capture the range of variation, is smaller than for other diseases.

The prevalence of diabetes, on which the costs are based, is particularly sensitive to variations in population BMI. This is illustrated most dramatically in Figure 26 by the results of Sim 5, with its widely separated cost trajectories and dramatic reduction in costs.

Although, technically, the microsimulation is able to do this, we have not yet tested class, ethnic or regional interventions, which will depend on coherent demographic or policy hypotheses.

The current Public Service Agreement target focuses on halting the year-on-year rise in obesity levels in children under 11. Simulating success with this target in our simulation program demonstrates that financial benefits will only begin to be reaped after 2050 or thereabouts. Preventing rises in obesity among the 6–10-year age group appears to produce similar delayed effects.

Each of the runs used to estimate figures 25 and 26 generated data on the incidence and prevalence of all BMI-related diseases as well as their estimated costs. In addition, statistics were generated that identify disease incidence that is

Figure 25: Batch 2, Predicted NHS stroke costs from targeted BMI reductions as listed in table 8

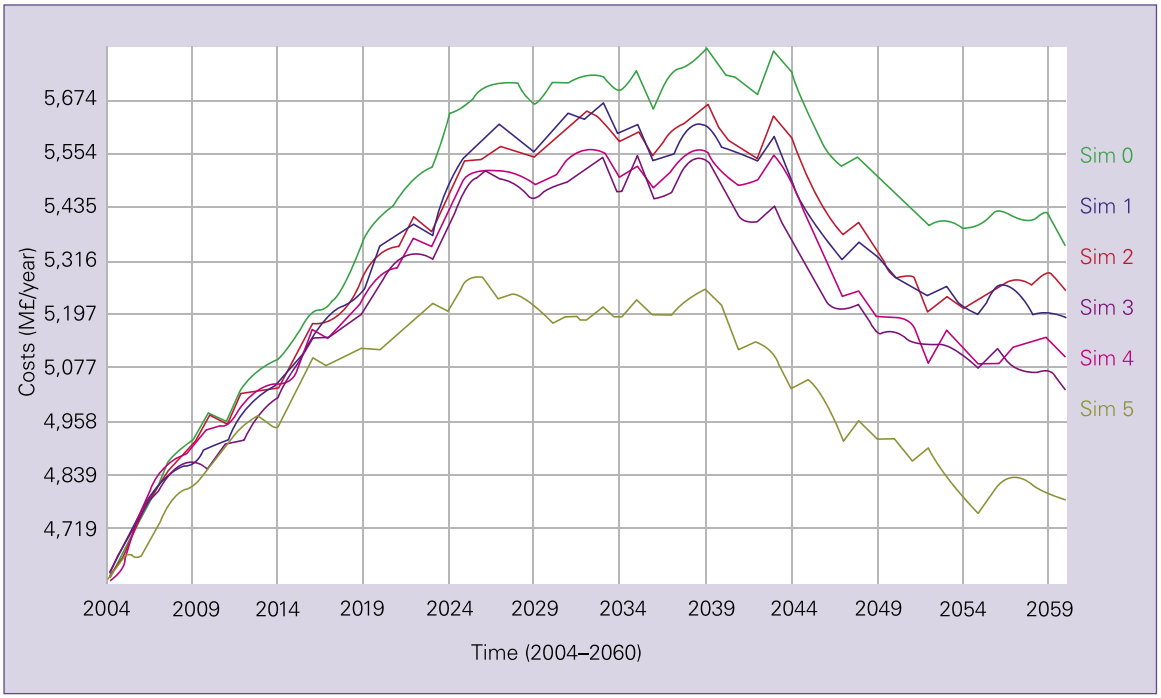
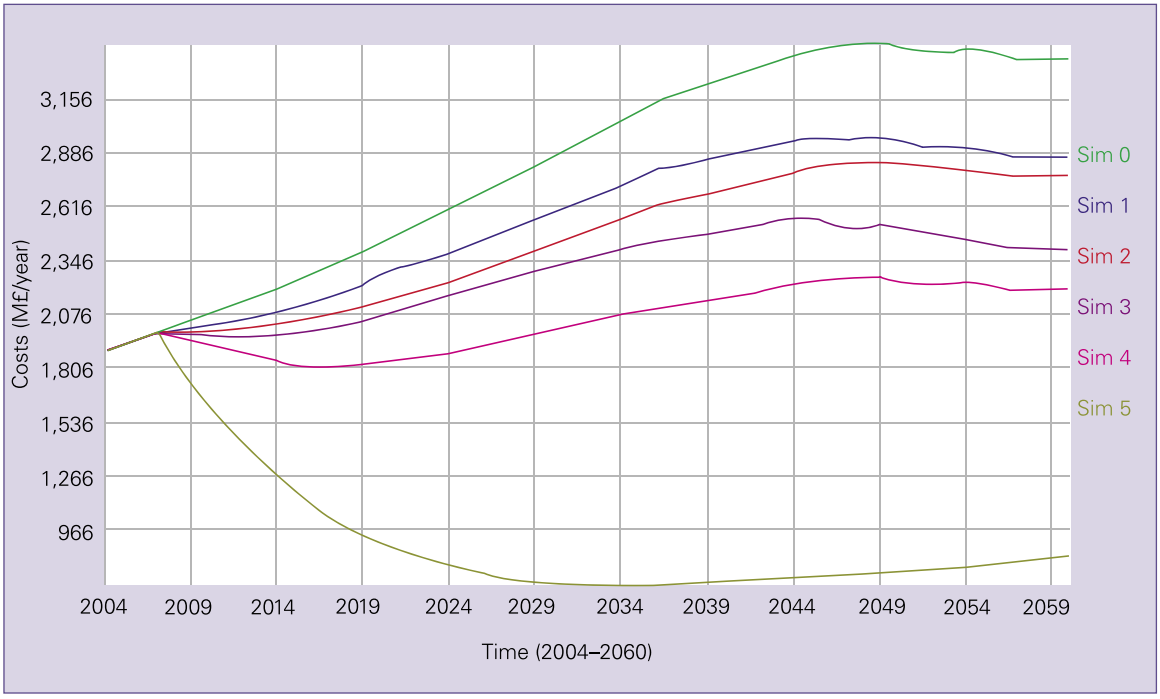


Figure 26: Batch 2,Predicted NHS diabetes costs from targeted BMI reductions as listed in table 8



strictly attributable to BMI at various levels. In real-life trials, attributable disease incidence is a statistic that must be inferred, though in microsimulation it can be measured directly. This is discussed further in Section 6.3.

6.3 Prevalence of disease attributable to overweight and obesity with different simulated interventions

Attributable disease refers to disease incidence that arises uniquely from the simulated subject being overweight or obese. In the microsimulation, events occur when probability thresholds are randomly achieved. It is worthwhile to explain the process briefly.

In any simulated disease incidence event, the host computer produces a new random probability – call it p. For the person that is the subject of the trial in progress and for each disease they might contract, two annual probability thresholds are produced: T_1, a baseline threshold – the probability of the person contracting the disease if not overweight or obese; and T_2, a higher threshold – the probability of the person contracting the disease if overweight or obese (in practice the thresholds are determined by the persons current BMI score). These thresholds depend on the person's age, gender and their disease history. If p is less than T_1, the person develops the disease. If p is greater than T_1 but less than T_2, the person develops the disease and it is attributed to their BMI. If p is greater than T_2, the person does not contract the disease. In this way, disease incidence, and its subsequent prevalence, is attributed to BMI.

The set of graphs in figures 27-34 are drawn for the same set of runs and simulations as those that produced the previous cost graphs (Figures 24-26). They show the numbers of cases per year of selected diseases (stroke, coronary heart disease, diabetes, arthritis) that are directly attributable to the constituent individual's BMI. These numbers are logged by the microsimulation only when the disease is contracted because of the increased risk due to the individual's BMI.

This set of eight graphs (figures 27-34) can be compared with the previous set (Figures 25 and 26), and there are obvious correlations. However, the cost graphs use the overall disease prevalence as the basis of their costing. The attributable disease graphs and associated statistics can be used to calculate costs that are directly attributable to BMI.

Figure 27: Batch 2: Sim 1 (purple) compared with Sim 0 (green), cases of BMI-attributable stroke, by year

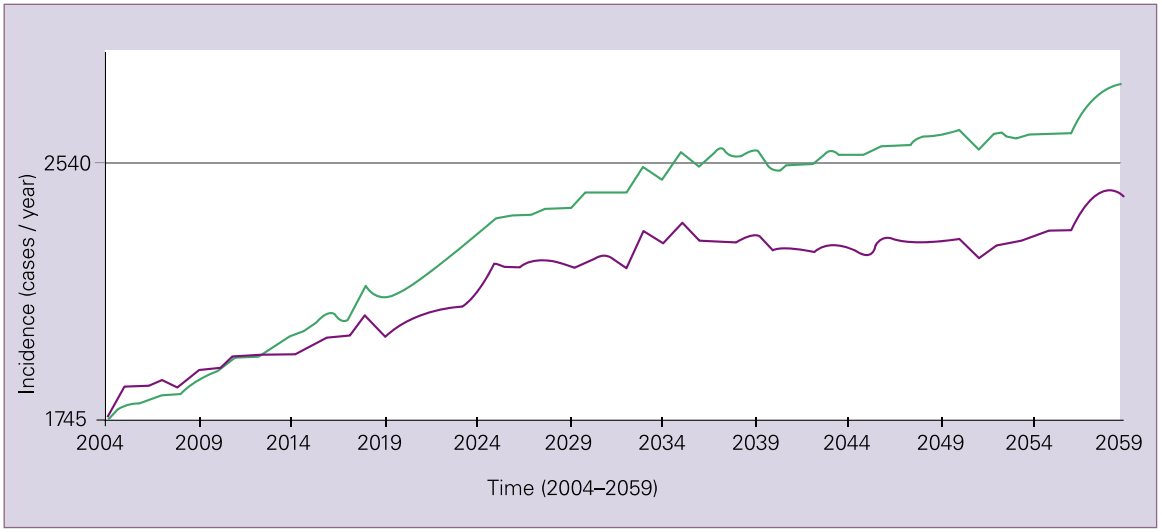


Figure 28: Batch 2: Sim 1 (purple) compared with Sim 0 (green), cases of BMI-attributable coronary heart disease, by year

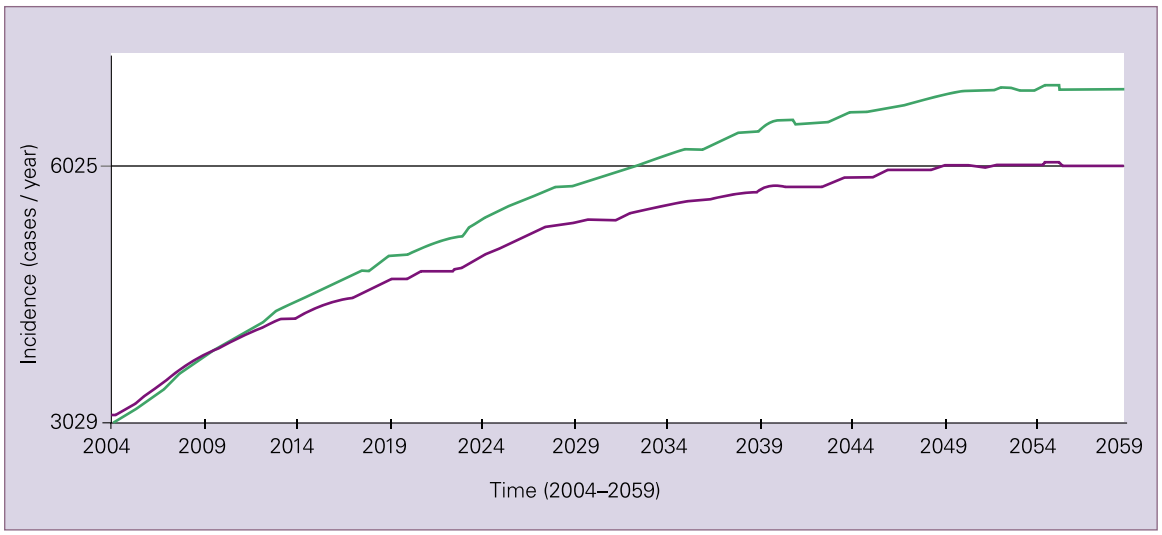


Figure 29: Batch 2: Sim 1 (purple) compared with Sim 0 (green), cases of BMI-attributable diabetes, by year

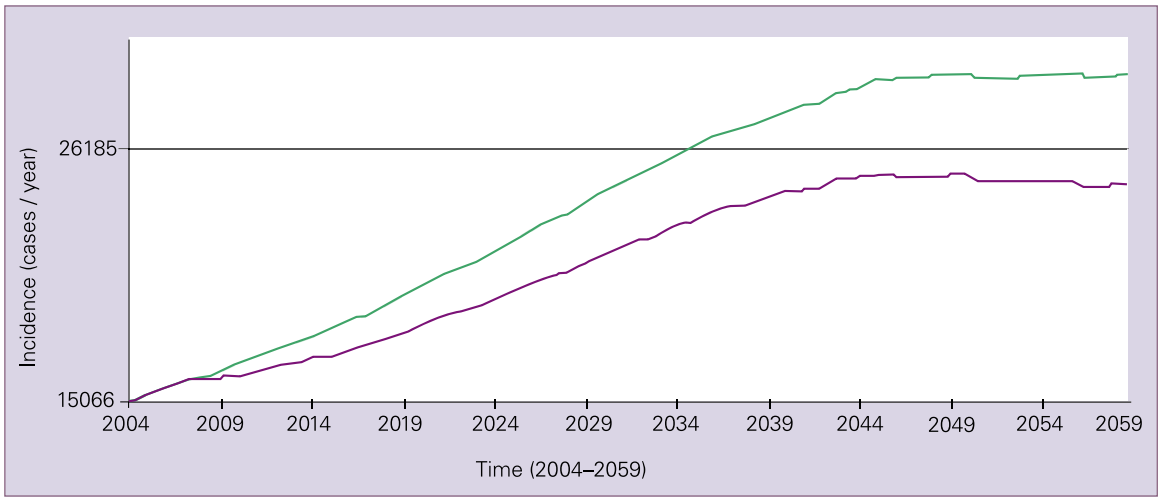


Figure 30: Batch 2: Sim 1 (purple) compared with Sim 0 (green), cases of BMI-attributable arthritis, by year

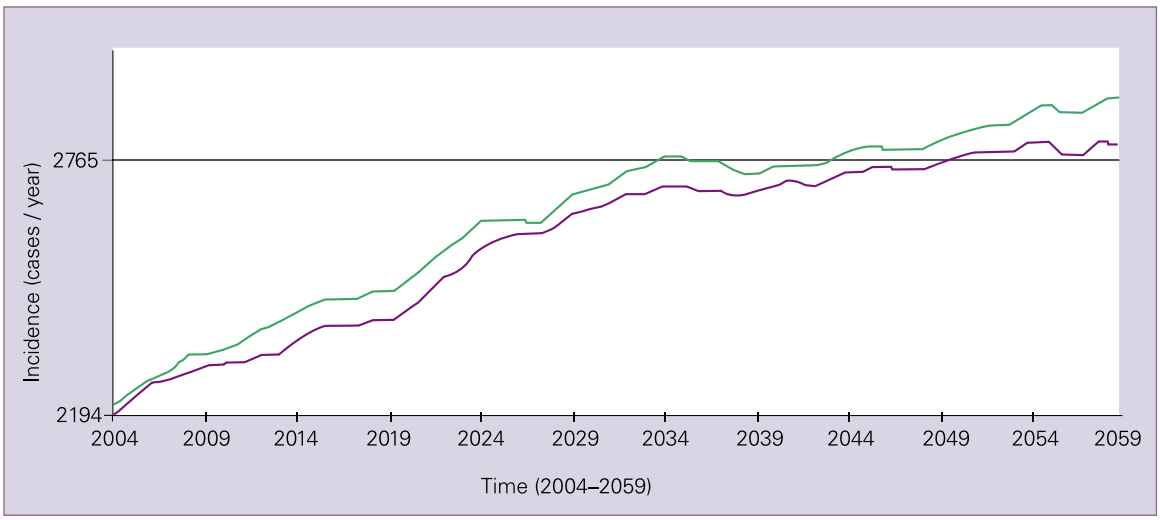


Figure 31: Batch 2: Sim 5 (purple) compared with Sim 0 (green), cases of BMI-attributable stroke, by year

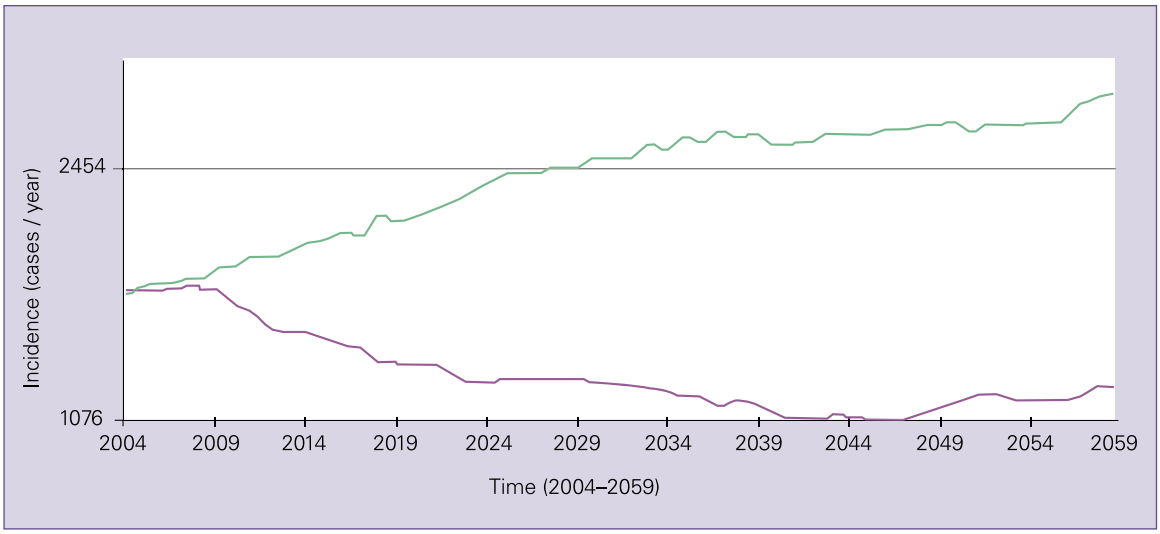


Figure 32: Batch 2 Sim 5 (purple) compared with Sim 0 (green), cases of BMI-attributable coronary heart disease, by year

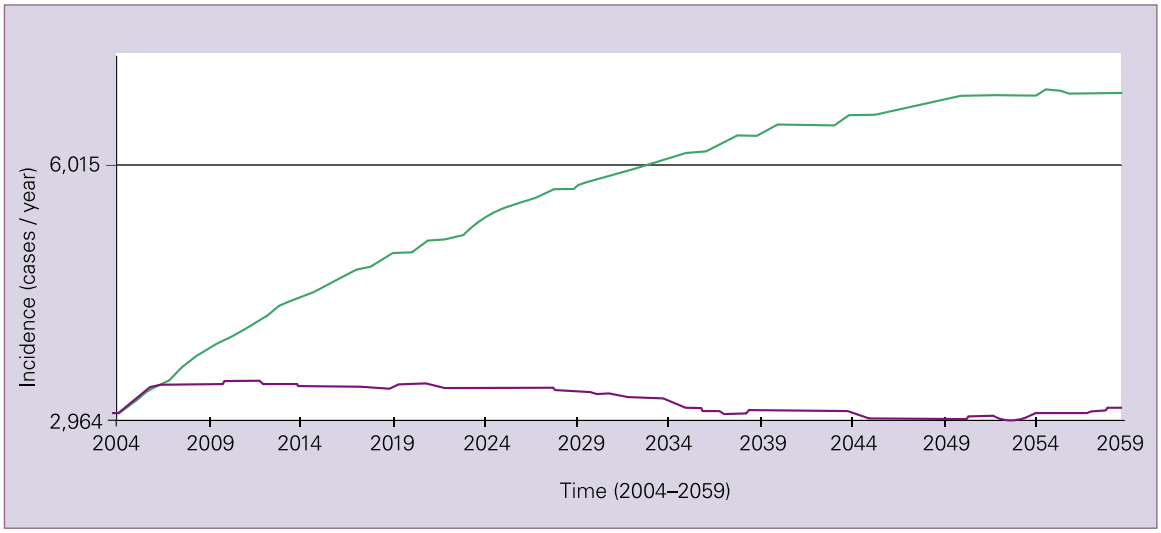


Figure 33: Batch 2: Sim 5 (purple) compared with Sim 0 (green), cases of BMI-attributable diabetes, by year

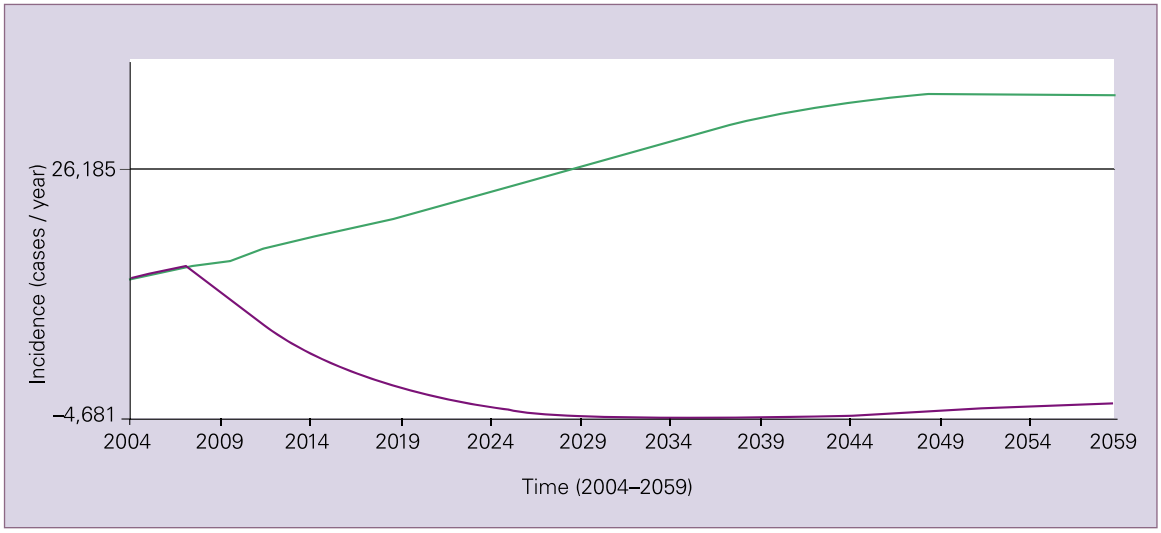
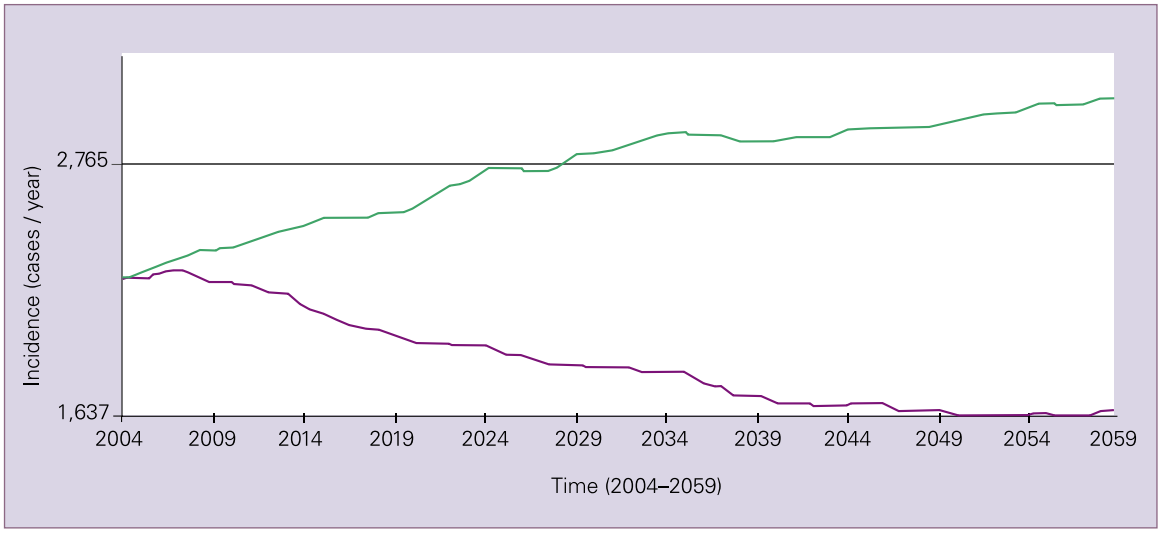


Figure 34: Batch 2: Sim 5 (purple) compared with Sim 0 (green), cases of BMI-attributable arthritis, by year



Examples of the associated attributable costs are shown in the following four graphs, figures 35–38. The shapes of the graphs are identical to their corresponding, defining disease prevalence graph.

Figure 35: Batch 2: Sim 5(purple) compared with Sim 0 (green), BMI-attributable stroke costs, by year

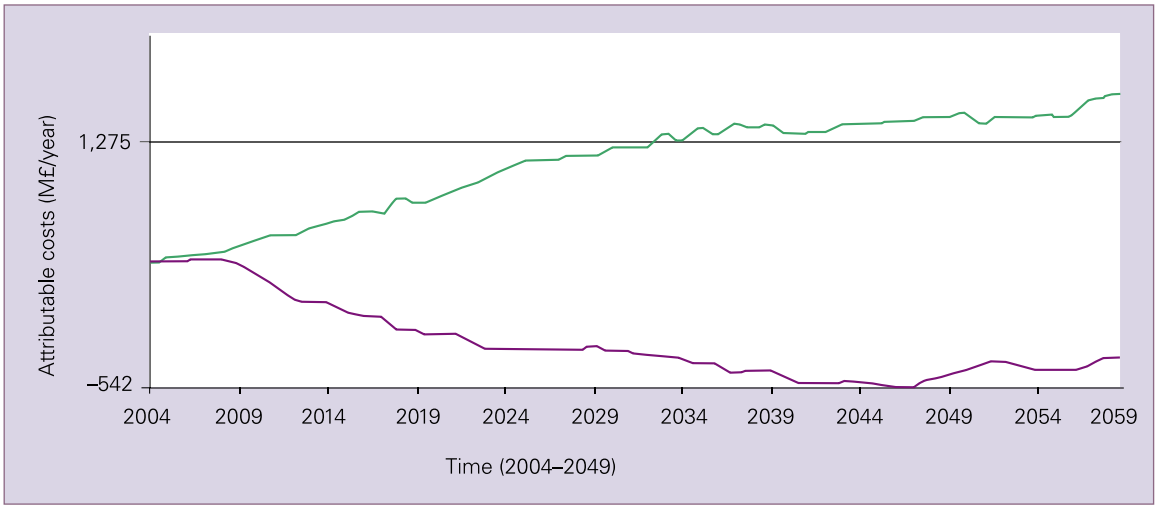


Figure 36: Batch 2 Sim 5 (purple) compared with Sim 0 (green): BMI-attributable coronary heart disease costs, by year

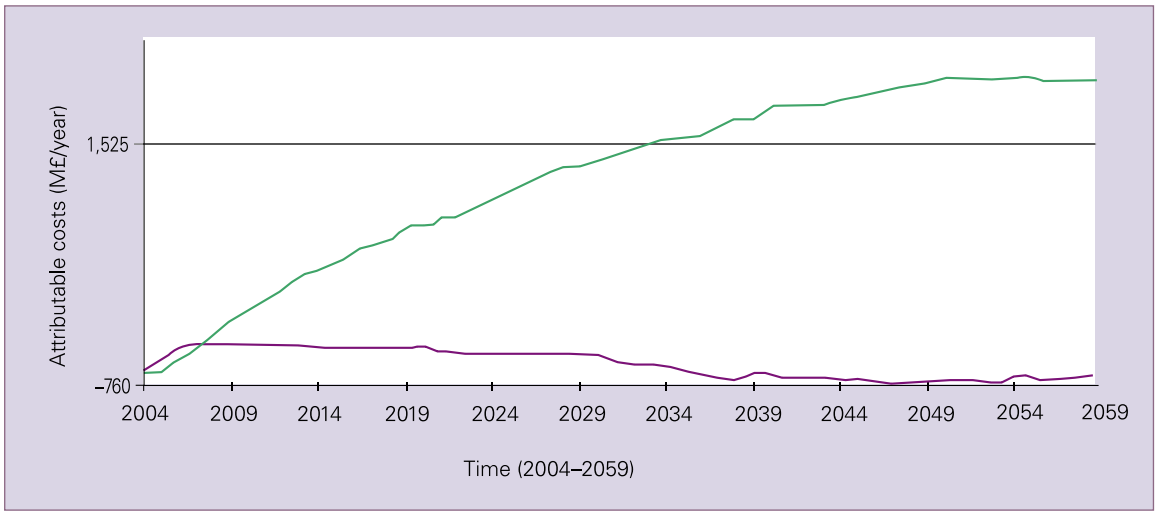


Figure 37: Batch 2: Sim 5 (purple) compared with Sim 0 (green), BMI-attributable diabetes costs, by year

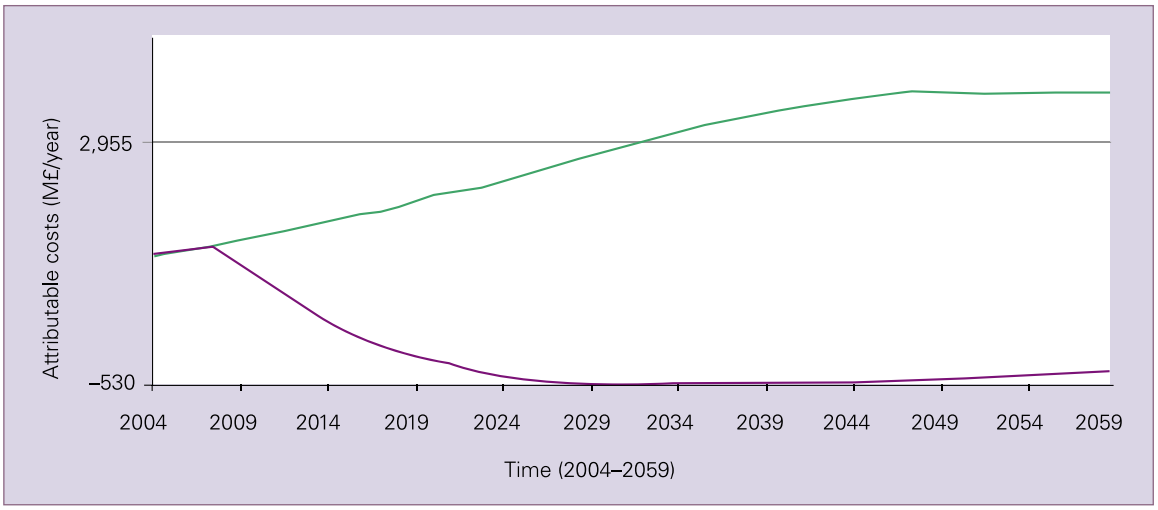
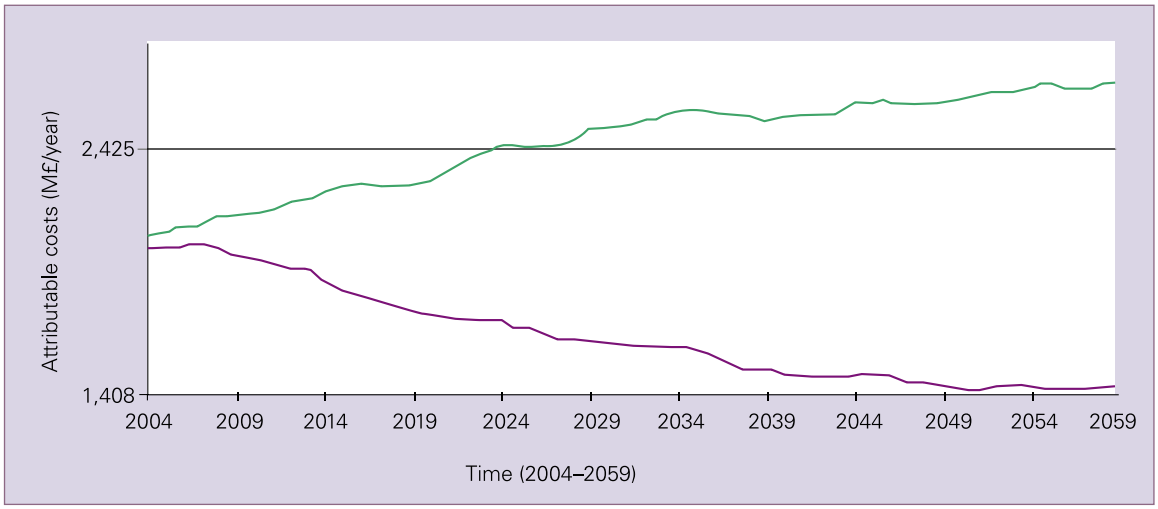


Figure 38: Batch 2: Sim 5 (purple) compared with Sim 0 (green), BMI-attributable arthritis costs, by year



6.4 Simulation testing of PSA targets

The PSA target is implemented as Sim 1 of Batch 3 in which four interventions were trialled. All are listed in Table 10. The endpoint of the run has been extended to 2070 so that the cost benefits from the simulation can be seen. As before, each run consists of 1.000,000 Monte Carlo trials.

Table 10: Batch 3: intervention parameters

Sim 0	2008 to 2070; No interventions
Sim 1	2008 to 2070; Age{6-10} Cla{all} Eth{all} no BMI growth
Sim 2	2008 to 2070; All ages; BMI cap 30; BMICaps 50%
Sim 3	2008 to 2070; 20<=Ages<=100; BMI shift -4.0

The total costs are shown in Figure 39. As expected, the effect of the intervention in 6-10 year olds (Sim 1) takes some time to manifest itself fully.

The attributable costs of coronary heart disease, diabetes, stroke, and arthritis for these different interventions are shown in Figures 40–43. Scale apart, these graphs can be considered as showing the relative prevalence of the individual diseases.

The behaviour of all interventions should be familiar by now. In particular it is worth noting that it will take about 40 years to realise any appreciable disease-related financial gains. Of course, there will be gains in quality of life[21].

Figure 39: Batch 3 NHS costs for four simulations as listed in table 10.

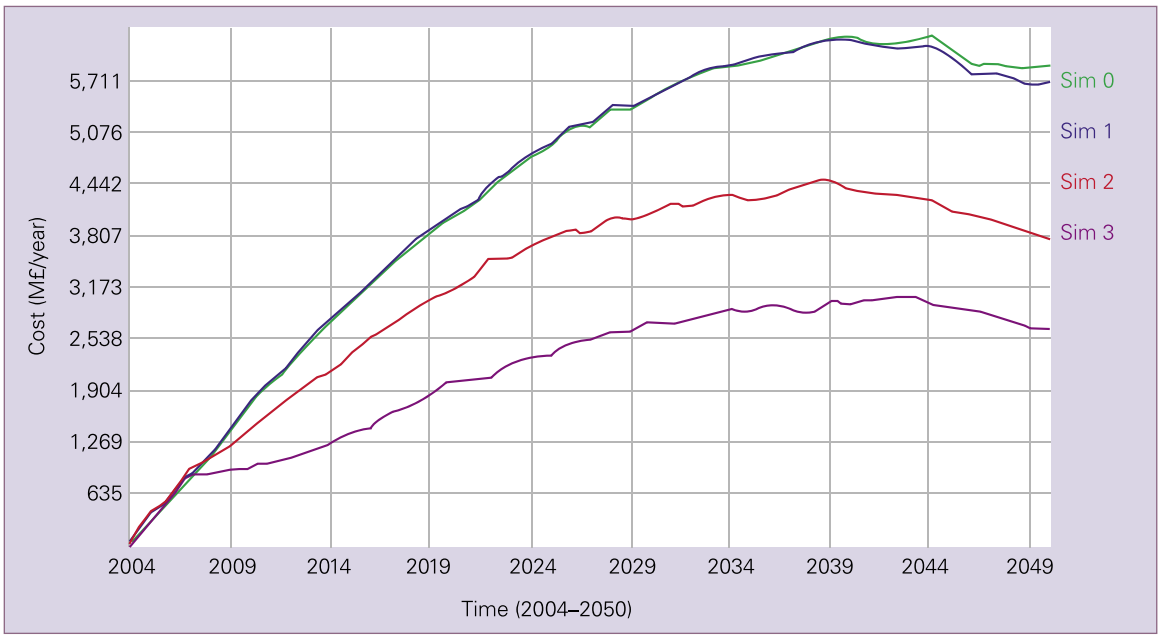


Figure 40: Batch 3 BMI attributable coronary heart disease costs by year

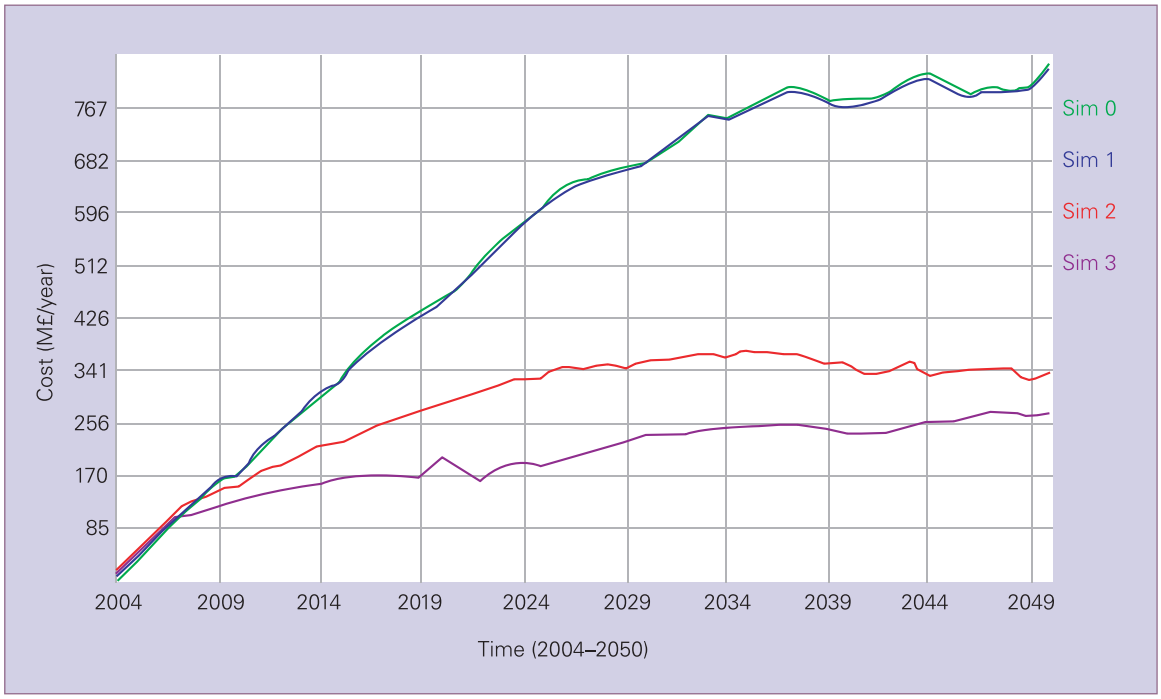
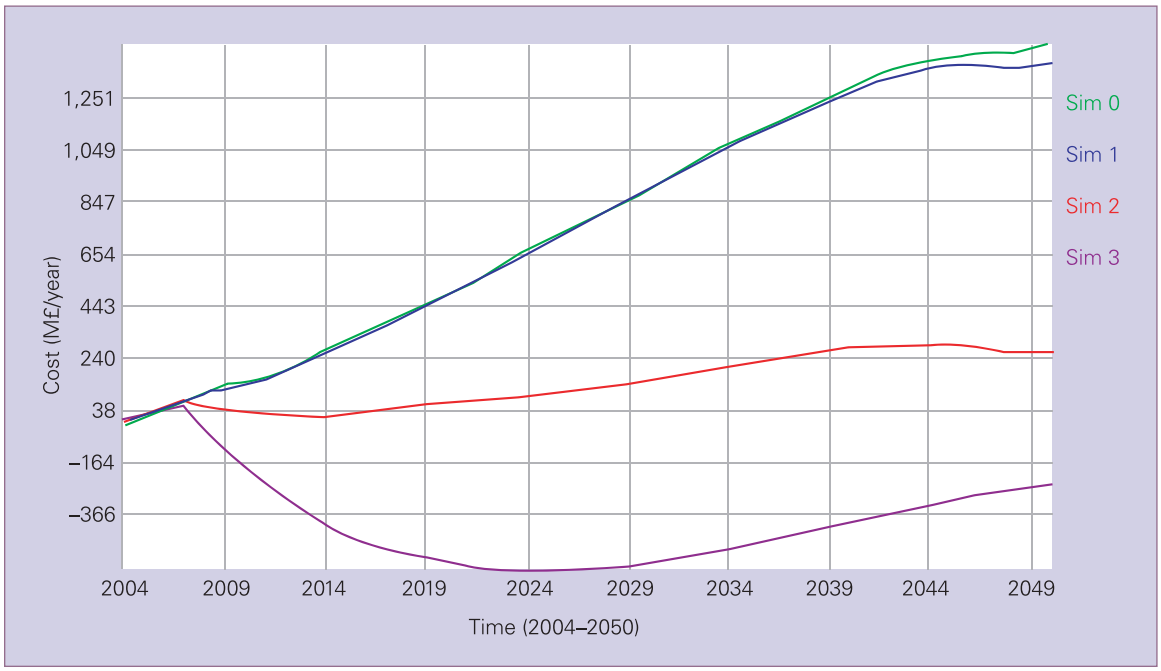


Figure 41: Batch 3 BMI attributable diabetes costs by year



Again, as should be familiar, the benefits of reduced BMI on the prevalence of arthritis can be seen in Figure 43. For Sim 1 there is only an appreciable effect 20 years after the intervention start.

Figure 42: Batch 3 BMI attributable stroke costs by year

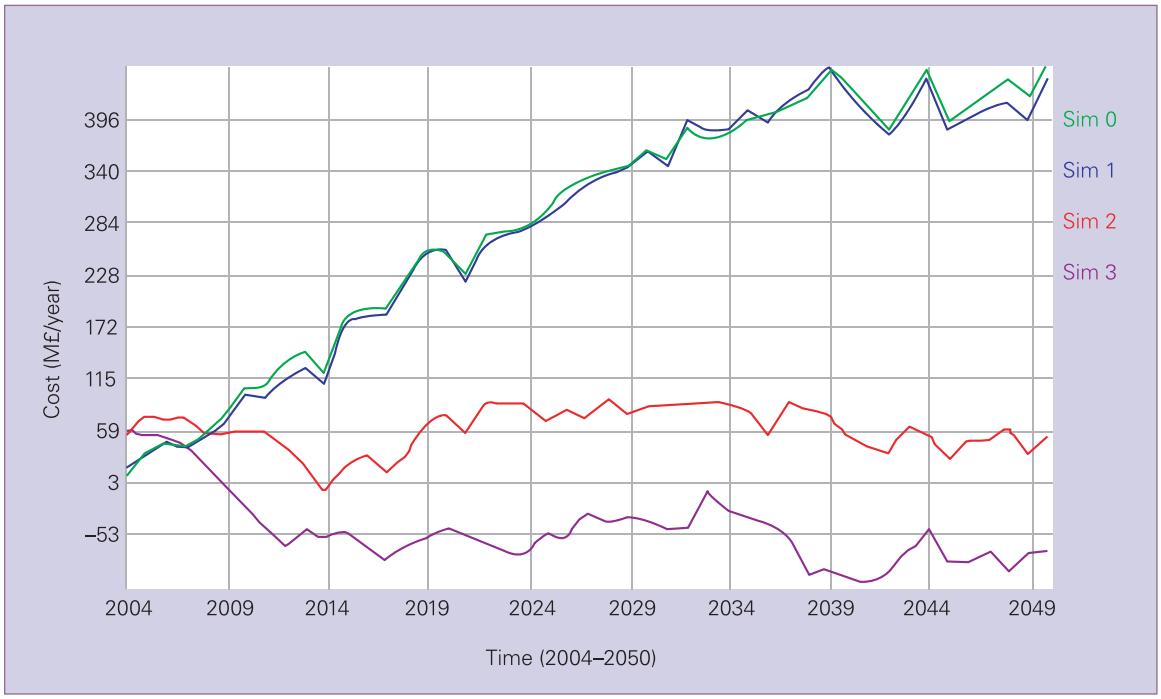
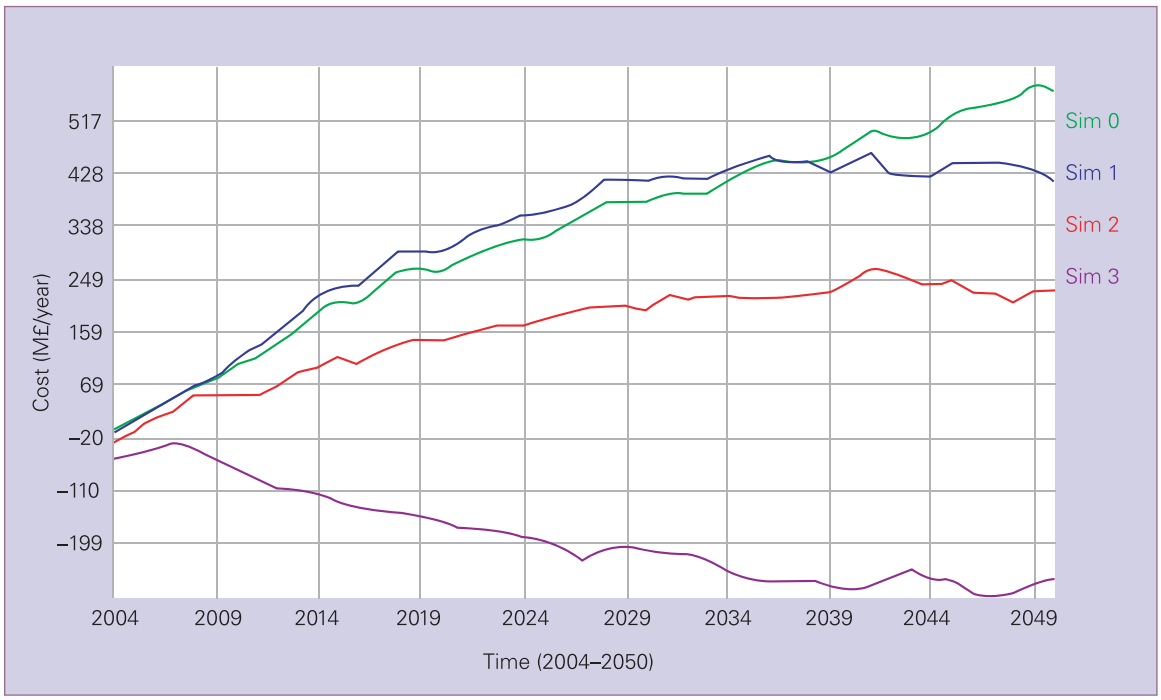


Figure 43: Batch 3 BMI attributable arthritis costs by year



As has been stated, incidence statistics are automatically logged by the microsimulation. At this point a useful example is provided by showing the incidence rates by age for coronary heart disease and arthritis. These are shown in Figure 44 and Figure 45.

Figure 44: Batch 3: Coronary heart disease incidence by age

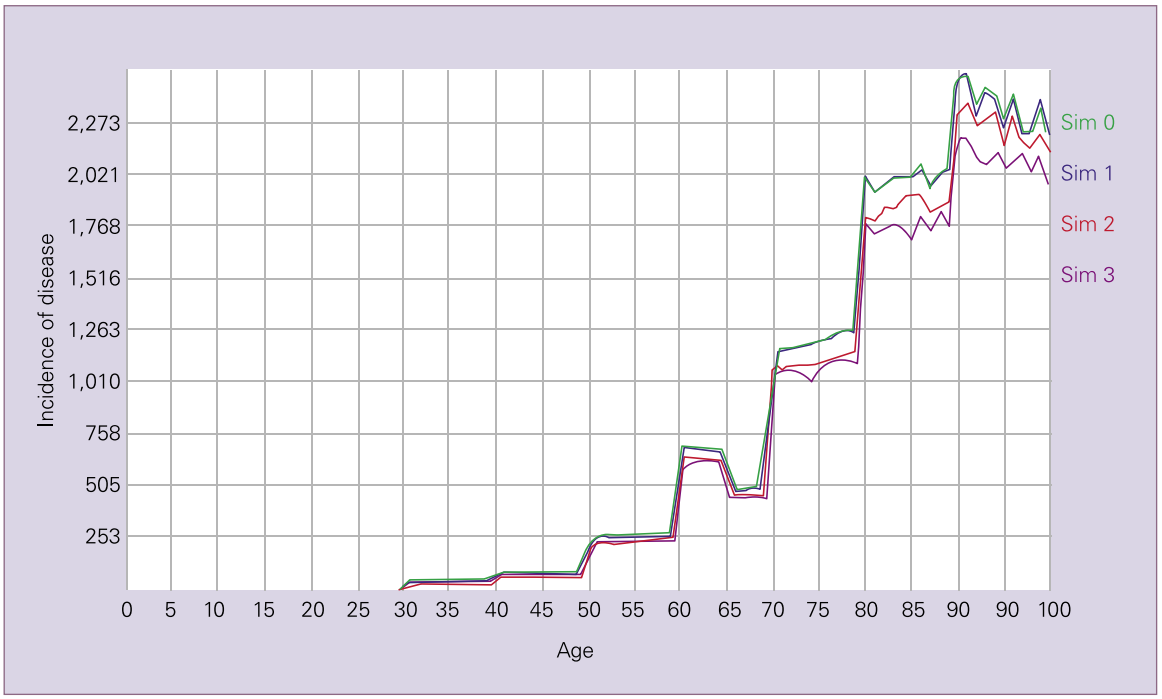
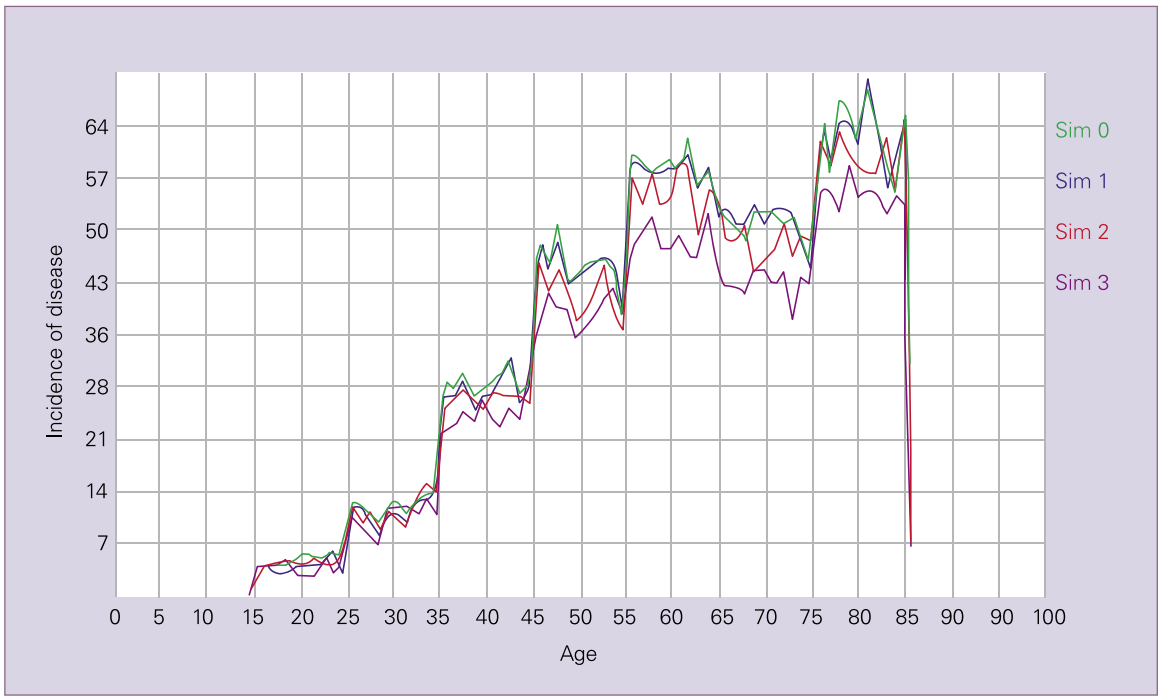


Figure 45: Batch 3: Arthritis incidence by age



Notice the earlier onset and greater difference between the simulations in the case of arthritis. The step-like character of these graphs directly reflects the step function character of the input statistics.

6.5 BMI distribution histories for PSA targets (Batch 3 simulations)

For Simulation 0, the microsimulation generates a random population whose BMI statistics reproduce (in a stochastic sense) the input BMI statistics. There are a number of ways in which this can be checked by the program. Perhaps the simplest is to plot the simulated distributions by year together with the input distributions. This is done in the pair of graphs, Figure 46 and Figure 47.

The graphs drawn are for the entire population. The program has the capability to draw the same comparison for any particular age group. The results are all much the same: there is good agreement consistent with the convergence expected for the number of Monte Carlo trials – 1,000,000 for the above.

Away from Sim 0, the changes to the disease incidence reported in Section 0 are fuelled by the reductions in the BMI as specified in the various simulations. The next six graphs show the simulation results for the three remaining simulations of the batch. In order not to clutter the graphs, only the three highest BMI categories are drawn. In each graph the grey curves of Sim 0 are shown to make possible a comparative assessment.

There are few surprises. The jump, most noticeable in figures 52 and 53, is caused by the start date of the simulated intervention being 2008 while the simulation run starts at 2004. The data used to draw these graphs is gathered at five-yearly intervals from the intervention start.

Figure 46: Batch 3, Simulation 0 BMI distributions [male]. Comparison of simulated (black) and input distributions (colour).

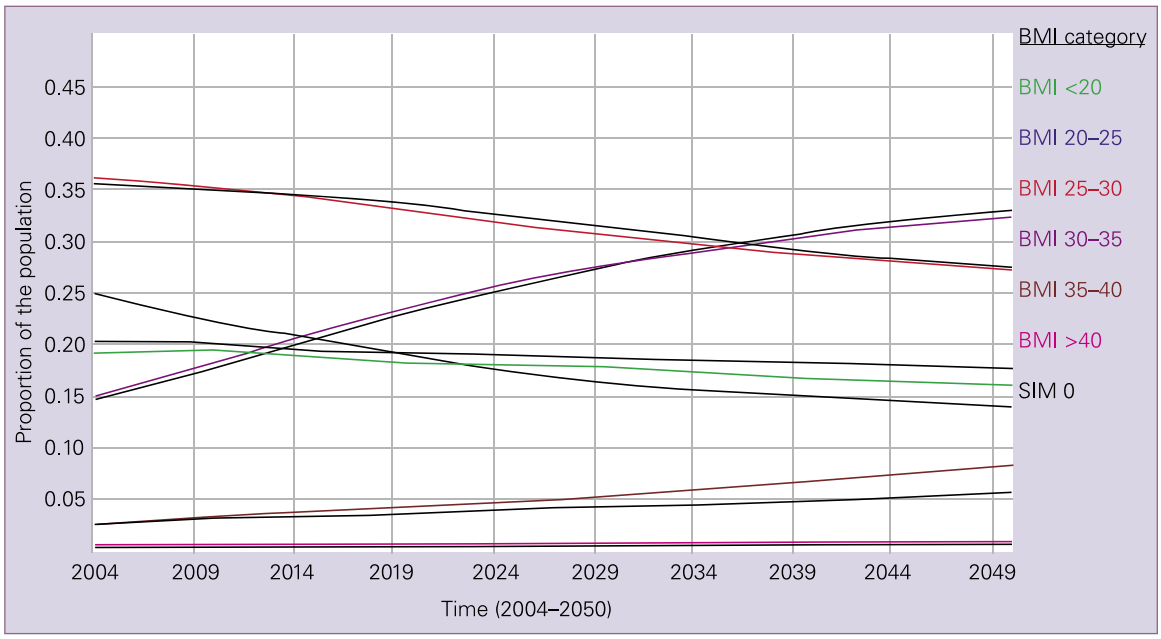


Figure 47: Batch 3, Simulation 0 BMI distributions [female]. Comparison of simulated (black) and input distributions (colour).

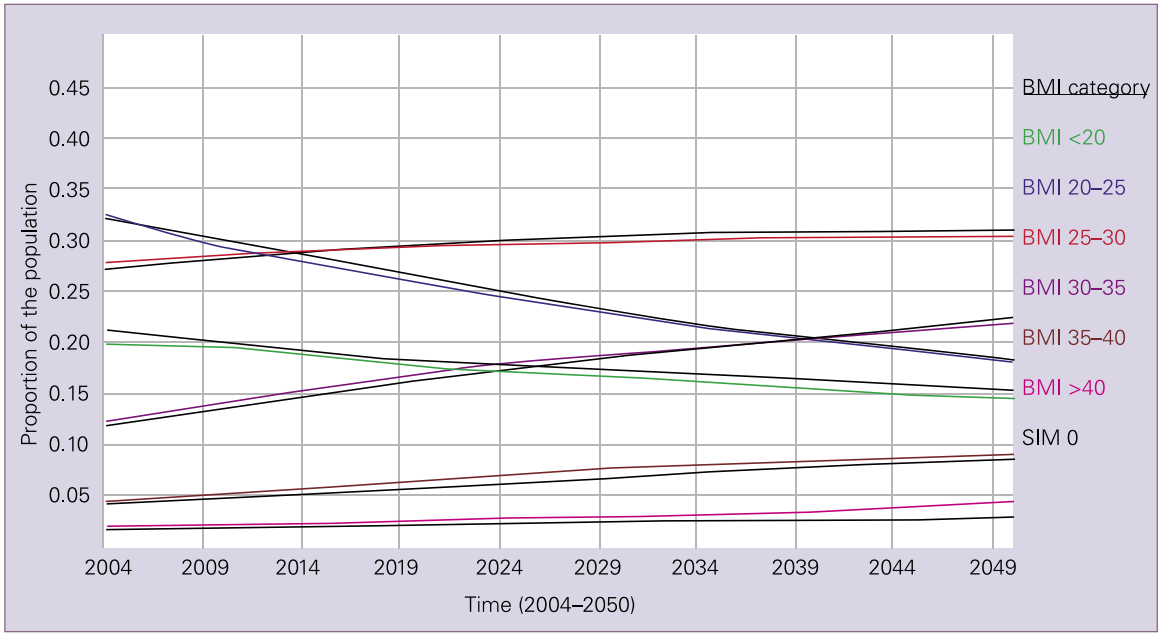


Figure 48: Batch 3, Simulation 1 BMI distributions [male]. Comparison of simulated (black) and input distributions (colour).

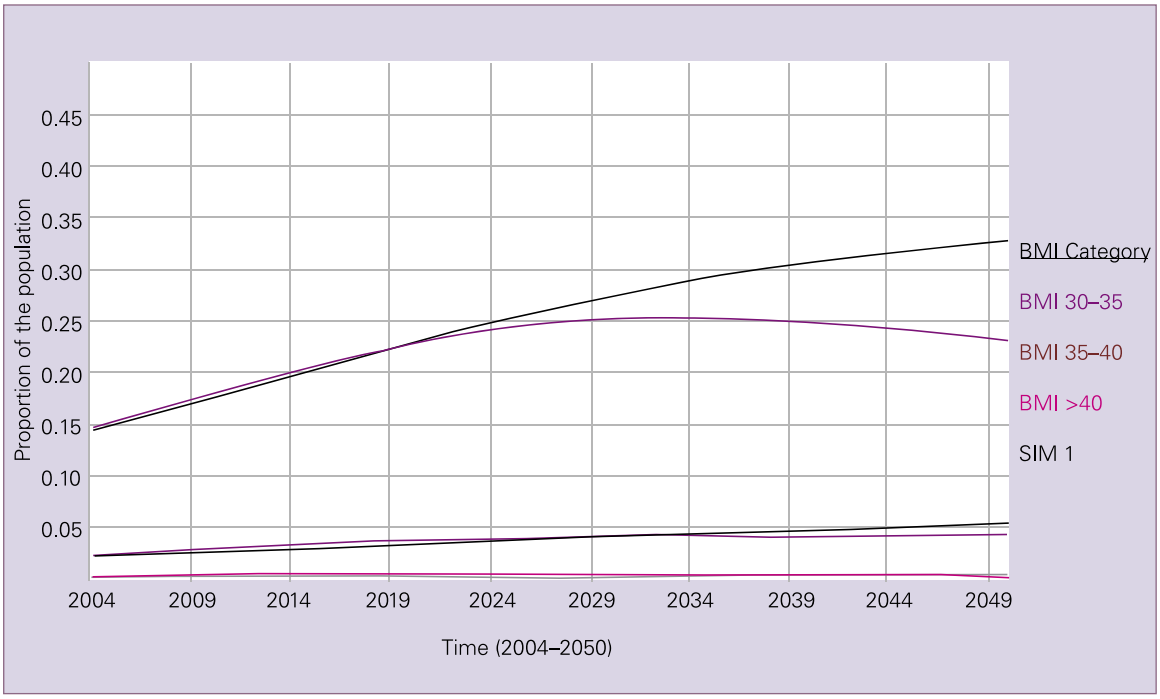


Figure 49: Batch 3, Simulation 1 BMI distributions [female]. Comparison of simulated (black) and input distributions (colour).

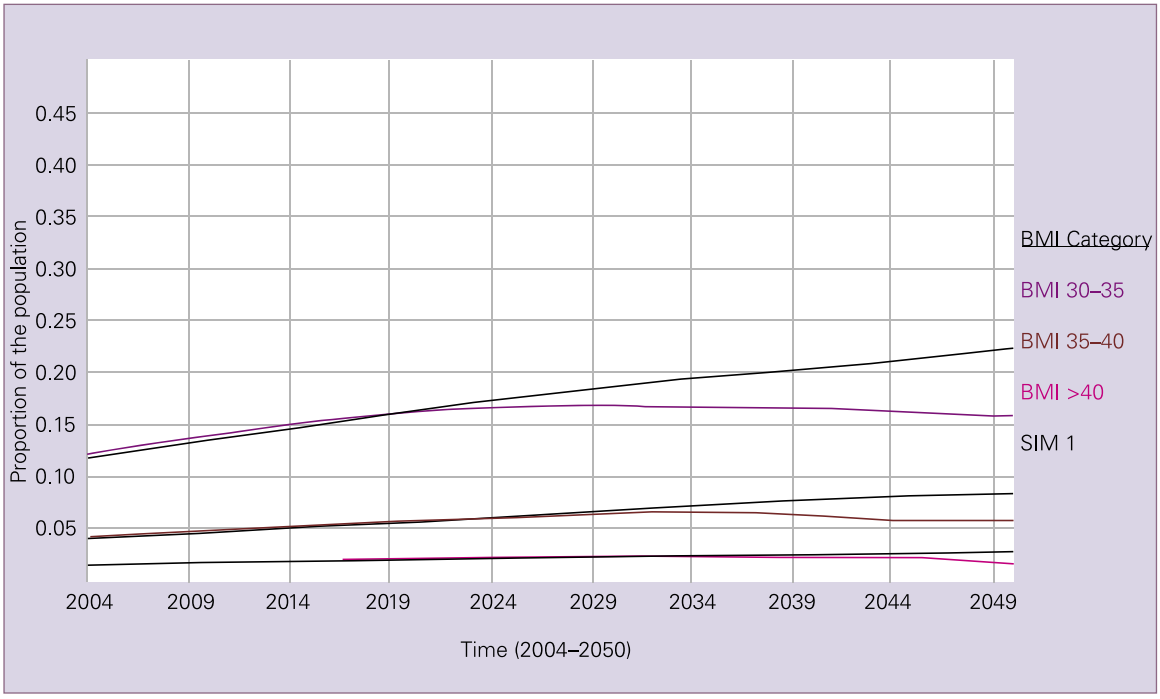


Figure 50: Batch 3, Simulation 2 BMI distributions [male]. Comparison of simulated (black) and input distributions (colour).

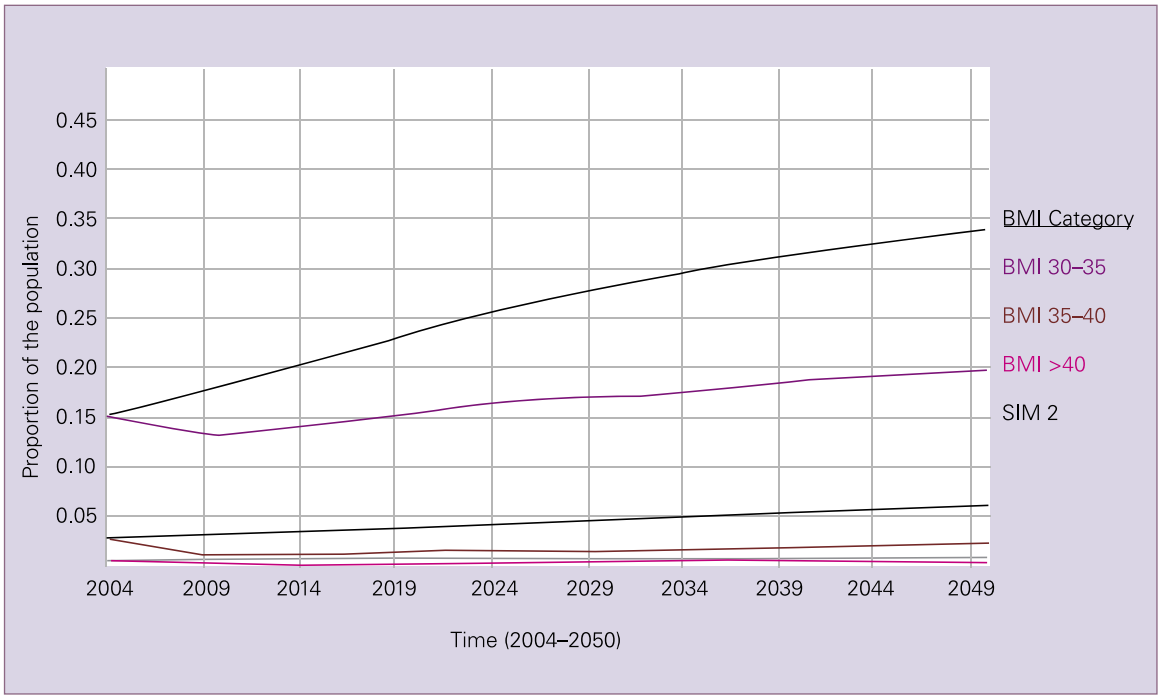


Figure 51: Batch 3, Simulation 2 BMI distributions [female]. Comparison of simulated (black) and input distributions (colour).

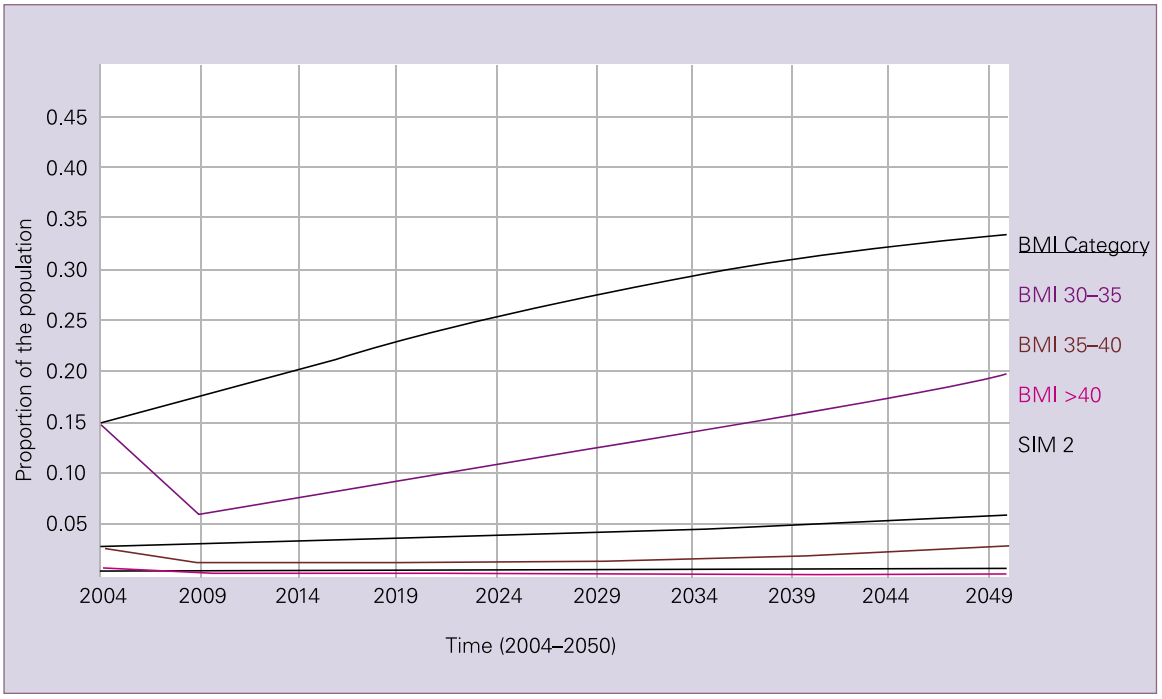


Figure 52: Batch 3, Simulation 3 BMI distributions [male]. Comparison of simulated (black) and input distributions (colour).

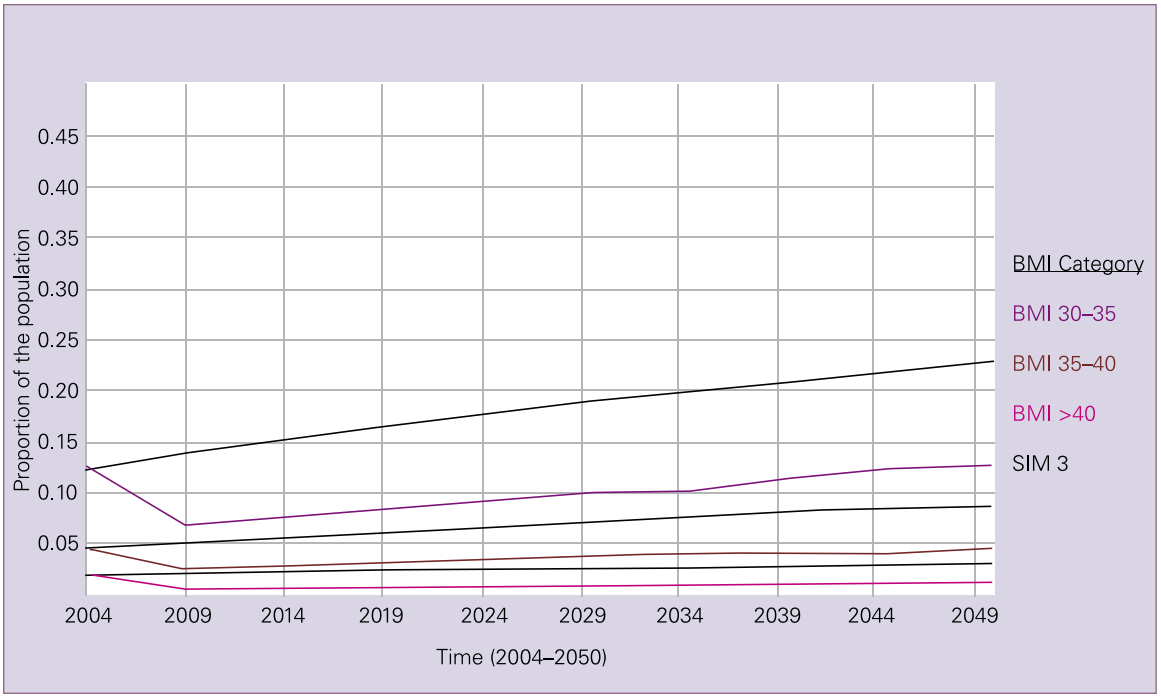
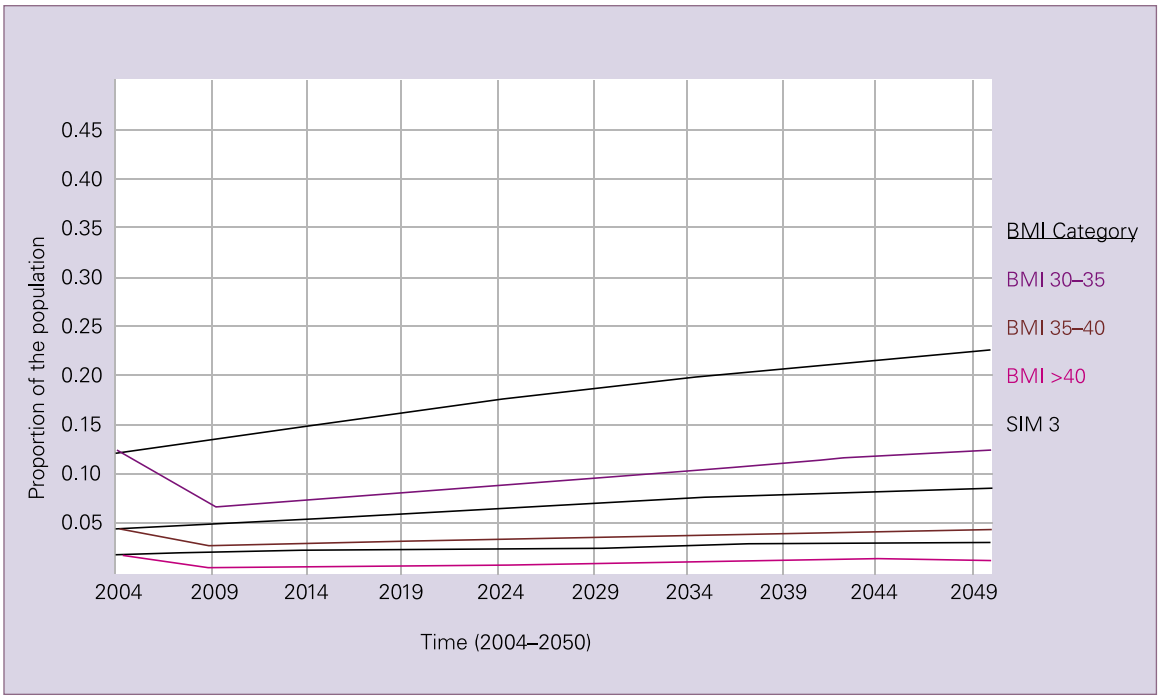


Figure 53: Batch 3, Simulation 3 BMI distributions [female]. Comparison of simulated (black) and input distributions (colour).



7 Discussion

Rates of obesity and overweight have been increasing in the UK since the mid-1980s. They are showing no evidence of a general decline and are projected to continue to rise until 2010[1]. This modelling exercise is intended to predict further the growth or otherwise of obesity rates through to 2050 and to predict the consequences for health, health costs and life expectancy. Understanding the consequences of this growth is complex and not amenable to normal epidemiological extrapolation.

Assessing the potential population benefit of a health intervention requires consideration of many elements, including disease prevalence, population characteristics, effectiveness and cost.[22] Modelling is being increasingly employed in policy making and resource allocation because it permits policy makers to simulate the effects of different sets of circumstances within a population and therefore to examine future policy options.[23] Weinstein defined a model as 'a logical mathematical framework that permits the integration of facts and values to produce outcomes of interest to clinicians and decision makers' or, alternatively, as 'an analytical methodology that accounts for events over time and across populations based on data drawn from primary or secondary sources'.[24]

In order to forecast BMI rates and the attributable disease risks over the next 50 years, it was clear that a cell-based macrosimulation would not be a satisfactory methodology, since too many risks apply differentially to too many groups to allow accurate computation. In addition, individuals accrue risks as individuals – for example, diabetics are at greater subsequent risk of coronary heart disease by an amount that depends on gender, while the baseline risk depends on age. The only way to make progress is to model the anticipated growth and apply it to simulated individuals in populations among which known risks apply.

A dynamic microsimulation model was developed to address the problem. While microsimulation modelling is relatively well employed in other areas of public policy, particularly in economics, its usage in health remains rare. This project probably represents the first major development and utilisation of a dynamic microsimulation of chronic disease in the UK.

The anticipated growth in obesity in almost all segments of the population is quite alarming, and the analyses of the Health Survey for England give a strong impression of continued inexorable growth. There is uncertainty about the growth in the numbers of morbidly obese people, particularly men. The weighing scales used by the Health Survey for England do not give accurate results over 130kg, so the data only provide estimated weights for people over 130kg. Since weight tends to be underestimated, the reported data on BMI is therefore likely to be on the low side and the Survey projections may underestimate the true position. If

so, the predictions made in this modelling exercise may be conservative. But of course, to increase appropriately the proportion of the population that is morbidly obese, if this was possible, would result in a commensurate reduction among the obese alone in these circumstances.

Any attempt to predict any trend so far into the future, whatever method is used, including a microsimulation, is always compromised by lack of knowledge and the questions this raises. But, although the ultimate determinants of a phenomenon like obesity are essentially social, albeit through biological mechanisms, the stability in the trends in BMI of the particular social and demographic subgroups with time has demonstrated a consistency within the Health Survey for England data set (1993–2004) that is difficult to ignore.

Clearly, important changes in these social determinants, whatever they may be, are unlikely in the absence of social, fiscal or legislative change. If they took place, this intrinsic stability would be compromised, with consequences for obesity, which is much more difficult to predict. The actual determinants of obesity are generally poorly measured and understood (i.e. determinants of the individual metabolic balance of energy input with output over long periods of time), and so predicting obesity from changes in measured determinants would be far less precise than predicting the outcome (BMI) among groups of the population, as has been done here.

Detailed epidemiological analyses of the pattern of change observed among subgroups with time are suggested, given the limitations of the data among small groups. This method of prediction will, however, take no account of the possibility of earlier death among the obese, which is why a microsimulation is, in principle, important.

We believe that this work demonstrates that a public-health-orientated, dynamic microsimulation model, although it certainly has its limitations, offers the best tool for estimating future levels of avoidable chronic disease and anticipating, as has been done here, the scale of interventions required to have a significant impact. So, while microsimulation modelling may not be as cheap to establish as macrosimulations, given their greater flexibility and taking into account the potential costs of the impacts they are forecasting, it still appears to be good value.

8 Next steps

This microsimulation Obesity 2 offers a demonstration of the potential application of microsimulation methods in public health. It needs to be consolidated and validated more rigorously than the time constraints of this project have so far allowed. There are probably some serious calibration issues to be undertaken to make, for example, deaths derived from incidence and survival among the obese consistent with current data, such as it is. It needs to be refined to accommodate more detailed specification of policy targets by age, gender, social class etc.

It has, as yet, only addressed future obesity rates in England, although the original intention was to include analysis of obesity in the devolved nations. However, time constraints and availability of data precluded this. The authors hope to be able to complete the picture for the UK at a future date and to further validate the microsimulation with data from other countries.

In addition, within this model, the implications of falling coronary heart disease, stroke and cancer rates have not been fully explored, nor have the possibilities of significant latent effects of obesity on disease been explored. For instance, familial patterns of obesity transfer, possibly moderated by class and gender, have not been incorporated.

Obesity is a complex, multifactorial disease that not only has a significant impact on physical health but also on psychosocial well-being and therefore on quality of life. Obese people experience substantial impairments in quality of life as a consequence of their weight, and these can impact significantly on their mental health, which in turn can further impact on their physical health. It is not yet clear how quality of life differs among different subsets of weight and genders, ethnicities etc., but the literature is growing and should be incorporated into future versions of the obesity model. This also applies to the economic modelling we have employed, which largely concerns itself with direct costs to the NHS attributable to BMI. The cost, or burden, of obesity and overweight should be measured both by the loss of life years and quality of life and by the financial impact of related disease on the health system (direct costs) and on society (indirect costs).

Another factor for future consideration is the relationship between BMI and blood pressure, which appears to be linear and exists throughout the non-obese range. We have modelled some of the interelationships between the relative risk factors related to obesity, but we may not have fully considered the strength of the association of obesity with hypertension, which varies among different racial and ethnic groups. Generally, risk estimates suggest that approximately 75% and 65% of the cases of hypertension in men and women, respectively, are directly attributable to overweight and obesity, particularly metabolic syndrome, which is present in approximately 20% of adult populations in developed countries and in approximately 80% of people with type 2 diabetes. Hypertension has not been

considered here, in part to prevent any double counting of the relative risks of disease.

However, future models should take a detailed account of this because, as levels of obesity are increasing, the occurrence of metabolic syndrome is also likely to increase with the rising levels of obesity and will contribute perhaps further to the epidemic of diabetes that has already been highlighted by the obesity model. Metabolic syndrome is likely to have a marked impact on the prevalence of heart disease and type 2 diabetes in the next two decades. Other medical conditions, such as fatty liver disease, polycystic ovary syndrome, gallstones, sleep apnoea and certain cancers, will also increase with the increased level of BMI.[25]

The influence of obesity on the incidence of metabolic syndrome has also been observed in children in recent years and obesity is now an important cause of type 2 diabetes occurring in children. Some ethnic groups have a higher predisposition to central obesity than others, for example, the amount of central obesity is greater in South Asians than Europeans and is greater in Europeans than African-Caribbeans. A future version of the model would examine these relationships in more detail. The Health Survey for England data employed in this model precludes this level of analysis within any acceptable confidence limits. However, the incorporation of other datasets into the model would enable greater understanding of ethnic differences in relative risks and, particularly from cohort data, would provide a better understanding of the BMI rates over the life course.

There also needs to be translation from the Foresight Tackling Obesities: Future Choices project qualitative scenarios[26] into a quantified specification of when and how obesity and its distribution might change, by how much it might change and who might change them. In principle, all such developments can readily be accommodated in the existing model, even if the current generous specification of each simulation is inadequate. The specifications can be modified appropriately.

The microsimulation has been developed to be flexible in its application. With further work, it could test other datasets from other countries. The model could be extended to further incorporate other behavioural risk factors notably associated with avoidable chronic disease, including smoking or alcohol consumption. This would provide a fuller picture of the attributable morbidities and mortalities associated with such health-related behaviours. All of these factors could be accommodated within the existing program's structure. As it is, the model has rapidly evolved into a powerful policy tool, which is easy to use and to interpret. It needs to be tested, used and further developed to achieve its full potential.

The results from the microsimulation demonstrate rapidly escalating health costs, and particularly an increasing burden from diabetes, if the prevalence of overweight and obesity continues on the trajectory observed in England in recent years. This will give rise to the increasing importance of obesity as a public health problem. These trends currently appear inexorable and BMI distributions

eminently predictable, in which case the importance of the policy implications for a considerable period are clear. The methods developed allow a more sensitive tool than has been available until now, both for the monitoring of obesity trends and analysing in real time the consequences of obesity growth and their policy implications. In this way, changes from the predicted trends can be evaluated and assessed. Any assumptions contained in these assessments can be revisited, altered and the consequences efficiently re-evaluated by policy makers.

Some of the predictions reported are surprising, particularly the low estimated effect on life expectancy. Only one set of epidemiological relationships has been examined and the sensitivity of these results to plausible variations in epidemiological knowledge would be interesting. Moreover, the relationship of more cardiovascular-sensitive measures of obesity (such as waist:hip ratio measurements) could, given the data, result in different predictions.

It is clear that reducing obesity levels and their consequences is complex and will require a much greater understanding of the effects of policy at all levels on obesity trends. Indeed, it will probably require entirely novel and comprehensive policies at many of these levels to have any important effect on burgeoning obesity.

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10 Appendix 1

10.1 Main epidemiological sources used in developing microsimulation

Books:

Obesity. BNF Blackwell 1999.

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10.2 Disease input incidence data

Table 11: Disease input data

Incidence									
Coronary heart disease									
AgeGp	30–40	40–50	50–60	60–70	70–80	80–90	>90		
Male rate/100000	38	61	236	595	1150	1864	2429		
Female rate/100000	10	31	90	275	859	1304	1615		
stroke									
AgeGp	45–55	55–65	65–75	75–85	>85				
Male rate/100000	84	281	788	1631	2600				
Female rate/100000	66	219	613	1269	2000				

diabetes									
AgeGp	0–35	35–45	45–55	55–65	65–75	>75			
Male rate/100000	0	2	5	10	42	122			
Female rate/100000	0	1	3	6	28	109			
cr_C									
AgeGp	0–35	35–45	45–55	55–65	65–75	>75			
Male rate/100000	0	3	13	45	118	173			
Female rate/100000	0	3	8	27	75	193			
br_C									
AgeGp	0–35	35–45	45–55	55–65	65–75	>75			
Male rate/100000	0	0	0	0	0	0			
Female rate/100000	1	17	43	76	116	225			
ki_C									
AgeGp	0–5	6–10	10–15	15–20	20–25	25–30	30–35	35–40	40–45
Male rate/100000	0	0	0	0	0	0	0	0	2
Female rate/100000	0	0	0	0	0	0	0	0	1
ki_C (cont'd)									
AgeGp	45–50	50–55	55–60	60–65	65–70	70–75	75–80	80–85	>85
Male rate/100000	3	7	12	16	22	31	44	50	69
Female rate/100000	1	3	5	7	13	16	20	25	30
oe_C									
AgeGp	25–30	30–35	35–40	40–45	45–50	50–55	55–60	60–65	65–70
Male rate/100000	0	0	1	3	8	17	28	38	55
Female rate/100000	0	0	0	1	2	5	8	13	21
oe_C (cont'd)									
AgeGp	70–75	75–80	80–85	>85					
Male rate/100000	72	94	101	126					
Female rate/100000	32	47	60	68					
en_C									
AgeGp	35–40	40–45	45–50	50–55	55–60	60–65	65–70	70–75	75–80
Male rate/100000	0	0	0	0	0	0	0	0	0
Female rate/100000	0	0	1	2	6	9	15	19	21
en_C (cont'd)									
AgeGp	80–85	>85							
Male rate/100000	0	0							
Female rate/100000	29	39							

gbd									
AgeGp	35–45	45–55	55–65	65–75	>75				
Male rate/100000	5	11	29	93	265				
Female rate/100000	7	11	33	95	517				
arthr									
AgeGp	15–25	25–35	35–45	45–55	55–65	65–75	75–85	>85	
Male rate/100000	3	3	3	9	19	13	54	0	
Female rate/100000	3	12	30	41	48	44	25	0	
unspec									
AgeGp	0–35	35–45	45–55	55–65	65–75	>75			
Male rate/100000	80	191	453	1094	3036	10474			
Female rate/100000	46	114	291	686	1953	9356			

Table 12: Relative risk data for BMI-related diseases

Coronary heart disease

Male	Age [0–65]	Age [>65]
BMI [25–30]	1.35	1.00
BMI [>30]	1.80	1.20

Coronary heart disease

Female	Age [0–65]	Age [>65]
BMI [25–30]	1.40	1.00
BMI [>30]	2.00	1.25

stroke

Male	Age [0–65]	Age [>65]
BMI [25–30]	1.35	1.00
BMI [>30]	1.50	1.15

stroke

Female	Age [0–65]	Age [>65]
BMI [25–30]	1.25	1.00
BMI [>30]	1.60	1.20

diabetes

Male	Age [0–100]
BMI [0–23]	1.00
BMI [23–24]	1.00
BMI [24–25]	1.50
BMI [25–27]	2.20
BMI [27–29]	12.00
BMI [29–31]	30.00
BMI [31–33]	40.00
BMI [33–35]	55.00
BMI [>35]	90.00

diabetes

Female	Age [0–100]
BMI [0–23]	0.80
BMI [23–24]	0.80
BMI [24–25]	0.90
BMI [25–27]	1.00
BMI [27–29]	4.40
BMI [29–31]	6.70
BMI [31–33]	11.60
BMI [33–35]	21.30
BMI [>35]	42.10

cr_C

Male	Age [0–100]
BMI [25–30]	1.15
BMI [>30]	1.33

cr_C

Female	Age [0–100]
BMI [25–30]	1.15
BMI [>30]	1.33

br_C

Male	Age [0–50]	Age [>50]
BMI [25–30]	1.00	1.00
BMI [>30]	1.00	1.00

br_C

Female	Age [0-50]	Age [>50]
BMI [25–30]	1.00	1.12
BMI [>30]	1.00	1.25

ki_C

Male	Age [0–100]
BMI [25–30]	1.36
BMI [>30]	1.84

ki_C

Female	Age [0–100]
BMI [25–30]	1.36
BMI [>30]	1.84

oe_C

Male	Age [0–100]
BMI [25–30]	1.00
BMI [>30]	1.00

oe_C

Female	Age [0–100]
BMI [25–30]	1.00
BMI [>30]	1.00

en_C

en_C

Female	Age [0–100]
BMI [25–30]	1.59
BMI [>30]	2.52

gbd

Male	Age [0–100]
BMI [25–30]	1.34
BMI [>30]	1.78

gbd

Female	Age [0–100]
BMI [25–30]	1.34
BMI [>30]	1.78

arthr

Male	Age [0–100]
BMI [0–18]	0.15
BMI [18–25]	1.00
BMI [25–30]	1.76
BMI [30–35]	1.80
BMI [35–40]	2.11
BMI [>40]	3.88

arthr

Female	Age [0–100]
BMI [0–18]	1.49
BMI [18–25]	1.00
BMI [25–30]	1.63
BMI [30–35]	1.90
BMI [35–40]	1.98
BMI [>40]	3.29

10.3 NHS costs by disease

Table 13: NHS costs – input data

Disease	Cost/year (£ billion)	Year of cost
coronary heart disease	3.45	2004
stroke	4.6	2004
diabetes	1.9	2004
cr_C	0.38	2004
br_C	0.24	2004
ki_C	0.1	2004
oe_C	0.1	2004
en_C	0.1	2004
gbd	0.1	2004
arthr	5.5	2004
unspec	0	2004

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10 The computer program Obesity 1 (*obesity_distribution.exe)* is described in the supporting documents to this report: *Obesity distribution: System definition* and *Obesity distribution: User Guide.*

11 The computer program Obesity 2 (*obesity.exe)* is described in the companion documents to this report: *Obesity: System definition* and *Obesity: User Guide*

12 The notation |B| means the number of elements in the group B.

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