Extending the Critical Path

A report from the Critical Illness Definitions and Geographical Variations Working Party

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The views expressed in this paper are those of some or all of the authors and do not necessarily represent the views of our employers. Any errors or omissions are our sole responsibility too.

Contents

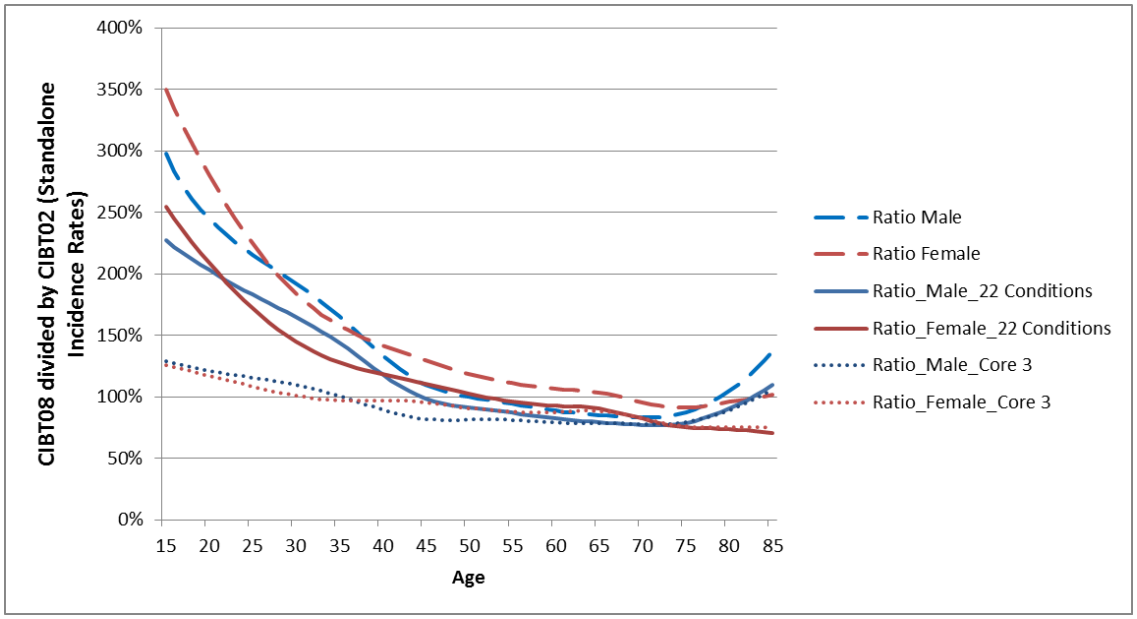
Abstract / Executive Summary

Overview

- This paper summarises our work to analyse a data set of seriatim Hospital Episode Statistics (HES) records. The data related to in-patient admissions in English hospitals between April 1997 and March 2010. Most significantly the data was at individual episode level and allowed us to identify patients across all their hospital admissions so we could build "medical histories" of patients. This in turn has allowed us to calculate "first ever" incidence rates for critical illness conditions directly from the data, rather than relying on approximations.
- In producing our analysis of the dataset we have concentrated on two areas:
    - Production of an updated set of benchmark population rates to replace CIBT02. This new set of rates is to be called CIBT08. It covers a wider range of conditions to reflect the expansion of the number of illnesses covered in the UK Critical Illness market now.
    - Demonstrating the potential effectiveness of geodemographic profilers in assessing morbidity risk. This was motivated by the use of geodemographic profilers to assess mortality rates in annuitants.

New Benchmark Rates: CIBT08

- This paper examined the use of HES data to produce rates for 39 conditions. 36 of these are aggregated to form CIBT08 and 3 are held out separately since they generally result in partial, rather than full sum assured, payments in current product designs.
- The new rates include 15 conditions not included in CIBT02.
- A substantial change to CIBT02 is our allowance for overlap between the different conditions. CIBT02 only allowed for overlaps with conditions with known co-morbidities. Our data allows us to identify all (hospital inpatient treated) morbidities a particular patient has had and count the first ever only. For this reason our allowance for overlaps tends to reduce our rates more so than CIBT02 did.
- Our approach to prevalence calculations also differs to CIBT02. As we have derived our rates allowing for the possibility of any other of the 39 conditions being diagnosed beforehand we must also reduce the exposure to allow for this. Deriving an appropriate reduction has proven difficult and the reader is encouraged to review our approach carefully, especially if older age rates are important to them. To some extent this prevalence effect offsets the overlap effect mentioned above.
- The chart below show the comparison of CIBT02 to CIBT08 for (i) all illnesses except TPD, (ii) the 22 illnesses that are in common between the two tables, and (iii) just Cancer, Heart Attack and Stroke combined.



Comparisons between illness-specific rates derived for CIBT08 and those derived from insured experience for the ACL04 tables suggest that the shapes of the CIBT08 rates are broadly reasonable.

Geodemographic Profilers

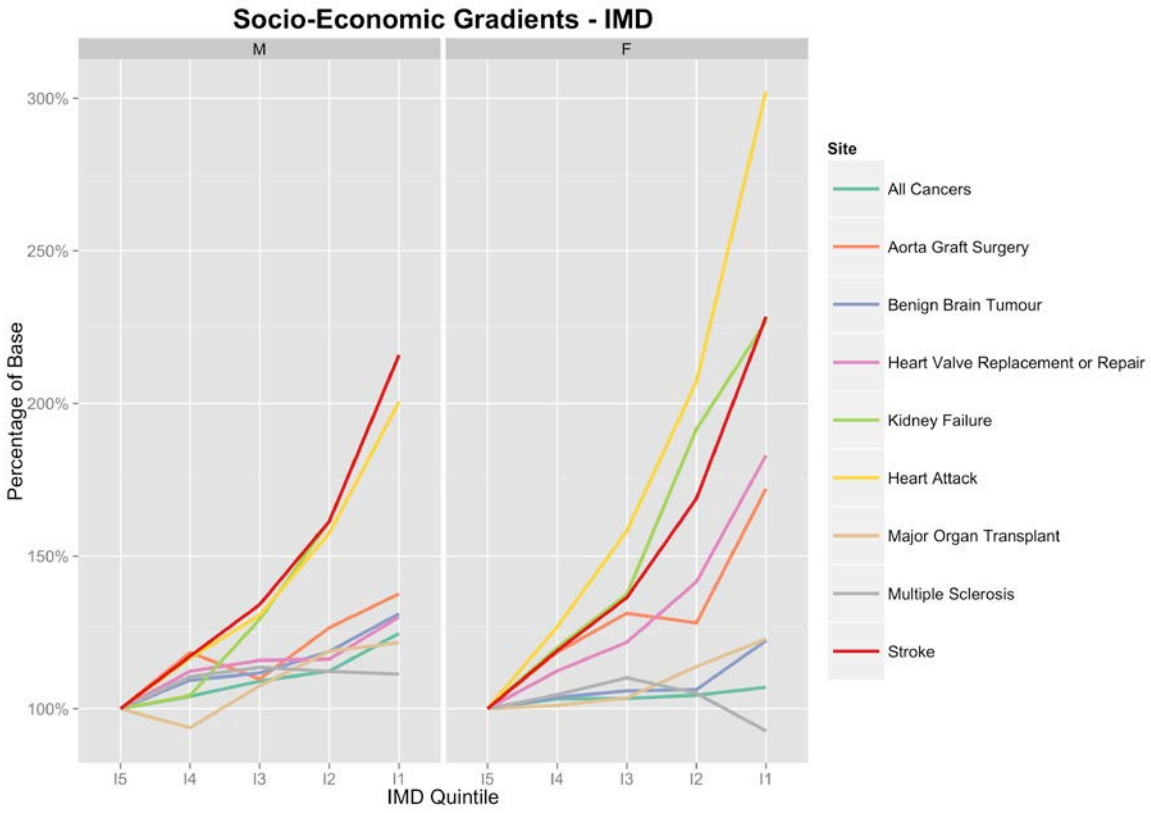
- The HES data we received was coded with the classifications from three geodemographic profilers:
    - The Index of Multiple Deprivations (IMD)[1].
    - CACI's ACORN[2]
    - Mosaic's Experian[3], and
- For each of ACORN and Experian we have created two sets of groupings each consisting, arbitrarily, of six groups. The two sets of groupings are:
    - Top-down, where we have simply used the names of the categories provided by the geodemographic profilers to create an intuitively grouped and ordered set of groups and then tested these to see if they do produce strong gradients in incidence rates;
    - Bottom-up, where we have used the data to produce incidence rates for each of the smallest sub-divisions of the geodemographic profilers and then grouped them to produce six groups which display minimum variance of incidence within the groups and overall. These Bottom-up groups were derived with reference to Heart Attack, Cancer and Stroke incidence combined and then these groupings were used unchanged for all the other conditions where we present geodemographic results.

[1] The 2004 version.

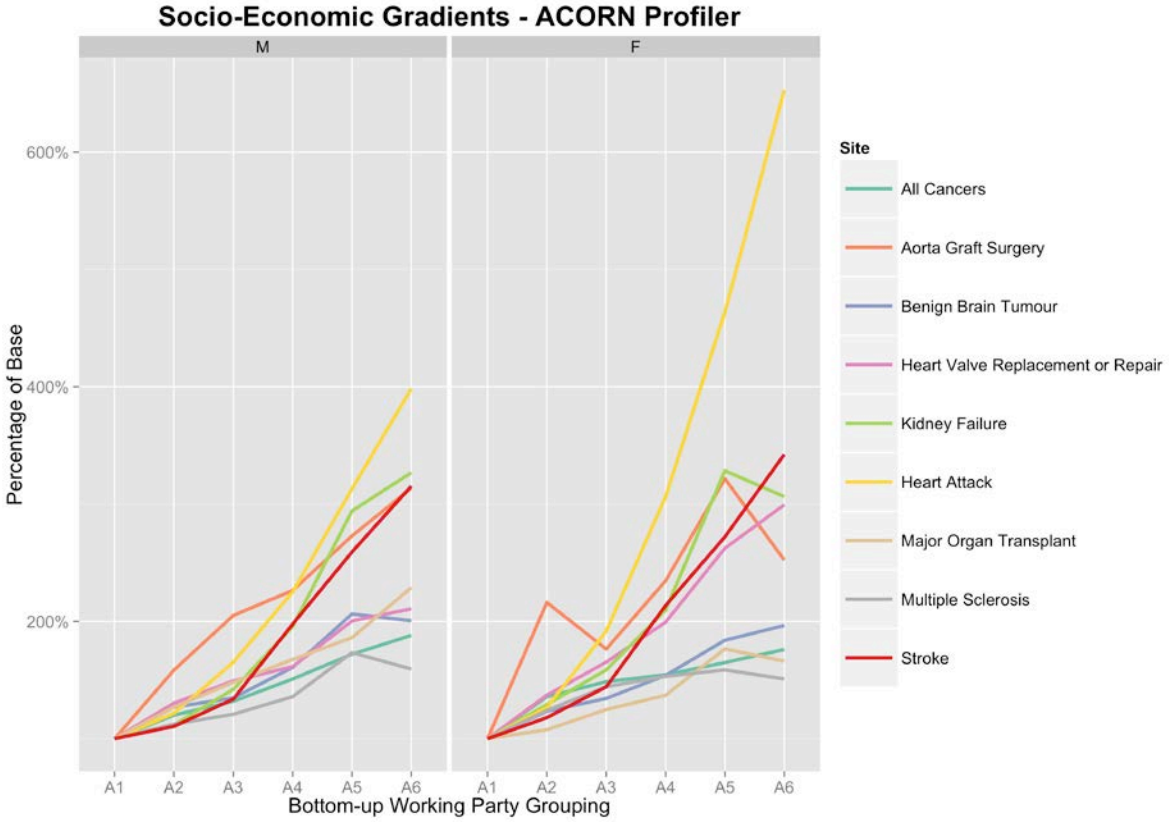
[2] © CACI 1979-2013. All rights reserved.

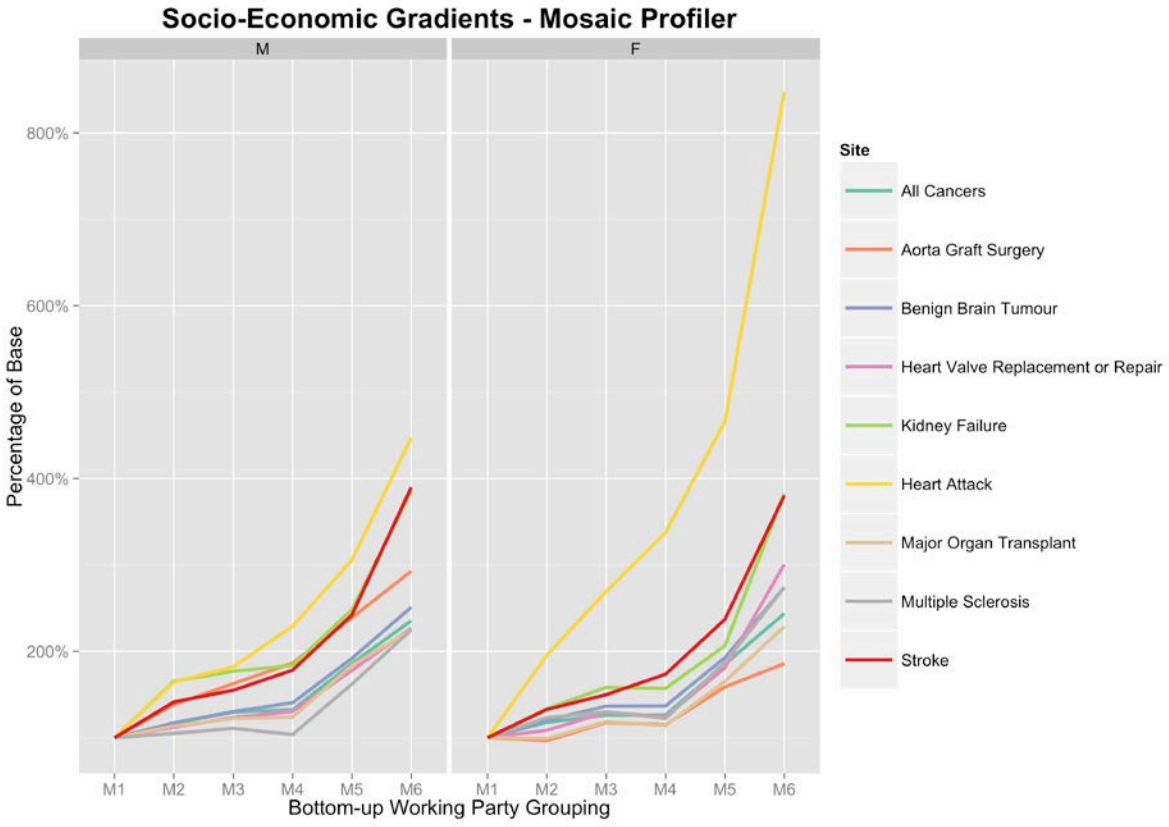
[3] © Experian 2013. All rights reserved.

- Since IMD is naturally an ordinal index of deprivation we have analysed the IMD results by quintile.
- Geodemographic results were produced for a selection of the main illnesses only.
- Our results by IMD quintile show:
    - A positive socio-economic gradient overall (i.e. rates are lower for areas with the lowest deprivation) although the variation for Critical illness incidence is lower than the variation seen for all-cause mortality.
    - Substantial variation by individual illness. For example, Cancer, which is the predominate cause of Critical Illness claims, shows very low variation in aggregate while specific sites show different degrees of variation with some cancers (e.g. Malignant Melanoma) showing even a negative socio-economic gradient. Substantially greater variation is seen for Heart Attack, Stroke, Heart Valve Replacement or Repair, Kidney Failure and Major Organ Transplant. These results are summarised in the graph below:



- Our analyses using Mosaic and ACORN revealed the following:
    - These geodemographic profilers tend to exhibit stronger socio-economic gradients than IMD, which is to be expected given Mosaic and ACORN are postcode level classification systems whereas our IMD results are at Super Output Area level. Whilst this is generally the case there are conditions where the stronger gradient isn't so obvious e.g. Aorta Graft Surgery.
    - Both the bottom-up and top-down groupings exhibit strong gradients. As expected the bottom-up results produce the stronger gradients of the two approaches. The results for the bottom-up results by illness are shown below. (Similar top-down results are included in Appendix 6 for completeness.)


Socio-Economic Gradients - ACORN Profiler
M
F
600%
400%
200%
Percentage of Base
A1
A2
A3
A4
A5
A6
Bottom-up Working Party Grouping
Site
All Cancers
Aorta Graft Surgery
Benign Brain Tumour
Heart Valve Replacement or Repair
Kidney Failure
Heart Attack
Major Organ Transplant
Multiple Sclerosis
Stroke


Socio-Economic Gradients - Mosaic Profiler
M
F
800%
600%
400%
200%
Percentage of Base
M1
M2
M3
M4
M5
M6
Bottom-up Working Party Grouping
Site
All Cancers
Aorta Graft Surgery
Benign Brain Tumour
Heart Valve Replacement or Repair
Kidney Failure
Heart Attack
Major Organ Transplant
Multiple Sclerosis
Stroke

  - For the individual cancer sites we analysed the picture is more mixed. We only used bottom up results here. Generally these have produced a positive socio-economic gradient except for ACORN where our groupings produced an "n" shape for Malignant Melanoma. For all other cases the gradient was positive and for lung cancer amazingly so.

- Overall we would conclude that the use of geodemographic profiling could refine Critical illness pricing bases.

1. Introduction

1.1. Background

This Working Party grew out of an initiative of the Health and Care Practice Executive Committee, as it was then, in 2009 to encourage member led research into Critical Illness insurance. No specific brief was given to the volunteers and initial discussions highlighted two areas where it was felt that practicing actuaries could benefit from additional research, namely:

- The provision of benchmarks to assist the pricing of the proliferation of minor and ABI+ conditions entering the market, and
- Quantification of the variation in experience by socio-economic indicators.

The volunteers initially split into two working parties to concentrate on each topic separately but have recombined to produce this paper for presentation to the Staple Inn Actuarial Society. In coming together the group decided they wanted to expand the remit of the group to include the production of a new population Base Table for UK Critical Illness business, which would be useful to benchmark pricing and valuation basis and provide an update to CIBT02, which was published in 2006.

When considering how to disseminate their work the Working Party decided a presentation to the Society was the obvious choice; SIAS having had the distinction of having been presented the three seminal papers on UK Critical Illness pricing in the past:

- Dread Disease Cover – An Actuarial Perspective, by Dash and Grimshaw (January 1990);
- A Critical Review, by Dinani et al, (March 2000) which included the table CIBT93;
- Exploring the Critical Path, by Robjohns et al, (December 2006) which included the table CIBT02.

All three papers concentrated on developing pricing assumptions for Critical Illness covers starting from population based data. This paper follows that heritage.

A Critical Review and Exploring the Critical Path established the use of Hospital Episode Statistics (HES) data as a valuable data source in developing pricing assumptions for Critical Illness cover. We have obtained more detailed HES data than was available to these previous working parties and this paper presents the results of our analyses.

We have not sought to repeat the broad scope of Exploring the Critical Path but rather to concentrate on the areas of work where possession of the more detailed HES data afforded the Working Party an advantage over what actuaries could achieve in their normal work. Despite this self-imposed restriction in scope we trust the length of this paper is testament to the breadth of the work that the Working Party did achieve.

Insured experience has emerged through the work of the Continuous Mortality Investigation Bureau (CMIB) in recent years. This work, principally and most recently, is contained in CMI Working Papers 50, 52 and 58 and covers the experience between 2003 and 2006. Subsequent years' experience is expected to be published during 2014. We propose that the work contained in this paper is a highly valuable supplement to this work as it provides, inter alia, rates for a wider range of conditions, more detail of socio-economic variations and provides a benchmark to compare population and insured experience which should be of interest to other markets.

1.2. Paper Layout

It is intended that this paper be a valuable reference resource for all actuaries involved in pricing Critical Illness Cover. Providing easy access to salient sections was of prime concern to the Working Party.

The early sections of this paper describe the data we have used:

- Section 2 describes the HES data, including the data manipulation and calculations on that data;
- We then provide an overview of our approach in deriving illness-specific incidence rates in Section 3;
- Section 4 describes the indicators of socio-economic status that we had available to us and the methods we employed to differentiate and group illness-specific incidence rates according to these indicators.
- Section 5 provides information on the construction of the aggregate CIBT08 table, including comparisons to other commonly used UK Critical Illness pricing tables.

The bulk of the paper is then taken up by having one section at a time dedicated to each condition. In an attempt to make this as easy to use as possible for practicing actuaries we have:

- Put the three main Critical Illness Conditions:
    - Cancer
    - Heart Attack
    - Stroke

  in their own section to provide suitable emphasis to the importance of these conditions which constitute between 80% and 90% of all CI claims.
- Arranged all the remaining conditions in alphabetical order in the next major section.

We believe the list of conditions covered includes all conditions in the UK market as at the start of 2013.

The layout of each condition section is standardised as far as possible.

- The first two sections “What is it?” and “Symptoms and Treatment” provide a plain English introduction to the condition.
- “Risk Factors” discusses the reasons why the condition develops, with particular to reference to which of those reasons can be screened out during the underwriting process.
- “Insurance Industry Definitions” catalogues the various definitions found in the UK market for this particular condition. In particular, we note where the definition is included in the ABI Statement of Best Practice for Critical Illness and where companies have exceeded the standardised ABI definition and so claim their definition to be “ABI+”. Given the current explosion of definition variations in the UK market we decided to stop updating the paper for new definitions at September 2013.
- “Derived Incidence Rates” provides the main findings of our work - the incidence rates for the given illness. Results are presented in the form of charts and higher level summary rates with detailed rates for the main conditions being provided in Appendix 6 to this paper. Wherever possible we draw comparison to the CIBT02 rates and also the ACL04 rates.

- Finally, for the conditions with higher incidence rates we include a section showing the results of our "Geodemographic Analysis". These analyses utilise the three socio-economic indicators that we have available to us as outlined in section 4.

1.3. Market Context

The UK Critical Illness market has continued to develop and evolve since the publication of CIBT02 in Exploring the Critical Path. Distribution, product design and pricing have all changed but market volumes have fallen. Critical Illness sales peaked in 2002, when over 1m policies were sold. Although by 2012 sales had fallen to around 550,000 policies the product still remains important at around 30% of new protection premiums.

Market prices for critical illness have come down significantly over that period. This has been driven by a number of different factors but particularly from a reduction in new business processing costs, resulting from increased use of automated underwriting systems, and from reductions in expected diagnosis rates and hence reinsurance rates. The improved price for consumers is even more remarkable given the factors acting in the opposite direction – the rapidly reducing claim declinature rates following the implementation of TCF guidance on claims assessment; and the increasing number of conditions that critical illness products cover. The latter is of course of particular relevance to this paper.

The ABI Statement of Best Practice for Critical Illness published in 1999 covered 20[4] different conditions. Further statements were published in 2004 and 2006 but rather than adding new conditions, these were focussed on introducing severity criteria to "future proof" the main definitions. This largely arose from fears that the trend risks on claims rates over a 25 year time horizon simply made guarantees unaffordable. Hence, severity criteria and exclusions were added improve clarity and reduce risk. For example, the Heart Attack definition became "Heart Attack – of specified severity" and required Troponin (a particular cardiac enzyme used as a diagnostic tool by the medical profession) to be raised above specified numerical levels.

These changes have of course been implemented by offices, but competition on product design has intensified and led to nearly all the products in the market offering materially more cover than the Statement of Best Practice requires. The number of conditions covered now appears to have peaked at around 40, but as a result providers have started to compete in two main areas: by relaxing the severity criteria in the ABI definition, making it so-called "ABI+"; and by adding partial or additional payments of 10-25% sum assured that do not terminate the policy. With these additional conditions, some policies cover up to 60 conditions in total.

In refreshing and extending the work done on CIBT02 and in producing CIBT08, we have attempted to cover as many of these new conditions as possible. However, we recognise that in a rapidly developing market we will always be playing catch-up.

[4] 7 core conditions and 13 additional conditions. The specimen list of conditions shown under paragraph 2.7.10 listed 23 conditions though – the extra 3 (for which there were no standardised definition wordings) were Alzheimer's, HIV via blood transfusion and TPD.

2. The HES Data

2.1. Overview

Hospital Episode Statistics (HES) is a data warehouse containing details of all admissions, outpatient appointments and A&E attendances at NHS hospitals in England. This data is collected during a patient's time at hospital and is submitted to allow hospitals to be paid for the care they deliver. HES data is designed to enable secondary use, that is use for non-clinical purposes, of this primarily administrative data[5].

HES is an episode level dataset where each record relates to one period of finished patient contact with the hospital services, defined as "the time a patient spends in the continuous care of one consultant" before being transferred to another consultant or discharged. Importantly the data is now available with an anonymised patient identifier (the "HESID") which does not allow personal identification of who the patient is but does allow all periods of care for that patient to be identified and linked. Thus we can build up a "medical history" of episodes of hospital admittance with this dataset. This is a significant enhancement to the data previous actuarial working parties had access to since it allows us to develop "first ever" rates directly, rather than having to approximate them.

The data is highly detailed and the data fields fall into four main categories:

- Clinical information about a patient's diagnoses and treatments;
- Demographic information about the patient, such as their age and gender
- Administrative information, for example date of admission and discharge; and
- Geographical information on the location of treatment and the area in which the patient lives.

Uniquely for our dataset, the last of these categories has been augmented with the addition of the geodemographic variables from Experian and CACI as discussed in section 4.

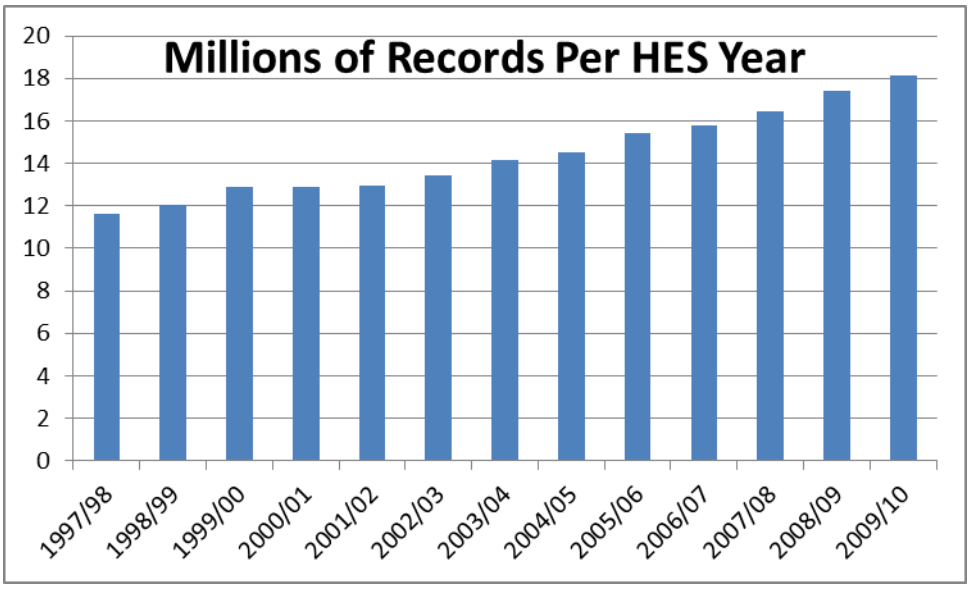
Full details of the data contained within HES can be found on the Health and Social Care Information Centre (HSCIC) website under http://www.hscic.gov.uk/hes

2.2. Our HES data

The HES data used in our analysis is the HES Admitted Patient Care dataset (so any hospital admission which requires a bed to be allocated to a patient), it provides information on admissions to NHS hospitals in England over the period April 1997 to March 2010.

The dataset is very large consisting of approximately 188 million records. The datasets have grown year on year as shown in the graph below:

[5] Source: http://www.hscic.gov.uk/hes accessed 3 November 2013.



We requested and received selected data fields for all the episode records from the HES Admitted Patient Care database, without any filters being applied. Previous IFoA Critical Illness working parties, when requesting their summarised HES data results, have applied filters and so in our work we have considered these differences. Full details of our methodology are provided in section 3.

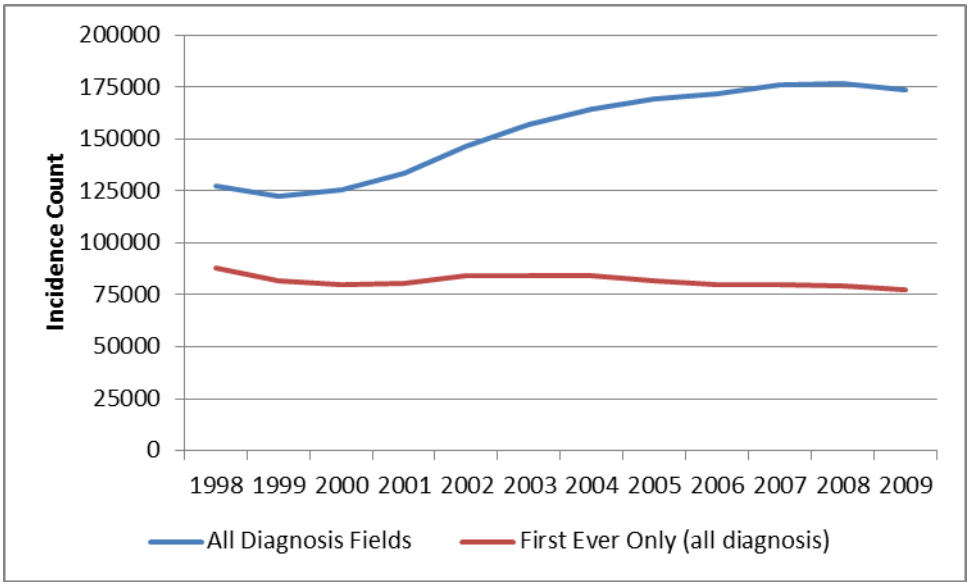
Upon receipt of the data, and throughout our data analysis, we have always sought to reconcile our results to the freely available summary results from the Health and Social Care Information Centre. Whilst these results are not as detailed as those we have generated, they did allow us to confirm that we were interpreting the data correctly.

2.3. Issues with the HES Data

Whilst the HES data represents an enormously useful source of data for generating population Critical Illness incidence rates, there are a range of issues that the user should be mindful of.

- There have been changes in recording practice over time and there are inconsistencies between different coders. Our data series is all post the change to ICD-10 but coding practices do differ between coders and have changed slightly over time. In particular medical definitions of some illnesses have changed over the period.
- The HES data is limited to England and is therefore not representative of the whole of the UK. Indeed, Exploring the Critical Path noted that incidence rates appear to be significantly higher in Scotland than in England.
- HES only include people who go to an NHS hospital. Therefore HES do not include people admitted to private hospitals for treatment nor people who are diagnosed and treated by their GP. This leads to under-reporting of some conditions, for example, dementias. There may be other reasons why the HES data under-reports certain illnesses – where we are aware of these issues we comment on them in the specific condition sections.

- There has been massive growth of the additional diagnosis or operation fields driven by a desire to record all clinically relevant information on the hospital admission record. We have used all the diagnosis and operation fields to be as inclusive as possible because not all CI's are recorded as the primary diagnosis. Given our work to determine first ever incidence of conditions we consider this less of an issue than it would otherwise be but we are very conscious that this change in recording practice could accentuate any upwards trends seen in the data. This is illustrated in the chart below which compares the incidence count for Heart Attacks by calendar year showing how the basic count of each occurrence of heart attack increases rapidly but the first ever incidence count remains relatively flat:



- In theory, there should be a one to one correspondence between individual patients and HESID. The HESID is derived using a matching algorithm mainly mapped to NHS number, but not all records contain an NHS number, especially in the early years, so full matching is not possible. In those cases HES use other patient identifiable fields (Date of Birth, Sex, Postcode, etc.) so imperfect matching may mean patients have more than one HESID. According to the NHS IC 83% of records had an NHS number in 2000/01 and this had grown to 97% by 2007/08, so the issue is clearly reducing. Indeed, our data contains 47.5m unique HESIDs which when compared to the English population of around 49m in 1997, and allowing for approximately 1m new lives a year due to births and inwards migration would suggest around 75% of people in England were admitted at least once during the 13 year period for which we have data. Our view is that this proportion seems a little high but we have been unable to verify that this proportion is reasonable against an independent source.
- HES do not capture data for individuals who die before arriving at hospital.

3. Methodology

For each CI condition we undertook desk-based research to understand the condition. We then used the HES data set to extract crude incidence rates. Finally we conducted further desk-based research to verify the HES data results and to obtain figures to adjust the crude incidence rates as was done for CIBT02.

3.1. Understanding each condition

We obtained descriptions and an overview of the medical management of and the risk factors associated with each condition from a wide range of medical papers and patient resources on the Internet.

We recorded insurance definitions from all released versions of the ABI Statement of Best Practice. We also recorded CI definitions contained in UK insurers' publicly accessible Critical Illness policy documents as at September 2013.

In preparation for the HES data analysis, we defined the ICD-10 diagnosis codes and/or OPCS procedure codes associated with each CI condition. We used the ABI Statement of Best Practice definition as our starting point for conditions included in the standard list and the market norm for non-ABI conditions. It was generally difficult to separate out cases that would meet the ABI+ definitions but not the ABI definition. However, where the HES data has allowed us to comment on the additional cost of ABI+ versions, we have done so in the illness-by-illness sections of this report.

Wherever possible the HES data codes were extracted at the 3-digit level in the interests of efficiency but in some cases we had to extend the definition to the 4-digit level to ensure that we did not include cases that were clearly not covered by the CI definitions. A full listing of the codes we have used is set out in Appendix 2. We have also included a list of the codes used in CIBT02 where appropriate.

3.2. HES data analysis for crude "first ever" incidence counts and overlap factors

The data was converted from a file where each line represents a hospital episode to a file where the history of each life, as they are admitted and discharged throughout their lifetime, can be tracked from 1997. A unique ID (the HESID) has been provided for each life which allows us to track lives across multiple episodes. We have used the first ten years of our data to build up these medical histories and have then only extracted incidence counts based on the last three years of data. We describe later in this section how we have done this.

Similarly to the work underlying the derivation of the CIBT02 tables, we removed unfinished hospital episodes on the grounds that this data is largely incomplete and therefore inaccurate. We also remove "day-cases" on the grounds that this avoids large volumes of duplicates for lives that are admitted but visit the hospital on a regular basis for treatment. In theory these duplicates would be removed as per the step above but this filter is retained to ensure we do not over-estimate counts materially should the unique ID be less than 100% accurate. Appendix 1 contains examples of the numerical impact of these filters. The work in Appendix 1 shows that for Cancer and Heart Attack the difference between excluding day cases or not is insignificant. However, in a condition like Kidney failure this filter may lead to under-estimation in the incidence of Kidney Failure.

Using the relevant ICD-10 and/or OPCS code filters we extracted episode counts for each condition, by age and sex at the time of the episode, in the following ways:

a) All counts by financial year – to allow a reconciliation with freely available HES data

b) All counts by calendar year – in contrast with CIBT02, where a crude adjustment was made from the financial year results, we could use the actual admission date to allocate episodes to each calendar year

c) First ever counts by calendar year i.e. each patient is permitted only a single count for the relevant condition in their lifetime

d) First ever CI counts by calendar year i.e. each patient is permitted only a single condition covered under a CI policy in their lifetime. This effectively allows for overlap across the conditions in c), Where more than one Critical Illness condition is recorded on a single episode record primacy is given to diagnoses over procedures and then by ordering of the HES coding within diagnoses and procedures.

Example

To help the reader understand the differences between the various counts, consider an example of a patient who:

- *Suffered a Heart Attack in 2005;*
- *Had Cancer in 2008;*
- *Had a second Heart Attack in 2009.*

In this case, the Heart Attack event in 2009 would not be counted in either of the first ever counts (c or d, above) because of the prior Heart Attack in 2005, which predates the 2007-2009 period of investigation but falls in our lead in period.

The cancer event would be counted in the first ever count (c, above) since there was no prior cancer event. However, it would not be counted in the first ever CI count (d, above) due to the prior Heart Attack event in 2005.

It should be noted that for each episode in the HES data, up to 20 diagnosis codes and 24 operation codes are permitted. The published free data only provides counts for the primary diagnosis code. For operation counts the free data provides for both main operations and also for all operations. For the purposes of determining incidence rates, we have included all diagnosis/operation codes although of course under c) and d) above, a patient is still only permitted a single contribution to the total count.

The reduction in rates in moving from b) to c) is very significant in some cases. We believe the size of this reduction has increased over the years as the use of more diagnosis fields has become commonplace as hospitals have sought to record all clinically relevant information on their records. The ratio of the rates calculated under c) divided by the rates calculated under b) are included in Appendix 3Appendix 1. But by way of indication the ratios range from 10% (for Multiple Sclerosis) to 99% (for Mastectomy for Ductal Carcinoma in Situ, which is a single operation so is less likely to be recorded multiple times).

We extracted counts across calendar years 2007 to 2009 to strike a balance between using recent data with a long enough lead in period to determine whether the event was the first ever and obtaining credible and smooth data. This used the 3 latest years available but allows a 10-year lead in period.

The resulting incidence rates therefore apply to calendar year 2008. Counts were grouped into 5-year age bands for each gender so that we are smoothing/extrapolated banded rates rather than individual rates.

Wherever possible we compared the results obtained from our analysis of HES data with other sources to determine reasonability.

3.3. Derivation of Standalone and Accelerated CI incidence rates

Once we obtained first ever counts and adjusted for overlap, our method continued to follow the broad approach used in the development of CIBT02 which is described briefly below.

3.3.1. Derived incidence rate

For each condition we determine an incidence rate at age x denoted "I_x" which is used as an input to both Standalone and Accelerated rates. It is calculated as follows:

Numerator

- Raw observed first ever count (count (c) above) LESS
- Reduction in raw counts due to overlap (derived from (d) above) PLUS
- Sudden death gross-up (desk-based research)

In general the overlap is greater at the older ages simply because older lives are much more likely to experience multiple morbidities.

As was done for CIBT02 we ignored other reasons for under-reporting of cases such as private hospital admissions.

Importantly we do not propose further adjustments if the HES data is not a suitable match to the insured definition. Such adjustments are left to the individual reader to develop.

Denominator

- Population estimate LESS
- Prevalence of all the Critical illnesses included in this paper.

For the population estimate we used ONS population estimates for England in 2007 to 2009 as our starting point. These were the values released during 2013 and include the revisions following the 2011 census[6].

Since we are adopting count type d), and the event of interest is the incidence of the first ever covered condition, the denominator should be reduced to reflect the prevalence of lives that have had any of the relevant conditions. We have considered only cancer, heart attack and stroke explicitly as these would have the most material impact in the resulting incidence rate. We considered making an additional small allowance for the prevalence of the other conditions, but this is considered immaterial when compared to the prevalence of the main three conditions. It is also offset by the fact that we are probably over estimating prevalence at the older ages because we have simply added independent estimates of prevalence of Cancer, Stroke and Heart attack and in reality some of these people will likely have had more than one of these three illnesses. Typically, prevalence data is difficult to find particularly if it is required by age but we have used the following sources:

[6] http://www.ons.gov.uk/ons/publications/re-reference-tables.html?edition=tcm%3A77-280885

Cancer	Cancer prevalence in the United Kingdom: estimates for 2008 Cancer prevalence in the United Kingdom: estimates for 2008 Cancer Prevalence in the UK, British Journal of Cancer - 31st December 2008[7]
Heart Attack	Prevalence of CHD, stroke, myocardial infarction and angina, by sex and age, England 2006 (www.bhf.org.uk)[8]
Stroke	

The resultant prevalence rates are shown below, by age band:

Age	Male	Female
20-39	0.7%	0.7%
40-59	5.5%	4.9%
60-79	25.3%	17.8%

3.3.2. Standalone CI incidence rates

The standalone rate for each CI condition is denoted "I'_x"and is determined by reducing I_x for the proportion of cases that result in death during the survival period. CI policies typically specify that claims will not be paid where death occurs within 14 or 28 days of the CI event. For the purposes of this work we have set rates to correspond to a 28-day survival rate.

We determined this value by undertaking desk-based research for significant conditions where this adjustment has a material impact on the resulting standalone incidence rate. For other conditions we have defaulted to four weeks of the annual aggregate population mortality rates although in some cases this will significantly underestimate short-term mortality.

The 6 conditions for which we have made a specific assumption are: Cancer, Heart Attack, Stroke, Major Organ Transplant, Kidney Failure and Coronary Artery Bypass Graft.

Ideally we would have derived these survival rates directly from our data. Unfortunately our HES data only shows deaths that occur in hospital. To obtain a complete view of mortality after hospital admission the HES-ONS dataset would have been required. This dataset links the HES data to the ONS death registry data. The availability of this data is more restricted than access to the HES data alone and we have been unable to obtain access to it directly, although the reader will note we did access it indirectly to help develop our Heart Attack assumptions.

3.3.3. Accelerated CI incidence rates

For accelerated rates we used the same model employed to develop CIBT02, which was that preferred by Dash and Grimshaw[9]. The (CI-specific) Addition for Accelerated Rates, over and above the mortality risk cost, for products which have an Accelerated CI benefit is estimated as:

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[7] http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2720244/

[8] http://www.bhf.org.uk/research/heart-statistics/morbidity/prevalence.aspx

[9] http://www.actuaries.org.uk/research-and-resources/documents/dread-disease-cover-actuarial-perspective

$$I_x - k_x.q_x$$

where

- I_x is the derived incidence rate for the CI concerned
- k_x is the proportion of deaths due to the CI concerned,
- q_x is the all-cause population mortality rate.

For q_x we substituted the m_x values from Interim Life Tables, 2007-09, for England, published by the ONS with adjustments for updated exposures arising out of the 2011 Census. m_x rather than q_x values were used for consistency with the incidence rates calculated.

We derived k_x values from the Mortality in the 21st Century dataset available from the ONS, ignoring the minor geographic inconsistency with the mortality data. Average values over the period 2007 to 2009 were calculated, consistent with the q_x values, in 5-year age-bands.

3.4. Graduation and final rates

The data we have used comes in many different forms, particularly as regards age-banding:

- HES data is available for individual ages but we grouped it into 5-year age bands as an input to our calculation for consistency with CIBT02 and to avoid spurious accuracy where observations are sparse
- Interim Life Tables provide mortality rates by individual age
- Mortality by cause data for k_x values is grouped into 5-year age bands
- Data from other sources was found in broader, irregular, age-bands with little consistency between sources.

We therefore need to:

- Graduate or smooth the data to remove variations caused by random ‘sampling error’
- Interpolate values to individual ages for the final table
- Extrapolate values to the full age range where occasionally necessary
- Convert rates and adjustment data to a common individual age structure to allow the calculations to proceed.

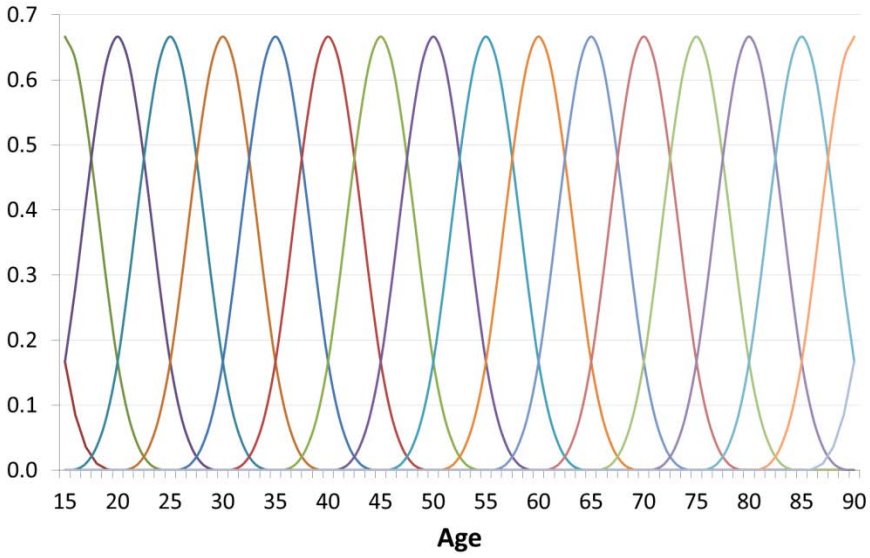
To meet these needs we have applied the same general penalised B-spline technique that was used in the development of CIBT02. The fitted rate for age x (incidence rate, k factor, prevalence etc.) is calculated as follows:

$$Rate(x) = \sum_{i=1}^{18} P_i N_{i,p}$$

where

- $N_{i,p}$ is the basis function of degree p at knot i
- P_i is the coefficient/control point at knot i
- In all cases, rates have been fitted to the age range x=15,…,90

In common with the development of CIBT02, we have chosen 18 cubic basis functions[10] with knots at 5-year intervals (ages 10, 15,…,95). This means that at each given age in the fitted range from 15 to 90 there are 4 non-zero basis functions, as shown in the chart below:



The coefficients are fitted by minimising the following statistic.

$$\sum_j \left(\frac{O_j - E_j}{\sigma_j} \right)^2 + \lambda \sum_{i=1}^{16} \left(\frac{P_{i+2} \div P_{i+1}}{P_{i+1} \div P_i} \right)^2$$

where:

- j is the age band (the number of age bands will depend on the source)
- O_j is the observed rate for age band j
- E_j is the fitted rate for age band j
- σ_i is the standard deviation associated with sampling error on the observed value for the age band j (so typically for rate $p_j = n_j \div N_j$, $\sigma_j \approx \sqrt{(p_j \div N_j)}$, where n_j is observed number of CI events and N_j is the sample population for age band j).
- λ is the smoothing parameter and determines the relative importance in the solution of goodness-of-fit and smoothness; we conducted an exercise to set these using statistical criterion [11] but the results suggested we could adopt a more simple approach of simply choosing λ to give results we felt were reasonable (generally using the same value of λ for all applications of this method to data from a single source).
- P_i are the 18 fitted coefficients, and so the ratio of the ratios of these terms is analogous to the 2nd order differential.

The main advantages of this method are perceived to be:

- The same approach can be applied to each set of data regardless of the underlying age-band structure.

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[10] http://mathworld.wolfram.com/B-Spline.html

[11] Our thanks go to Richard Russell for his work in conducting this exercise.

- The fit is not constrained to belong to a particular class of formulae and so does not require a prior view on form and is less likely to override genuine features of the data.
- The fit is tested across the whole of each age-band in which the data is available, rather than assigning the age-band average arbitrarily to a specific age and interpolating from point to point.
- The fit allows appropriately for the random sampling error / sample size for each data observation and can effectively trade smoothness and goodness-of-fit.

We have generally applied this technique to smooth/extrapolate age-banded rates to individual-age rates at the Crude Rates stage and also to convert all the subsequent adjustments to an individual-age basis before applying them.

Following the method set out above we first arrive central m_x-type rates. We have converted these to initial q_x-type rates using the formula:

$$1 - e^{-m_x}$$

Where x is age last birthday.

3.5. Comparison with CIBT02

As a final step we compared our graduated results to results obtained in CIBT02 and specific commentary on deviations can be found in the illness-by-illness section of this report. The comparison takes the form of two graphs showing:

(1) An age related comparison of the natural logarithm of the standalone CI rates;

(2) An age banded comparison of the various allowances made to move from crude rates to final accelerated CI rates, an example of which is shown below:

The following table describes what has been included in each of the categories in drawing the comparisons in the graph above:

Category	CIBT02	CIBT08
Smoothed, Interpolated Crude Rate (A)	First ever incidence counts including sudden death gross-ups as a % of the total gross population	As per CIBT02, although the first ever counts have been derived directly from the data
Adjustment for Overlap (B)	Reduction to the incidence rates to reflect counts that will already be included in other conditions. Developed cumulatively in order by condition.	All incidence counts have been produced on a “first ever CI” basis and so overlap is automatically incorporated. There is no “order” in the calculation of by-condition rates although the extent of overlap will depend on the covered conditions.
Prevalence Rate (C)	Represents the proportion of the population with the specified condition and is used to reduce the gross population totals. Again this is developed cumulatively in order by condition.	Represents the proportion of the population with a past history of any CI condition and is used to reduce the gross population totals. Hereafter we refer to this as the “Combined Prevalence Rate”.
Other Adjustments (D)	Miscellaneous adjustments used for various reasons including geographical differences of required rates vs. the data source and/or adjustments to reflect insured definitions rather than diagnoses	Not used
Derived Incidence Rate Ix (E)	A × (1-B) / (1-C) × D	
28 Day Mortality Rates (F)	As stated	
Stand Alone Rates I'x (G)	E × (1-F)	
Mortality Rates (H)	Population mortality rates	
Proportions of Deaths kx (I)	Proportion of the population mortality rates associated with each condition	
Addition for Accelerated Rates (J)	E – I × H	

For presentational reasons in the tables of summary rates in the condition-specific sections we show the Adjustment of Overlaps and 28 day mortality rates as negative numbers, because they act to reduce the rates.

Please note that the summary tables in the condition specific sections show age-banded summary results for each set of adjustments made during the rate calculation process. Therefore, lines in the tables which represent the results of calculations based on figures elsewhere in the table may not appear to follow logically. This is purely a function of the calculations being done for individual ages first and then summarised into age bands.

4. Geodemographic Profilers

Geodemographic segmentation is a multivariate statistical classification technique which divides a population into segments where individuals are grouped according to demographic variables, such as income and age, and simultaneously identified by a geographic variable, such as postcode.

Our work has been motivated by the growing use of postcodes for annuity business in recent years. A body of work relating to the use of postcodes when analysing mortality already exists, however, we believe the relationship with morbidity is less established.

There is a number of competing geodemographic segmentation systems available. Within our work we have restricted our focus to the ACORN and Mosaic geodemographic profilers.

4.1. ACORN

ACORN (A Classification of Residential Neighbourhoods) is CACI's geodemographic segmentation system of the UK population. We have used the 2010 version of ACORN which segments postcodes into 5 Categories, 17 Groups and 57 Types. Full details of these types can be found in the Appendices. A new version of ACORN was published in 2013.

4.2. Mosaic UK

Mosaic UK is Experian's geodemographic segmentation system of the UK population. We have used the 2009 version of Mosaic UK which segments postcodes into 15 Groups and 67 Household Types. Full details of these types can be found in the Appendices. A new version of Mosaic is expected in 2014.

4.3. Socio-Economic Analysis

The HES data records have been encoded with both an ACORN Type and a Mosaic UK Household Type. This enables hospital admissions to be split by ACORN and Mosaic Type. This covers the "claims" side of an incidence rate calculation. In order to determine the exposure, both CACI and Experian were able to provide us with the population of England, as at 2009 and 2010 respectively, split by gender, age band and profiler. We used these population counts without adjustment to calculate our rates as at 2008. While we know this to be incorrect we only present relative values and understand that the structure of the UK population by ACORN/Mosaic type did not shift significantly in the intervening period.

This provides all the ingredients to enable an analysis of incidence rates by socio-economic analysis to be performed. However, given the large number of Types we need to group them together in some way to get meaningful results. We have adopted two approaches for grouping.

4.3.1. Derivation of "Top Down" Groupings

The "Top Down" approach is a qualitative method that relies upon the descriptions provided by the segmentation systems for each Group. We start with the 15 Groups provided by Mosaic and combine them based on the description of each. An analogous process is followed for the 17 Groups provided by ACORN.

For both profilers we have aggregated the Groups into 6 "top down" groups, as illustrated in Sections 4.3.1.1 and 0. The following points should be noted:

- The decision to use 6 groups is somewhat arbitrary. Our main motivation was to work with a manageable number that still had a significant number of claims in each group, after allowing for other rating factors;
- The ACORN and Mosaic exercises have been performed independently. We have not attempted to compare the composition of our ACORN groups against our Mosaic groups;
- The process of combining Groups based on descriptions is clearly highly subjective. Many of the decisions could be challenged and a different final set of 6 groups could be obtained.

The key objective here is to investigate the socio-economic gradients we see if, instead of being guided by the data, we are guided purely by the descriptions of different geodemographic profiles as they are suggestive of socio-economic differences. Many of the categories are also suggestive of age but we have not taken that into account in our work since age will be an independent co-variate when we derive rates.

4.3.1.1. Mosaic UK Derivation

For Mosaic UK we aggregate the 15 Groups into 6 "top down" groups as shown below. Based on the Group descriptions we would expect that lower "top down" grouping numbers to correspond with lighter incidence experience.

Top Down Group	Description	% of Population
M1	Alpha Territory Liberal Opinions Professional Rewards	22.7%
M2	Careers and Kids Rural Solitude Suburban Mindsets	23.6%
M3	Active Retirement Small Town Diversity	12.5%
M4	Industrial Heritage New Homemakers	12.1%
M5	Elderly Needs Ex-Council Community	12.5%
M6	Claimant Cultures Terraced Melting Pot Upper Floor Living	16.5%

4.3.1.2. ACORN Derivation

For ACORN we aggregate the 17 Groups into 6 "top down" groupings. We, once again, would expect the lower "top down" grouping numbers to correspond with lighter incidence experience.

Top Down Group	Description	% of Population
A1	Affluent Greys Prosperous Professionals Wealthy Executives	18.5%
A2	Educated Urbanites Flourishing Families	15.0%
A3	Secure Families Starting Out	19.7%
A4	Aspiring Singles Prudent Pensioners Settled Suburbia	12.6%
A5	Asian Communities Blue Collar Roots Post Industrial Families	13.8%
A6	Burdened Singles High Rise Hardship Inner City Adversity Struggling Families Unclassified	20.3%

4.3.2. Derivation of "Bottom Up" Groupings

The "bottom up" grouping is a quantitative approach. We identified a random two-thirds of the patients in our HES dataset and calculated a combined set of first ever incidence rates covering Heart Attack, Stroke and Cancer split by age band and geodemographic type. The exposures used in this calculation were taken to be two-thirds of the total population counts.

This has enabled the calculation of incidence rates, which have then been standardised using a business mix provided by the CMIB. The output is a set of 57 incidence rates for ACORN and 67 incidence rates for Mosaic.

As with the top-down approach, we have presupposed that 6 groups will be an appropriate number. To enable the optimal grouping the incidence rates have been placed in ascending order. A step function has been fitted to the data, where the location of the steps has been varied in order to minimise the sum of the squares of the difference between the incidence rates and the step function. The process has been run separately for ACORN and Mosaic.

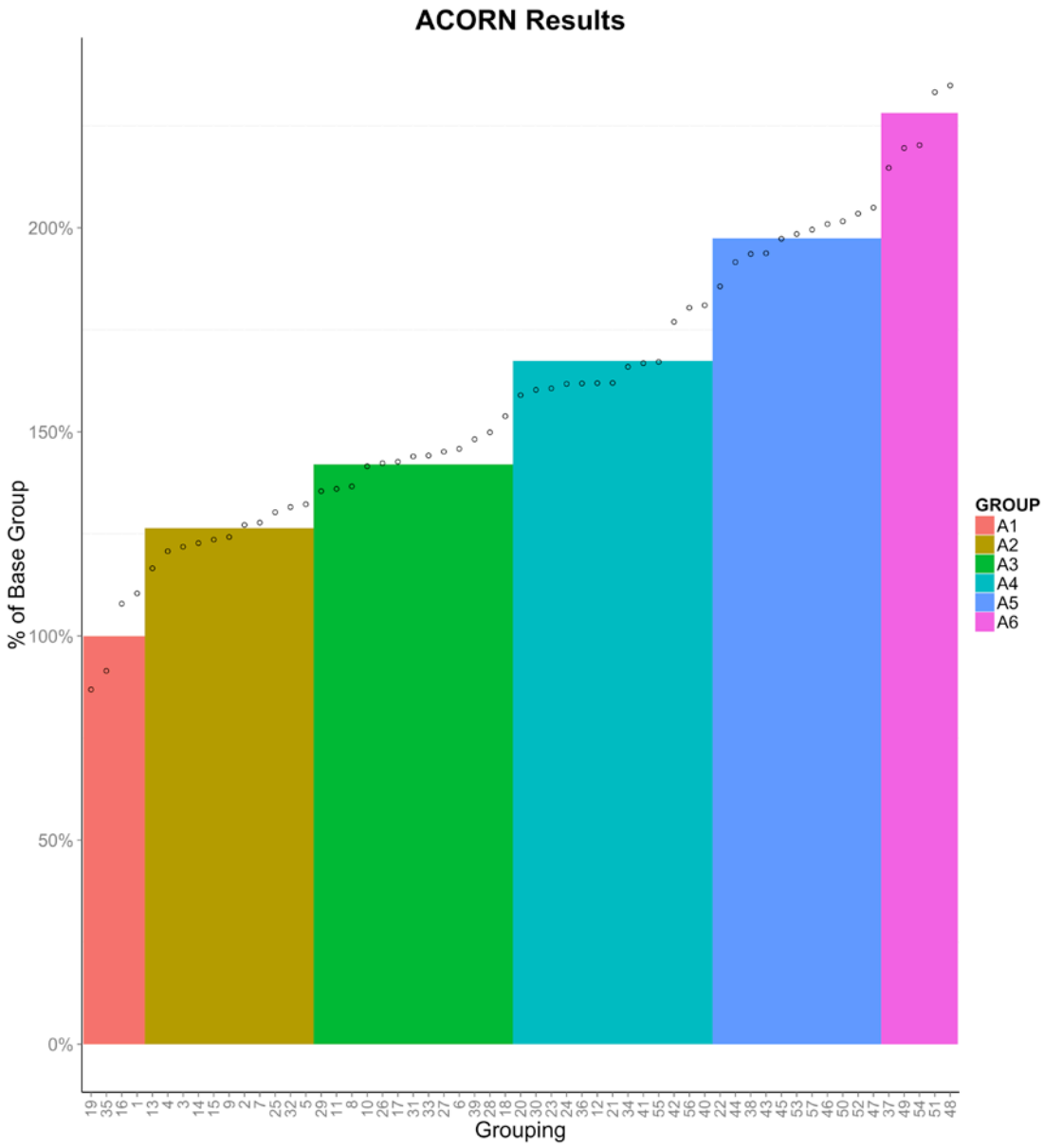
We believe that this approach is consistent with that commonly used within annuity pricing. In contrast to the Top-down approach, here our groupings are driven purely by the mathematics. The following points should be noted:

- We have not analysed the geodemographic groups that have fallen into each of our larger groups as this is not the main purpose of our investigation;
- No analysis of the constituent parts of the ACORN bottom up groups against the Mosaic bottom up groups has been performed.

The optimization exercise has been performed using MS Excel Solver. Note that versions of

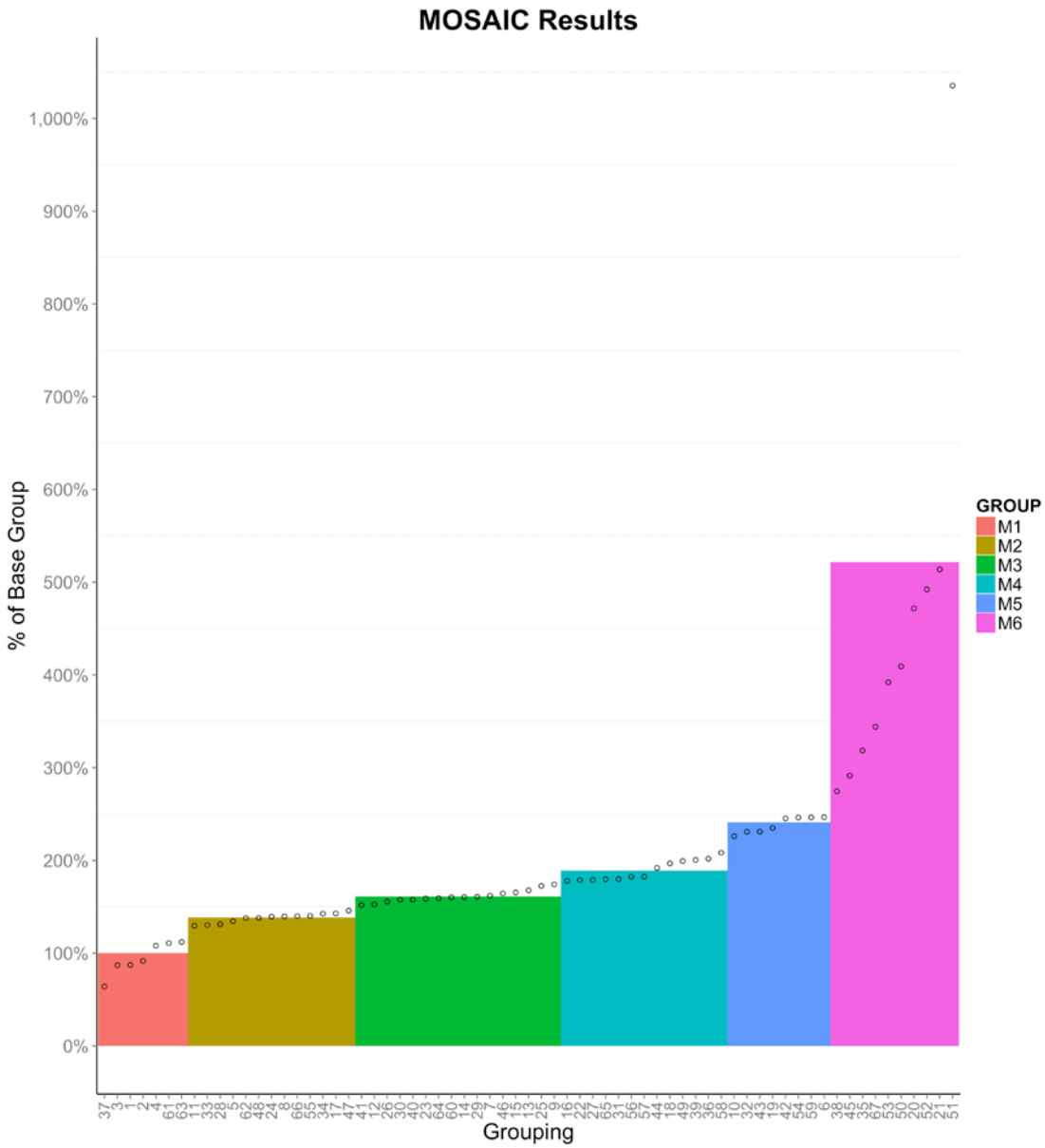
Solver prior to 2010 cannot successfully optimise such a problem. The raw incidence rates and "bottom up" grouping for ACORN and Mosaic are shown in the charts below. Results are shown as a percentage of the Group 1 level of each classification system.

A table detailing the mapping from type to group can be found in Appendix 5.



The percentage of the population falling into each of the "bottom up" groups is shown in the following table:

Group	Population %
A1	7.7%
A2	21.5%
A3	28.4%
A4	20.4%
A5	16.1%
A6	5.8%



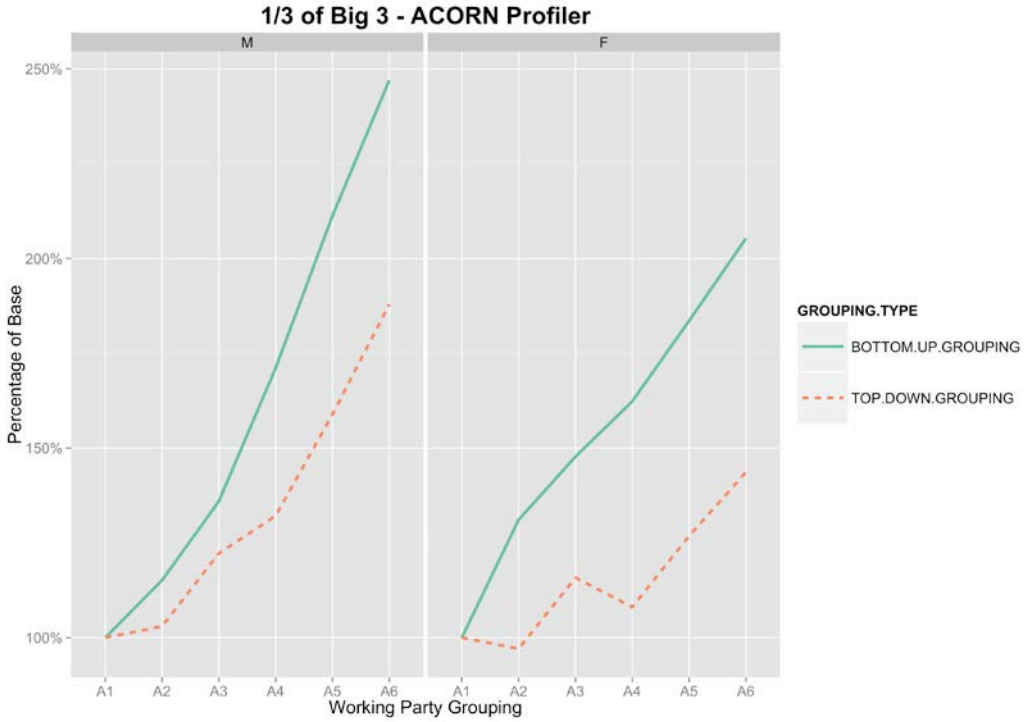
The percentage of the population falling into each of the "bottom up" groups is shown in the following table:

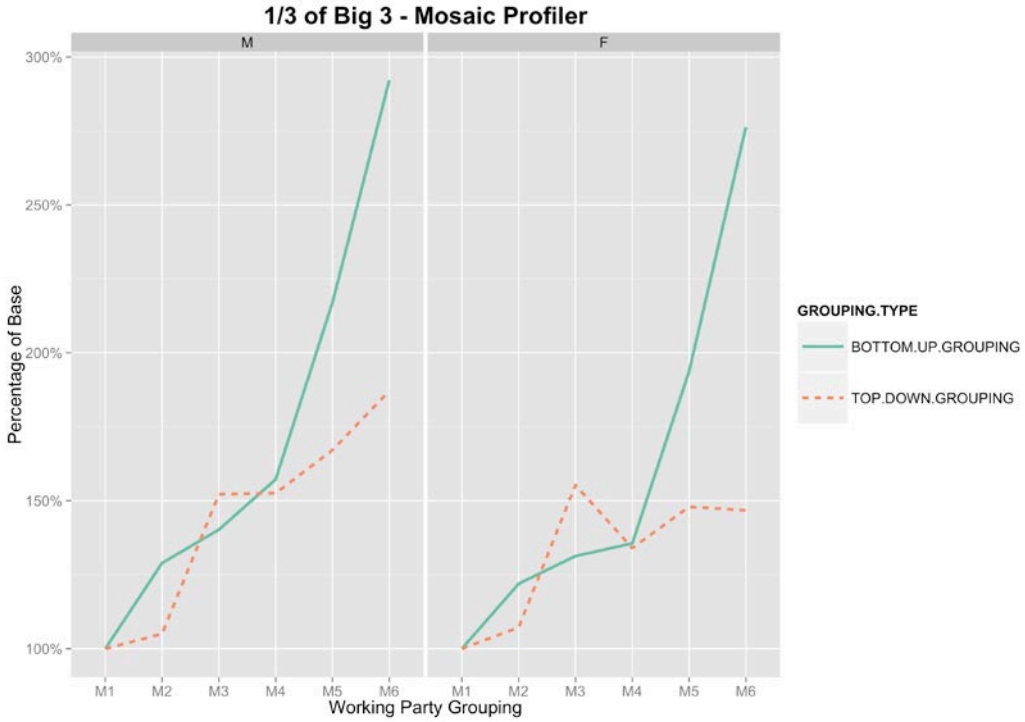
Group	% Population
M1	7.9%
M2	21.2%
M3	24.9%
M4	22.0%
M5	14.3%
M6	9.6%

4.3.2.1. Testing "Bottom Up" Groupings

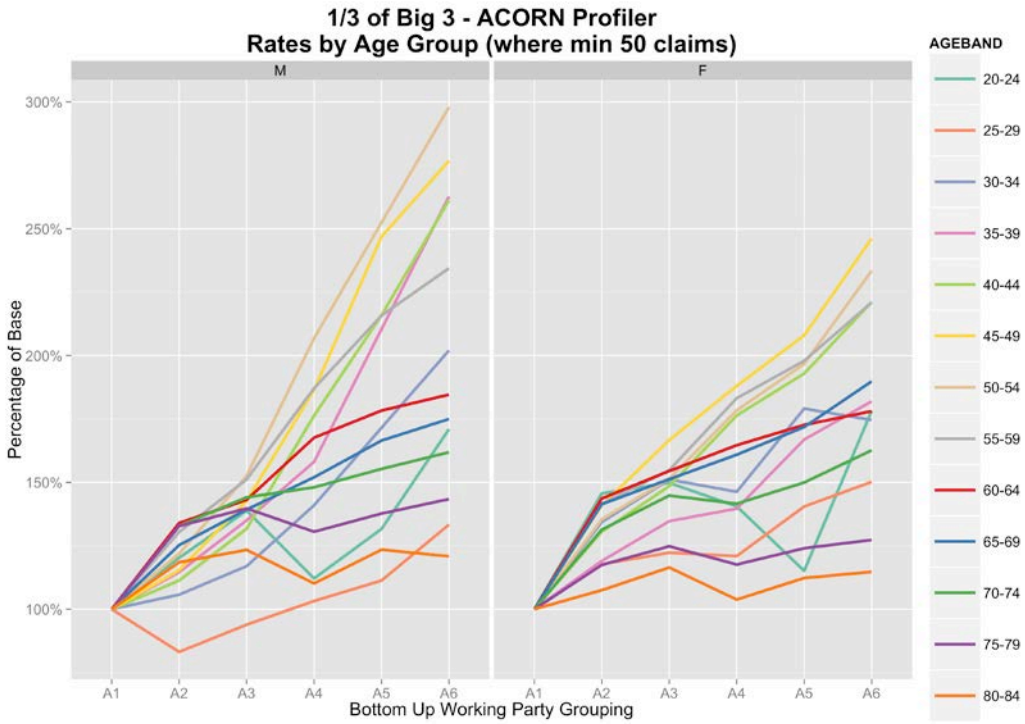
Following derivation of the "bottom up" groupings we have tested them by applying the derived groupings to first ever incidence rates for Cancer, Heart Attack and Stroke combined calculated for the remaining one third of the patients. As envisaged we see a strong gradient

for both ACORN and Mosaic across both genders. For the Top-down approach, for both profilers, the curve is not monotonically increasing though – in both cases (by co-incidence) the rate for group 3 is higher than the rate for group 4. It isn't entirely clear to us why this would be the case but may have something to do with the differing age profiles within the groups.





This gradient is also visible when results are considered by age band. For illustrative purposes we show the results for ACORN. Figures are shown as a percentage of group A1 and a positive gradient is visible for most bands.



We note that once past middle age the curves tend to flatten with increasing age. Again we are not sure of what is driving this and could be due to the differing age profiles within the various types.

4.4. Index of Multiple Deprivation

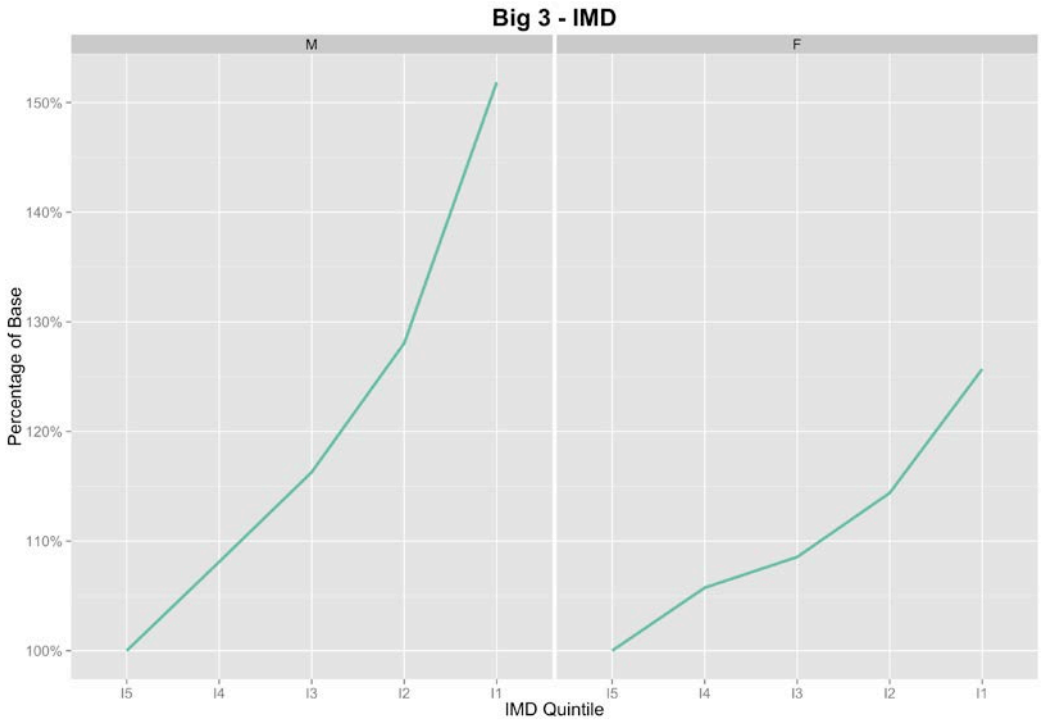
In addition to the geodemographic coding, the HES seriatim data also contains an IMD quintile. As population data split by IMD quintile is readily available, we have included an analysis by IMD quintile.

There have been several versions of Indices of Deprivation issued. Our HES data was coded with the 2004 version. For our work we have used the overall ranking made by combining the seven IMD Domain scores in defined weights.

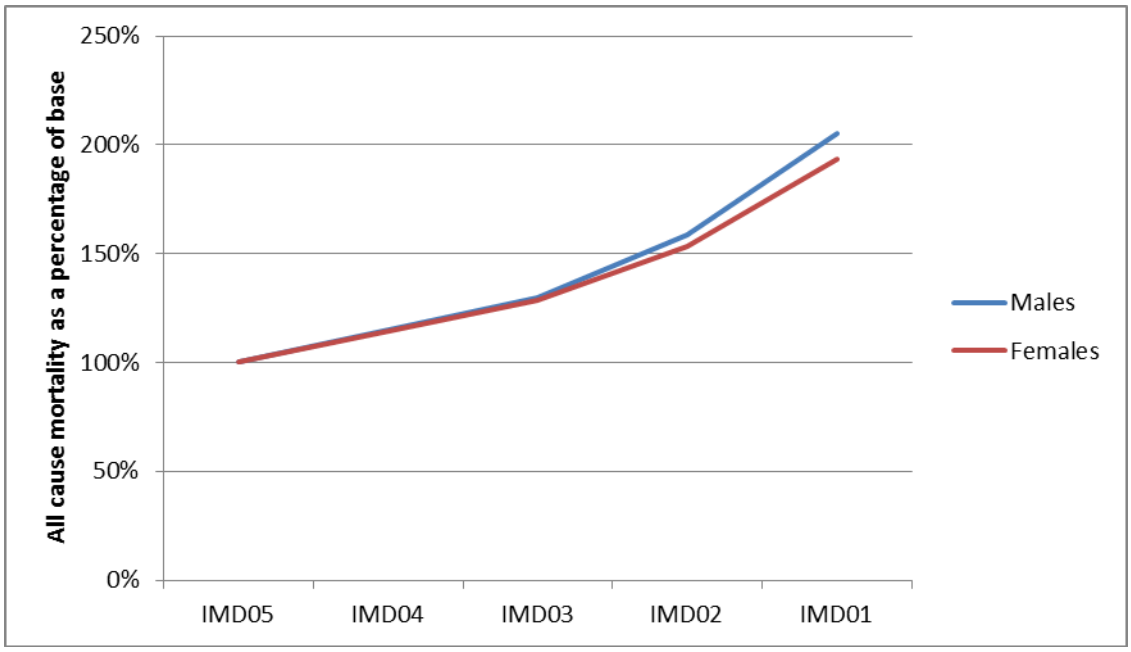
The reader should note that whereas ACORN and Mosaic are postcode level profilers, the IMD results that we have are at Lower Super Output Area (LSOA) level. There are 32,482 of these compared to approximately 1.8m postcodes in use at the moment. As such, ACORN and Mosaic would naturally be expected to be more discriminatory.

In our results I5 is the considered to be the least deprived quintile and I1 the most deprived quintile. For that reason the x-axis on our graphs run from I5 on the left to I1 on right, so from least deprived to most deprived.

For the sake of completeness, here we show the gradient for IMD for the Big 3 dataset in its entirety.



By way of comparison to our rates we have been able to obtain all-cause mortality rates divided by IMD quintile[12] to provide a benchmark for our rates. These are shown in the graph below. The mortality range shown is much greater than the range shown in the "big 3" results. The wider mortality differential is particularly evident for females where cancer dominates the cause of claim to a greater degree and as we shall see the gradient for cancers is particularly small.



[12] Personal communication: Maddy Bajekal, Nov 2013

5. The Combined Table

The proposed new table, CIBT08, includes rates for 36 conditions. This compares to 24 conditions in CIBT02. 22 conditions are common between the two tables, 2 are included in CIBT02 but not in CIBT08 and 15 are new conditions.

This paper proposes rates for three other conditions (Coronary Angioplasty, Ductal Carcinoma in Situ and Prostate Cancer) but these are not included in the table because these are most commonly partial payment conditions on Critical Illness contracts at the time of writing.

As with CIBT02 we calculate the addition for accelerated rates and so tables can be produced for both standalone and accelerated covers.

We set out below the comparison of conditions between the two tables with some commentary:

Conditions Included in Both Tables

Aorta graft surgery	Loss of limb(s)*
Alzheimer's disease	Loss of speech
Benign brain tumour	Heart attack
Blindness	Motor neurone disease
Coronary artery bypass graft	Major organ transplant
Cancer	Multiple sclerosis
Coma	Parkinson's disease
Deafness	Paralysis of two limbs
HIV infection	Stroke
Heart value replacement or repair	Third degree burns
Kidney failure	Traumatic head injury

* Note our Loss of limb rates are set more akin to loss of one limb whereas CIBT02 calibrated to loss of two limbs.

Conditions Not In CIBT08

Coronary angioplasty

Total and Permanent Disability (TPD)

As noted above Coronary angioplasty rates are derived but are simply not included in the table because it is most commonly covered as a partial CI benefit at the time of writing.

For TPD we have elected not to calculate rates because:

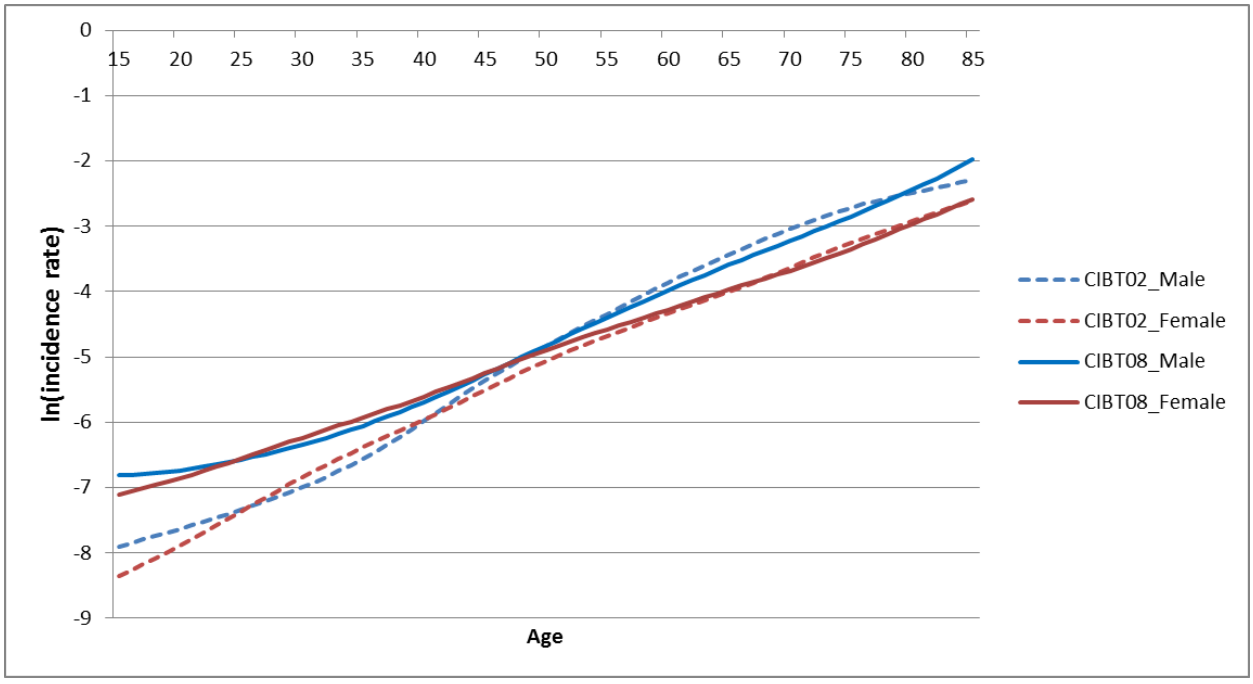
- There is no concept in HES that lends itself to calculating a TPD rate and so we have no new information to add to that utilised in the production of CIBT02.
- Insured experience on TPD appears to continue to be at a level well below that indicated by the work in CIBT02.

New Conditions in CIBT08

Aplastic anaemia	Loss of one limb
Bacterial meningitis	Multiple system atrophy
Cardiomyopathy	Open heart surgery
Creutzfeldt-Jakob disease	Primary pulmonary hypertension
Rheumatoid arthritis	Progressive supranuclear palsy
Dementia	Respiratory failure
Encephalitis	Systemic lupus erythematous
Liver failure	

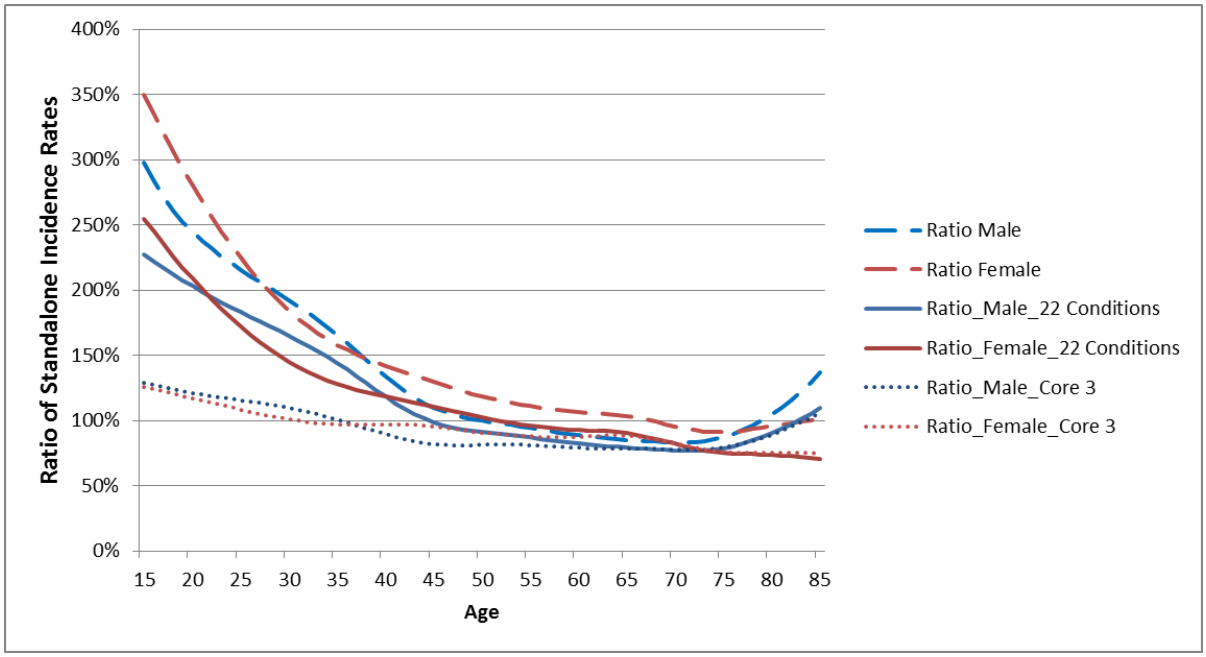
5.1. Comparison to CIBT02

The graphs below compare CIBT08 to CIBT02. For the purposes of comparison we have used the CIBT02 extended rates, on a standalone basis, excluding TPD. This graph shows the comparison of the logarithm of the rates:



The following graph shows the ratio of the rates. Three sets of comparisons are shown:

1. The full CIBT08 table to the Extended CIBT02 table less the rate for TPD, since this is not included in CIBT08;
2. The 22 conditions that are common between the two tables;
3. The big three conditions only – Cancer, Heart Attack and Stroke.



All the comparisons show the CIBT08 rates are higher than the CIBT02 rates at younger ages. The ratio decreases at first until the CIBT02 rates exceed the CIBT08 rates. The crossover occurs at age 45 for males and age 53 for females, on the like for like comparison of 22 illnesses. CIBT08 rates the rise above the CIBT02 rates at the older ages for some of the comparisons, in particular for the males.

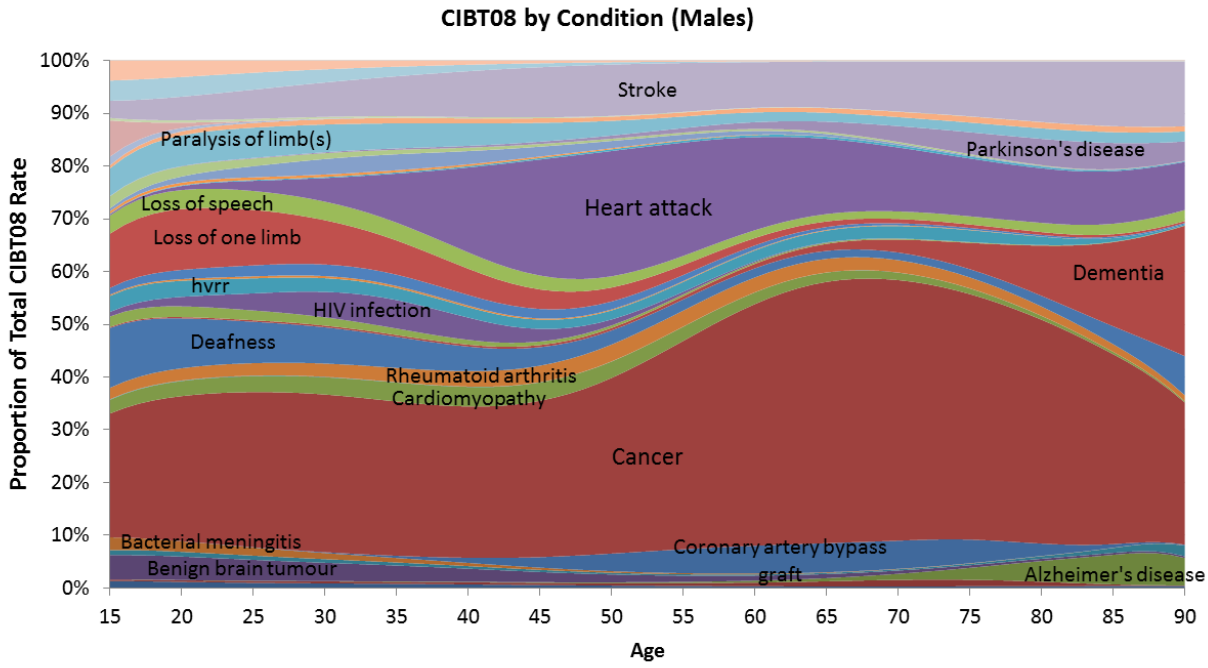
From the comparison of the core 3 conditions we can see the main difference in the rates is driven by the 19 other conditions that are common between the two rate tables. This is heavily influenced by our decision not to apply the adjustments to reflect differences between the medical definitions and the insured definitions. These adjustments are left to the reader to apply.

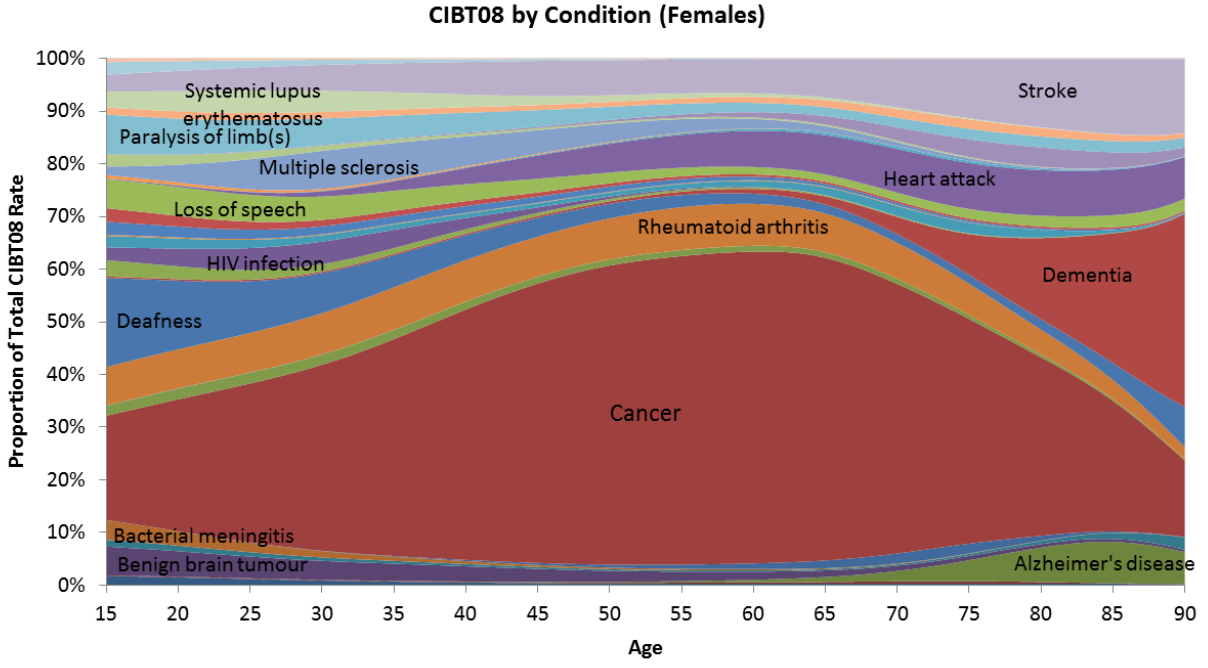
At the older ages the lower rates in CIBT08 will be largely driven by how we have reflected overlaps with all other conditions covered, as per the HES data, in deriving our new rates.

The increase in ratios at the extreme older ages, particularly for males, is thought to be a function of our treatment of prevalence. We have been unable to derive satisfactory prevalence rates at these old ages and we are concerned our approach may be too prudent for the males, where cardio-vascular risks dominate. We also note that our prevalence adjustments do not include allowances for neurodegenerative disease prevalence. As we see in the next section the incidence of these does become significant above age 75.

5.2. Composition of CIBT08 by Illness

The graphs below show how the total CIBT08 is made up of the individual conditions. Not all conditions are labelled to make the graph more readable. Labels are located at or around the point where that illnesses constitutes the most significant proportion of the risk. The colours representing the difference illnesses are the same on both charts.





The graphs above again show the significant influence of some of the minor illnesses at young ages as a result of our decision not to apply adjustments to reflect the insured

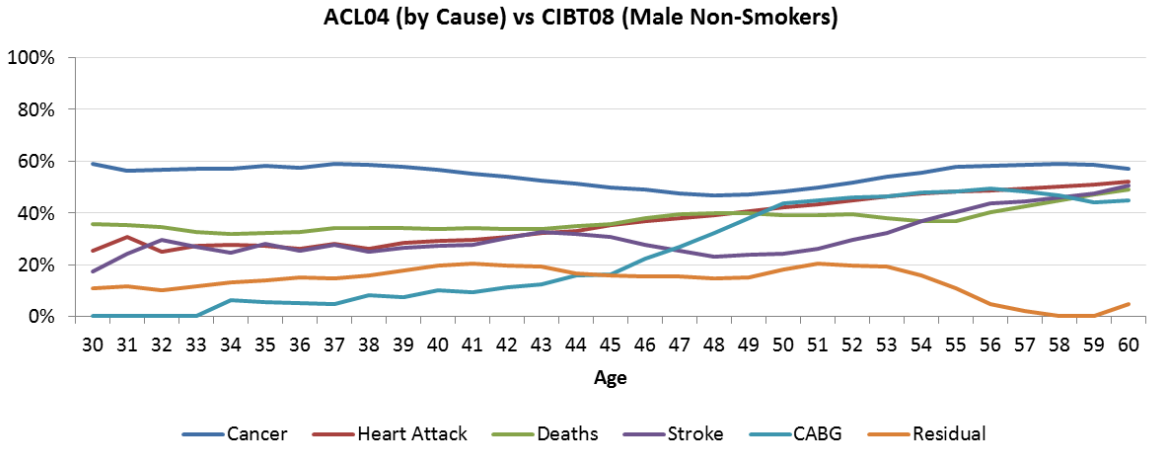
definitions. In particular rates for Benign Brain Tumour (where we have also included an additional significant ICD-10 code over what was included in CIBT02), Bacterial Meningitis, Deafness, HIV Infection, Loss of Speech, Paralysis and Lupus all look very significant at the young ages as a result. For males, Loss of Limb also looks disproportionately high.

For females the high proportion of rate for Rheumatoid Arthritis is also due to us not applying an adjustment for differences between the insured and medical definitions.

As noted in the previous section the neurodegenerative diseases start to become a significant proportion of the rate above age 75.

5.3. Comparison to ACL04 by Cause

The graphs below compare the insured lives rate tables for specific illnesses published by the CMI in Working Paper 52[13].

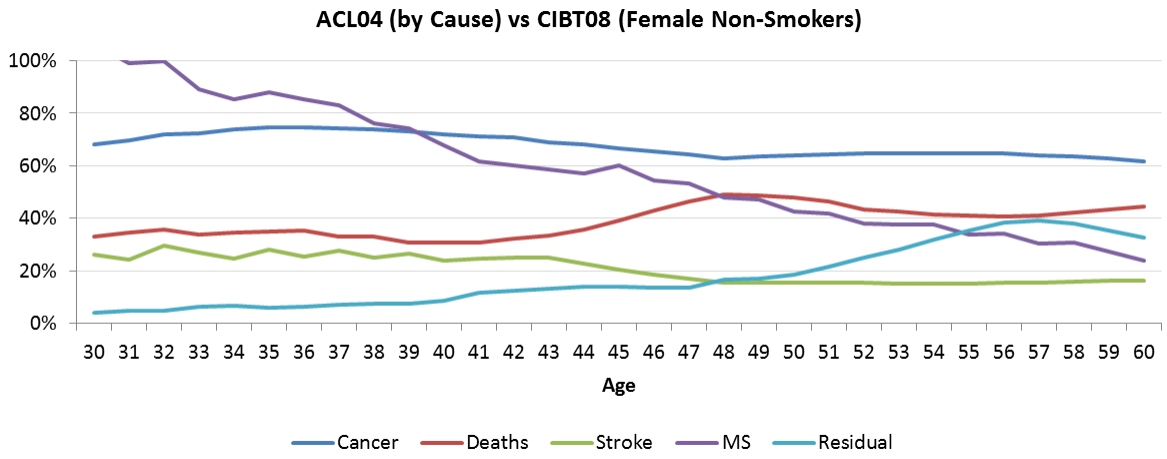


Overall we note the relatively flat profiles of the curves showing that our rates share similar profiles by age to the insured rates. We note that the (orange) residual line is relatively low. This suggests we are materially over-estimating the incidence of the less common conditions. This is to be expected to some degree as, except in a limited number of cases where an opportunity was presented by the HES data, we have not tried to adjust for the differences between the insured definition and the medical definition. To some extent we also note that some of these illnesses are relatively new to the market and so may be underexposed in the historic experience.

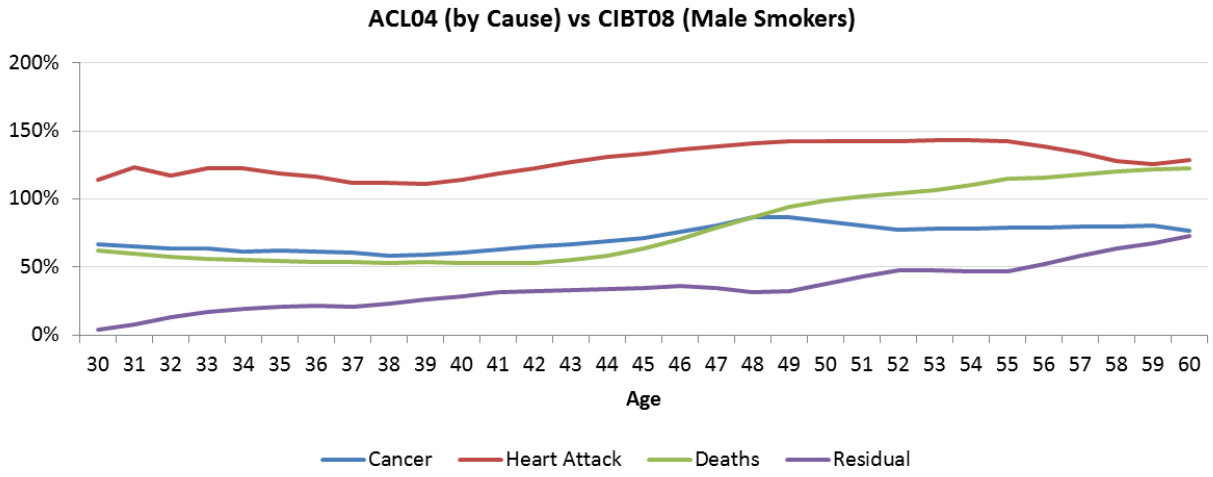
The CABG curve does increase with age. We are not sure as to the reason for that.

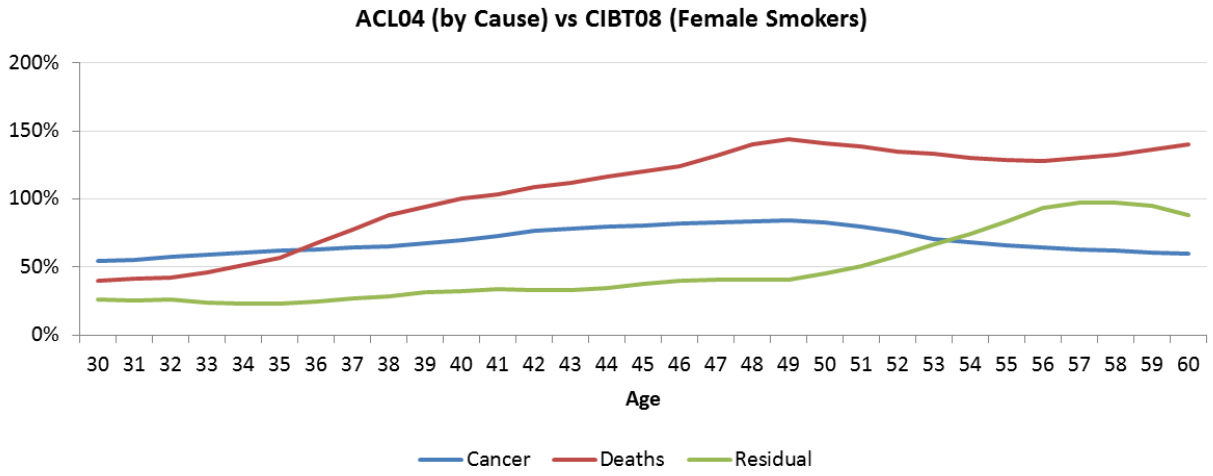
For non-smokers the rates by illnesses are generally always less than 100% as expected. This reflects the differences between insured lives and general population as well as the fact that CIBT08 is a smoker aggregated table. Conversely for smokers some ratios are over 100%.

[13] © Institute and Faculty of Actuaries.



The MS curve shows that our shape of rates differs to the insured experience quite markedly. We believe this is indicative of the HES data not being a suitable source of data for pricing MS given that hospital inpatient stays are not the primary way of delivering treatment to MS sufferers.





6. Illness by Illness Review

6.1. Cancer

6.1.1. What is it?

Cancer is a condition where cells in a specific part of the body grow and reproduce uncontrollably. The cancerous cells can invade and destroy surrounding healthy tissue, including organs.

Cancer sometimes begins in one part of the body before spreading to other areas. This process is known as metastasis.

There are over 200 different types of cancer, each with its own methods of diagnosis and treatment.

Whilst many tumours take the form of a solid growth, others do not such as leukaemia, which is a cancer of blood-forming cells. Lymphomas are cancers of the lymphatic (glandular system).

Cancer is a common condition with over 300,000 new cases of cancer diagnosed each year in the UK. More than one in three people will develop some form of cancer during their lifetime.

In the UK, the most common types of cancer are:

- breast cancer;
- prostate cancer;
- lung cancer;
- bowel cancer;
- bladder cancer;
- uterine (womb) cancer.

6.1.2. Symptoms and Treatment

When cancer begins it invariably produces no symptoms with signs and symptoms only appearing as the mass continues to grow or ulcerates. The findings that result depend on the type and location of the cancer. Few symptoms are specific, with many of them also frequently occurring in individuals who have other conditions. Cancer is the new "great imitator". Thus it is not uncommon for people diagnosed with cancer to have been treated for other diseases to which it was assumed their symptoms were due.

Local symptoms may occur due to the mass of the tumour or its ulceration. For example, mass effects from lung cancer can cause blockage of the bronchus resulting in cough or pneumonia; oesophageal cancer can cause narrowing of the oesophagus, making it difficult or painful to swallow; and colorectal cancer may lead to narrowing or blockages in the bowel, resulting in changes in bowel habits. Masses in breasts or testicles may be easily felt. Ulceration can cause bleeding which, if it occurs in the lung, will lead to coughing up blood, in the bowels to anaemia or rectal bleeding, in the bladder to blood in the urine, and in the uterus to vaginal bleeding. Although localised pain may occur in advanced cancer, the initial swelling is usually painless. Some cancers can cause build-up of fluid within the chest or abdomen.

General symptoms occur due to distant effects of the cancer that are not related to direct or metastatic spread. These may include: unintentional weight loss, fever, being excessively tired, and changes to the skin. Hodgkin disease (a type of lymphoma), leukaemia, and cancers of the liver or kidney can cause a persistent fever of unknown origin.

Each specific type of cancer has its own set of treatment methods. However, many cases of cancer are treated using chemotherapy (powerful cancer-killing medication) and radiotherapy (the controlled use of high energy X-rays). Surgery is also often carried out to remove cancerous tissue.

6.1.3. Risk Factors

The causes of cancer are diverse, complex, and only partially understood. Many things are known to increase the risk of cancer, including tobacco use, dietary factors, certain infections, exposure to radiation, lack of physical activity, obesity, and environmental pollutants. These factors can directly damage genes or combine with existing genetic faults within cells to cause cancerous mutations. Approximately 5–10% of cancers can be traced directly to inherited genetic defects.

6.1.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Cancer (1999)

Any malignant tumour characterised by the uncontrolled growth and spread of malignant cells and invasion of tissue. The term cancer includes leukaemia and Hodgkin's disease but the following are excluded:

- All tumours which are histologically described as pre-malignant, as non-invasive or as cancer in situ.
- All forms of lymphoma in the presence of any Human Immunodeficiency Virus.
- Kaposi's sarcoma in the presence of any Human Immunodeficiency Virus.
- Any skin cancer other than invasive malignant melanoma.

Cancer (2002)

Any malignant tumour characterised by the uncontrolled growth and spread of malignant cells and invasion of tissue. The term cancer includes leukaemia and Hodgkin's disease but the following are excluded:

- All tumours which are histologically described as pre-malignant, as non-invasive or as cancer in situ.
- All tumours of the prostate unless histologically classified as having a Gleason score greater than 6 or having progressed to at least TNM classification T2N0M0.
- All forms of lymphoma in the presence of any Human Immunodeficiency Virus.
- Kaposi's sarcoma in the presence of any Human Immunodeficiency Virus.
- Any skin cancer other than invasive malignant melanoma.

Cancer – excluding less advanced cases (2006)

Any malignant tumour positively diagnosed with histological confirmation and characterised by the uncontrolled growth of malignant cells and invasion of tissue.

The term malignant tumour includes leukaemia, lymphoma and sarcoma.

For the above definition, the following are not covered:

- All cancers which are histologically classified as any of the following:
    - pre-malignant, for example essential thrombocythaemia and polycythaemia rubra vera;
    - non-invasive;
    - cancer in situ;
    - having either borderline malignancy; or
    - having low malignant potential.
- All tumours of the prostate unless histologically classified as having a Gleason score greater than 6 or having progressed to at least clinical TNM classification T2N0M0.
- Chronic lymphocytic leukaemia unless histologically classified as having progressed to at least Binet Stage A.
- Any skin cancer other than malignant melanoma that has been histologically classified as having caused invasion beyond the epidermis (outer layer of skin).

Cancer – excluding less advanced cases (2011)

Any malignant tumour positively diagnosed with histological confirmation and characterised by the uncontrolled growth of malignant cells and invasion of tissue.

The term malignant tumour includes leukaemia, sarcoma and lymphoma **except cutaneous lymphoma (lymphoma confined to the skin).**

For the above definition, the following are not covered:

- All cancers which are histologically classified as any of the following:
    - pre-malignant;
    - non-invasive;
    - cancer in situ;
    - having borderline malignancy; or
    - having low malignant potential;
- All tumours of the prostate unless histologically classified as having a Gleason score greater than 6 or having progressed to at least clinical TNM classification T2N0M0.
- Chronic lymphocytic leukaemia unless histologically classified as having progressed to at least Binet Stage A.
- Any skin cancer (including cutaneous lymphoma) other than malignant melanoma that has been histologically classified as having caused invasion beyond the epidermis (outer layer of skin).

Some companies have enhanced benefit under this definition in order to claim ABI+ status by explicitly covering skin cancers that have metastasised to lymph glands or distant organs.

The key specific change for cancer has been the inclusion of an overlap factor for cancer. For CIBT02 the authors noted limited co-morbidities with other CI conditions and so assumed no overlap. The lack of co-morbidity with cancer is driven by the high mortality rate of some cancers while other CIs share similar pre-disposing risk factors.

Our data enabled us to identify cancer sufferers who had also been diagnosed with other CI conditions purely due to people suffering multiple unconnected conditions and so we could explicitly allow for these. Given morbidity increases with age this impact becomes more significant with increasing age.

6.1.5. Derived Incidence Rates

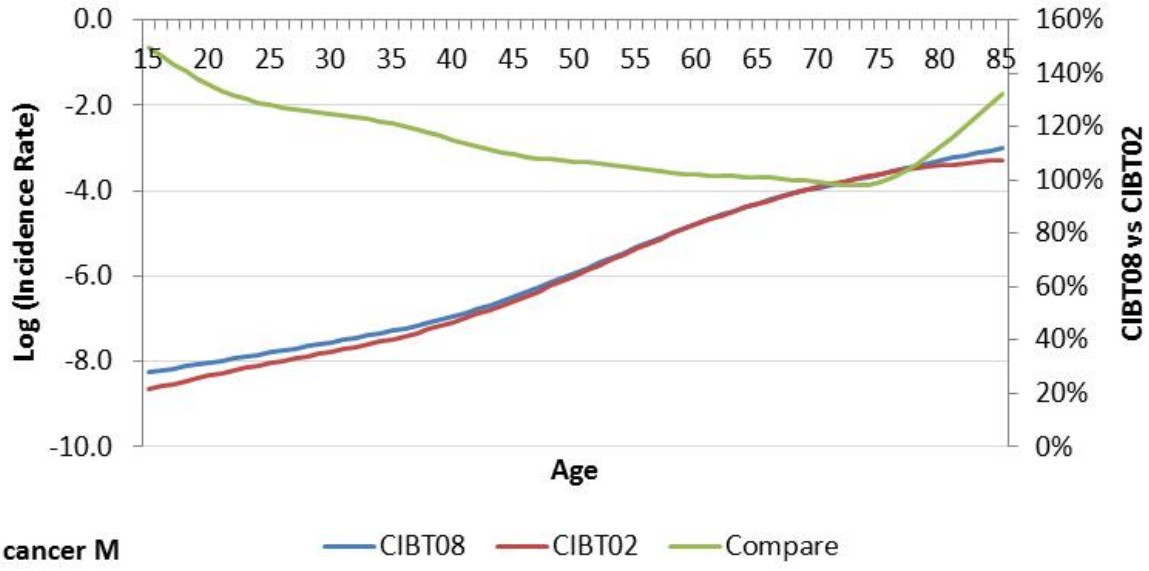
6.1.5.1. Male Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

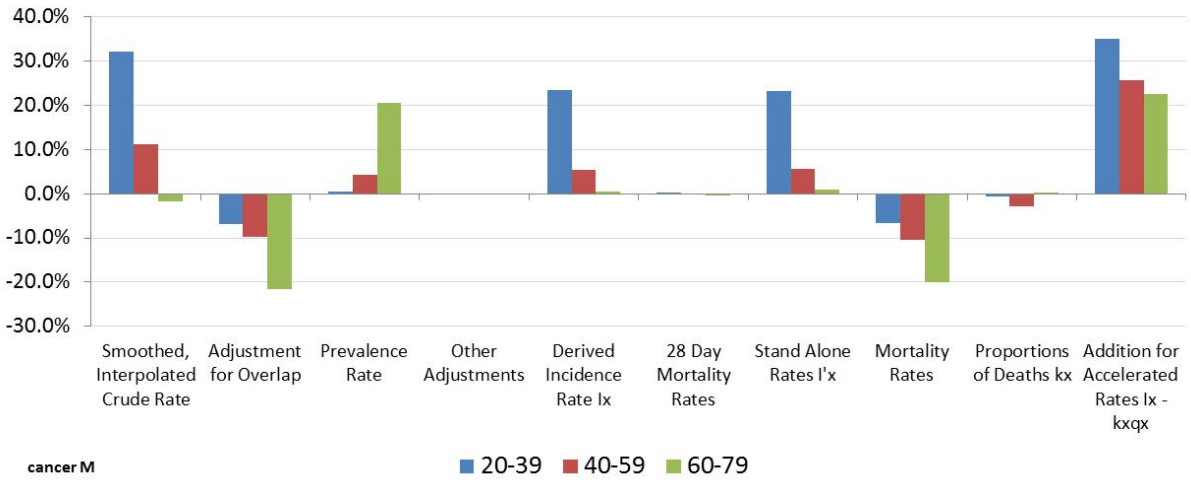
Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	5.92	31.25	166.96
Adjustment for Overlap	-6.9%	-9.8%	-21.5%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	5.55	30.11	180.57
28 Day Mortality Rates	-0.6%	-0.9%	-1.1%
Stand Alone Rates I'x	5.51	29.83	178.42
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	10.1%	26.0%	37.2%
Addition for Accelerated Rates Ix - kxqx	4.57	19.30	103.74

cancer _ M

The standalone incidence rates for Cancer are shown in the chart below for both the newly derived rates (CIBT08) and those from CIBT02. The green line, with values on the 2nd axis, displays CIBT08 as a percentage of CIBT02.



The following chart illustrates the movement of each of these components relative to CIBT02, for the same 3 age bands:



We note that the CIBT08 standalone rates are higher than CIBT02 in the lowest age band but similar in the higher age bands. The higher rates at younger ages are largely driven by the crude rates being higher in the new HES data which we believe is explained by recent cancer trends. For the older ages the reduction is due to the allowance for overlap with other conditions, which we have been able to derive directly from our HES data.

Note that we have included some additional ICD codes for cancer relative to CIBT02 but overall we believe the additional codes add only around 3% to the rates. We have added the following codes:

- D45
- D46
- D47

These additions have been made due to changes in the ICD classification over the intervening period.

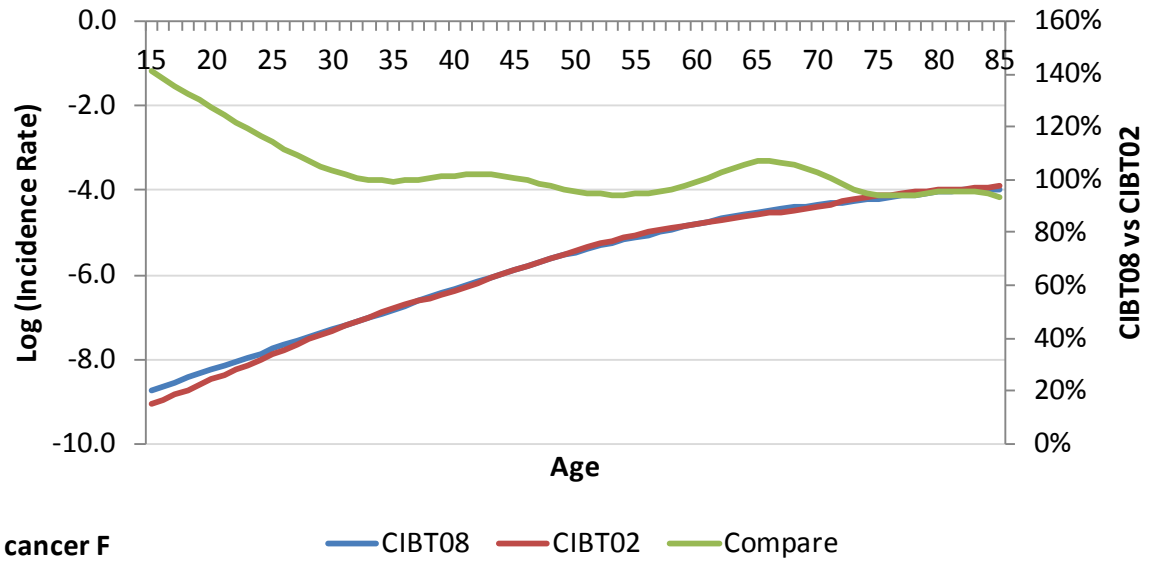
We have compared our crude cancer rates to ONS Cancer Registry statistics (which we consider to be the gold standard for cancer incidence in England) over the corresponding period and we found that the aggregate cancer rates were in reasonably good agreement – in aggregate our rates were within 1% of ONS rates. By individual site the correspondence is more variable which is to be expected given not all cancers are treated during in-patient hospital stays.

6.1.5.2. Female Results

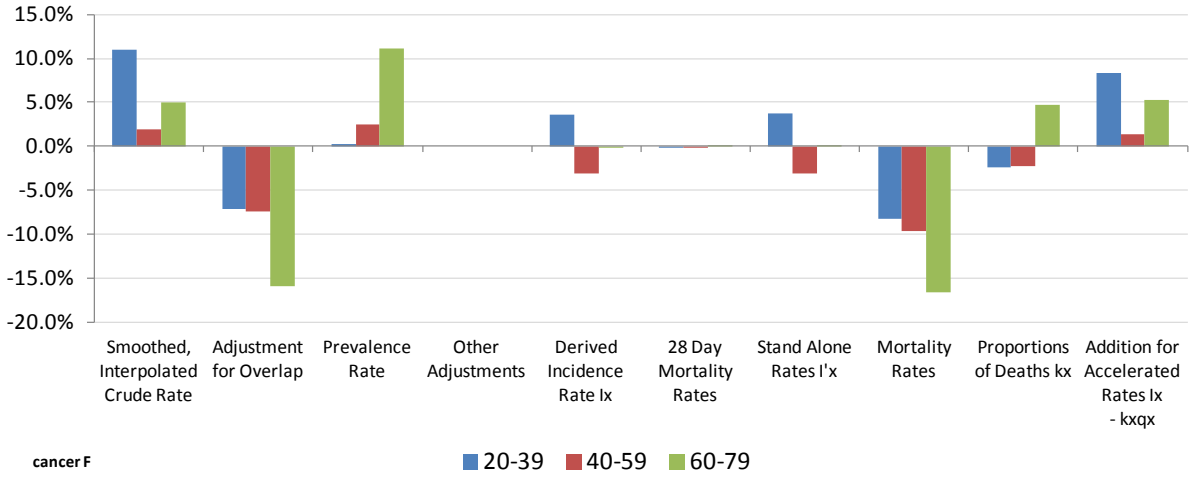
The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	8.21	42.33	120.30
Adjustment for Overlap	-7.2%	-7.4%	-16.0%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	7.71	41.52	123.08
28 Day Mortality Rates	-0.3%	-0.5%	-1.1%
Stand Alone Rates I'x	7.69	41.31	121.71
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	24.1%	46.8%	45.7%
Addition for Accelerated Rates Ix - kxqx	6.55	29.90	61.92

As with males the blue line reflects the standalone rates (CIBT08) and these are compared against those from CIBT02. The green line displays CIBT08 as a percentage of CIBT02.



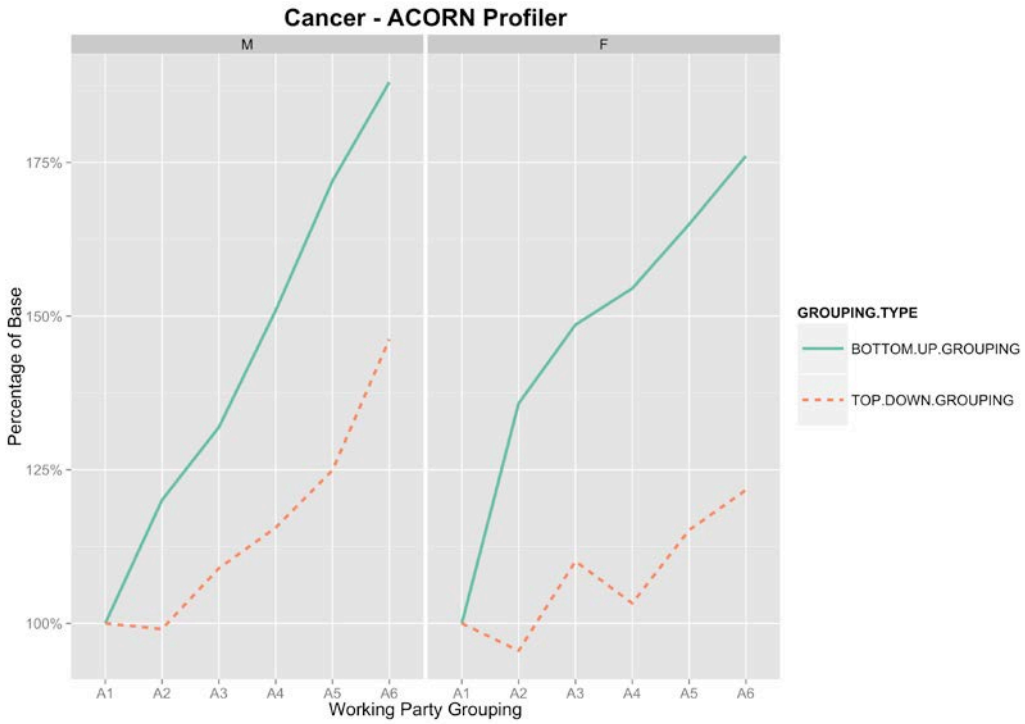
The following chart illustrates the movement of each of these components relative to CIBT02, for the same 3 age bands:



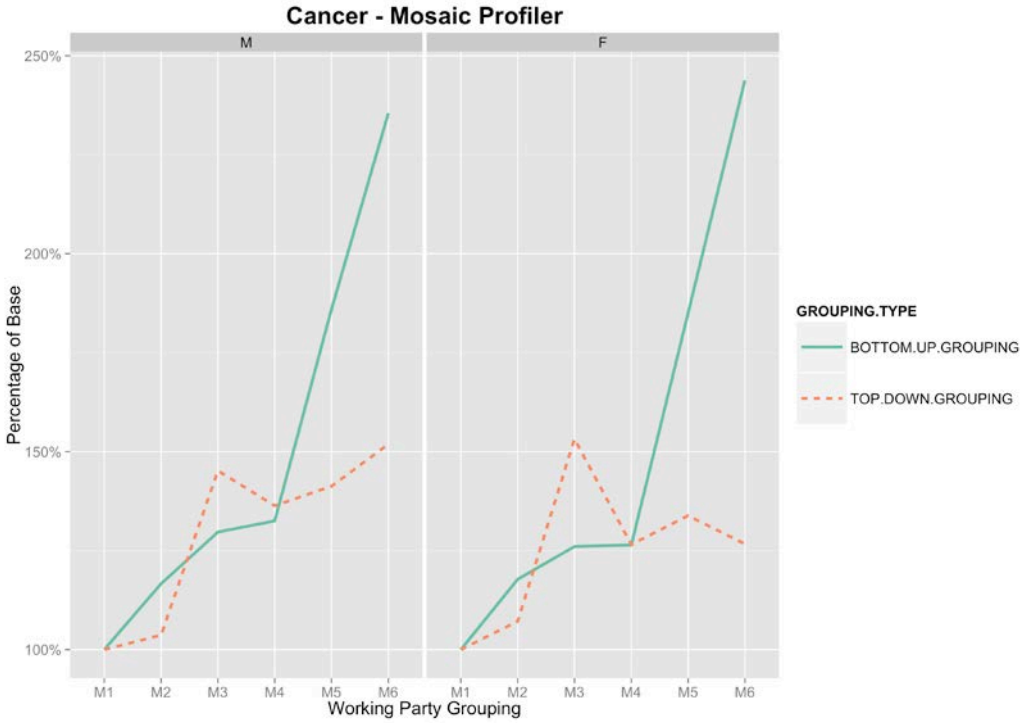
Compared to the males the overall magnitude of change in crude incidence is much smaller. The overlap adjustment is still significant although this is offset by a large prevalence adjustment.

6.1.6. Geodemographic Analysis

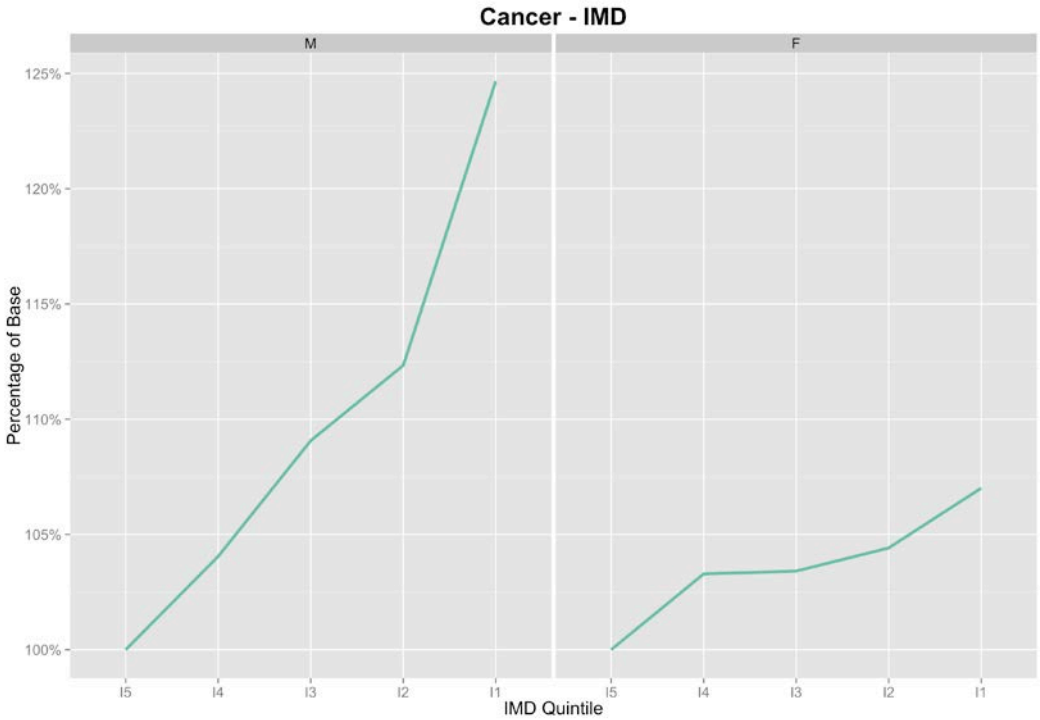
6.1.6.1. ACORN



6.1.6.2. Mosaic



6.1.6.3. Index of Multiple Deprivation



The ratios shown above tend to agree with the results presented in National Cancer Intelligence Network's (NCIN's) 2008 publication 'Cancer Incidence by Deprivation, England,

1995-2004'[14]. This reported the following relative age standardised incidence rate ratios from least deprived to most deprived for all cancers combined, excluding non-melanoma skin cancer:

Period	Males	Females
1995-1999	1.3	1.1
2000-2004	1.2	1.1

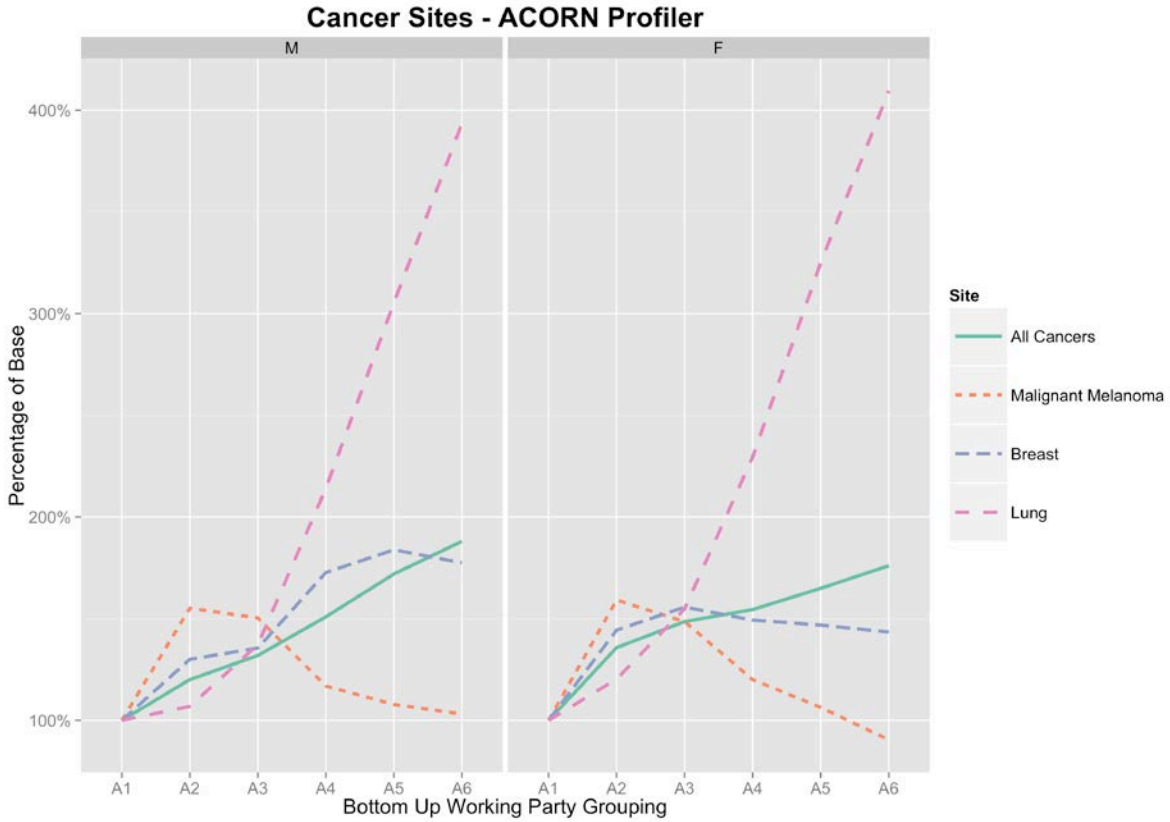
6.1.7. Geodemographic Analysis - Individual Cancer Sites

In this section we show a comparison of the gradients seen for individual cancer sites against the overall cancer gradient. Specifically we consider the gradients for the following specific sites:

- Breast;
- Lung;
- Malignant Melanoma.

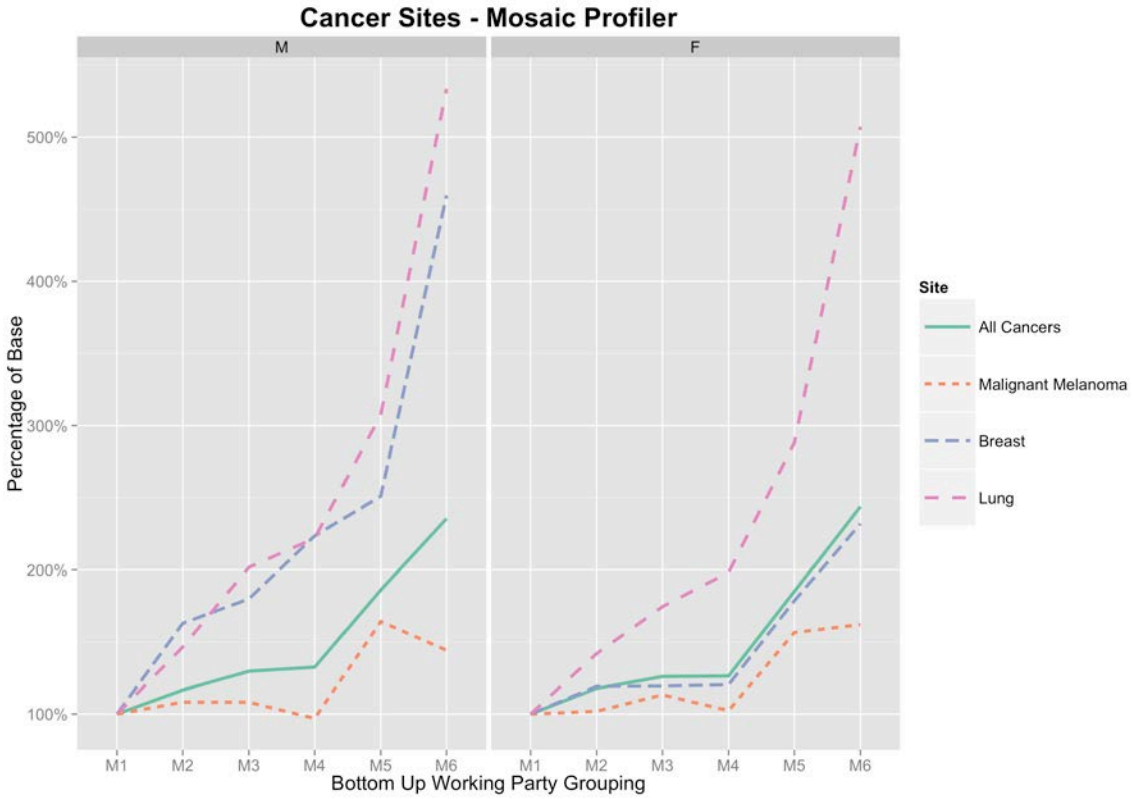
These are shown for ACORN, Mosaic and IMD. Note, that for the sake of brevity, we have only shown the bottom up results.

6.1.7.1. ACORN

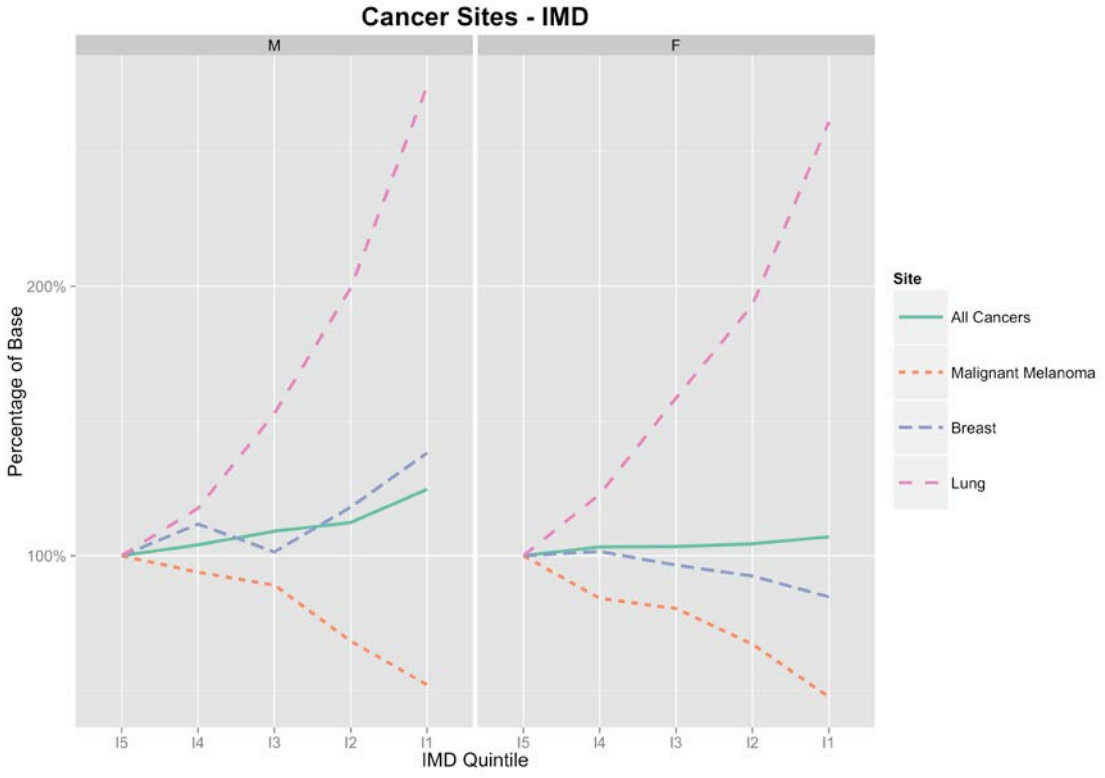


[14] http://www.ncin.org.uk/view?rid=73

6.1.7.2. Mosaic



6.1.7.3. Index of Multiple Deprivation



Again these results correspond well to those released by the NCIN. The NCIN figures suggest that the most deprived groups should have an age standardised incidence rate relative to the least deprived of approximately:

- 50% for Malignant melanoma;
- 80% for Breast cancer (for females)
- 240% for males suffering lung cancer and between 250% and 270% for females.

6.2. Heart Attack

6.2.1. What is it?

Myocardial Infarction (MI) is the medical term for Heart Attack.

Most heart attacks involve a sudden blockage of the coronary artery in connection with coronary heart disease. This is when coronary arteries, which supply blood to the heart, narrow due to a gradual build-up of a fatty substance called atheroma within their walls. If the atheroma becomes unstable, a piece may break off, exposing the soft inner portions of the build-up to the blood. This triggers the blood's clotting mechanism. This clot can block the coronary artery, starving the heart of blood and oxygen and causing death of heart muscle.[15]

While rare, MI may arise for a variety of other reasons where the heart's demand for oxygen increases or where the oxygen supply is restricted.[16] These include arterial spasm for which smoking is a risk factor, use of cocaine which causes arteries to constrict, cardiac embolism whereby a blood clot develops for other reasons, anaemia and other heart abnormalities.

6.2.2. Symptoms and Treatment

Symptoms include sudden chest pain, shortness of breath, sweating, nausea, vomiting, abnormal heartbeats and anxiety. Loss of consciousness and sudden death can also occur with MI, although survival rates following MI are improving.

Women experience fewer of these symptoms than men, but usually have shortness of breath, weakness, a feeling of indigestion, and fatigue.

In some cases MI may occur in the absence of symptoms and the heart damage is discovered on subsequent investigation or at autopsy. This is known as "silent Heart Attack". It is most common among the elderly and diabetics. A number of studies have sought to estimate the proportion of MI that is silent and have reported results ranging from negligible to well over half of cases, depending on the study subjects and methods.

The diagnosis of MI can be made after assessing the patient's complaints and physical status, ECG changes, coronary angiogram and levels of cardiac markers help to confirm the diagnosis. Immediate treatments for a suspected acute myocardial infarction include oxygen to help with breathing, aspirin, which prevents the blood from clotting and forming further blockages, and nitro-glycerine to treat chest pain. Other treatments will commonly include the following:

- Thrombolysis - a combination of medication to dissolve the blood clot and restore the flow of blood to the heart (this is known as thrombolysis);
- Coronary Angioplasty - surgery to widen the coronary artery. A stent is usually inserted into the artery to help keep it open following surgery. This is now more often referred to as Percutaneous Coronary Intervention (PCI);
- Coronary Artery Bypass Graft (CABG) – performed when coronary angioplasty is not technically possible. A CABG involves taking a blood vessel from another part of the body, usually the chest or leg, to use as a graft and to replace any hardened or narrowed coronary arteries.

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[15] Sources include http://www.nhs.uk/Conditions/Heart-attack/Pages/Introduction.aspx; http://www.bhf.org.uk/heart-health/conditions/heart-attack.aspx; and http://www.patient.co.uk/health/myocardial-infarction-heart-attack#

[16] British Heart Foundation "Factfile: Non-atherosclerotic causes of myocardial infarction (MI)" May 2011

In recent years angioplasty has become the preferred primary intervention for cases that present early enough. The UK now has a network of catheterisation laboratories which are examination rooms in a hospital or clinic with diagnostic imaging equipment used to visualise the arteries of the heart and the chambers of the heart. They also have all the equipment needed to perform coronary angioplasty.

After MI there are various treatments that are prescribed to reduce the risk of recurrence and of further development of the heart disease such as statins to control cholesterol, anti-hypertensives and platelet thinning drugs to reduce the risk of thrombus.

6.2.3. Risk Factors

Risk factors that increase the risk of ischemic heart disease developing and subsequent MI include age, sex, high blood pressure, diabetes, high cholesterol, tobacco smoking (including passive smoking), family history of ischaemic heart disease or MI, obesity, lack of physical activity and alcohol consumption. Smoking, obesity and lack of exercise are the most significant risk factors for MI.

6.2.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Heart Attack (1999)

The death of a portion of heart muscle as a result of inadequate blood supply as evidenced by an episode of typical chest pain, new electrocardiographic changes and by the elevation of cardiac enzymes. The evidence must be consistent with the diagnosis of a heart attack.

Heart Attack (2002)

The death of a portion of heart muscle as a result of inadequate blood supply, that has resulted in all of the following evidence of acute myocardial infarction;

- typical chest pain.
- new characteristic electrocardiograph changes
- the characteristic rise of cardiac enzymes, troponins or biochemical markers, where all of the above shows a definite acute myocardial infarction.

Other acute coronary syndromes, including but not limited to angina, are not covered under this definition.

Heart Attack – of specified severity (2006/2011)

Death of heart muscle, due to inadequate blood supply, that has resulted in all of the following evidence of acute myocardial infarction:

- Typical clinical symptoms (for example characteristic chest pain).
- New characteristic electrocardiographic changes.
- The characteristic rise of cardiac enzymes or Troponins recorded at the following levels or higher;
    - Troponin T>1.0 ng/ml
    - AccuTnl>0.5 ng/ml or equivalent threshold with other Troponin I methods.

The evidence must show a definite acute myocardial infarction.

For the above definition, the following are not covered:

- Other acute coronary syndromes including but not limited to angina.

Many companies in the market claim an "ABI+" definition by removing the requirement for 'typical clinical symptoms (for example characteristic chest pain)' to be present. By doing so they claim that the cover is extended because this change results in "silent" MI being covered.

Recently these have been developments in the market where benefits are payable using similar criteria with the removal of stated levels of troponin, effectively extending cover for all cases of acute MI. This has been done in some cases offering a full benefit whilst another option is to pay full benefit with stated troponin levels and MI with lower troponin levels as a partial/additional benefit.

6.2.5. Comparison with Clinical Definitions

Until 2000 events recorded as myocardial infarction in clinical practice and which would qualify for claim in terms of insurance definitions were broadly similar, where diagnosis was based on presence of typical symptoms, ECG changes and elevation of cardiac enzymes. Clinical practice was based on the World Health Organization definition of 1979 and would treat presence of two out of the three requirements as probable heart attack while the insurance requirement was for all three to be met so that payment was triggered for the WHO classification for definite MI.

The WHO definition began to be replaced in medical practice from 2000 onwards following the publication of the paper "Myocardial infarction redefined — a consensus document of The Joint European Society of Cardiology (ESC)/American College of Cardiology Committee (ACC) for the Redefinition of Myocardial Infarction".

One of the major differences in the new definition was placing biochemical markers, with the specific mention of troponin, at the cornerstone of how a diagnosis of MI is made. Cardiac troponins are chemicals found exclusively in heart muscle and are released into the blood when heart muscle is damaged. Troponin concentration in the blood is negligible in the absence of heart muscle damage. Blood tests are available to detect concentrations of either Troponin I or Troponin T. Troponins are much more sensitive in detecting damage to heart tissue than the cardiac enzymes used historically and have been used widely in the diagnosis of MI for over 10 years.

Following the release of the paper in 2000, there was some resistance to adopt its recommendations where troponins were found to be minimally elevated. The concern was that it was not appropriate to label as MI those cases involving minimal heart damage and which would not have been detected in the absence of a troponin assay.

This led to a paper published in 2004 by a British Cardiac Society working group that suggested that certain thresholds of concentration should be exceeded before recognising clinical MI. Thresholds for assays in use at the time were Troponin T > 1.0 ng/ml and AccuTnI > 0.5 ng/ml. The paper's authors postulated that these levels equated to the severity of events previously detectable by the less sensitive cardiac enzymes. These thresholds were used in the 2006 ABI Statement of Best Practice definition of heart attack as it was felt appropriate to do so until there had been universal agreement upon the re-definition of myocardial infarction.

The ECS and ACC published the Second Universal Definition of MI in 2007 which was then universally accepted and began to be more readily used in medical practice. There have been two further updates to this paper, with the last being in 2012 and known as the "Third Universal Definition of Myocardial Infarction". The first part of the definition of MI from this paper is reproduced below.

The term acute myocardial infarction (MI) should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischaemia. Under these conditions any one of the following criteria meets the diagnosis for MI:

Detection of a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:

- *Symptoms of ischaemia;*
- *New or presumed new significant ST-segment–T wave (ST–T) changes or new left bundle branch block (LBBB);*
- *Development of pathological Q waves in the ECG;*
- *Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality;*
- *Identification of an intracoronary thrombus by angiography or autopsy.*

The URL is the "upper reference limit" and is the 99th percentile of concentrations observed in a normal reference population studied by the test's manufacturers i.e. people without MI. If the test is unreliable at these levels, as denoted by a coefficient of variation in excess of 10%, a cut-off higher than the 99th percentile should be used. Clinicians using older troponin assays made MI diagnoses at cut-offs in excess of the 99th percentile because of reliability issues but the newer highly sensitive troponin assays are more reliable at these levels.

The concentrations at which MI is diagnosed in clinical practice are as small as 1% of the ABI definition concentration cut-offs. The incidence rates we have derived from HES data are therefore higher than we would expect from insured lives experience. As the troponin tests become more sensitive and reliable, more cases will be labelled as clinical MI but will not meet the severity criteria set out in the ABI definition. Furthermore the ABI definition does not make specific mention of how imaging evidence and angiography should be taken into account.

6.2.6. Derived Incidence Rates

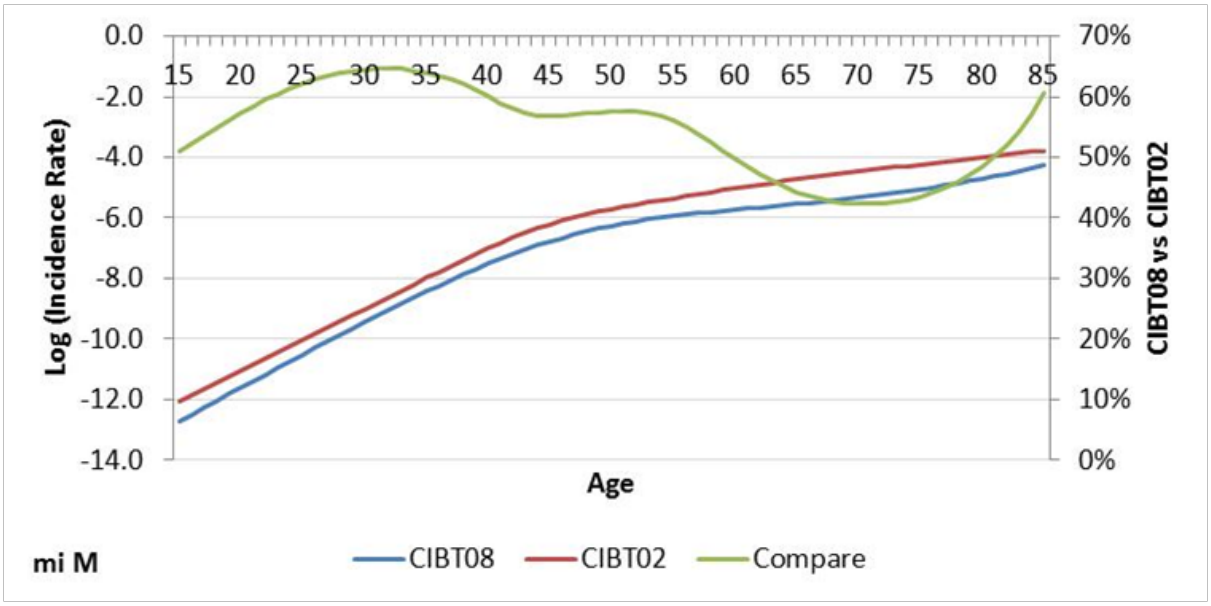
6.2.6.1. Male Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.66	21.76	75.07
Adjustment for Overlap	-6.2%	-8.9%	-28.6%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	1.58	20.99	72.14
28 Day Mortality Rates	-19.8%	-16.3%	-31.8%
Stand Alone Rates I'x	1.33	17.35	47.34
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	1.6%	7.1%	7.5%
Addition for Accelerated Rates Ix - kxqx	1.40	18.23	56.02

mi _ M

The smoothed crude rates are shown in the chart below for both the newly derived rates (CIBT08) and those from CIBT02. The green line, with values on the 2nd axis, displays CIBT08 as a percentage of CIBT02.



The adjustment for first ever heart attack is included in the crude incidence shown above. By comparing the first ever to all diagnosis counts from our HES data set we conclude that this adjustment reduces the incidence rate by between 40% and 60%, with the reduction increasing with age. The "first ever" reduction was between 7% and 17% in the construction of CIBT02 and was based on a comparison of ICD-10 codes I21 and I22.

The smoothed crude rate also includes a gross-up factor for sudden deaths which was obtained with reference to a paper that linked records between HES data and ONS death data for England in calendar year 2010.[17] This study by Smolina et al exposes cases where death occurs in the absence of hospitalisation. There were over 82,000 MIs in this data set and the author kindly provided us with the raw data to deduce the unsmoothed gross-up factors to arrive at all MIs from hospitalised cases. The paper noted no difference in fatality rates between first ever and subsequent MIs so we made no further adjustments for differences in deaths between first ever and recurrent MI. A further difference worth noting is that this paper excluded MI cases that were discharged alive within 1 day because of concerns that these are cases of suspected MI. Our derived incidence rates may include such cases.

The table below shows the resulting gross up factors derived from the 2010 data with the factors used in the development of CIBT02 are shown to the right. The banded values are not age-standardised.

Sudden death gross-up factors (males)

Age band	2010 data used for CIBT08	CIBT02
20-39	114%	106%
40-59	115%	107%
60-79	128%	109%

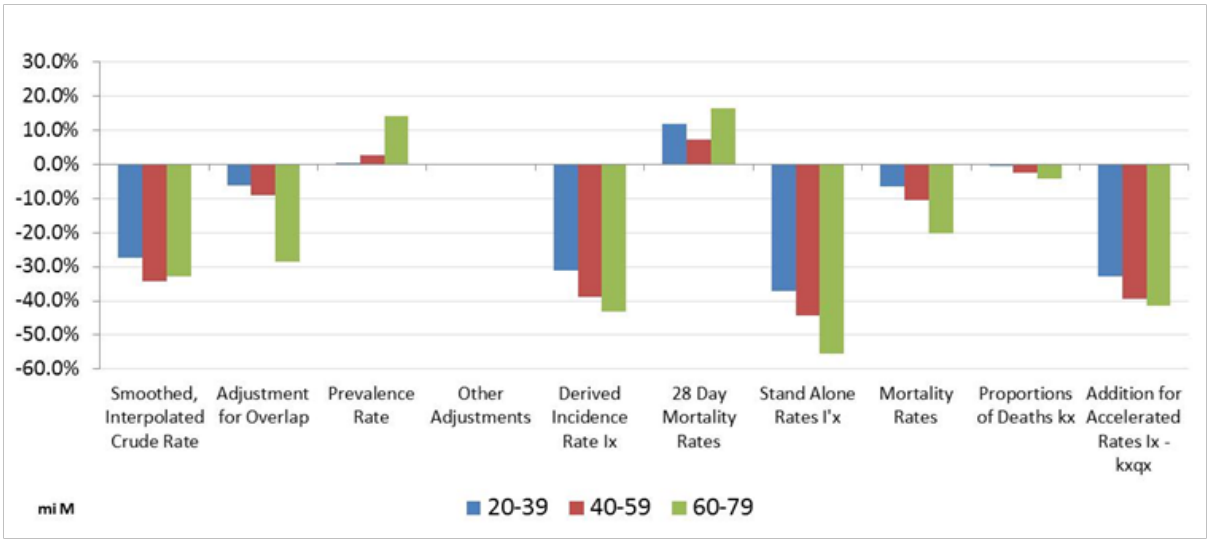
The gross-up using the actual 2010 data is higher than in CIBT02. This partially offsets some of the reduction in the raw incidence rates.

We performed some higher level comparisons between incidence rates implied for the period 2001 to 2003 and 2007 to 2009 within our data set and found that the levels were relatively flat for males below age 50 but that incidence appears to have reduced by up to 20% at older ages.

Therefore we conclude that a key driver of the change against CIBT02 is the new method of calculation of first-ever incidence.

The following chart illustrates the movement of each of these components relative to CIBT02, for the same 3 age bands:

[17] Smolina K et al, "Incidence and 30-day case fatality for acute myocardial infarction in England in 2010: national-linked database study"; European Journal of Public Health, 1–6; doi:10.1093/eurpub/ckr196



The CIBT02 tables included no adjustment for overlap as heart attack was one of the first conditions analysed and overlap was removed in a step-wise fashion. The CIBT08 overlap adjustment uses the HES data to strip out the background level of all other CIs among heart attack patients. This adjustment is substantial and, as expected, the effect increases with age. This adjustment further reduced the incidence rates in CIBT08 vs. CIBT02.

Correspondingly we have included all CIs in our prevalence adjustment which partially offsets the reduction in rates coming from a higher overlap adjustment.

To derive 28-day mortality rates we again referenced the data from Smolina et al which recorded deaths among hospital admissions within 30 days. We made no further adjustment to reduce the death rates from 30 to 28 days as we believe that the deaths are skewed towards the earlier part of this period. The resulting reductions are shown in the age banded summary at the start of this section. Our updated evidence suggests a higher 30-day mortality rate than was used for CIBT02 which in turn reduces standalone MI incidence.

We observe that population death rates have reduced since 2002, as has the proportion of deaths attributed to MI.

The addition for accelerated Rates, $I_x - k_x.q_{x,}$ has reduced as a result of the substantial reduction in crude incidence with some dampening coming from reduced overlap with death.

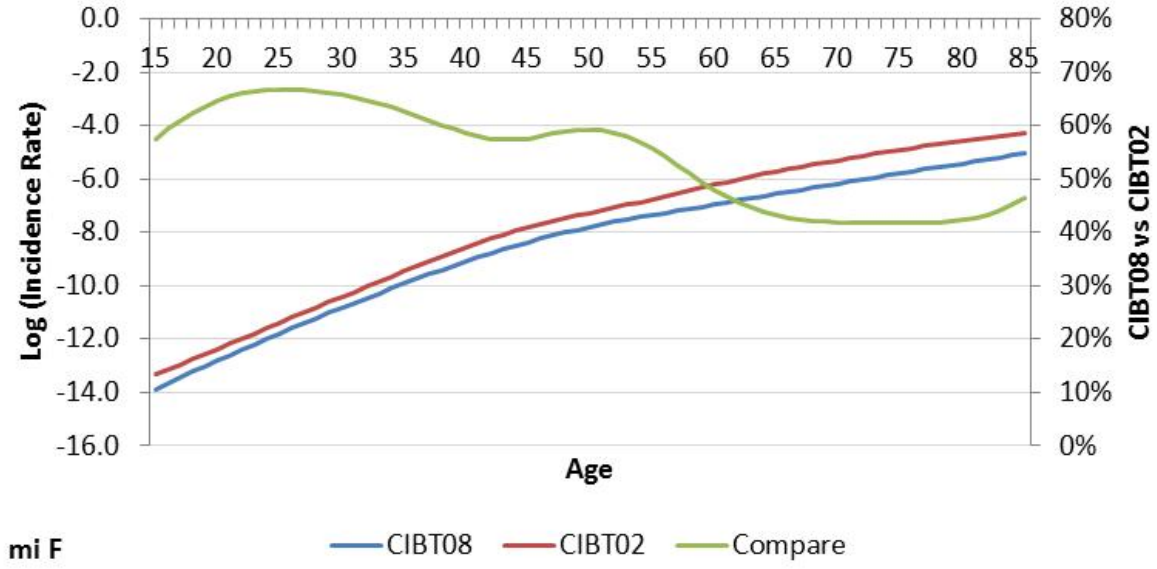
6.2.6.2. Female Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.42	5.34	35.00
Adjustment for Overlap	-15.2%	-15.2%	-29.5%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.37	4.76	29.59
28 Day Mortality Rates	-18.6%	-14.6%	-27.9%
Stand Alone Rates I'x	0.30	4.04	20.27
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.8%	2.4%	4.3%
Addition for Accelerated Rates Ix - kxqx	0.32	4.15	22.37

mi _ F

As with males the blue line reflects the new smoothed crude rates (CIBT08) and these are compared against those from CIBT02. The green line displays CIBT08 as a percentage of CIBT02.



As for males we observe a substantial reduction in the crude rate.

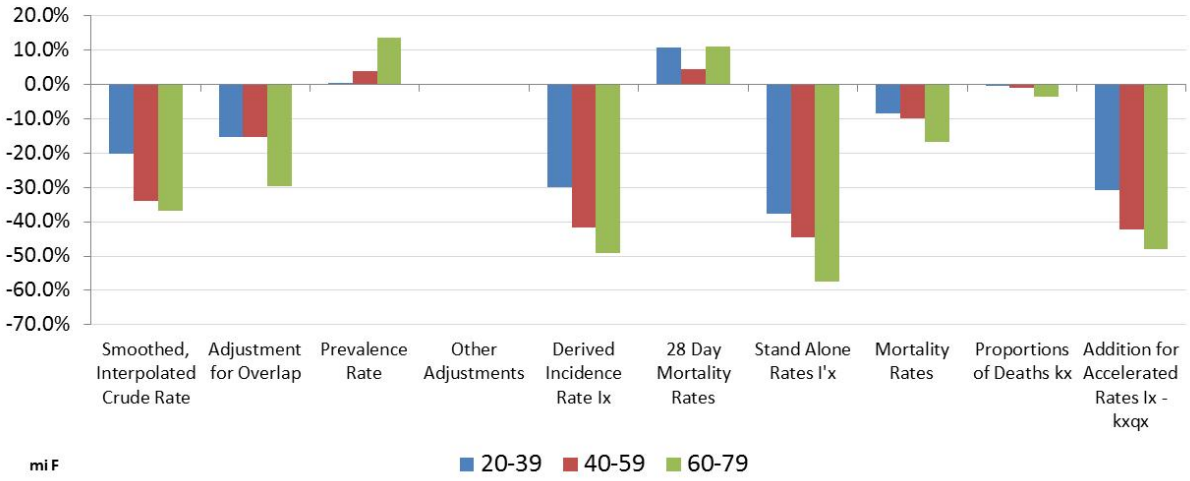
The table below contrasts the sudden death gross up factors derived for CIBT08 with those used for CIBT02. The figures have not been age-standardised.

Sudden death gross-up factors (females)

Age band	2010 data used for CIBT08	CIBT02
20-39	116%	106%
40-59	112%	107%
60-79	124%	110%

As for males the factors used to construct CIBT08 partial offset the reduction in first ever hospitalised MI rates.

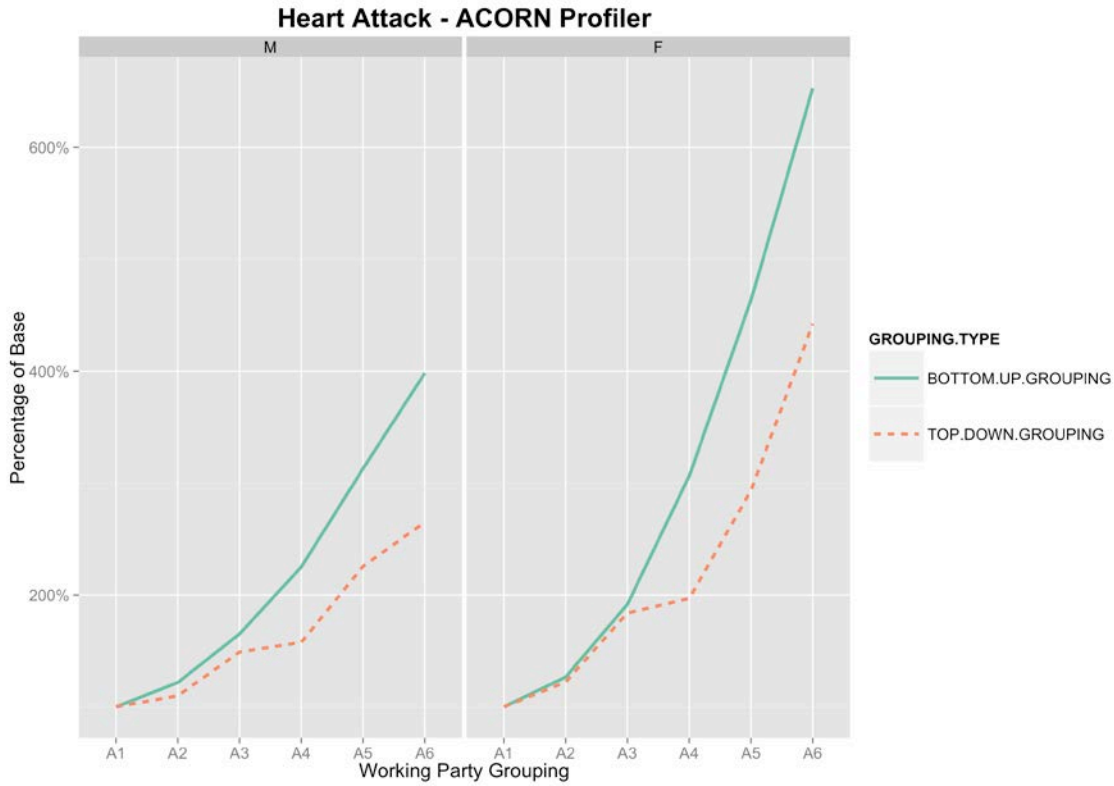
The following chart illustrates the movement of each of these components relative to CIBT02, for the same 3 age bands:



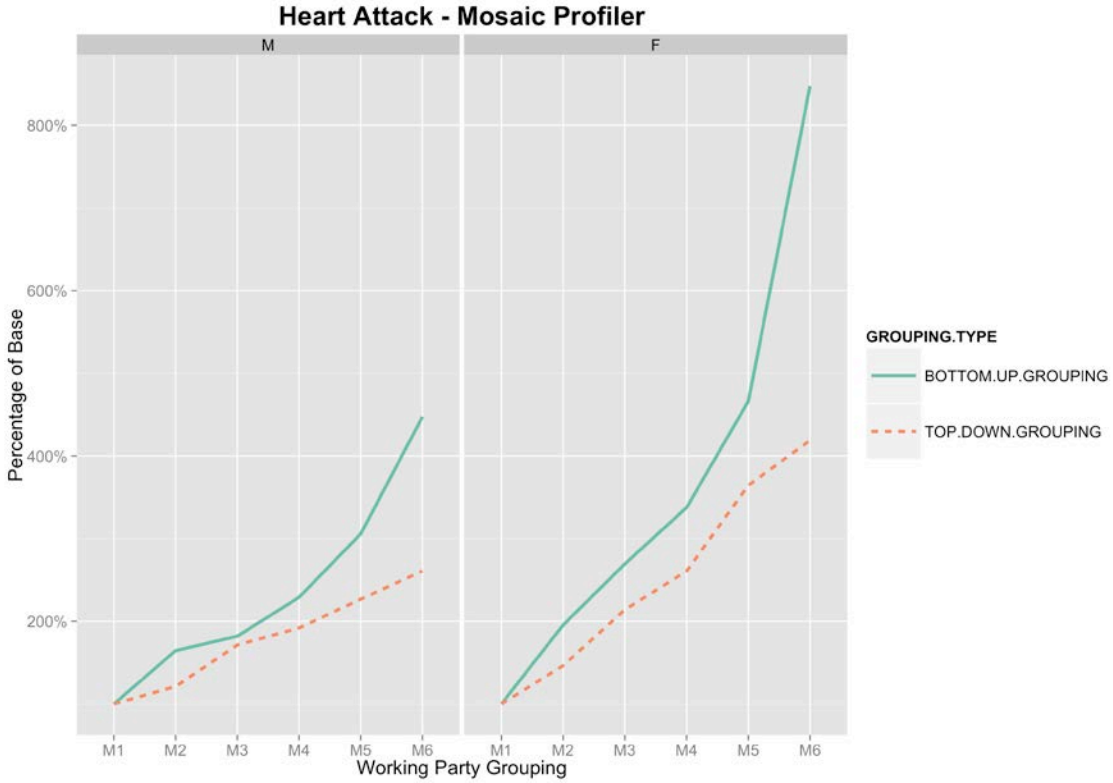
The changes are broadly consistent with those observed for males.

6.2.7. Geodemographic Analysis

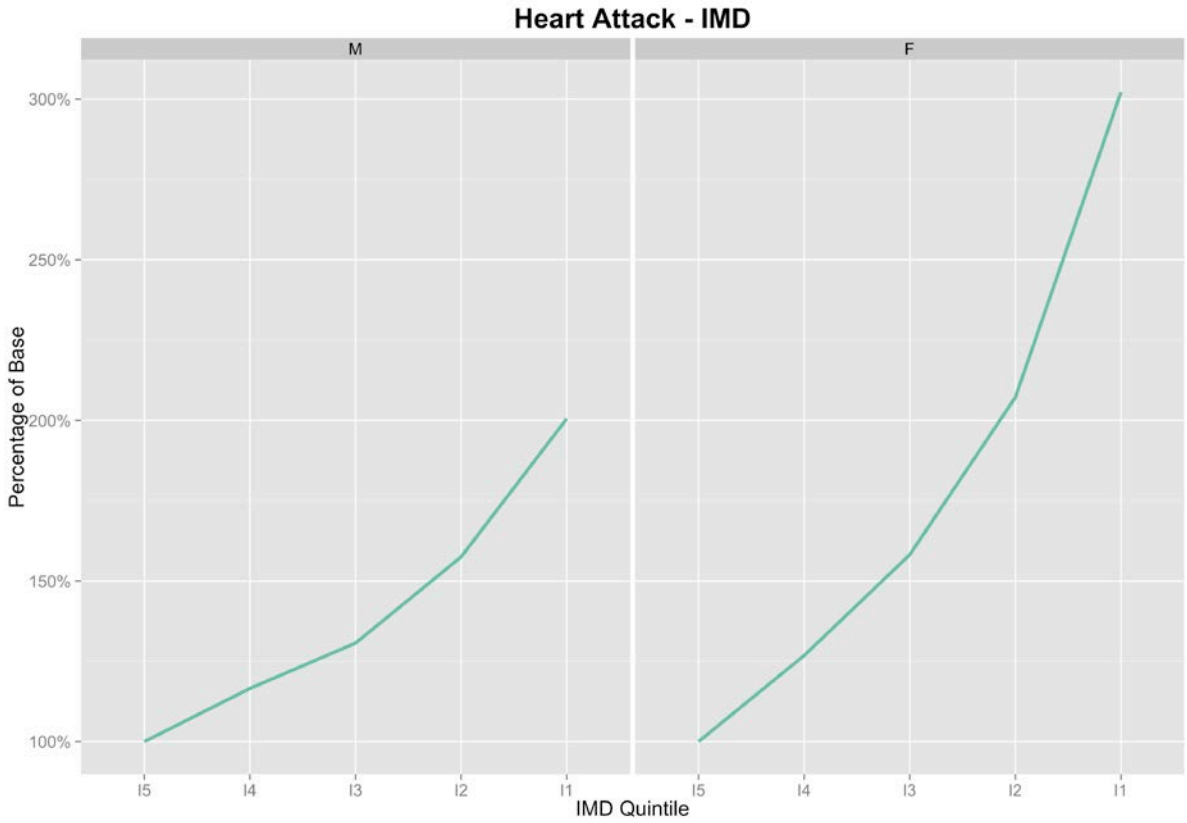
6.2.7.1. ACORN



6.2.7.2. Mosaic



6.2.7.3. Index of Multiple Deprivation



We observe a stronger positive gradient in incidence against deprivation for MI than we see for the top 3 CI conditions combined. The ACORN and Mosaic results have many similarities. The IMD measure does not appear to be as effective at isolating socio-economic differences in MI incidence.

6.3. Stroke

6.3.1. What is it?

A stroke means that the blood supply to a part of the brain is suddenly cut off. The brain cells need a constant supply of oxygen from the blood. Soon after the blood supply is cut off, the cells in the affected area of brain become damaged, or die. A stroke is sometimes called a “brain attack”. A stroke is a medical emergency and can cause permanent neurological damage and death.

The two most common types of stroke are ischaemic and haemorrhagic strokes:

- **Ischaemic strokes** happen when the artery that supplies blood to the brain is blocked, for example by a blood clot.
- **Haemorrhagic strokes** happen when a blood vessel bursts and bleeds into the brain, damaging brain tissue and starving some of the brain cells of blood and oxygen.

Acute ischaemic stroke can be classified according to various systems. Classification can be based on initial symptoms, based on the extent of the symptoms, some take into consideration the area of the brain affected, the underlying cause and the prognosis; some also based on clinical symptoms as well as results of further investigations.

Each year around 120,000 people in the UK have a first stroke, and about 30,000 have a recurrent stroke. Stroke is the largest cause of disability in the UK, and the third most common cause of death (after heart disease and cancer). Most cases occur in people aged over 65. Each year about 1 in 100 people over the age of 75 will have a stroke. But a stroke can occur at any age - even in babies. About one million people in the UK are living with the effects of stroke. Half of these people depend on others for help with everyday activities.

6.3.2. Symptoms and Treatment

Symptoms can include numbness, hyperaesthesia (increased sensitivity), paralysis, localised weakness, dysarthria (difficulty with speech), aphasia (inability to speak), dysphagia (difficulty in swallowing), visual impairment, difficulty in walking, lack of coordination, tremor, seizures, lethargy, dementia, delirium and coma.

Stroke symptoms typically start suddenly, over seconds to minutes, and in most cases do not progress further. The symptoms depend on the area of the brain affected. The more extensive the area of brain affected, the more functions that are likely to be lost. Some forms of stroke can cause additional symptoms. For example, in intracranial haemorrhage, the affected area may compress other structures. Most forms of stroke are not associated with headache, apart from subarachnoid haemorrhage and cerebral venous thrombosis and occasionally intracerebral haemorrhage.

Initially an ischemic stroke can be treated in a hospital with thrombolysis (also known as a "clot buster"), and some haemorrhagic strokes benefit from neurosurgery. The main intention is to re-establish the blood supply to the brain as quickly as possible in order to restrict the amount of brain treatment so as to recover any lost function - this is termed as stroke rehabilitation. Stroke rehabilitation should ideally be in a stroke unit and involving health professions such as speech and language therapy, physical therapy and occupational therapy. Prevention of recurrence may involve the administration of antiplatelet drugs such as aspirin and dipyridamole, control and reduction of high blood pressure, and the use of statins. Selected patients may benefit from carotid endarterectomy and the use of anticoagulants.

6.3.3. Risk Factors

Risk factors for stroke include old age, high blood pressure, previous stroke or transient ischemic attack (TIA), diabetes, high cholesterol, tobacco smoking and atrial fibrillation. High blood pressure is the most important modifiable risk factor of stroke. It is the second leading cause of death worldwide.

6.3.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

> **Stroke (1999/2002)**
>
> A cerebrovascular incident resulting in permanent neurological damage. Transient Ischaemic Attacks are specifically excluded.

It is death of brain tissue due to inadequate blood supply or haemorrhage within the skull resulting in permanent neurological deficit with persisting clinical symptoms. Symptoms of dysfunction in the nervous system that are present on clinical examination and expected to last throughout the insured person's life.

> **Stroke – resulting in permanent symptoms (2006/2011)**
>
> Death of brain tissue due to inadequate blood supply or haemorrhage within the skull resulting in permanent neurological deficit with persisting clinical symptoms.
>
> For the above definition, the following are not covered:
>
> - Transient ischaemic attack
> - Traumatic injury to brain tissue or blood vessels.

Most insurers have definitions consistent with the ABI above. Many have also removed the exclusion relating to "traumatic injury to brain tissue or blood vessels" in order to claim that benefit is enhanced to gain "ABI+" status.

Some companies have extended cover by not including criteria of "permanent neurological deficit". Several pay full benefit with the more generous definition, whilst others have chosen to pay a partial benefit where, following a stroke, a complete recovery is made and continue to pay full benefit where there are permanent symptoms.

6.3.5. Derived Incidence Rates

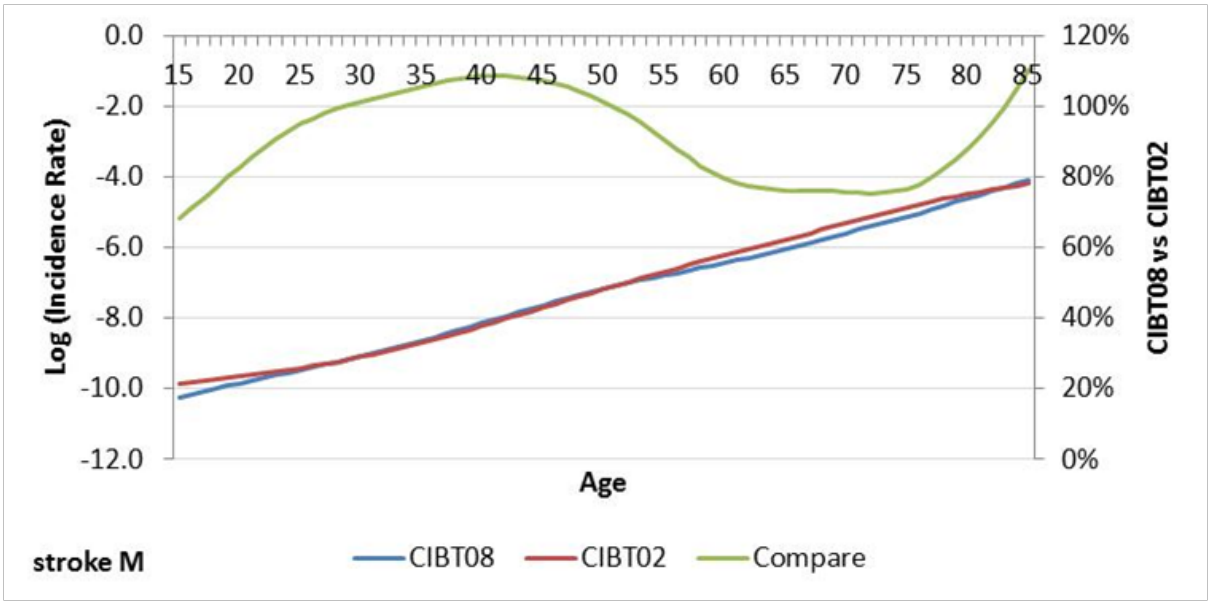
6.3.5.1. Male Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.64	9.67	46.47
Adjustment for Overlap	-14.7%	-18.3%	-34.9%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	1.42	8.32	41.78
28 Day Mortality Rates	-8.9%	-8.7%	-11.4%
Stand Alone Rates I'x	1.29	7.59	36.49
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	1.7%	3.4%	4.2%
Addition for Accelerated Rates Ix - kxqx	1.25	7.07	31.52

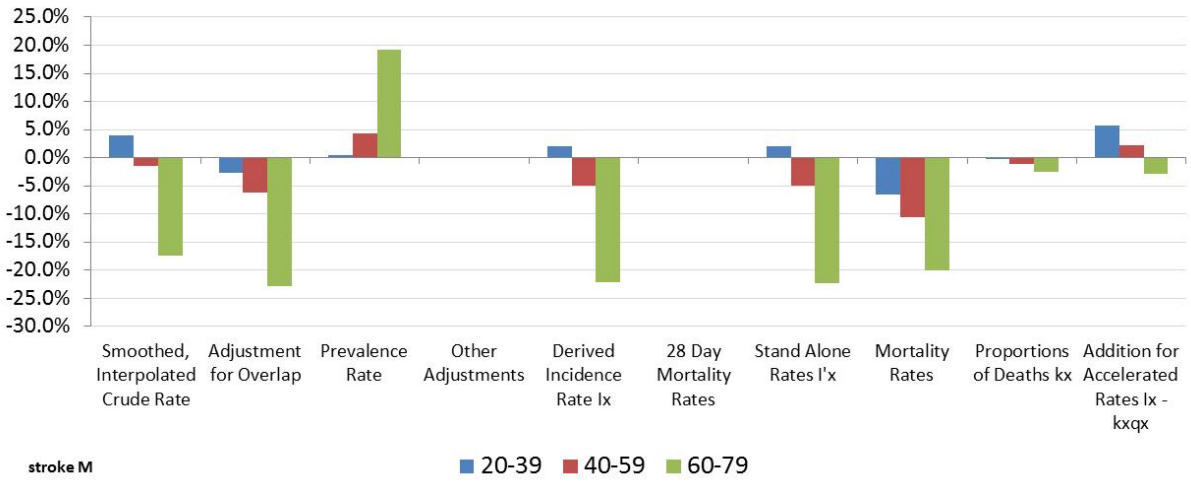
stroke _ M

The smoothed crude rates are shown in the chart below for both the newly derived rates (CIBT08) and those from CIBT02. The green line, with values on the 2nd axis, displays CIBT08 as a percentage of CIBT02.



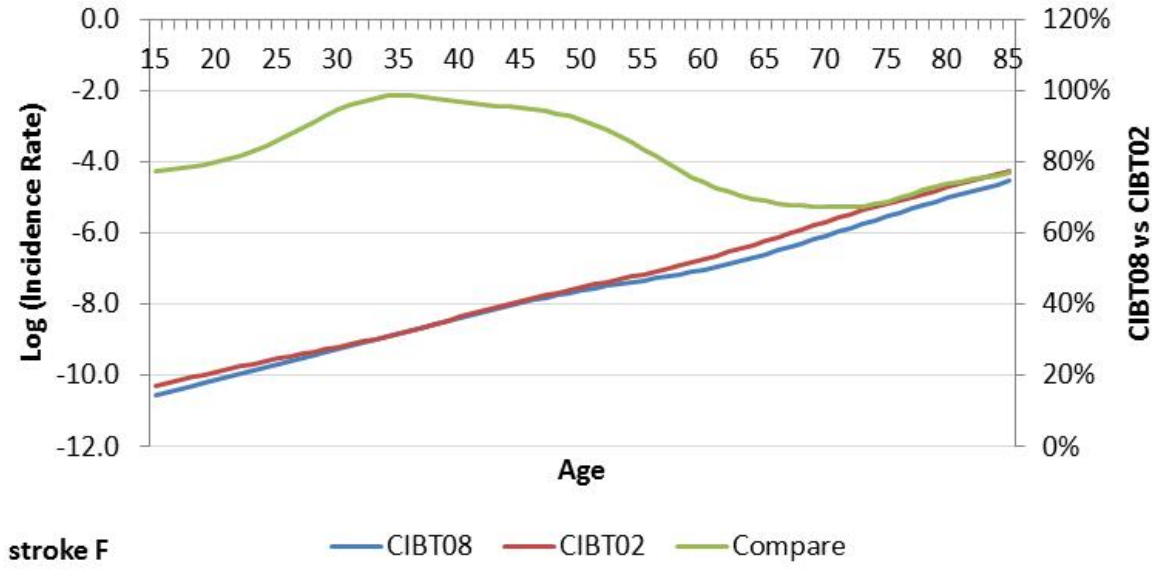
There is a distinct age-shape to the comparison of CIBT08 and CIBT02. CIBT08 is generally lower than CIBT02 at very young ages (less than 25) and ages 55-75, comparable over ages 25-45 and much higher at older ages. Excluding these extreme ages, the new incidence rates are broadly comparable to those in CIBT02.

The following chart illustrates the movement of each of these components relative to CIBT02, for the same 3 age bands:



6.3.5.2. Female Results

As with males the blue line reflects the new smoothed crude rates (CIBT08) and these are compared against those from CIBT02. The green line displays CIBT08 as a percentage of CIBT02.

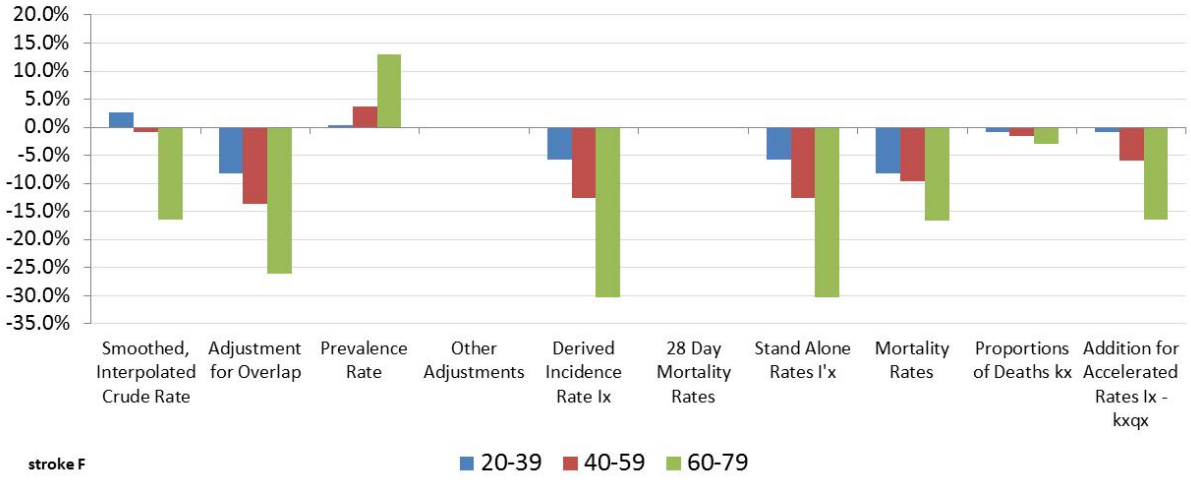


In order to move from a smoothed crude rate to a standalone and accelerate CI rate, we have followed an equivalent process to that used in Exploring the Critical Path. The following table below provides a summary of the adjustments by 3 age bands.

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.35	6.37	33.82
Adjustment for Overlap	-14.2%	-19.6%	-32.0%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	1.17	5.35	28.16
28 Day Mortality Rates	-9.8%	-10.6%	-14.2%
Stand Alone Rates I'x	1.05	4.78	23.77
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	2.5%	4.2%	5.1%
Addition for Accelerated Rates Ix - kxqx	1.05	4.37	19.21

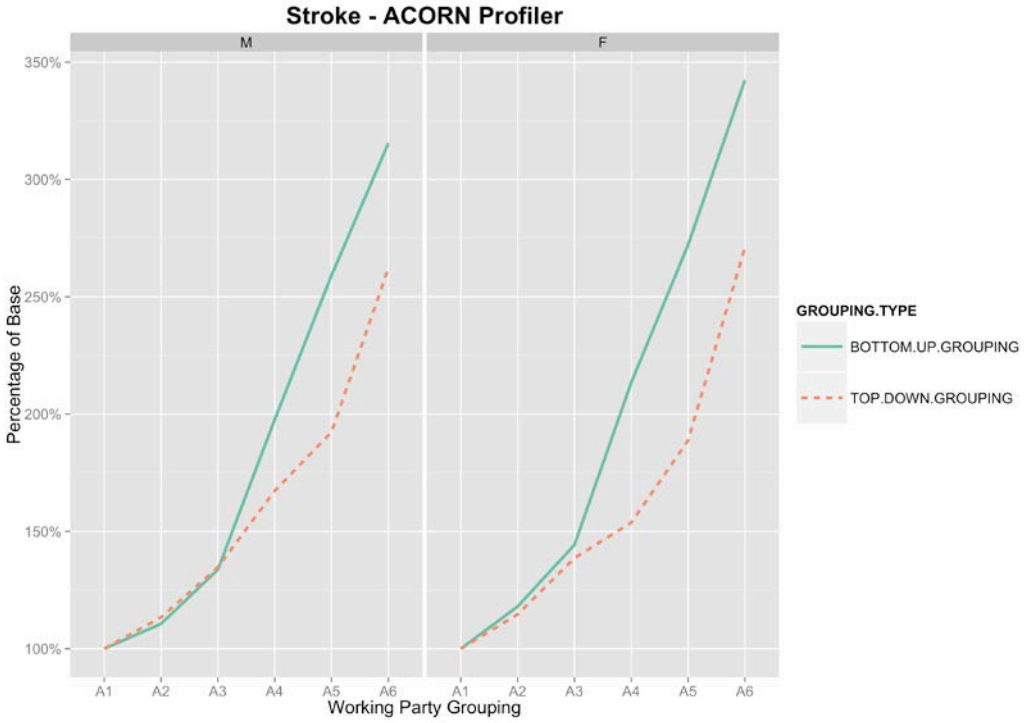
stroke _ F

The following chart illustrates the movement of each of these components relative to CIBT02, for the same 3 age bands:



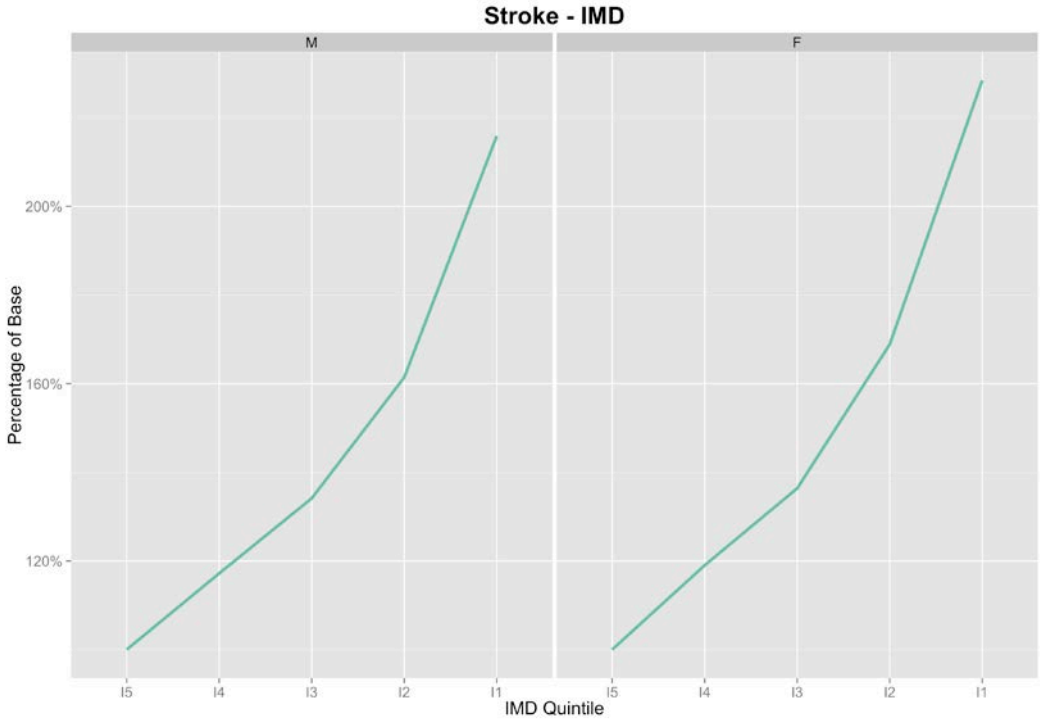
6.3.6. Geodemographic Analysis

6.3.6.1. ACORN



6.3.6.2. Mosaic

6.3.6.3. Index of Multiple Deprivation



6.4. Alzheimer’s Disease

6.4.1. What is it?

Alzheimer's disease is the most common cause of dementia. It is a progressive illness that destroys brain cells and nerve cells thereby disrupting the transmitters which carry messages in the brain, particularly those responsible for storing memories.

During the course of Alzheimer's disease, nerve cells die in particular regions of the brain. The brain shrinks as gaps develop in the temporal lobe and hippocampus, which are responsible for storing and retrieving new information. This in turn affects people's ability to remember, speak, think and make decisions. It is not known what causes nerve cells to die but there are characteristic appearances of the brain after death. In particular, 'tangles' and 'plaques' made from protein fragments are observed under the microscope in damaged areas of brain. This confirms the diagnosis of Alzheimer's disease

Alzheimer's disease is most common in people over 65 years of age, and affects slightly more women than men.

The Alzheimer’s Society estimates that dementia affects around 650,000 people in England, with Alzheimer’s disease responsible for around 62% of dementia cases.

Dementia in people under 65 years of age, known as early-onset dementia, is less common. In the UK, around 2% of all dementia cases are early-onset dementia.

The risk increases with age, and people who are over 80 years of age are thought to have a one in six chance of developing the condition.

6.4.2. Symptoms and Treatment

Although Alzheimer's disease develops differently for every individual, there are many common symptoms. Early symptoms are often mistakenly thought to be 'age-related' concerns, or manifestations of stress. In the early stages, the most common symptom is difficulty in remembering recent events. When Alzheimer’s disease is suspected, the diagnosis is usually confirmed with tests that evaluate behaviour and thinking abilities, often followed by a brain scan if available. As the disease advances, symptoms can include confusion, irritability, aggression, mood swings, trouble with language, and long-term memory loss. Gradually, bodily functions are lost, ultimately leading to death. Since the disease is different for each individual, predicting how it will affect the person is difficult. Alzheimer’s disease develops for an unknown and variable amount of time before becoming fully apparent, and it can progress undiagnosed for years. On average, the life expectancy following diagnosis is approximately seven years. Fewer than 3% of individuals live more than fourteen years after diagnosis.

The cause and progression of Alzheimer's disease are not well understood. Current treatments only help with the symptoms of the disease. There are no available treatments that stop or reverse the progression of the disease. Mental stimulation, exercise, and a balanced diet have been suggested as ways to delay cognitive symptoms (though not brain pathology) in healthy older individuals, but there is no conclusive evidence supporting an effect.

Because Alzheimer’s disease cannot be cured and is degenerative, the sufferer relies on others for assistance. The role of the main caregiver is often taken by the spouse or a close relative. Alzheimer's disease is known for placing a great burden on caregivers; the pressures can be wide-ranging, involving social, psychological, physical, and economic elements of the caregiver's life. In developed countries, Alzheimer’s disease is one of the most costly diseases to society.

6.4.3. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

> **Alzheimer's Disease [before age x] – resulting in permanent symptoms (2006/2011)**
>
> A definite diagnosis of Alzheimer's disease [before age x] by a Consultant Neurologist, Psychiatrist or Geriatrician. There must be permanent clinical loss of the ability to do all of the following:
> - remember;
> - reason; and
> - perceive, understand, express and give effect to ideas.
>
> For the above definition, the following are not covered:
> - Other types of dementia.

6.4.4. Derived Incidence Rates

Our Alzheimer's rates are significantly lower than those derived in Exploring the Critical Path as we would expect given the majority of Alzheimer's cases would not be treated as in-patient admissions.

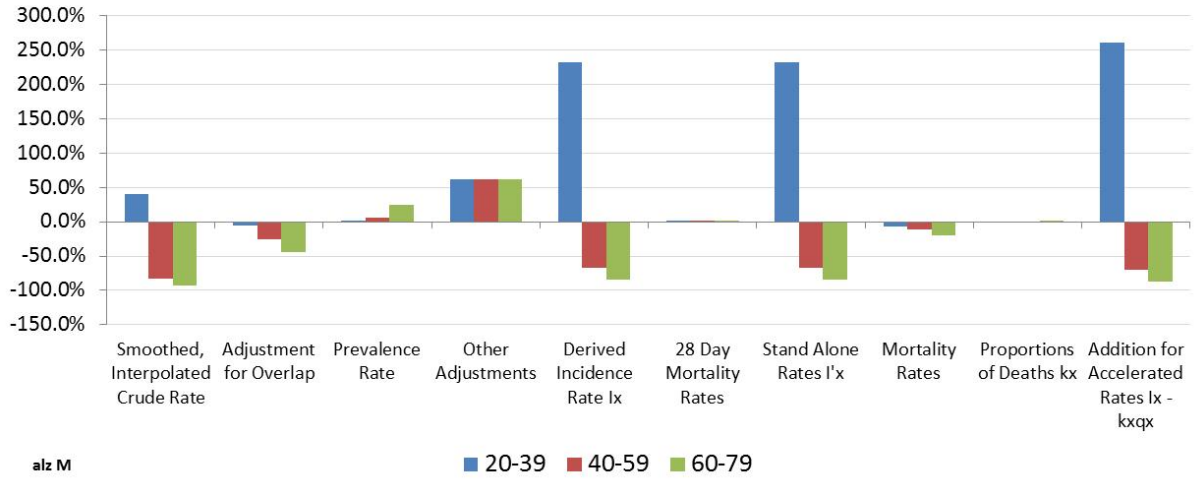
Overall we are not comfortable with these rates as true estimates of the incidence of Alzheimer's disease. We have included them for consistency with our overall methodology and in particular because of the interaction with our first ever CI calculations.

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.01	0.22	7.50	0.01	0.22	8.95
Adjustment for Overlap	-6.0%	-25.7%	-44.3%	-24.2%	-32.6%	-37.5%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.01	0.22	7.50	0.01	0.22	8.95
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.01	0.17	5.94	0.01	0.16	6.85
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.4%	0.0%	0.1%	0.8%
Addition for Accelerated Rates Ix - kxqx	0.01	0.15	4.67	0.01	0.13	5.25

alz

The following graphs compare the rates we have derived with those in CIBT02.



6.5. Aorta Graft Surgery

6.5.1. What is it?

Aorta Graft Surgery is for disease to thoracic and abdominal aorta with excision and surgical replacement of a portion of the diseased aorta with a graft. Three common diseases of the Aorta are Aortic aneurysm, Aortic dissection and Aortic coarctation.

Aneurysm is a permanent localised dilation of the artery and is classified by shape, location along the aorta, and how it is formed. There are two types: Abdominal aneurysm and Thoracic aneurysm.

Dissection is the tearing of the inner layer of the aortic wall, allowing blood to leak into the wall itself and cause the separation of the inner and outer layers. There are three types: Type A dissection; Type B dissection; Thoracic Aortic injury.

Coarctation is a narrowing of a section of the aorta, just beyond the aortic arch as it bends down to descend to the lower body. This is a congenital condition and can be diagnosed and treated in childhood or in adult patients with the treatment of hypertension.

6.5.2. Symptoms and Treatment

Symptoms of the disease vary depending on whether it is an aneurysm or a dissection.

The risk of an aneurysm rupturing and causing life threatening internal bleeding increases as the size of the dilation increases.

Dissection is usually associated with severe chest pain and back pain.

Traumatic injury is associated with pain and can be fatal.

The treatment of aorta is a combination of medical treatment and lifestyle changes.

The medical treatment is aimed at protecting the graft, the remaining aorta, the aortic valve and the cardiovascular system. The aorta can undergo medical treatment at a number of different sites depending on the condition. The operational procedures vary depending on the condition. The operational procedure can also be an emergency or interventional.

Lifestyle recommendations includes blood pressure optimisation, review of the lifestyle management and addresses diet, exercise and smoking cessation.

6.5.3. Risk Factors

The risk factors contributing to the disease of Aorta are:

- High blood pressure or hypertension;
- Atherosclerosis with the hardening of the arteries due to build-up of the cholesterol and other fatty deposits;
- Congenital weakness of the artery wall from infectious and inflammatory conditions;
- Weakening of the artery wall from smoking and other unhealthy lifestyle choices;
- Dissection or tearing of the artery walls due to traumatic injury such as slips and falls; motor vehicle accidents;
- Increasing age;
- Genetic factors.

6.5.4. Insurance Industry Definitions

The following are the definitions that have been included in ABI Statements of Best Practice in the past:

Aorta Graft Surgery (1999/2002)

Undergoing of surgery for disease of the aorta needing surgical replacement of a portion of the diseased aorta with a graft.. For this definition, aorta means the thoracic and abdominal aorta but not its branches.

Aorta Graft Surgery –*for disease* (2006/2011)

The undergoing of surgery for disease to the aorta with excision and surgical replacement of a portion of the diseased aorta with a graft.

The term aorta includes the thoracic and abdominal aorta but not its branches.

For the above definition, the following are not covered:

- Any other surgical procedure, for example the insertion of stents or endovascular repair.
- Surgery following traumatic injury to the aorta.

Most companies have enhanced this definition to include coverage for traumatic injury to the aorta in order to gain ABI+ status.

6.5.5. Derived Incidence Rates

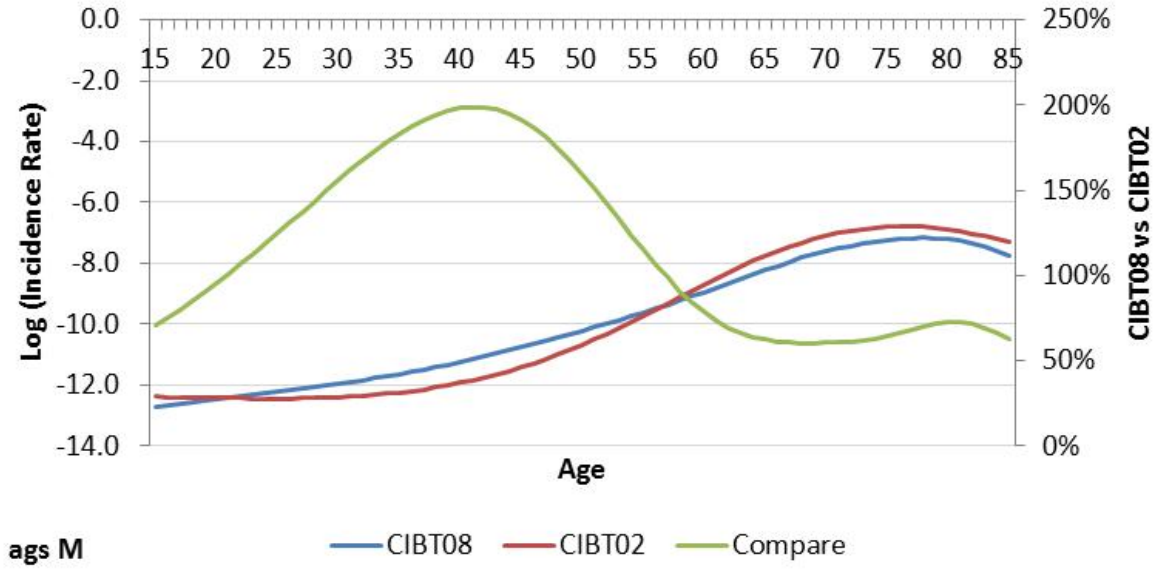
6.5.5.1. Male Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000):

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.09	0.66	7.42
Adjustment for Overlap	-22.5%	-34.8%	-58.1%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.07	0.42	4.22
28 Day Mortality Rates	-0.6%	-0.8%	-2.2%
Stand Alone Rates I'x	0.07	0.42	4.11
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.07	0.42	4.15

ags _ M

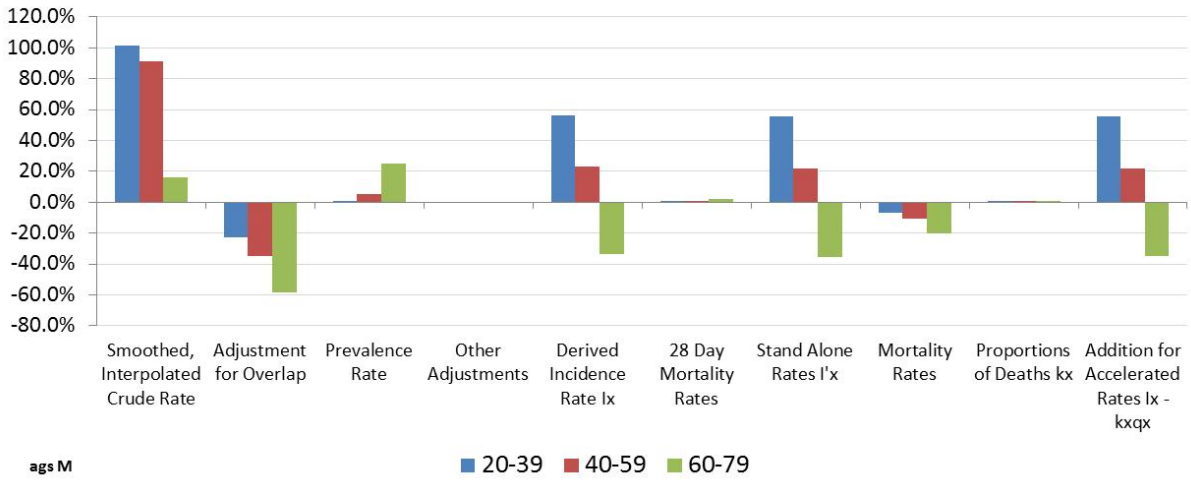
The standalone incidence rates for Aorta Graft Surgery are shown in the chart below for both the newly derived rates (CIBT08) and those from CIBT02. The green line, with values on the 2nd axis, displays CIBT08 as a percentage of CIBT02.



The CIBT08 rates profile is similar in shape to that of CIBT02 rates. The CIBT08 standalone rates are higher than CIBT02 rates in the lower and middle age bands but lower in the higher age band.

As we can see the CIBT08 rates range between 50% and 200% of the CIBT02 rates, although the difference does seem to reduce with age.

The following chart illustrates the movement of each of these components relative to CIBT02, for the same 3 age bands. As we can see the increase in rates can be largely attributed to a significant increase in the crude rates, although at the older ages this is offset by the increased adjustment for overlaps.



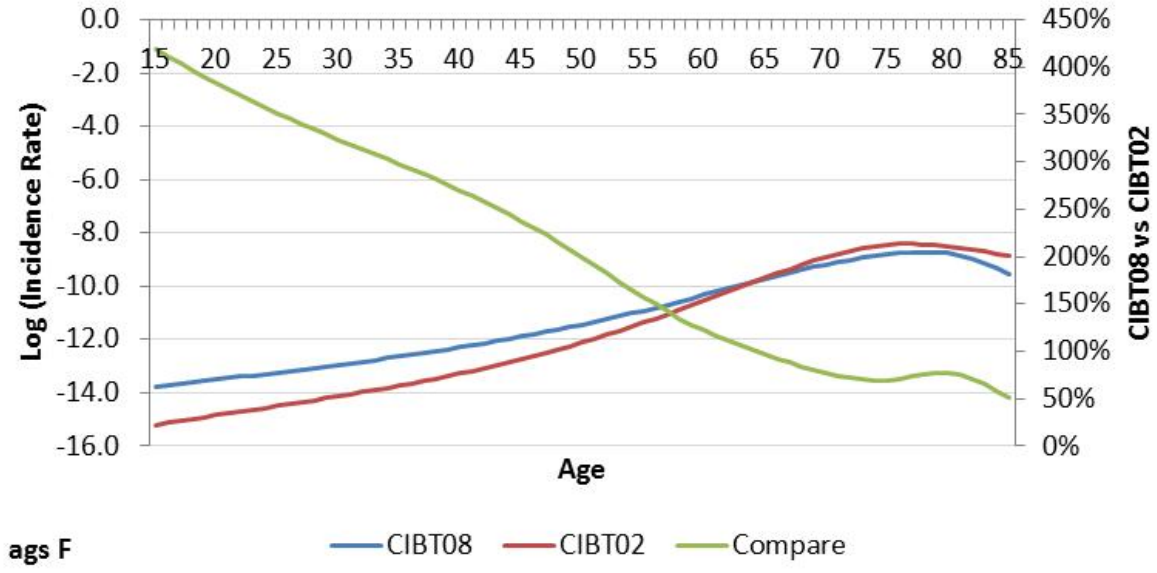
6.5.5.2. Female Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

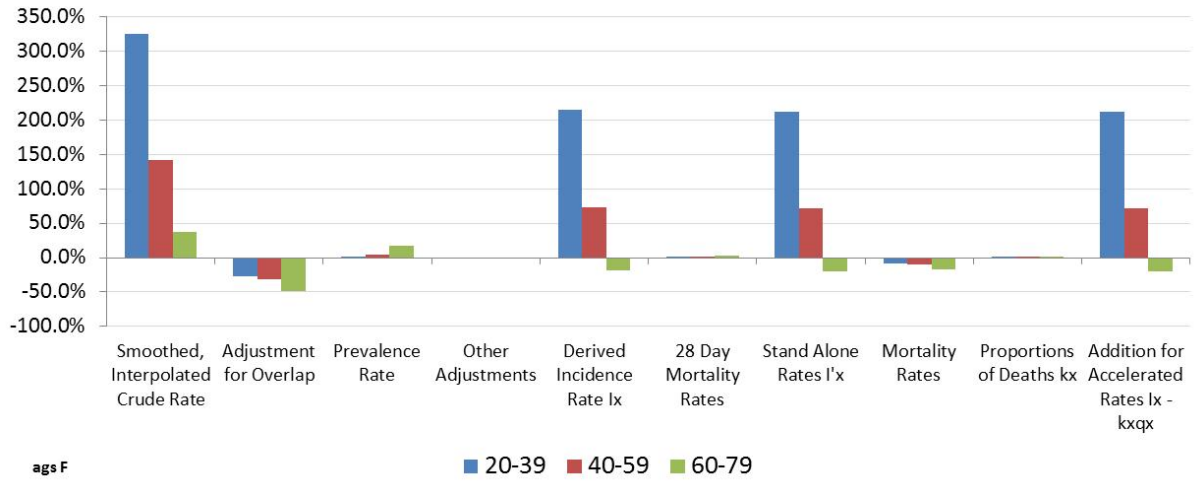
Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.03	0.17	1.59
Adjustment for Overlap	-26.8%	-30.9%	-48.8%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.03	0.12	0.94
28 Day Mortality Rates	-0.6%	-0.8%	-2.3%
Stand Alone Rates I'x	0.03	0.12	0.91
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.03	0.12	0.92

ags _ F

As with males the blue line reflects the standalone rates (CIBT08) and these are compared against those from CIBT02. The green line displays CIBT08 as a percentage of CIBT02.

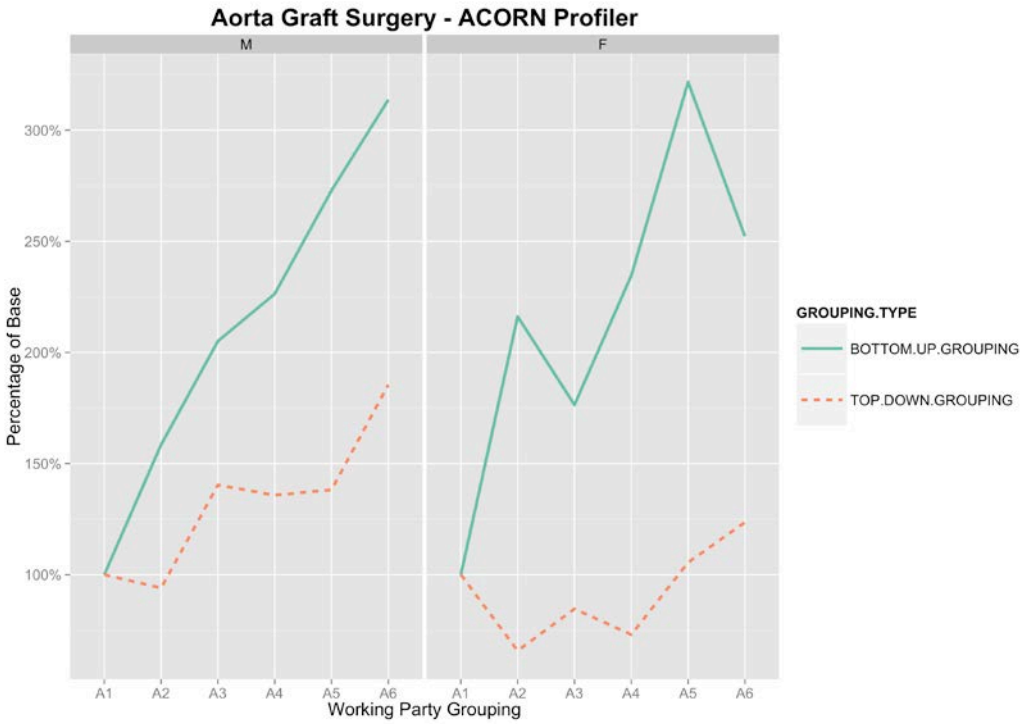


As we saw with the male rates the new CIBT08 rates are significantly higher than the old CIBT02 rates at the younger ages, although the difference reduces with age. The table below shows this difference is again due largely to the increase in the crude rates.

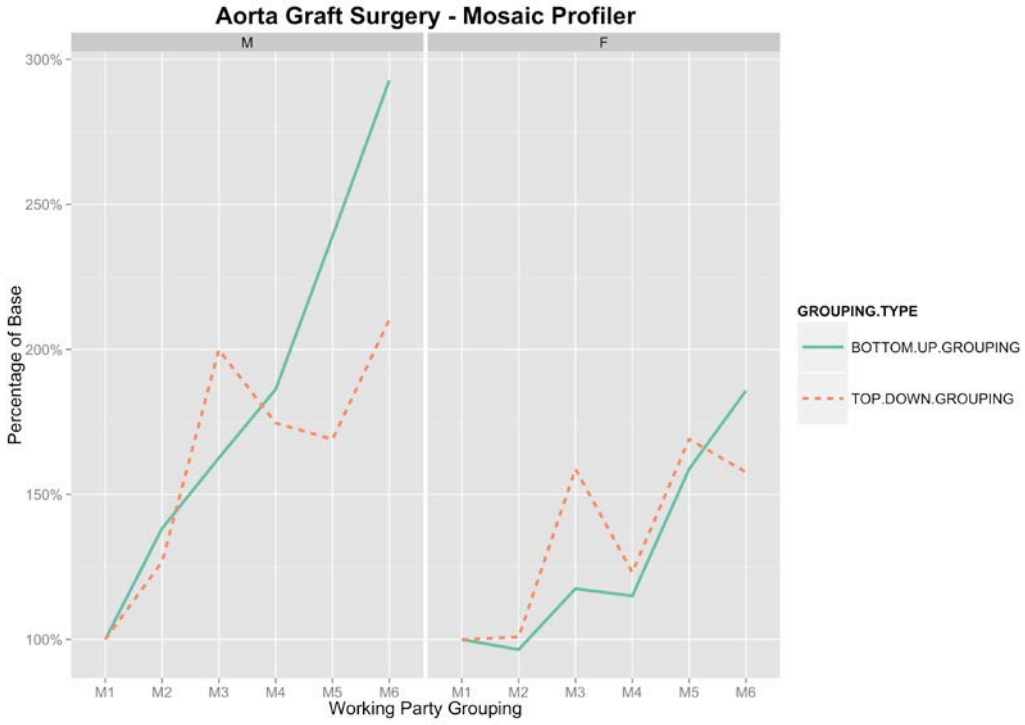


6.5.6. Geodemographic Analysis

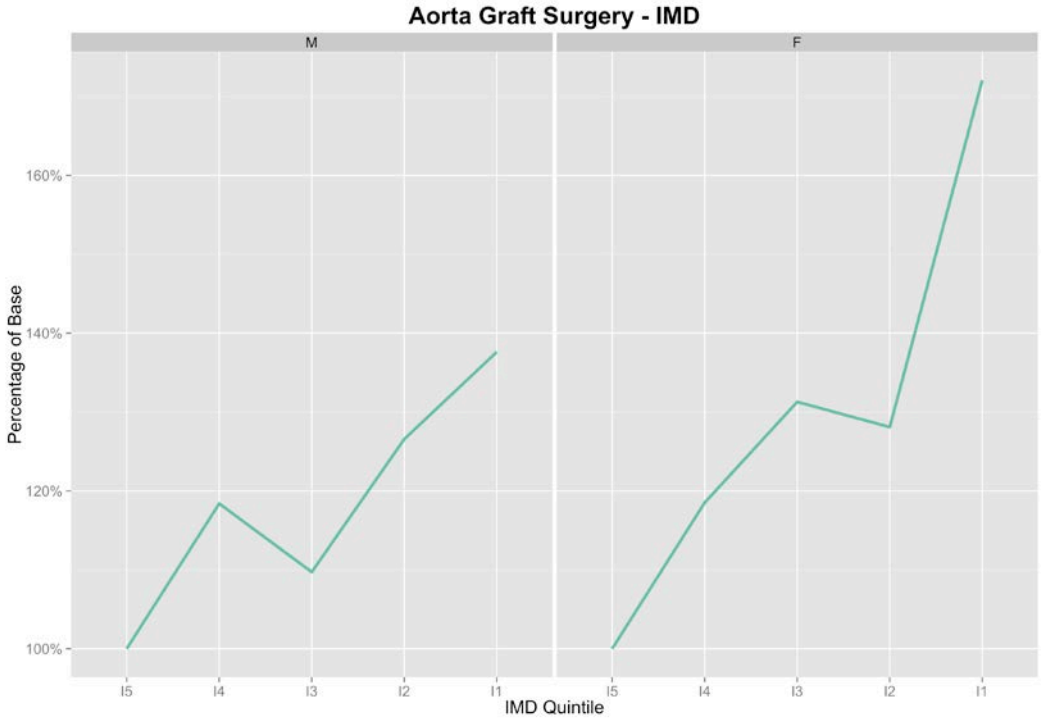
6.5.6.1. ACORN



6.5.6.2. Mosaic



6.5.6.3. Index of Multiple Deprivation



6.6. Aplastic Anaemia

6.6.1. What is it?

Aplastic anaemia is a disease in which the bone marrow, and the blood stem cells that reside there, are damaged. This causes a deficiency of all three blood cell types: red blood cells, white blood cells and platelets. Aplastic refers to inability of the stem cells to generate the mature blood cells.

It occurs most commonly in the teens and twenties, and also among the elderly. It can be caused by exposure to chemicals, drugs, radiation, infection, immune disease, and heredity; in about half the cases, the cause is unknown.

The definitive diagnosis is by bone marrow biopsy; normal bone marrow has 30-70% blood stem cells, but in aplastic anaemia, these cells are mostly gone and replaced by fat.

Although there are some similarities with cancer or leukaemia, aplastic anaemia is generally not considered to be a malignant condition. However, it may be associated with cancer and leukaemia treatments and occasionally it may also develop into leukaemia,

6.6.2. Symptoms and Treatment

Symptoms of aplastic anaemia are as follows:

- Anaemia with malaise, pallor and associated symptoms such as palpitations
- Thrombocytopenia (low platelet counts), leading to increased risk of haemorrhage, bruising and petechiae.
- Leukopenia (low white blood cell count), leading to increased risk of infection
- Reticulocytopenia (low counts of immature red blood cells)

Aplastic anaemia is treated with immunosuppressive drugs, typically either anti-lymphocyte globulin or anti-thymocyte globulin, combined with corticosteroids and cyclosporine, with a response rate of about 70%; this indicates that aplastic anaemia has an auto-immune component. Stem cell transplant is also used, especially for patients younger than 30 years of age.

6.6.3. Insurance Industry Definitions

Aplastic anaemia does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market. However approximately 70% of companies do cover aplastic anaemia in their CI policies illnesses.

A few examples of the current definitions used by insurers are as follows:

Aplastic Anaemia - of specified severity

Confirmation by a consultant haematologist of a definite diagnosis of complete bone marrow failure which results in anaemia, neutropenia and thrombocytopenia and requires as a minimum, one of the following treatments:

- blood transfusion
- bone-marrow transplantation
- immunosuppressive agents
- marrow stimulating agents

All other forms of anaemia are specifically excluded.

Aplastic Anaemia – with permanent bone marrow failure

A definite diagnosis of aplastic anaemia by a consultant haematologist. There must be permanent bone marrow failure with anaemia, neutropenia and thrombocytopenia.

6.6.4. Derived Incidence Rates

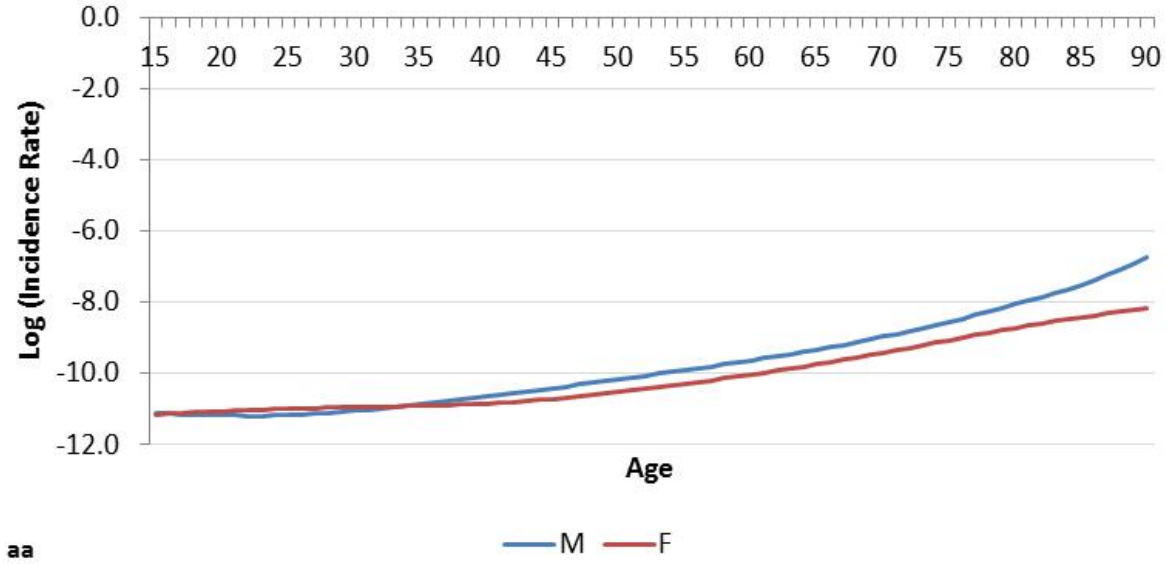
Aplastic Anaemia rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2.

Rates shown below are per 10,000.

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.28	0.73	2.60	0.29	0.62	1.82
Adjustment for Overlap	-40.5%	-48.7%	-65.2%	-39.7%	-56.6%	-64.8%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.17	0.38	1.26	0.18	0.27	0.81
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.17	0.38	1.26	0.18	0.27	0.81
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.1%	0.0%	0.0%	0.1%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.16	0.38	1.21	0.17	0.27	0.76

aa

The graph below shows a smooth progression of rates by age:



aa

6.7. Bacterial Meningitis

6.7.1. What is it?

Bacterial meningitis is inflammation of the meninges which are protective membranes covering the brain and spinal cord. In some cases the inflammation can damage the nerves and brain resulting in permanent disability or death in the most severe cases.

There are several different types of bacteria that can cause bacterial meningitis and are the same bacteria that can cause septicaemia. These bacteria may be transmitted from prolonged close contact with an infected person or may be carried ordinarily by healthy people but cause a problem when the immune system is compromised.[18]

Vaccinations are available for some but not all types of bacterial meningitis.

Viral meningitis is the most common and less serious type of meningitis.

6.7.2. Symptoms and Treatment

Symptoms include sudden onset of fever, headache, stiff neck, nausea, vomiting, increased sensitivity to light and confusion. As the infection progresses, seizures and coma may occur. The infection can progress rapidly and in the most severe cases can cause death or permanent disability within hours of onset of symptoms.

Treatment will usually begin immediately, even for suspected cases, because diagnostic tests can take several hours to complete and delays are dangerous.[19] Diagnostic tests may include:

- a blood test – to check for the presence of bacteria or viruses that can cause meningitis;
- a lumbar puncture – where a sample of cerebrospinal fluid (CSF) is taken from the base of the spine and checked for the presence of bacteria or viruses;
- a computerised tomography (CT) scan – if there are any other suspected problems, such as damage to the brain;
- a chest X-ray to look for signs of infection.

Bacterial meningitis is treated with antibiotics. It is important to establish which type of bacteria is involved in order to prescribe the most effective antibiotic.

6.7.3. Risk Factors

- Age – while people of all ages are at risk, incidence rates are highest among infants and young children. The elderly are also at increased risk.
- Living in a community setting where germs are transmitted easily.
- Other medical conditions which weaken the immune system.

[18] http://www.cdc.gov/meningitis/bacterial.html

[19] http://www.nhs.uk/Conditions/Meningitis/Pages/diagnosis.aspx

6.7.4. Insurance Industry Definitions

Bacterial meningitis does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market. Definitions used in the market are very similar and an example is as follows:

> **Bacterial meningitis - resulting in permanent symptoms**
> A definite diagnosis of bacterial meningitis by a Consultant Neurologist resulting in permanent neurological deficit with persisting clinical symptoms.
>
> For the above definition, the following is not covered:
>
> • all other forms of meningitis other than those caused by bacterial infection.

6.7.5. Derived incidence rates

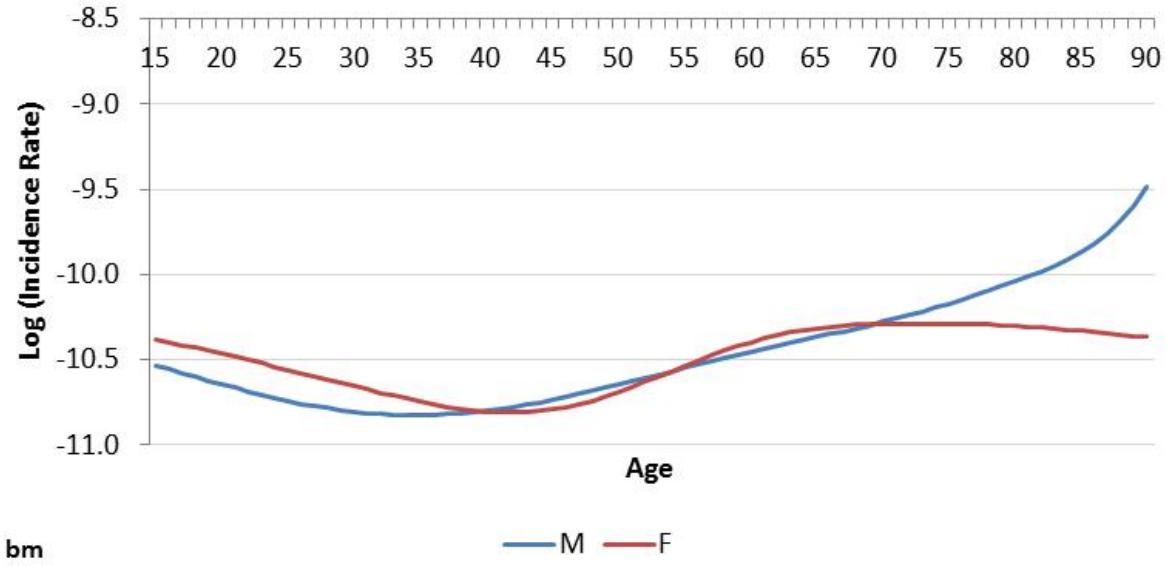
Bacterial meningitis rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2.

The HES data is not a good match for the insurance definition because in most cases the patient will make a full recovery. The HES data could have been queried to only include cases with hospital stays over a certain length as a proxy for severity. We have not done this.

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.25	0.30	0.44	0.28	0.30	0.43
Adjustment for Overlap	-15.0%	-25.2%	-43.1%	-14.2%	-26.5%	-36.6%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.21	0.24	0.34	0.24	0.23	0.33
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.21	0.24	0.34	0.24	0.23	0.33
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.1%	0.1%	0.0%	0.2%	0.1%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.20	0.20	0.29	0.23	0.21	0.28

bm



It is clear from the chart that, although there is an elevated incidence at the youngest and oldest ages, it is broadly level across the key insured ages with little difference between males and females.

6.8. Benign Brain Tumour

6.8.1. What is it?

A brain tumour is an intracranial solid neoplasm – an abnormal growth of cells – within the brain. The terms includes all tumours within the cranium

A **primary** tumour arises when brain cells grow and multiply abnormally. If the abnormal cells have spread to the brain from a cancerous tumour in another part of the body, this is referred to as a **secondary** or **metastatic** tumour.

Benign brain tumours are non-cancerous. They are always primary tumours but do not spread into and invade the brain tissue surrounding them. Furthermore they do not send secondary tumours to other parts of the body. However, benign tumours can grow to a considerable size creating pressure on and damaging the surrounding brain tissue.

Each year in the UK, about 5,000 people are diagnosed with a malignant brain tumour and about 4,300 with a benign brain tumour. Brain tumours can occur at any age but are more common in older people. About 300 children are diagnosed with a brain tumour each year.

There are different types of benign brain tumours, depending on the type of brain cells they have grown from. Common examples are:

- **Gliomas** - these are tumours of the glial (grey matter) tissue, which binds nerve cells and fibres together.
- **Meningiomas** - these are tumours of the membranes that cover the brain.
- **Acoustic neuromas** - these tumours grow in the acoustic nerve, which helps to control hearing and balance.
- **Craniopharyngiomas** - these tumours grow near the base of the brain and are most often diagnosed in children, teenagers and young adults.
- **Pituitary adenomas** - these are tumours of the pituitary gland (the pea-sized gland below the brain). However, these tumours are generally excluded from cover.

Whilst virtually all malignant brain tumours arise from glial brain tissue itself, by contrast, approximately only 15% of less aggressive tumours are within the brain, with almost 50% occurring in the meninges, 15% in other parts of the central nervous system and the remaining 25% in the intracranial endocrine glands such as the pituitary and pineal glands.

6.8.2. Symptoms and Treatment

The symptoms vary depending largely upon where the tumour is situated in the brain, its size and which parts of the brain it is affecting. Symptoms may include: memory problems; headaches; drowsiness; visual problems; speech problems; numbness or weakness; seizures (epilepsy).

Some people might also experience hormonal and personality changes. After the onset of symptoms, most people would usually see their GP who then refers them to a local hospital, or directly to a neuroscience centre, for tests and investigations. Generally diagnosis will be confirmed by an MRI or CT scan.

Treatment of benign brain tumours is similar to other brain tumour treatments except that chemotherapy is seldom done. Treatment protocols are based on the patient's age, the location and size of the tumour, and the patient's overall condition. Brain surgery (craniotomy) with surgical removal of tumour and/or radiation therapy (for example, conventional radiation, gamma knife, proton beam) are the main treatments. Often other drugs such as corticosteroids that reduce oedema (swelling) and help the brain heal are part of the treatment plan. Rarely are benign tumours untreatable. Where complete removal of

the tumour is possible, the prognosis is generally very good with the risk of recurrence being small. However, the outlook is very different where the tumour cannot be removed, which can occur if the tumour is situated in a site that is inaccessible. In such cases sub-total removal combined with radiotherapy is usual with the outcome being more guarded.

6.8.3. Risk Factors

There are only a few known risk factors that have been established by research. For example children who receive radiation to the head have a higher risk of developing a brain tumour as adults.

Similarly people who have certain rare genetic conditions such as neurofibromatosis also have a higher risk. However, these cases represent a fraction of the new primary brain tumours diagnosed each year

Research is still being conducted into the long-term impact of heavy mobile-phone usage.

6.8.4. Insurance Industry Definitions

Benign Brain Tumour has an ABI standard definition since the first Statement of best practice. The standard definitions have been:

Benign Brain Tumour – resulting in permanent symptoms (1999/2002)

A non-malignant tumour in the brain resulting in permanent deficit to the neurological system. Tumours or lesions in the pituitary gland are not covered.

Benign Brain Tumour – resulting in permanent symptoms (2006/2011)

A non-malignant tumour or cyst in the brain, cranial nerves or meninges within the skull, resulting in permanent neurological deficit with persisting clinical symptoms.

For the above definition, the following are not covered:

- Tumours in the pituitary gland.
- Angiomas.

“Permanent neurological deficit with persisting clinical symptoms” is a generic term within the SoBP. The insurance industry has evolved this definition in a number of ways in order to gain “ABI+” status. For example the following extends cover for cases where there has been surgical removal even if there is no residual neurological deficit.

Benign Brain Tumour – resulting in permanent symptoms

A non-malignant tumour or cyst in the brain, cranial nerves or meninges within the skull, resulting in **either surgical removal or** permanent neurological deficit with persisting clinical symptoms.

For the above definition, the following are not covered:

- Tumours in the pituitary gland.
- Angiomas

Other definitions in the Irish market have further extended cover to include radiotherapy treatments.

Although pituitary tumours are medically recognised as brain tumours, they are generally excluded form cover and have been since BBT was first introduced as a critical illness condition. The reasons being that these tumours usually have a very good prognosis and can often be more easily treated with drugs that suppress tumour growth. In addition, they also have a very good prognosis when surgically treated. Recently several offices have added "pituitary tumour" as a specific additional condition, usually as additional benefit.

Angiomas also excluded as these are not really tumours where there is new cellular growth; rather they are congenital collections of abnormal blood vessels.

6.8.5. Derived Incidence Rates

Benign Brain Tumour was considered in CIBT02. However, it should be noted that we have extended the list of ICD codes so that the HES counts are not 2.5 times higher than those included in the CIBT02 tables.

In particular we have included D32 - benign neoplasm of meninges. It is generally viewed that this is also accepted for claims purposes as Benign Brain Tumours and, as already mentioned, "meningioma" is the most common benign brain tumour. Inclusion of D32 increases the expected rate by between 75% and 100% compared with the Exploring the Critical Path figures.

Other inclusions which increase rates by a further 33% against codes D32, D33, D42 and D43 are:

- Cerebral cysts - while these would not be considered to be benign tumours in a clinical context, the insurance definition specifically refers to cysts;
- Syringomyelia and syringobulbia;
- Neurofibromatosis (non-malignant).

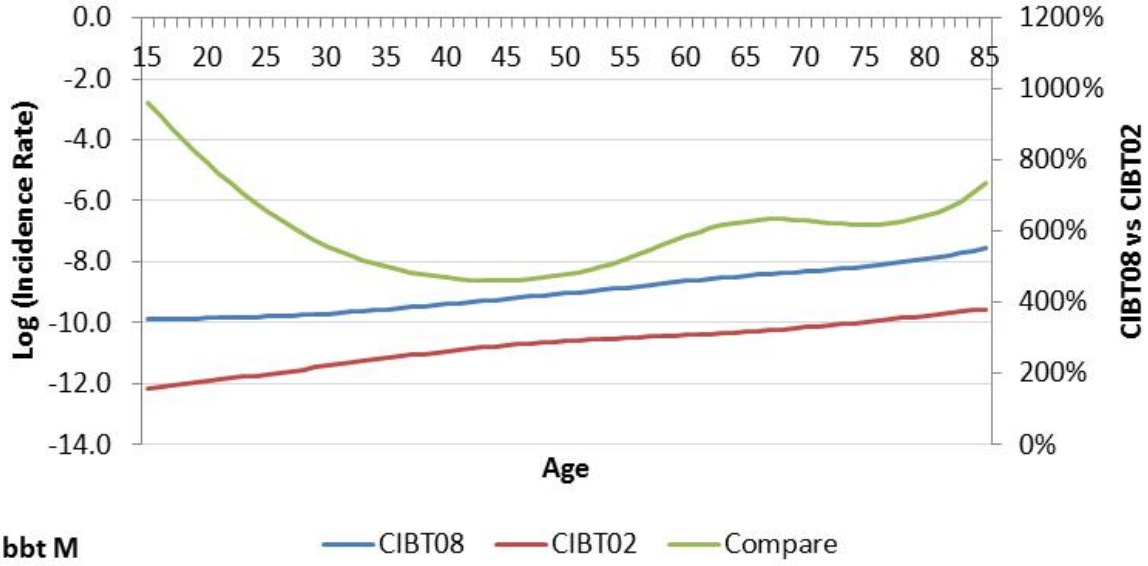
6.8.5.1. Male Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.74	1.42	2.93
Adjustment for Overlap	-15.5%	-21.5%	-39.8%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.63	1.18	2.37
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.63	1.18	2.36
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.2%	0.2%	0.2%
Addition for Accelerated Rates Ix - kxqx	0.61	1.11	1.95

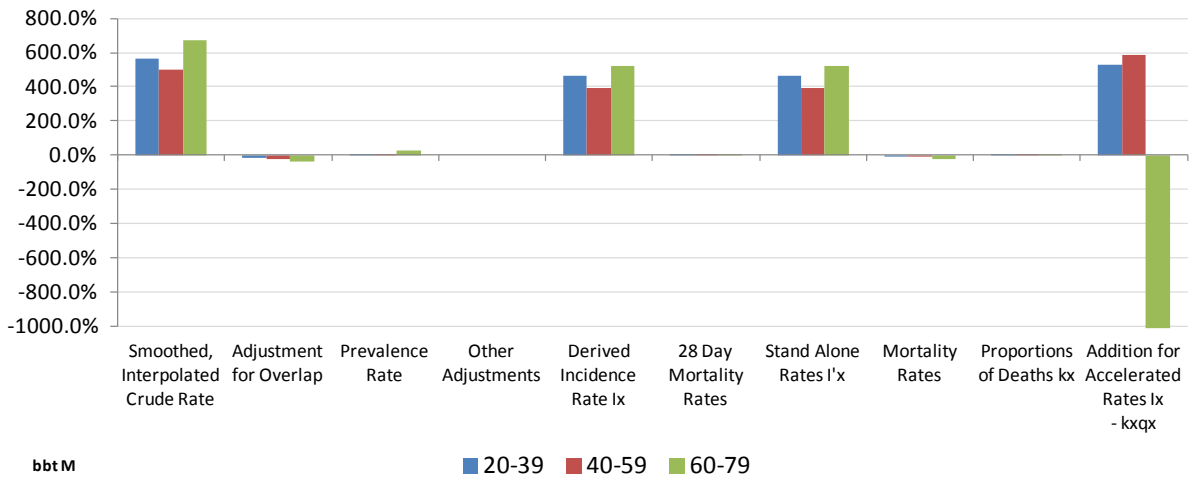
bbt _ M

The standalone incidence rates for Cancer are shown in the chart below for both the newly derived rates (CIBT08) and those from CIBT02. The green line, with values on the 2nd axis, displays CIBT08 as a percentage of CIBT02.



While 250% of the increase is explained by the change in ICD codes included, there is still a large residual change.

The following chart illustrates the movement of each of these components relative to CIBT02, for the same 3 age bands. Please note that the negative seen in the band "60-79" for the Addition for Accelerated rates is a consequence of this being negative in CIBT02. Because of this the chart below has had the range on the y-axis restricted – the final green bar extends down to -54000%!

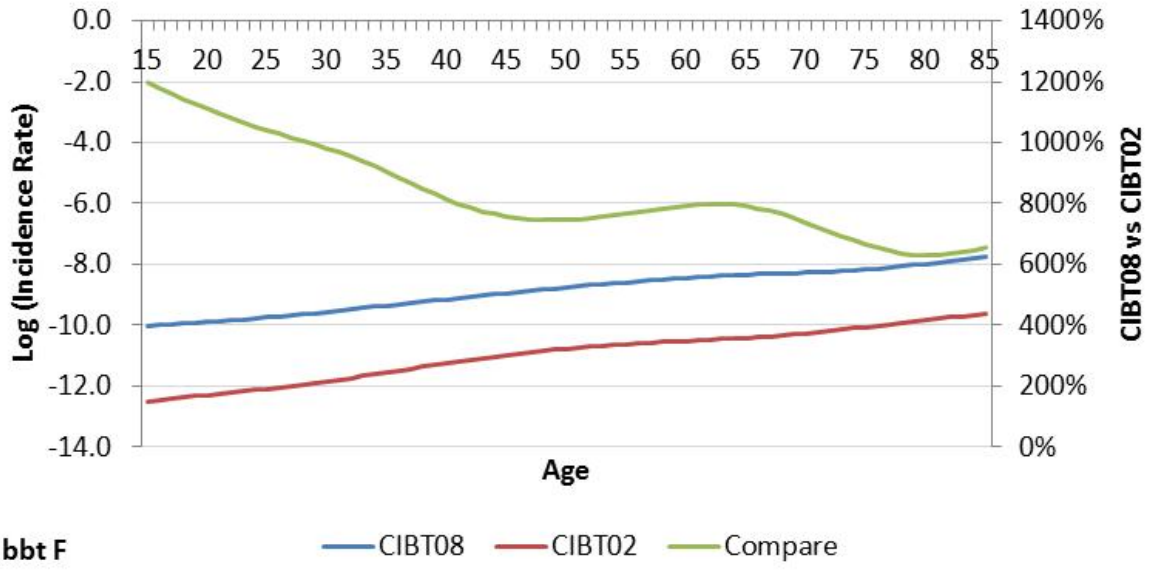


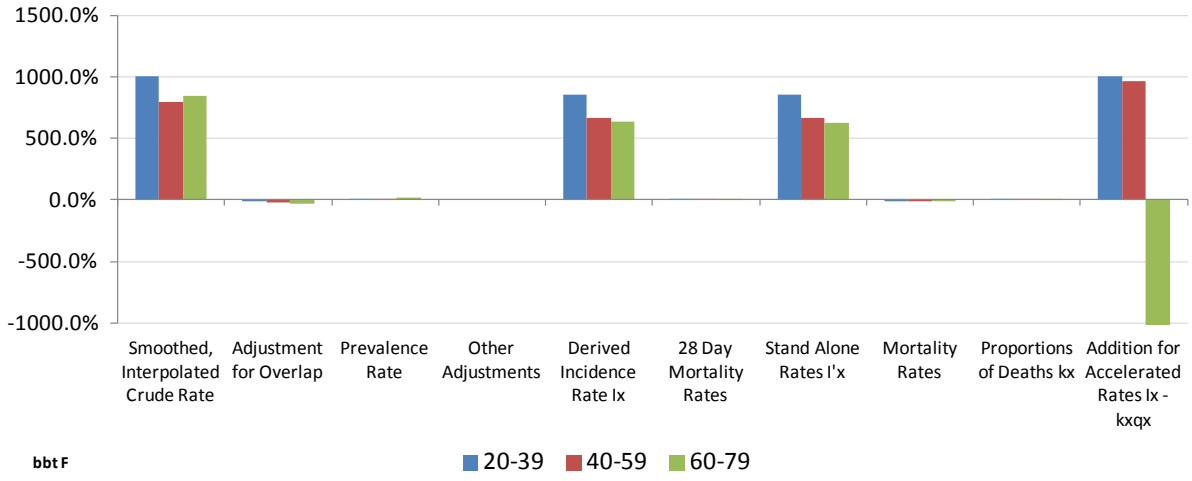
6.8.5.2. Female Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.84	1.79	3.30
Adjustment for Overlap	-14.3%	-18.8%	-35.8%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.72	1.53	2.55
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.72	1.53	2.54
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.4%	0.3%	0.2%
Addition for Accelerated Rates Ix - kxqx	0.71	1.46	2.18

bbt _ F



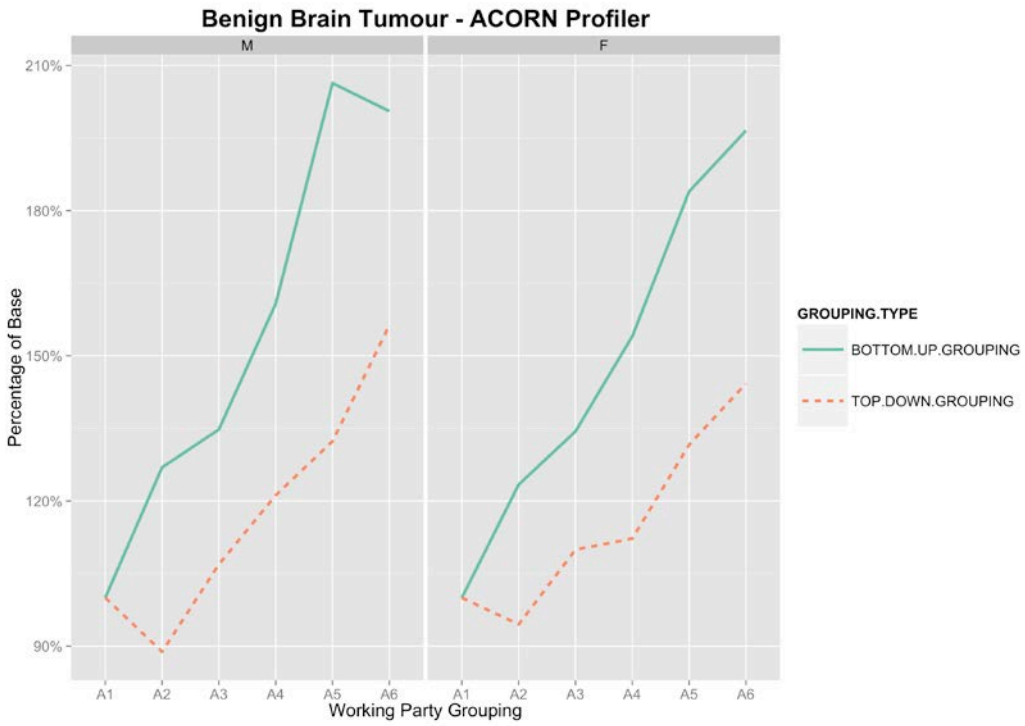


As per the males, the range on the y-axis has been restricted.

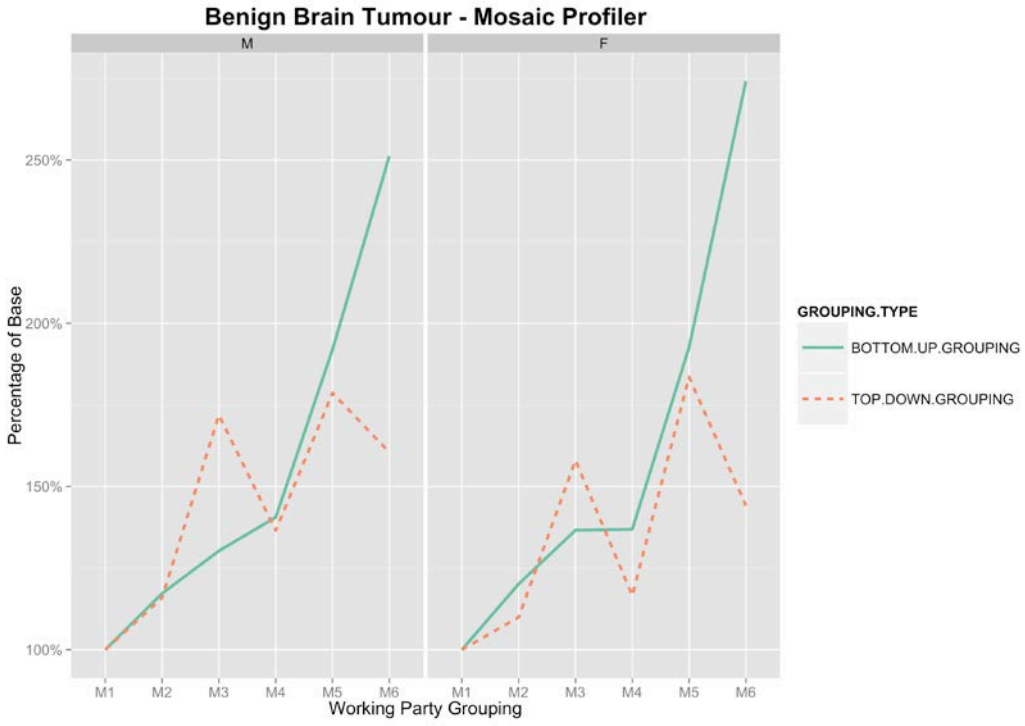
Comments are as per the males.

6.8.6. Geodemographic Analysis

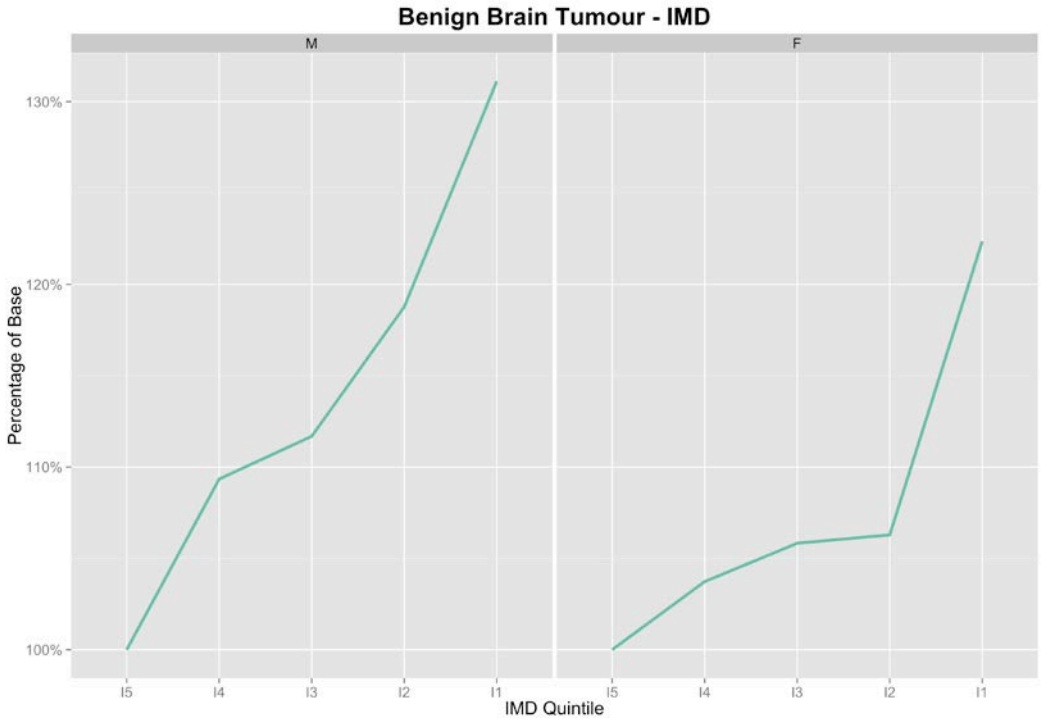
6.8.6.1. Acorn



6.8.6.2. Mosaic



6.8.6.3. Index of Multiple Deprivation



6.9. Blindness

6.9.1. What is it?

Blindness is defined as the loss of sight or in the state of being sightless or to the extent of vision impairment. A blind individual is unable to see. It denotes the condition of total blackness of vision with the inability of a person to distinguish darkness from bright light in either eye.

In the UK, people can register as being “blind” or suffering with “severe visual loss” where visual acuity is measured as being 3/60 on a Snellen eye chart, or 6/60 if there is in addition loss of visual fields. This entitles them to receive specific state benefits.

Up until 2006, the standard ABI definition used basic criteria of being “completely blind” though claims practices usually reflected and applied the criteria used to assess state benefits. However, in 2006, the ABI model wording was changed to align the criteria with the state benefits criteria and claims practices.

In addition, social benefits can be obtained for less severe losses of vision that are categorised as “partially sighted” with specific criteria relating to visual acuity and field loss.

Numbers of people in the UK that are registered as having a degree of significant visual impairment are as follows:

- 299,000 in England (NHS Information Centre, 2011);
- 34,500 in Scotland (Scottish Government, 2010);
- 16,000 in Wales (Welsh Assembly Government, 2010);
- 8,000 in Northern Ireland (Royal National Institute for the Blind- estimate).

Half of those on the register are blind, and half are partially sighted.

Incidence increases rapidly with age with 1 in 5 people aged 75 living with sight loss.

6.9.2. Symptoms and Treatment

Common symptom is difficulty in seeing. People with similar levels of visual loss may have very different responses to that symptom. If one is born blind, there is much less adjustment to a non-seeing world than there is for people who lose their vision late in life, where there may be limited ability to cope with that visual loss. Support systems available to individuals and their psychological make-up will also modify the symptom of lack of sight. People who lose their vision suddenly, rather than over a period of years, also can have more problems from their visual loss.

Associated symptoms, such a discomfort in the eyes, awareness of the eyes, foreign body sensation, and pain in the eyes or discharge from the eyes may be present or absent, depending on the underlying cause of the blindness.

A blind person may have no visible signs of any abnormalities when sitting in a chair and resting. If blindness is a result of infection of the cornea (the dome in front of the eye), the transparent cornea may become white. This opaque cornea can make it difficult to view the coloured part of the eye. In blindness from cataract, the normally black pupil may appear white. Depending on the degree of blindness, the affected individual will exhibit signs of visual loss when attempting to ambulate. Some blind people have learned to look directly at a source of conversation.

There is no cure or treatment for permanent and/or irreversible blindness. The treatment of some loss of vision or poor sight depends on the cause. In third-world nations where there are many people who have poor vision as a result of a refractive error, merely prescribing

and giving glasses will alleviate the problem. Nutritional causes of blindness can be addressed by dietary changes. There are hundreds of thousands of people who are blind from cataracts. In these patients, cataract surgery would, in most cases, restore their sight. Inflammatory and infectious causes of blindness can be treated with medication in the form of drops or pills.

6.9.3. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

> **Blindness (1999/2002)**
>
> Total permanent and irreversible loss of all sight in both eyes.

> **Blindness – *permanent and irreversible* (2006/2011)**
>
> Permanent and irreversible loss of sight to the extent that even when tested with the use of visual aids, vision is measured at 3/60 or worse in the better eye using a Snellen eye chart.

Some companies have extended the definition to offer ABI+ by including claims criteria that make reference to visual field loss in addition to visual acuity. This in effect makes the wording very close to that used to pay social benefits.

6.9.4. Derived Incidence Rates

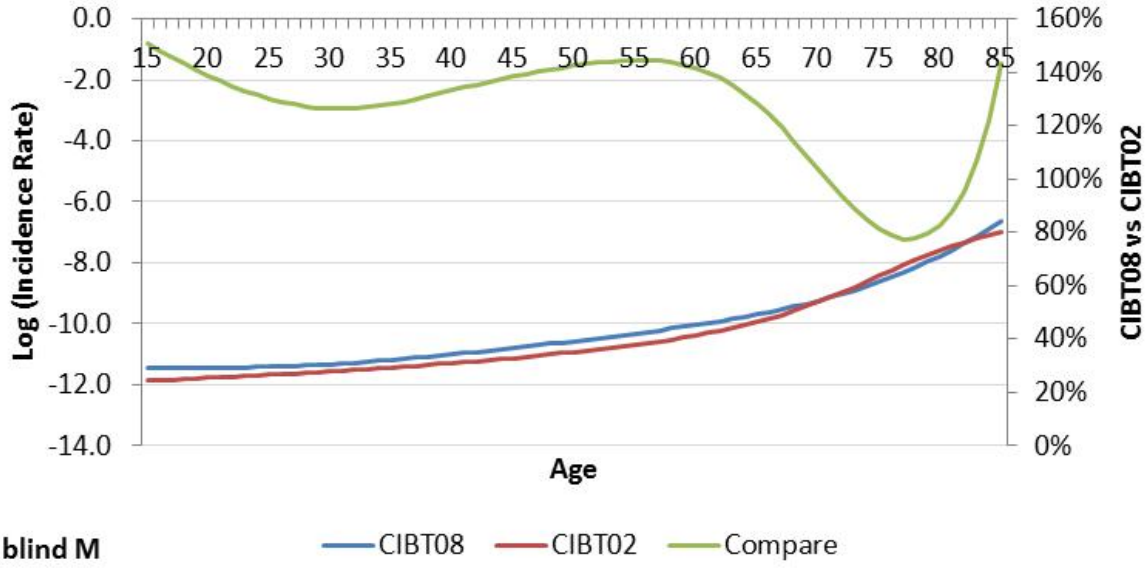
6.9.4.1. Male Results

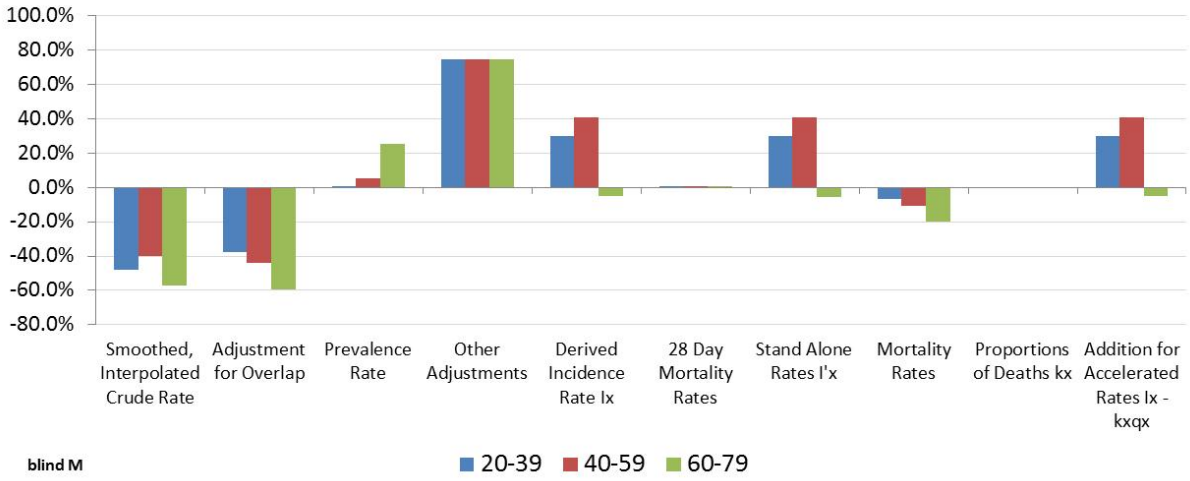
The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.20	0.44	1.90
Adjustment for Overlap	-37.9%	-44.2%	-59.5%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.13	0.26	1.06
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.13	0.26	1.05
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.13	0.26	1.06

blind _ M

The graph below shows a smooth progression of rates by age:

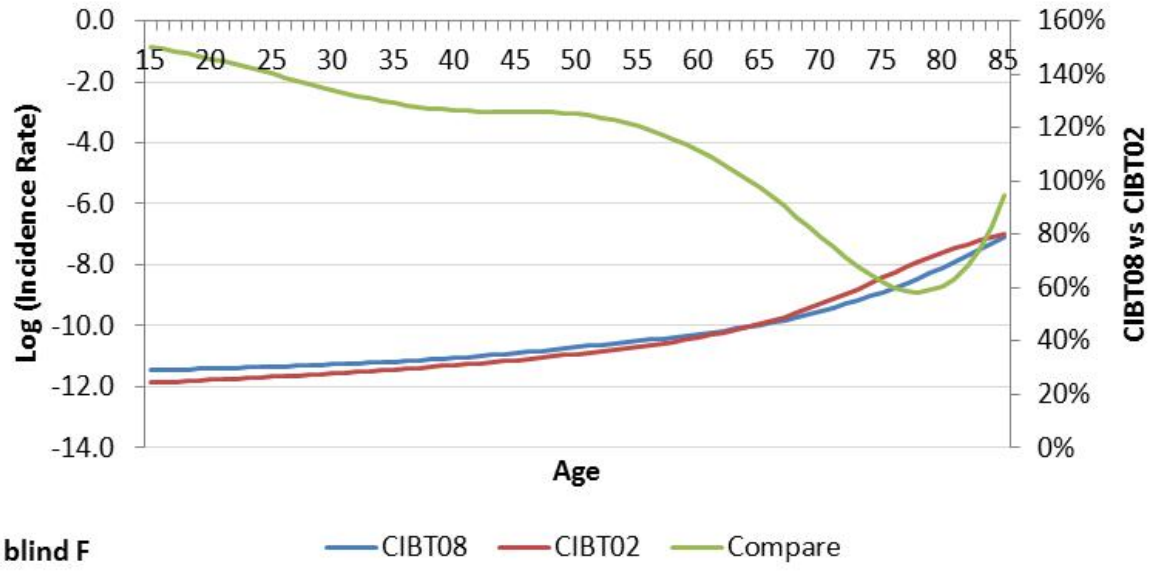


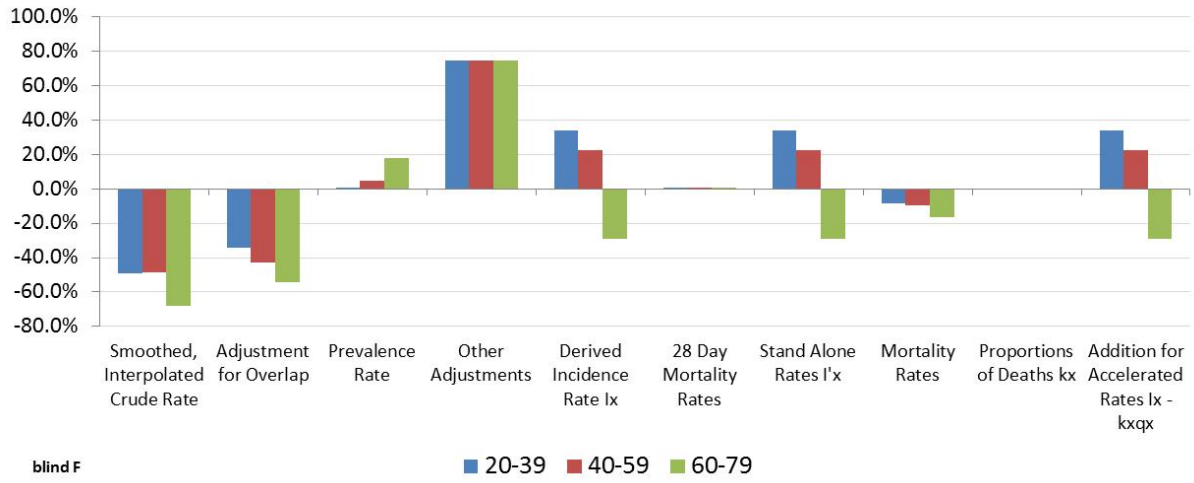


6.9.4.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.20	0.38	1.52
Adjustment for Overlap	-34.0%	-43.1%	-54.1%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.13	0.22	0.84
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.13	0.22	0.84
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.13	0.22	0.84

blind _ F




100.0%
80.0%
60.0%
40.0%
20.0%
0.0%
-20.0%
-40.0%
-60.0%
-80.0%
Smoothed, Interpolated Crude Rate
Adjustment for Overlap
Prevalence Rate
Other Adjustments
Derived Incidence Rate Ix
28 Day Mortality Rates
Stand Alone Rates l'x
Mortality Rates
Proportions of Deaths kx
Addition for Accelerated Rates Ix - kxqx
20-39
40-59
60-79

blind F

6.10. Cardiomyopathy

6.10.1. What is it?

Cardiomyopathy is a weakening of the heart muscle or another problem with the heart muscle. It often occurs when the heart cannot pump as well as it should, or with other heart function problems. The four major types are: dilated cardiomyopathy, hypertrophic cardiomyopathy, ischemic cardiomyopathy and restrictive cardiomyopathy.

- **Dilated cardiomyopathy** is a condition in which the heart becomes weak and the chambers get large resulting in the heart unable to pump enough blood out to the body.
- **Hypertrophic cardiomyopathy** is a condition in which the heart muscle becomes thick which makes it harder for blood to leave the heart.
- **Ischemic cardiomyopathy** is caused by narrowing in the arteries that supply the heart with blood, making the heart walls thin so they do not pump well.
- **Restrictive cardiomyopathy** is a group of disorders. The heart chambers are unable to properly fill with blood because the heart muscle is stiff.

6.10.2. Symptoms and Treatment

Symptoms of heart failure are most common. Usually, they develop slowly over time. However, sometimes symptoms start very suddenly and are severe. The common symptoms are: chest pain, cough, shortness of breath, loss of appetite, swelling of feet and ankles, swelling of the abdomen, palpitations, fatigue, weakness and faintness.

The main goal of treatment is to control symptoms and improve quality of life.

The following treatments may be used to control symptoms or prevent problems:

- Blood thinning medications to reduce the risk of blood clots;
- Chemotherapy (in some situations);
- Diuretics to remove fluid and help improve breathing;
- Medications to prevent or control uneven or abnormal heart rhythms;
- Steroids for some causes;
- A heart transplant may be considered if the heart function is very poor and the patient has severe symptoms;
- Self-monitoring of symptoms, heart rate and blood pressure by the patients;
- Change in diet including managing alcohol and salt intake;
- A permanent pacemaker to control the heartbeat;
- An implanted defibrillator that recognizes life-threatening heart rhythms and sends an electrical pulse to stop them;
- Heart bypass surgery or angioplasty to improve blood flow to the damaged or weakened heart muscle.

6.10.3. Risk Factors

The risk factors vary depending on the type of cardiomyopathy, however in general they include:

- Alcohol or drug abuse, or medicines that can be toxic to the heart;
- Autoimmune illnesses which weaken the immune system;
- Genetic factors;
- Heart valves that are either too narrow or too leaky (regurgitant);
- Infections that involve the heart muscle;
- Amyloidosis and scarring of the heart from an unknown cause;
- Diseases of the heart lining (endocardium);

  Iron overload (hemochromatosis).

6.10.4. Insurance Industry Definitions

Cardiomyopathy does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market. An example of the definition used is as follows:

Cardiomyopathy – of specified severity

A definite diagnosis of cardiomyopathy by a Consultant Cardiologist that has resulted in permanent damage to the heart muscle and function resulting in both of the following:

- a reduced ejection fraction of 35%
- impairment to the degree of class 3 New York Heart Association classification of cardiac impairment*

For the above definition, the following aren't covered:

- Cardiomyopathy directly related to alcohol or drug abuse.
- All other forms of heart disease, heart enlargement and myocarditis.

*New York Heart Association Class 3 – heart disease resulting in marked limitation of physical activities where less than ordinary activity causes fatigue, palpitation, breathlessness or chest pain.

The only significant variation from the example shown is that some companies do not include reference to "ejection fraction" in their definitions.

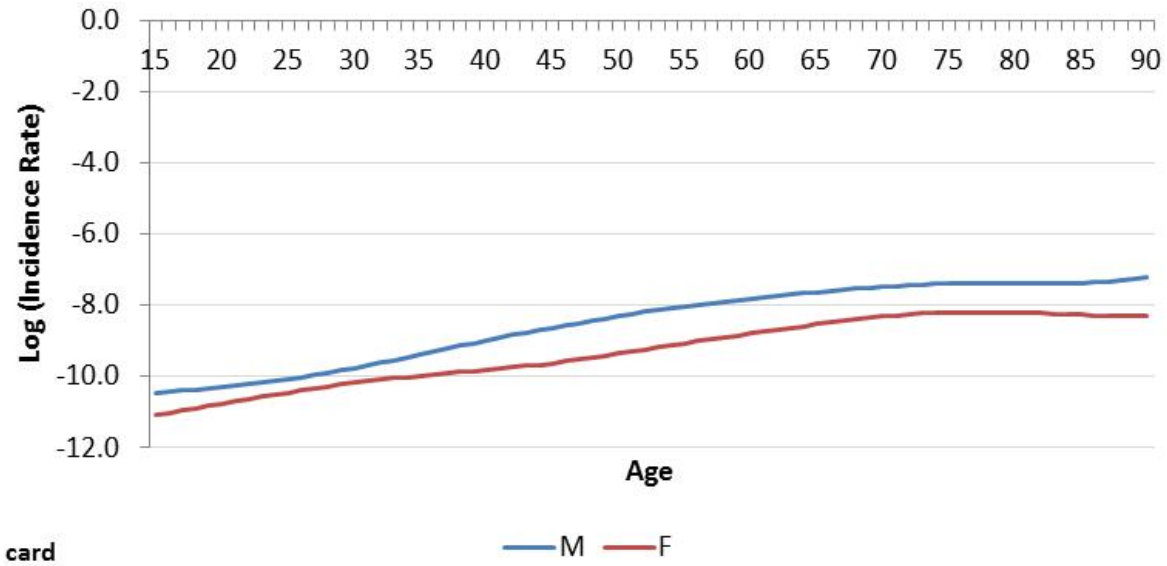
6.10.5. Derived Incidence Rates

The HES data is potentially not a good match for the insurance definition because in many cases the patient will not fulfil the insured criteria.

Cardiomyopathy rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.75	3.10	8.26	0.44	1.18	3.37
Adjustment for Overlap	-16.1%	-25.8%	-51.3%	-14.9%	-28.6%	-44.9%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.64	2.39	5.25	0.37	0.87	2.23
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.64	2.39	5.24	0.37	0.87	2.23
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	1.2%	1.3%	0.6%	1.0%	0.6%	0.3%
Addition for Accelerated Rates Ix - kxqx	0.52	1.97	4.15	0.33	0.75	1.85

card



As expected, the incidence rate of Cardiomyopathy increase materially with age in a broadly log-linear fashion. Incidence is much higher for males in common with other cardio-vascular conditions such as heart attack.

6.11. Coma

6.11.1. What is it?

A coma is a state of unconsciousness lasting more than six hours, in which a person cannot be awakened, fails to respond normally to painful stimuli, light, or sound, lacks a normal sleep-wake cycle and does not initiate voluntary actions. A person in a state of coma is described as being comatose.

Comas are caused by an injury to the brain. Brain injury can be due to increased pressure, bleeding, loss of oxygen, or build-up of toxins. The injury can be temporary and reversible. It also can be permanent.

More than 50% of comas are related to head trauma or disturbances in the brain's circulatory system. Repeated seizures can prevent the brain from recovering in between seizures. This will cause prolonged unconsciousness and coma.

There is a universal method of measuring the neurological state of a person suffering from coma known as the **Glasgow Coma Scale** (**GCS).** This is a neurological scale that aims to give a reliable, objective way of recording the conscious state of a person for initial as well as subsequent assessment. The degree of coma is assessed against the criteria of the scale, and the resulting points give a patient score between 3 (indicating deep unconsciousness) and either 14 (original scale) or 15 (the more widely used modified or revised scale).

6.11.2. Symptoms and Treatment

Coma may have developed in humans as a response to injury to allow the body to pause bodily actions and heal the most immediate injuries - if at all - before waking. It therefore could be a compensatory state in which the body's expenditure of energy is not superfluous.

Treatment for a coma depends on the cause. Prompt medical attention is vital to treat potentially reversible conditions. For example, if there is an infection that's affecting the brain, antibiotics may be needed. Glucose may be required in the event of a diabetic shock. Surgery may also be necessary to relieve the pressure on the brain due to swelling or to remove a tumour.

Certain drugs may also help relieve the swelling. Medication may also be given to stop seizures if necessary.

In general, treatment for a coma is supportive. People in comas are looked after in an intensive care unit (ICU) and may often require full life support until their situation improves.

6.11.3. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Coma – resulting in permanent symptoms (1999/2002)

A state of unconsciousness, with no reaction to external stimuli or internal needs persisting continuously with the use of life support systems for a period of at least 96 hours and resulting in permanent neurological deficit. Coma secondary to alcohol and drug misuse is not covered.

Coma – resulting in permanent symptoms (2006)

A state of unconsciousness, which:

- requires the use of life support systems,
- results in permanent neurological deficit with persisting clinical symptoms.

For the above definition, the following is not covered:

- Coma secondary to alcohol or drug abuse.

Coma – resulting in permanent symptoms (2011)

A state of unconsciousness resulting in no reaction to external stimuli or internal needs which

- requires the use of life support systems for a continuous period of at least 96 hours and
- results in permanent neurological deficit with persisting clinical symptoms.

For the above definition, the following is not covered:

- Coma secondary to alcohol or drug abuse.

The "ABI+" definition removes the requirement for the use of life support systems for 'a continuous period of at least 96 hours'. The ABI+ definition thus requires no minimum length of time in a coma on life support and is as follows:

Coma – resulting in permanent symptoms (2011/ABI+)

A state of unconsciousness resulting in no reaction to external stimuli or internal needs which

- requires the use of life support systems and
- results in permanent neurological deficit with persisting clinical symptoms.

For the above definition, the following is not covered:

- Coma secondary to alcohol or drug abuse

6.11.4. Derived Incidence Rates

Exploring the Critical Path set the Coma rates to zero, after consideration of overlaps with other CIs.

For our analysis we have used ICD codes in combination with OPCS codes to require life support. This has resulted in very low incidence rates which effectively translate into a zero incidence rate. This is demonstrated in the tables that follow. Rates are per 10,000 population.

6.11.4.1. Male Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.01	0.02	0.03
Adjustment for Overlap	-8.0%	-39.6%	-67.0%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.01	0.01	0.01
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.01	0.01	0.01
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.01	0.00	-0.02

coma _ M

6.11.4.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.01	0.02	0.03
Adjustment for Overlap	-23.1%	-40.0%	-42.0%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.01	0.01	0.02
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.01	0.01	0.02
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.1%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.00	0.00	-0.02

coma _ F

As such in accordance with CIBT02 we propose a zero rate for coma.

In future we recommend an approach whereby HES data is used without the life support requirement through OPCS with a corresponding adjustment made severity using other data sources.

6.12. Coronary Angioplasty

6.12.1. What is it?

A coronary angioplasty (stent) is a procedure used to widen blocked or narrowed coronary arteries. A short wire-mesh tube, called a stent, is inserted into an artery to allow blood to flow more freely through it. Coronary angioplasty is sometimes known as percutaneous transluminal coronary angioplasty (PTCA) or percutaneous coronary intervention (PCI).

Like all organs in the body, the heart needs a constant supply of blood. This is supplied by two large blood vessels called the left and right coronary arteries. In older people, these arteries can become narrowed and hardened. This is known as atherosclerosis. Hardening of the coronary arteries can restrict the flow of blood to the heart, which can lead to angina.

6.12.2. Symptoms and Treatment

The most common symptom of angina is chest pain, which is usually triggered by physical activity. While many cases of angina can be treated with medication, a coronary angioplasty may be required to restore the blood supply to the heart in severe angina.

A coronary angioplasty is one of the most common types of treatment for the heart. In 2012 there were over 90,000 coronary angioplasties performed in the UK. Coronary angioplasties are most commonly performed in people who are 65 years of age or older as they are more likely to have angina.

A coronary angioplasty does not involve making major incisions in the body and is usually carried out safely in most people. Doctors refer to this as a minimally invasive form of treatment.

Coronary angioplasties are also often used as an emergency treatment after a heart attack.

6.12.3. Risk Factors

Important risk factors for coronary heart disease are:

- High blood pressure;
- High blood cholesterol;
- Diabetes;
- Smoking;
- Being overweight;
- Being physically inactive;
- Having a family history of early heart disease;
- Age (55 or older for women);
- Unhealthy diet;
- Stress.

The risk of coronary heart disease and heart attack increases with the number of risk factors and their severity. Also, some risk factors put one at greater risk of CHD and heart attack than others. Examples of these risk factors include smoking and diabetes.

Many coronary heart disease risk factors start during childhood. This is even more common now because many children are overweight and do not get enough physical activity. Some risk factors can even develop within the first 10 years of life.

Risk factors such as age and family history of early heart disease cannot be changed. For women, age becomes a risk factor at 55. After menopause, women are at greater risk of heart disease, in part because their body's production of oestrogen drops. Women who have gone through early menopause, either naturally or because they have had a hysterectomy, are twice as likely to develop heart disease as women of the same age who have not yet gone through menopause. Another reason for the increasing risk is that middle age is a time when women tend to develop risk factors for heart disease. Family history of early heart disease is another risk factor that cannot be changed. One is more likely to get heart disease if close family members such as parents have the disease.

6.12.4. Insurance Industry Definitions

Coronary angioplasty does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market However, some insurers offer cover which is usually a partial/additional benefit.

Two examples of the current definitions used by the insurers are set out below:

Coronary angioplasty – *of 2 or more coronary arteries*

Undergoing on 2 or more coronary arteries: balloon angioplasty; atherectomy; rotablation; laser treatment; or the application of stents; for coronary artery disease. There must be angiographic or equivalent evidence of the underlying disease which shows there is stenosis of at least 50% narrowing of 2 or more coronary arteries. The disease must be considered uncontrollable by non-invasive medical therapy.

Coronary angioplasty – *of 2 or more coronary arteries*

We will pay the lower of 25 percent of the benefit and £25,000 if a member undergoes any of the following:

- balloon angioplasty
- atherectomy
- rotablation
- laser treatment and / or
- insertion of stents

The above operations must have been carried out on the advice of a consultant cardiologist to treat severe coronary artery disease in two or more main coronary arteries. The above operation must be to treat at least 70 percent diameter narrowing. If an operative procedure is only performed on one main coronary artery there must be at least 70 percent diameter narrowing in another main coronary artery.

For the purposes of this definition main coronary arteries are described as one or more of the following:

- right coronary artery
- left main stem
- left anterior descending
- circumflex

The following is not covered: procedures to any branches of any of the main coronary arteries.

6.12.5. Derived Incidence Rates

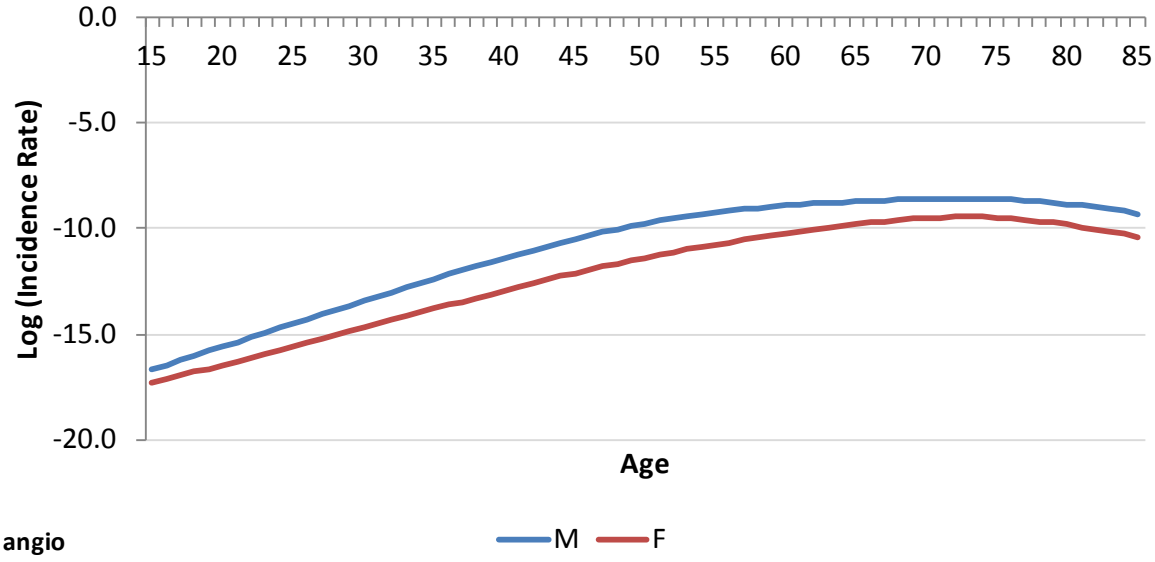
6.12.5.1. Single Vessel Angioplasty

Single Vessel Angioplasty rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2.

Rates shown below are per 10,000.

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.10	1.43	4.19	0.03	0.35	1.50
Adjustment for Overlap	-80.8%	-64.4%	-69.8%	-78.2%	-68.0%	-66.7%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.03	0.57	1.66	0.01	0.12	0.61
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.03	0.57	1.66	0.01	0.12	0.61
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.03	0.57	1.66	0.01	0.12	0.61

angio



In common with other cardiovascular diseases, male rates are materially higher then female rates. We also observe the expected flattening by age from around age 60-70 indicating that less angioplasties are carried possible because by-pass grafts may be more effective.

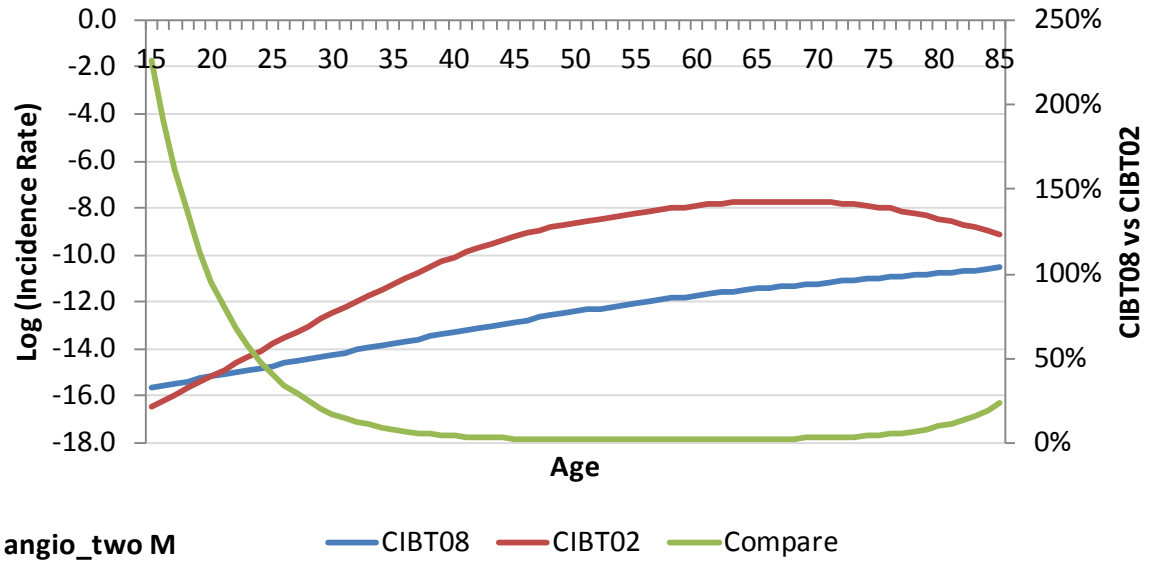
6.12.5.2. Two or more Coronary Arteries

Our dataset does enable us to analyse the incidence of two or more coronary arteries. The results obtained here are low and consequently the graduation is somewhat spurious. We therefore set the rates to zero for CIBT08 and rely upon the single vessel angioplasty results discussed in Section 6.12.4.1.

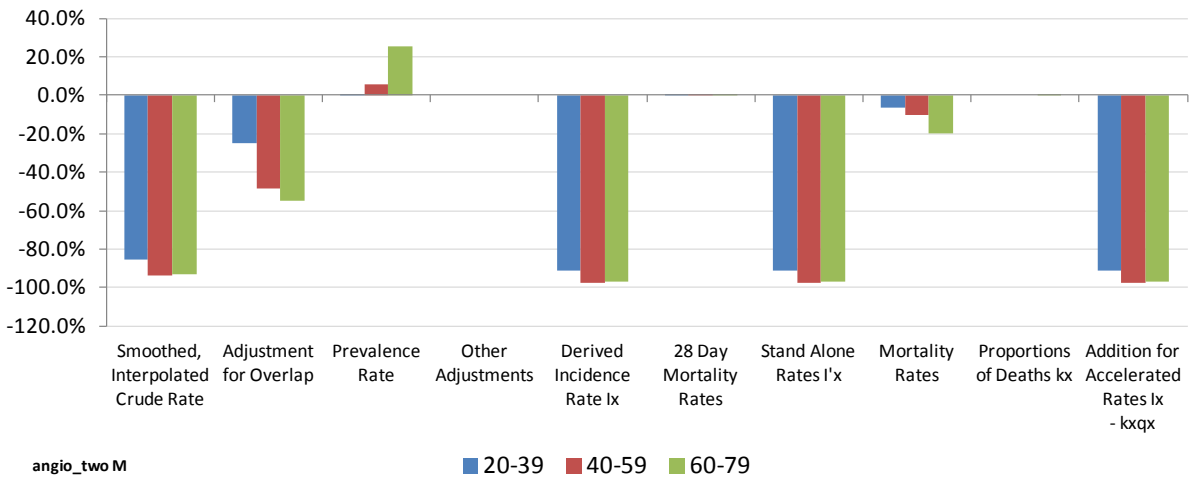
Rates are illustrated below for males. Similar comments apply to the female results, although these are not shown for the sake of brevity.

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.01	0.13	0.36
Adjustment for Overlap	-45.0%	-68.6%	-74.8%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.01	0.04	0.13
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.01	0.04	0.13
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.01	0.04	0.13

The standalone incidence rates for two-vessel angioplasty are shown in the chart below for both the newly derived rates (CIBT08) and those from CIBT02. The green line, with values on the 2nd axis, displays CIBT08 as a percentage of CIBT02.



As we can see our rates are significantly lower than those computed in CIBT02. The same result is seen for females. The following chart shows that this occurs due to a significant reduction in the crude rate and also in the allowance for overlaps at all ages.



6.13. Coronary Artery By-pass Grafts ("CABG")

6.13.1. What is it?

Coronary Artery Bypass Graft is for the treatment of coronary artery disease. Coronary artery disease is where one or more of the coronary arteries are partly or totally blocked and the heart does not get enough blood.

Coronary artery disease is different from person to person. CABG creates a new route for blood and oxygen to reach the heart.

6.13.2. Symptoms and Treatment

Symptoms may be very noticeable, but sometimes you can have the disease and not have any symptoms. This is especially true in the early stages of heart disease.

Chest pain or discomfort (angina) is the most common symptom. The pain varies from person to person and occurs with activity or emotion. Other symptoms include shortness of breath, fatigue and general weakness.

The treatment of this disease is a combination of medical treatment and lifestyle changes. Treatment depends on your symptoms and how severe the disease is.

The medical treatment will include one or more medicines to treat heart disease, blood pressure, diabetes or high cholesterol. In other cases, it requires operational procedures which varies depending on the condition and includes CABG and angioplasty.

Lifestyle recommendations includes blood pressure optimization, review of the lifestyle management and addresses diet, exercise and smoking cessation. This includes cardiac rehabilitation program recommended by the doctor.

6.13.3. Risk Factors

The risk factors are:

- High blood pressure;
- Atherosclerosis with the hardening of the arteries due to build up plaque;
- Weakening of the artery wall from smoking and other unhealthy lifestyle choices;
- Increasing age;
- Genetic factors;
- Lack of exercise and poor diet;
- Other health conditions such as diabetes.

6.13.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Coronary Artery By-pass Grafts (1999/2002)

The undergoing of open heart surgery on the advice of a Consultant Cardiologist to correct narrowing or blockage of one or more coronary arteries with by-pass grafts but excluding balloon angioplasty, laser relief or any other procedures.

Coronary Artery By-pass Grafts – *with surgery to divide the breastbone* (2006/2011)

The undergoing of surgery requiring median sternotomy (surgery to divide the breastbone) on the advice of a Consultant Cardiologist to correct narrowing or blockage of one or more coronary arteries with by-pass grafts.

Many companies enhance cover in order to gain ABI+ status by removing the requirement of “median sternotomy” so that other forms of less radical surgery are also covered.

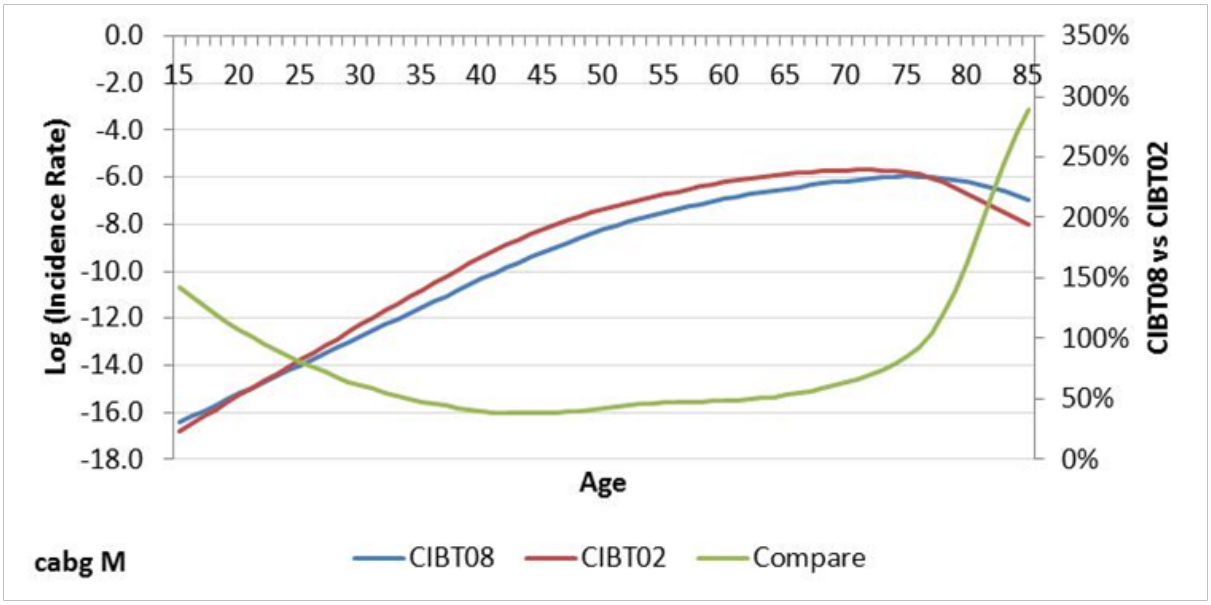
6.13.5. Derived Incidence Rates

6.13.5.1. Male Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

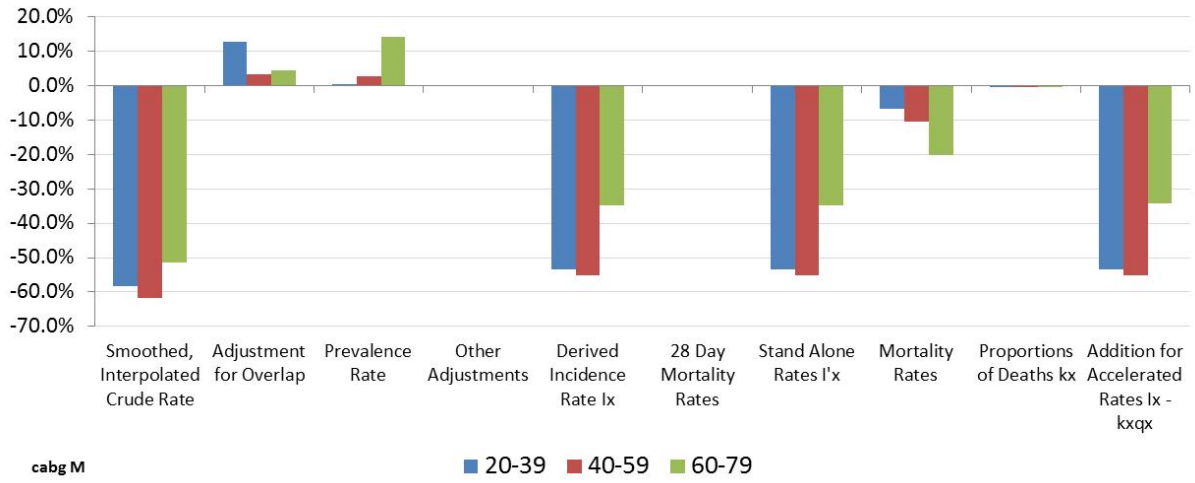
Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.13	5.89	29.10
Adjustment for Overlap	-45.2%	-54.7%	-53.6%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.06	3.02	18.47
28 Day Mortality Rates	-0.6%	-0.8%	-2.2%
Stand Alone Rates I'x	0.06	3.00	18.02
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.1%	0.1%
Addition for Accelerated Rates Ix - kxqx	0.06	3.00	18.16

cabg _ M



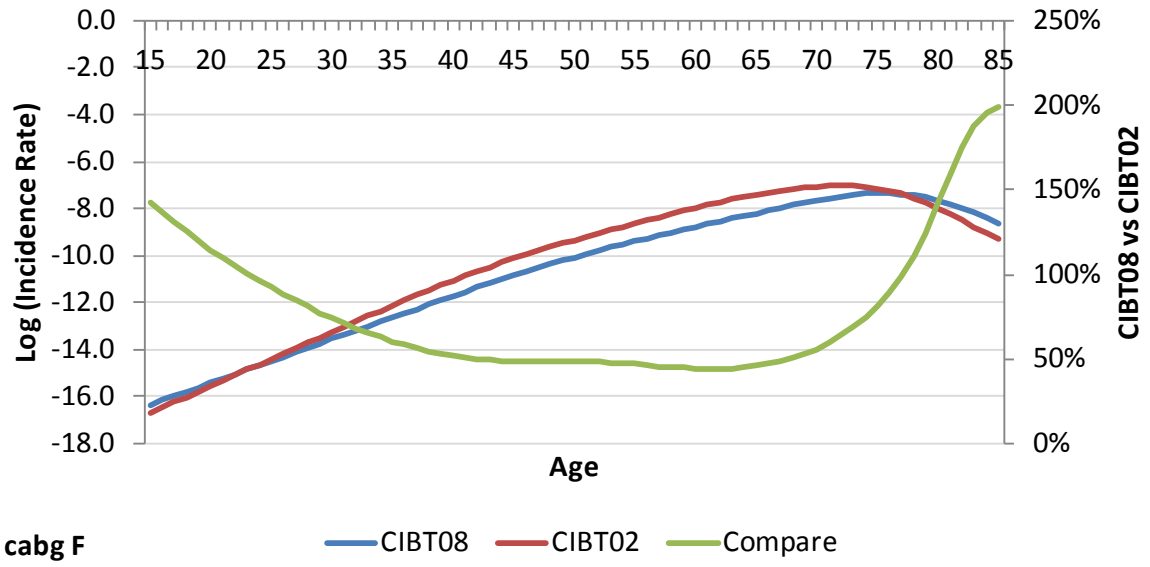
The CIBT08 rates profile is similar in shape to that of CIBT02 rates through most of the age range but increases substantially at the extreme ends of the age range.

The decrease in rates can be largely attributable to a significant decrease in the crude rates, although it is slightly offset by the increased adjustment for overlaps. The rates in the higher age bands have also been offset by increase in prevalence rates.

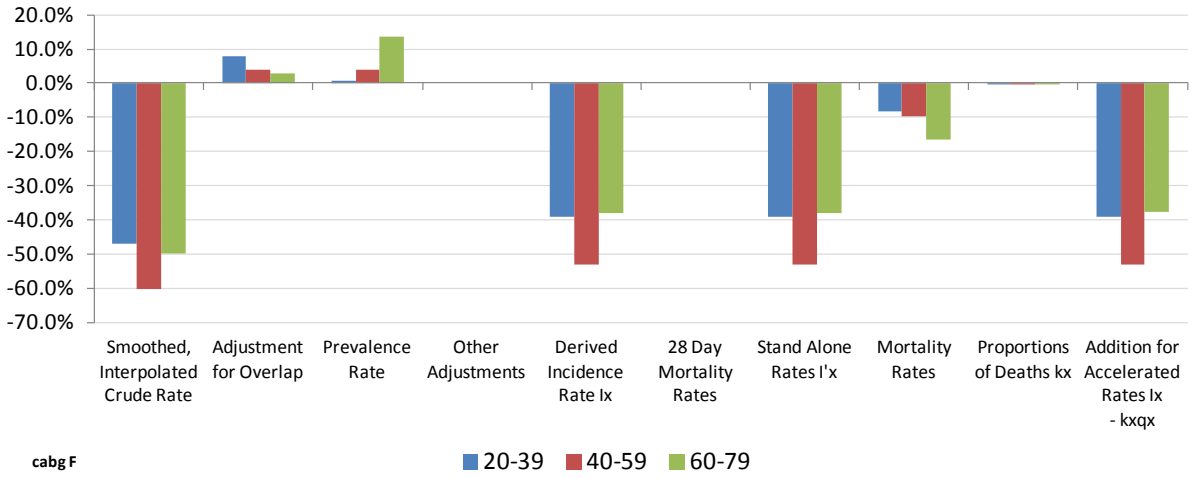


6.13.5.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.04	0.96	7.48
Adjustment for Overlap	-50.3%	-54.0%	-55.3%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.02	0.48	4.07
28 Day Mortality Rates	-0.6%	-0.8%	-2.3%
Stand Alone Rates I'x	0.02	0.48	3.96
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.1%
Addition for Accelerated Rates Ix - kxqx	0.02	0.48	3.98



The female results relative to CIBT02 are very similar to what was observed with the males.



We have compared our crude CABG rates to benchmark rates derived from The Epidemiology and Economics of Cardiothoracic Surgery in the Elderly (David A. Etzioni and Vaughn A. Starnes[20])

The benchmark rates have been apportioned into male and female across all age bands using the exposure and incidence rate profile used in our dataset. Results are shown below:

Incidence Rates	**M**			**F**		
	20-39	40-59	60-79	20-39	40-59	60-79
Derived Crude Rate	0.13	5.89	29.10	0.04	0.96	7.48
Benchmark	0.20	8.91	43.99	0.06	1.45	11.31
Ratio	65%	66%	66%	62%	66%	66%

The benchmark rates sourced are not comparable to the derived rates. It can be attributed to the variability in the population data, size and profile used in deriving the benchmark rates. It can also be due to the demography of the study group used that is not representative of the wider population.

[20] http://www.springer.com/cda/content/document/cda_downloaddocument/9781441908919-c1.pdf?SGWID=0-0-45-1140938-p174061371

6.14. Creutzfeldt-Jakob Disease (CJD)

6.14.1. What is it?

CJD is the most common human form of a group of rare, fatal brain disorders known as “prion diseases”, that is incurable and invariably fatal. CJD is at times called a human form of mad cow disease (bovine spongiform encephalopathy or BSE) even though classic CJD is not related to BSE; however, given that BSE is believed to be the cause of variant CJD (vCJD) disease in humans, the two are often confused.

Prion diseases occur when prion protein which is found throughout the body but whose normal function is not yet known, begins folding into an abnormal three-dimensional shape. This shape change gradually triggers prion protein in the brain to fold into the same abnormal shape.

Scientists do not yet understand the process of such folding but it is understood that mis-folded prion protein would destroy brain cells. The resulting damage leads to rapid decline in thinking and reasoning as well as involuntary muscle movements, confusion, difficulty, walking and mood changes.

CJD is rare, occurring in about one in 1 million people annually worldwide.

The disease causes a type of dementia that gets worse unusually fast. More common causes of dementia, such as Alzheimer's, dementia with Lewy bodies and frontotemporal dementia, typically progress more slowly.

Sporadic Creutzfeldt-Jakob disease has no known cause. Most scientists believe the disease begins when prion protein somewhere in the brain spontaneously misfolds, triggering a "domino effect" that misfolds prion protein throughout the brain. Genetic variation in the prion protein gene may affect risk of this spontaneous misfolding.

6.14.2. Symptoms and Treatment

Specific symptoms experienced by an individual and the order in which they appear can differ significantly. Some common symptoms include:

- Depression;
- Agitation, apathy and mood swings;
- Rapidly worsening confusion, disorientation, and problems with memory, thinking, planning and judgment;
- Difficulty walking;
- Muscle stiffness, twitches and involuntary jerky movements.

Typically, onset of symptoms occurs about age 60, and about 90% of patients die within 1 year. In the early stages of disease, people may have failing memory, behavioural changes, lack of coordination and visual disturbances. As the illness progresses, mental deterioration becomes pronounced and involuntary movements, blindness, weakness of extremities, and coma may occur.

There is currently no effective treatment available that can prevent the development of the condition.

6.14.3. Risk Factors

Most cases of Creutzfeldt-Jakob disease occur for unknown reasons, and no risk factors can be identified. However, a few factors seem to be associated with different kinds of CJD.

- Age - Sporadic CJD tends to develop later in life, usually around the age of 60. Onset of familial CJD occurs only slightly earlier. On the other hand, vCJD has affected people at a much younger age, usually in their late 20s.
- Genetics - People with familial CJD have a genetic mutation that causes the disease. The disease is inherited in an autosomal dominant fashion, which means only one copy of the mutated gene from either parent needed to develop the disease. If there is mutation, the chance of passing it on to the next generation is 50%. Genetic analysis in people with iatrogenic and variant CJD suggest that inheriting identical copies of certain variants of the prion gene may predispose a person to developing CJD if exposed to contaminated tissue.
- Exposure to contaminated tissue - People who have received human growth hormone derived from human pituitary glands or who have had dura mater grafts may be at risk of iatrogenic CJD. The risk of contracting vCJD from eating contaminated beef is difficult to determine. In general, if countries are effectively implementing public health measures, the risk is virtually non-existent.

6.14.4. Insurance Industry Definitions

CJD does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market. However, common examples of current definitions used by insurers are set out below. They vary from a straightforward diagnosis to inclusions of permanent clinical loss of ability criterion in the definitions.

Creutzfeldt-Jakob Disease (CJD)

Confirmation by a Consultant Physician of a definite diagnosis of Creutzfeldt-Jakob disease.

Creutzfeldt-Jakob Disease (CJD)

Confirmation by a Consultant Physician of a definite diagnosis of Creutzfeldt-Jakob disease resulting in permanent neurological deficit with persisting clinical symptoms.

Creutzfeldt-Jakob Disease (CJD)

A definite diagnosis of Creutzfeldt-Jakob disease by a Consultant Neurologist. There must be permanent clinical loss of the ability to do all of the following:

- remember;
- reason; and
- perceive, understand, express and give effect to ideas.

For the above definition, the following are not covered:
other types of dementia (these are covered under the dementia definition).

Creutzfeldt-Jakob Disease (CJD)

A definite diagnosis of Creutzfeldt-Jakob disease by a Consultant Neurologist. There must be permanent clinical impairment of motor function and loss of the ability to do all of the following:

- Remember
- reason, and
- perceive, understand, express and give effect to ideas

For the above definition, the following aren't covered: - Other types of dementia.

Creutzfeldt-Jakob Disease (CJD)

A definite diagnosis of Creutzfeldt-Jakob disease made by a consultant neurologist. There must be permanent clinical loss of the ability in mental and social functioning to the extent that permanent supervision or assistance by a third party is required.

6.14.5. Derived Incidence Rates

CJD rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000) although the rates are effectively zero. We therefore, do not spend any further time here.

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.00	0.02	0.05	0.01	0.00	0.00
Adjustment for Overlap	-0.4%	-19.8%	0.0%	-85.2%	-0.3%	-21.4%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.00	0.02	0.05	0.01	0.00	0.00
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.00	0.02	0.07	0.00	0.00	0.00
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.00	0.00	0.00	0.00	-0.01	-0.05

cjd

We believe that CJD is a genuinely rare condition and these rates confirm this. As such the results of the graduation are somewhat spurious. We therefore, set the rates to zero for CIBT08.

6.15. Deafness

6.15.1. What is it?

Deafness, hearing impairment, or hearing loss is a partial or total inability to hear. It is caused by many different factors, including but not limited to age, noise, illness, chemicals and physical trauma. There is a diagnosis to determine the severity of the hearing impairment, and it is measured in decibels. The severity of a hearing impairment is ranked according to the additional intensity above a nominal threshold that a sound must be before being detected by an individual; it is (measured in decibels of hearing loss, or dB HL). Hearing impairment may be ranked as mild, moderate, moderately severe, severe or profound as defined below:

- Mild:
    - for adults: between 26 and 40 dB HL
    - for children: between 20 and 40 dB HL[2]
- Moderate: between 41 and 54 dB HL[2]
- Moderately severe: between 55 and 70 dB HL[2]
- Severe: between 71 and 90 dB HL[2]
- Profound: 91 dB HL or greater[2]
- Totally Deaf: Have no hearing at all.

There are two main types of hearing loss, depending on where the problem lies:

- **sensorineural hearing loss –** caused by damage to the sensitive hair cells inside part of the inner ear called the cochlea or the auditory nerve; this occurs naturally with age or as a result of injury;
- **conductive hearing loss** – when sounds are unable to pass from the outer ear to the inner ear, often as the result of a blockage such as earwax, glue ear or a build-up of fluid due to an ear infection, a perforated ear drum or a disorder of the hearing bones.

It is also possible to have both these types of hearing loss. This is known as mixed hearing loss. Some people are born with hearing loss, but most cases develop with age.

6.15.2. Symptoms and Treatment

Early or general signs of hearing loss can include:

- difficulty hearing other people clearly and misunderstanding what they say;
- asking people to repeat themselves;
- listening to music or watching television with the volume turned up high;
- difficulty hearing the telephone or doorbell;
- regularly feeling tired or stressed, due to having to concentrate closely while listening.

Hearing loss can occur suddenly, but usually develops gradually.

The way hearing loss is treated depends on the cause and how severe it is.

In cases of sensorineural hearing loss, there are several options that may help to improve a

person's ability to hear and communicate. These include:

- **digital hearing aids;**
- **middle ear implants** – surgically implanted devices suitable for some people who are unable to use hearing aids;
- **cochlear implants** – small hearing devices that are surgically implanted inside the inner ear for people who find that hearing aids are not powerful enough;
- **lip reading or sign language** - such as British Sign Language (BSL).

Conductive hearing loss is sometimes temporary and can be treated with medication or minor surgery if necessary. However, more major surgery may be required to fix the ear drum or hearing bones. If conventional hearing aids do not work, there are also some implantable devices for this type of hearing loss, such as a Bone Anchored Hearing Aids (BAHAs).

6.15.3. Risk Factors

Age and loud noises are the most common causes of hearing loss.

Age: Age is the biggest single cause of hearing loss. Hearing loss that develops as a result of getting older is often known as age-related hearing loss or presbycusis.

Most people begin to lose a small amount of their hearing when they are 30 to 40 years old. This hearing loss increases as people get older. By the age of 80 most people will have significant hearing problems.

Loud noises: Another common cause of hearing loss is damage to the ear due to repeated exposure to loud noises over time. This is known as noise-induced hearing loss and it occurs when the sensitive hair cells inside the cochlea become damaged (known as sensorineural hearing loss).

Other types of sensorineural hearing loss

Sensorineural hearing loss occurs if the sensitive hair cells inside the cochlea are damaged, or as a result of damage to the auditory nerve (the nerve that transmits sound to brain). In some cases, both may be damaged.

Hearing loss caused by age and exposure to loud noises are both types of sensorineural hearing loss.

Sensorineural hearing loss can also be caused by:

- **genetic hearing loss** – some people may be born deaf or become deaf over time due to a genetic abnormality, although there is not always a family history;
- **viral infections of the inner ear**, such as mumps or measles;
- **viral infections of the auditory nerve**, such as mumps or rubella;
- **Ménière's disease** – where a person suffers with vertigo (spinning dizziness), hearing loss which can come and go, tinnitus and a feeling of a blockage in the ear;
- **acoustic neuroma** – a non-cancerous (benign) growth on or near the auditory nerve;
- **meningitis** – an infection of the protective membranes that surround the brain and spinal cord;
- **encephalitis** – inflammation of the brain;
- **multiple sclerosis** – a neurological condition affecting the central nervous system (the brain and spinal cord);

- **stroke** – where the blood supply to the brain is cut off or interrupted.

Some medications, such as certain chemotherapy medicines and certain antibiotics can also damage the cochlea and the auditory nerve, causing sensorineural hearing loss.

Sensorineural hearing loss is permanent and hearing aids are often required to improve hearing in these cases.

Conductive hearing loss

Conductive hearing loss occurs when sounds are unable to pass into the inner ear. This is usually due to a blockage, such as having too much ear wax, a build-up of fluid in the ear (glue ear), or an ear infection.

Conductive hearing loss can also be caused by:

- **a perforated eardrum** – where the eardrum is torn or has a hole in it;
- **otosclerosis** – an abnormal growth of bone in the middle ear which causes the inner hearing bone (the stapes) to be less mobile and less effective at transmitting sound;
- **damage** to the hearing bones from injury, a collapsed ear drum or conditions such as cholesteatoma (an abnormal collection of skin cells inside your ear).

Conductive hearing loss is usually temporary and it can often be treated with medication or minor surgery.

6.15.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Deafness – (1999/2002)

Total permanent and irreversible loss of all hearing in both ears.

Deafness – permanent and irreversible (2006/2011)

Permanent and irreversible loss of hearing to the extent that the loss is greater than 95 decibels across all frequencies in the better ear using a pure tone audiogram.

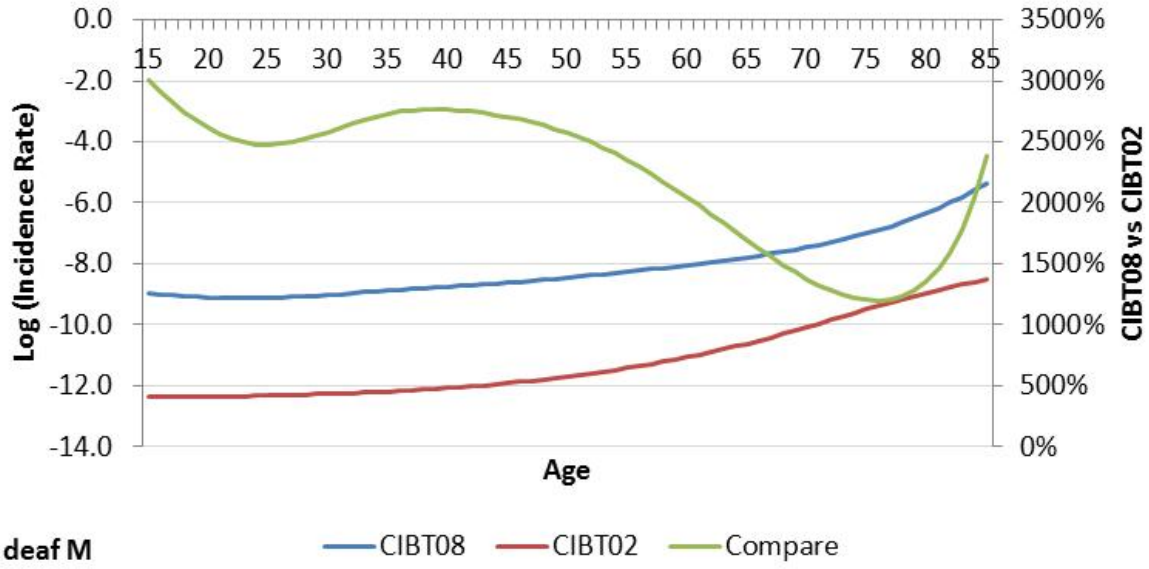
6.15.5. Derived Incidence Rates

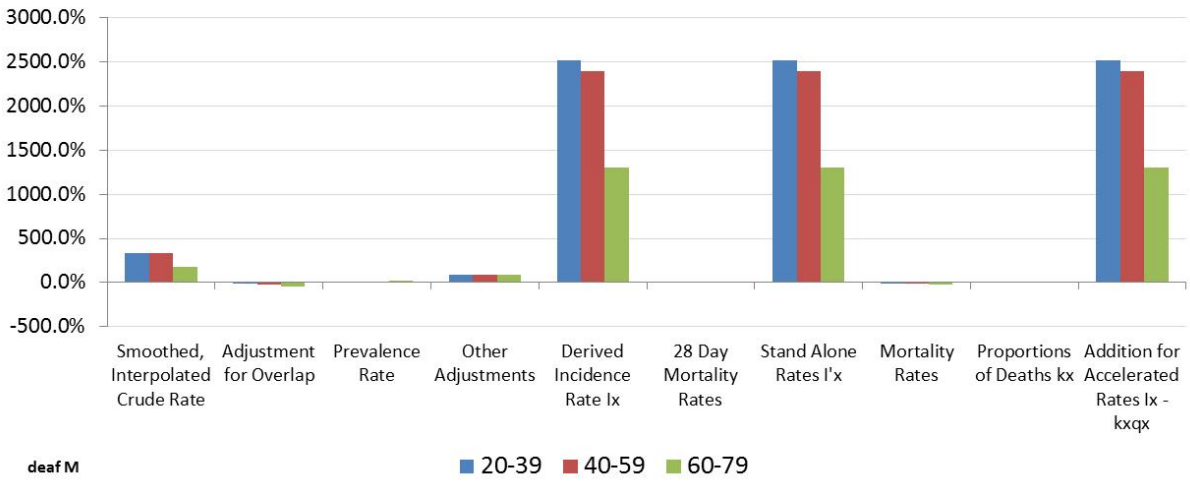
The ICD-10 codes used for this condition are shown in Appendix 2. The tables below provide a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

6.15.5.1. Male Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.38	2.49	7.88
Adjustment for Overlap	-9.9%	-18.4%	-42.7%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	1.25	2.13	5.95
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	1.25	2.13	5.93
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	1.25	2.13	5.95

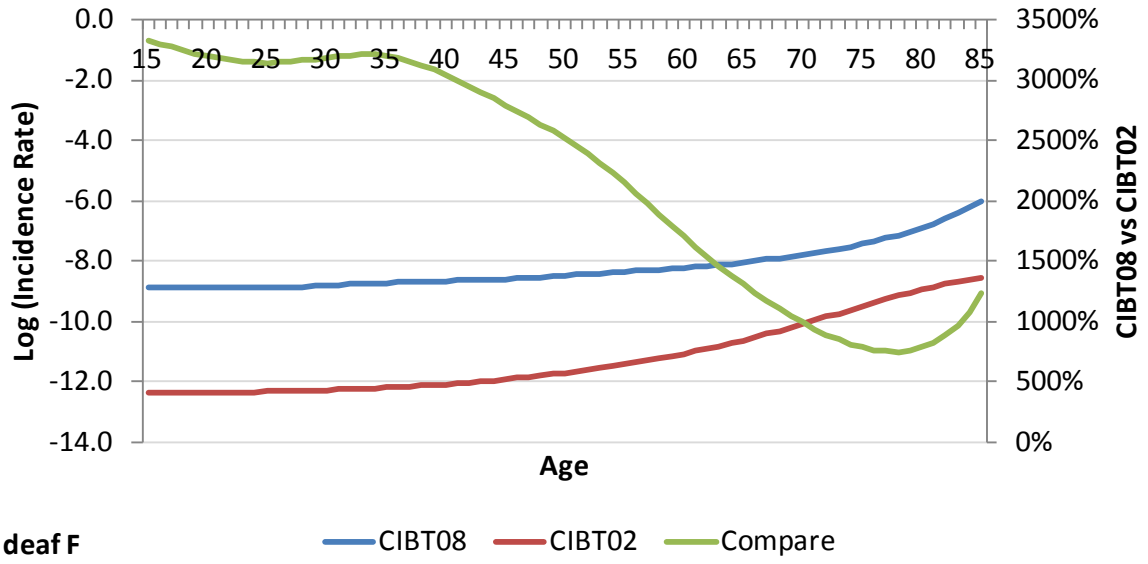
deaf _ M

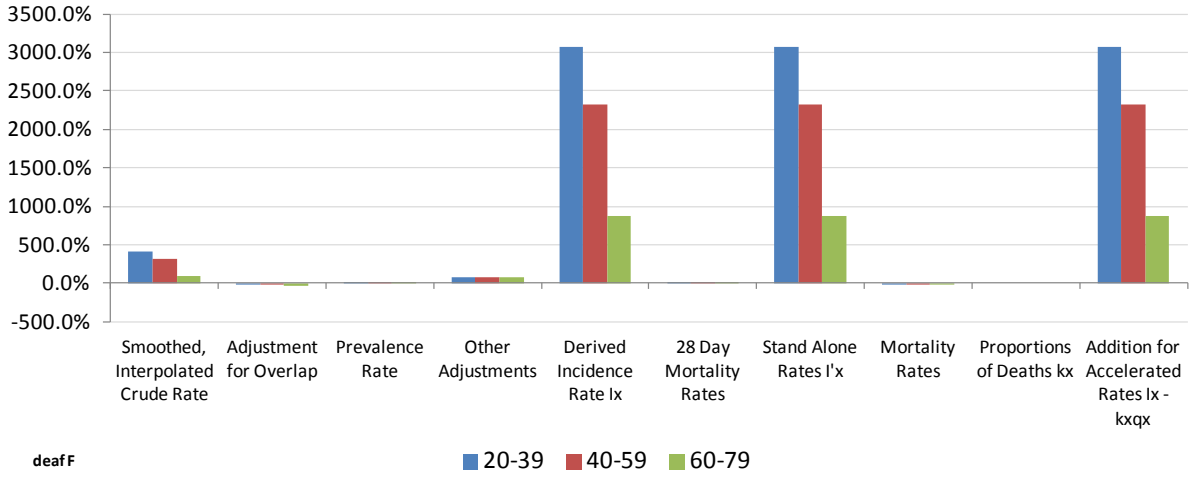




6.15.5.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.64	2.42	5.75
Adjustment for Overlap	-8.6%	-18.1%	-36.4%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	1.51	2.07	4.31
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	1.51	2.07	4.31
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	1.51	2.07	4.31





The CIBT08 rates are significantly higher than the CIBT02 rates. The CIBT02 rates are based on the HES data whereas for CIBT02 the data used was based on individuals registered as deaf based on reports from the Department of Health. CIBT02 also contained an adjustment to allow for the stricter definition that the hearing loss is greater than 95 db HL. The CIBT08 rates include less severe hearing loss.

6.16. Dementia

6.16.1. What is it?

Dementia is a serious loss of global cognitive ability in a previously unimpaired person, beyond what might be expected from normal aging. It may be static, the result of a unique global brain injury, or progressive, resulting in long-term decline due to damage or disease in the body. Although dementia is far more common in the geriatric population it can occur before the age of 65, in which case it is termed "early onset dementia"

Dementia is a syndrome (a group of related symptoms) associated with an ongoing decline of the brain and its abilities.

Some of the most common forms of dementia are: Alzheimer's disease, vascular dementia, frontotemporal dementia, semantic dementia and dementia with Lewy bodies.

6.16.2. Symptoms and Treatment

Dementia is not a disease but a collection of symptoms that result from damage to the brain. These symptoms can be caused by a number of conditions. The most common cause of dementia is Alzheimer's disease.

Common symptoms of Alzheimer's disease and other forms of dementia include:

- memory loss, especially problems with memory for recent events, such as forgetting messages, remembering routes or names, and asking questions repetitively;
- increasing difficulties with tasks and activities that require organisation and planning;
- becoming confused in unfamiliar environments;
- difficulty finding the right words;
- difficulty with numbers and/or handling money in shops;
- changes in personality and mood;
- depression.

Symptoms in the later stages of dementia

Dementia is progressive. This means that the person's brain will become more damaged and will work less well over time, and their symptoms will tend to change and become more severe.

As dementia progresses, memory loss and difficulties with communication often become very severe. In the later stages, the person is likely to neglect their own health and require constant care and attention.

Treatment

Currently, no medications have been shown to prevent or cure dementia. Medications are used to treat the behavioural and cognitive symptoms and have no effect on the underlying pathophysiology.

Cognitive and behavioural interventions may be appropriate. Educating and providing emotional support to the caregiver is of importance, along with the care itself.

6.16.3. Risk Factors

Age is the most significant risk factor for dementia. While it is possible to develop dementia early in life, the chances of doing so increase dramatically with age.

Other risk factors are mainly associated with lifestyle and very rarely from genes that we inherit. These risk factors include:

- Cardiovascular risk factors – Brain infarcts, heart disease and mid-life hypertension increase the risk of Alzheimer's disease and Vascular dementia. Smoking has also been identified as a risk factor;
- Diabetes – A recent study found that having diabetes increases the risk of developing Alzheimer's disease by 65%. This risk can be reduced by careful management of diabetes with medications that maintain blood glucose levels within a healthy range;
- High cholesterol – Cholesterol is essential to brain function – it is a component of cell membranes (structures that enclose nerve cells), and it is required for the repair and establishment of new connections between nerve cells. However, studies have shown that, high cholesterol in mid-life and late-life can increase the risk of Alzheimer's disease. Subsequent studies have indicated that cholesterol lowering drugs may lower the risk of developing Alzheimer's disease;
- High homocysteine levels – Homocysteine is a by-product of many metabolic reactions occurring in our body. Some studies have found that high homocysteine levels are associated with an increased risk of Alzheimer's disease and other dementias. Adequate intake of vitamin B and folate can help reduce homocysteine levels;
- Head injury – A study of World War II veterans indicated that moderate to severe head injury increased risk of developing Alzheimer's disease and other dementias. Another study found that this risk is further increased if the head injury resulted in loss of consciousness;
- Family history – A family history of dementia increases one's risk of developing dementia. This is probably due to genetic factors that have not yet been discovered;
- Genetic factors – Some risk factors predisposing to dementia are associated with genetic inheritance or previous life events.

6.16.4. Insurance Industry Definitions

Dementia does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market.

The definitions used in the market are generally similar and examples are shown below:

Dementia – resulting in permanent symptoms

A definite diagnosis of dementia by a consultant neurologist, psychiatrist or geriatrician. The diagnosis must be supported by evidence of progressive loss of ability to do all of the following:

- remember;
- to reason; and
- to perceive, understand, express and give effect to ideas.

For the above definition, the following is not covered:

- Dementia secondary to alcohol or drug abuse.

Dementia (including senile dementia) – resulting in permanent symptoms

A definite diagnosis of dementia by a Consultant Neurologist, Psychiatrist, or Geriatrician.

There must be permanent clinical loss of the ability to do all of the following:

- Remember
- Reason; and
- Perceive, understand, express and give effect to ideas.

Pre-senile dementia before age 65 – resulting in permanent symptoms

A definite diagnosis before your 65th birthday, by a consultant neurologist, psychiatrist or geriatrician, of pre-senile dementia. The diagnosis must, at the time it is made, be supported by evidence of progressive deterioration of memory and of the ability to reason and to perceive, understand, express and give effect to ideas.

Pre-senile dementia – resulting in permanent symptoms

Definite diagnosis of pre-senile dementia supported by evidence of progressive loss of ability to:

- remember;
- reason;
- perceive, understand, express and give effect to ideas,

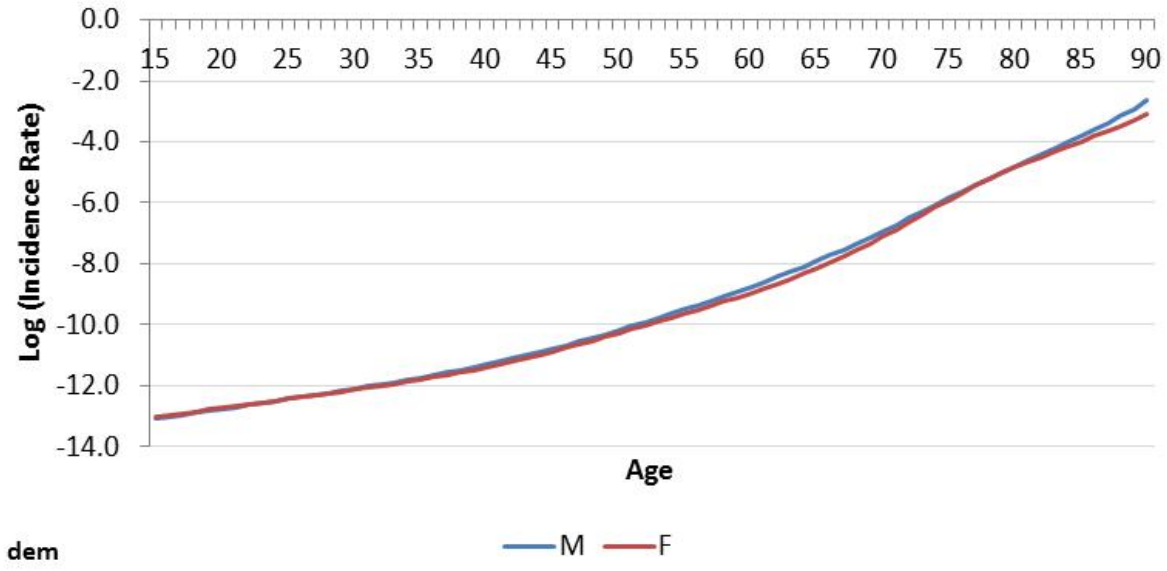
which causes a significant reduction in mental and social functioning, requiring the continuous supervision of the person covered.

6.16.5. Derived Incidence Rates

Dementia rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The following table provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.08	0.71	21.13	0.07	0.58	20.38
Adjustment for Overlap	-20.8%	-36.7%	-56.0%	-18.7%	-31.4%	-47.3%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.06	0.46	13.38	0.06	0.40	13.51
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.06	0.46	13.34	0.06	0.40	13.48
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.1%	0.8%	0.0%	0.1%	1.2%
Addition for Accelerated Rates Ix - kxqx	0.06	0.42	10.79	0.06	0.37	10.68

dem



6.17. Ductal Carcinoma in Situ / Mastectomy

6.17.1. What is it?

When a person has ductal carcinoma in situ (DCIS), it means that cells inside some of the ducts of the breast have started to turn into cancer cells. These cells are all contained inside the ducts and have not started to spread into the surrounding breast tissue. So, there is very little chance that any of the cells have spread to the lymph nodes or elsewhere in the body. Where DCIS is confined it may be described as pre-invasive, non-invasive, or intra ductal cancer.

DCIS is a very early form of breast cancer where cancer cells are confined to the epithelial linings. If it is not treated, then DCIS can spread into the surrounding breast tissue. Therefore, it may become an “invasive” cancer and would become payable under the cancer benefit of a critical illness plan.

DCIS is being found more often than in the past which is largely because of routine National Health System screening programmes that is available for all women over the age of 50 are screened for breast cancer by mammography.

6.17.2. Symptoms and Treatment

Because most forms of DCIS have a high probability of progression into invasive carcinoma, doctors will usually recommend that the lesion be completely removed. Therefore, DCIS is usually treated in much the same way as a malignant tumour.

Surgery is the main treatment for DCIS. Many women have removal of the area of DCIS, with a border of healthy tissue around it. This is called wide local excision or conservative surgery or sometimes lumpectomy. After wide local excision surgery, radiotherapy to the rest of the breast tissue may be performed if the DCIS.

Some women have removal of the whole breast (mastectomy). One may be advised to have a mastectomy if:

- the area of the DCIS is large;
- there are several areas of DCIS within the breast;
- the area of breast tissue is too significant affected by DCIS to make wide local excision possible.

After being explained the surgical options, some women prefer to have the whole breast removed rather than wide local excision as it makes them feel more confident that the DCIS is cured.

If DCIS cells have oestrogen receptors, further treatment may be given in the form of tamoxifen (a type of hormone therapy) to try to reduce the risk of developing an invasive breast cancer in the future

6.17.3. Risk Factors

The risk factors for DCIS are:

- **Getting older**: The risk of DCIS increases with age – it’s rare in young women.
- **Hormonal factors**: Long, uninterrupted periods of exposure to the hormones oestrogen and progesterone can influence the risk of breast cancer.
- **Some breast conditions**: The risk is also higher if where there has been a type benign breast disease called atypical hyperplasia.

- **Family history of breast cancer**: Sometimes, DCIS is linked to an inherited breast cancer gene. This is more likely if there's a strong history of breast cancer in a family. The chances of there being a breast cancer gene increases where:
    - several close relatives have had breast or ovarian cancer – close relatives, sometimes called first degree relatives, are your parents, children, brothers and sisters;
    - a close relative has had breast or ovarian cancer at a young age (under 40);
    - a close relative has had breast cancer in both breasts.

The genes most commonly linked to an increased risk of breast cancer in families are BRCA1 and BRCA2.

6.17.4. Insurance Industry Definitions

Ductal cancer in situ does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market.

Benefits are typically partial and do not pay full benefits in view of the better prognosis DCIS has when compared to invasive breast cancer.

There are some variations in the market as shown by the following examples which are typical of definitions used in the market:

Carcinoma in situ of the breast – requiring mastectomy or lumpectomy

The undergoing of a mastectomy or lumpectomy operation following the diagnosis of carcinoma in situ of the breast. In situ means tumours of the breast which are histologically confirmed as carcinoma in situ.

For the above definition, the following are not covered:

- Prophylactic mastectomy or lumpectomy without histological confirmation.

Ductal carcinoma in situ - *with surgery to remove the tumour*

The undergoing of a mastectomy, partial mastectomy, segmentectomy or lumpectomy operation on the advice of a consultant oncologist following a histologically confirmed diagnosis of ductal carcinoma in situ (DCIS) of the breast.

Specifically excluded are:

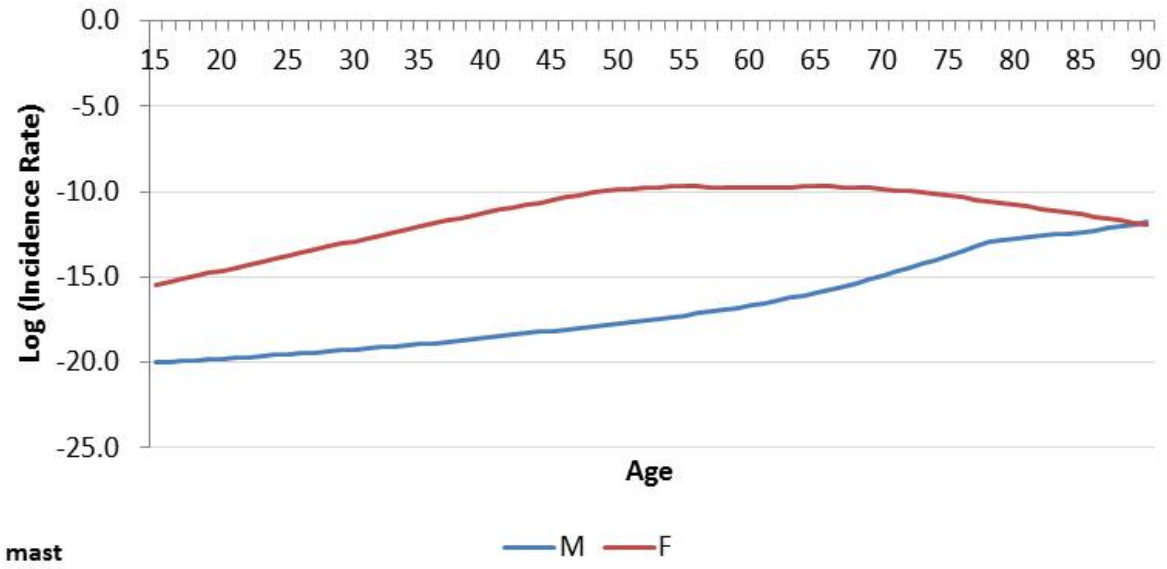
- mastectomy, partial mastectomy, segmentectomy or lumpectomy operations for reasons other than DCIS, for example, prophylactic mastectomy or lobular carcinoma in situ (LCIS).

6.17.5. Derived Incidence Rates

DCIS rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.00	0.00	0.01	0.05	0.55	0.75
Adjustment for Overlap	-98.4%	-96.0%	-72.7%	-26.3%	-28.2%	-44.6%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.00	0.00	0.01	0.04	0.41	0.50
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.00	0.00	0.01	0.04	0.41	0.50
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.00	0.00	0.01	0.04	0.41	0.50

mast



Mastectomy rates peak at around age 55 before flattening off and reducing at the very eldest ages. The flattening is in common with the incidence rate of all breast cancers and the reduction is expected where there is some type of operation/procedure that will be carried out less frequently in the elderly.

6.18. Emphysema (respiratory failure or chronic lung disease)

6.18.1. What is it?

Emphysema is a long-term, progressive disease of the lungs that primarily causes shortness of breath. In people with emphysema the lung tissues necessary to support the physical shape and function of the lung are damaged. It is included in a group of diseases called Chronic Obstructive Pulmonary Disease or COPD (pulmonary refers to the lungs). Emphysema is called an obstructive lung disease because the destruction of lung tissue around smaller airways, called bronchioles, makes these airways unable to hold their shape properly when one exhales. This makes them inefficient at transferring oxygen into the blood, and in taking carbon dioxide out of the blood.

The most common cause is cigarette smoking. Prolonged exposure to smoke gradually causes enough lung destruction to cause the characteristic cough and shortness of breath. Affected individuals with alpha-1 antitrypsin deficiency tend to develop symptoms of emphysema at earlier ages. Emphysema is a subtype of chronic obstructive pulmonary disease (COPD in the US; COLD in the United Kingdom).

6.18.2. Symptoms and Treatment

Treatment is based on whether symptoms are mild, moderate or severe. Treatments include inhalers, oxygen, medications and sometimes surgery to relieve symptoms and prevent complications.

Emphysema is a progressive disease with symptoms beginning in patients after 50 years of age. Most patients, except in those in whom disease is the result of a genetic deficiency (alpha-1 antitrypsin deficiency), have variable manifestations of the different components of COPD which include:

- chronic bronchitis;
- asthma;
- emphysema; and
- bronchiectasis.

Each of the subtypes has characteristic symptoms; those primarily associated with emphysema are shortness of breath and wheezing. Initially the shortness of breath (dyspnoea) occurs with activity; as time continues and the disease progresses, the episodes of dyspnoea occur more frequently eventually occurring at rest making routine daily activities difficult to perform.

6.18.3. Risk Factors

The main risk factor for emphysema is smoking, which activates inflammatory cells in the lung. This inflammation causes swelling within the bronchioles, and activation of enzymes called proteases which attack and destroy lung tissue. This leads to centriacinar emphysema, which begins in the bronchioles and gradually spreads peripherally to the far reaches of the lung. There may be a genetic contribution to the development of emphysema, since not all people who smoke suffer from emphysema. The risk for all types of smokers increases with the number of years and amount of tobacco smoked.

There is also an inherited form of emphysema. The relatively rare condition known as alpha 1-antitrypsin deficiency is the genetic deficiency of a chemical that protects the lung from damage by proteases. This results in panacinar emphysema, which destroys the alveoli throughout the lung uniformly.

Aging is also a risk factor. As the lungs get older, the elastic properties decrease, and the tensions that develop can result in small areas of emphysema. Although the lung damage that occurs in emphysema develops gradually, most people with tobacco-related emphysema begin to experience symptoms of the disease between the ages of 40 and 60.

There are other risk factors such as:

- Exposure to second-hand smoke - being around second-hand smoke increases the risk of emphysema;
- Occupational exposure to fumes or dust – exposure to certain chemicals or dust from grain, cotton, wood or mining products is more likely to develop emphysema. This risk is even greater if one smokes;
- Exposure to indoor and outdoor pollution - car exhaust, for instance, increases the risk of emphysema.

6.18.4. Insurance Industry Definitions

Respiratory failure/emphysema does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market.

A few examples of the current definitions used by the insurers are set out below.

Emphysema

Advanced stage emphysema or other chronic lung disease, resulting in all of the following:

- The need for regular oxygen treatment on a permanent basis.
- The permanent impairment of lung function tests as follows;
- Forced Vital Capacity (FVC) and Forced Expiratory Volume at 1 second (FEV1) being less than 50% of normal.

Chronic lung disease

Confirmation by a Consultant Physician of chronic lung disease resulting in all of the following:
the need for continuous daily oxygen therapy on a permanent basis;
FEV1 being **less than 40% of normal**; and Vital Capacity less than 50% of normal.

Severe lung disease

Confirmation by a Consultant Physician of severe lung disease where there is permanent impairment of lung function evidenced by all of the following:

- The need for daily oxygen therapy for at least 15 hours per day for a minimum of six months,
- Forced Vital Capacity (FVC) being less than 50% of normal, and
- Forced Expiratory Volume at 1 second (FEV1) being less than 40% of normal.

Emphysema

Confirmation by a consultant physician of severe lung disease which is evidenced by all of the following:

- the need for continuous daily oxygen therapy on a permanent basis
- evidence that oxygen therapy has been required for a minimum period of six months
- FEV1 being less than 40 percent of normal; and
- vital capacity less than 50 percent of normal

Emphysema

Advanced stage emphysema or other chronic lung disease, resulting in all of the following:

- The need for oxygen therapy for a minimum of 15 hours a day and evidence that daily oxygen therapy has been required for a minimum period of 6 months.
- The permanent impairment of lung function tests as follows:
    - Forced vital capacity (FVC) and forced expiratory volume at 1 second (FEV1) being less than 40% of normal. (AEGON's definition)

6.18.5. Derived Incidence Rates

Emphysema / Respiratory Failure rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2 but importantly we have attempted to apply the severity underpin by requiring the ICD-10 codes to appear in conjunction with OPCS codes indicating the need for permanent oxygen support.

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000). However, these rates are effectively zero so we will treat them as zero going forward.

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.17	0.00	0.00	0.00	0.00	0.01
Adjustment for Overlap	-64.5%	0.0%	0.0%	-4.6%	-45.2%	-32.1%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.02	0.00	0.00	0.00	0.00	0.01
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.02	0.00	0.00	0.00	0.00	0.01
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.02	0.00	0.00	0.00	0.00	0.00

rf

6.19. Encephalitis

6.19.1. What is it?

Encephalitis is irritation and swelling (inflammation) of the brain, most often due to infections. It is a rare condition and occurs mostly in the first year of life. It is caused by a viral infection and many types of viruses can cause it.

The virus causes inflammation of brain tissue. The brain tissue swells (cerebral oedema), which may destroy nerve cells, cause bleeding in the brain (intracerebral haemorrhage), and brain damage.

Permanent brain damage may occur in severe cases of encephalitis. It can affect hearing, memory, muscle control, sensation, speech and vision.

Some cases are mild and short, and the person fully recovers. Other cases are severe, and permanent impairment or death is possible.

6.19.2. Symptoms and Treatment

Some patients may have symptoms of a cold or stomach infection before encephalitis symptoms begin. When a case of encephalitis is not very severe, the symptoms may be similar to those of other illnesses, including: fever, mild headache, low energy and a poor appetite.

Other symptoms include: clumsiness, disorientation, drowsiness, irritability and confusion, light sensitivity, stiff neck and back and vomiting.

Symptoms in newborns and younger infants may not be as easy to recognise.

Emergency symptoms include: loss of consciousness, muscle weakness, seizures and sudden change in mental functions such as amnesia, impaired judgement and low mood.

The treatment of encephalitis aims to provide supportive care (rest, nutrition, fluids) to help the body fight the infection, and to relieve symptoms. Hence, it includes:

- Reorientation and emotional support for confused or delirious patients;
- Antiviral medications to treat herpes encephalitis or other severe viral antibiotics if the infection is caused by certain bacteria;
- Physical therapy and speech therapy after the illness to restore brain function;
- Anti-seizure medications to prevent seizures;
- Sedatives to treat irritability or restlessness.

6.19.3. Risk Factors

The risk factor is the exposure to viruses through:

- Breathing in respiratory droplets from an infected person;
- Contaminated food or drink;
- Skin contact;
- Mosquito, tick and other insect bites.

Other factors include: allergic reactions to vaccinations, autoimmune disease and bacterial infection.

6.19.4. Insurance Industry Definitions

Encephalitis does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market. Definitions used in the market are very similar and an example is as follows:

> **Encephalitis - resulting in permanent symptoms**
>
> A definite diagnosis of encephalitis by a Consultant Neurologist resulting in permanent neurological deficit with persisting clinical symptoms.

6.19.5. Derived Incidence Rates

Encephalitis rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.31	0.48	0.79	0.33	0.43	0.62
Adjustment for Overlap	-13.8%	-24.7%	-41.8%	-15.9%	-25.9%	-41.5%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.27	0.38	0.62	0.28	0.33	0.44
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.27	0.38	0.62	0.28	0.33	0.44
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.1%	0.0%	0.0%	0.1%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.27	0.37	0.57	0.27	0.33	0.40

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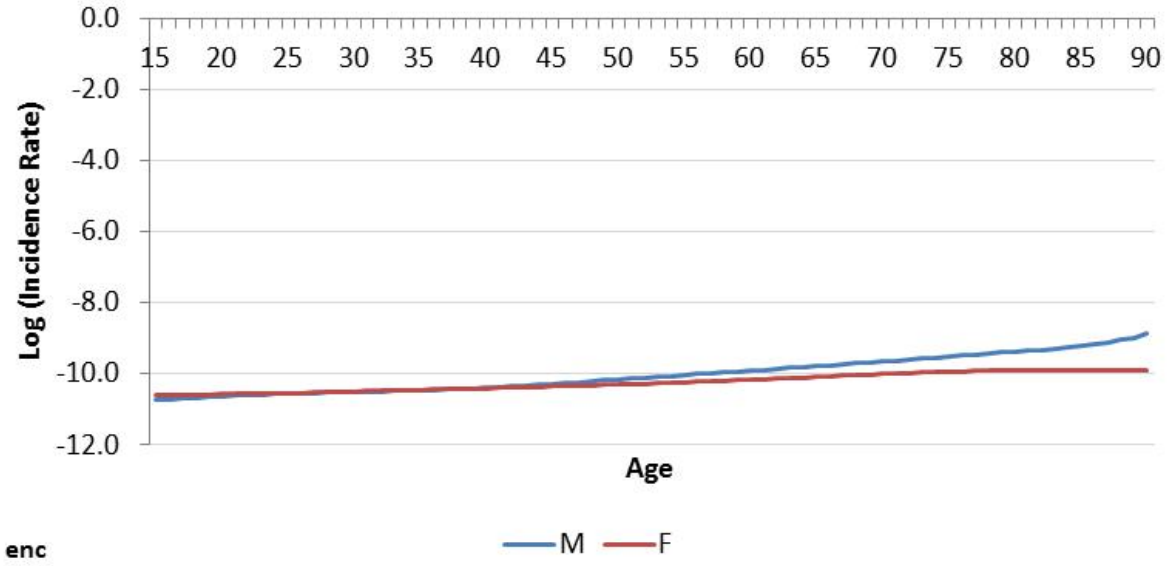
Results are broadly of the same order as the Bacterial Meningitis results derived in section 6.7. As with Bacterial Meningitis the likelihood is that a relatively small proportion of hospital admissions would result in permanent neurological deficit.



We have compared our crude encephalitis rates to benchmark rates derived from "New Estimates of Incidence of Encephalitis in England"[21]. Their method also used HES data and resulted in a rate not disimilar to ours.

[21] Granerod J, Cousens S, Davies NWS, Crowcroft NS, Thomas SL. New estimates of incidence of encephalitis in England. Emerg Infect Dis [Internet]. 2013 Sep. http://dx.doi.org/10.3201/eid1909.130064

6.20. Heart Valve Replacement or Repair

6.20.1. What is it?

There are four valves in the heart that ensure that the blood flows through the heart in the correct direction.

If one or more of the valves is diseased or damaged, it can affect blood flows through the heart in two ways:

- If the valve does not open fully, it will obstruct the flow of blood. This is called valve stenosis or narrowing;
- If the valve does not close properly, it will allow blood to leak backwards. This is called valve incompetence, or regurgitation, or a leaky valve.

There are two options for valve surgery: valve repair and valve replacement.

- **Valve repair** is often used for mitral valves that become floppy and leak but are not seriously damaged.
- **Valve replacement** is when the diseased valve is replaced with a new valve. The most common types of replacement valves are mechanical (artificial) valves or tissue (animal) valves.

In some cases, a Transcatheter Aortic Valve Implantation (TAVI) procedure may be used if the patient is an adult and is not well enough to have traditional heart surgery.

Whether or not it should be a heart valve surgery, and/or whether the operation is a repair or a replacement will depend on many factors, including the cause of the problem, which valve is affected, how badly the valve is affected, how many valves are affected, symptoms, and general health.

Heart valve repair or replacement surgery is a treatment option for valvular heart disease. When heart valves become damaged or diseased, they may not function properly. Conditions which may cause heart valve dysfunction are valvular stenosis and valvular insufficiency (regurgitation). Heart valve disease may cause the heart to pump less efficiently. If blood does not flow through the heart properly, extra strain is put on it.

When one (or more) valve(s) becomes stenotic (stiff), the heart muscle must work harder to pump the blood through the valve. Some reasons why heart valves become stenotic include infection (such as rheumatic fever or staphylococcus infections) and aging. If one or more valves become insufficient (leaky), blood leaks backwards, this means that less blood is pumped in the proper direction.

6.20.2. Symptoms and Treatment

Symptoms include breathlessness, tiredness and swollen ankles. If the heart valve(s) becomes damaged or diseased, a person may experience the following symptoms:

- Dizziness;
- Chest pain;
- Breathing difficulties;
- Palpitations;
- Oedema (swelling) of the feet, ankles, or abdomen;
- Rapid weight gain due to fluid retention.

Many people with heart valve disease need little or no treatment. However, the patient may be advised to have surgery on his/her valve, which can greatly improve the symptoms and quality of life.

Heart valve surgery is an operation used to treat leaking or narrowed valves. It can improve or get rid of symptoms and may prevent permanent damage to the heart.

Heart valve replacement surgery involves the removal of the badly damaged valve. The valve is replaced with a plastic or metal mechanical valve, or a bio-prosthetic valve, which is usually made from pig tissue. The damaged valve is cut out, and the new valve is sewn into place.

People who receive a mechanical heart valve are more likely than those who receive a bio-prosthetic heart valve to develop blood clots in the heart. The clots may break loose, travel to the brain, and cause a stroke. So it is a mechanical heart valve to treat severe MR, the patient will need to take medicine for the rest of his/her life to prevent clots from forming (anticoagulant medicine).

In some cases, a plastic or metal valve may be preferred if the patient is already taking anticoagulants for other reasons, such as atrial fibrillation.

Traditionally, repair or replacement of heart valves has involved open-heart surgery. In order to open the chest, the breastbone, or sternum, is cut in half and spread apart. Once the heart is exposed, large tubes are inserted into the heart so that the blood can be pumped through the body during the surgery by a cardiopulmonary bypass machine (heart-lung machine). The bypass machine is necessary to pump blood because the heart is stopped and kept still while the surgeon performs the valve repair or replacement procedure.

Newer, less invasive techniques have been developed to replace or repair heart valves. Minimally invasive procedures make smaller incisions–and mean less pain afterward and shorter hospital stays. Balloon valvuloplasty is one such procedure. It's used to treat some cases of valve stenosis, and is done as part of a catheterization procedure, rather than as part of open heart surgery.

Transcatheter aortic valve replacement, or TAVR, is a new alternative for some cases of aortic valve stenosis. This hybrid procedure typically is done by a cardiac surgeon and an interventional cardiologist.

6.20.3. Risk Factors

Advancing age and congenital heart problems (present from birth) are common risk factors. These are factors which are not controllable. Factors one can control include infections and untreated strep throat, which can lead to rheumatic fever.

6.20.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

> **Heart valve replacement or repair (1999/2002)**
>
> Undergoing open heart surgery from medical necessity to replace one or more heart valves.

Most insurers adopted the following definition but there is no clear indication if the surgery required is the median sternotomy.

> **Heart valve replacement or repair – with surgery to divide the breastbone (2006/2011)**
>
> The undergoing of surgery requiring median sternotomy (surgery to divide the breastbone) on the advice of a Consultant Cardiologist to replace or repair one or more heart valves.

There are, however, some insurers using the following definitions which enhance benefit to ABI+ by removing the requirement for surgery to include median sternotomy as follows.

> **Heart valve replacement or repair**
>
> The undergoing of surgery on the advice of a consultant cardiologist to replace or repair one or more heart valves.

> **Heart valve replacement or repair**
>
> The undergoing of surgery requiring **thoracotomy (keyhole surgery** or median sternotomy) on the advice of a consultant cardiologist to replace or repair one or more heart valves.

6.20.5. Derived Incidence Rates

6.20.5.1. Male Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.58	2.06	11.04
Adjustment for Overlap	-18.6%	-29.3%	-43.5%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.47	1.52	8.46
28 Day Mortality Rates	-0.6%	-0.8%	-2.2%
Stand Alone Rates I'x	0.47	1.51	8.24
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.1%
Addition for Accelerated Rates Ix - kxqx	0.47	1.51	8.31

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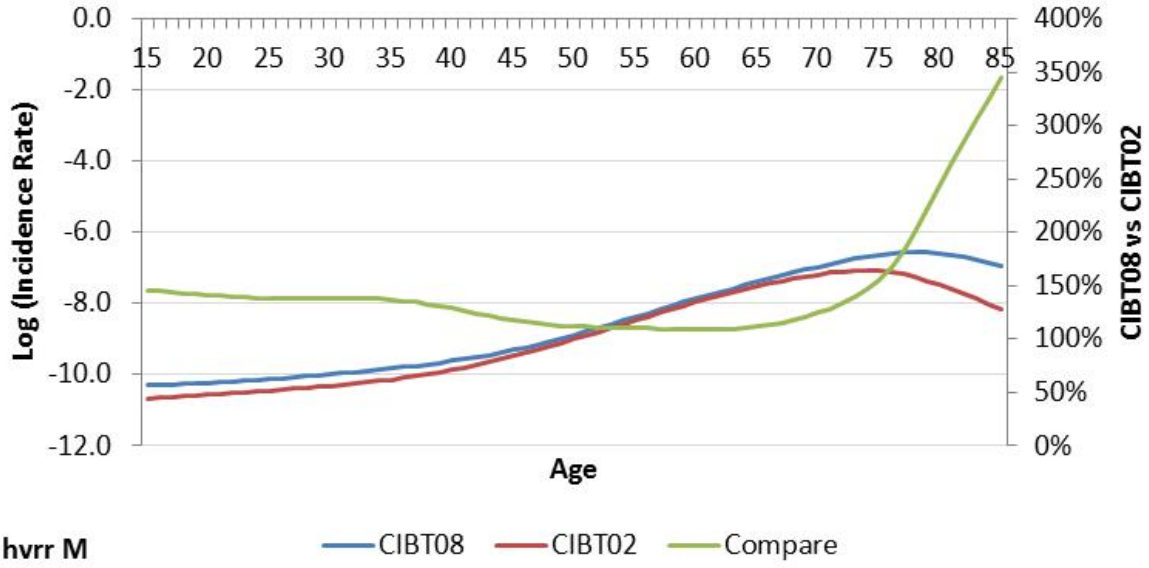
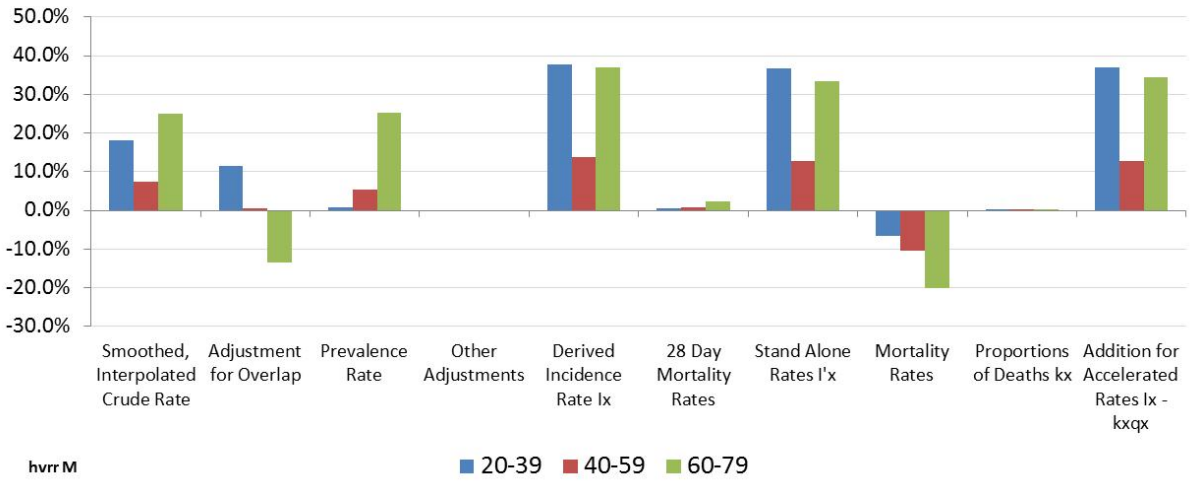
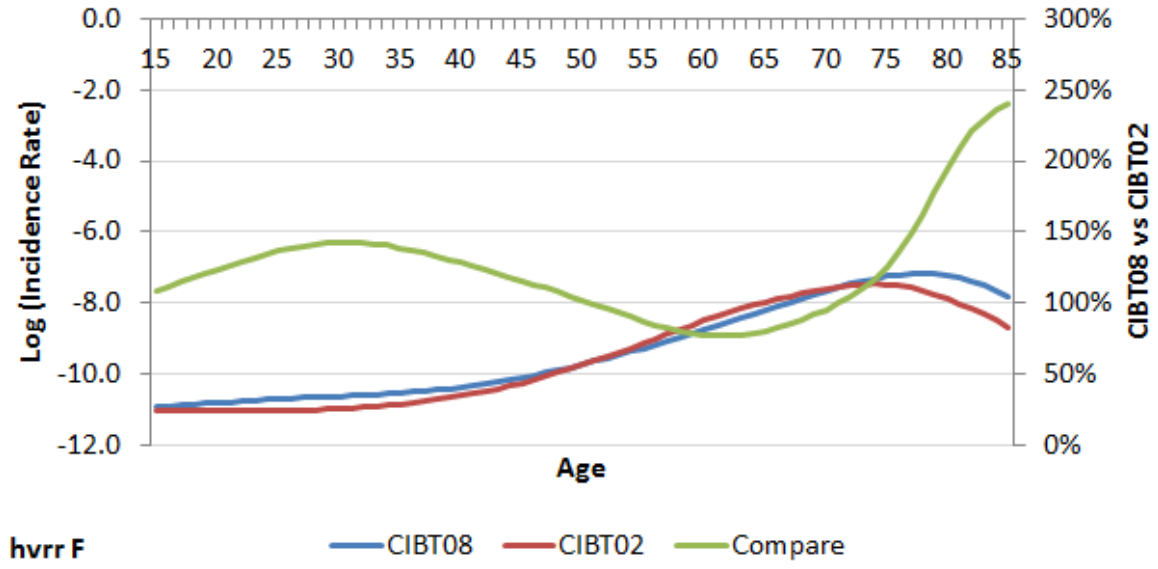




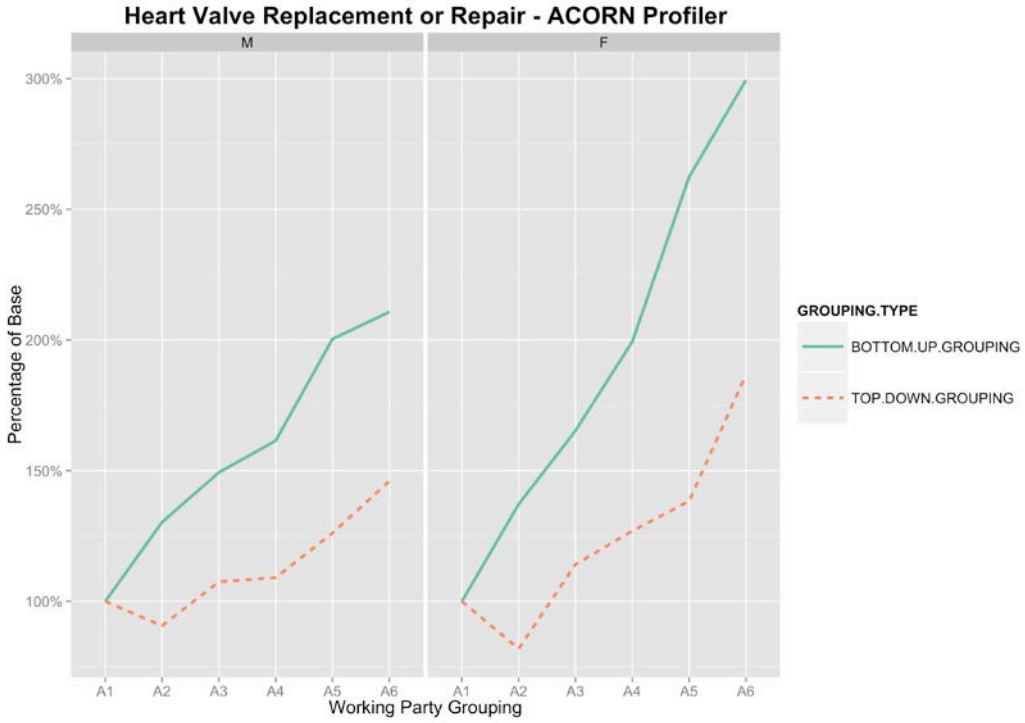
6.20.5.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.33	0.96	6.46
Adjustment for Overlap	-25.4%	-35.6%	-44.1%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.25	0.65	4.41
28 Day Mortality Rates	-0.6%	-0.8%	-2.3%
Stand Alone Rates I'x	0.25	0.65	4.29
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.1%
Addition for Accelerated Rates Ix - kxqx	0.25	0.65	4.31

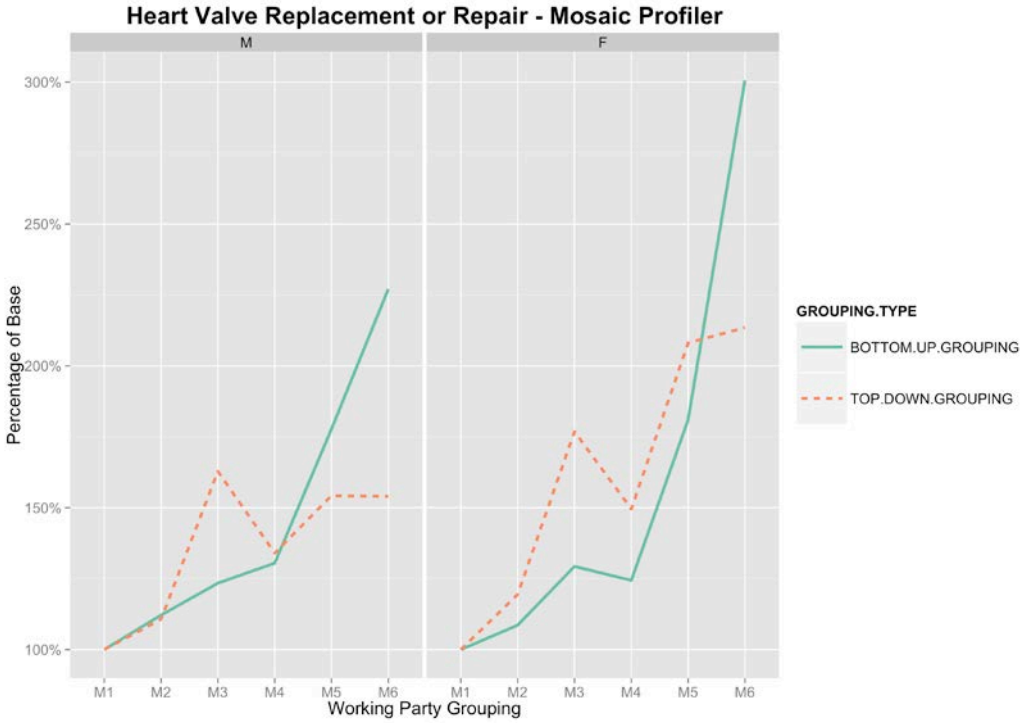


6.20.6. Geodemographic Analysis

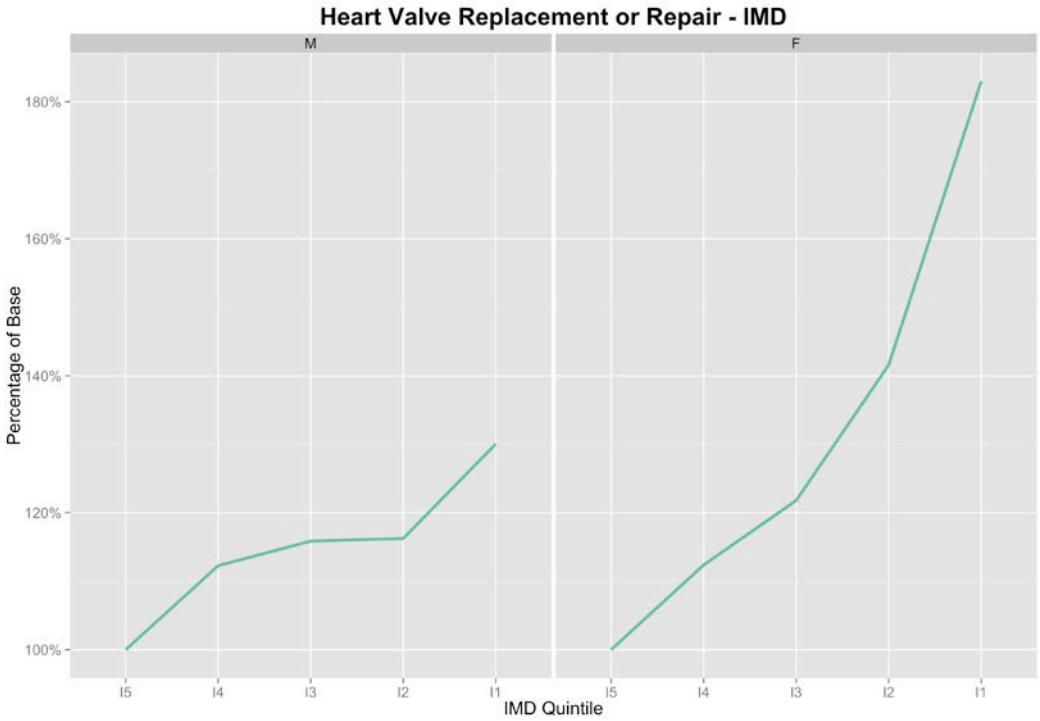
6.20.6.1. ACORN



6.20.6.2. Mosaic



6.20.6.3. Index of Multiple Deprivation



6.21. HIV Infection

6.21.1. What is it?

HIV infection is a condition caused by the human immunodeficiency virus (HIV). The condition gradually destroys the immune system, which makes it harder for the body to fight infections.

Human immunodeficiency virus (HIV) causes AIDS. The virus attacks the immune system and leaves the body vulnerable to a variety of life-threatening infections and cancers.

HIV is found in saliva, tears, nervous system tissue and spinal fluid, blood, semen, vaginal fluid and breast milk. However blood, semen, vaginal secretions and breast milk have been shown to transmit infection to others.

6.21.2. Symptoms and Treatment

People who become infected with HIV may not have any symptoms for up to 10 years yet can pass the infection to others.

Symptoms related to HIV are usually due to a different infection in the body as the immune system is weakened to fight against opportunistic infections. These symptoms include diarrhoea, fatigue, fever, mouth sores, muscle stiffness, skin rashes, sore throat, swollen lymph glands and weight loss.

There is no cure for HIV at this time. A range of treatments are available to manage symptoms at and improve the quality and length of life.

Antiretroviral therapy suppresses the replication of the HIV virus in the body. A combination of several antiretroviral drugs, called highly active antiretroviral therapy has been very effective in reducing the number of HIV particles in the bloodstream. It is extremely important for people with HIV to take all doses of their medications; otherwise the virus may become resistant to the drugs.

Medications are also used to prevent opportunistic infections.

People with HIV infection are educated about the disease and treatment so that they can be active participants in making decisions with their health care provider.

6.21.3. Risk Factors

The HIV virus can be transmitted:

- Through sexual contact -- including oral, vaginal, and anal sex
- Through blood -- via blood transfusions or needle sharing
- From mother to child -- a pregnant woman can transmit the virus to her foetus through their shared blood circulation, or a nursing mother can transmit it to her baby in her breast milk

Other methods of spreading the virus are rare and include accidental needle injury, artificial insemination with infected donated semen, and organ transplantation with infected organs.

6.21.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

HIV Infection *- caught in the UK from a blood transfusion, a physical assault or at work in an eligible occupation* **(2006/2011)**

Infection by Human Immunodeficiency Virus resulting from:

- a blood transfusion given as part of medical treatment;
- a physical assault; or
- an incident occurring during the course of performing normal duties of employment from the eligible occupations if applicable;

after the start of the policy and satisfying all of the following:

- The incident must have been reported to appropriate authorities and have been investigated in accordance with the established procedures.
- Where HIV infection is caught through a physical assault or as a result of an incident occurring during the course of performing normal duties of employment, the incident must be supported by a negative HIV antibody test taken within 5 days of the incident.
- There must be a further HIV test within 12 months confirming the presence of HIV or antibodies to the virus.
- The incident causing infection must have occurred in the UK including in geographic limits as applicable

The above definition does not cover:

- HIV infection resulting from any other means, including sexual activity or drug abuse.

Companies often enhance benefit in order to gain ABI+ status by providing wider geographical coverage of the countries in which the incident causing infection occurs other than just in the UK.

6.21.5. Derived Incidence Rates

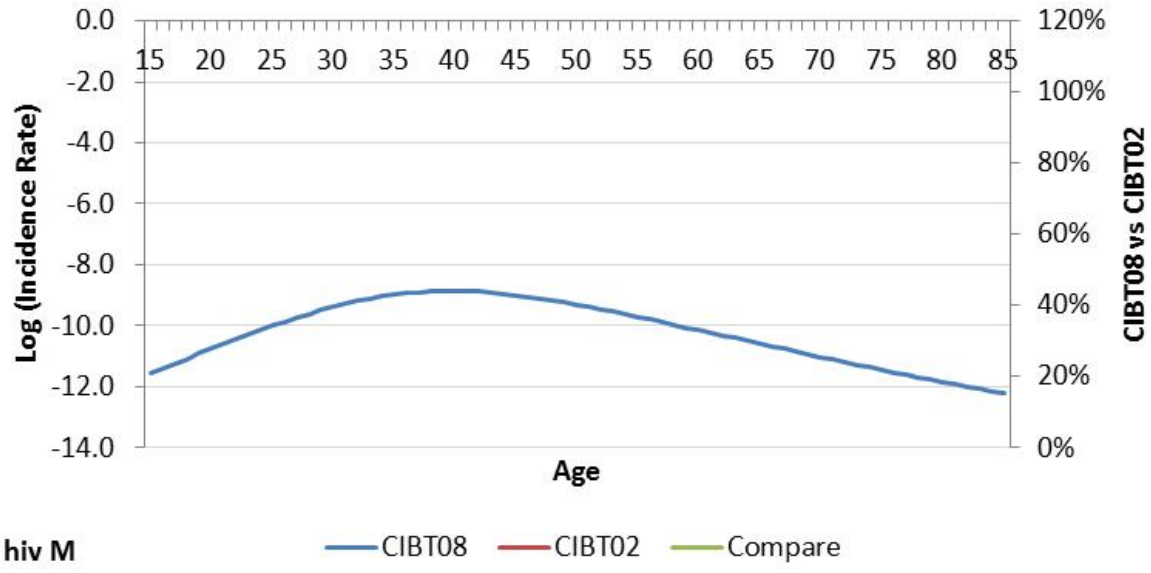
6.21.5.1. Male Results

For CIBT02 all rates were set to zero as incidence is believed to be negligible in context of overall CI rates.

The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

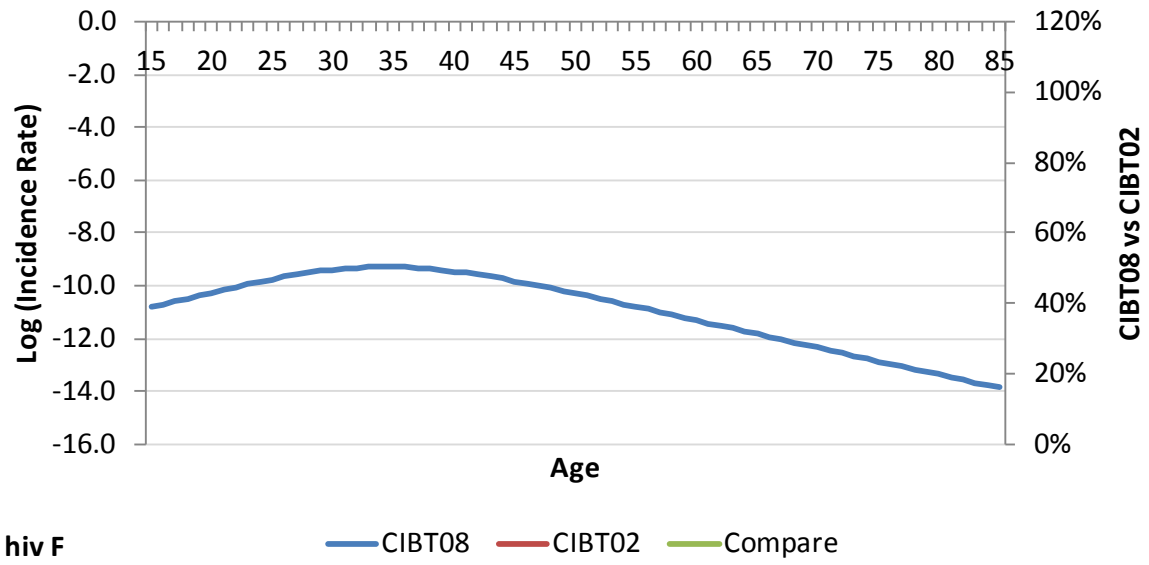
Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.88	1.05	0.23
Adjustment for Overlap	-5.2%	-12.8%	-25.3%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.83	0.97	0.22
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.83	0.97	0.22
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.5%	0.5%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.78	0.85	0.15

hiv _ M



6.21.5.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.77	0.44	0.06
Adjustment for Overlap	-5.0%	-9.9%	-17.4%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.73	0.42	0.06
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.73	0.42	0.06
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.9%	0.3%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.69	0.37	0.05



6.22. Kidney Failure

6.22.1. What is it?

Kidney failure is a medical condition in which the kidneys fail to adequately filter waste products from the blood. The function of the kidneys is, among other things, to get rid of the waste products that result from the body's metabolism. One of the major by-products of the metabolism of protein is urea. The kidneys remove the waste products by extracting them from the blood and sending them along the ureter to the bladder, from where they are excreted in the urine.

If the kidneys fail, there is a harmful build-up of the body’s waste products. In severe cases it may be necessary for the filtering to be done by a dialysis machine or, in some cases, a transplant may be needed. Kidney failure can become life threatening.

The two main forms are acute kidney injury, which is often reversible with adequate treatment, and chronic kidney disease, which is often not reversible.

Kidney failure is also known as renal failure or renal insufficiency.

6.22.2. Symptoms and Treatment

Most people with kidney failure have no symptoms because the body can tolerate even a large reduction in kidney function.

If kidney failure does occur, the symptoms may include:

- weight loss and poor appetite;
- swollen ankles, feet or hands (due to water retention);
- shortness of breath;
- blood or protein in urine;
- an increased need to urinate, particularly at night;
- itchy skin;
- muscle cramps;
- high blood pressure (hypertension);
- nausea;
- erectile dysfunction in men (an inability to get or maintain an erection);
- tiredness.

A change in kidney function is usually discovered through a routine blood or urine test. If you are diagnosed with kidney disease, your kidney function will be monitored with regular blood tests, and treatment aims to keep any symptoms to a minimum.

But with long-term kidney disease, if the kidneys deteriorate and can no longer function at all, there are only two treatment options:

- dialysis, which uses an artificial device to clean the blood of waste products or
- a kidney transplant.

People with Chronic Kidney Disease (CKD) are known to have an increased risk of a stroke or heart attack because of changes that occur to the circulation.

Medication, especially those that control diabetes and high blood pressure, can sometimes help slow the progress of chronic kidney disease. A sudden loss of kidney function may improve if the underlying cause -- such as a pregnancy complication -- is resolved.

6.22.3. Risk Factors

People are at higher risk if they have:

- high blood pressure (hypertension);
- diabetes;
- a family history of Chronic Kidney Disease (CKD).

Kidney failure is mainly associated with ageing. The older you get, the more likely you are to have some degree of kidney disease.

Kidney failure is more common in people of south Asian origin (those from India, Bangladesh, Sri Lanka and Pakistan) and black people than the general population. The reasons for this include higher rates of diabetes in south Asian people and higher rates of high blood pressure in African or Caribbean people.

6.22.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Kidney Failure – (1999/2002)

End stage renal failure presenting as chronic irreversible failure of both kidneys to function, as a result of which either regular renal dialysis or renal transplant is initiated.

Kidney Failure – requiring dialysis (2006/2011)

Chronic and end stage failure of both kidneys to function, as a result of which regular dialysis is necessary.

6.22.5. Derived Incidence Rates

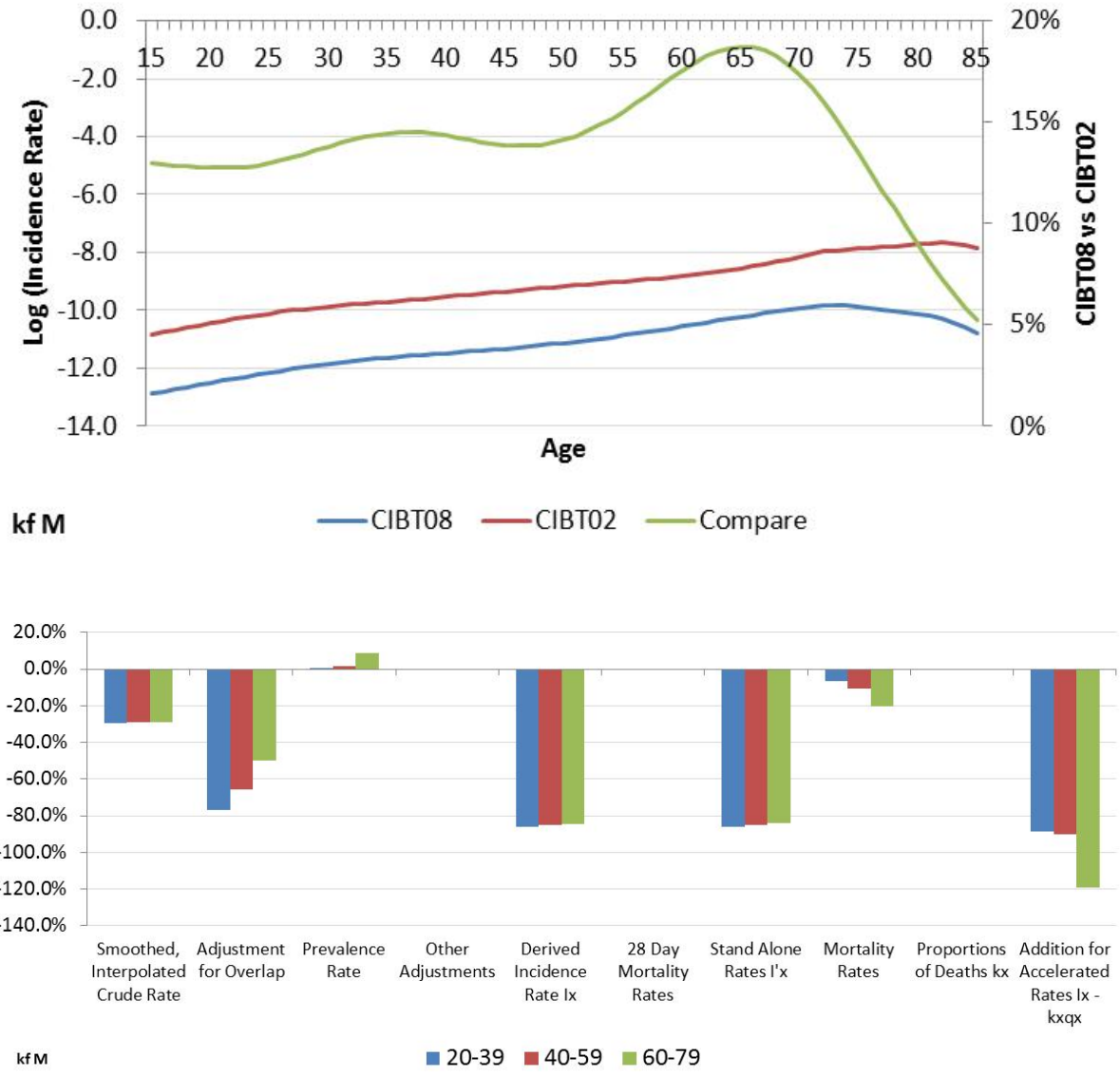
The ICD-10 codes used for this condition are shown in Appendix 2.

6.22.5.1. Male Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.37	0.87	2.90
Adjustment for Overlap	-81.4%	-83.1%	-87.5%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.07	0.15	0.48
28 Day Mortality Rates	-0.6%	-1.9%	-11.4%
Stand Alone Rates I'x	0.07	0.15	0.41
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.1%	0.2%	0.3%
Addition for Accelerated Rates Ix - kxqx	0.06	0.09	-0.35

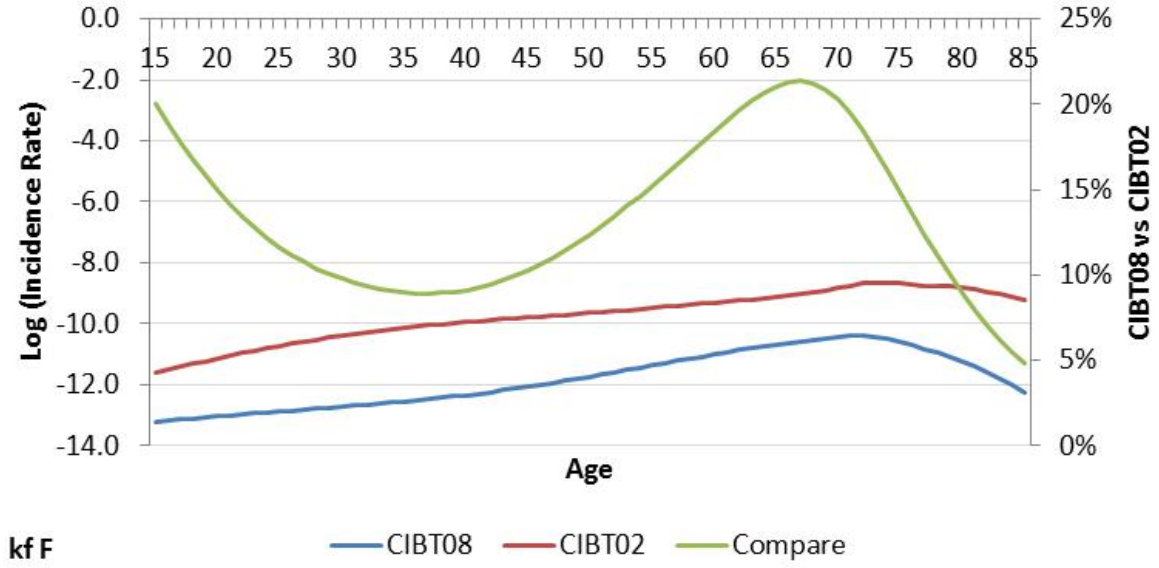
kf _ M



6.22.5.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.29	0.59	1.65
Adjustment for Overlap	-88.7%	-87.0%	-85.6%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.03	0.08	0.28
28 Day Mortality Rates	-0.6%	-1.9%	-14.6%
Stand Alone Rates I'x	0.03	0.08	0.23
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.2%	0.2%	0.3%
Addition for Accelerated Rates Ix - kxqx	0.02	0.04	-0.32

kf _ F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-2.0
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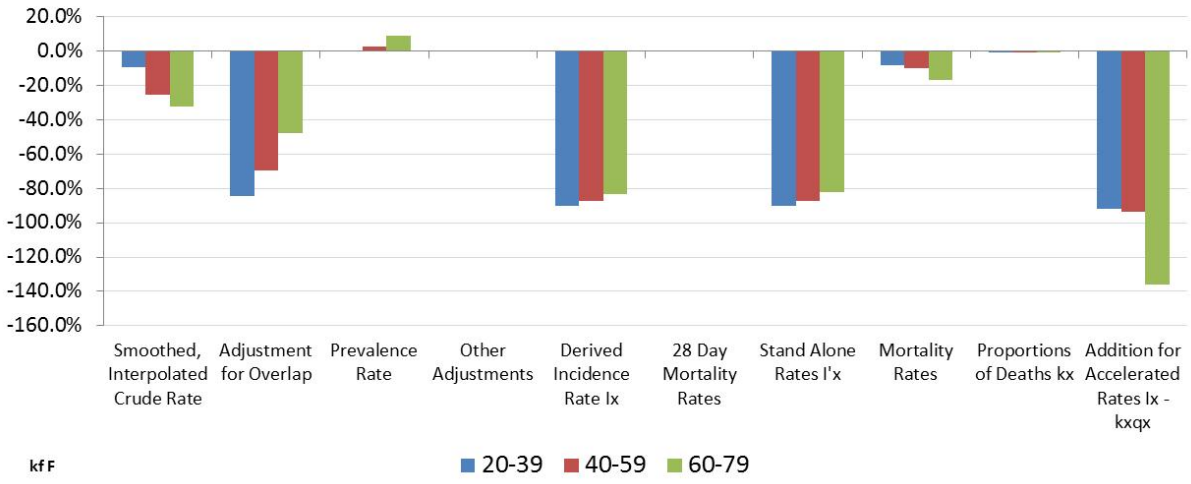
20.0%
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-140.0%
-160.0%
Smoothed, Interpolated Crude Rate
Adjustment for Overlap
Prevalence Rate
Other Adjustments
Derived Incidence Rate Ix
28 Day Mortality Rates
Stand Alone Rates I'x
Mortality Rates
Proportions of Deaths kx
Addition for Accelerated Rates Ix - kxqx
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20-39
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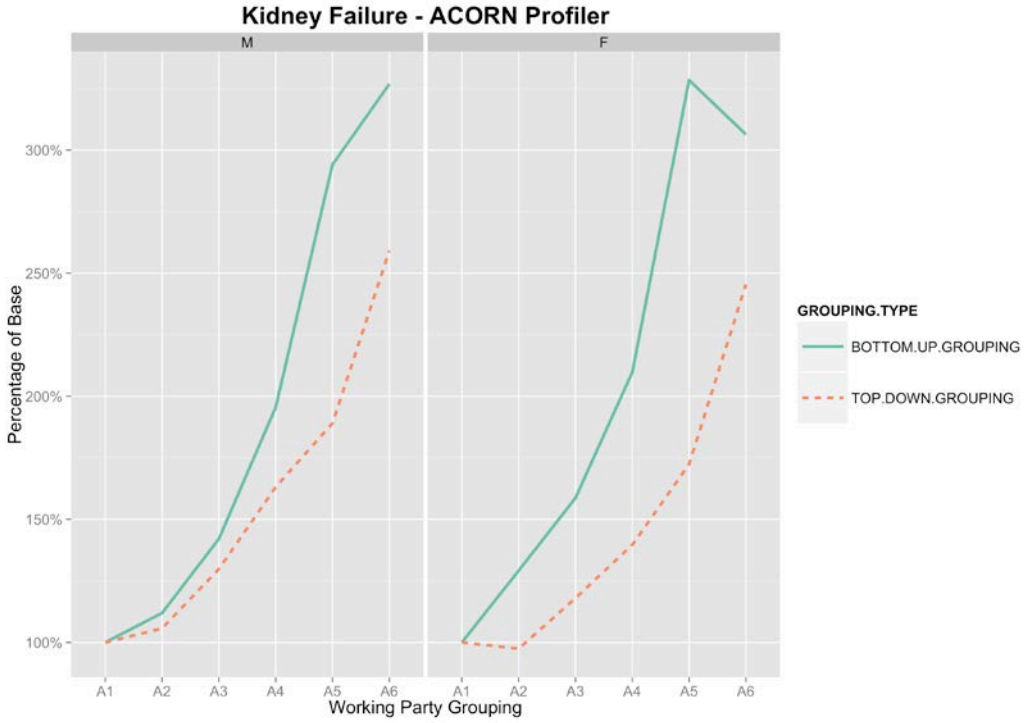
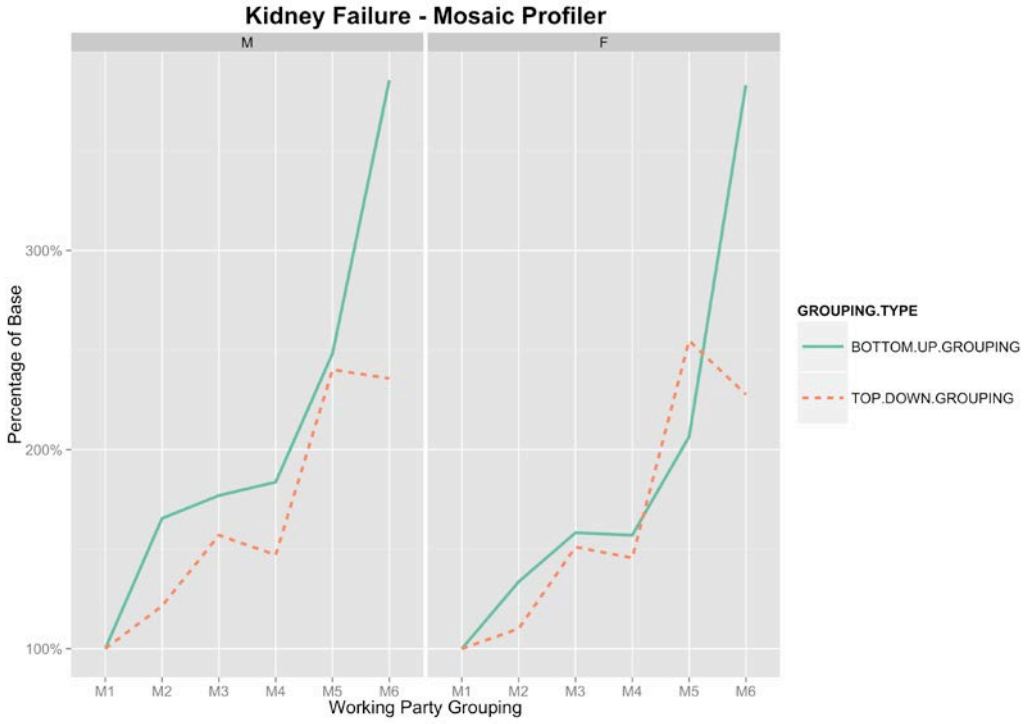
60-79

6.22.6. Geodemographic Analysis

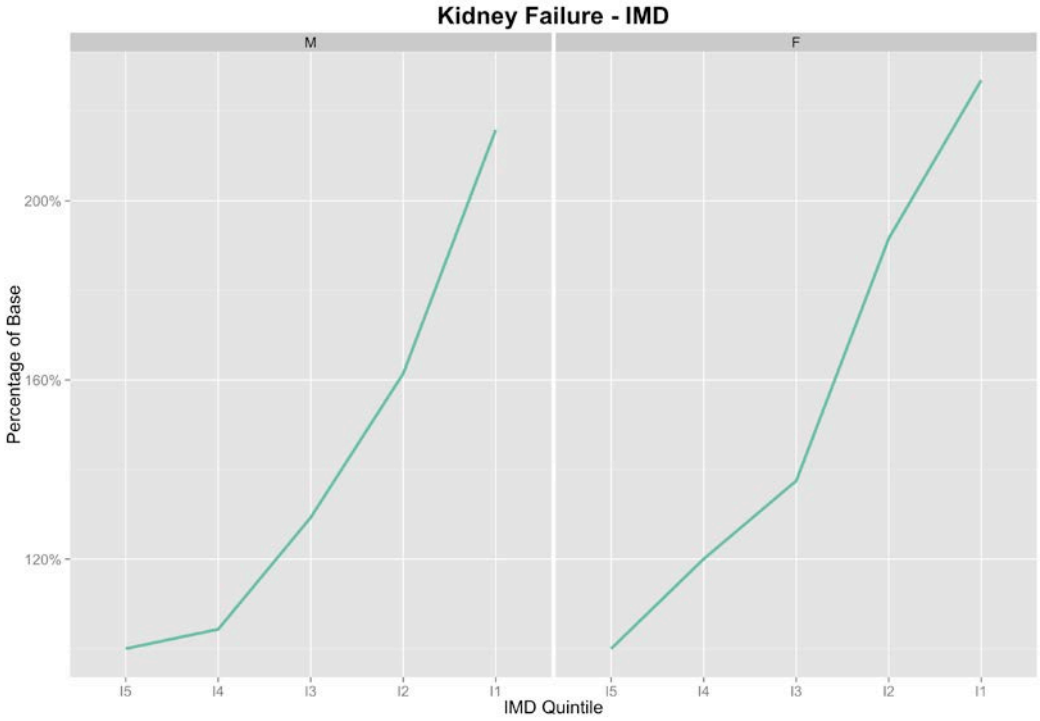
6.22.6.1. ACORN



6.22.6.2. Mosaic



6.22.6.3. Index of Multiple Deprivation



6.23. Liver failure

6.23.1. What is it?

Liver failure occurs when large parts of the liver become damaged beyond repair and the liver is no longer able to function. The liver is an important organ, which carries out several of the body's vital functions such as helping with digestion and clearing toxins. This definition covers liver failure at an advanced stage. This type of liver failure must lead to permanent jaundice (yellow discolouration of the skin), ascites (build-up of fluid in the abdomen), and encephalopathy (brain disease or damage).

Liver failure is a life-threatening condition that demands urgent medical care. Most often, liver failure occurs gradually and over many years. However, a more rare condition known as acute liver failure occurs rapidly (in as little as 48 hours) and can be difficult to detect initially.

The diagnosis of acute liver failure is based on physical exam, laboratory findings, patient history, and past medical history to establish mental status changes, coagulopathy, rapidity of onset, and absence of known prior liver disease respectively.

The most common causes of chronic liver failure include:

- Hepatitis B;
- Hepatitis C;
- Long term alcohol consumption;
- Cirrhosis;
- Hemochromatosis (an inherited disorder that causes the body to absorb and store too much iron);
- Malnutrition.

The causes of acute liver failure, when the liver fails rapidly, however, are often different. These include:

- Acetaminophen (Tylenol) overdose;
- Viruses including hepatitis A, B, and C (especially in children);
- Reactions to certain prescription and herbal medications.

Chronic liver failure usually occurs in the context of cirrhosis, itself potentially the result of many possible causes, such as excessive alcohol intake, hepatitis B or C, autoimmune, hereditary and metabolic causes (such as iron or copper overload, Steatohepatitis or non-alcoholic fatty liver disease).

This definition does not cover liver disease that is secondary to alcohol or drug abuse.

6.23.2. Symptoms and Treatment

Liver failure may be initially difficult to diagnose. Early symptoms include nausea, loss of appetite, fatigue, and diarrhoea. However, as liver failure progresses, the symptoms become more serious, requiring urgent care. These symptoms include:

- Jaundice;
- Bleeding easily;
- Swollen abdomen;
- Mental disorientation or confusion (known as hepatic encephalopathy);
- Sleepiness;
- Coma.

If detected early enough, acute liver failure caused by an overdose of acetaminophen can sometimes be treated and its effects reversed. Likewise, if a virus causes liver failure, supportive care can be given at a hospital to treat the symptoms until the virus runs its course. In these cases, the liver will sometimes recover on its own.

For liver failure that is the result of long-term deterioration, the initial treatment goal may be to save whatever part of the liver is still functioning. If this is not possible, then a liver transplant is required. Fortunately, liver transplant is a common procedure that is often successful.

6.23.3. Insurance Industry Definitions

Liver failure does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market. However, common examples of current definitions used by insurers are as set out below.

A few examples of the current definitions used by insurers are as follows:

Liver failure – of advanced stage

Liver failure due to cirrhosis and resulting in all of the following:

- Permanent jaundice
- Ascites
- Encephalopathy

For the above definition, the following is not covered:

- Liver disease secondary to alcohol or drug abuse

Liver failure

A **definite diagnosis, by a Consultant Physician, of irreversible end stage** liver failure due to cirrhosis resulting in all of the following:

- permanent jaundice;
- ascites; and
- encephalopathy.

For the above definition, the following is not covered:

- liver failure secondary to alcohol or drug abuse.

Liver failure

A definite diagnosis, by a Consultant Physician or other appropriately qualified medical professional, of irreversible end stage liver failure due to cirrhosis resulting in all of the following:

- permanent jaundice;
- ascites; and
- encephalopathy.

For the above definition, the following is not covered:

- liver failure secondary to alcohol or drug abuse.

There are further small variations in the definitions where “chronic liver failure” or “advanced stage liver failure” are used to reflect severity of liver failure.

Liver failure

Advanced stage liver failure due to cirrhosis and resulting in all of the following:

- permanent jaundice
- ascites
- encephalopathy

For the above definition, the following isn't covered:

- Liver disease secondary to alcohol or drug abuse.

The following example does not specify the exclusion of “liver disease secondary to alcohol or drug abuse”.

Liver failure

Chronic liver failure due to cirrhosis and resulting in all of the following:

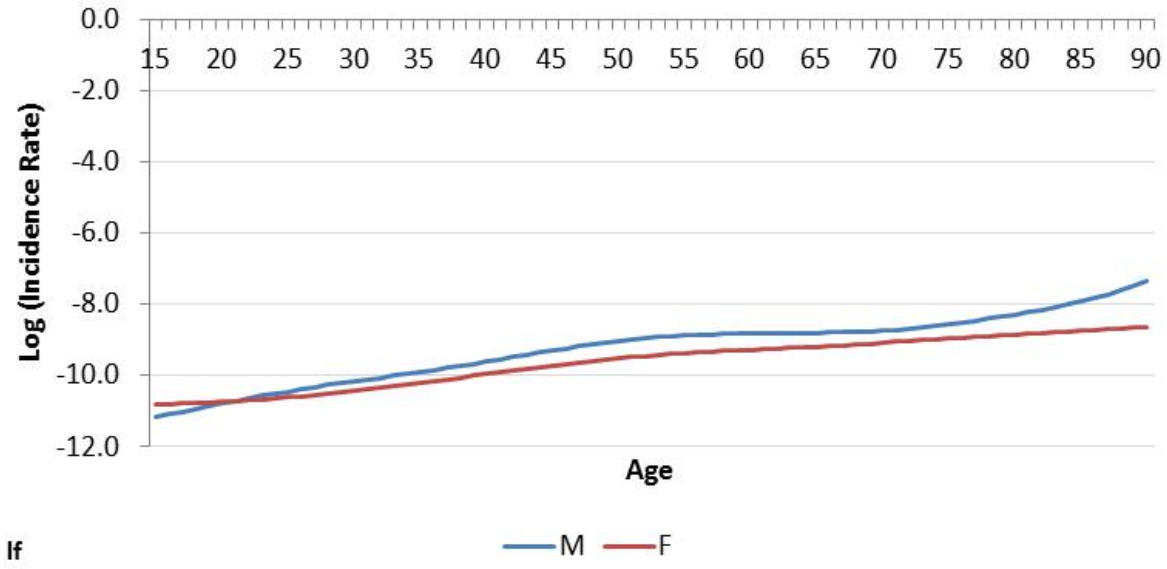
- permanent jaundice
- ascites
- encephalopathy

6.23.4. Derived Incidence Rates

Liver failure rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2.

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.49	1.60	3.16	0.38	0.99	1.99
Adjustment for Overlap	-19.5%	-32.6%	-60.2%	-17.7%	-32.1%	-52.9%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.40	1.11	1.66	0.31	0.70	1.12
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.40	1.11	1.66	0.31	0.70	1.12
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	1.0%	2.4%	0.9%	1.2%	1.8%	1.0%
Addition for Accelerated Rates Ix - kxqx	0.29	0.32	0.06	0.25	0.27	-0.08

If



If

In line with expectations, the incidence rate of liver failure increases with age. With the exception of the very youngest ages, incidence rates are higher for males than for females. The rate of increase in incidence with age is also more pronounced for males with a significant elevation in male incidence rates at the eldest ages.

6.24. Loss of Hands or Feet

6.24.1. What is it?

It is the loss of part of an arm or leg as a result of circulation problems from atherosclerosis or diabetes resulting in amputation; traumatic injuries; cancer and birth defects.

6.24.2. Symptoms and Treatment

The symptoms include:

- A body part that has been completely or partially cut off
- Bleeding depending on the location and nature of the injury or loss
- Pain though the degree is not related to the severity of the injury
- Crushed body tissue
- Phantom pain which is the feeling of pain in the missing limb.
- Grief and emotional distress
- Skin problems

The treatment includes early emergency and critical care management, new surgical techniques to minimise the loss of body parts, new prosthetic designs and management post amputation. Other treatment may include new limb replantation. The treatment also includes a patient care programme covering rehabilitation and psychological support.

6.24.3. Risk Factors

The risk factors are:

- Health conditions such as diabetes
- Natural disasters that can cause traumatic amputations
- Motor vehicle accidents
- Factory, farm or power tools accidents
- Genetic factors

6.24.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Loss of limbs (1999/2002)

Permanent physical severance of any combination of 2 or more limbs above the [elbow/wrist] or the [knee/ankle] joint.

Loss of Hands or Feet – *permanent physical severance* (2006/2011)

Permanent physical severance of any combination of 2 or more hands or feet at or above the wrist or ankle joints.

The above definition has remained unchanged for coverage provided by the companies. Historically, the definition was the same as that is currently in use.

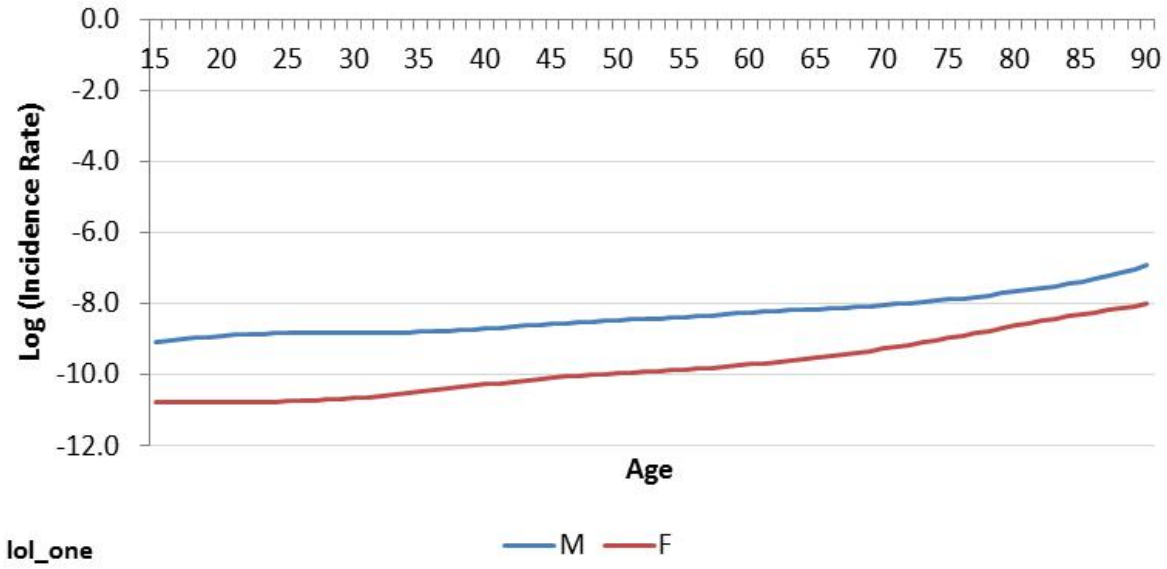
6.24.5. Derived Incidence Rates

6.24.5.1. Loss of One Limb

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.56	2.43	5.12	0.29	0.64	1.81
Adjustment for Overlap	-4.7%	-18.1%	-52.3%	-14.4%	-30.1%	-55.6%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	1.49	2.07	3.20	0.25	0.46	0.95
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	1.49	2.07	3.19	0.25	0.46	0.95
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	1.49	2.07	3.20	0.25	0.46	0.95

lol_one



It can be seen that the male and female rates follow a similar pattern, with male rates being constantly above the female equivalent.

In CIBT02 operation codes were selected that related to the amputation of an arm or leg. It was noted that no information was found on multiple amputations and, consequently, the raw counts will overstate the incidence of a definition requiring a combination of 2 or more severances. In order to allow for this a subjective severity factor of 25% was applied.

Furthermore in order to allow for the overlap with TPD, the authors allowed for an overlap of 15% up to age 17 rising linearly to 75% for ages 47 and above. Again it was recognised that this is highly subjective.

6.24.5.2. Loss of Two Limbs Results

Using our dataset we have been able to analyse the incidence of two or more limbs. We believe that loss of two limbs is genuinely rare and the rates obtained confirm this.

Rates are illustrated below for male. Note that due to the low nature of the rates the graduation is somewhat spurious. We therefore set the rates to zero for CIBT08 and rely upon the one limb rates discussed in Section 6.24.5.1.

Similar comments apply to the female results, although these are not shown for the sake of brevity.

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.00	-0.01	0.01
Adjustment for Overlap	-56.8%	-48.7%	-34.8%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.00	-0.01	0.01
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.00	-0.01	0.01
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.00	-0.01	0.01

lol_two _ M

6.25. Loss of Speech

6.25.1. What is it?

For the medical definition, loss of speech can refer to a number of different types of speech impediment. Some of these may be permanent and some may refer to disrupted speech and some may not be complete loss of speech. Many terms have been used to describe various speech disorders, for example:

- Aphasia: Difficulty in using and understanding language, although thought and hearing processes remain intact. This is also known as dysphasia.
- Dysarthria: Articulation problems resulting from weak muscles in the throat and mouth. Results in slurred speech and poor breath control when speaking.
- Aphonia: Loss of voice
- Dysphonia: An impairment to the voice (e.g. chronic hoarseness).
- Dyspraxia: Difficulty in co-ordinating muscles to produce speech.
- Dyslexia: Difficulty with reading, spelling and written language.

The medical definition of loss of speech is wider than the insurance industry definition.

For the insurance industry definition the loss of speech is when a person is not able to talk again.

Loss of speech has a variety of causes, some of which are listed below:

- Damage to or removal of vocal cords because of a tumour, inflammation or a serious injury;
- Problems with the brain or nerves that control the facial muscles, larynx, and vocal cords necessary for speech e.g. as a result of stroke or neurological disease;
- Congenital anatomical abnormalities.

6.25.2. Symptoms and Treatment

Symptoms have been described above within the different types of speech impediment. Most people who lose their speech are still able to communicate, although they may need a communication aid.

Speech therapy is a common treatment for communication impairments but if the loss of speech is irreversible, the patient may need to learn sign languages.

6.25.3. Risk Factors

Risk factors can be social, environmental and / or literacy in nature depending on types of impairments. Loss of speech may also be due to conditions present at birth, increasing age and / or is generally triggered by other conditions such as stroke.

6.25.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Loss of speech (1999/2002)

Total permanent and irreversible loss of the ability to speak as a result of physical injury or disease.

Loss of speech – permanent and irreversible (2006/2011)

Total permanent and irreversible loss of the ability to speak as a result of physical injury or disease.

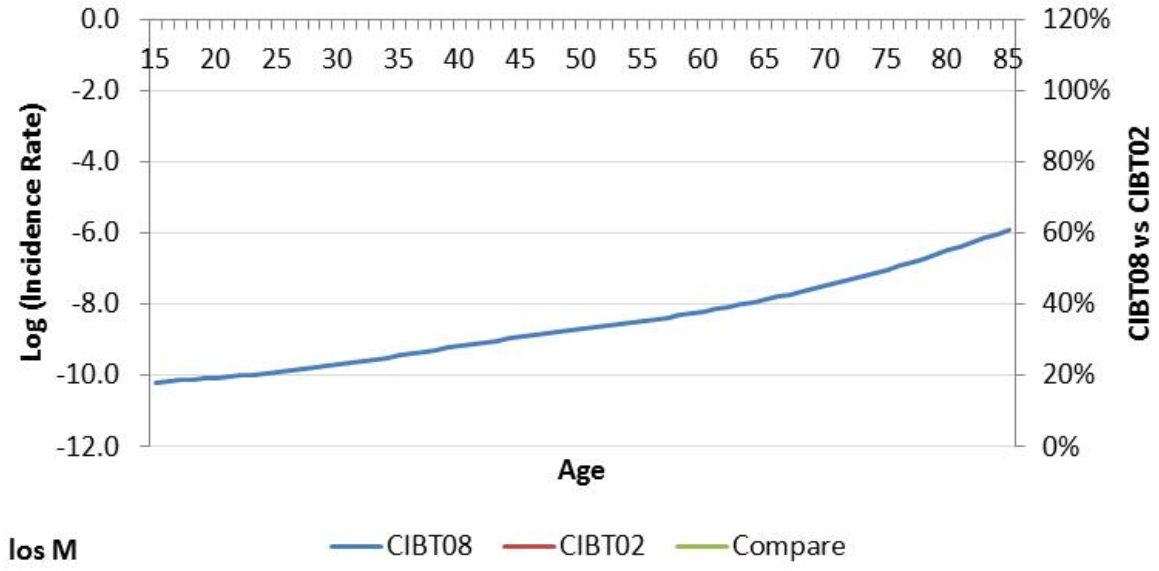
6.25.5. Derived Incidence Rates

6.25.5.1. Male Rates

For CIBT02 all Rates set to zero as residual incidence after removing overlap with other CIs is believed to be negligible in context of overall CI rates. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.89	2.89	11.22
Adjustment for Overlap	-27.7%	-43.3%	-63.1%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.65	1.67	5.63
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.65	1.67	5.62
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.65	1.67	5.63

los _ M



6.25.5.2. Female Rates

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.12	2.45	8.31
Adjustment for Overlap	-23.1%	-40.9%	-59.1%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.86	1.49	4.08
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.86	1.49	4.07
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.86	1.49	4.08

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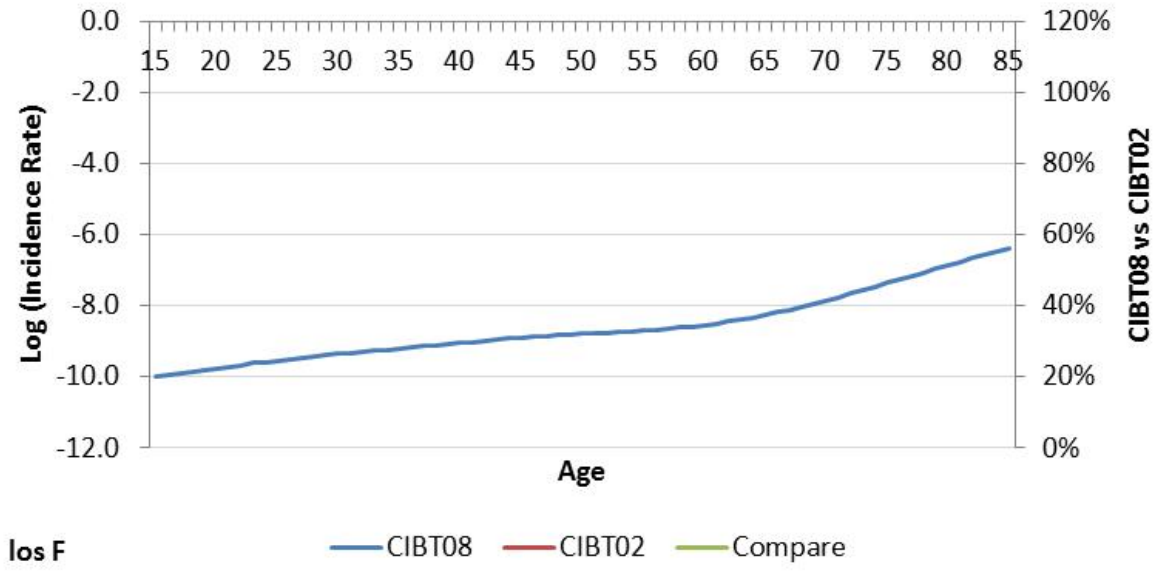


6.26. Low Grade Prostate Cancer

6.26.1. What is it?

Prostate cancer is a malignancy of one of the major male sex glands. Along with the testicles and the seminal vesicles, the prostate secretes the fluid that makes up semen. The prostate is about the size of a walnut and lies just behind the urinary bladder. A tumour in the prostate interferes with proper control of the bladder and normal sexual functioning.

With regards to Critical Illness, prostate cancer is covered under the cancer benefit category unless it is discovered early at an early stage and is of “low grade”.

6.26.1.1. Low Grade

The "stage" of a cancer indicates how far the cancer has spread, whilst the "grade" of a cancer describes the cellular characteristics and provides information relating to how aggressive the rate of tumour growth is likely to be. The grading system used with regards to prostate cancer is the "Gleason" grading system/score.

Gleason grade or score

The Gleason grading examines the tumour cells microscopically of 2 separate samples with regards to the frequency of malignant cells that can be recognised. The results from each sample are added together that form the "Gleason score".

A Gleason score of between 2 and 6 indicates that the tumour is a "low grade" prostate cancer. Therefore, it is likely to grow very slowly. A Gleason score of 7 is an intermediate grade that will grow at a moderate rate. A Gleason score of 8 to 10 is a high grade cancer that is likely to grow more quickly.

Prostate cancer staging

Prostate cancer is staged using the TNM system. This is used all over the world. It separately assesses the tumour (T), lymph nodes (N) and secondary cancer (metastases - M).

T1 tumours are too small to be seen on scans or felt during examination of the prostate - they may have been discovered by needle biopsy, after finding a raised "Prostatic Specific Antigen” (PSA) level in the blood (a test that can be increased with prostate cancer).

T2 tumours are completely inside the prostate gland and are divided into 3 groups:

- T2a - The tumour is in only half of one of the lobes of the prostate gland;
- T2b - The tumour is in more than half of one of the lobes; and
- T2c - The tumour is in both lobes but is still inside the prostate gland.

Currently, tumours that are staged as T2 can claim benefit under the current cancer definition within a CI plan. The "Low grade prostate cancer" cover offers a reduced benefit amount in keeping with the better prognosis it has with less likelihood of it impacting on either quality of life or life expectancy.

6.26.2. Symptoms and Treatment

Often the first symptom of prostate cancer is difficulty in urinating. However, because a very common, non-cancerous condition of the prostate, benign prostatic hyperplasia (BPH) also causes the same problem, difficulty in urination is not necessarily due to cancer. The symptoms of growths in the prostate are similar whether they are non-cancerous (benign) or cancerous (malignant). The symptoms include:

- Having to rush to the toilet to pass urine;
- Difficulty passing urine;
- Passing urine more often than usual, especially at night;
- Pain when passing urine;
- Blood in the urine or semen.

The last two symptoms - pain and bleeding are very rare in prostate cancer. They are more often a symptom of non-cancerous prostate conditions.

Cancer of the prostate gland often grows slowly, especially in older men. Symptoms may be mild and occur over many years. Sometimes the first symptoms are from prostate cancer cells which have spread to bones but this is not common. Cancer cells in the bone may cause pain in the back, hips, pelvis and other bony areas.

Cancer that has spread to other areas of the body is called metastatic or secondary prostate cancer.

Other symptoms that may occur are weight loss, particularly in elderly men, and difficulty getting an erection.

In 2008, the National Institute for Health and Clinical Excellence (NICE) issued guidance about treatment options for prostate cancer.

If the cancer is in its early stages and not causing symptoms, it is often the case that no treatment is given in order to wait to see if any symptoms of progressive cancer develop and this is particularly the case for “low grade” tumours. If the cancer does develop, then other treatments to control prostate cancer are usually used. Tumours may be actively surveyed with the aim to avoid unnecessary treatment of harmless cancers, while still providing timely treatment for men who need it with around half to two-thirds of men with low-risk prostate cancer never needing any treatment.

Active surveillance involves regular PSA testing and biopsies to ensure any signs of progression are found as early as possible. Other treatments involve surgery and radiotherapy.

Surgery in the form of “radical prostatectomy” is the surgical removal of the prostate gland and is a treatment option for curing localised prostate cancer and locally-advanced prostate cancer; this is particularly where there are other unfavourable features such as a high Gleason score.

Radiotherapy involves using radiation to kill cancerous cells. This treatment is another option for curing localised prostate cancer and locally-advanced prostate cancer. Brachytherapy is a form of radiotherapy where a number of tiny radioactive seeds are surgically implanted into the tumour. It is also known as internal radiotherapy.

This method has the advantage of delivering a high dose of radiation to the prostate, while minimising damage to other tissues. However, the risk of sexual dysfunction and urinary problems is the same as with radiotherapy, although the risk of bowel problems is slightly lower.

6.26.3. Risk Factors

Prostate cancer is now the most common cancer in men in the UK (not counting non melanoma skin cancer). More than 40,800 men are diagnosed each year. That is almost a quarter of all cancers diagnosed in men.

Prostate cancer is quite rare in men under 50. More than half of all cases are diagnosed in

men over 70. Age is the most significant risk factor of all for prostate cancer. With increasing age, the greater the risk there is. In old age, up to 8 out of 10 men have prostate cancer cells in the prostate.

In the UK, about 1 in 9 men will get prostate cancer at some point in their lives. However, many will have prostate cancer during their lifetime and because of the indolent nature of the condition will never be aware they have it.

Other main factors that influence the risk:

- Family history of prostate cancer;
- Race - black (of African or Caribbean ancestry) – prostate cancer is more common in black and mixed race men than white or Asian men. African men are more likely to develop prostate cancer compared with Caucasian men and are nearly 2.5 times as likely to die from the disease.

6.26.4. Insurance Industry Definitions

Low grade prostate cancer does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market. However, common examples of current definitions used by insurers are as follows.

Low grade prostate cancer

The undergoing of treatment on the advice of your hospital consultant following the diagnosis of a malignant tumour of the prostate positively diagnosed and histologically classified as having a Gleason score between 2 and 6 inclusive and having progressed to clinical TNM classification T1N0M0.

For the above definition, the following are not covered:

- Prostatic intraepithelial neoplasia (PIN).
- Observation or surveillance.
- Surgical biopsy.

Low grade prostate cancer - additional cover condition

Tumours of the prostate histologically classified as having a Gleason score between 2 and 6 inclusive, provided the tumour has progressed to **at least** clinical TNM classification T1N0M0; **and treatment included the complete removal of the prostate or external beam or interstitial implant radiotherapy.**

For the above definition the following are not covered:

- **treatment of the tumour by any procedures other than complete removal of the prostate, external beam or interstitial implant radiotherapy. For example: cases treated with cryotherapy, other less radical treatment such as transurethral resection of the prostate,**
- **'experimental' treatments, or**
- **hormone therapy.**

Low grade prostate cancer

We will pay the lower of 25 percent of the benefit and £25,000 if a member is diagnosed with a tumour of the prostate histologically classified as having a Gleason score between 2 and 6 inclusive provided the tumour has progressed to at least clinical TNM classification T1N0M0; and treatment included the complete removal of the prostate or external beam or interstitial implant radiotherapy.

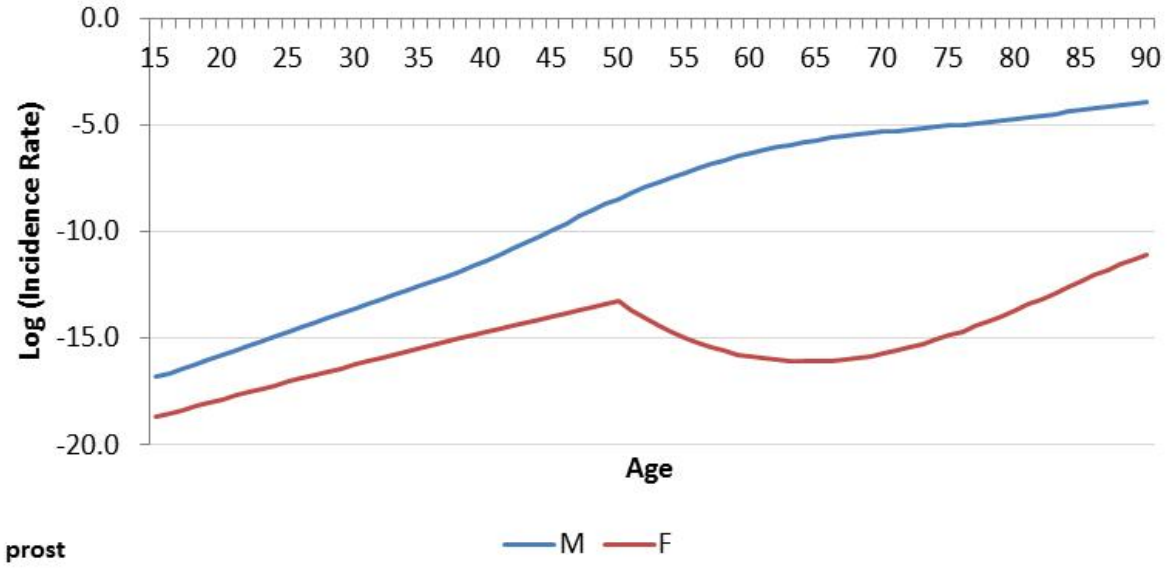
For clarity, cases treated with cryotherapy, other less radical treatment (e.g. transurethral resection of the prostate), experimental treatments or hormone therapy are not included

6.26.5. Derived Incidence Rates

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.03	3.97	44.75	0.00	0.02	0.14
Adjustment for Overlap	-12.3%	-18.7%	-27.2%	-25.6%	-33.1%	-98.7%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.02	3.69	43.68	0.00	0.01	0.00
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.02	3.69	43.58	0.00	0.01	0.00
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.6%	3.6%	1.5%	1.6%	1.5%
Addition for Accelerated Rates Ix - kxqx	0.02	3.36	34.59	-0.06	-0.36	-2.28

prost



As expected, prostate cancer incidence rates increase materially by age. We have left the female curve in this chart but the rates are trivial as we would expect.

6.27. Major Organ Transplant

6.27.1. What is it?

Organ transplantation is the moving of an organ from one body to another or from a donor site to another location on the patient's own body, for the purpose of replacing the recipient's damaged or absent organ. Organs and/or tissues that are transplanted within the same person's body are called autografts. Transplants that are recently performed between two subjects of the same species are called allografts. Allografts can either be from a living or cadaveric source.

For the purpose of critical illness cover, it is only the organs that are considered to be “major” in the sense that they are more likely to impact upon quality of life and mortality that are covered that are heart, lung, liver, pancreas, kidney or bone marrow.

Other organs that can be transplanted include intestines and thymus. In addition, there can also be transplantation of other damaged tissues such as bones, tendons (both referred to as musculoskeletal grafts), cornea, skin, nerves and veins. However, these do not currently form part of any cover being provided.

In the UK, the kidneys are the most commonly transplanted organs, followed by the liver and heart that have similar numbers. The cornea and musculoskeletal grafts are the most commonly transplanted tissues; these outnumber organ transplants by more than tenfold.

A bone marrow transplant, more commonly known as a stem cell transplant, replaces damaged bone marrow with healthy bone marrow stem cells. Usually this damage is resulting from cancer or leukaemia.

6.27.2. Symptoms and Treatment

Symptoms requiring major organ transplantation include the irreversible failure of the heart, both lungs, liver, both kidneys or bone marrow. Transplantation must be medically necessary.

The greatest risk in this procedure is rejection by the recipient's tissues of the transplanted organ.

The emerging field of regenerative medicine is allowing scientists and engineers to create organs to be re-grown from the patient's own cells (stem cells, or cells extracted from the failing organs).

6.27.3. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Major Organ Transplant (1999/2002)

The actual undergoing as a recipient of a transplant of, or inclusion on an official UK waiting list for a transplant of a heart, liver, lung, pancreas or bone marrow.

Major Organ Transplant (2006/ 2011)

The undergoing as a recipient of a transplant of bone marrow or of a complete heart, kidney, liver, lung or pancreas, or inclusion on an official UK waiting list for such a procedure.

For the above definition, the following is not covered:

- Transplant of any other organs, parts of organs, tissues or cells.

Some companies now extend cover to in order to gain ABI+ status by also covering partial transplants of lungs and liver rather than complete organ transplants.

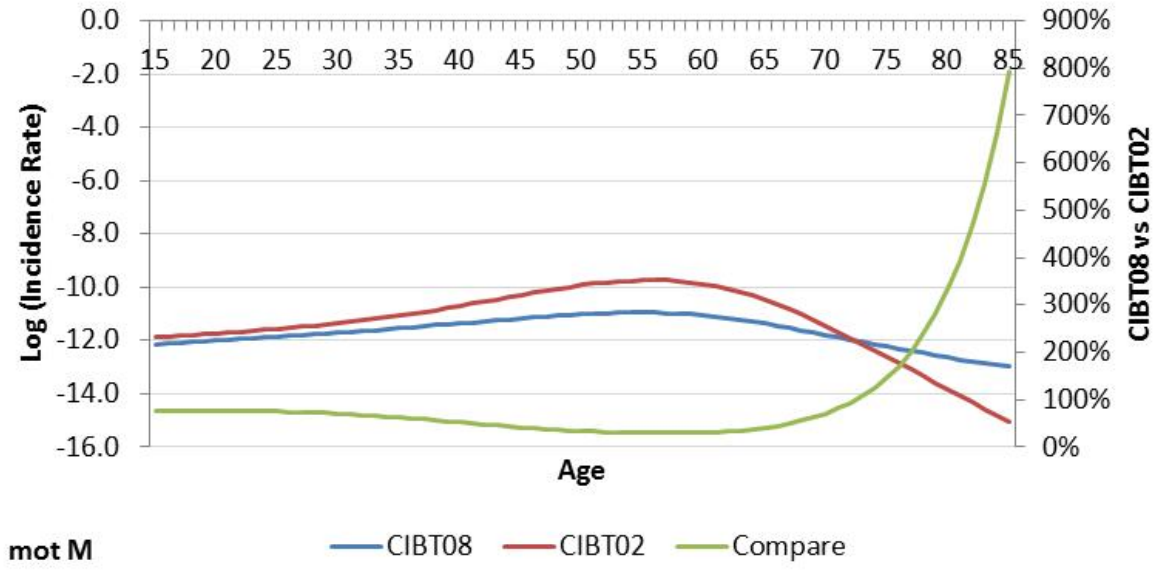
6.27.4. Derived Incidence Rates

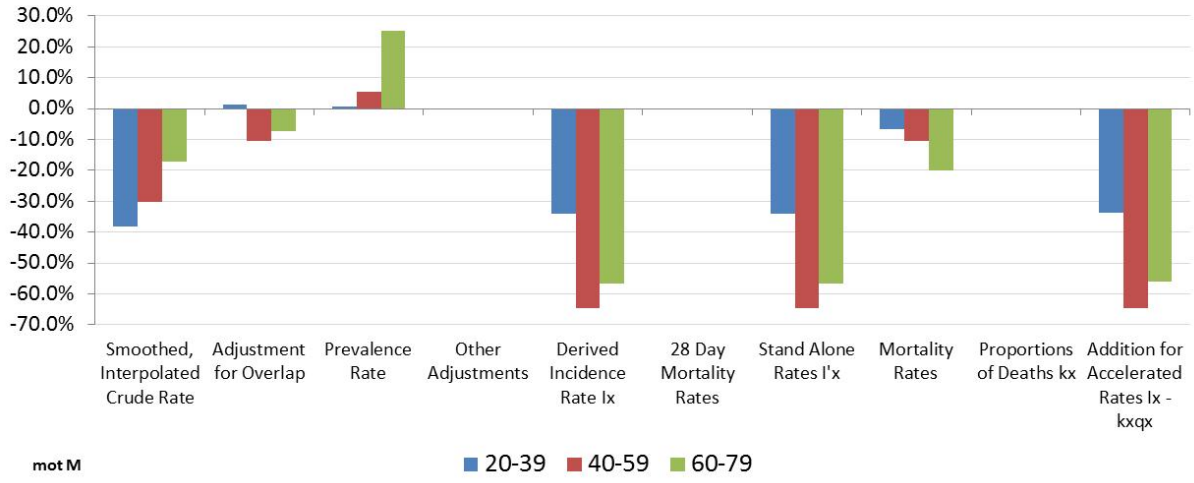
6.27.4.1. Male Rates

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.69	1.48	1.04
Adjustment for Overlap	-87.3%	-89.7%	-91.0%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.09	0.16	0.10
28 Day Mortality Rates	-4.0%	-4.0%	-4.0%
Stand Alone Rates I'x	0.08	0.15	0.10
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.08	0.15	0.10

mot _ M

The shape of the CIBT08 rates profile for MOT is quite different to the corresponding CIBT02 profile. CIBT08 rates are much flatter for males than suggested by CIBT02. Incidence rates are also lower below age 70. The peak in rates at around age 55 is still evident in CIBT08 but to a much lesser degree. As expected, there is a gradual decrease in incidence rates beyond age 55 to 60 as operations are carried out less frequently at older ages. However this reduction is much less severe than suggested by CIBT02. This is due to the increased prevalence rate per CIBT08.

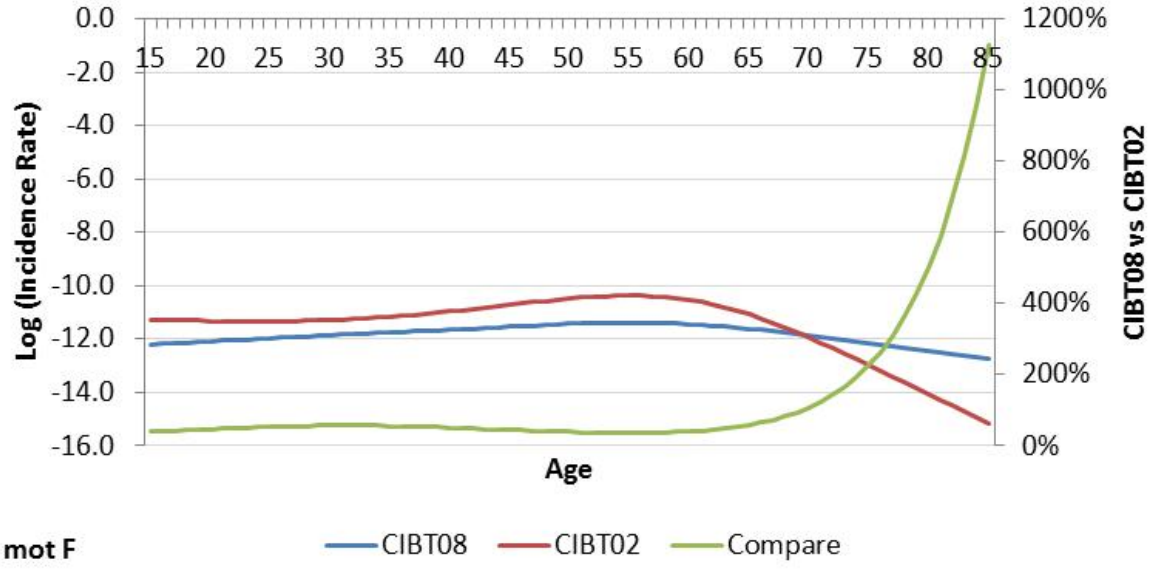




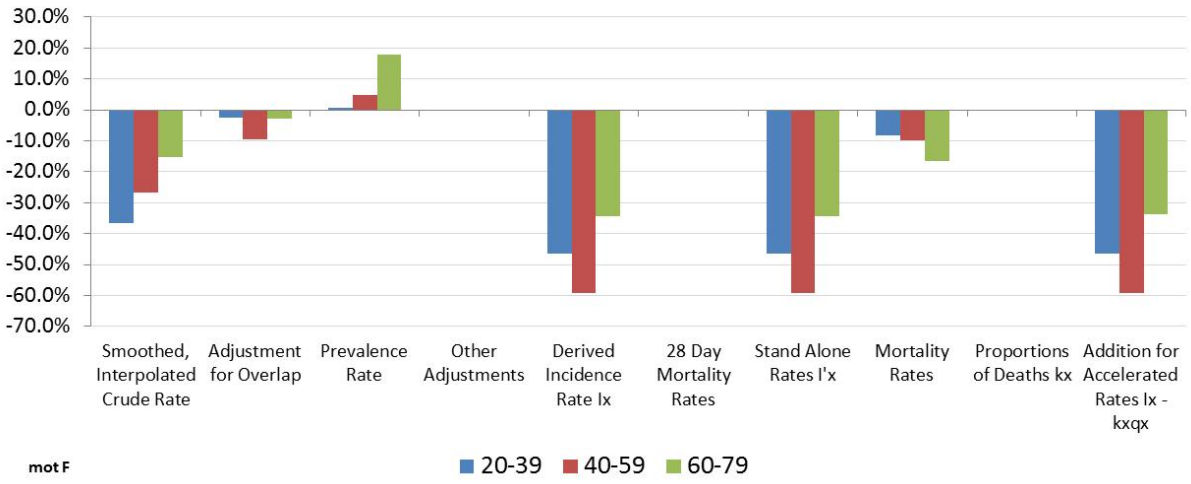
6.27.4.2. Female Rates

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.49	0.94	0.52
Adjustment for Overlap	-84.7%	-89.1%	-81.8%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.07	0.11	0.08
28 Day Mortality Rates	-4.0%	-4.0%	-4.0%
Stand Alone Rates I'x	0.07	0.10	0.08
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.1%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.07	0.10	0.08

mot _ F

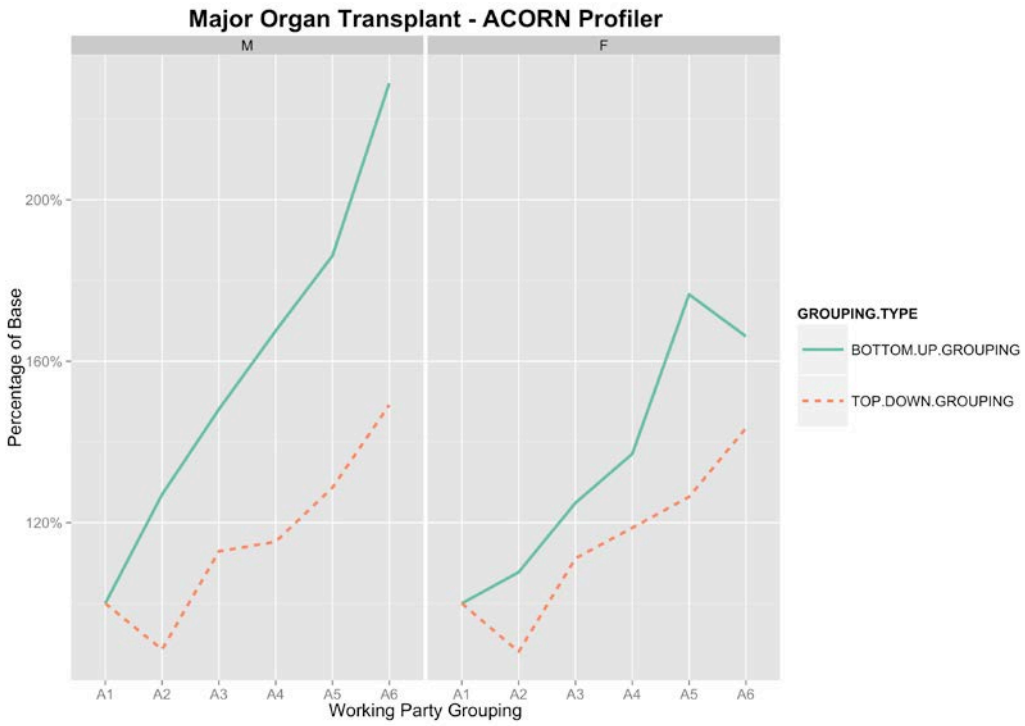


CIBT08 also exhibits much flatter MOT incidence rates for females than CIBT02. CIBT08 incidence rates are lower below age 70 with the gap less pronounced than for males however. The peak in rates at around age 55 is also still evident but again, to a much lesser degree. As expected, there is a gradual decrease in incidence rates beyond age 55 to 60 as operations are carried out less frequently at older ages. However this reduction is much less severe than suggested by CIBT02. This is due to the increased prevalence rate per CIBT08.

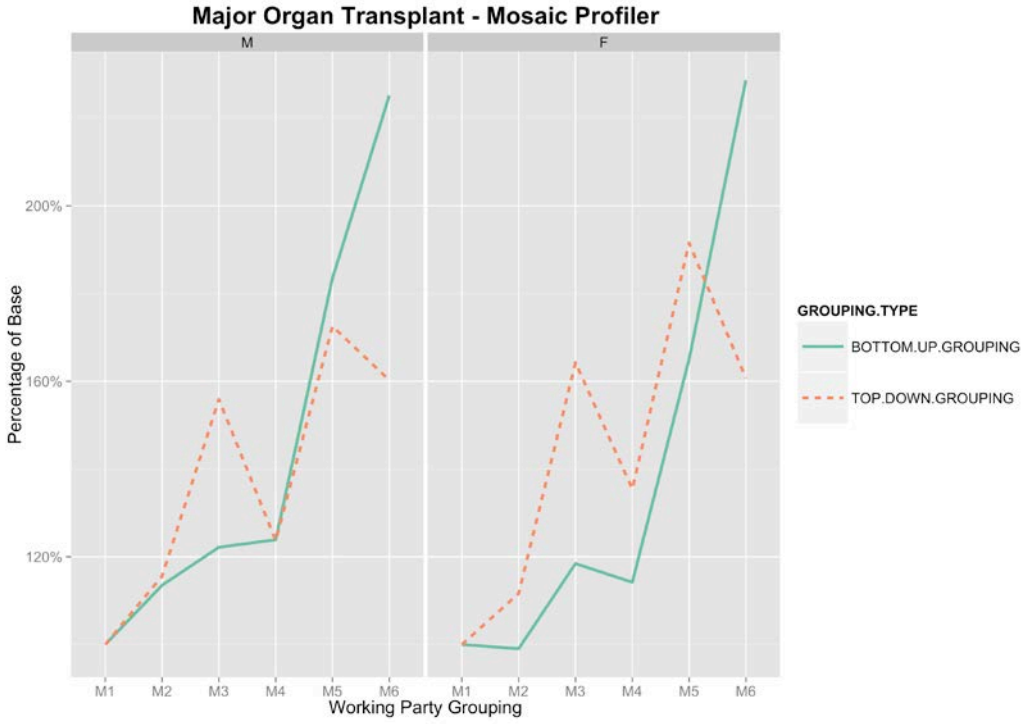


6.27.5. Geodemographic Analysis

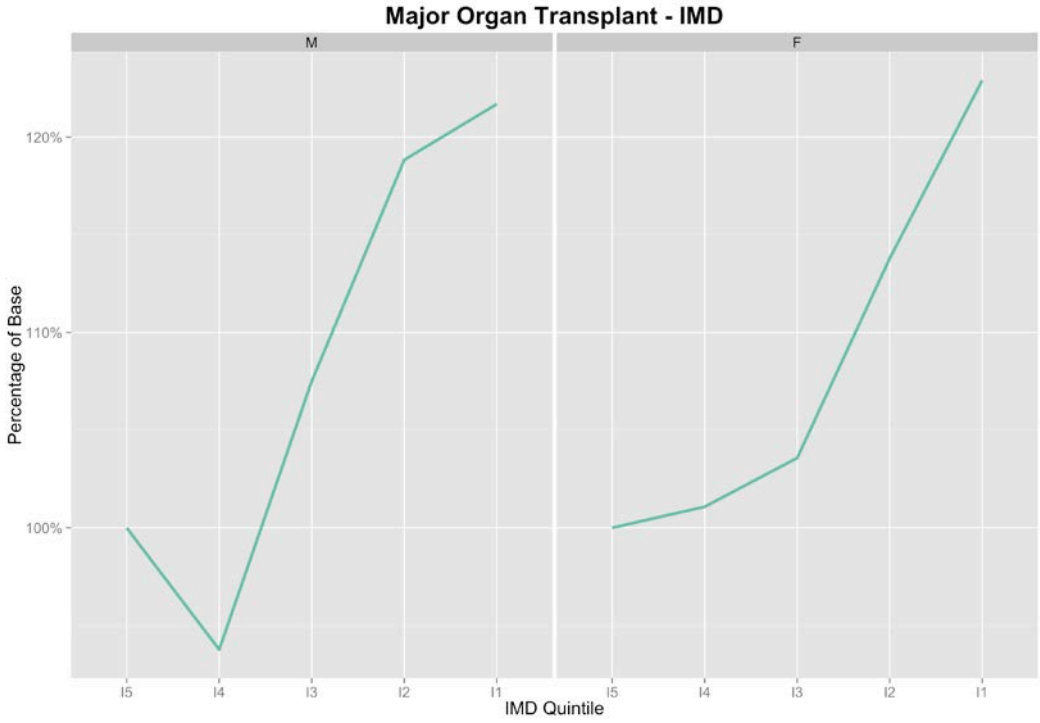
6.27.5.1. ACORN



6.27.5.2. Mosaic



6.27.5.3. Index of Multiple Deprivation



6.28. Motor Neurone Disease

6.28.1. What is it?

The motor neurone diseases (MND) are a group of neurological disorders that selectively affect motor neurons, the cells that control voluntary muscle activity including speaking, walking, swallowing, and general movement of the body. They are generally progressive in nature, and cause increasingly debilitating disability and, eventually, death.

There are four main types of MND, each affecting people in different ways. There can be a great deal of overlap between all of these forms, so, while it is useful to separate the various types of the disease, in practice it is not always possible to be so specific for classification purposes.

Brief descriptions of the recognised types of MND follow:

Amyotrophic lateral sclerosis (ALS)

This is the most common form, with both upper (part of the body) and lower motor neurone involvement. This form of the disease is characterised by weakness and wasting in the limbs. Average life expectancy is from two to five years from onset of symptoms.

Progressive bulbar palsy (PBP)

Affects about a quarter of people diagnosed, and involves both the upper and lower motor neurones. Life expectancy is between six months and three years from onset of symptoms.

Progressive muscular atrophy (PMA)

Affects only a small proportion of people, mainly causing damage to the lower motor neurones. Most people live for more than five years.

Primary lateral sclerosis (PLS)

A rare form of MND involving the upper motor neurones only, causing mainly weakness in the lower limbs, although some people may experience clumsiness in the hands or speech problems. Life span could essentially be normal, although it may be life-limiting, depending on whether it remains as pure PLS or develops into ALS.

6.28.2. Symptoms and Treatment

Early symptoms of MND are often mild. They may include:

- stumbling due to weakness of the leg muscles;
- difficulty holding objects due to weakness of the hand muscles;
- slurring of speech or swallowing difficulties due to weakness of the tongue and throat muscles;
- cramps and muscle twitching (fasciculation).

For most people with MND, the intellect and memory are not significantly affected, nor are the senses of sight, hearing, taste, smell and touch.

Fronto-temporal cognitive change (a type of dementia) has been associated with MND with one in five experiencing these changes. Neuro-psychological studies also suggest that approximately one in three people with MND may have very mild changes in cognitive skills and processes.

The effects of motor neurone disease - initial symptoms, rate and pattern of progression, and survival time after diagnosis vary significantly from person to person.

Although there is no cure for MND, treatments can help both to slow the disease and also to improve symptoms.

6.28.3. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Motor Neurone Disease (1999/2002)

Confirmation by a consultant neurologist of a definite diagnosis of Motor Neurone disease.

Motor Neurone Disease [before age x] – resulting in permanent symptoms (2006/2011)

A definite diagnosis of motor neurone disease [before age x] by a Consultant Neurologist. There must be permanent clinical impairment of motor function.

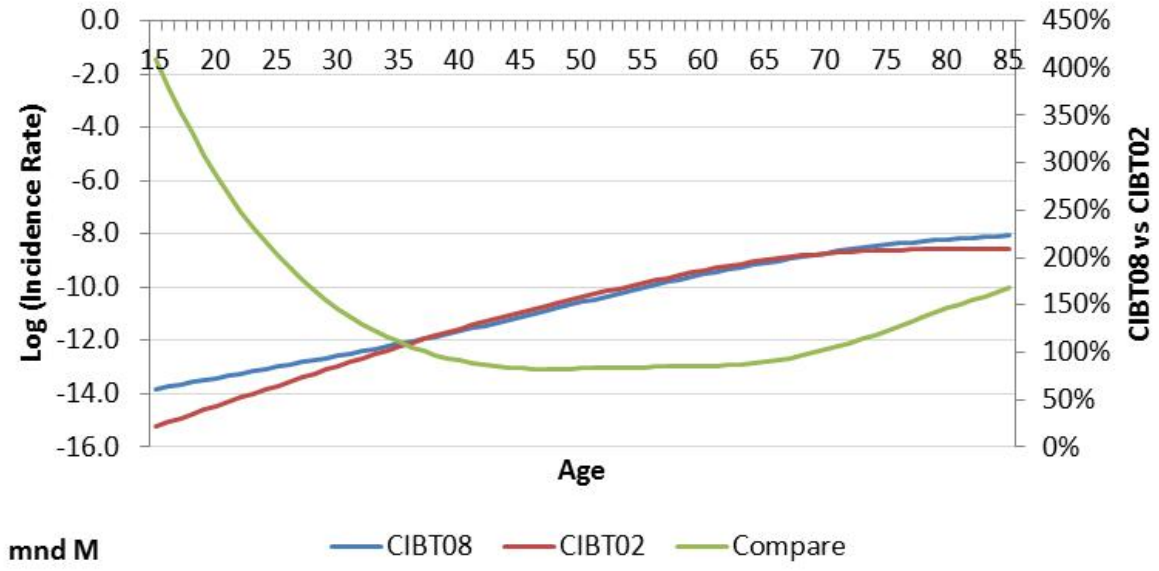
6.28.4. Derived Incidence Rates

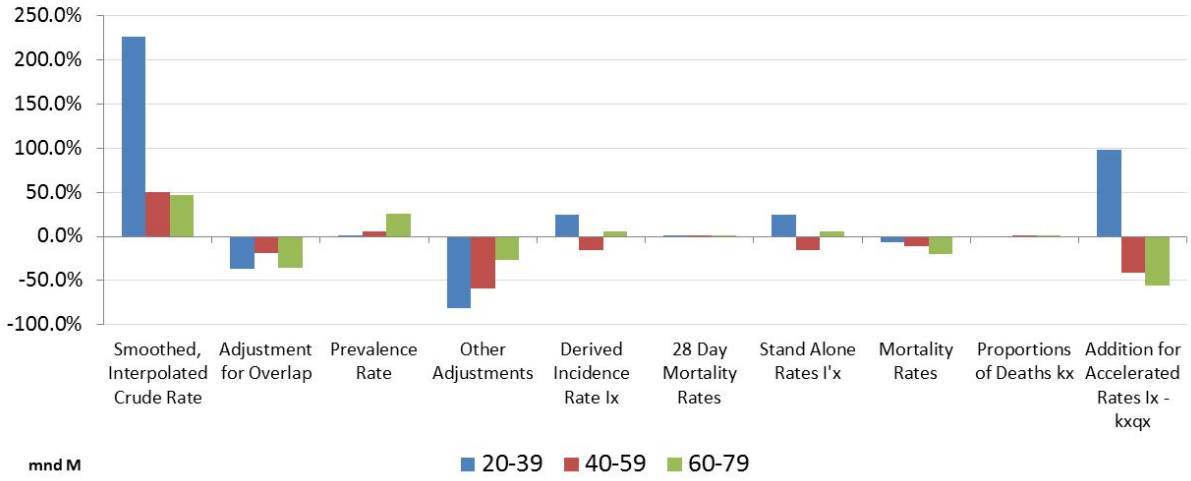
6.28.4.1. Male Rates

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.06	0.32	1.68
Adjustment for Overlap	-36.2%	-18.7%	-35.3%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.04	0.28	1.48
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.04	0.28	1.47
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.1%	0.5%	0.7%
Addition for Accelerated Rates Ix - kxqx	0.03	0.07	0.11

mnd _ M



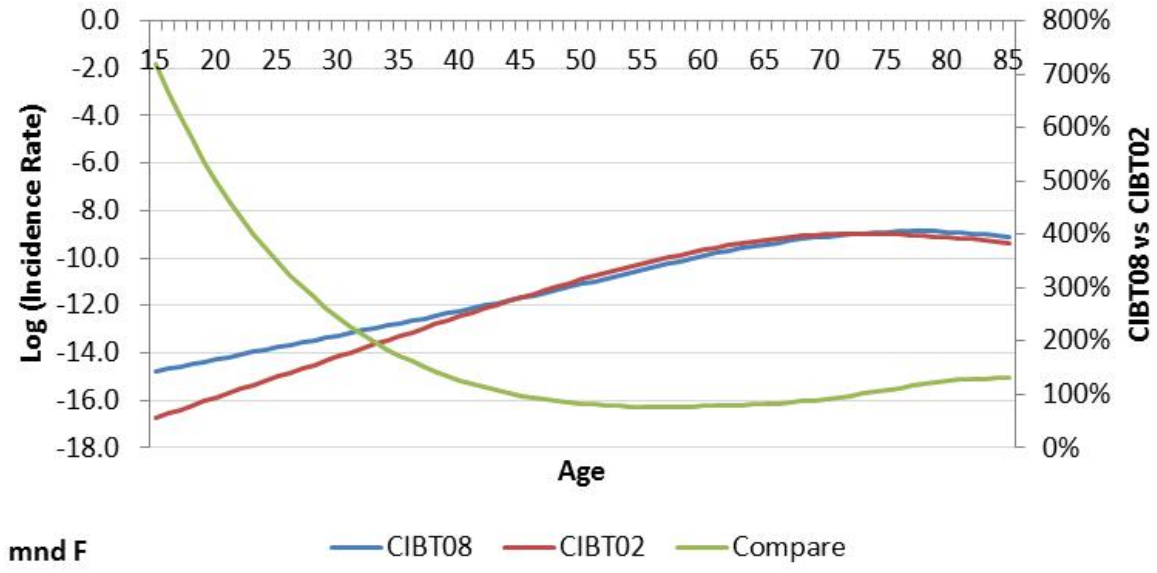


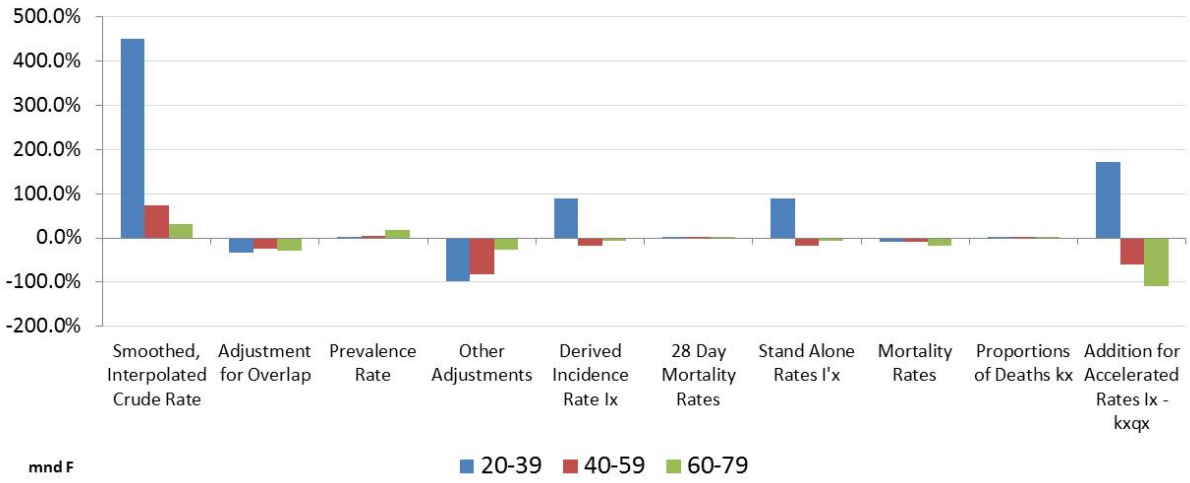
Between ages 35 and 65, the CIBT08 derived MND incidence rates for males are very similar to those derived per CIBT02. CIBT08 rates have a flatter profile than CIBT02 across all age bands however. CIBT08 displays a significant elevation in incidence rates at either side of these ages with the increase most pronounced at the very youngest and eldest ages. Although there is a marked increase in rates at younger ages, incidence of MND below age 40 remains very rare however.

6.28.4.2. Female Rates

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.03	0.21	1.15
Adjustment for Overlap	-34.2%	-24.7%	-28.9%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.02	0.17	0.98
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.02	0.17	0.98
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.1%	0.5%	0.7%
Addition for Accelerated Rates Ix - kxqx	0.01	0.04	-0.02

mnd _ F





CIBT08 derived MND incidence rates for females are also much higher at the younger ages than those derived per CIBT02, although again, incidence below age 40 remains very rare. Unlike the male results however, the increased Female incidence per CIBT08 is much less pronounced at the older ages.

6.29. Multiple Sclerosis

6.29.1. What is it?

Multiple sclerosis (“MS”) is an autoimmune disease that affects the brain and spinal cord (central nervous system).

MS is caused by damage to the myelin sheath, the protective covering that surrounds nerve cells. When this nerve covering is damaged, nerve signals slow down or stop.

The nerve damage is caused by inflammation. Inflammation occurs when the body's own immune cells attack the nervous system. This can occur along any area of the brain, optic nerve and spinal cord.

6.29.2. Symptoms and Treatment

Patients with multiple sclerosis can have symptoms in many parts of the body because nerves in any part of the brain or spinal cord can be damaged.

The common symptoms include: fatigue, loss of balance, muscle spasm, numbness, loss of movement in arms or legs, constipation, incontinence, vision loss, rapid eye movements, reduced attention span, memory loss, dizziness, hearing loss and speech impairment.

It is common for the disease to return (relapse). However, the disease may continue to get worse without periods of remission. People who have a form of MS called relapsing-remitting may have a history of at least two attacks, separated by a period of reduced or no symptoms.

The goal of treatment is to control symptoms and help the patient maintain a normal quality of life as there is no cure for MS. Hence it includes:

- Medications used to slow the progression of MS taken on a long-term basis;
- Medicines to reduce muscle spasms;
- Cholinergic medications to reduce urinary problems;
- Physical therapy, speech therapy, occupational therapy, and support groups;
- A planned exercise program early in the course of the disorder;
- A healthy lifestyle, with good nutrition and enough rest and relaxation;
- Making changes around the home to prevent falls.

6.29.3. Risk Factors

Multiple sclerosis (MS) affects women more than men.

It is unknown what exactly causes MS. The key risk factors are:

- Exposure to virus;
- Genetic defect with a family history of MS;
- Adverse environmental factors can exacerbate existing conditions;
- Fever, hot baths and sun exposure can trigger attacks.

6.29.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Multiple Sclerosis (1999/2002)

A definite diagnosis of Multiple Sclerosis by a Consultant Neurologist which satisfies all of the following criteria:

- There must be current impairment of motor or sensory function, which must have persisted for a continuous period of at least six months.
- The diagnosis must be confirmed by diagnostic techniques current at the time of claim.

Multiple Sclerosis - with persisting symptoms (2006/2011)

A definite diagnosis of Multiple Sclerosis by a Consultant Neurologist. There must be current clinical impairment of motor or sensory function, which must have persisted for a continuous period of at least 6 months.

Some companies have enhanced this definition to attain ABI+ status by allowing the period of persistent symptoms of 3 months in order to claim

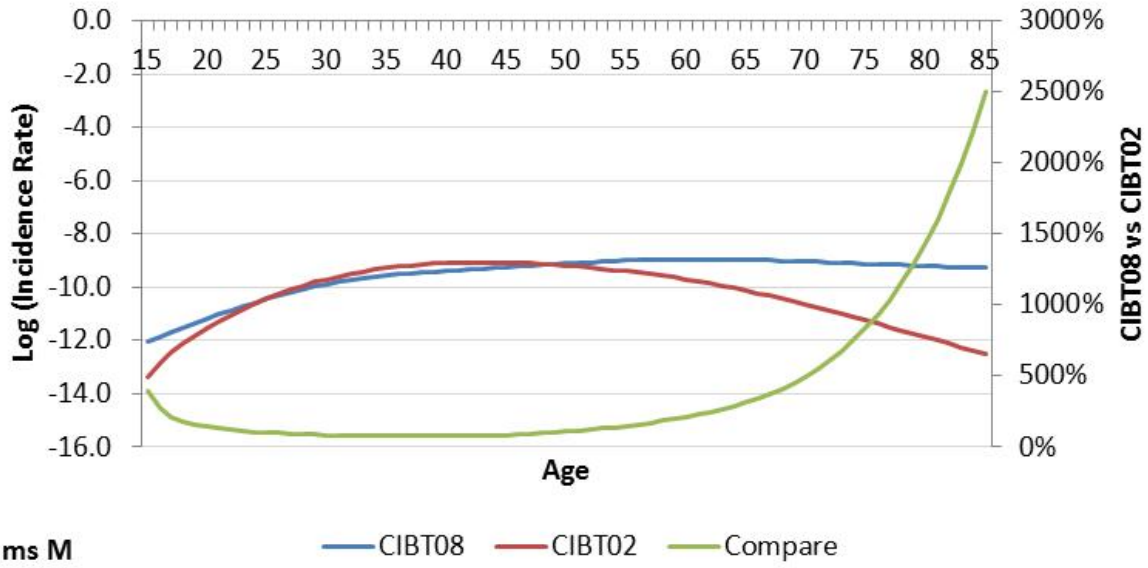
6.29.5. Derived Incidence Rates

6.29.5.1. Male Results

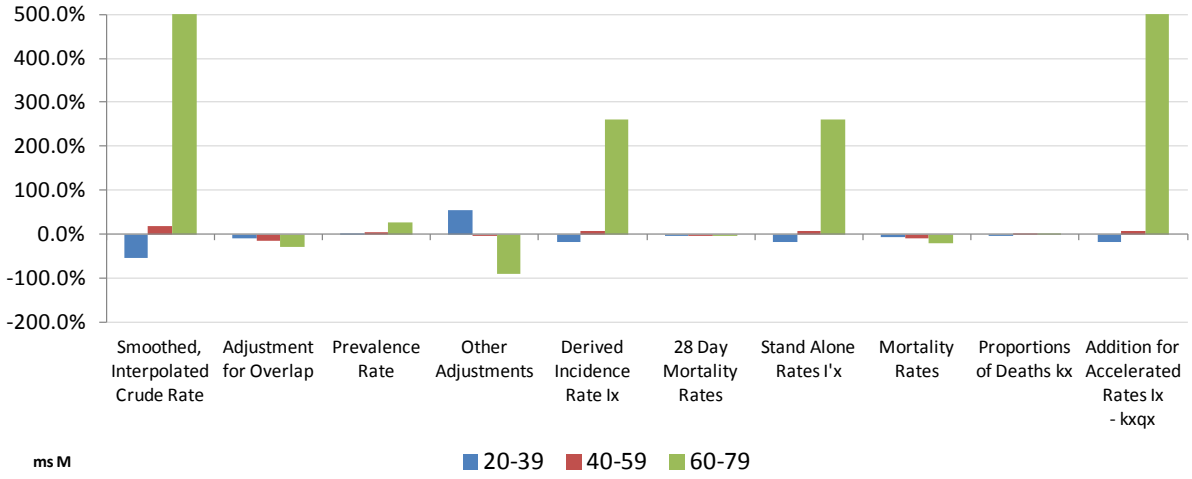
The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.54	1.17	1.26
Adjustment for Overlap	-9.3%	-14.0%	-28.8%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.49	1.07	1.20
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.49	1.07	1.20
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.2%	0.5%	0.2%
Addition for Accelerated Rates Ix - kxqx	0.47	0.87	0.81

ms _ M



The CIBT08 rates profile is different in shape to that of CIBT02 rates. The CIBT08 standalone rates are similar to the CIBT02 rates up until age 50 but significantly higher thereafter. CIBT02 did not rely on the HES data for their work citing concerns over multiple admissions during the relapsing/remitting stage of the disease and also concerns around many sufferers never being hospitalised. Our first ever algorithm should, to some extent, deal with the multiple admissions concern.

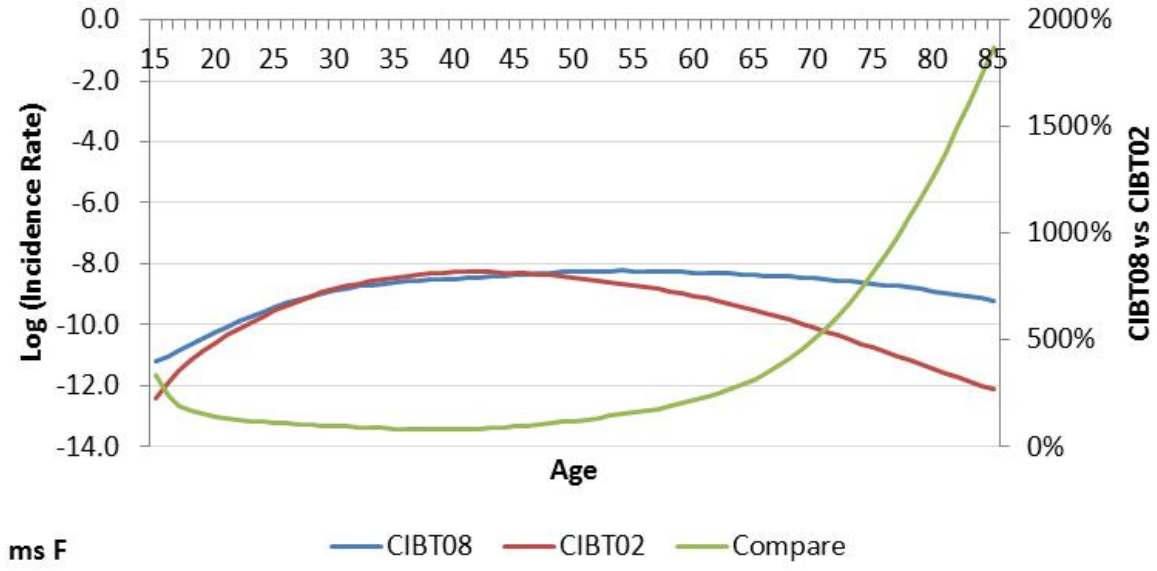


The age range on this graph has been restricted because the change in the "Additional for Accelerated Rates" for the age range 60-79 shows a change of approximately 51000%. This is a function of CIBT02 having a small negative value for the "Addition for Accelerated CI".

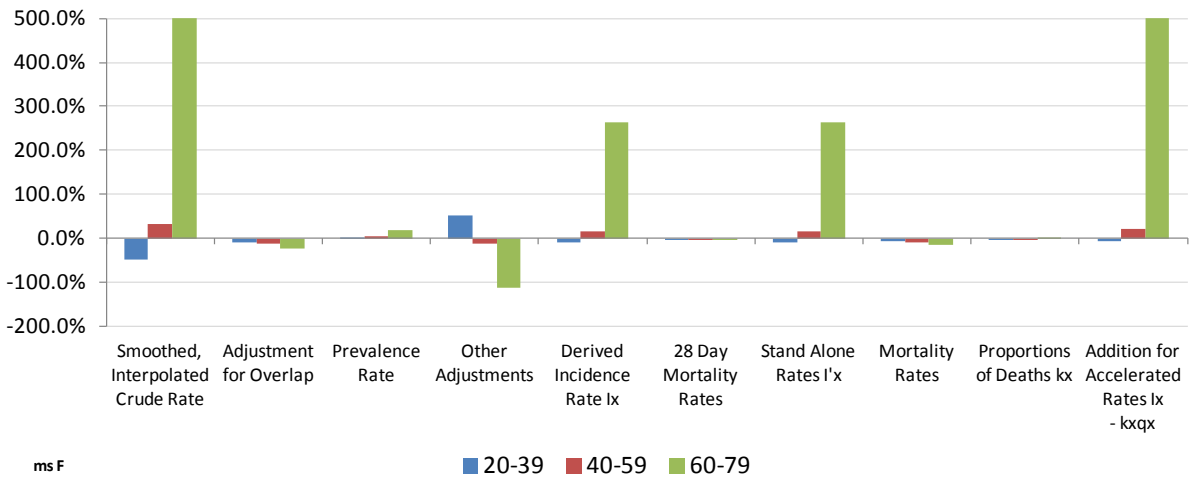
6.29.5.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.39	2.67	2.28
Adjustment for Overlap	-8.8%	-12.9%	-23.4%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	1.29	2.45	2.12
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	1.29	2.44	2.12
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.5%	1.3%	0.6%
Addition for Accelerated Rates Ix - kxqx	1.26	2.13	1.49

ms _ F



As per the males, The CIBT08 rates profile is different in shape to that of CIBT02 rates. The CIBT08 standalone rates are similar to the CIBT02 rates up until age 50 but significantly higher thereafter.



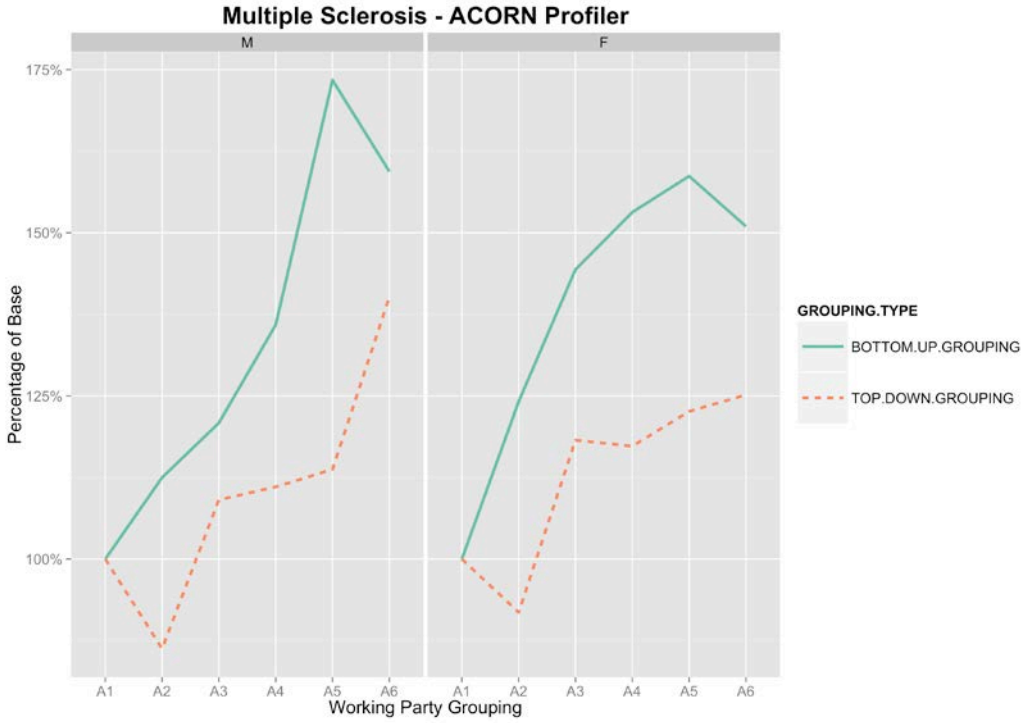
As per the males, the age range on this graph has been restricted because the change in the "Additional for Accelerated Rates" for the age range 60-79 shows a change of approximately 8000%. This is a function of CIBT02 having a small negative value for the "Addition for Accelerated CI".

We have compared our crude MS rates to benchmark rates. The benchmark rate was sourced from an analysis of incidence and prevalence of MS in the UK (1990-2010) using GPRD data[22]. By comparison to the rates presented in the paper our rates for the lowest and middle age bands do not appear unreasonable but our rate at the oldest age appears too high. For example, in age band 60-69 the incidence rates in the paper are 0.52 (compared to 1.26) for Males and 1.04 (compared to 2.28) for females, per 10,000. At age 40-49 the rates are 0.902 (compared to 1.17) and 2.4 (compared to 2.67) respectively which are much closer to our results. We note that the paper itself uses HES data to identify additional cases not included in GPRD but the differences do not account for size of difference we see at the oldest ages.

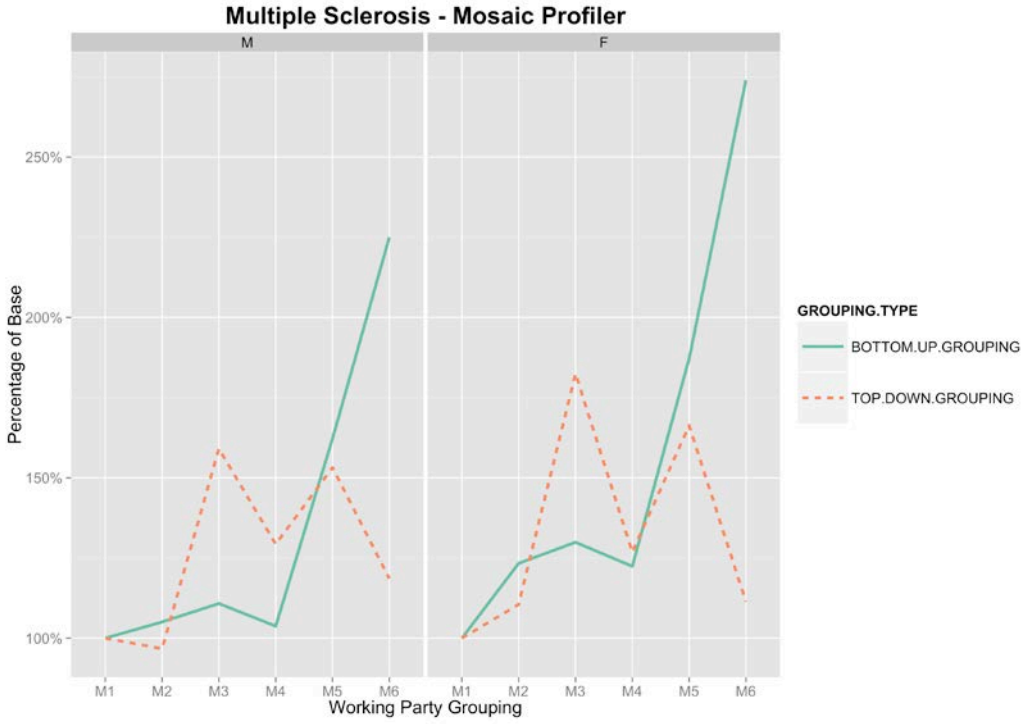
[22] Incidence and prevalence of multiple sclerosis in the UK 1990-2010: a descriptive study in the General Practice Research Database. Mackenzie et al.
http://www.ncbi.nlm.nih.gov/pubmed/24052635

6.29.6. Geodemographic Analysis

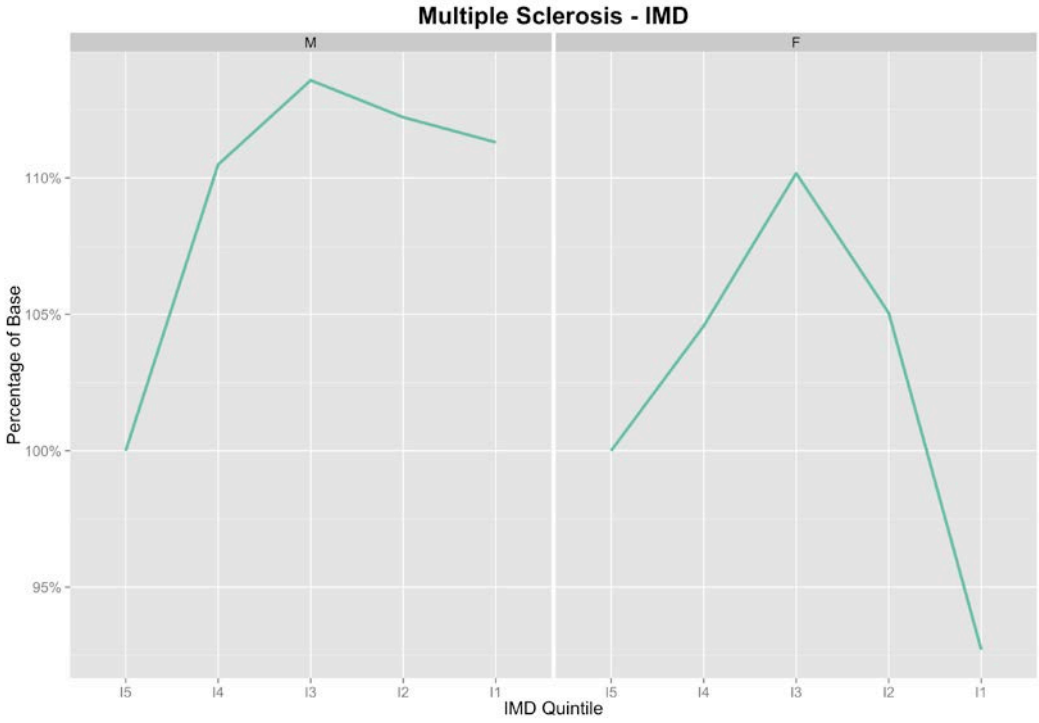
6.29.6.1. ACORN



6.29.6.2. Mosaic



6.29.6.3. Index of Multiple Deprivation



6.30. Multiple System Atrophy

6.30.1. What is it?

Multiple System Atrophy (MSA) is a progressive neurodegenerative disease, caused by cell loss in certain areas of the brain and spinal cord. There is no known cause for this. MSA is characterised by problems with movement, balance and automatic functions of the body such as bladder and blood pressure control.

There are 2 different types of MSA, depending on the most prominent symptoms present at the time of evaluation:

- The Parkinsonian Type (MSA-P) has primary characteristics of Parkinson's disease, such as moving slowly, stiff muscles, tremor and problems with balance and coordination;
- The Cerebellar Type (MSA-C) has primary characteristics featuring difficulty in swallowing, slurred speech, and ataxia (problems with balance and coordination).

6.30.2. Symptoms and Treatment

The initial symptoms of MSA are often difficult to distinguish from the initial symptoms of Parkinson's disease and include:

- decreased spontaneous movement, tremor, or rigid muscles;
- clumsiness, loss of balance, and frequent falls;
- slurred speech, a croaky, quivering voice, or difficulty swallowing;
- fainting or light-headedness due to orthostatic hypotension, a condition in which blood pressure drops rapidly when rising from a seated or lying down position; and
- bladder control problems, such as a sudden urge to urinate or difficulty emptying the bladder completely.

MSA is progressive. Currently, there are no treatments to delay the progress of neurodegeneration in the brain. There are ongoing treatment trials to evaluate drugs that could potentially help delay progression.

There are treatments available to help people cope with some of the more disabling symptoms of MSA.

The fainting and light-headedness from orthostatic hypotension is often treated successfully with simple interventions such as adding extra salt to the diet and avoiding heavy meals and alcohol. Some people with MSA sleep with the head of the bed tilted up or use a compression body stocking. Drinking a glass or two of water before getting out of bed in the morning can also help raise blood pressure.

Bladder control problems are treated according to the nature of the problem. Limiting fluid intake after the evening meal and taking desmopressin at night can reduce episodes of night-time bedwetting.

Difficulties with swallowing and breathing eventually require that people with MSA use an artificial feeding tube or breathing tube.

Muscle spasms and contractures usually benefit from physical therapy that builds strength and encourages people to remain mobile for as long as possible.

6.30.3. Risk Factors

The cause of MSA is unknown. Age appears to be a risk factor with most people developing the disease when older than age 40 and the mean age at onset is between ages 50 – 70.

6.30.4. Insurance Industry Definitions

Multiple system atrophy does not have a standard ABI model wording, however, many providers do cover the condition. Definitions tend to be similar with only minor variations.

An example definition currently found in the market is:

> **Multiple system atrophy – resulting in permanent symptoms**
>
> A definite diagnosis of multiple system atrophy by a consultant neurologist. There must be evidence of permanent clinical impairment of either:
>
> - motor function with associated rigidity of movement; or
> - the ability to coordinate muscle movement; or
> - bladder control and postural hypotension.

Some providers explicitly state there must be evidence of disease progression, as demonstrated in the following example:

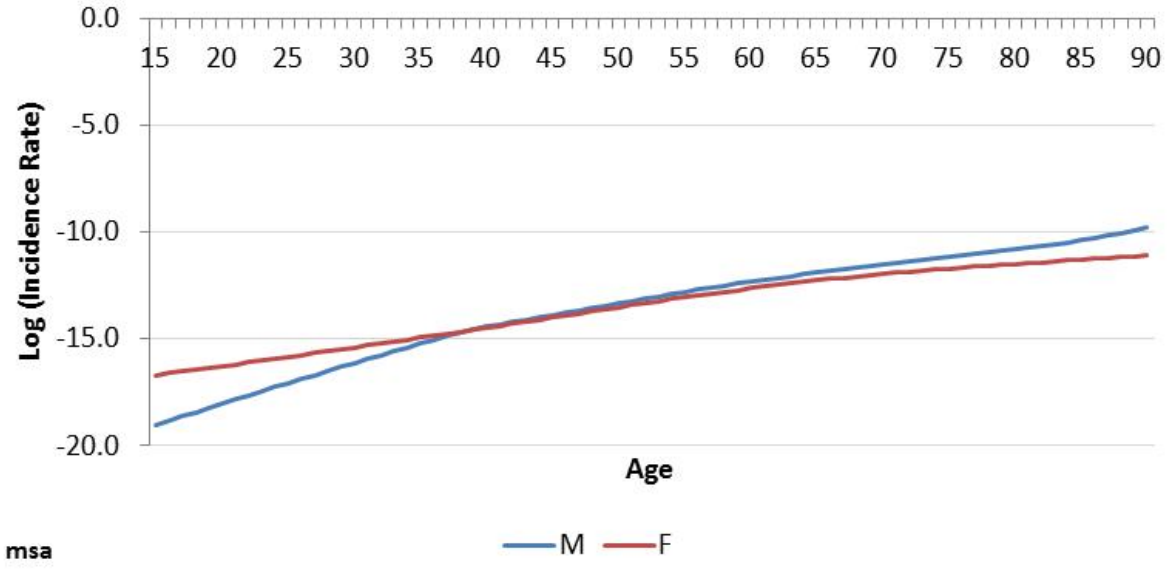
> **Multiple system atrophy – resulting in progressive and permanent symptoms**
>
> A definite diagnosis of multiple system atrophy by a consultant neurologist. There must be evidence of ***disease progression and*** permanent clinical impairment of either:
>
> - motor function with associated rigidity of movement; or
> - the ability to coordinate muscle movement; or
> - bladder control and postural hypotension.

6.30.5. Derived Incidence Rates

MSA rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The following table provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.00	0.04	0.20	0.00	0.03	0.17
Adjustment for Overlap	-0.2%	-44.8%	-66.3%	-37.7%	-58.9%	-69.4%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.00	0.02	0.09	0.00	0.01	0.06
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.00	0.02	0.09	0.00	0.01	0.06
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.1%	0.6%	0.1%	0.1%
Addition for Accelerated Rates Ix - kxqx	0.00	0.01	-0.01	-0.02	0.00	-0.01

msa



Figures from 2009[23] put the MSA incidence rate at 0.3 per 10,000 in the population aged over 50 years. We see that our crude rates in age band 60-79 of 0.20 for males and 0.17 for females are of the same order of magnitude.

Furthermore our crude rates support the fact that most patients with MSA develop the disease when older than age 40.

[23] Stefanova N, Bucke P, Duerr S, et al; Multiple system atrophy: an update. Lancet Neurol. 2009 Dec;8(12):1172-8.

6.31. Open (Structural) Heart Surgery

6.31.1. What is it?

The original purpose of this definition was to cover procedures to the heart requiring what was often referred to historically as “Open heart surgery” which indicated that median sternotomy would performed together with support during the surgery with a heart-lung by-pass machine.

However, the term “open heart” in this context is not medically defined and is open to interpretation. Therefore, it can be argued that any surgery to the heart, could be described as "open heart", particularly if access is gained via an excision to the chest wall (thoracotomy). Therefore, there has been a tendency in the market to change the heading and definitions to “structural heart surgery” or just “heart surgery – requiring median sternotomy”.

6.31.2. Symptoms and Treatment

Open heart surgery can treat a variety of diseases and conditions of the heart. The surgery is often recommended for:

- Congenital defect repair, which is done to correct a variety of heart problems that are present at birth;
- Coronary artery bypass graft (CABG). CABG provides a new route around diseased coronary arteries with healthy vessels taken from other places in the body. This is the most common heart surgery in adults;
- Heart transplant, to remove a severely damaged heart and replace it with a donor heart;
- Heart valve repair or replacement, when a heart valve does not function properly. Heart valves control the flow of blood into and out of the heart.

Other non-surgical treatments include lifestyle changes, medicines and medical procedures.

Some of these other surgical procedures are typically covered under other CI definitions. Further it is likely that congenital defects will be underwritten out. The HES data does allow us to distinguish between congenital and other cases to some extent but we have included those associated with congenital problems on the bases that some congenital conditions are not apparent until later in life. Our resulting rates may still be higher than insured experience as a result.

6.31.3. Risk Factors

Risk factors include family history of heart problems, other health problems such as diabetes or high blood pressure and age.

6.31.4. Insurance Industry Definitions

Structural/open heart surgery does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market.

There are some variations in the market as shown by the following examples which are typical of definitions used in the market:

> **Heart surgery - with surgery to divide the breastbone**
>
> The undergoing of open heart surgery requiring median sternotomy (surgery to divide the breastbone) on the advice of a Consultant Cardiologist to correct a structural abnormality of the heart.

The following example uses a different heading and wording. However, the cover being provided is the same:

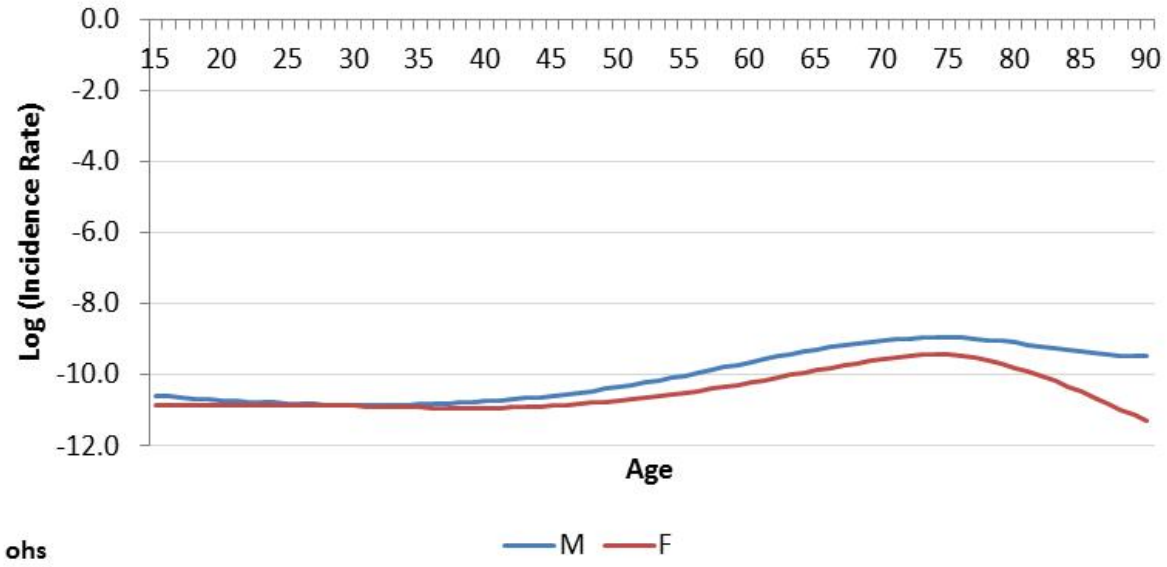
> **Open heart surgery – with surgery to divide the breastbone**
>
> The undergoing of open heart surgery requiring median sternotomy (surgery to divide the breastbone) on the advice of a Consultant Cardiologist to correct a disease or defect of the heart.
>
> For the above definition, the following are not covered:
>
> - any percutaneous, transluminal or other procedure that does not involve median sternotomy
> - investigative procedures

6.31.5. Derived Incidence Rates

Open Heart Surgery rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.27	0.60	1.92	0.25	0.37	1.19
Adjustment for Overlap	-24.8%	-45.2%	-59.4%	-26.0%	-40.7%	-56.4%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.20	0.34	1.05	0.19	0.23	0.61
28 Day Mortality Rates	-0.6%	-0.8%	-2.2%	-0.6%	-0.8%	-2.3%
Stand Alone Rates I'x	0.20	0.34	1.03	0.19	0.23	0.60
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.20	0.34	1.03	0.19	0.23	0.60

ohs



6.32. Paralysis of Limbs

6.32.1. What is it?

Paralysis is loss of muscle function for one or more muscles. Paralysis can be accompanied by a loss of feeling (sensory loss) in the affected area if there is sensory damage as well as motor damage.

Muscle is a type of tissue that enables our bodies to move. It is under the control of the nervous system, which processes messages to and from all parts of the body. Sometimes the nerve cells, or neurons, that control the muscles become diseased or injured. When that happens, a person loses the ability to move the muscles voluntarily, and that person is said to be paralysed.

Damage to the nerves of the spinal cord affects different parts of the body, depending on the amount of damage and where it occurred. Paralysis may be temporary or permanent, depending on the disease or injury. Paralysis can either be localised, where a specific section of the body, such as the face or hand, is paralysed, or it can be generalised, where a larger area of the body is affected.

There are a number of medical terms used to describe different types of paralysis. For example:

- **monoplegia** – where one limb is paralysed
- **hemiplegia** – where the arm and leg on one side of the body are paralysed
- **paraplegia** – where both legs and sometimes the pelvis and some of the lower body are paralysed
- **tetraplegia** – where both the arms and legs are paralysed (also known as quadriplegia)

6.32.2. Symptoms and Treatment

The signs and symptoms of paralysis vary. When the spinal cord is crushed, a person is immediately paralysed and loses feeling in the affected limbs. When damage to the muscles or central nervous system is caused by a progressive disease or disorder, such as muscular dystrophy or multiple sclerosis, symptoms are gradual and often start with muscle fatigue and weakness. With poliomyelitis and stroke, paralysis comes on suddenly, with little or no warning.

It is usually not possible to prevent the conditions that cause paralysis, and most of the time there is no specific treatment. Therefore, in cases of permanent paralysis treatment aims to:

- help a person live as independently as possible
- address any associated complications that arise from paralysis, such as pressure ulcers (sores that develop when the affected area of tissue is placed under too much pressure)
- address bladder and bowel problems that are secondary to paralysis
- treat spasms and complications resulting from paralysis

Mobility aids, such as wheelchairs and orthoses, can help a person with paralysis.

Manual wheelchairs are designed for people with good upper body strength. Electric wheelchairs are designed for people with poor upper body muscle strength or paralysis in all four limbs.

Steroid medications are sometimes given at the time of spinal cord injury to reduce inflammation in an attempt to limit the amount of damage to the spinal nerves. For people with paralysis who must use wheelchairs, treatment emphasises exercises and special care to avoid infections and pressure sores.

Paralysis can also cause a number of associated secondary conditions, such as urinary incontinence (an inability to control the flow of urine) and bowel incontinence (where stools leak uncontrollably from the back passage). It may also affect sexual function in both men and women.

6.32.3. Risk Factors

The four most common causes of paralysis are:

- Stroke,
- Head injury,
- Spinal cord injury and
- Multiple sclerosis.

There are also a number of less common causes, these are listed below.

- Cancer
- Cerebral palsy
- Friedreich’s ataxia
- Guillain-Barré syndrome
- Lyme disease
- Motor neurone disease
- Spina bifida
- Poliomyelitis
- Peripheral neuropathy,
- Parkinson's disease,
- Botulism

Drugs that interfere with nerve function, such as curare, can also cause paralysis. There are many known causes for paralysis, and perhaps more yet to be discovered.

6.32.4. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

> **Paralysis / Paraplegia – (1999/2002)**
>
> Total irreversible loss of muscle function or sensation to the whole of any two limbs as a result of injury or disease. The disability must be permanent and supported by appropriate neurological evidence.

> **Paralysis of limbs – total and irreversible (2006/2011)**
>
> Total and irreversible loss of muscle function to the whole of any 2 limbs.

Many companies enhance benefit in order to ABI+ status by proving cover where only one limb has been paralysed as shown in the following example:

> **Paralysis of a limb – total and irreversible (ABI+)**
>
> Total and irreversible loss of muscle function to the whole of any one limb.

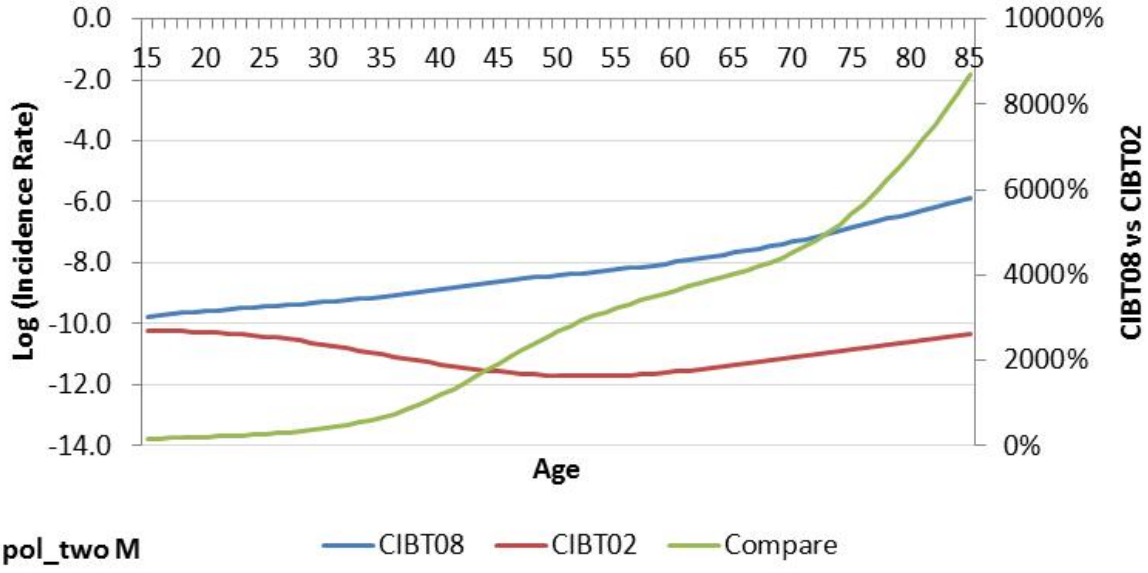
6.32.5. Derived Incidence Rates

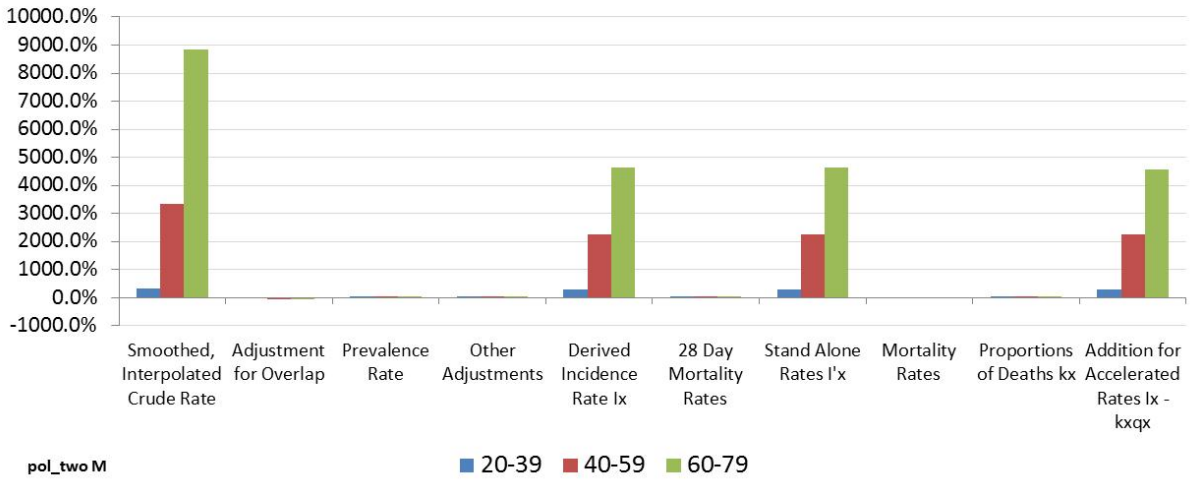
Incidence rates have been derived for the paralysis of one limb and also for paralysis of 2 limbs, in order to give an indication of the impact of the ABI+ definition. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

6.32.5.1. Male Results (Paralysis of two limbs)

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.29	3.96	15.85
Adjustment for Overlap	-26.2%	-45.3%	-68.2%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.96	2.18	6.71
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.96	2.18	6.69
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.1%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.95	2.16	6.63

pol_two _ M

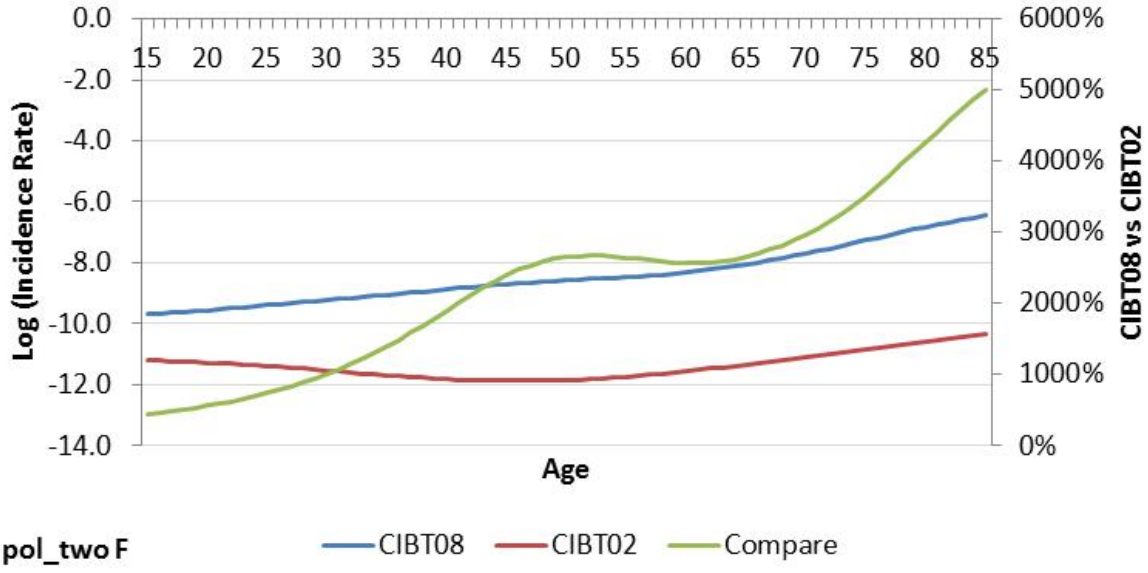


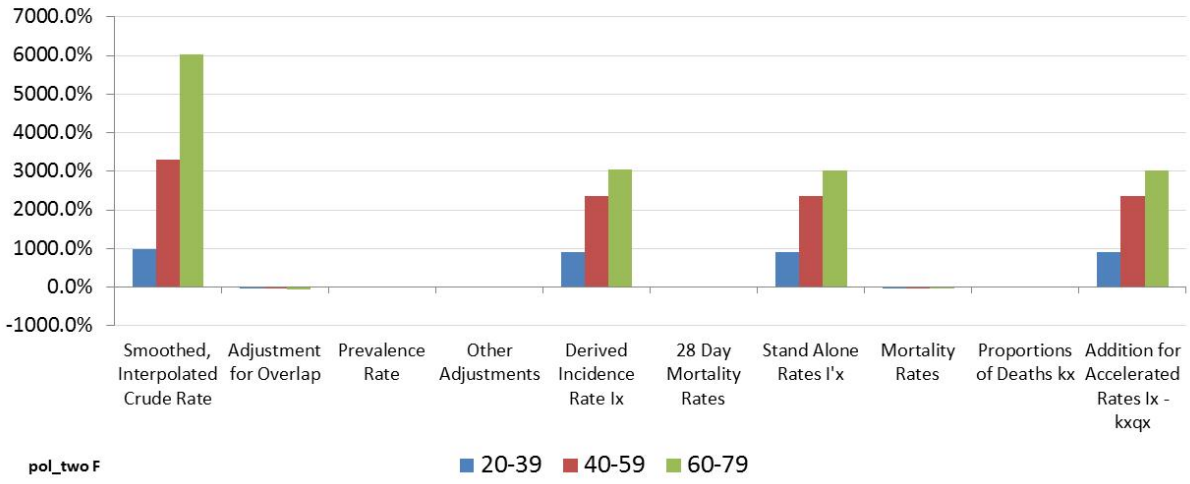


6.32.5.2. Female Results (Paralysis of two limbs)

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.34	3.18	11.40
Adjustment for Overlap	-25.4%	-43.3%	-64.9%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	1.00	1.84	4.67
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	1.00	1.84	4.66
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	1.00	1.83	4.63

pol_two _ F



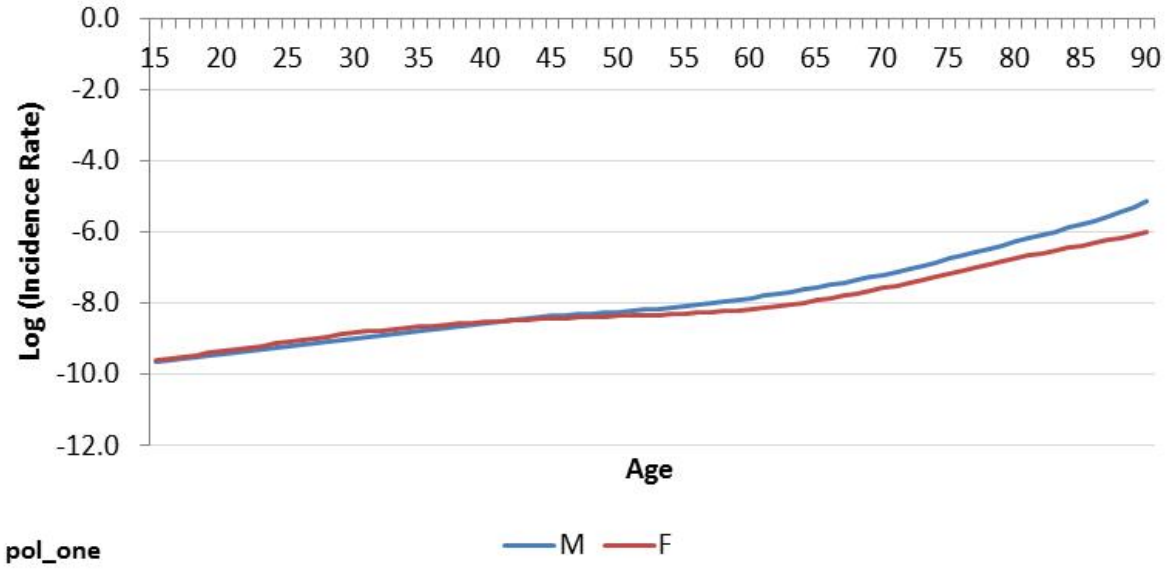


CIBT02 used data from the Spinal Injuries Association and only included paralysis due to injury. CIBT08 could include some paralysis due to medical conditions such as bleeding; as such CIBT08 could include some non-permanent paralysis.

6.32.5.3. Paralysis of One Limb Results

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	1.63	4.55	17.09	1.80	3.78	12.30
Adjustment for Overlap	-22.5%	-42.1%	-67.0%	-21.0%	-39.8%	-63.6%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	1.27	2.66	7.51	1.43	2.32	5.24
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	1.27	2.66	7.50	1.43	2.32	5.23
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	1.27	2.66	7.51	1.43	2.32	5.24

pol_one



6.33. Parkinson's Disease (PD)

6.33.1. What is it?

PD is a condition in which part of the brain becomes progressively more damaged over many years (a progressive neurological condition). It is caused by a loss of nerve cells in part of the brain called the substantia nigra. This leads to a reduction in the amount of a chemical called dopamine in the brain.

Dopamine plays a vital role in regulating the movement of the body and this reduction in dopamine is responsible for many of the symptoms of Parkinson's Disease.

What causes the loss of nerve cells is currently unclear. Most experts think that a combination of genetic and environmental factors is responsible.

PD mainly develops in people over the age of 50. It becomes more common with increasing age. About 5 in 1,000 people in their 60s, and about 40 in 1,000 people in their 80s have PD. It affects both men and women but is a little more common in men.

PD is not usually inherited, and it can affect anyone. However, genetic (hereditary) factors may be important in the small number of people who develop PD before the age of 50.

6.33.2. Symptoms and Treatment

The three main symptoms of Parkinson's Disease are related to movement:

- involuntary shaking of particular parts of the body – known as tremor;
- muscle stiffness that can make everyday tasks such as getting out of a chair very difficult – this is known as rigidity;
- physical movements become very slow – known as bradykinesia.

A person with Parkinson's Disease can also experience a wide range of symptoms unrelated to movement (non-motor symptoms) such as:

- depression;
- daytime sleepiness;
- dysphagia (difficulties swallowing).

There is currently no cure for Parkinson's Disease though a medication called "levodopa" has proved effective in relieving symptoms.

Unfortunately after around 3-5 years of use, the effectiveness of levodopa is reduced.

After this time people can experience a sudden return of symptoms (this is known as an 'off episode') as well as an involuntary jerking of their muscles (dyskinesias). At this point additional medication is usually required.

A range of non-pharmaceutical treatments can be used to manage symptoms, such as speech and language therapy, physiotherapy and surgery.

6.33.3. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Parkinson's disease [before age x] (1999/2002)

Confirmation by a Consultant Neurologist of a definite diagnosis of Parkinson's Disease [before age x].

Parkinson's Disease secondary to alcohol or drug misuse is not covered.

Parkinson's disease [before age x] – resulting in permanent symptoms (2006)

A definite diagnosis of Parkinson's disease [before age x] by a Consultant Neurologist.

There must be permanent clinical impairment of motor function with associated tremor, muscle rigidity and postural instability.

For the above definition, the following are not covered:

- Parkinson's disease secondary to drug abuse

Parkinson's disease [before age x] – resulting in permanent symptoms (2011)

A definite diagnosis of Parkinson's disease [before age x] by a Consultant Neurologist.

There must be permanent clinical impairment of motor function with associated tremor, muscle rigidity and postural instability.

For the above definition, the following are not covered:

- Parkinson's disease secondary to drug abuse
- Other Parkinsonian syndromes

The ABI definition includes an age limit for PD which is optional. Most insurance companies that cover PD in their CI policies do not include age limits.

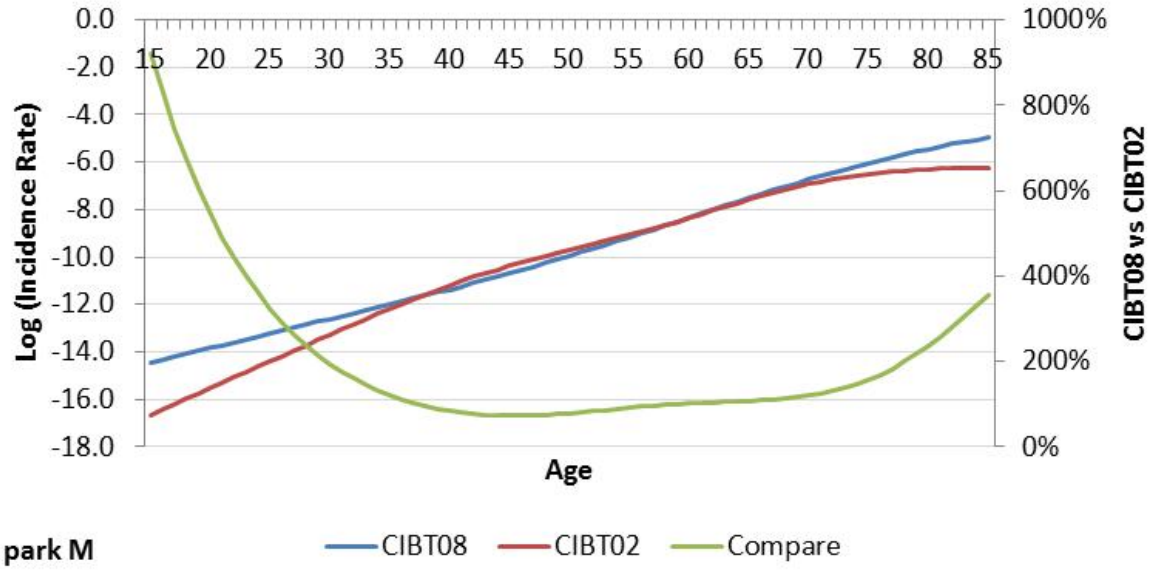
6.33.4. Derived Incidence Rates

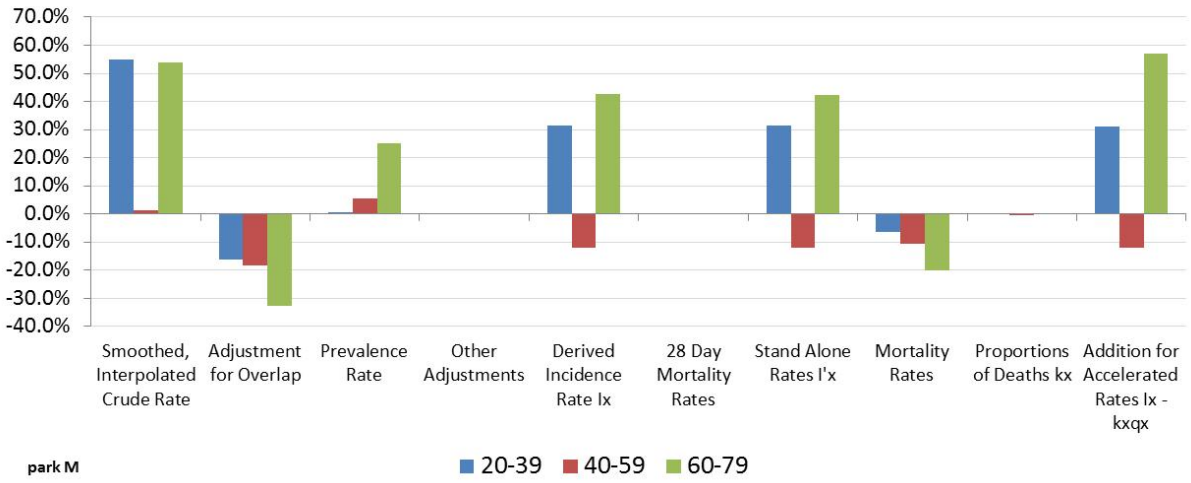
6.33.4.1. Male Results

The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.05	0.70	12.89
Adjustment for Overlap	-16.1%	-18.2%	-32.8%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.04	0.61	11.96
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.04	0.61	11.93
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.7%
Addition for Accelerated Rates Ix - kxqx	0.04	0.59	9.70

park _ M



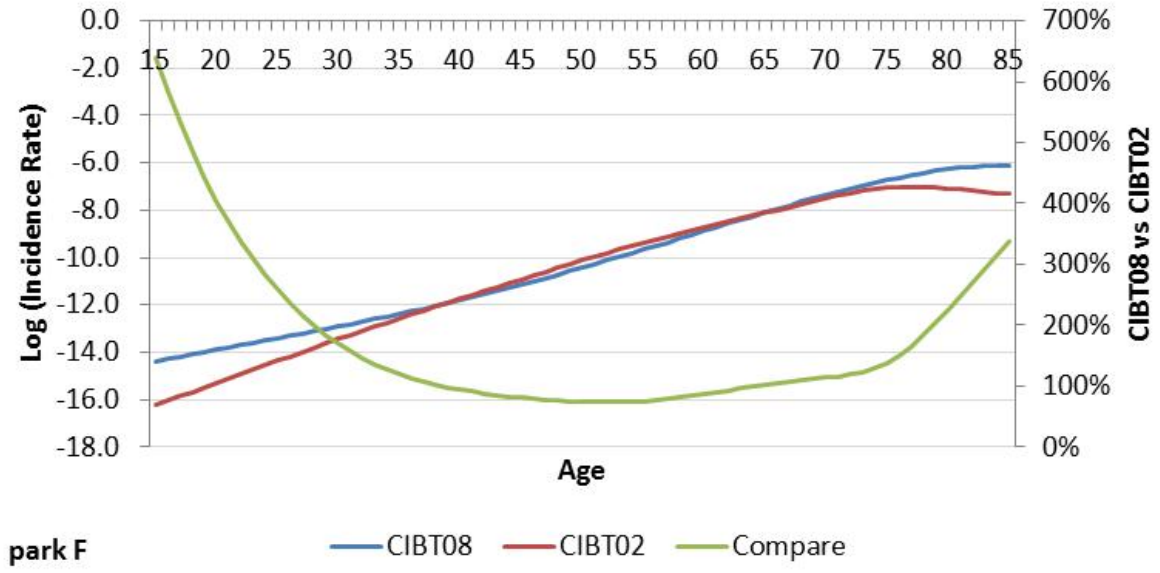


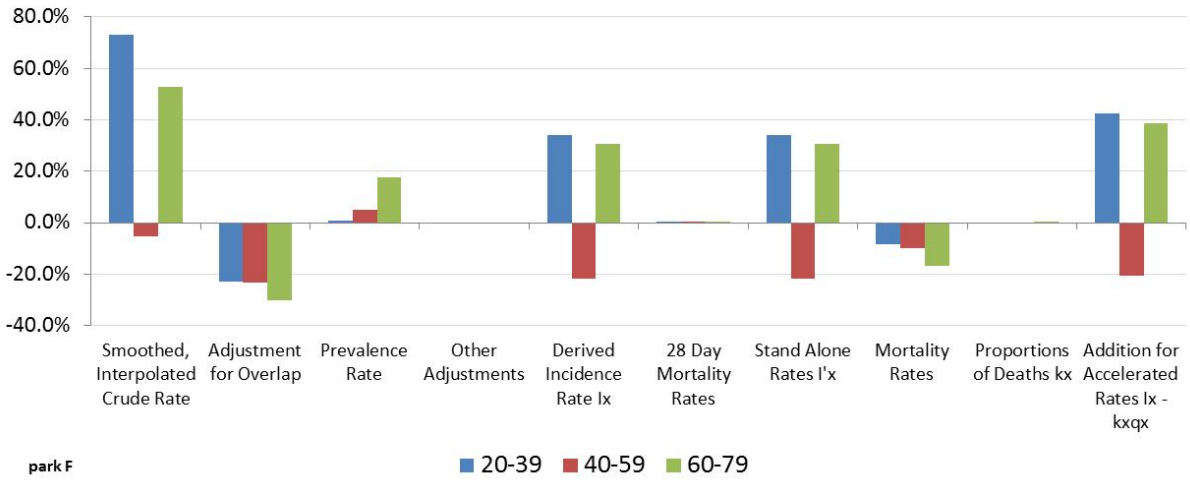
Between ages 35 and 65, male Parkinson's incidence rates per CIBT08 are close to those derived per CIBT02. The CIBT08 rates profile relative to CIBT02 is very similar to that obtained for Motor Neurone Disease. CIBT08 also has a flatter profile than CIBT02 across all age bands with CIBT08 displaying higher incidence rates at younger and older ages. This is primarily due to an increase in crude rates at these age bands. Although CIBT08 exhibits significantly higher rates at younger ages, overall incidence below age 40 remains extremely rare.

6.33.4.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.04	0.45	7.51
Adjustment for Overlap	-22.8%	-23.4%	-30.2%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.03	0.37	6.42
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.03	0.37	6.41
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.5%
Addition for Accelerated Rates Ix - kxqx	0.03	0.36	5.35

park _ F





As expected, female incidence rates for Parkinson's Disease are lower than for males across all age groups. The female profile relative to CIBT02 is also very similar to that generated for males. Between ages 35 and 70, female incidence rates per CIBT08 are very similar to those derived per CIBT02. CIBT08 incidence rates are higher at either side of these ages however, with the elevation becoming more pronounced in the tails. This is primarily due to an increase in crude rates at these age bands Again, although CIBT08 exhibits significantly higher rates at younger ages, overall incidence below age 40 remains extremely rare.

6.34. Primary Pulmonary Hypertension ("PPH")

6.34.1. What is it?

Pulmonary hypertension is abnormally high blood pressure in the arteries of the lungs. It makes the right side of the heart work harder than normal.

The small arteries (blood vessels) of the lung become narrowed, they cannot carry as much blood and hence the heart needs to work harder to pump the blood through the vessel against this pressure. Over time, this causes the right side of the heart to become larger.

6.34.2. Symptoms and Treatment

Shortness of breath or light-headedness during activity is often the first symptom. Fast heart rate may be present. Over time, symptoms occur with lighter activity or even while at rest.

Other symptoms may include dizziness, fatigue, increased abdomen size, feeling faint, swelling of the feet or ankles, and chest pain (particularly during exercise).

People with pulmonary hypertension often have symptoms that come and go.

There is no cure for pulmonary hypertension. The goal of treatment is to control symptoms and prevent more lung damage thereby improving quality of life. Some can slow the progression of PH and can also help reverse damage to the heart and lungs. Hence, the treatments include:

- Blood thinners to reduce the risk of blood clots
- Oxygen therapy at home
- Heart-lung transplant, if medicines do not work
- Getting a yearly flu vaccine and other vaccines
- Stop smoking
- Managing heavy physical lifting and other strenuous activities

6.34.3. Risk Factors

The conditions that increase risk of causing PPH include:

- Birth defects of the heart
- Autoimmune disease that damage the lungs such as HIV infection
- Blood clots in the lung
- Heart valve disease
- Low oxygen levels in the blood for a long period of time
- Lung disease
- Medicines
- Obstructive sleep apnoea
-

6.34.4. Insurance Industry Definitions

Primary pulmonary hypertension does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market.

Definitions used in the UK market are similar and an example is as follows:

Primary pulmonary hypertension – of specified severity

A definite diagnosis of primary pulmonary hypertension by a Consultant Cardiologist or specialist in respiratory medicine. There must be clinical impairment of heart function resulting in the permanent loss of ability to perform physical activities to at least Class 3 of the New York Heart Association classification of functional capacity*.

For the above definition, the following isn't covered:

- Pulmonary hypertension secondary to any other known cause i.e. not primary.

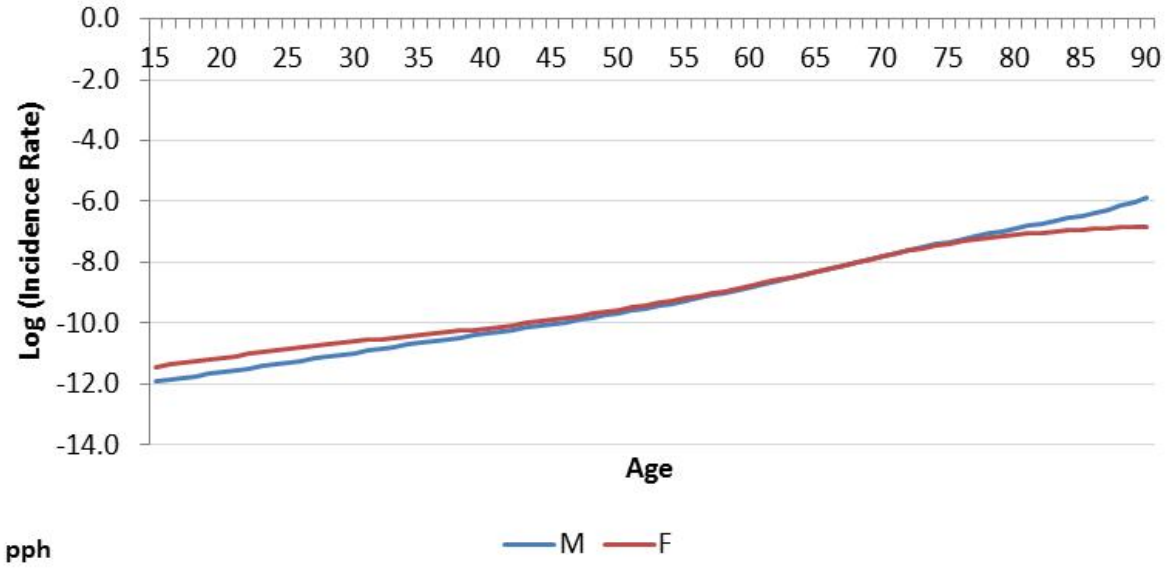
*New York Heart Association Class 3 – heart disease resulting in marked limitation of physical activities where less than ordinary activity causes fatigue, palpitation, breathlessness or chest pain.

6.34.5. Derived Incidence Rates

PPH rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.24	0.94	5.76	0.31	1.02	5.87
Adjustment for Overlap	-24.6%	-32.2%	-50.4%	-20.8%	-30.9%	-45.0%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.18	0.66	3.84	0.25	0.73	3.87
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.18	0.66	3.83	0.25	0.73	3.87
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.1%	0.0%	0.0%	0.1%	0.1%	0.1%
Addition for Accelerated Rates Ix - kxqx	0.18	0.65	3.75	0.24	0.70	3.76

pph



As expected, incidence rates increase significantly by age and a broadly similar across males and females.

It is understood that genuine “idiopathic” pulmonary hypertension is a very rare condition with incidence rates far below what the HES data is suggesting. It is feasible that the ICD-10 coding for PPH is being used for other forms of pulmonary hypertension which are far more common.

We have not taken into account the effect of the CI definition’s severity criterion of impairment at NYHA 3 or more. We would expect insured live claim rates to be lower than our derived rates and leave it to the reader to make the appropriate adjustments.

6.35. Progressive Supranuclear Palsy

6.35.1. What is it?

Progressive supranuclear palsy (PSP) is a rare and progressive condition in which increasing numbers of brain cells become damaged over time. This is known as neuro-degeneration and leads to difficulty with balance, movement, vision, speech and swallowing. It is so called because it's:

- **Progressive** – it gets steadily worse over time
- **Supranuclear** – it damages parts of the brain above the pea-sized 'nuclei' that control eye movements
- **a Palsy** – it causes weakness

Progressive supranuclear palsy, is also known as Steele-Richardson-Olszewski syndrome. In addition, some of the symptoms are similar to those of Parkinson's disease and it is also known as one of the conditions recognised and referred to as "Parkinson Plus".

PSP is associated with an over-production of a protein called tau in certain areas of the brain. In PSP, it forms into clumps – or neurofibrillary tangles – which are believed to damage nerve cells.

6.35.2. Symptoms and Treatment

Early symptoms may include loss of balance and unexpected falls (usually backwards), stiffness and eye problems. These can include difficulties in looking up or down, focusing, double or tunnel vision and dislike of bright lights.

Some people can experience behavioural and cognitive changes – depression, apathy, clumsiness, or tiny, cramped handwriting. Early on, symptoms may resemble those of other neuro-degenerative diseases such as Parkinson's disease, Alzheimer's disease, Motor Neurone disease or Multiple System Atrophy. As a result many people are initially misdiagnosed.

As PSP progresses, symptoms increase. There may be problems with swallowing, slurred speech, recurrent falls, irritability and apathy, slowness of response and severe difficulties walking. Sometimes the eyelids close involuntarily and it becomes increasingly difficult to look up or down. Emotional lability, such as laughing or crying inappropriately, may be another feature and there can be incontinence.

The average life expectancy is around seven years from onset. However, every case is different – there is considerable variation in the symptoms and rate of progression in individuals.

There are no simple tests to diagnose PSP. Brain scans are often used by neurologists for diagnosis as they help by excluding other conditions.

Due to the slowness of movement and balance problems, PSP is often initially diagnosed as Parkinson's disease, a stroke or a brain tumour. Sometimes it is misdiagnosed as Alzheimer's because of changes in mood, intellect and personality that can occur. PSP often goes undiagnosed in the elderly, especially for those living in care homes.

Although research into PSP continues, there is currently no cure for the condition. Treatment focuses on relieving symptoms while trying to ensure that someone with PSP has the best possible quality of life.

Treatment for PSP is provided by a team of health and social care professionals working together, as a person may be affected in many different ways. Specific symptoms of PSP might be treated with:

- **medication** - to improve balance, stiffness and other symptoms
- **botulinum injections or special glasses** - to help with eye problems
- **feeding tubes** - to help manage dysphagia and avoid malnutrition or dehydration

6.35.3. Risk Factors

There is conflicting information about whether or not the disease is inherited. Some research indicates that some people may have a genetic susceptibility that puts them more at risk of developing the condition than others. Other research states that there is no evidence that PSP can be passed down from a parent to their child, and that it is unlikely that PSP is an inherited condition.

Further research is ongoing to identify what other factors may trigger PSP. Suggested environmental triggers include:

- an, as yet, unidentified virus or other type of infection, which may slowly infect the brain over the course of many years
- an unidentified neurotoxin (a poison that damages brain and nerve cells) that may be present in the environment

Males and females are affected approximately equally and there is no racial, geographical or occupational predilection.

6.35.4. Insurance Industry Definitions

PSP does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market.

The definitions used in the market are generally similar and some examples are shown below:

Progressive Supranuclear Palsy – *resulting in permanent symptoms*

A definite diagnosis by a consultant neurologist of progressive supranuclear palsy.

Progressive Supranuclear Palsy – *resulting in permanent symptoms*

A definite diagnosis of progressive supranuclear palsy by a consultant neurologist. There must be permanent clinical impairment of eye movements and motor function.

Progressive Supranuclear Palsy – resulting in permanent symptoms

A definite diagnosis, by a Consultant Neurologist, of Progressive Supranuclear Palsy. There must be permanent clinical impairment of eye movement and motor function with associated tremor, rigidity of movement and postural instability.

Progressive Supranuclear Palsy

A definite diagnosis of Progressive Supranuclear Palsy by a Consultant Neurologist which satisfies all of the following criteria:

- there must be current clinical impairment of motor function,
- there must be current clinical impairment of eye movements, and
- the diagnosis must be confirmed by diagnostic techniques current at the time of claim.

6.35.5. Derived Incidence Rates

PSP rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.17	0.00	0.00	0.00	0.00	0.01
Adjustment for Overlap	-64.5%	0.0%	0.0%	-4.6%	-45.2%	-32.1%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.02	0.00	0.00	0.00	0.00	0.01
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.02	0.00	0.00	0.00	0.00	0.01
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.02	0.00	0.00	0.00	0.00	0.00

rf

We believe that PSP is a genuinely rare condition and these rates confirm this. As such the results of the graduation are somewhat spurious. We therefore, set the rates to zero for CIBT08.

6.36. Rheumatoid Arthritis

6.36.1. What is it?

Rheumatoid arthritis (“RA”) is a chronic, progressive auto-immune disease. It results in inflammation in the joints causing pain, swelling, stiffness and fatigue.

The commonly affected joints are in the hands, feet and wrists. However, it is a systemic disease which means it can affect the whole body and internal organs such as lungs, heart and eyes. The aggressiveness and progression of the disease varies between individuals.

6.36.2. Symptoms and Treatment

RA usually affects joints on both sides of the body equally. Wrists, fingers, knees, feet, and ankles are the most commonly affected. The disease often begins slowly, usually with only minor joint pain, stiffness, and fatigue. An individual can have flare ups when the symptoms become worse than normal.

The common symptoms include: joint pain and swelling, stiffness in the joints after inactivity, lack of or poor sleep patterns, fatigue and tiredness, flu like symptoms, anaemia, psychological effects such as depression and anxiety, weight loss, eye inflammation, inflammation of other body parts and rheumatoid nodules.

RA has no cure. It is manageable and a variety of treatments are available that can slow down the condition and keep the joint damage to a minimum. Hence, the treatment includes:

- Disease modifying anti rheumatic drugs (“DMARDs”) to reduce pain, swelling and stiffness.
- Anti-inflammatory drugs to reduce pain and swelling.
- Surgery is occasionally required ranging from minor procedures such as the release of a nerve or tendon to major ones such as joint replacement.
- Occupational therapy to adjust daily activities to avoid placing too much strain on the joints..

Physiotherapy with exercises to ease symptoms and keep joints healthy.

6.36.3. Risk Factors

RA can occur at any age, but is more common in middle age.

RA is not necessarily caused by genetic factors. However, it can be a contributory factor in developing the condition. There is some evidence that lifestyle factors may increase the risk of developing the condition.

6.36.4. Insurance Industry Definitions

Rheumatoid arthritis does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market.

Definitions used in the market are similar and include within the criteria the impact the condition has in performing activities as shown in the following example:

Rheumatoid Arthritis – resulting in a loss of the ability to do specified physical activities

A definite diagnosis by a Consultant Rheumatologist of chronic rheumatoid arthritis as evidenced by widespread joint destruction with major clinical deformity.
In addition the life assured must permanently be unable to perform three or more of the following activities:

- Bending – The ability to get into or out of a standard saloon car, or to bend or kneel to pick up a tea cup (or similar object) from the floor and straighten up again without the assistance of another person but including the use of appropriate aids
- Dexterity – The physical ability to use hands and fingers, such as being able to communicate effectively using a pen, pencil or keyboard
- Lifting – The ability to lift, carry or otherwise move everyday objects by hand. Everyday objects include a kettle of water, a bag of shopping or an overnight bag or briefcase
- Walking – The ability to walk a distance of 200 metres on a level surface without the assistance of another person, but including the use of appropriate aids, for example a walking stick

6.36.5. Derived Incidence Rates

Rheumatoid Arthritis rates were not calculated for CIBT02. The ICD-10 codes used for this condition are shown in Appendix 2. The table below provides a summary, by 3 age bands, of the adjustments made in calculating these rates (rates shown are per 10,000).

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.53	2.84	10.02	1.71	6.43	18.83
Adjustment for Overlap	-8.1%	-15.7%	-36.1%	-6.8%	-13.8%	-28.3%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.49	2.50	8.65	1.60	5.82	16.29
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.49	2.50	8.63	1.60	5.81	16.27
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.1%	0.0%	0.1%	0.3%
Addition for Accelerated Rates Ix - kxqx	0.49	2.49	8.45	1.60	5.79	15.83

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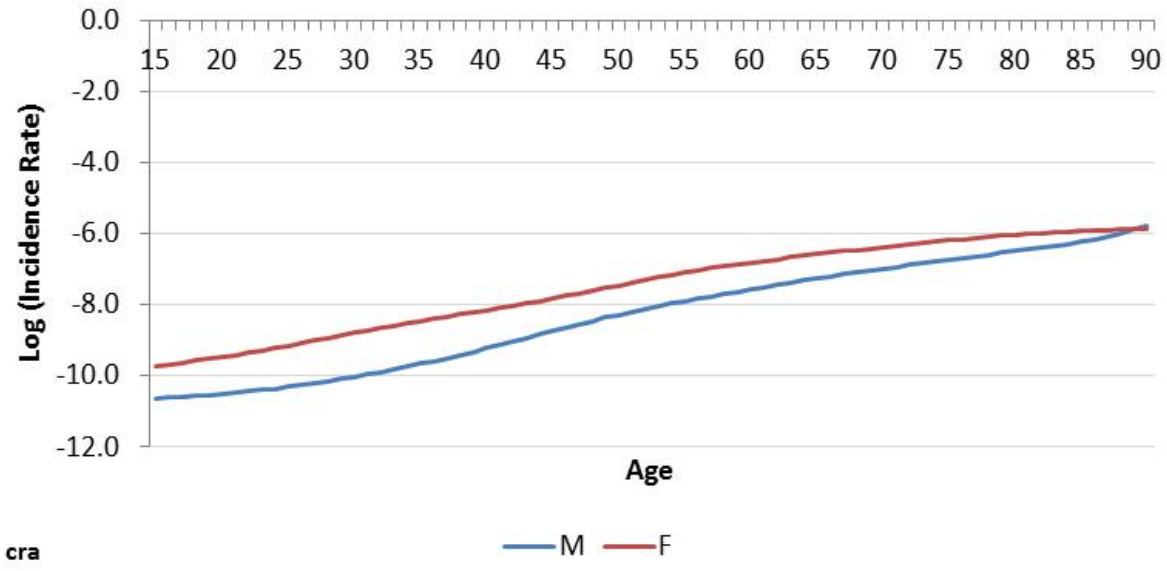

Our rates are have not been adjusted to reflect the severity of the insured definition and so these will overstate the cost of Rheumatoid Arthritis in a Critical Illness policy.

We have compared our crude Rheumatoid Arthritis rates to benchmark rates. The benchmark rate was sourced from the Arthritis Research UK[24] website. The table below draws some comparisons, although the age bands are not the same:

Males

Our Age Band	Their Age Band	Our Rate	Their Rate
20-39	25-34	0.53	0.56
40-59	45-54	2.84	3.13
60-79	65-74	10.02	6.66

As you can see our rates are not dissimilar except at the oldest ages where our rates are considerably higher.

Females

Our Age Band	Their Age Band	Our Rate	Their Rate
20-39	25-34	1.71	2.9
40-59	45-54	6.43	9.2
60-79	65-74	18.83	9.4

Here we see our rates are very different with our rates initially much lower but for the final age band much higher.

[24] http://www.arthritisresearchuk.org/arthritis-information/data-and-statistics/rheumatoid-arthritis.aspx

6.37. Systemic Lupus Erythematosus

6.37.1. What is it?

Systemic lupus erythematosus (SLE) is a chronic disease that causes inflammation in various parts of the body. It is commonly just called SLE or 'lupus'. SLE affects the skin and joints and may involve internal organs..

SLE is a systemic autoimmune disease whereby as in other autoimmune diseases, the immune system attacks the body's cells and tissue, resulting in inflammation and tissue damage.

The disease occurs nine times more often in women than in men, especially in women in child-bearing years ages 15 to 35, and is also more common in those of non-European descent.

6.37.2. Symptoms and Treatment

In some cases, the symptoms develop quite slowly. At first they may be confused with other problems, as there are many possible causes of joint pains and tiredness. Sometimes several symptoms occur together. Symptoms range from mild to severe. For example:

- **Mild SLE**. Many people with SLE just have joint and/or skin symptoms with tiredness. These are unpleasant but are not serious or life-threatening.
- **Moderate SLE**. This includes some inflammation of other parts of the body apart from joints and skin. This may include pleurisy, pericarditis or mild kidney inflammation.
- **Severe SLE**. In some cases, severe inflammation develops which can cause damage to organs such as the heart, lung, brain (central nervous system) or kidneys. This can be life-threatening.

Typically, there are times when the disease flares up (relapses) and symptoms become worse for a few weeks, sometimes longer. These relapses tend to alternate with times when symptoms settle down (remission). The reason why symptoms flare up or settle down is not yet fully understood.

Arthritis is also common but rarely causes permanent deformity as in Rheumatoid Arthritis. Frequently a rash appears on the face, neck and hands. However, no one individual will ever have all the possible symptoms of SLE.

Most people with SLE lead active, normal lives. The outlook for people with SLE is much better than it was in the past. Modern treatments are more effective. For many people with SLE, symptoms are mild or moderate with little risk to life. The joint and skin symptoms may persist but can usually be eased with treatment. For a few people, SLE is severe and can be life-threatening. Severe inflammation of the kidneys, leading to kidney failure, can rarely occur. Severe brain involvement is also rare but can be very serious. However, modern immunosuppressive treatments have improved the outlook, even for people with severe disease. Some people find that symptoms settle in their middle age and they can come off all treatment.

There is no cure for SLE. It is treated mainly with cyclophosphamide, corticosteroids and other immunosuppressants. The leading cause of death is from cardiovascular diseases acquired from corticosteroid therapy. Survival for people with SLE in the United States, Canada, and Europe has risen to approximately 95% at five years, 90% at 10 years, and 78% at 20 years, and now approaches that of matched controls without lupus.

6.37.3. Insurance Industry Definitions

SLE does not have a standard ABI model wording as it does not meet the criteria of being on 75% of products in the UK market. However, a common example of a current definition used by insurers is as follows:

> **Systemic Lupus Erythematosus – *of specified severity***
>
> A definite diagnosis of systemic lupus erythematosus by a consultant rheumatologist resulting in either of the following:
>
> - Permanent neurological deficit with persisting clinical symptoms,
> - the permanent impairment of kidney function tests as follows; Glomerular Filtration Rate (GFR) below 30 ml/min.

The example definition shows that cover is intended to provide benefits for severe SLE that have the very serious complications where the condition has resulted in permanent damage to either the kidneys or the brain.

6.37.4. Derived Incidence Rates

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.08	0.15	0.30	0.81	1.11	1.13
Adjustment for Overlap	-19.6%	-31.5%	-52.4%	-11.5%	-21.1%	-37.5%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.06	0.11	0.19	0.72	0.92	0.86
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.06	0.11	0.19	0.72	0.92	0.86
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.3%	0.1%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.06	0.10	0.19	0.71	0.91	0.86

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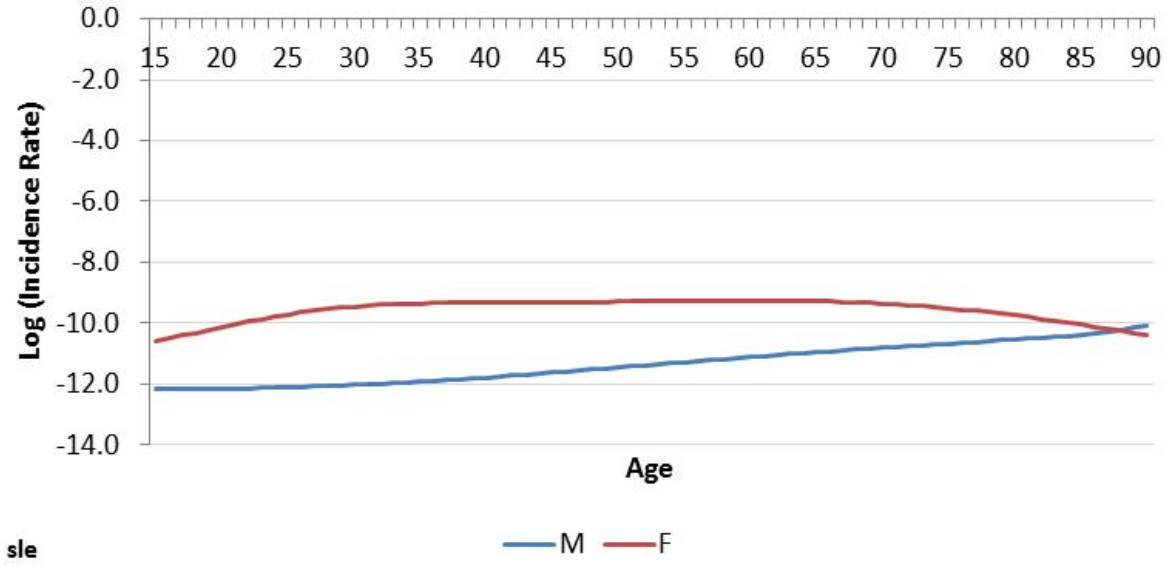

As expected, incidence is much higher for females than for males at most ages, with the variance particularly pronounced within the child-bearing range. While female rates begin to reduce at older ages, male incidence rates gradually increase with age. .At the very highest ages, incidence is higher for males than for females.

6.38. Third Degree Burns

6.38.1. What is it?

A burn is a type of injury to flesh or skin caused by heat, electricity, chemicals, friction or radiation

Burns are classified according to the depth and extent of the skin damage, in the following way.

- **First-degree burns:** the skin is red, painful and very sensitive to touch. The damaged skin may be slightly moist from leakage of the fluid in the deeper layers of the skin.
- **Second-degree burns:** the damage is deeper and blisters usually appear on the skin. The skin is still painful and sensitive.
- **Third-degree burns:** the tissues in all layers of the skin are dead. Usually there are no blisters.

6.38.2. Symptoms and Treatment

The characteristics of a burn depend upon its depth. Third-degree burns destroy the different skin layers through the epidermis and dermis. They may also damage the underlying bones, muscles and tendons. The burn site appears white or charred. There is no sensation in the area since the nerve endings are destroyed. Burns may also produce emotional and psychological distress.

The treatment required depends on the severity of the burn. Third degree burns usually require surgical treatments, such as skin grafting. Extensive burns often require large amounts of intravenous fluids because the subsequent inflammatory response will result in significant capillary fluid leakage and oedema. The most common complications of burns are related to infection.

6.38.3. Insurance Industry Definitions

There have been the following ABI SoBP definitions:

Third Degree Burns – covering 20% of the body's surface area (1999/2002)

Third Degree Burns covering at least 20% of the body's surface area.

Third Degree Burns – covering 20% of the body's surface area (2006/2011)

Burns that involve damage or destruction of the skin to its full depth through to the underlying tissue and covering at least 20% of the body's surface area.

The “ABI+” definitions in the market consider burns to the face, head and neck separately and will payment will be triggered if there is a substantial burn to these areas.

Third Degree Burns (ABI+)

Burns that involve damage or destruction of the skin to its full depth through to the underlying tissue and covering at least 20% of the body's surface area or covering 20% of the area of the face or head.

Third Degree Burns – covering 20% of the body’s surface area (ABI+)

Burns that involve damage or destruction of the skin to its full depth through to the underlying tissue and covering

- at least 20% of the body’s surface area or
- 30% of the head and neck or
- 50% of the face.

6.38.4. Derived Incidence Rates

6.38.4.1. Male Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.45	0.37	0.34
Adjustment for Overlap	-3.1%	-9.3%	-31.8%
Combined Prevalence Rate	0.7%	5.5%	25.3%
Derived Incidence Rate Ix	0.44	0.35	0.31
28 Day Mortality Rates	0.0%	0.0%	-0.2%
Stand Alone Rates I'x	0.44	0.35	0.31
Mortality Rates	9.15	36.28	219.94
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.44	0.35	0.31

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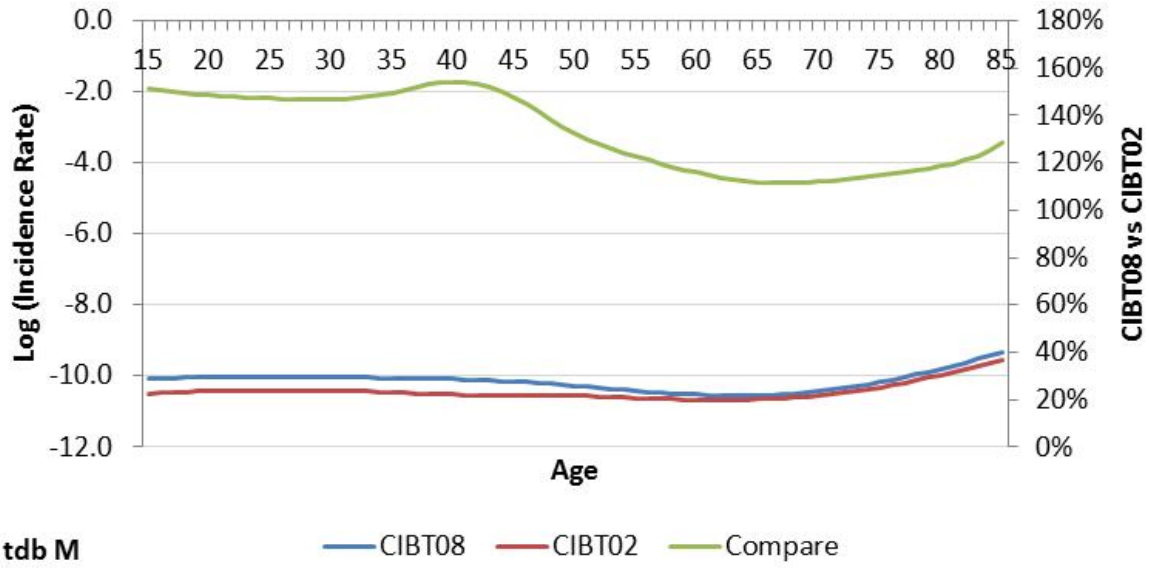
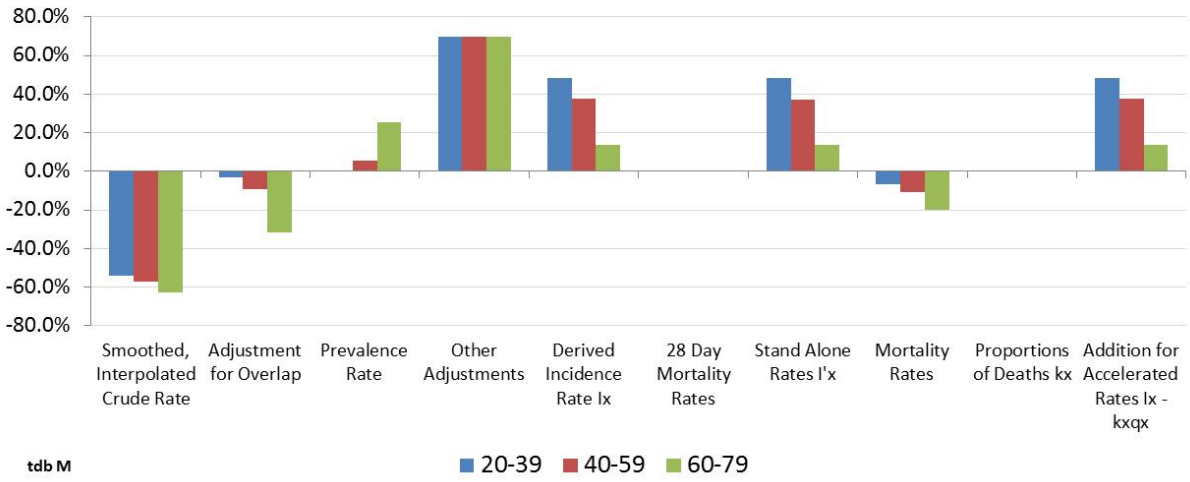


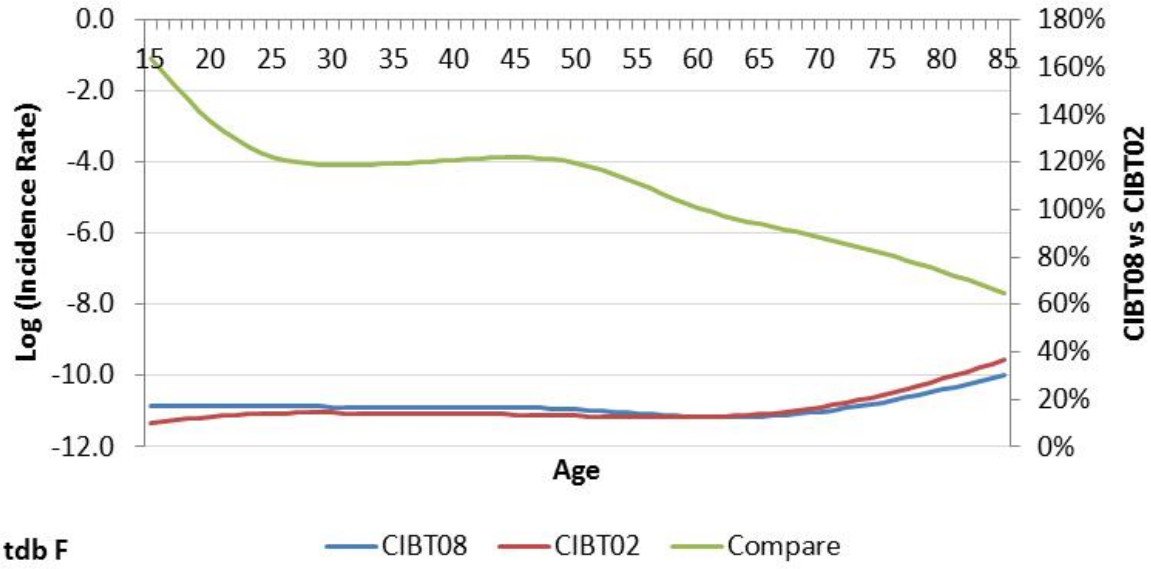


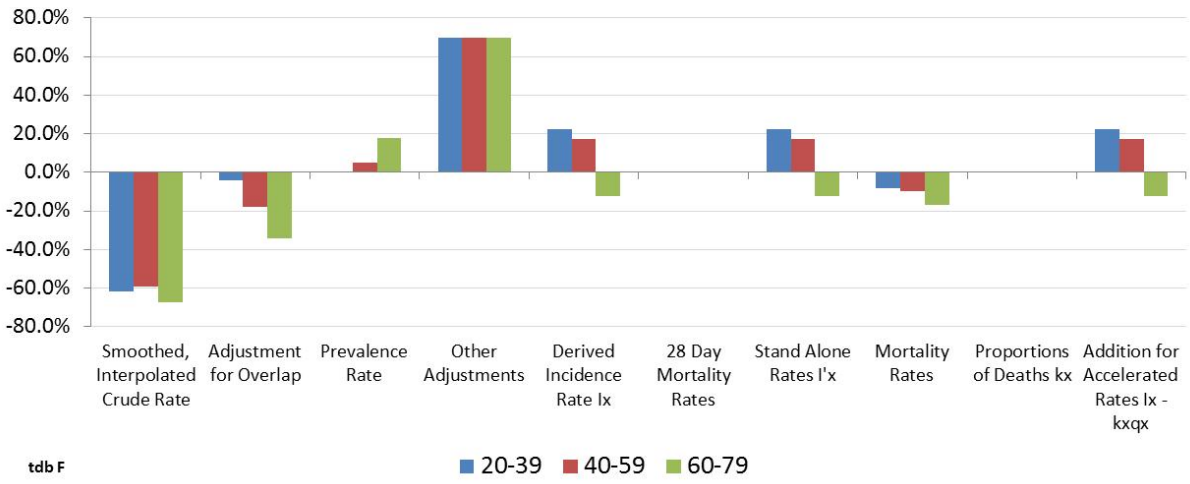
The key reason for the rates increasing over CIBT02 is because we have not made any adjustments for the severity underpin the insured definition relative to the medical definition, nor have we made any allowance for overlap with TPD. Exploring the Critical Path reduced the rates by 30% for severity and by a similar amount again on average for TPD overlap (although this varied by age).

6.38.4.2. Female Results

Age Band	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.20	0.20	0.22
Adjustment for Overlap	-4.4%	-17.8%	-34.3%
Combined Prevalence Rate	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.19	0.17	0.17
28 Day Mortality Rates	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.19	0.17	0.17
Mortality Rates	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.19	0.17	0.17

tdb _ F





6.39. Traumatic head injury

6.39.1. What is it?

Traumatic head injury or traumatic brain injury (TBI) is an injury to the brain caused by a trauma to the head. There are many possible causes, including road traffic accidents, assaults, falls and accidents at home or at work. The injury would have sustained permanent clinical symptoms.

TBI can be classified based on severity, mechanism (closed or penetrating head injury), or other features (e.g. occurring in a specific location or over a widespread area). Head injury usually refers to TBI, but is a broader category because it can involve damage to structures other than the brain, such as the scalp and skull.

TBI is a major cause of death and disability worldwide, especially in children and young adults. Males sustain traumatic brain injuries more frequently than do females. Prevention measures include use of technology to protect those suffering from automobile accidents, such as seat belts and sports or motorcycle helmets, as well as efforts to reduce the number of traffic accidents, such as safety education programs and enforcement of traffic laws.

In addition to the damage caused at the moment of injury, brain trauma causes secondary injury, a variety of events that take place in the minutes and days following the injury. These processes, which include alterations in cerebral blood flow and the pressure within the skull, contribute substantially to the damage from the initial injury.

TBI can cause a host of physical, cognitive, social, emotional and behavioural effects. The outcome can range from complete recovery to permanent disability or death.

6.39.2. Symptoms and Treatment

Symptoms can depend on the type of TBI and the part of the brain that is affected. Unconsciousness tends to last longer for people with injuries on the left side of the brain than for those with injuries on the right.

Symptoms can also depend on the injury's severity. With mild TBI, the patient may remain conscious or may lose consciousness for a short period of time. Other symptoms of mild TBI include headache, vomiting, nausea, lack of motor coordination, dizziness, difficulty balancing, light-headedness, blurred vision or tired eyes, ringing in the ears, bad taste in the mouth, fatigue or lethargy, and changes in sleep patterns.

Cognitive and emotional symptoms include behavioural or mood changes, confusion, and trouble with memory, concentration, attention, or thinking. Mild TBI symptoms may also be present in moderate and severe injuries.

With a moderate or severe TBI, the injured person may have a headache that does not go away, repeated vomiting or nausea, convulsions, an inability to awaken, dilation of one or both pupils, slurred speech, difficulties with words, muscle weakness that causes disordered speech, weakness or numbness in the limbs, loss of coordination, confusion, restlessness, or agitation. Common long-term symptoms of moderate to severe TBI are changes in appropriate social behaviour, deficits in social judgment, and cognitive changes, especially problems with sustained attention, processing speed, and executive functioning. A deficiency in identifying, understanding, processing, and describing emotions occurs in 60% of individuals with TBI. Cognitive and social deficits have long-term consequences for the daily lives of people with moderate to severe TBI, but can be improved with appropriate rehabilitation.

There are diagnosis and treatment that decreased death rates and improved outcome. Some of the current imaging techniques used for diagnosis and treatment include CT scans computed tomography and MRIs magnetic resonance imaging. Depending on the injury,

treatment required may be minimal or may include interventions such as medications, emergency surgery or surgery years later. Physical therapy, speech therapy, recreation therapy, occupational therapy and vision therapy may be employed for rehabilitation.

6.39.3. Insurance Industry Definitions

The ABI SoBP definition is as follows and has not been changed since it was introduced in the 2006 SoBP:

> **Traumatic Head Injury – resulting in permanent symptoms (2006/2011)**
>
> Death of brain tissue due to traumatic injury resulting in permanent neurological deficit with persisting clinical symptoms.

6.39.4. Derived Incidence Rates

For these rates we have produced two sets of rates showing the effect of accepting either of the two criteria or presence of both on an episode:

- ICD-10 codes showing external head injury causes (S06 or T06 codes which indicate a head injury, which could be quite mild);
- OPCS codes indicating operations on the brain.

The rates resulting from requiring either of these criteria are quite significant and so we propose that only those episodes indicate by both criteria being fulfilled are shown here:

	Males			Females		
Age Band	20-39	40-59	60-79	20-39	40-59	60-79
Smoothed, Interpolated Crude Rate	0.41	0.38	0.69	0.07	0.11	0.29
Adjustment for Overlap	-26.8%	-34.7%	-49.2%	-27.6%	-39.5%	-52.1%
Combined Prevalence Rate	0.7%	5.5%	25.3%	0.7%	4.9%	17.8%
Derived Incidence Rate Ix	0.30	0.26	0.47	0.05	0.07	0.16
28 Day Mortality Rates	0.0%	0.0%	-0.2%	0.0%	0.0%	-0.1%
Stand Alone Rates I'x	0.30	0.26	0.47	0.05	0.07	0.16
Mortality Rates	9.15	36.28	219.94	4.31	23.61	150.28
Proportions of Deaths kx	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Addition for Accelerated Rates Ix - kxqx	0.30	0.26	0.47	0.05	0.07	0.16

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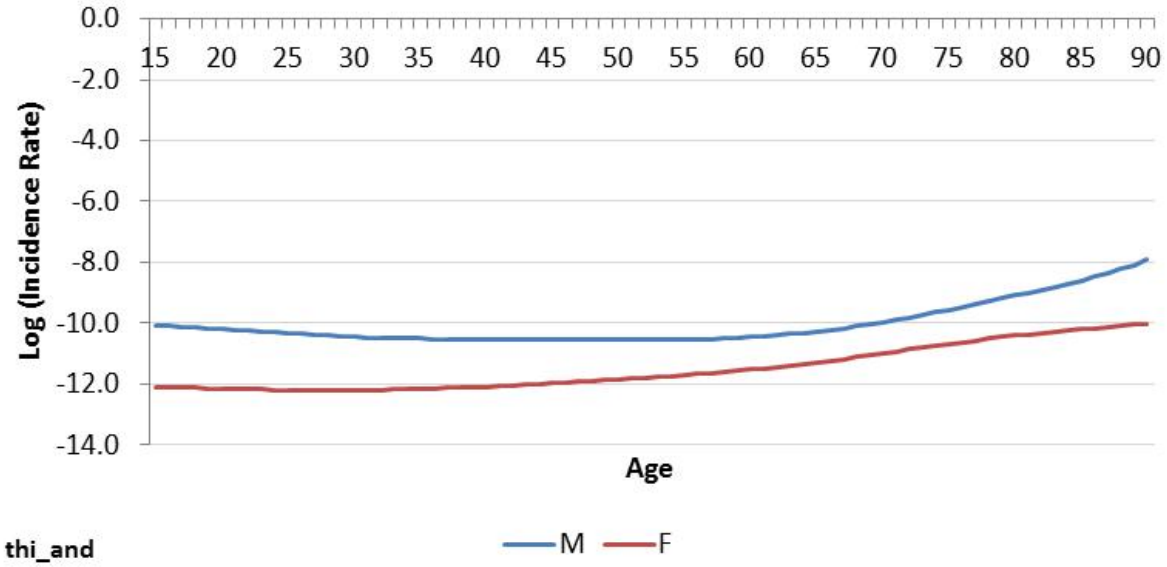
These rates still seem quite high in relation to emerging insured experience, especially at the younger ages.


0.0
-2.0
-4.0
-6.0
-8.0
-10.0
-12.0
-14.0
15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90
Log (Incidence Rate)
Age
M
F
thi_and

Appendix 1 Effect of Unfinished and Day Cases in HES

As noted in section 3.2, in calculating our rates we have excluded:

- Unfinished episodes (EpiStat =1, in the HES data)
- Regular day or night attenders (CLASSPAT =3 or 4, in the HES data).

For previous working parties, these filters were essential to ensure incidence counts were not overstated for either of the following reasons:

- Two incidence counts for the same event could be included if an episode was not completed at the end of the financial year. In this case there would be an unfinished record for the prior financial year and a finished record in the following financial year. In practice the impact of this is naturally mitigated because most unfinished episodes do not have any diagnoses or procedures coded [25] - for example there are approximately 21,000 unfinished episodes in 2007 and only approximately 5500 have anything entered into the first diagnosis field.
- For regular day or night attenders anything other than the first episode is clearly not a first ever event and for some illnesses patients could be admitted many times on a day/night basis and there would be no way to accurately determine the average number of admittances to remove this bias in the absence of patient identifiers.

With our data this time we could in theory relax these filters since we should be able to determine first ever episode of a particular condition for a patient due to matching on HESID. We did not, however do this due to concerns around how perfect the matching by patient was and also due to concerns about accuracy of data entered on unfinished episodes.

The short section demonstrates the effect of applying these filters for three conditions:

- Kidney failure, for which we expect a very large number of regular day attenders;
- Heart Attack, for whom we expect a relatively low number of regular day attenders;
- Cancer, simply because it is so significant for Critical Illness covers.

In the tables below we show the ratio of incidence count for episodes with the appropriate ICD-10 codes for the given condition allowing sequentially for the two filters, relative to the incidence count without any filters applied (so the tables show the results with the stated filter applied divided by the total count with no filter applied).

[25] As the HES data dictionary states: “Because hospital providers are advised not to include clinical data (diagnosis and operation codes) in unfinished records, these are normally excluded from analyses. Also, if unfinished episodes are included in time series analyses - where data for more than one year is involved - there is a danger of counting the same episode twice.”

Starting with Kidney failure, and looking simply at incidence count as recorded in the Primary Diagnosis field without any attempt to calculate first ever incidence we see:

Ratio to:	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Finished Episodes Only	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%
Finished Episodes Excluding Regular Cases	11%	5%	6%	6%	7%	10%	9%	8%	5%	5%	5%

The effect of removing unfinished cases is negligible as shown by the ratios which round to 100%. The effect of excluding regular cases though is more significant though – between 89% and 95% of incidence is removed.

If we now shift to looking at these ratios based on incidence counts having applied our first ever algorithm we see the following:

Ratio to:	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Finished Episodes Only	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%
Finished Episodes Excluding Regular Cases	62%	61%	65%	62%	62%	61%	55%	60%	65%	62%	61%

As before, and as expected, the ratio to finished episodes is still 100%. Also as expected the effect of removing regular attendees is greatly reduced. The reduction though is still significant and so this may suggest:

- Some Kidney Failure suffers have only ever attended as regular attendees and if that is the case we may be understating the true incidence here;
- The use of HESID may indeed not be perfect in matching lives; or
- Some lives may have had their first admission prior to 1997, in which case it would be right to exclude them.

Switching our attention to Heart Attack the same analysis results in ratios of effectively 100% in all cases (some ratios are 99.9% to 1 decimal place).

For Cancer, the results for Primary Diagnosis only, with no first ever algorithm are:

Ratio to:	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Finished Episodes Only	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%
Finished Episodes Excluding Regular Cases	96%	92%	91%	90%	88%	89%	88%	87%	86%	85%	84%

But when we apply our first ever algorithm we see the effect of excluding regular cases drop significantly to well within the acceptable levels of materiality:

Ratio to:	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
Finished Episodes Only	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%
Finished Episodes Excluding Regular Cases	99%	99%	99%	99%	99%	98%	98%	98%	98%	98%	98%

Appendix 2 Diagnosis and Procedure Codes

Illness	Diag or procedure priority?	ICD-10 codes	OPCS codes	CIBT02 - ICD 10 Codes	CIBT02 - OPCS codes
Cancer - All		All C ICD-10 codes excl C44 and including D45, D46, D47		C00 - C97 (excl C44)	
Cancer – Breast	Diagnosis	C50		Not covered in CIBT02	
Cancer- Lung	Diagnosis	C34		Not covered in CIBT02	
Cancer - Melanoma	Diagnosis	C43		Not covered in CIBT02	
Heart attack	Diagnosis	I21		I21, I22	
Stroke	Diagnosis	I60, I61, I62, I63, I64		I60 - I69	
Alzheimer's Disease	Diagnosis	F00, G30		G30	
Aorta graft surgery	Procedure	I71	L16, L18, L19, L20, L21,L23		L183-185, l192-195, l202-205, L212-215, L231, L233, L451-L452
Aplastic anaemia	Diagnosis	D60, D61		Not covered in CIBT02	
Bacterial meningitis	Diagnosis	G00, G03, G042		Not covered in CIBT02	
Benign brain tumour	Diagnosis	D320, D329, D330, D331, D332, D333, D354, D420, D429, D430, D431, D432, D433, D445, G930, G950, Q850	A01, A02, A295, A381, A382, A383, A384, A385, A386, A388, A389, A431, A432, A438, A439	D33, D43	
Blindness	Diagnosis	H540, H541, H542, H543		None used	

Illness	Diag or procedure priority?	ICD-10 codes	OPCS codes	CIBT02 - ICD 10 Codes	CIBT02 - OPCS codes
Cardiomyopathy	Diagnosis	I255, I420, I421, I422, I423, I424, I425, I428, I429, I43, O903, O909		Not covered in CIBT02	
Coma	Diagnosis and procedure together	B150, B160, B162, B190, E035, E100, E110, E120, E130, E140, E15, R402, S067	E85	None used	
Coronary Angioplasty	Procedure		**Multi-vessel:** K492, **Single vessel:** K491, **Unknown no. of vessels:** other K49 codes, K75		K492 (Multiple vessel)
CABG	Procedure		K40, K41, K42, K43, K44, K45		K401 - K404, K408, K409, K411 - K414, K418, K419, K421 - K424, K428, K429, K431 - K434, K438, K439, K441, K442, K448, K449, K451, K452 - K455, K458, K459, K461 - K464, K468, K469
CJD	Diagnosis	A810, F021		Not covered in CIBT02	
Deafness	Diagnosis	H90, H91		None used	
Dementia	Diagnosis	F01, F02, F03		Not covered in CIBT02	
DCIS / Mastectomy	Diagnosis and procedure together	D05	B27	Not covered in CIBT02	
Emphysema / Respiratory failure	Diagnosis and procedure together	J96	E87	Not covered in CIBT02	

Illness	Diag or procedure priority?	ICD-10 codes	OPCS codes	CIBT02 - ICD 10 Codes	CIBT02 - OPCS codes
Encephalitis	Diagnosis	A83, A84, A85, A86, G04, G05		Not covered in CIBT02	
Heart valve replacement or repair	Procedure		K25, K26, K27, K28, K29, K31, K32, K33, K34, K35, K36		K251-255, K258-259, K261-265, K268-269, K271-276, K278-279, K281-285, K288-289, K291-295, K298-299, K311-314 ,K318-319, K321-324, K328-329, K341-344, K348-349, K351-355, K358-359
HIV infection	Diagnosis	B20, B21, B22, B23, B24, R75, Z21		None used	
Kidney failure	Procedure	N17, N18, N19	M01, X40	N17, N18, N19	
Liver failure	Diagnosis	K72		Not covered in CIBT02	
Loss of limb(s)	Procedure	**ABI: T05 ABI+:** S48, S58, S68, S78, S88, S98	X07, X081, X09, X101, X108, X109		X073-075, X093-095
Loss of speech	Diagnosis	F80, R47		None used	
Low grade prostate cancer	Diagnosis	C61	M61	Not covered in CIBT02	
Major organ transplant	Procedure		E53, J01, J54, K01, K02, M01, W34, W99		K011, K018, K019, K021, K022, K023, K024, K028, K029, E538, E539, J011, J012, J541, J542, J543, J548, J549, M011, M012, M013,

Illness	Diag or procedure priority?	ICD-10 codes	OPCS codes	CIBT02 - ICD 10 Codes	CIBT02 - OPCS codes
					M018, M019, W341, W342, W348, W349
Motor Neurone Disease	Diagnosis	G122		G122	
Multiple sclerosis	Diagnosis	G35		G35	
Multiple system atrophy	Diagnosis	G903		Not covered in CIBT02	
Open heart surgery	Procedure		**CI-qualifying:** K14, K48, K52, K54, K55, **Congenital:** K04, K05, K06, K07, K08, K09, K10, K11, K12, K17, K18, K19, K20, K22, K23, K24, L01, L02, L04, L05, L06, L07, L08, L09, L10, L12		
Paralysis of limb(s)	Diagnosis	**ABI:** G81, G82; **ABI+:** G831, G832, G833, G834, G838, G839		None used	
Parkinson's Disease	Diagnosis	F023, G20		G20, G21	
Primary pulmonary hypertension	Diagnosis	I270		Not covered in CIBT02	
Progressive supranuclear palsy	Diagnosis	G231		Not covered in CIBT02	

Illness	Diag or procedure priority?	ICD-10 codes	OPCS codes	CIBT02 - ICD 10 Codes	CIBT02 - OPCS codes
Rheumatoid arthritis	Diagnosis	M053, M058, M059, M06, M08		Not covered in CIBT02	
Systemic lupus erythematosus	Diagnosis	M32		Not covered in CIBT02	
Third degree burns	Diagnosis	T203, T207, T213, T217, T223, T227,T243, T247, T293, T297, T312, T313, T314, T315, T316, T317, T318, T319, T322, T323, T324, T325, T326, T327, T328, T329		T203, T213, T223, T243, T293, T210, T220, T240, T300	
Traumatic head injury	Diagnosis	S06, T060	A052, A053, A054, A401, A408, A409, A411, V031, V053, V054	None used	

Appendix 3 Ratio of First Ever Diagnosis to Count of All Diagnosis

Condition	First ever as a % of all diagnosis
Alzheimer's disease	24%
Angioplasty	81%
Aorta graft surgery	93%
Aplastic anaemia	24%
Bacterial meningitis	56%
Benign brain tumour	39%
Blindness	38%
Cancer	11%
Cardiomyopathy	30%
Coma	91%
Coronary Artery Bypass Graft	54%
Creutzfeldt-Jakob Disease	47%
Deafness	50%
Dementia	32%
Ductal Carcinoma in Situ / Mastectomy	99%
Encephalitis	48%
Heart attack	45%
Heart value replacement or repair	76%
HIV infection	66%
Kidney failure	11%
Liver failure	53%
Loss of One Limb	84%
Loss of speech	55%
Lung cancer	22%
Major organ transplant	89%
Motor Neurone Disease	29%
Multiple sclerosis	10%
Multiple system atrophy	38%
Open Heart Surgery	62%
Paralysis of limb(s)	32%
Parkinson's Disease	18%
Primary pulmonary hypertension	35%

Progressive supranuclear palsy	41%
Prostate cancer	24%
Respiratory failure	97%
Rheumatoid arthritis	20%
Stroke	43%
Systemic lupus erythematous	16%
Third degree burns	44%
Traumatic Head Injury	87%

Appendix 4 Geodemographic Profiler Types

ACORN 2010 Profiler

CATEGORY	GROUP	TYPE
Wealthy Achievers	Wealthy Executives	1 Wealthy mature professionals, large houses
		2 Wealthy working families with mortgages
		3 Villages with wealthy commuters
		4 Well-off managers, larger houses
	Affluent Greys	5 Older affluent professionals
		6 Farming communities
		7 Old people, detached homes
		8 Mature couples, smaller detached homes
	Flourishing Families	9 Older families, prosperous suburbs
		10 Well-off working families with mortgages
		11 Well-off managers, detached houses
		12 Large families and houses in rural areas
Urban Prosperity	Prosperous Professionals	13 Well-off professionals, larger houses and converted flats
		14 Older professionals in suburban houses and apartments
	Educated Urbanites	15 Affluent urban professionals, flats
		16 Prosperous young professionals, flats
		17 Young educated workers, flats
		18 Multi-ethnic young, converted flats
		19 Suburban privately renting professionals
	Aspiring Singles	20 Student flats and cosmopolitan sharers
		21 Singles and sharers, multi-ethnic areas
		22 Low income singles, small rented flats
		23 Student terraces
Comfortably Off	Starting Out	24 Young couples, flats and terraces
		25 White-collar singles/sharers, terraces
	Secure Families	26 Younger white-collar couples with mortgages
		27 Middle income, home owning areas
		28 Working families with mortgages
		29 Mature families in suburban semis
		30 Established home owning workers
		31 Home owning Asian family areas
	Settled Suburbia	32 Retired home owners
		33 Middle income, older couples
		34 Lower incomes, older people, semis
	Prudent Pensioners	35 Elderly singles, purpose built flats
		36 Older people, flats

CATEGORY	GROUP	TYPE
Moderate Means	Asian Communities	37 Crowded Asian terraces
	Asian Communities	38 Low income Asian families
	Post Industrial Families	39 Skilled older families, terraces
		40 Young working families
	Blue Collar Roots	41 Skilled workers, semis and terraces
		42 Home owning families, terraces
		43 Older people, rented terraces
Hard Pressed	Struggling Families	44 Low income larger families, semis
		45 Low income, older people, smaller semis
		46 Low income, routine jobs, terraces and flats
		47 Low income families, terraced estates
		48 Families and single parents, semis and terraces
		49 Large families and single parents, many children
	Burdened Singles	50 Single elderly people, council flats
		51 Single parents and pensioners, council terraces
		52 Families and single parents, council flats
	High Rise Hardship	53 Old people, many high-rise flats
		54 Singles and single parents, high-rise estates
	Inner City Adversity	55 Multi-ethnic purpose built estates
		56 Multi-ethnic crowded flats
Unclassified	Unclassified	57 Unclassified

Mosaic United Kingdom 2009

Group	Type	Description
Alpha Territory	A01	Global Power Brokers
	A02	Voices of Authority
	A03	Business Class
	A04	Serious Money
Professional Rewards	B05	Mid-Career Climbers
	B06	Yesterday's Captains
	B07	Distinctive Success
	B08	Dormitory Villagers
	B09	Escape to the Country
	B10	Parish Guardians
Rural Solitude	C11	Squires Among Locals
	C12	Country Loving Elders
	C13	Modern Agribusiness
	C14	Farming Today
	C15	Upland Struggle
Small Town Diversity	D16	Side Street Singles
	D17	Jacks of All Trades
	D18	Hardworking Families
	D19	Innate Conservatives
Active Retirement	E20	Golden Retirement
	E21	Bungalow Quietude
	E22	Beachcombers
	E23	Balcony Downsizers
Suburban Mindsets	F24	Garden Suburbia
	F25	Production Managers
	F26	Mid-Market Families
	F27	Shop Floor Affluence
	F28	Asian Attainment
Careers and Kids	G29	Footloose Managers
	G30	Soccer Dads and Mums
	G31	Domestic Comfort
	G32	Childcare Years
	G33	Military Dependants
New Homemakers	H34	Buy-to-Let Territory
	H35	Brownfield Pioneers
	H36	Foot on the Ladder
	H37	First to Move In
Ex-Council Community	I38	Settled Ex-Tenants
	I39	Choice Right to Buy
	I40	Legacy of Labour
	I41	Stressed Borrowers

Group	Type	Description
Claimant Cultures	J42	Worn-Out Workers
	J43	Streetwise Kids
	J44	New Parents in Need
Upper Floor Living	K45	Small Block Singles
	K46	Tenement Living
	K47	Deprived View
	K48	Multicultural Towers
	K49	Re-Housed Migrants
Elderly Needs	L50	Pensioners in Blocks
	L51	Sheltered Seniors
	L52	Meals on Wheels
	L53	Low Spending Elders
Industrial Heritage	M54	Clocking Off
	M55	Backyard Regeneration
	M56	Small Wage Owners
Terraced Melting Pot	N57	Back-to-Back Basics
	N58	Asian Identities
	N59	Low-Key Starters
	N60	Global Fusion
Liberal Opinions	O61	Convivial Homeowners
	O62	Crash Pad Professionals
	O63	Urban Cool
	O64	Bright Young Things
	O65	Anti-Materialists
	O66	University Fringe
	O67	Study Buddies

Appendix 5 Working Party Groupings of Geodemographic Profilers

The following table details the mappings that have been used by the Working Party:

Bottom Up			
ACORN		Mosaic	
TYPE	GROUP	TYPE	GROUP
1	A1	1	M1
2	A2	2	M1
3	A2	3	M1
4	A2	4	M1
5	A2	5	M2
6	A3	6	M5
7	A2	7	M3
8	A3	8	M2
9	A2	9	M3
10	A3	10	M5
11	A3	11	M2
12	A4	12	M3
13	A2	13	M3
14	A2	14	M3
15	A2	15	M3
16	A1	16	M4
17	A3	17	M2
18	A3	18	M4
19	A1	19	M5
20	A4	20	M6
21	A4	21	M6
22	A5	22	M4
23	A4	23	M3
24	A4	24	M2
25	A2	25	M3
26	A3	26	M3
27	A3	27	M4
28	A3	28	M2
29	A3	29	M3
30	A4	30	M3
31	A3	31	M4
32	A2	32	M5
33	A3	33	M2
34	A4	34	M2
35	A1	35	M6
36	A4	36	M4
37	A6	37	M1

Top Down			
ACORN		Mosaic	
TYPE	GROUP	TYPE	GROUP
1	A1	1	M1
2	A1	2	M1
3	A1	3	M1
4	A1	4	M1
5	A1	5	M1
6	A1	6	M1
7	A1	7	M1
8	A1	8	M1
9	A2	9	M1
10	A2	10	M1
11	A2	11	M2
12	A2	12	M2
13	A1	13	M2
14	A1	14	M2
15	A2	15	M2
16	A2	16	M3
17	A2	17	M3
18	A2	18	M3
19	A2	19	M3
20	A4	20	M3
21	A4	21	M3
22	A4	22	M3
23	A4	23	M3
24	A3	24	M2
25	A3	25	M2
26	A3	26	M2
27	A3	27	M2
28	A3	28	M2
29	A3	29	M2
30	A3	30	M2
31	A3	31	M2
32	A4	32	M2
33	A4	33	M2
34	A4	34	M4
35	A4	35	M4
36	A4	36	M4
37	A5	37	M4

Bottom Up			
ACORN		Mosaic	
TYPE	GROUP	TYPE	GROUP
38	A5	38	M6
39	A3	39	M4
40	A4	40	M3
41	A4	41	M3
42	A4	42	M5
43	A5	43	M5
44	A5	44	M4
45	A5	45	M6
46	A5	46	M3
47	A5	47	M2
48	A6	48	M2
49	A6	49	M4
50	A5	50	M6
51	A6	51	M6
52	A5	52	M6
53	A5	53	M6
54	A6	54	M5
55	A4	55	M2
56	A4	56	M4
57	A5	57	M4
		58	M4
		59	M5
		60	M3
		61	M1
		62	M2
		63	M1
		64	M3
		65	M4
		66	M2
		67	M6

Top Down			
ACORN		Mosaic	
TYPE	GROUP	TYPE	GROUP
38	A5	38	M5
39	A5	39	M5
40	A5	40	M5
41	A5	41	M5
42	A5	42	M6
43	A5	43	M6
44	A6	44	M6
45	A6	45	M6
46	A6	46	M6
47	A6	47	M6
48	A6	48	M6
49	A6	49	M6
50	A6	50	M5
51	A6	51	M5
52	A6	52	M5
53	A6	53	M5
54	A6	54	M4
55	A6	55	M4
56	A6	56	M4
57	A6	57	M6
		58	M6
		59	M6
		60	M6
		61	M1
		62	M1
		63	M1
		64	M1
		65	M1
		66	M1
		67	M1

Appendix 6 CIBT08 Calculation Details and Tables

Appendix 6.01 CIBT08 Male Rates – By condition

All rates per 10,000.

Age	Cancer	Heart attack	Stroke	Aplastic anaemia	Aorta graft surgery	Alzheimer's disease	Benign brain tumour	Blindness	Bacterial meningitis	Coronary artery bypass graft
18	2.98	0.06	0.46	0.14	0.03	0.00	0.52	0.11	0.25	0.00
19	3.11	0.07	0.49	0.14	0.04	0.00	0.52	0.11	0.24	0.00
20	3.26	0.09	0.53	0.14	0.04	0.00	0.53	0.11	0.24	0.00
21	3.42	0.11	0.57	0.14	0.04	0.00	0.53	0.11	0.23	0.00
22	3.58	0.14	0.62	0.14	0.04	0.00	0.54	0.11	0.23	0.00
23	3.75	0.18	0.67	0.14	0.04	0.01	0.54	0.11	0.22	0.01
24	3.93	0.22	0.72	0.14	0.05	0.01	0.55	0.11	0.22	0.01
25	4.12	0.27	0.78	0.14	0.05	0.01	0.56	0.11	0.22	0.01
26	4.32	0.34	0.84	0.14	0.05	0.01	0.56	0.11	0.21	0.01
27	4.53	0.42	0.90	0.15	0.05	0.01	0.57	0.11	0.21	0.01
28	4.76	0.52	0.98	0.15	0.06	0.01	0.58	0.12	0.21	0.02
29	5.00	0.64	1.06	0.15	0.06	0.01	0.60	0.12	0.21	0.02
30	5.25	0.79	1.15	0.16	0.06	0.01	0.61	0.12	0.20	0.03
31	5.52	0.97	1.24	0.16	0.07	0.01	0.63	0.12	0.20	0.04
32	5.82	1.20	1.36	0.17	0.07	0.01	0.64	0.13	0.20	0.05
33	6.14	1.47	1.48	0.18	0.08	0.02	0.66	0.13	0.20	0.06
34	6.49	1.80	1.62	0.18	0.08	0.02	0.68	0.13	0.20	0.08
35	6.87	2.19	1.78	0.19	0.09	0.02	0.71	0.14	0.20	0.10
36	7.31	2.66	1.96	0.20	0.10	0.02	0.73	0.14	0.20	0.13
37	7.79	3.21	2.17	0.21	0.10	0.02	0.76	0.15	0.20	0.16
38	8.34	3.85	2.40	0.22	0.11	0.03	0.78	0.15	0.20	0.20
39	8.97	4.59	2.65	0.23	0.12	0.03	0.81	0.16	0.20	0.26
40	9.69	5.45	2.94	0.24	0.13	0.03	0.84	0.17	0.20	0.33
41	10.51	6.41	3.26	0.25	0.14	0.04	0.87	0.17	0.21	0.42
42	11.45	7.49	3.61	0.26	0.16	0.04	0.90	0.18	0.21	0.53
43	12.54	8.67	3.99	0.27	0.17	0.05	0.93	0.19	0.21	0.66
44	13.80	9.95	4.42	0.29	0.19	0.06	0.96	0.20	0.21	0.83
45	15.25	11.32	4.88	0.30	0.21	0.06	1.00	0.20	0.22	1.03
46	16.93	12.77	5.39	0.32	0.23	0.07	1.03	0.21	0.22	1.27
47	18.86	14.28	5.93	0.33	0.26	0.08	1.07	0.22	0.23	1.55

Age	Cancer	Heart attack	Stroke	Aplastic anaemia	Aorta graft surgery	Alzheimer's disease	Benign brain tumour	Blindness	Bacterial meningitis	Coronary artery bypass graft
48	21.10	15.84	6.52	0.35	0.29	0.10	1.11	0.23	0.23	1.87
49	23.67	17.41	7.14	0.37	0.33	0.11	1.15	0.24	0.23	2.25
50	26.64	18.99	7.79	0.39	0.37	0.13	1.19	0.26	0.24	2.67
51	30.03	20.54	8.46	0.41	0.41	0.15	1.23	0.27	0.24	3.14
52	33.88	22.06	9.16	0.43	0.46	0.18	1.28	0.28	0.25	3.67
53	38.22	23.55	9.88	0.45	0.52	0.21	1.33	0.30	0.25	4.24
54	43.10	24.99	10.62	0.47	0.59	0.25	1.38	0.31	0.26	4.87
55	48.55	26.38	11.39	0.50	0.67	0.29	1.44	0.33	0.26	5.56
56	54.59	27.72	12.18	0.53	0.76	0.34	1.50	0.35	0.27	6.30
57	61.26	29.01	13.01	0.56	0.87	0.40	1.57	0.37	0.27	7.09
58	68.55	30.26	13.92	0.59	0.99	0.48	1.64	0.39	0.28	7.93
59	76.47	31.49	14.92	0.62	1.14	0.57	1.71	0.41	0.28	8.83
60	85.05	32.72	16.03	0.66	1.31	0.67	1.78	0.44	0.29	9.78
61	94.29	33.95	17.29	0.70	1.51	0.80	1.85	0.47	0.29	10.79
62	104.13	35.20	18.70	0.74	1.74	0.96	1.92	0.50	0.30	11.84
63	114.49	36.49	20.28	0.78	2.01	1.15	1.99	0.53	0.30	12.93
64	125.28	37.83	22.05	0.83	2.31	1.38	2.06	0.57	0.31	14.05
65	136.44	39.25	24.04	0.89	2.67	1.67	2.12	0.62	0.31	15.20
66	147.89	40.77	26.26	0.95	3.07	2.02	2.19	0.67	0.32	16.37
67	159.62	42.40	28.73	1.02	3.52	2.46	2.25	0.73	0.33	17.56
68	171.64	44.18	31.45	1.10	3.99	3.01	2.31	0.79	0.33	18.76
69	183.96	46.12	34.44	1.18	4.49	3.69	2.38	0.87	0.34	19.99
70	196.59	48.25	37.70	1.28	4.99	4.55	2.45	0.96	0.34	21.25
71	209.57	50.61	41.27	1.38	5.49	5.61	2.52	1.07	0.35	22.51
72	223.05	53.24	45.20	1.50	5.98	6.92	2.60	1.20	0.36	23.71
73	237.23	56.18	49.56	1.63	6.43	8.55	2.69	1.36	0.37	24.75
74	252.32	59.48	54.45	1.78	6.85	10.56	2.78	1.55	0.37	25.50
75	268.59	63.21	59.97	1.95	7.20	13.02	2.89	1.78	0.38	25.85
76	286.24	67.42	66.21	2.14	7.48	16.01	3.02	2.07	0.39	25.68
77	305.28	72.17	73.25	2.36	7.66	19.58	3.16	2.42	0.40	25.00
78	325.62	77.51	81.17	2.60	7.72	23.79	3.32	2.88	0.41	23.86
79	347.11	83.49	90.01	2.87	7.65	28.66	3.50	3.46	0.42	22.31
80	369.53	90.14	99.80	3.18	7.40	34.25	3.70	4.18	0.44	20.43
81	392.68	97.51	110.62	3.53	6.98	40.61	3.93	5.10	0.45	18.30
82	416.77	105.76	122.67	3.92	6.41	47.89	4.19	6.30	0.46	16.03
83	442.24	115.12	136.24	4.37	5.74	56.31	4.48	7.89	0.48	13.72
84	469.75	125.89	151.83	4.90	5.02	66.19	4.82	10.02	0.50	11.49
85	500.27	138.53	170.07	5.53	4.34	77.95	5.23	12.88	0.52	9.47

Age	Cardiomyopathy	CJD	Coma	Deafness	Dementia	Encephalitis	HIV infection	Heart Value replacement or repair	Kidney failure	Liver failure
18	0.32	0.00	0.01	1.16	0.03	0.23	0.16	0.35	0.03	0.18
19	0.33	0.00	0.01	1.14	0.03	0.24	0.18	0.36	0.03	0.19
20	0.34	0.00	0.01	1.12	0.03	0.24	0.21	0.36	0.04	0.21
21	0.35	0.00	0.01	1.11	0.03	0.25	0.25	0.37	0.04	0.22
22	0.37	0.00	0.01	1.10	0.03	0.25	0.29	0.37	0.04	0.24
23	0.38	0.00	0.01	1.09	0.03	0.25	0.34	0.38	0.05	0.26
24	0.40	0.00	0.01	1.09	0.04	0.26	0.39	0.39	0.05	0.27
25	0.42	0.00	0.01	1.10	0.04	0.26	0.45	0.40	0.05	0.29
26	0.45	0.00	0.01	1.11	0.04	0.26	0.52	0.41	0.06	0.31
27	0.47	0.00	0.01	1.13	0.05	0.27	0.60	0.42	0.06	0.33
28	0.50	0.00	0.01	1.15	0.05	0.27	0.68	0.43	0.06	0.35
29	0.54	0.00	0.01	1.18	0.05	0.27	0.76	0.44	0.07	0.37
30	0.58	0.00	0.01	1.21	0.06	0.27	0.85	0.46	0.07	0.39
31	0.62	0.00	0.01	1.24	0.06	0.28	0.94	0.47	0.07	0.41
32	0.67	0.00	0.01	1.28	0.06	0.28	1.03	0.49	0.08	0.43
33	0.72	0.00	0.01	1.31	0.07	0.28	1.12	0.50	0.08	0.45
34	0.78	0.00	0.01	1.35	0.07	0.28	1.20	0.52	0.09	0.48
35	0.85	0.00	0.01	1.39	0.08	0.29	1.27	0.54	0.09	0.50
36	0.92	0.01	0.01	1.42	0.09	0.29	1.33	0.56	0.09	0.53
37	0.99	0.01	0.01	1.46	0.09	0.29	1.37	0.58	0.09	0.56
38	1.07	0.01	0.01	1.49	0.10	0.30	1.40	0.61	0.10	0.60
39	1.16	0.01	0.01	1.53	0.11	0.30	1.42	0.64	0.10	0.64
40	1.25	0.01	0.01	1.57	0.12	0.31	1.42	0.67	0.10	0.68
41	1.35	0.01	0.01	1.61	0.14	0.31	1.41	0.70	0.11	0.72
42	1.45	0.01	0.01	1.66	0.15	0.32	1.38	0.74	0.11	0.77
43	1.56	0.01	0.01	1.70	0.16	0.33	1.34	0.79	0.11	0.82
44	1.67	0.01	0.01	1.75	0.18	0.33	1.29	0.84	0.12	0.87
45	1.79	0.01	0.01	1.81	0.20	0.34	1.23	0.90	0.12	0.93
46	1.92	0.01	0.01	1.86	0.23	0.35	1.17	0.97	0.13	0.98
47	2.05	0.01	0.01	1.92	0.26	0.36	1.10	1.05	0.13	1.04
48	2.18	0.01	0.01	1.99	0.29	0.37	1.03	1.15	0.14	1.10
49	2.33	0.01	0.01	2.06	0.33	0.38	0.97	1.25	0.14	1.15
50	2.48	0.01	0.01	2.13	0.38	0.39	0.90	1.38	0.15	1.21
51	2.63	0.01	0.01	2.21	0.43	0.40	0.83	1.52	0.15	1.26
52	2.80	0.02	0.01	2.30	0.50	0.41	0.77	1.68	0.16	1.30
53	2.96	0.02	0.01	2.39	0.57	0.42	0.71	1.86	0.17	1.34
54	3.12	0.02	0.01	2.48	0.66	0.43	0.66	2.06	0.18	1.38

Age	Cardiomyopathy	CJD	Coma	Deafness	Dementia	Encephalitis	HIV infection	Heart Value replacement or repair	Kidney failure	Liver failure
55	3.29	0.02	0.01	2.58	0.76	0.44	0.60	2.29	0.19	1.41
56	3.45	0.03	0.01	2.69	0.87	0.45	0.56	2.54	0.20	1.43
57	3.61	0.03	0.01	2.81	1.01	0.46	0.51	2.82	0.22	1.45
58	3.77	0.03	0.01	2.93	1.17	0.47	0.47	3.13	0.23	1.46
59	3.93	0.04	0.01	3.07	1.36	0.49	0.43	3.46	0.24	1.47
60	4.08	0.04	0.01	3.21	1.59	0.50	0.39	3.83	0.26	1.48
61	4.24	0.04	0.01	3.37	1.86	0.51	0.36	4.22	0.28	1.49
62	4.40	0.04	0.01	3.53	2.19	0.52	0.33	4.64	0.30	1.49
63	4.56	0.05	0.01	3.72	2.59	0.54	0.30	5.09	0.32	1.50
64	4.72	0.05	0.01	3.93	3.08	0.55	0.28	5.58	0.34	1.50
65	4.88	0.05	0.01	4.15	3.68	0.56	0.25	6.11	0.36	1.51
66	5.04	0.06	0.01	4.40	4.42	0.58	0.23	6.69	0.39	1.52
67	5.20	0.06	0.01	4.68	5.34	0.59	0.21	7.30	0.41	1.53
68	5.36	0.06	0.01	4.99	6.50	0.61	0.19	7.95	0.44	1.55
69	5.51	0.07	0.01	5.35	7.96	0.63	0.18	8.65	0.47	1.58
70	5.65	0.07	0.01	5.75	9.79	0.64	0.16	9.39	0.49	1.61
71	5.78	0.08	0.01	6.21	12.08	0.66	0.15	10.16	0.52	1.65
72	5.90	0.08	0.01	6.76	14.95	0.68	0.14	10.95	0.55	1.70
73	6.01	0.08	0.01	7.39	18.57	0.70	0.13	11.72	0.54	1.76
74	6.10	0.09	0.01	8.15	23.12	0.72	0.12	12.43	0.53	1.83
75	6.18	0.09	0.01	9.05	28.81	0.74	0.11	13.06	0.51	1.92
76	6.25	0.10	0.01	10.14	35.86	0.76	0.10	13.56	0.49	2.01
77	6.30	0.10	0.01	11.47	44.53	0.78	0.09	13.91	0.47	2.12
78	6.33	0.10	0.01	13.10	55.07	0.80	0.08	14.08	0.45	2.25
79	6.35	0.11	0.01	15.14	67.78	0.83	0.08	14.04	0.42	2.39
80	6.36	0.11	0.01	17.65	82.98	0.85	0.07	13.77	0.40	2.55
81	6.34	0.12	0.01	20.78	101.04	0.87	0.07	13.24	0.37	2.72
82	6.33	0.12	0.01	24.82	122.74	0.90	0.06	12.51	0.34	2.91
83	6.31	0.13	0.01	30.19	149.10	0.92	0.06	11.62	0.29	3.12
84	6.30	0.13	0.01	37.39	181.51	0.95	0.05	10.67	0.25	3.37
85	6.32	0.14	0.01	47.08	221.83	0.99	0.05	9.72	0.20	3.66

Age	Loss of one limb	Loss of speech	Motor Neurone Disease	Major Organ Transplant	Multiple sclerosis	Multiple system atrophy	Open Heart Surgery	Parkinson's disease	Paralysis of limb(s)	Primary pulmonary hypertension
18	1.28	0.41	0.01	0.06	0.10	0.00	0.23	0.01	0.64	0.08
19	1.33	0.42	0.01	0.06	0.12	0.00	0.23	0.01	0.67	0.09
20	1.37	0.43	0.02	0.06	0.14	0.00	0.22	0.01	0.69	0.09
21	1.40	0.44	0.02	0.06	0.16	0.00	0.22	0.01	0.71	0.10
22	1.43	0.45	0.02	0.06	0.19	0.00	0.21	0.01	0.73	0.10
23	1.45	0.47	0.02	0.07	0.22	0.00	0.21	0.01	0.76	0.11
24	1.47	0.48	0.02	0.07	0.26	0.00	0.21	0.02	0.78	0.12
25	1.48	0.50	0.02	0.07	0.30	0.00	0.20	0.02	0.80	0.12
26	1.48	0.52	0.02	0.07	0.34	0.00	0.20	0.02	0.82	0.13
27	1.48	0.54	0.03	0.07	0.38	0.00	0.20	0.02	0.85	0.14
28	1.48	0.56	0.03	0.08	0.43	0.00	0.20	0.03	0.87	0.15
29	1.47	0.59	0.03	0.08	0.47	0.00	0.19	0.03	0.90	0.16
30	1.47	0.62	0.03	0.08	0.52	0.00	0.19	0.03	0.92	0.17
31	1.48	0.65	0.04	0.08	0.56	0.00	0.19	0.04	0.95	0.18
32	1.48	0.68	0.04	0.09	0.60	0.00	0.19	0.04	0.99	0.20
33	1.49	0.71	0.05	0.09	0.64	0.00	0.19	0.05	1.02	0.21
34	1.51	0.75	0.05	0.09	0.68	0.00	0.20	0.05	1.07	0.22
35	1.52	0.79	0.05	0.10	0.71	0.00	0.20	0.06	1.11	0.24
36	1.55	0.84	0.06	0.10	0.74	0.00	0.20	0.07	1.17	0.25
37	1.58	0.88	0.07	0.10	0.77	0.00	0.20	0.08	1.23	0.27
38	1.61	0.93	0.07	0.11	0.79	0.00	0.21	0.09	1.29	0.29
39	1.64	0.99	0.08	0.11	0.82	0.00	0.21	0.10	1.36	0.30
40	1.68	1.04	0.09	0.12	0.84	0.01	0.22	0.12	1.43	0.32
41	1.73	1.10	0.10	0.12	0.87	0.01	0.22	0.13	1.51	0.34
42	1.77	1.16	0.11	0.13	0.89	0.01	0.23	0.15	1.59	0.37
43	1.81	1.22	0.12	0.13	0.91	0.01	0.24	0.17	1.67	0.39
44	1.86	1.29	0.13	0.14	0.94	0.01	0.24	0.20	1.75	0.42
45	1.90	1.35	0.15	0.14	0.96	0.01	0.25	0.23	1.83	0.44
46	1.95	1.42	0.17	0.15	0.99	0.01	0.26	0.26	1.91	0.47
47	1.99	1.49	0.18	0.15	1.02	0.01	0.28	0.30	1.99	0.51
48	2.03	1.56	0.21	0.15	1.05	0.01	0.29	0.35	2.07	0.55
49	2.07	1.63	0.23	0.16	1.07	0.01	0.30	0.41	2.15	0.59
50	2.11	1.70	0.26	0.16	1.10	0.02	0.32	0.48	2.23	0.63
51	2.15	1.78	0.29	0.17	1.13	0.02	0.34	0.56	2.31	0.68
52	2.19	1.85	0.32	0.17	1.16	0.02	0.36	0.65	2.39	0.74
53	2.24	1.94	0.36	0.17	1.19	0.02	0.39	0.76	2.48	0.80
54	2.29	2.02	0.40	0.17	1.21	0.03	0.42	0.89	2.58	0.87
55	2.33	2.12	0.45	0.17	1.23	0.03	0.45	1.05	2.69	0.95

Age	Loss of one limb	Loss of speech	Motor Neurone Disease	Major Organ Transplant	Multiple sclerosis	Multiple system atrophy	Open Heart Surgery	Parkinson's disease	Paralysis of limb(s)	Primary pulmonary hypertension
56	2.39	2.22	0.50	0.17	1.25	0.03	0.48	1.24	2.81	1.03
57	2.44	2.33	0.55	0.17	1.26	0.03	0.52	1.46	2.94	1.13
58	2.50	2.46	0.61	0.17	1.28	0.04	0.56	1.72	3.09	1.24
59	2.56	2.60	0.67	0.16	1.28	0.04	0.60	2.03	3.25	1.36
60	2.62	2.75	0.73	0.16	1.29	0.05	0.65	2.40	3.43	1.49
61	2.68	2.93	0.80	0.15	1.29	0.05	0.70	2.83	3.63	1.64
62	2.73	3.12	0.88	0.14	1.29	0.05	0.76	3.34	3.85	1.80
63	2.79	3.33	0.96	0.13	1.28	0.06	0.82	3.93	4.09	1.99
64	2.85	3.57	1.04	0.12	1.27	0.06	0.87	4.64	4.36	2.19
65	2.91	3.84	1.13	0.12	1.26	0.07	0.93	5.46	4.66	2.42
66	2.97	4.13	1.22	0.11	1.25	0.07	1.00	6.42	4.98	2.68
67	3.03	4.46	1.32	0.10	1.23	0.08	1.05	7.53	5.35	2.97
68	3.10	4.82	1.42	0.09	1.22	0.09	1.11	8.80	5.76	3.29
69	3.17	5.23	1.53	0.08	1.20	0.09	1.16	10.27	6.22	3.64
70	3.24	5.68	1.64	0.08	1.18	0.10	1.21	11.94	6.73	4.03
71	3.33	6.18	1.76	0.07	1.16	0.11	1.25	13.84	7.32	4.46
72	3.42	6.75	1.88	0.06	1.14	0.12	1.27	15.97	7.99	4.93
73	3.53	7.39	1.99	0.06	1.13	0.12	1.29	18.37	8.74	5.44
74	3.65	8.12	2.11	0.05	1.11	0.13	1.30	21.03	9.59	5.99
75	3.78	8.95	2.23	0.05	1.09	0.14	1.30	23.98	10.54	6.58
76	3.94	9.89	2.34	0.05	1.07	0.15	1.28	27.24	11.62	7.20
77	4.11	10.97	2.46	0.04	1.06	0.17	1.25	30.82	12.84	7.87
78	4.29	12.20	2.56	0.04	1.04	0.18	1.22	34.72	14.18	8.59
79	4.50	13.61	2.66	0.04	1.02	0.19	1.17	38.95	15.67	9.36
80	4.72	15.21	2.75	0.03	1.00	0.21	1.12	43.50	17.29	10.19
81	4.96	17.04	2.83	0.03	0.98	0.22	1.07	48.33	19.06	11.07
82	5.22	19.12	2.90	0.03	0.97	0.24	1.02	53.38	21.00	12.03
83	5.51	21.52	2.97	0.03	0.95	0.26	0.97	58.58	23.15	13.07
84	5.85	24.31	3.04	0.02	0.94	0.29	0.92	63.87	25.57	14.24
85	6.25	27.61	3.12	0.02	0.93	0.31	0.87	69.21	28.35	15.57

Age	Progressive supranuclear palsy	Respiratory failure	Rheumatoid arthritis	Systemic lupus erythematosus	Third Degree Burns	Traumatic Head Injury	Total Standalone Rate	Total Addition for Accelerated	Total Accelerated Rate
18	0.11	0.25	0.26	0.05	0.43	0.39	11.33	4.62	15.95
19	0.09	0.17	0.27	0.05	0.44	0.38	11.57	5.21	16.78
20	0.08	0.12	0.27	0.05	0.44	0.37	11.86	5.63	17.49
21	0.07	0.08	0.28	0.05	0.44	0.36	12.20	5.88	18.08
22	0.06	0.05	0.29	0.05	0.45	0.35	12.58	6.00	18.58
23	0.05	0.04	0.30	0.05	0.45	0.34	13.01	6.03	19.04
24	0.04	0.03	0.32	0.05	0.45	0.33	13.49	6.04	19.53
25	0.04	0.02	0.33	0.06	0.45	0.32	14.02	6.08	20.10
26	0.03	0.01	0.35	0.06	0.45	0.32	14.60	6.19	20.78
27	0.03	0.01	0.37	0.06	0.45	0.31	15.23	6.36	21.60
28	0.02	0.01	0.39	0.06	0.44	0.30	15.94	6.61	22.54
29	0.02	0.01	0.41	0.06	0.44	0.30	16.72	6.91	23.63
30	0.02	0.00	0.44	0.06	0.44	0.29	17.58	7.27	24.85
31	0.02	0.00	0.47	0.06	0.44	0.29	18.53	7.68	26.21
32	0.01	0.00	0.51	0.06	0.44	0.28	19.58	8.13	27.71
33	0.01	0.00	0.54	0.06	0.43	0.28	20.75	8.60	29.35
34	0.01	0.00	0.59	0.06	0.43	0.28	22.06	9.09	31.15
35	0.01	0.00	0.64	0.07	0.43	0.27	23.52	9.58	33.10
36	0.01	0.00	0.69	0.07	0.43	0.27	25.14	10.08	35.21
37	0.01	0.00	0.75	0.07	0.42	0.27	26.94	10.60	37.54
38	0.01	0.00	0.82	0.07	0.42	0.27	28.97	11.15	40.12
39	0.01	0.00	0.90	0.07	0.42	0.27	31.24	11.75	42.99
40	0.01	0.00	0.99	0.08	0.41	0.27	33.78	12.42	46.20
41	0.01	0.00	1.09	0.08	0.41	0.27	36.61	13.17	49.79
42	0.01	0.00	1.20	0.08	0.41	0.27	39.77	13.99	53.76
43	0.01	0.00	1.32	0.08	0.40	0.26	43.28	14.86	58.14
44	0.01	0.00	1.46	0.09	0.39	0.26	47.17	15.78	62.95
45	0.01	0.00	1.61	0.09	0.39	0.26	51.46	16.73	68.19
46	0.01	0.00	1.77	0.09	0.38	0.26	56.19	17.72	73.91
47	0.01	0.00	1.95	0.10	0.37	0.26	61.37	18.79	80.16
48	0.01	0.00	2.14	0.10	0.36	0.26	67.05	20.00	87.05
49	0.01	0.00	2.34	0.10	0.35	0.26	73.24	21.40	94.64
50	0.01	0.00	2.55	0.11	0.34	0.26	79.96	23.06	103.02
51	0.01	0.00	2.77	0.11	0.33	0.26	87.26	24.99	112.25
52	0.01	0.00	3.00	0.11	0.33	0.26	95.16	27.15	122.31
53	0.01	0.00	3.23	0.12	0.32	0.26	103.70	29.47	133.17
54	0.01	0.00	3.48	0.12	0.31	0.26	112.92	31.90	144.82
55	0.01	0.00	3.73	0.13	0.30	0.26	122.87	34.39	157.25

Age	Progressive supranuclear palsy	Respiratory failure	Rheumatoid arthritis	Systemic lupus erythematosus	Third Degree Burns	Traumatic Head Injury	Total Standalone Rate	Total Addition for Accelerated	Total Accelerated Rate
56	0.01	0.00	4.00	0.13	0.29	0.27	133.59	36.90	170.50
57	0.01	0.00	4.28	0.14	0.28	0.27	145.16	39.58	184.74
58	0.02	0.00	4.57	0.14	0.28	0.28	157.64	42.54	200.18
59	0.02	0.00	4.88	0.15	0.27	0.28	171.10	45.95	217.05
60	0.02	0.00	5.21	0.15	0.27	0.29	185.62	49.96	235.58
61	0.02	0.00	5.56	0.15	0.27	0.30	201.29	54.70	255.98
62	0.02	0.00	5.92	0.16	0.26	0.31	218.10	60.18	278.28
63	0.03	0.00	6.30	0.16	0.26	0.32	236.07	66.39	302.46
64	0.03	0.00	6.70	0.17	0.26	0.33	255.19	73.31	328.50
65	0.03	0.00	7.10	0.17	0.26	0.35	275.49	80.91	356.41
66	0.02	0.00	7.52	0.18	0.27	0.37	297.01	89.21	386.22
67	0.01	0.00	7.95	0.19	0.27	0.39	319.86	98.37	418.23
68	0.01	0.00	8.38	0.19	0.28	0.41	344.20	108.63	452.83
69	0.00	0.00	8.84	0.20	0.29	0.44	370.19	120.27	490.46
70	0.00	0.00	9.31	0.20	0.29	0.47	398.03	133.61	531.64
71	0.00	0.00	9.79	0.21	0.31	0.51	427.97	149.02	576.99
72	0.00	0.00	10.29	0.21	0.32	0.55	460.38	166.91	627.30
73	0.00	0.00	10.82	0.22	0.34	0.59	495.69	187.78	683.46
74	0.00	0.00	11.37	0.23	0.36	0.65	534.44	212.02	746.46
75	0.00	0.00	11.95	0.23	0.38	0.71	577.24	240.09	817.33
76	0.00	0.00	12.56	0.24	0.41	0.77	624.71	272.48	897.19
77	0.00	0.00	13.21	0.25	0.44	0.85	677.37	309.87	987.24
78	0.00	0.00	13.89	0.25	0.47	0.93	735.73	353.06	1088.80
79	0.00	0.00	14.59	0.26	0.51	1.02	800.19	403.11	1203.30
80	0.00	0.00	15.33	0.27	0.55	1.13	871.11	461.18	1332.30
81	0.00	0.00	16.10	0.27	0.61	1.24	949.10	528.62	1477.72
82	0.00	0.00	16.90	0.28	0.66	1.36	1036.25	606.83	1643.08
83	0.00	0.00	17.79	0.29	0.73	1.50	1135.66	697.41	1833.07
84	0.00	0.00	18.78	0.30	0.80	1.66	1251.63	802.01	2053.64
85	0.00	0.00	19.95	0.31	0.90	1.85	1390.03	922.63	2312.65

Appendix 6.02 Additional Male Rates for Conditions not Included in CIBT08

All rates per 10,000.

Age	Coronary angioplasty (single vessel)	Ductal carcinoma in situ	Prostate cancer
18	0.00	0.00	0.00
19	0.00	0.00	0.00
20	0.00	0.00	0.00
21	0.00	0.00	0.00
22	0.00	0.00	0.00
23	0.00	0.00	0.00
24	0.00	0.00	0.00
25	0.01	0.00	0.00
26	0.01	0.00	0.01
27	0.01	0.00	0.01
28	0.01	0.00	0.01
29	0.01	0.00	0.01
30	0.01	0.00	0.01
31	0.02	0.00	0.01
32	0.02	0.00	0.02
33	0.03	0.00	0.02
34	0.03	0.00	0.03
35	0.04	0.00	0.04
36	0.05	0.00	0.04
37	0.06	0.00	0.06
38	0.08	0.00	0.07
39	0.09	0.00	0.09
40	0.11	0.00	0.12
41	0.14	0.00	0.15
42	0.16	0.00	0.20
43	0.19	0.00	0.26
44	0.23	0.00	0.36
45	0.27	0.00	0.49
46	0.32	0.00	0.66
47	0.37	0.00	0.89
48	0.43	0.00	1.19
49	0.49	0.00	1.58
50	0.55	0.00	2.06

51	0.62	0.00	2.67
52	0.69	0.00	3.41
53	0.76	0.00	4.31
54	0.83	0.00	5.40
55	0.90	0.00	6.70
56	0.97	0.00	8.22
57	1.03	0.00	9.94
58	1.08	0.00	11.84
59	1.14	0.00	13.88
60	1.18	0.00	16.03
61	1.22	0.00	18.28
62	1.25	0.00	20.56
63	1.28	0.00	22.84
64	1.31	0.00	25.06
65	1.33	0.00	27.18
66	1.34	0.00	29.16
67	1.35	0.00	30.99
68	1.35	0.00	32.68
69	1.34	0.00	34.22
70	1.33	0.00	35.61
71	1.30	0.00	36.87
72	1.27	0.00	38.01
73	1.23	0.00	39.05
74	1.18	0.01	40.03
75	1.12	0.01	40.97
76	1.06	0.01	41.90
77	0.98	0.01	42.88
78	0.90	0.01	43.97
79	0.82	0.01	45.23
80	0.74	0.01	46.73
81	0.66	0.02	48.49
82	0.58	0.02	50.40
83	0.51	0.02	52.31
84	0.44	0.02	54.07
85	0.38	0.02	55.54

Appendix 6.03 CIBT08 Female Rates – By condition

All rates per 10,000.

Age	Cancer	Heart attack	Stroke	Aplastic anaemia	Aorta graft surgery	Alzheimer's disease	Benign brain tumour	Blindness	Bacterial meningitis	Coronary artery bypass graft
18	2.17	0.02	0.34	0.15	0.01	0.00	0.48	0.11	0.29	0.00
19	2.39	0.02	0.37	0.15	0.01	0.00	0.49	0.11	0.29	0.00
20	2.63	0.03	0.40	0.16	0.01	0.00	0.51	0.11	0.28	0.00
21	2.90	0.03	0.44	0.16	0.01	0.00	0.52	0.11	0.28	0.00
22	3.19	0.04	0.48	0.16	0.02	0.00	0.54	0.11	0.27	0.00
23	3.52	0.05	0.52	0.16	0.02	0.00	0.55	0.12	0.27	0.00
24	3.88	0.06	0.57	0.17	0.02	0.00	0.57	0.12	0.26	0.00
25	4.28	0.08	0.62	0.17	0.02	0.00	0.59	0.12	0.26	0.01
26	4.72	0.09	0.68	0.17	0.02	0.00	0.61	0.12	0.25	0.01
27	5.20	0.11	0.74	0.17	0.02	0.00	0.63	0.12	0.25	0.01
28	5.72	0.13	0.81	0.17	0.02	0.00	0.65	0.12	0.24	0.01
29	6.29	0.16	0.88	0.17	0.02	0.00	0.68	0.13	0.24	0.01
30	6.92	0.20	0.96	0.18	0.02	0.00	0.70	0.13	0.24	0.01
31	7.59	0.24	1.05	0.18	0.03	0.00	0.73	0.13	0.23	0.02
32	8.33	0.29	1.14	0.18	0.03	0.01	0.76	0.13	0.23	0.02
33	9.13	0.34	1.24	0.18	0.03	0.01	0.80	0.13	0.22	0.02
34	10.01	0.41	1.35	0.18	0.03	0.01	0.83	0.14	0.22	0.03
35	10.98	0.49	1.47	0.18	0.03	0.01	0.87	0.14	0.22	0.03
36	12.04	0.58	1.60	0.19	0.03	0.01	0.90	0.14	0.21	0.04
37	13.20	0.69	1.75	0.19	0.04	0.01	0.94	0.15	0.21	0.05
38	14.49	0.82	1.91	0.19	0.04	0.01	0.98	0.15	0.21	0.06
39	15.91	0.96	2.08	0.19	0.04	0.02	1.02	0.15	0.20	0.07
40	17.48	1.13	2.27	0.20	0.05	0.02	1.06	0.16	0.20	0.08
41	19.20	1.32	2.48	0.20	0.05	0.02	1.11	0.16	0.20	0.10
42	21.10	1.53	2.71	0.21	0.05	0.03	1.15	0.17	0.20	0.12
43	23.16	1.77	2.96	0.21	0.06	0.03	1.20	0.17	0.20	0.14
44	25.42	2.03	3.22	0.22	0.06	0.04	1.25	0.18	0.20	0.17
45	27.86	2.31	3.50	0.22	0.07	0.05	1.30	0.19	0.21	0.20
46	30.49	2.62	3.79	0.23	0.07	0.06	1.35	0.19	0.21	0.23
47	33.30	2.96	4.09	0.24	0.08	0.07	1.40	0.20	0.21	0.27
48	36.26	3.31	4.39	0.25	0.09	0.08	1.46	0.21	0.22	0.32
49	39.35	3.69	4.69	0.26	0.10	0.10	1.52	0.22	0.22	0.37
50	42.54	4.09	4.99	0.27	0.11	0.11	1.57	0.23	0.23	0.43

Age	Cancer	Heart attack	Stroke	Aplastic anaemia	Aorta graft surgery	Alzheimer's disease	Benign brain tumour	Blindness	Bacterial meningitis	Coronary artery bypass graft
51	45.81	4.51	5.29	0.28	0.12	0.14	1.63	0.23	0.23	0.49
52	49.19	4.95	5.59	0.29	0.13	0.16	1.69	0.24	0.24	0.56
53	52.68	5.41	5.90	0.31	0.15	0.19	1.75	0.25	0.25	0.64
54	56.31	5.90	6.23	0.32	0.16	0.23	1.81	0.26	0.26	0.73
55	60.10	6.41	6.58	0.34	0.18	0.28	1.86	0.27	0.27	0.83
56	64.07	6.95	6.95	0.36	0.20	0.33	1.92	0.29	0.27	0.94
57	68.24	7.53	7.36	0.38	0.23	0.40	1.98	0.30	0.28	1.06
58	72.65	8.15	7.82	0.40	0.26	0.48	2.04	0.31	0.29	1.20
59	77.32	8.82	8.34	0.42	0.29	0.57	2.10	0.33	0.30	1.35
60	82.28	9.53	8.95	0.44	0.33	0.69	2.15	0.34	0.31	1.52
61	87.53	10.31	9.65	0.47	0.37	0.82	2.20	0.36	0.31	1.72
62	92.95	11.15	10.47	0.50	0.42	0.99	2.25	0.38	0.32	1.94
63	98.40	12.07	11.42	0.53	0.47	1.20	2.30	0.41	0.32	2.18
64	103.71	13.06	12.51	0.56	0.53	1.45	2.34	0.44	0.33	2.46
65	108.73	14.13	13.77	0.59	0.60	1.77	2.38	0.47	0.33	2.76
66	113.32	15.29	15.21	0.63	0.68	2.15	2.41	0.51	0.33	3.11
67	117.55	16.55	16.85	0.67	0.76	2.64	2.45	0.55	0.34	3.48
68	121.51	17.90	18.69	0.72	0.85	3.25	2.48	0.60	0.34	3.88
69	125.32	19.36	20.76	0.77	0.94	4.01	2.52	0.66	0.34	4.31
70	129.09	20.93	23.08	0.82	1.03	4.97	2.56	0.73	0.34	4.77
71	132.92	22.61	25.66	0.88	1.13	6.15	2.60	0.82	0.34	5.24
72	136.88	24.42	28.55	0.94	1.23	7.61	2.66	0.92	0.34	5.70
73	141.01	26.36	31.79	1.01	1.32	9.38	2.71	1.04	0.34	6.09
74	145.37	28.43	35.43	1.08	1.42	11.52	2.78	1.19	0.34	6.37
75	150.00	30.66	39.49	1.16	1.50	14.08	2.85	1.36	0.34	6.50
76	154.93	33.03	44.02	1.25	1.57	17.11	2.94	1.58	0.34	6.44
77	160.03	35.59	49.02	1.34	1.62	20.61	3.03	1.84	0.34	6.20
78	165.13	38.33	54.47	1.44	1.64	24.54	3.14	2.16	0.34	5.82
79	170.05	41.29	60.39	1.54	1.62	28.90	3.26	2.57	0.34	5.33
80	174.59	44.48	66.77	1.65	1.56	33.67	3.39	3.07	0.34	4.76
81	178.57	47.92	73.64	1.75	1.43	38.79	3.53	3.69	0.33	4.15
82	181.89	51.65	81.05	1.86	1.27	44.10	3.69	4.47	0.33	3.52
83	184.46	55.72	89.02	1.98	1.08	49.42	3.87	5.49	0.33	2.91
84	186.20	60.17	97.71	2.09	0.88	54.54	4.05	6.80	0.33	2.33
85	187.01	65.05	107.21	2.21	0.70	59.24	4.25	8.48	0.33	1.82

Age	Cardiomyopathy	CJD	Coma	Deafness	Dementia	Encephalitis	HIV infection	Heart Value replacement or repair	Kidney failure	Liver failure
18	0.19	0.00	0.00	1.38	0.03	0.26	0.28	0.19	0.02	0.21
19	0.20	0.00	0.00	1.37	0.03	0.26	0.32	0.20	0.02	0.21
20	0.21	0.00	0.00	1.37	0.03	0.26	0.35	0.20	0.02	0.22
21	0.23	0.00	0.01	1.37	0.03	0.26	0.39	0.21	0.02	0.22
22	0.24	0.00	0.01	1.37	0.03	0.26	0.44	0.21	0.02	0.23
23	0.26	0.00	0.01	1.38	0.04	0.26	0.48	0.22	0.02	0.23
24	0.27	0.00	0.01	1.39	0.04	0.26	0.53	0.22	0.02	0.24
25	0.29	0.00	0.01	1.40	0.04	0.26	0.58	0.22	0.03	0.25
26	0.31	0.00	0.01	1.41	0.04	0.27	0.64	0.23	0.03	0.26
27	0.33	0.00	0.01	1.43	0.04	0.27	0.69	0.23	0.03	0.26
28	0.34	0.00	0.01	1.45	0.05	0.27	0.75	0.24	0.03	0.27
29	0.36	0.00	0.01	1.47	0.05	0.27	0.79	0.24	0.03	0.28
30	0.38	0.00	0.01	1.49	0.05	0.28	0.84	0.24	0.03	0.30
31	0.40	0.00	0.01	1.52	0.06	0.28	0.87	0.25	0.03	0.31
32	0.41	0.00	0.01	1.54	0.06	0.28	0.90	0.25	0.03	0.32
33	0.43	0.00	0.01	1.57	0.07	0.29	0.92	0.26	0.03	0.34
34	0.45	0.00	0.01	1.60	0.07	0.29	0.93	0.26	0.03	0.35
35	0.47	0.00	0.01	1.62	0.08	0.29	0.93	0.27	0.04	0.37
36	0.48	0.00	0.01	1.65	0.08	0.29	0.92	0.28	0.04	0.39
37	0.50	0.00	0.01	1.67	0.09	0.30	0.89	0.28	0.04	0.41
38	0.51	0.00	0.01	1.69	0.09	0.30	0.86	0.29	0.04	0.43
39	0.53	0.00	0.01	1.71	0.10	0.30	0.82	0.30	0.04	0.45
40	0.55	0.00	0.01	1.73	0.11	0.30	0.78	0.32	0.04	0.47
41	0.57	0.00	0.01	1.76	0.12	0.31	0.74	0.33	0.05	0.50
42	0.59	0.00	0.01	1.78	0.14	0.31	0.69	0.35	0.05	0.52
43	0.61	0.00	0.01	1.80	0.15	0.31	0.64	0.37	0.05	0.55
44	0.64	0.00	0.01	1.83	0.17	0.31	0.59	0.39	0.05	0.57
45	0.66	0.00	0.01	1.86	0.19	0.32	0.54	0.41	0.06	0.60
46	0.70	0.00	0.01	1.90	0.21	0.32	0.50	0.44	0.06	0.63
47	0.73	0.00	0.01	1.94	0.24	0.33	0.45	0.47	0.07	0.65
48	0.77	0.00	0.01	1.99	0.27	0.33	0.41	0.51	0.07	0.68
49	0.82	0.00	0.01	2.04	0.31	0.33	0.38	0.55	0.07	0.71
50	0.86	0.00	0.01	2.09	0.35	0.34	0.34	0.60	0.08	0.73
51	0.92	0.00	0.01	2.14	0.39	0.34	0.31	0.65	0.09	0.76
52	0.97	0.00	0.01	2.19	0.45	0.35	0.28	0.71	0.09	0.78
53	1.03	0.00	0.01	2.25	0.51	0.35	0.25	0.78	0.10	0.81
54	1.09	0.00	0.01	2.31	0.58	0.35	0.23	0.85	0.11	0.83
55	1.16	0.00	0.01	2.36	0.66	0.36	0.21	0.94	0.12	0.85

Age	Cardiomyopathy	CJD	Coma	Deafness	Dementia	Encephalitis	HIV infection	Heart Value replacement or repair	Kidney failure	Liver failure
56	1.23	0.00	0.01	2.42	0.75	0.36	0.19	1.04	0.13	0.87
57	1.30	0.00	0.01	2.48	0.85	0.37	0.17	1.15	0.13	0.89
58	1.37	0.00	0.01	2.54	0.97	0.37	0.15	1.28	0.14	0.91
59	1.45	0.00	0.01	2.60	1.12	0.38	0.14	1.42	0.16	0.92
60	1.53	0.00	0.01	2.68	1.29	0.39	0.12	1.59	0.17	0.94
61	1.61	0.00	0.01	2.76	1.49	0.39	0.11	1.78	0.18	0.95
62	1.70	0.00	0.01	2.85	1.74	0.40	0.10	1.99	0.19	0.97
63	1.79	0.00	0.01	2.96	2.05	0.40	0.09	2.23	0.20	0.99
64	1.88	0.00	0.01	3.08	2.43	0.41	0.08	2.49	0.22	1.00
65	1.98	0.00	0.01	3.21	2.91	0.42	0.07	2.80	0.23	1.02
66	2.08	0.00	0.01	3.36	3.51	0.42	0.07	3.13	0.24	1.04
67	2.17	0.00	0.01	3.52	4.28	0.43	0.06	3.50	0.26	1.06
68	2.27	0.00	0.02	3.71	5.29	0.44	0.05	3.90	0.27	1.09
69	2.37	0.00	0.02	3.92	6.60	0.45	0.05	4.33	0.28	1.11
70	2.46	0.00	0.02	4.15	8.28	0.45	0.04	4.79	0.29	1.14
71	2.54	0.00	0.02	4.41	10.42	0.46	0.04	5.28	0.31	1.17
72	2.61	0.00	0.02	4.71	13.19	0.47	0.04	5.77	0.32	1.20
73	2.67	0.00	0.02	5.06	16.78	0.47	0.03	6.26	0.30	1.23
74	2.72	0.00	0.02	5.46	21.40	0.48	0.03	6.71	0.27	1.26
75	2.76	0.00	0.02	5.92	27.25	0.48	0.03	7.10	0.25	1.29
76	2.78	0.00	0.02	6.48	34.54	0.49	0.02	7.42	0.22	1.33
77	2.78	0.00	0.02	7.14	43.35	0.49	0.02	7.64	0.20	1.36
78	2.78	0.00	0.02	7.95	53.75	0.50	0.02	7.73	0.18	1.39
79	2.76	0.00	0.02	8.95	65.80	0.50	0.02	7.66	0.15	1.42
80	2.74	0.00	0.02	10.18	79.56	0.50	0.02	7.41	0.13	1.45
81	2.71	0.00	0.02	11.70	95.17	0.51	0.01	6.95	0.11	1.47
82	2.68	0.00	0.02	13.68	113.05	0.51	0.01	6.33	0.09	1.50
83	2.65	0.00	0.02	16.33	133.71	0.51	0.01	5.60	0.08	1.53
84	2.61	0.00	0.02	19.85	157.71	0.50	0.01	4.84	0.06	1.56
85	2.58	0.00	0.02	24.49	185.64	0.50	0.01	4.10	0.05	1.59

Age	Loss of one limb	Loss of speech	Motor Neurone Disease	Major Organ Transplant	Multiple sclerosis	Multiple system atrophy	Open Heart Surgery	Parkinson's disease	Paralysis of limb(s)	Primary pulmonary hypertension
18	0.21	0.52	0.01	0.05	0.24	0.00	0.19	0.01	0.67	0.13
19	0.21	0.55	0.01	0.06	0.29	0.00	0.20	0.01	0.69	0.14
20	0.21	0.58	0.01	0.06	0.35	0.00	0.20	0.01	0.71	0.15
21	0.21	0.61	0.01	0.06	0.42	0.00	0.20	0.01	0.74	0.16
22	0.21	0.64	0.01	0.06	0.51	0.00	0.20	0.01	0.76	0.16
23	0.21	0.66	0.01	0.06	0.60	0.00	0.20	0.01	0.78	0.17
24	0.21	0.69	0.01	0.06	0.70	0.00	0.20	0.01	0.81	0.18
25	0.22	0.72	0.01	0.06	0.81	0.00	0.20	0.01	0.84	0.19
26	0.22	0.75	0.01	0.06	0.93	0.00	0.20	0.02	0.87	0.20
27	0.22	0.78	0.01	0.07	1.05	0.00	0.19	0.02	0.90	0.21
28	0.23	0.81	0.01	0.07	1.18	0.00	0.19	0.02	0.93	0.23
29	0.23	0.84	0.02	0.07	1.30	0.00	0.19	0.02	0.96	0.24
30	0.24	0.87	0.02	0.07	1.41	0.00	0.19	0.02	0.99	0.25
31	0.24	0.89	0.02	0.07	1.51	0.00	0.19	0.03	1.02	0.26
32	0.25	0.92	0.02	0.07	1.60	0.00	0.19	0.03	1.05	0.27
33	0.26	0.95	0.02	0.08	1.68	0.00	0.18	0.03	1.09	0.28
34	0.27	0.98	0.03	0.08	1.76	0.00	0.18	0.04	1.13	0.29
35	0.28	1.01	0.03	0.08	1.82	0.00	0.18	0.04	1.16	0.31
36	0.29	1.04	0.03	0.08	1.87	0.00	0.18	0.05	1.21	0.32
37	0.31	1.08	0.04	0.08	1.92	0.00	0.18	0.05	1.25	0.34
38	0.32	1.11	0.04	0.08	1.97	0.00	0.18	0.06	1.30	0.35
39	0.33	1.14	0.04	0.08	2.02	0.00	0.18	0.07	1.35	0.37
40	0.35	1.18	0.05	0.09	2.06	0.01	0.18	0.08	1.40	0.39
41	0.36	1.22	0.05	0.09	2.11	0.01	0.18	0.09	1.45	0.41
42	0.38	1.26	0.06	0.09	2.17	0.01	0.18	0.10	1.51	0.43
43	0.39	1.29	0.07	0.09	2.22	0.01	0.18	0.11	1.56	0.45
44	0.40	1.33	0.08	0.09	2.28	0.01	0.19	0.13	1.61	0.48
45	0.42	1.37	0.08	0.10	2.33	0.01	0.19	0.15	1.66	0.51
46	0.43	1.40	0.09	0.10	2.39	0.01	0.20	0.17	1.71	0.54
47	0.44	1.43	0.11	0.10	2.45	0.01	0.20	0.19	1.76	0.58
48	0.46	1.47	0.12	0.10	2.50	0.01	0.21	0.22	1.80	0.62
49	0.47	1.49	0.13	0.11	2.55	0.01	0.21	0.26	1.85	0.66
50	0.48	1.52	0.15	0.11	2.59	0.01	0.22	0.30	1.89	0.71
51	0.49	1.55	0.17	0.11	2.62	0.01	0.23	0.34	1.93	0.76
52	0.50	1.57	0.19	0.11	2.64	0.02	0.24	0.40	1.97	0.81
53	0.51	1.60	0.22	0.11	2.66	0.02	0.25	0.47	2.01	0.87
54	0.52	1.63	0.25	0.11	2.66	0.02	0.26	0.54	2.06	0.94
55	0.53	1.67	0.28	0.11	2.66	0.02	0.27	0.64	2.10	1.02

Age	Loss of one limb	Loss of speech	Motor Neurone Disease	Major Organ Transplant	Multiple sclerosis	Multiple system atrophy	Open Heart Surgery	Parkinson's disease	Paralysis of limb(s)	Primary pulmonary hypertension
56	0.54	1.71	0.31	0.11	2.64	0.02	0.29	0.74	2.16	1.10
57	0.56	1.76	0.35	0.11	2.62	0.03	0.30	0.87	2.21	1.20
58	0.57	1.82	0.40	0.11	2.59	0.03	0.32	1.02	2.28	1.30
59	0.59	1.89	0.45	0.11	2.56	0.03	0.34	1.20	2.36	1.42
60	0.61	1.96	0.50	0.11	2.52	0.03	0.37	1.40	2.45	1.55
61	0.63	2.06	0.56	0.11	2.49	0.04	0.39	1.65	2.56	1.70
62	0.66	2.16	0.61	0.10	2.45	0.04	0.42	1.93	2.68	1.87
63	0.68	2.29	0.68	0.10	2.41	0.04	0.45	2.26	2.83	2.06
64	0.71	2.43	0.74	0.09	2.37	0.04	0.48	2.64	3.00	2.27
65	0.74	2.60	0.80	0.09	2.32	0.05	0.52	3.08	3.19	2.51
66	0.78	2.79	0.87	0.09	2.28	0.05	0.56	3.58	3.40	2.77
67	0.81	3.02	0.94	0.08	2.23	0.05	0.60	4.15	3.65	3.07
68	0.85	3.27	1.00	0.08	2.18	0.06	0.64	4.80	3.93	3.39
69	0.90	3.57	1.07	0.07	2.13	0.06	0.67	5.53	4.24	3.74
70	0.95	3.90	1.13	0.07	2.08	0.06	0.71	6.34	4.58	4.11
71	1.00	4.29	1.19	0.07	2.02	0.07	0.75	7.25	4.97	4.51
72	1.06	4.72	1.24	0.06	1.96	0.07	0.77	8.26	5.40	4.93
73	1.13	5.21	1.29	0.06	1.90	0.08	0.79	9.36	5.88	5.36
74	1.20	5.76	1.33	0.06	1.83	0.08	0.80	10.56	6.41	5.80
75	1.29	6.36	1.36	0.05	1.76	0.08	0.79	11.86	6.99	6.23
76	1.37	7.04	1.39	0.05	1.68	0.09	0.77	13.25	7.65	6.66
77	1.47	7.79	1.40	0.05	1.61	0.09	0.73	14.69	8.36	7.08
78	1.58	8.61	1.40	0.04	1.53	0.10	0.68	16.13	9.13	7.49
79	1.69	9.52	1.38	0.04	1.45	0.10	0.62	17.52	9.95	7.89
80	1.81	10.52	1.36	0.04	1.37	0.10	0.56	18.81	10.83	8.26
81	1.93	11.61	1.33	0.04	1.29	0.11	0.49	19.94	11.76	8.62
82	2.07	12.82	1.29	0.03	1.22	0.11	0.44	20.88	12.73	8.96
83	2.21	14.14	1.24	0.03	1.14	0.12	0.38	21.62	13.76	9.27
84	2.35	15.61	1.19	0.03	1.07	0.12	0.33	22.12	14.84	9.56
85	2.50	17.23	1.13	0.03	1.01	0.13	0.28	22.38	15.97	9.83

Age	Progressive supranuclear palsy	Respiratory failure	Rheumatoid arthritis	Systemic lupus erythematous	Third Degree Burns	Traumatic Head Injury	Total Standalone Rate	Total Addition for Accelerated	Total Accelerated Rate
18	0.00	0.00	0.70	0.33	0.20	0.05	9.46	1.81	11.27
19	0.00	0.00	0.74	0.37	0.20	0.05	9.95	1.91	11.86
20	0.00	0.00	0.78	0.40	0.20	0.05	10.51	1.95	12.46
21	0.00	0.00	0.82	0.44	0.19	0.05	11.12	1.96	13.08
22	0.00	0.00	0.87	0.48	0.19	0.05	11.79	1.96	13.75
23	0.00	0.00	0.93	0.52	0.19	0.05	12.52	1.96	14.48
24	0.00	0.00	1.00	0.56	0.19	0.05	13.32	1.97	15.29
25	0.00	0.00	1.07	0.60	0.19	0.05	14.20	2.02	16.21
26	0.00	0.00	1.14	0.64	0.19	0.05	15.14	2.11	17.25
27	0.00	0.00	1.23	0.68	0.19	0.05	16.16	2.24	18.40
28	0.00	0.00	1.32	0.72	0.19	0.05	17.24	2.39	19.63
29	0.00	0.00	1.42	0.75	0.19	0.05	18.38	2.56	20.94
30	0.00	0.00	1.53	0.78	0.19	0.05	19.59	2.73	22.32
31	0.00	0.00	1.64	0.81	0.19	0.05	20.85	2.89	23.75
32	0.00	0.00	1.76	0.83	0.19	0.05	22.18	3.07	25.25
33	0.00	0.00	1.88	0.85	0.19	0.05	23.58	3.26	26.84
34	0.00	0.00	2.01	0.86	0.19	0.05	25.07	3.47	28.54
35	0.00	0.00	2.14	0.87	0.19	0.05	26.66	3.71	30.37
36	0.00	0.00	2.28	0.87	0.19	0.05	28.36	3.98	32.34
37	0.00	0.00	2.42	0.88	0.19	0.05	30.19	4.26	34.45
38	0.00	0.00	2.57	0.88	0.19	0.05	32.19	4.55	36.74
39	0.00	0.00	2.72	0.88	0.19	0.06	34.37	4.86	39.23
40	0.00	0.00	2.89	0.88	0.19	0.06	36.76	5.18	41.94
41	0.00	0.00	3.08	0.88	0.19	0.06	39.39	5.52	44.91
42	0.00	0.00	3.28	0.88	0.19	0.06	42.27	5.89	48.16
43	0.00	0.00	3.50	0.89	0.19	0.06	45.41	6.28	51.69
44	0.00	0.00	3.74	0.89	0.19	0.06	48.83	6.71	55.53
45	0.00	0.00	4.01	0.89	0.19	0.06	52.53	7.17	59.70
46	0.00	0.00	4.31	0.90	0.18	0.06	56.52	7.66	64.18
47	0.01	0.00	4.64	0.90	0.18	0.07	60.79	8.20	68.98
48	0.01	0.00	5.00	0.91	0.18	0.07	65.29	8.79	74.08
49	0.01	0.00	5.40	0.92	0.18	0.07	70.02	9.46	79.47
50	0.01	0.00	5.83	0.92	0.17	0.07	74.93	10.20	85.14
51	0.01	0.00	6.29	0.93	0.17	0.07	80.03	11.04	91.07
52	0.01	0.00	6.78	0.93	0.17	0.08	85.31	11.97	97.28
53	0.01	0.00	7.30	0.94	0.16	0.08	90.83	12.96	103.79
54	0.01	0.00	7.82	0.95	0.16	0.08	96.60	14.02	110.62
55	0.01	0.00	8.36	0.95	0.16	0.08	102.64	15.13	117.77

Age	Progressive supranuclear palsy	Respiratory failure	Rheumatoid arthritis	Systemic lupus erythematous	Third Degree Burns	Traumatic Head Injury	Total Standalone Rate	Total Addition for Accelerated	Total Accelerated Rate
56	0.01	0.01	8.89	0.96	0.15	0.09	109.01	16.29	125.30
57	0.01	0.01	9.43	0.96	0.15	0.09	115.76	17.53	133.30
58	0.01	0.01	9.97	0.96	0.15	0.09	122.97	18.94	141.90
59	0.01	0.01	10.51	0.96	0.14	0.10	130.70	20.55	151.26
60	0.01	0.01	11.06	0.96	0.14	0.10	139.04	22.46	161.50
61	0.01	0.01	11.61	0.96	0.14	0.10	148.04	24.72	172.76
62	0.01	0.01	12.18	0.96	0.14	0.11	157.65	27.38	185.03
63	0.01	0.01	12.75	0.95	0.14	0.11	167.78	30.47	198.25
64	0.01	0.01	13.34	0.95	0.14	0.12	178.34	34.03	212.37
65	0.02	0.01	13.94	0.94	0.14	0.13	189.25	38.08	227.33
66	0.02	0.01	14.56	0.93	0.15	0.13	200.46	42.68	243.14
67	0.02	0.01	15.19	0.91	0.15	0.14	212.14	47.93	260.07
68	0.02	0.00	15.83	0.90	0.15	0.15	224.50	54.00	278.50
69	0.02	0.00	16.49	0.88	0.16	0.16	237.80	61.07	298.86
70	0.02	0.00	17.16	0.86	0.17	0.17	252.27	69.32	321.59
71	0.02	0.00	17.85	0.84	0.17	0.18	268.18	79.00	347.19
72	0.02	0.00	18.54	0.82	0.18	0.19	285.80	90.39	376.19
73	0.02	0.01	19.25	0.79	0.19	0.20	305.39	103.74	409.13
74	0.03	0.01	19.97	0.77	0.20	0.22	327.27	119.28	446.55
75	0.03	0.02	20.69	0.74	0.21	0.23	351.74	137.22	488.97
76	0.03	0.02	21.41	0.71	0.23	0.24	379.07	157.75	536.83
77	0.03	0.02	22.13	0.68	0.24	0.26	409.24	181.29	590.53
78	0.03	0.01	22.85	0.65	0.26	0.27	442.09	208.47	650.56
79	0.03	0.01	23.55	0.62	0.28	0.29	477.52	240.04	717.56
80	0.03	0.01	24.24	0.59	0.31	0.30	515.41	276.98	792.39
81	0.03	0.01	24.89	0.55	0.33	0.32	555.72	320.49	876.21
82	0.03	0.00	25.51	0.52	0.36	0.33	599.02	371.65	970.67
83	0.04	0.00	26.09	0.49	0.39	0.34	645.96	431.57	1077.53
84	0.04	0.00	26.61	0.46	0.42	0.36	697.39	501.17	1198.56
85	0.04	0.00	27.06	0.43	0.46	0.37	754.13	581.34	1335.47

Appendix 6.04 Additional Female Rates for Conditions not Included in CIBT08

All rates per 10,000.

Age	Coronary angioplasty (single vessel)	Ductal carcinoma in situ
18	0.05	0.67
19	0.05	0.69
20	0.05	0.71
21	0.05	0.74
22	0.05	0.76
23	0.05	0.78
24	0.05	0.81
25	0.05	0.84
26	0.05	0.87
27	0.05	0.90
28	0.05	0.93
29	0.05	0.96
30	0.05	0.99
31	0.05	1.02
32	0.05	1.05
33	0.05	1.09
34	0.05	1.13
35	0.05	1.16
36	0.05	1.21
37	0.05	1.25
38	0.05	1.30
39	0.06	1.35
40	0.06	1.40
41	0.06	1.45
42	0.06	1.51
43	0.06	1.56
44	0.06	1.61
45	0.06	1.66
46	0.06	1.71
47	0.07	1.76
48	0.07	1.80
49	0.07	1.85
50	0.07	1.89

Age	Coronary angioplasty (single vessel)	Ductal carcinoma in situ
51	0.07	1.93
52	0.08	1.97
53	0.08	2.01
54	0.08	2.06
55	0.08	2.10
56	0.09	2.16
57	0.09	2.21
58	0.09	2.28
59	0.10	2.36
60	0.10	2.45
61	0.10	2.56
62	0.11	2.68
63	0.11	2.83
64	0.12	3.00
65	0.13	3.19
66	0.13	3.40
67	0.14	3.65
68	0.15	3.93
69	0.16	4.24
70	0.17	4.58
71	0.18	4.97
72	0.19	5.40
73	0.20	5.88
74	0.22	6.41
75	0.23	6.99
76	0.24	7.65
77	0.26	8.36
78	0.27	9.13
79	0.29	9.95
80	0.30	10.83
81	0.32	11.76
82	0.33	12.73
83	0.34	13.76
84	0.36	14.84
85	0.37	15.97

Appendix 6.05 Detailed Calculations by Age for Cancer, Heart Attack and Stroke

Male Cancer Rates, per 10,000

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates I'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
18	3.19	6.5%	0.2%	100.0%	2.99	0.4%	2.98	5.22	9.1%	2.51
19	3.34	6.5%	0.2%	100.0%	3.13	0.5%	3.11	5.84	8.6%	2.63
20	3.50	6.5%	0.2%	100.0%	3.28	0.5%	3.26	6.28	8.2%	2.76
21	3.66	6.5%	0.2%	100.0%	3.43	0.5%	3.42	6.53	8.1%	2.91
22	3.84	6.5%	0.3%	100.0%	3.60	0.5%	3.58	6.65	8.0%	3.06
23	4.02	6.6%	0.3%	100.0%	3.77	0.5%	3.75	6.69	8.1%	3.23
24	4.22	6.6%	0.3%	100.0%	3.95	0.5%	3.93	6.70	8.2%	3.40
25	4.42	6.6%	0.3%	100.0%	4.14	0.5%	4.12	6.75	8.4%	3.57
26	4.64	6.7%	0.4%	100.0%	4.35	0.5%	4.32	6.89	8.7%	3.75
27	4.87	6.7%	0.4%	100.0%	4.56	0.6%	4.53	7.12	9.0%	3.92
28	5.11	6.7%	0.5%	100.0%	4.79	0.6%	4.76	7.44	9.3%	4.10
29	5.37	6.8%	0.5%	100.0%	5.03	0.6%	5.00	7.83	9.6%	4.28
30	5.64	6.9%	0.6%	100.0%	5.28	0.6%	5.25	8.30	9.9%	4.46
31	5.93	6.9%	0.7%	100.0%	5.56	0.6%	5.52	8.85	10.2%	4.66
32	6.25	7.0%	0.7%	100.0%	5.85	0.6%	5.82	9.45	10.4%	4.87
33	6.59	7.1%	0.8%	100.0%	6.18	0.6%	6.14	10.09	10.7%	5.09
34	6.97	7.2%	0.9%	100.0%	6.53	0.7%	6.49	10.77	11.1%	5.34
35	7.39	7.3%	1.0%	100.0%	6.92	0.7%	6.87	11.47	11.5%	5.61
36	7.85	7.4%	1.1%	100.0%	7.36	0.7%	7.31	12.19	11.9%	5.90
37	8.37	7.5%	1.3%	100.0%	7.85	0.7%	7.79	12.93	12.5%	6.23
38	8.96	7.6%	1.4%	100.0%	8.40	0.7%	8.34	13.74	13.2%	6.60
39	9.64	7.7%	1.6%	100.0%	9.04	0.7%	8.97	14.63	13.9%	7.00
40	10.40	7.9%	1.8%	100.0%	9.76	0.8%	9.69	15.63	14.8%	7.45
41	11.28	8.0%	2.0%	100.0%	10.59	0.8%	10.51	16.77	15.7%	7.95
42	12.28	8.2%	2.3%	100.0%	11.54	0.8%	11.45	18.03	16.8%	8.52
43	13.44	8.3%	2.5%	100.0%	12.64	0.8%	12.54	19.41	17.9%	9.17
44	14.77	8.5%	2.9%	100.0%	13.91	0.8%	13.80	20.89	19.1%	9.92
45	16.30	8.7%	3.2%	100.0%	15.38	0.8%	15.25	22.47	20.4%	10.79
46	18.06	8.8%	3.6%	100.0%	17.07	0.8%	16.93	24.14	21.8%	11.81
47	20.09	9.0%	4.0%	100.0%	19.03	0.9%	18.86	26.01	23.2%	12.98
48	22.43	9.3%	4.4%	100.0%	21.28	0.9%	21.10	28.17	24.7%	14.33

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate lx	28 Day Mortality Rates	Stand Alone Rates l'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates lx - kxqx
49	25.11	9.5%	4.9%	100.0%	23.88	0.9%	23.67	30.73	26.2%	15.84
50	28.19	9.8%	5.4%	100.0%	26.88	0.9%	26.64	33.81	27.7%	17.52
51	31.70	10.1%	5.9%	100.0%	30.30	0.9%	30.03	37.47	29.1%	19.39
52	35.66	10.4%	6.5%	100.0%	34.19	0.9%	33.88	41.61	30.6%	21.48
53	40.12	10.7%	7.1%	100.0%	38.58	0.9%	38.22	46.12	31.9%	23.86
54	45.09	11.0%	7.8%	100.0%	43.51	0.9%	43.10	50.86	33.3%	26.60
55	50.60	11.4%	8.5%	100.0%	49.01	0.9%	48.55	55.71	34.5%	29.79
56	56.68	11.8%	9.3%	100.0%	55.12	1.0%	54.59	60.59	35.6%	33.52
57	63.33	12.3%	10.2%	100.0%	61.85	1.0%	61.26	65.71	36.7%	37.75
58	70.55	12.8%	11.1%	100.0%	69.21	1.0%	68.55	71.29	37.6%	42.40
59	78.34	13.3%	12.1%	100.0%	77.22	1.0%	76.47	77.59	38.4%	47.41
60	86.70	13.9%	13.1%	100.0%	85.89	1.0%	85.05	84.86	39.1%	52.72
61	95.61	14.5%	14.2%	100.0%	95.23	1.0%	94.29	93.30	39.6%	58.27
62	105.00	15.2%	15.4%	100.0%	105.18	1.0%	104.13	102.92	40.0%	64.01
63	114.78	16.0%	16.6%	100.0%	115.65	1.0%	114.49	113.69	40.2%	69.89
64	124.86	16.9%	18.0%	100.0%	126.57	1.0%	125.28	125.58	40.4%	75.90
65	135.13	17.8%	19.4%	100.0%	137.86	1.0%	136.44	138.56	40.3%	81.99
66	145.52	18.8%	20.9%	100.0%	149.46	1.0%	147.89	152.63	40.2%	88.16
67	155.98	19.8%	22.5%	100.0%	161.34	1.1%	159.62	168.00	39.8%	94.40
68	166.46	20.9%	24.1%	100.0%	173.53	1.1%	171.64	184.89	39.4%	100.73
69	176.92	22.1%	25.9%	100.0%	186.02	1.1%	183.96	203.55	38.7%	107.19
70	187.31	23.2%	27.7%	100.0%	198.85	1.1%	196.59	224.19	37.9%	113.85
71	197.61	24.5%	29.6%	100.0%	212.04	1.2%	209.57	247.13	36.9%	120.81
72	207.91	25.7%	31.6%	100.0%	225.75	1.2%	223.05	272.95	35.8%	128.09
73	218.29	26.9%	33.6%	100.0%	240.19	1.2%	237.23	302.27	34.6%	135.71
74	228.88	28.2%	35.7%	100.0%	255.57	1.3%	252.32	335.76	33.3%	143.70
75	239.76	29.4%	37.8%	100.0%	272.15	1.3%	268.59	374.04	32.1%	152.10
76	251.00	30.6%	40.0%	100.0%	290.17	1.4%	286.24	417.75	30.9%	160.94
77	262.49	31.8%	42.2%	100.0%	309.63	1.4%	305.28	467.44	29.8%	170.28
78	274.09	33.0%	44.4%	100.0%	330.43	1.5%	325.62	523.67	28.7%	180.28
79	285.65	34.2%	46.6%	100.0%	352.43	1.5%	347.11	586.97	27.5%	191.15
80	297.03	35.3%	48.8%	100.0%	375.42	1.6%	369.53	657.89	26.2%	203.18
81	308.06	36.3%	50.9%	100.0%	399.19	1.6%	392.68	737.05	24.8%	216.76
82	318.58	37.4%	52.9%	100.0%	423.96	1.7%	416.77	825.39	23.2%	232.33
83	328.38	38.4%	55.0%	100.0%	450.19	1.8%	442.24	923.90	21.6%	250.42
84	337.27	39.3%	57.2%	100.0%	478.56	1.8%	469.75	1033.61	20.0%	271.73
85	345.08	40.2%	59.5%	100.0%	510.04	1.9%	500.27	1155.50	18.4%	297.16

Female Cancer Rates, per 10,000

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates I'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
18	2.38	8.7%	0.2%	100.0%	2.17	0.2%	2.17	2.27	13.7%	1.86
19	2.61	8.5%	0.3%	100.0%	2.39	0.2%	2.39	2.38	13.8%	2.07
20	2.87	8.3%	0.3%	100.0%	2.64	0.2%	2.63	2.43	14.1%	2.29
21	3.15	8.1%	0.3%	100.0%	2.90	0.2%	2.90	2.45	14.6%	2.55
22	3.47	7.9%	0.3%	100.0%	3.20	0.2%	3.19	2.46	15.4%	2.82
23	3.81	7.7%	0.3%	100.0%	3.53	0.2%	3.52	2.47	16.3%	3.13
24	4.20	7.6%	0.3%	100.0%	3.89	0.2%	3.88	2.52	17.3%	3.45
25	4.62	7.4%	0.4%	100.0%	4.29	0.2%	4.28	2.62	18.5%	3.81
26	5.08	7.3%	0.4%	100.0%	4.73	0.2%	4.72	2.78	19.7%	4.18
27	5.59	7.2%	0.4%	100.0%	5.21	0.2%	5.20	3.00	20.9%	4.58
28	6.15	7.1%	0.5%	100.0%	5.74	0.3%	5.72	3.27	22.1%	5.02
29	6.75	7.0%	0.5%	100.0%	6.31	0.3%	6.29	3.55	23.3%	5.48
30	7.41	7.0%	0.6%	100.0%	6.93	0.3%	6.92	3.85	24.4%	5.99
31	8.12	6.9%	0.6%	100.0%	7.61	0.3%	7.59	4.15	25.4%	6.56
32	8.90	6.9%	0.7%	100.0%	8.35	0.3%	8.33	4.46	26.5%	7.17
33	9.75	6.8%	0.8%	100.0%	9.16	0.3%	9.13	4.81	27.5%	7.83
34	10.68	6.8%	0.9%	100.0%	10.04	0.3%	10.01	5.22	28.6%	8.55
35	11.69	6.7%	1.0%	100.0%	11.01	0.3%	10.98	5.69	29.8%	9.31
36	12.80	6.7%	1.1%	100.0%	12.07	0.3%	12.04	6.26	31.2%	10.12
37	14.02	6.6%	1.2%	100.0%	13.25	0.3%	13.20	6.90	32.8%	10.98
38	15.35	6.6%	1.4%	100.0%	14.54	0.3%	14.49	7.62	34.4%	11.92
39	16.83	6.6%	1.5%	100.0%	15.96	0.3%	15.91	8.41	35.9%	12.94
40	18.45	6.5%	1.7%	100.0%	17.54	0.4%	17.48	9.24	37.5%	14.07
41	20.23	6.5%	1.9%	100.0%	19.27	0.4%	19.20	10.12	38.9%	15.34
42	22.17	6.5%	2.1%	100.0%	21.17	0.4%	21.10	11.06	40.1%	16.73
43	24.29	6.5%	2.4%	100.0%	23.25	0.4%	23.16	12.08	41.3%	18.26
44	26.60	6.6%	2.6%	100.0%	25.52	0.4%	25.42	13.18	42.5%	19.92
45	29.09	6.6%	2.9%	100.0%	27.97	0.4%	27.86	14.39	43.6%	21.69
46	31.76	6.7%	3.3%	100.0%	30.62	0.4%	30.49	15.72	44.8%	23.59
47	34.61	6.9%	3.6%	100.0%	33.45	0.4%	33.30	17.19	45.9%	25.56
48	37.60	7.0%	4.0%	100.0%	36.42	0.4%	36.26	18.84	46.9%	27.58
49	40.69	7.2%	4.4%	100.0%	39.53	0.5%	39.35	20.69	47.9%	29.61
50	43.87	7.3%	4.9%	100.0%	42.74	0.5%	42.54	22.76	48.8%	31.63
51	47.11	7.5%	5.4%	100.0%	46.04	0.5%	45.81	25.07	49.6%	33.60
52	50.43	7.7%	5.9%	100.0%	49.44	0.5%	49.19	27.60	50.3%	35.56

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates l'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
53	53.83	7.9%	6.4%	100.0%	52.97	0.5%	52.68	30.32	50.9%	37.53
54	57.34	8.2%	7.0%	100.0%	56.63	0.6%	56.31	33.19	51.5%	39.54
55	60.98	8.4%	7.6%	100.0%	60.45	0.6%	60.10	36.19	52.0%	41.62
56	64.76	8.7%	8.2%	100.0%	64.45	0.6%	64.07	39.30	52.6%	43.79
57	68.71	9.0%	8.9%	100.0%	68.67	0.6%	68.24	42.62	53.1%	46.05
58	72.88	9.3%	9.6%	100.0%	73.12	0.6%	72.65	46.23	53.5%	48.40
59	77.30	9.7%	10.3%	100.0%	77.84	0.7%	77.32	50.26	53.8%	50.83
60	82.01	10.1%	11.0%	100.0%	82.86	0.7%	82.28	54.79	53.9%	53.35
61	87.01	10.5%	11.7%	100.0%	88.18	0.7%	87.53	59.92	53.7%	55.98
62	92.18	11.1%	12.5%	100.0%	93.67	0.8%	92.95	65.70	53.4%	58.58
63	97.38	11.6%	13.2%	100.0%	99.20	0.8%	98.40	72.16	52.9%	61.01
64	102.46	12.2%	14.0%	100.0%	104.59	0.8%	103.71	79.36	52.3%	63.11
65	107.28	12.9%	14.8%	100.0%	109.69	0.9%	108.73	87.32	51.5%	64.75
66	111.72	13.6%	15.6%	100.0%	114.37	0.9%	113.32	96.10	50.6%	65.79
67	115.84	14.3%	16.4%	100.0%	118.69	1.0%	117.55	105.85	49.5%	66.29
68	119.74	15.1%	17.2%	100.0%	122.75	1.0%	121.51	116.74	48.3%	66.36
69	123.52	15.9%	18.0%	100.0%	126.66	1.1%	125.32	128.94	46.9%	66.15
70	127.27	16.8%	18.9%	100.0%	130.53	1.1%	129.09	142.60	45.4%	65.83
71	131.08	17.7%	19.8%	100.0%	134.48	1.2%	132.92	157.96	43.6%	65.58
72	135.02	18.6%	20.6%	100.0%	138.56	1.2%	136.88	175.51	41.7%	65.31
73	139.14	19.5%	21.6%	100.0%	142.81	1.3%	141.01	195.80	39.8%	64.93
74	143.50	20.5%	22.5%	100.0%	147.30	1.3%	145.37	219.39	37.8%	64.30
75	148.15	21.4%	23.5%	100.0%	152.08	1.4%	150.00	246.83	36.0%	63.32
76	153.12	22.5%	24.5%	100.0%	157.17	1.4%	154.93	278.65	34.2%	61.83
77	158.28	23.5%	25.5%	100.0%	162.44	1.5%	160.03	315.26	32.6%	59.80
78	163.47	24.6%	26.5%	100.0%	167.73	1.5%	165.13	357.05	30.9%	57.30
79	168.54	25.8%	27.6%	100.0%	172.83	1.6%	170.05	404.43	29.3%	54.51
80	173.33	26.9%	28.7%	100.0%	177.56	1.7%	174.59	457.77	27.5%	51.72
81	177.68	28.1%	29.7%	100.0%	181.72	1.7%	178.57	517.58	25.6%	49.31
82	181.43	29.4%	30.8%	100.0%	185.21	1.8%	181.89	584.79	23.6%	47.38
83	184.42	30.6%	31.9%	100.0%	187.96	1.9%	184.46	660.45	21.5%	45.91
84	186.50	31.8%	33.0%	100.0%	189.85	1.9%	186.20	745.59	19.4%	44.84
85	187.50	33.0%	34.1%	100.0%	190.81	2.0%	187.01	841.26	17.5%	44.00

Male Heart Attack Rates, per 10,000

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates I'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
18	0.09	7.0%	0.2%	100.0%	0.08	27.7%	0.06	5.22	0.1%	0.07
19	0.11	7.0%	0.2%	100.0%	0.10	27.1%	0.07	5.84	0.2%	0.09
20	0.13	6.9%	0.2%	100.0%	0.12	26.6%	0.09	6.28	0.2%	0.11
21	0.16	6.8%	0.2%	100.0%	0.15	26.1%	0.11	6.53	0.3%	0.14
22	0.20	6.8%	0.3%	100.0%	0.19	25.5%	0.14	6.65	0.3%	0.17
23	0.25	6.7%	0.3%	100.0%	0.23	25.0%	0.18	6.69	0.4%	0.21
24	0.31	6.6%	0.3%	100.0%	0.29	24.4%	0.22	6.70	0.5%	0.26
25	0.38	6.5%	0.3%	100.0%	0.36	23.7%	0.27	6.75	0.6%	0.32
26	0.46	6.4%	0.4%	100.0%	0.44	23.0%	0.34	6.89	0.7%	0.39
27	0.57	6.3%	0.4%	100.0%	0.54	22.2%	0.42	7.12	0.8%	0.48
28	0.70	6.3%	0.5%	100.0%	0.66	21.4%	0.52	7.44	1.0%	0.58
29	0.85	6.2%	0.5%	100.0%	0.80	20.5%	0.64	7.83	1.2%	0.71
30	1.04	6.1%	0.6%	100.0%	0.98	19.6%	0.79	8.30	1.3%	0.87
31	1.27	6.0%	0.7%	100.0%	1.20	18.6%	0.97	8.85	1.5%	1.06
32	1.53	6.0%	0.7%	100.0%	1.45	17.6%	1.20	9.45	1.8%	1.29
33	1.86	5.9%	0.8%	100.0%	1.76	16.7%	1.47	10.09	2.0%	1.56
34	2.25	5.9%	0.9%	100.0%	2.14	15.8%	1.80	10.77	2.3%	1.89
35	2.71	5.9%	1.0%	100.0%	2.58	15.1%	2.19	11.47	2.6%	2.29
36	3.27	5.8%	1.1%	100.0%	3.11	14.5%	2.66	12.19	2.9%	2.76
37	3.92	5.8%	1.3%	100.0%	3.74	14.1%	3.21	12.93	3.3%	3.32
38	4.68	5.9%	1.4%	100.0%	4.47	13.8%	3.85	13.74	3.6%	3.97
39	5.57	5.9%	1.6%	100.0%	5.32	13.7%	4.59	14.63	4.0%	4.73
40	6.59	6.0%	1.8%	100.0%	6.31	13.7%	5.45	15.63	4.5%	5.61
41	7.77	6.1%	2.0%	100.0%	7.44	13.8%	6.41	16.77	4.9%	6.63
42	9.09	6.2%	2.3%	100.0%	8.72	14.1%	7.49	18.03	5.3%	7.76
43	10.55	6.4%	2.5%	100.0%	10.13	14.4%	8.67	19.41	5.7%	9.02
44	12.14	6.7%	2.9%	100.0%	11.67	14.7%	9.95	20.89	6.1%	10.39
45	13.86	6.9%	3.2%	100.0%	13.32	15.0%	11.32	22.47	6.5%	11.85
46	15.68	7.2%	3.6%	100.0%	15.08	15.3%	12.77	24.14	7.0%	13.40
47	17.59	7.6%	4.0%	100.0%	16.92	15.6%	14.28	26.01	7.3%	15.01
48	19.55	8.0%	4.4%	100.0%	18.82	15.8%	15.84	28.17	7.7%	16.65
49	21.54	8.4%	4.9%	100.0%	20.75	16.1%	17.41	30.73	8.0%	18.30
50	23.54	8.8%	5.4%	100.0%	22.69	16.3%	18.99	33.81	8.2%	19.93
51	25.52	9.2%	5.9%	100.0%	24.62	16.6%	20.54	37.47	8.3%	21.51
52	27.48	9.7%	6.5%	100.0%	26.53	16.8%	22.06	41.61	8.3%	23.07

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates l'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
53	29.42	10.3%	7.1%	100.0%	28.43	17.2%	23.55	46.12	8.3%	24.60
54	31.35	10.9%	7.8%	100.0%	30.31	17.5%	24.99	50.86	8.2%	26.12
55	33.28	11.5%	8.5%	100.0%	32.20	18.0%	26.38	55.71	8.2%	27.63
56	35.21	12.2%	9.3%	100.0%	34.08	18.7%	27.72	60.59	8.2%	29.13
57	37.18	13.0%	10.2%	100.0%	36.00	19.4%	29.01	65.71	8.1%	30.65
58	39.23	13.9%	11.1%	100.0%	37.97	20.3%	30.26	71.29	8.1%	32.17
59	41.39	15.0%	12.1%	100.0%	40.02	21.3%	31.49	77.59	8.1%	33.73
60	43.69	16.1%	13.1%	100.0%	42.18	22.4%	32.72	84.86	8.0%	35.35
61	46.19	17.4%	14.2%	100.0%	44.49	23.7%	33.95	93.30	8.0%	37.05
62	48.89	18.7%	15.4%	100.0%	46.95	25.0%	35.20	102.92	7.9%	38.84
63	51.78	20.2%	16.6%	100.0%	49.57	26.4%	36.49	113.69	7.8%	40.72
64	54.87	21.8%	18.0%	100.0%	52.32	27.7%	37.83	125.58	7.7%	42.68
65	58.12	23.4%	19.4%	100.0%	55.21	28.9%	39.25	138.56	7.6%	44.70
66	61.53	25.2%	20.9%	100.0%	58.22	30.0%	40.77	152.63	7.5%	46.76
67	65.13	26.9%	22.5%	100.0%	61.39	30.9%	42.40	168.00	7.4%	48.88
68	68.95	28.7%	24.1%	100.0%	64.80	31.8%	44.18	184.89	7.4%	51.13
69	73.07	30.5%	25.9%	100.0%	68.53	32.7%	46.12	203.55	7.4%	53.57
70	77.56	32.3%	27.7%	100.0%	72.68	33.6%	48.25	224.19	7.3%	56.29
71	82.51	34.0%	29.6%	100.0%	77.38	34.6%	50.61	247.13	7.3%	59.39
72	88.00	35.6%	31.6%	100.0%	82.74	35.7%	53.24	272.95	7.2%	62.96
73	94.12	37.2%	33.6%	100.0%	88.92	36.8%	56.18	302.27	7.2%	67.11
74	100.99	38.8%	35.7%	100.0%	96.09	38.1%	59.48	335.76	7.2%	71.96
75	108.72	40.2%	37.8%	100.0%	104.48	39.5%	63.21	374.04	7.2%	77.70
76	117.46	41.6%	40.0%	100.0%	114.32	41.0%	67.42	417.75	7.1%	84.52
77	127.27	42.9%	42.2%	100.0%	125.81	42.6%	72.17	467.44	7.1%	92.62
78	138.17	44.0%	44.4%	100.0%	139.15	44.3%	77.51	523.67	7.1%	102.17
79	150.17	45.1%	46.6%	100.0%	154.49	46.0%	83.49	586.97	7.0%	113.36
80	163.26	46.0%	48.8%	100.0%	171.99	47.6%	90.14	657.89	6.9%	126.36
81	177.37	46.9%	50.9%	100.0%	191.80	49.2%	97.51	737.05	6.8%	141.37
82	192.50	47.6%	52.9%	100.0%	214.35	50.7%	105.76	825.39	6.7%	158.84
83	208.66	48.2%	55.0%	100.0%	240.34	52.1%	115.12	923.90	6.6%	179.44
84	225.86	48.7%	57.2%	100.0%	270.69	53.5%	125.89	1033.61	6.4%	204.13
85	244.08	49.1%	59.5%	100.0%	306.73	54.8%	138.53	1155.50	6.3%	234.26

Female Heart Attack Rates, per 10,000

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates I'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
18	0.03	21.1%	0.2%	100.0%	0.02	24.3%	0.02	2.27	0.4%	0.02
19	0.04	20.5%	0.3%	100.0%	0.03	22.6%	0.02	2.38	0.4%	0.02
20	0.04	19.9%	0.3%	100.0%	0.03	21.2%	0.03	2.43	0.4%	0.03
21	0.05	19.4%	0.3%	100.0%	0.04	20.0%	0.03	2.45	0.4%	0.03
22	0.06	18.8%	0.3%	100.0%	0.05	19.1%	0.04	2.46	0.4%	0.04
23	0.08	18.2%	0.3%	100.0%	0.06	18.3%	0.05	2.47	0.4%	0.05
24	0.09	17.6%	0.3%	100.0%	0.08	17.8%	0.06	2.52	0.4%	0.07
25	0.11	17.1%	0.4%	100.0%	0.09	17.5%	0.08	2.62	0.4%	0.08
26	0.13	16.5%	0.4%	100.0%	0.11	17.4%	0.09	2.78	0.4%	0.10
27	0.16	16.0%	0.4%	100.0%	0.13	17.5%	0.11	3.00	0.5%	0.12
28	0.19	15.5%	0.5%	100.0%	0.16	17.8%	0.13	3.27	0.5%	0.15
29	0.23	15.0%	0.5%	100.0%	0.20	18.1%	0.16	3.55	0.6%	0.18
30	0.28	14.5%	0.6%	100.0%	0.24	18.6%	0.20	3.85	0.6%	0.22
31	0.34	14.1%	0.6%	100.0%	0.29	19.0%	0.24	4.15	0.7%	0.26
32	0.41	13.7%	0.7%	100.0%	0.35	19.4%	0.29	4.46	0.9%	0.32
33	0.49	13.3%	0.8%	100.0%	0.43	19.7%	0.34	4.81	1.0%	0.38
34	0.58	13.0%	0.9%	100.0%	0.51	19.8%	0.41	5.22	1.1%	0.45
35	0.69	12.7%	1.0%	100.0%	0.61	19.6%	0.49	5.69	1.3%	0.54
36	0.82	12.5%	1.1%	100.0%	0.72	19.1%	0.58	6.26	1.4%	0.63
37	0.96	12.4%	1.2%	100.0%	0.85	18.3%	0.69	6.90	1.5%	0.74
38	1.12	12.3%	1.4%	100.0%	0.99	17.4%	0.82	7.62	1.7%	0.86
39	1.30	12.2%	1.5%	100.0%	1.15	16.5%	0.96	8.41	1.8%	1.00
40	1.50	12.3%	1.7%	100.0%	1.34	15.6%	1.13	9.24	1.9%	1.16
41	1.73	12.3%	1.9%	100.0%	1.55	14.9%	1.32	10.12	2.0%	1.35
42	2.00	12.5%	2.1%	100.0%	1.79	14.4%	1.53	11.06	2.1%	1.56
43	2.29	12.7%	2.4%	100.0%	2.05	13.9%	1.77	12.08	2.2%	1.79
44	2.62	12.9%	2.6%	100.0%	2.35	13.6%	2.03	13.18	2.2%	2.06
45	2.99	13.2%	2.9%	100.0%	2.67	13.5%	2.31	14.39	2.3%	2.35
46	3.39	13.6%	3.3%	100.0%	3.03	13.4%	2.62	15.72	2.3%	2.67
47	3.82	13.9%	3.6%	100.0%	3.41	13.4%	2.96	17.19	2.3%	3.01
48	4.29	14.3%	4.0%	100.0%	3.83	13.5%	3.31	18.84	2.4%	3.38
49	4.79	14.8%	4.4%	100.0%	4.27	13.6%	3.69	20.69	2.4%	3.78
50	5.32	15.2%	4.9%	100.0%	4.74	13.8%	4.09	22.76	2.4%	4.19
51	5.88	15.6%	5.4%	100.0%	5.24	13.9%	4.51	25.07	2.5%	4.62
52	6.47	16.1%	5.9%	100.0%	5.77	14.2%	4.95	27.60	2.5%	5.07

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates l'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
53	7.10	16.6%	6.4%	100.0%	6.32	14.4%	5.41	30.32	2.6%	5.54
54	7.77	17.2%	7.0%	100.0%	6.92	14.8%	5.90	33.19	2.6%	6.05
55	8.49	17.7%	7.6%	100.0%	7.56	15.2%	6.41	36.19	2.7%	6.59
56	9.27	18.3%	8.2%	100.0%	8.25	15.7%	6.95	39.30	2.8%	7.17
57	10.12	18.9%	8.9%	100.0%	9.01	16.4%	7.53	42.62	2.8%	7.80
58	11.07	19.6%	9.6%	100.0%	9.83	17.1%	8.15	46.23	2.9%	8.49
59	12.12	20.4%	10.3%	100.0%	10.75	18.0%	8.82	50.26	3.0%	9.24
60	13.29	21.2%	11.0%	100.0%	11.76	18.9%	9.53	54.79	3.1%	10.05
61	14.60	22.1%	11.7%	100.0%	12.88	20.0%	10.31	59.92	3.2%	10.95
62	16.07	23.0%	12.5%	100.0%	14.13	21.1%	11.15	65.70	3.4%	11.92
63	17.70	24.0%	13.2%	100.0%	15.51	22.2%	12.07	72.16	3.5%	12.98
64	19.52	25.0%	14.0%	100.0%	17.03	23.3%	13.06	79.36	3.7%	14.13
65	21.52	26.0%	14.8%	100.0%	18.68	24.4%	14.13	87.32	3.8%	15.35
66	23.71	27.1%	15.6%	100.0%	20.48	25.3%	15.29	96.10	4.0%	16.67
67	26.12	28.2%	16.4%	100.0%	22.44	26.3%	16.55	105.85	4.1%	18.06
68	28.76	29.2%	17.2%	100.0%	24.59	27.2%	17.90	116.74	4.3%	19.56
69	31.68	30.3%	18.0%	100.0%	26.94	28.1%	19.36	128.94	4.5%	21.18
70	34.90	31.4%	18.9%	100.0%	29.53	29.1%	20.93	142.60	4.6%	22.94
71	38.49	32.4%	19.8%	100.0%	32.41	30.2%	22.61	157.96	4.8%	24.86
72	42.45	33.5%	20.6%	100.0%	35.60	31.4%	24.42	175.51	4.9%	26.95
73	46.84	34.5%	21.6%	100.0%	39.14	32.7%	26.36	195.80	5.1%	29.22
74	51.68	35.5%	22.5%	100.0%	43.05	33.9%	28.43	219.39	5.2%	31.65
75	56.99	36.4%	23.5%	100.0%	47.36	35.3%	30.66	246.83	5.3%	34.26
76	62.81	37.3%	24.5%	100.0%	52.13	36.6%	33.03	278.65	5.4%	37.06
77	69.19	38.2%	25.5%	100.0%	57.39	38.0%	35.59	315.26	5.5%	40.10
78	76.16	39.0%	26.5%	100.0%	63.22	39.4%	38.33	357.05	5.5%	43.44
79	83.78	39.8%	27.6%	100.0%	69.69	40.7%	41.29	404.43	5.6%	47.17
80	92.10	40.5%	28.7%	100.0%	76.86	42.1%	44.48	457.77	5.6%	51.40
81	101.20	41.1%	29.7%	100.0%	84.84	43.5%	47.92	517.58	5.5%	56.28
82	111.22	41.7%	30.8%	100.0%	93.78	44.9%	51.65	584.79	5.4%	61.95
83	122.34	42.2%	31.9%	100.0%	103.89	46.4%	55.72	660.45	5.3%	68.63
84	134.79	42.6%	33.0%	100.0%	115.41	47.9%	60.17	745.59	5.2%	76.57
85	148.82	43.1%	34.1%	100.0%	128.63	49.4%	65.05	841.26	5.1%	86.07

Male Stroke Rates, per 10,000

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates I'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
18	0.62	19.0%	0.2%	100.0%	0.50	9.0%	0.46	5.22	1.1%	0.44
19	0.66	18.6%	0.2%	100.0%	0.54	9.0%	0.49	5.84	1.0%	0.48
20	0.71	18.2%	0.2%	100.0%	0.58	9.0%	0.53	6.28	1.0%	0.52
21	0.76	17.7%	0.2%	100.0%	0.63	9.0%	0.57	6.53	0.9%	0.57
22	0.82	17.2%	0.3%	100.0%	0.68	9.0%	0.62	6.65	0.9%	0.62
23	0.88	16.7%	0.3%	100.0%	0.73	9.0%	0.67	6.69	0.9%	0.67
24	0.94	16.2%	0.3%	100.0%	0.79	9.0%	0.72	6.70	1.0%	0.73
25	1.01	15.7%	0.3%	100.0%	0.85	9.0%	0.78	6.75	1.0%	0.78
26	1.08	15.2%	0.4%	100.0%	0.92	9.0%	0.84	6.89	1.1%	0.84
27	1.16	14.7%	0.4%	100.0%	0.99	9.0%	0.90	7.12	1.2%	0.91
28	1.25	14.3%	0.5%	100.0%	1.07	9.0%	0.98	7.44	1.4%	0.97
29	1.34	14.0%	0.5%	100.0%	1.16	9.0%	1.06	7.83	1.5%	1.04
30	1.45	13.7%	0.6%	100.0%	1.26	9.0%	1.15	8.30	1.6%	1.12
31	1.57	13.4%	0.7%	100.0%	1.37	9.0%	1.24	8.85	1.8%	1.21
32	1.71	13.3%	0.7%	100.0%	1.49	9.0%	1.36	9.45	2.0%	1.30
33	1.86	13.2%	0.8%	100.0%	1.63	8.9%	1.48	10.09	2.1%	1.41
34	2.03	13.2%	0.9%	100.0%	1.78	8.9%	1.62	10.77	2.3%	1.54
35	2.23	13.2%	1.0%	100.0%	1.96	8.8%	1.78	11.47	2.4%	1.68
36	2.46	13.3%	1.1%	100.0%	2.15	8.8%	1.96	12.19	2.5%	1.85
37	2.71	13.5%	1.3%	100.0%	2.38	8.8%	2.17	12.93	2.6%	2.04
38	3.00	13.7%	1.4%	100.0%	2.62	8.7%	2.40	13.74	2.6%	2.26
39	3.32	13.9%	1.6%	100.0%	2.90	8.7%	2.65	14.63	2.7%	2.51
40	3.68	14.1%	1.8%	100.0%	3.21	8.6%	2.94	15.63	2.8%	2.78
41	4.08	14.4%	2.0%	100.0%	3.56	8.6%	3.26	16.77	2.9%	3.08
42	4.52	14.7%	2.3%	100.0%	3.94	8.5%	3.61	18.03	3.0%	3.40
43	5.01	15.1%	2.5%	100.0%	4.37	8.6%	3.99	19.41	3.1%	3.76
44	5.55	15.4%	2.9%	100.0%	4.83	8.6%	4.42	20.89	3.2%	4.16
45	6.14	15.8%	3.2%	100.0%	5.34	8.6%	4.88	22.47	3.3%	4.60
46	6.79	16.2%	3.6%	100.0%	5.90	8.7%	5.39	24.14	3.4%	5.07
47	7.49	16.6%	4.0%	100.0%	6.50	8.7%	5.93	26.01	3.5%	5.59
48	8.23	17.1%	4.4%	100.0%	7.14	8.7%	6.52	28.17	3.6%	6.13
49	9.02	17.6%	4.9%	100.0%	7.81	8.6%	7.14	30.73	3.6%	6.70
50	9.85	18.2%	5.4%	100.0%	8.52	8.6%	7.79	33.81	3.7%	7.28
51	10.73	18.8%	5.9%	100.0%	9.26	8.6%	8.46	37.47	3.7%	7.88
52	11.64	19.5%	6.5%	100.0%	10.02	8.6%	9.16	41.61	3.7%	8.50

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates l'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
53	12.58	20.2%	7.1%	100.0%	10.82	8.7%	9.88	46.12	3.6%	9.14
54	13.57	20.9%	7.8%	100.0%	11.64	8.7%	10.62	50.86	3.6%	9.81
55	14.59	21.7%	8.5%	100.0%	12.49	8.8%	11.39	55.71	3.6%	10.51
56	15.65	22.6%	9.3%	100.0%	13.37	8.9%	12.18	60.59	3.5%	11.24
57	16.78	23.4%	10.2%	100.0%	14.30	9.0%	13.01	65.71	3.5%	12.02
58	17.99	24.3%	11.1%	100.0%	15.31	9.1%	13.92	71.29	3.4%	12.86
59	19.32	25.3%	12.1%	100.0%	16.42	9.1%	14.92	77.59	3.4%	13.78
60	20.81	26.2%	13.1%	100.0%	17.66	9.2%	16.03	84.86	3.4%	14.79
61	22.47	27.2%	14.2%	100.0%	19.06	9.3%	17.29	93.30	3.4%	15.90
62	24.34	28.2%	15.4%	100.0%	20.64	9.4%	18.70	102.92	3.4%	17.13
63	26.45	29.3%	16.6%	100.0%	22.42	9.6%	20.28	113.69	3.5%	18.49
64	28.82	30.5%	18.0%	100.0%	24.44	9.8%	22.05	125.58	3.5%	20.01
65	31.49	31.7%	19.4%	100.0%	26.70	10.0%	24.04	138.56	3.6%	21.69
66	34.48	32.9%	20.9%	100.0%	29.25	10.2%	26.26	152.63	3.7%	23.55
67	37.78	34.1%	22.5%	100.0%	32.10	10.5%	28.73	168.00	3.9%	25.60
68	41.37	35.4%	24.1%	100.0%	35.26	10.8%	31.45	184.89	4.0%	27.82
69	45.26	36.5%	25.9%	100.0%	38.76	11.1%	34.44	203.55	4.2%	30.24
70	49.42	37.7%	27.7%	100.0%	42.61	11.5%	37.70	224.19	4.4%	32.84
71	53.84	38.7%	29.6%	100.0%	46.87	11.9%	41.27	247.13	4.5%	35.66
72	58.60	39.7%	31.6%	100.0%	51.60	12.4%	45.20	272.95	4.7%	38.73
73	63.74	40.7%	33.6%	100.0%	56.91	12.9%	49.56	302.27	4.9%	42.12
74	69.35	41.6%	35.7%	100.0%	62.93	13.5%	54.45	335.76	5.1%	45.91
75	75.47	42.5%	37.8%	100.0%	69.79	14.1%	59.97	374.04	5.2%	50.18
76	82.18	43.3%	40.0%	100.0%	77.64	14.7%	66.21	417.75	5.4%	55.03
77	89.49	44.1%	42.2%	100.0%	86.61	15.4%	73.25	467.44	5.6%	60.53
78	97.41	44.8%	44.4%	100.0%	96.79	16.1%	81.17	523.67	5.7%	66.74
79	105.97	45.5%	46.6%	100.0%	108.32	16.9%	90.01	586.97	5.9%	73.74
80	115.19	46.1%	48.8%	100.0%	121.29	17.7%	99.80	657.89	6.0%	81.59
81	125.07	46.6%	50.9%	100.0%	135.87	18.6%	110.62	737.05	6.2%	90.38
82	135.61	47.1%	52.9%	100.0%	152.38	19.5%	122.67	825.39	6.3%	100.39
83	146.82	47.5%	55.0%	100.0%	171.37	20.5%	136.24	923.90	6.4%	112.12
84	158.68	47.8%	57.2%	100.0%	193.52	21.5%	151.83	1033.61	6.5%	126.21
85	171.17	48.0%	59.5%	100.0%	219.86	22.6%	170.07	1155.50	6.6%	143.64

Female Stroke Rates, per 10,000

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates I'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
18	0.45	15.5%	0.2%	100.0%	0.38	9.7%	0.34	2.27	1.0%	0.35
19	0.48	15.2%	0.3%	100.0%	0.41	9.7%	0.37	2.38	1.2%	0.38
20	0.53	14.9%	0.3%	100.0%	0.45	9.8%	0.40	2.43	1.3%	0.42
21	0.57	14.7%	0.3%	100.0%	0.49	9.8%	0.44	2.45	1.4%	0.45
22	0.62	14.5%	0.3%	100.0%	0.53	9.9%	0.48	2.46	1.6%	0.49
23	0.68	14.4%	0.3%	100.0%	0.58	9.9%	0.52	2.47	1.7%	0.54
24	0.74	14.3%	0.3%	100.0%	0.63	10.0%	0.57	2.52	1.9%	0.59
25	0.80	14.2%	0.4%	100.0%	0.69	10.0%	0.62	2.62	2.0%	0.64
26	0.88	14.2%	0.4%	100.0%	0.76	10.0%	0.68	2.78	2.1%	0.70
27	0.96	14.1%	0.4%	100.0%	0.82	10.1%	0.74	3.00	2.2%	0.76
28	1.04	14.1%	0.5%	100.0%	0.90	9.9%	0.81	3.27	2.3%	0.82
29	1.13	14.1%	0.5%	100.0%	0.98	9.8%	0.88	3.55	2.5%	0.89
30	1.23	14.1%	0.6%	100.0%	1.07	9.6%	0.96	3.85	2.6%	0.97
31	1.34	14.0%	0.6%	100.0%	1.16	9.4%	1.05	4.15	2.7%	1.05
32	1.46	14.0%	0.7%	100.0%	1.26	9.3%	1.14	4.46	2.8%	1.14
33	1.58	14.0%	0.8%	100.0%	1.37	9.4%	1.24	4.81	2.9%	1.23
34	1.72	13.9%	0.9%	100.0%	1.50	9.5%	1.35	5.22	3.1%	1.34
35	1.87	13.9%	1.0%	100.0%	1.63	9.6%	1.47	5.69	3.2%	1.45
36	2.04	13.9%	1.1%	100.0%	1.78	9.7%	1.60	6.26	3.3%	1.57
37	2.23	14.0%	1.2%	100.0%	1.94	9.8%	1.75	6.90	3.5%	1.70
38	2.43	14.1%	1.4%	100.0%	2.12	9.9%	1.91	7.62	3.6%	1.84
39	2.66	14.3%	1.5%	100.0%	2.31	10.0%	2.08	8.41	3.7%	2.00
40	2.91	14.6%	1.7%	100.0%	2.53	10.1%	2.27	9.24	3.9%	2.17
41	3.19	15.0%	1.9%	100.0%	2.77	10.2%	2.48	10.12	4.0%	2.36
42	3.50	15.4%	2.1%	100.0%	3.02	10.3%	2.71	11.06	4.1%	2.57
43	3.83	15.9%	2.4%	100.0%	3.30	10.3%	2.96	12.08	4.2%	2.79
44	4.19	16.5%	2.6%	100.0%	3.59	10.3%	3.22	13.18	4.3%	3.02
45	4.56	17.1%	2.9%	100.0%	3.90	10.2%	3.50	14.39	4.4%	3.27
46	4.96	17.7%	3.3%	100.0%	4.22	10.2%	3.79	15.72	4.5%	3.52
47	5.37	18.4%	3.6%	100.0%	4.55	10.2%	4.09	17.19	4.5%	3.78
48	5.80	19.0%	4.0%	100.0%	4.89	10.3%	4.39	18.84	4.5%	4.04
49	6.22	19.7%	4.4%	100.0%	5.23	10.4%	4.69	20.69	4.5%	4.30
50	6.65	20.3%	4.9%	100.0%	5.57	10.5%	4.99	22.76	4.5%	4.56
51	7.08	20.9%	5.4%	100.0%	5.92	10.6%	5.29	25.07	4.4%	4.81
52	7.50	21.4%	5.9%	100.0%	6.26	10.7%	5.59	27.60	4.3%	5.07

Age	Smoothed, Interpolated Crude Rate	Adjustment for Overlap	Combined Prevalence Rate	Other Adjustments	Derived Incidence Rate Ix	28 Day Mortality Rates	Stand Alone Rates l'x	Mortality Rates	Proportions of Deaths kx	Addition for Accelerated Rates Ix - kxqx
53	7.94	22.0%	6.4%	100.0%	6.62	10.8%	5.90	30.32	4.2%	5.33
54	8.39	22.6%	7.0%	100.0%	6.99	10.9%	6.23	33.19	4.2%	5.61
55	8.88	23.2%	7.6%	100.0%	7.38	10.9%	6.58	36.19	4.1%	5.91
56	9.40	23.8%	8.2%	100.0%	7.81	11.0%	6.95	39.30	4.0%	6.25
57	9.98	24.4%	8.9%	100.0%	8.28	11.1%	7.36	42.62	3.9%	6.62
58	10.64	25.1%	9.6%	100.0%	8.81	11.3%	7.82	46.23	3.8%	7.03
59	11.39	25.8%	10.3%	100.0%	9.42	11.5%	8.34	50.26	3.8%	7.51
60	12.27	26.5%	11.0%	100.0%	10.13	11.7%	8.95	54.79	3.8%	8.04
61	13.29	27.2%	11.7%	100.0%	10.95	11.9%	9.65	59.92	3.8%	8.65
62	14.47	28.0%	12.5%	100.0%	11.91	12.1%	10.47	65.70	3.9%	9.34
63	15.84	28.7%	13.2%	100.0%	13.02	12.3%	11.42	72.16	4.0%	10.13
64	17.42	29.4%	14.0%	100.0%	14.30	12.5%	12.51	79.36	4.1%	11.04
65	19.25	30.2%	14.8%	100.0%	15.78	12.8%	13.77	87.32	4.2%	12.07
66	21.35	30.9%	15.6%	100.0%	17.49	13.0%	15.21	96.10	4.4%	13.26
67	23.73	31.5%	16.4%	100.0%	19.43	13.3%	16.85	105.85	4.6%	14.59
68	26.42	32.1%	17.2%	100.0%	21.65	13.7%	18.69	116.74	4.8%	16.09
69	29.44	32.7%	18.0%	100.0%	24.16	14.1%	20.76	128.94	5.0%	17.75
70	32.80	33.3%	18.9%	100.0%	26.98	14.5%	23.08	142.60	5.2%	19.57
71	36.54	33.8%	19.8%	100.0%	30.15	14.9%	25.66	157.96	5.4%	21.57
72	40.71	34.3%	20.6%	100.0%	33.72	15.3%	28.55	175.51	5.7%	23.72
73	45.39	34.8%	21.6%	100.0%	37.73	15.7%	31.79	195.80	6.0%	26.04
74	50.65	35.3%	22.5%	100.0%	42.26	16.2%	35.43	219.39	6.3%	28.49
75	56.56	35.9%	23.5%	100.0%	47.36	16.6%	39.49	246.83	6.6%	31.07
76	63.21	36.5%	24.5%	100.0%	53.11	17.1%	44.02	278.65	6.9%	33.75
77	70.59	37.2%	25.5%	100.0%	59.53	17.7%	49.02	315.26	7.3%	36.50
78	78.72	37.8%	26.5%	100.0%	66.66	18.3%	54.47	357.05	7.7%	39.33
79	87.60	38.4%	27.6%	100.0%	74.50	18.9%	60.39	404.43	8.0%	42.22
80	97.24	39.1%	28.7%	100.0%	83.08	19.6%	66.77	457.77	8.3%	45.24
81	107.63	39.7%	29.7%	100.0%	92.44	20.3%	73.64	517.58	8.5%	48.45
82	118.80	40.2%	30.8%	100.0%	102.69	21.1%	81.05	584.79	8.7%	51.94
83	130.82	40.7%	31.9%	100.0%	114.01	21.9%	89.02	660.45	8.8%	55.81
84	143.70	41.0%	33.0%	100.0%	126.57	22.8%	97.71	745.59	8.9%	60.15
85	157.49	41.2%	34.1%	100.0%	140.55	23.7%	107.21	841.26	9.0%	65.08

Appendix 7 Top Down Geodemographic Analysis Summary