Potential Population Health Gain of the Quality and Outcomes Framework

Report to Department of Health 2007

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Acknowledgements

This project has been funded by the Department of the Health and the views expressed in this publication are those of the authors and not necessarily of the Department of Health.

We would like to acknowledge and thank our colleagues from the School of Medicine, Health Policy and Practice for their expert assistance. We also would like to thank Anne Mason and Professor Mark Sculpher of the Centre for Health Economics at York University for their advice in specific areas of literature searching. The responsibility for the report remains with the authors.
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List of Acronyms

A2RA    Angiotensin II receptor antagonists
ACE    Angiotensin converting enzyme
AF    Atrial fibrillation
AHCPR    Agency for healthcare policy & research
ARB    Angiotensin receptor blocker
ARR    Absolute risk reduction
BMI    Body mass index
BNF    British national formulary
BP    Blood pressure
CER    Control event rate (PCT)
CHD    Coronary heart disease
CI    Confidence interval
CKD    Chronic kidney disease
COPD    Chronic obstructive pulmonary disease
CT    Computed tomography
CVD    Cardiovascular disease
DEM    Dementia
DEP    Depression
DM    Diabetes mellitus
DoH    Department of health
eGFR    Estimated glomerular filtration rate
FeV1    Forced expiratory volume
FFS    Fee for service
GMS    General medical services
GP    General practitioner
PHG    Population health gain
IER    Intervention event rate
IHD    Ischaemic heart disease
LVD    Left ventricular disease
MI    Myocardial infarction
MRC    Medical research Council
<table>
<thead>
<tr>
<th>Acronym</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
</tr>
<tr>
<td>NICE</td>
<td>National Institute of Health and Clinical Excellence</td>
</tr>
<tr>
<td>NNT</td>
<td>Number needed to treat</td>
</tr>
<tr>
<td>NRT</td>
<td>Nicotine replacement therapy</td>
</tr>
<tr>
<td>OR</td>
<td>Odds ratio</td>
</tr>
<tr>
<td>P&lt;sub&gt;CT&lt;/sub&gt;</td>
<td>Patient control event rate (CER)</td>
</tr>
<tr>
<td>P4P</td>
<td>Pay for performance</td>
</tr>
<tr>
<td>PMS</td>
<td>Personal medical services</td>
</tr>
<tr>
<td>PTCA</td>
<td>Percutaneous transluminal coronary angioplasty</td>
</tr>
<tr>
<td>QALYs</td>
<td>Quality adjusted life years</td>
</tr>
<tr>
<td>QOF</td>
<td>Quality outcomes framework</td>
</tr>
<tr>
<td>RCT</td>
<td>Randomized controlled trial</td>
</tr>
<tr>
<td>RRR</td>
<td>Relative risk reduction</td>
</tr>
<tr>
<td>SE</td>
<td>Standard error</td>
</tr>
<tr>
<td>TIA</td>
<td>Transient ischaemic attack</td>
</tr>
<tr>
<td>TSH</td>
<td>Thyroid stimulating hormone</td>
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### List of Definitions

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<th>Term</th>
<th>Definition</th>
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<tr>
<td>$\text{ARR}_{T}$</td>
<td>Absolute risk reduction in risk of dying over next $T$ years</td>
</tr>
<tr>
<td>General Medical Services (GMS)</td>
<td>A typical contract that a GP holds with their Primary care trust</td>
</tr>
<tr>
<td>$N$</td>
<td>Number of eligible patients in 100,000 populations</td>
</tr>
<tr>
<td>$P_{CT}$</td>
<td>Baseline risk of dying over $T$ years in control patients the control event rate (CER)</td>
</tr>
<tr>
<td>Personal Medical Services (PMS)</td>
<td>A locally negotiated contract between the general practice and its primary care trust which is an alternative to GMS for providers of general practice.(^{(1)})</td>
</tr>
<tr>
<td>Potential Health Gain</td>
<td>The difference between 100% indicator achievement and zero achievement.</td>
</tr>
<tr>
<td>QALYs</td>
<td>An indication of the benefits gained from medical procedures in terms of quality and life and survival of patient.(^{(2)})</td>
</tr>
<tr>
<td>QOF</td>
<td>Annual reward and incentive programme measuring GP practice and achievement results.(^{(3)})</td>
</tr>
<tr>
<td>$T$</td>
<td>Length of study (years)</td>
</tr>
</tbody>
</table>
Executive Summary

Background

The new General Medical Service (GMS) contract was introduced into UK primary care in April 2004. The contract was supported by a significant investment, estimated to be £8 billion by the Department of Health (DoH) in the first 3 years\(^4\). It includes payment for performance against criteria in 4 areas: clinical, organisational, patient experience and additional services. There were 10 clinical domains in the original contract, and this was revised to include a further 8 clinical domains in 2006.\(^4\), \(^5\) Points are allocated on the basis of expected workload and quality of care.\(^4\) Practices achieved a high standard of performance, with practices in England scoring an average of 959 points out of a possible 1050 in 2004/5, rising to 1011 points out of a possible 1050 points in 2005/6 excluding exception reported patients\(^6\). The revision to the contract in 2006 has increased the points allocated to clinical indicators to 665.\(^5\)

Remit agreed with the Department of Health

This research formed a larger part of the project with the University of York and the University of Manchester. The University of York have estimated the cost effectiveness of a subset of clinical indicators, and the University of Manchester are exploring the trends in the performance in a subset of indicators inside and outside the Quality Outcomes Framework (QOF). Our research aims to estimate the potential population health gain of the full implementation of the interventions in the new GMS contract, both in its original form (2003)\(^4\) and its revised form (2006).\(^5\) The number of lives saved is the chosen measure of health gain, as many of the clinical interventions in the new GMS contract have potential to save lives. The “potential” health gain is based on the difference between 100% indicator achievement and zero achievement; however this will be greater than the actual health gain as there was a substantial indicator achievement before the contract was introduced. Secondly, we aimed to construct the aggregate of the number of lives saved per 100,000 populations per year at two levels namely; an aggregate at the domain level and an overall aggregate of all clinical indicators. The research was conducted in the following way:
1. A search was made for the most robust level of evidence for lives saved in terms of risk reduction. Sources used were the supporting documentation for the GMS contract\(^4\), \(^5\), The National Institute for Health and Clinical Excellence (NICE)\(^7\), Clinical Evidence (CE) database\(^8\) and the Cochrane Library\(^9\). Where the absolute risk reduction (ARR) was not reported in a clinical trial, then this was calculated where possible from other measures of health gain such as relative risk reduction (RRR) and odds ratio (OR). This is combined with the UK population prevalence data to calculate the baseline risk.

2. The risk reduction in mortality was approximated to a standardised year, with the assumption of a linear relationship between mortality and time. An adjustment was made for the prevalence of each condition to estimate the maximum health gain across a standardized population of 100,000 in terms of lives saved per year.

**Methods**

The research includes four stages that mirror the aims of this project, these being search strategy, inclusion and exclusion criteria, sensitivity analysis and strategies for combining data.

1. **Search Strategy**

Two researchers (RF and SP) independently reviewed four sources for the highest level of evidence for lives saved in terms of absolute risk reduction for all cause mortality for each clinical indicator in the GMS contract 2003 and 2006 versions\(^4\), \(^5\). The sources for evidence were as previously described. A detailed search strategy has been defined in **Appendix A**. The level of evidence was determined by using the classic “hierarchy of evidence” grading scale designed by the US Agency for Health Care Policy and Research (AHCPR 1992)\(^10\) and adopted by the Cochrane Library\(^9\).
2. **Inclusion and exclusion of studies**

Studies included have the following characteristics:

- Clinical interventions were compared with a placebo control arm.
- Studies had clinical interventions that closely matched the clinical indicators.
- Studies had all cause mortality as an outcome.

Evidence for effectiveness for each clinical indicator in the new GMS contract was sought in each of the 4 sources independently by each author. Each study that had been identified by this process was then again reviewed by two researchers against the inclusion and exclusion criteria in developing the final list of appropriate studies. 28 studies were included in the final analysis.

3. **Sensitivity analysis**

Where several studies of similar level were identified for a particular clinical indicator, then the upper and lower risk reductions estimated by these studies were used to estimate an upper and lower limit to the population health gain. The 95% confidence intervals were given for each study included where this has been reported.

4. **Results**

Evidence for lives saved was found for 19 indicators in the 2006 revised contract. A further 23 indicators were indirectly linked to a reduction in mortality, as they are necessary processes to deliver results for the directly linked 19 indicators. The number of potential lives saved by the 19 directly linked indicators ranged from 1.0 to 62.8 per 100,000 people per year.

In the 2003 GMS contract the potential lives saved per 100,000 populations per year aggregated at the domain level are: 163.2 lives in coronary heart disease, 105.2 lives in diabetes, 51.5 lives in hypertension, 49.7 lives in stroke, 27.0 lives...
in chronic obstructive pulmonary disease, 11.9 lives in left ventricular dysfunction, and 8.8 lives in asthma.

In the 2006 revised GMS contract the potential lives saved per 100,000 populations per year aggregated at domain level are: 160.9 lives in coronary heart disease, 103.0 lives in diabetes, 48.7 lives in stroke, 46.3 lives in hypertension, 24.4 lives in chronic obstructive pulmonary disease, 19.2 lives in atrial fibrillation, 12.8 lives in chronic kidney disease, 12 lives in smoking cessation and 11.9 lives in heart failure or left ventricular dysfunction.

In the 2003 contract there was potential for 415.0 lives saved per 100,000 per year (406.1-423.9) aggregated across all clinical indicators and domains. In the 2006 contract this increased by 24.3 lives to a potential for 439.3 lives per 100,000 people per year saved (426.1-455.2) aggregated across all clinical indicators and domains.

Conclusions

Full implementation of the quality indicators in the original GMS contract (2003) can be expected to result in a potential 415.0 lives being saved per 100,000 populations per year. However the actual number of lives saved was lower for two reasons. Firstly, there was a significant baseline activity in primary care before the implementation of the new GMS contract. Secondly, less than 100% of the target population received the intervention due to exclusions due to exception reporting and less than full implementation of the contract. Full implementation of the revised GMS contract (2006) increases the potential lives saved to 439.3 per 100,000 populations per year. This equates to approximately 262,855 lives saved per 100,000 people in one year for the whole of the United Kingdom population.

Lives saved are a direct outcome of 19 of the 2006 GMS contract indicators, and an indirect outcome of a further 23 indicators. Health gain represents a possible additional criterion to be used when allocating points to indicators and conditions in future revisions of the Quality Outcomes Framework (QOF). Limitations of this study include the use of mortality as a relatively narrow measure of health gain, the
assumption of linearity between mortality reduction and time, an incomplete evidence base for some interventions in the GMS contract, the changing prevalence of disease and the assumption that patients in the clinical trials are representative of the UK population. Further research includes developing other measures of quality such as QALYs\textsuperscript{(2)} for as many clinical indicators as possible to inform development and weighting of both current and new indicators in future revisions of the GMS contract.
Summary of results

Table 1: Health gain league table, estimated lives saved per 100,000 populations per year by indicator for 2003\(^{(4)}\) and 2006 contract\(^{(5)}\).

<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>CHD 12</td>
<td>Influenza immunization</td>
<td>62.8</td>
<td>62.8</td>
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<tr>
<td>DM 18</td>
<td>Influenza immunization</td>
<td>57.6</td>
<td>57.6</td>
</tr>
<tr>
<td>BP 5</td>
<td>Hypertension, BP,150/90 in past 9 months</td>
<td>46.3</td>
<td>46.3</td>
</tr>
<tr>
<td>CHD 10</td>
<td>Beta blocker</td>
<td>44.9</td>
<td>44.9</td>
</tr>
<tr>
<td>Stroke 10</td>
<td>Influenza immunization</td>
<td>26.2</td>
<td>26.2</td>
</tr>
<tr>
<td>CHD 9</td>
<td>Aspirin</td>
<td>25.2</td>
<td>25.2</td>
</tr>
<tr>
<td>COPD 8</td>
<td>Influenza immunization</td>
<td>24.4</td>
<td>24.4</td>
</tr>
<tr>
<td>DM 6</td>
<td>HbA1c&lt;7.4</td>
<td>24.0</td>
<td>24.0</td>
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<tr>
<td>Stroke 9</td>
<td>Antiplatelet/ anticoagulant</td>
<td>22.5</td>
<td>22.5</td>
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<tr>
<td>AF3</td>
<td>Anticoagulant</td>
<td>N/A</td>
<td>21.6</td>
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<td>CHD 8</td>
<td>Cholesterol &lt; 5 mmol</td>
<td>16.1</td>
<td>16.1</td>
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<td>CKD 3</td>
<td>BP&lt;140/85</td>
<td>N/A</td>
<td>12.8</td>
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<tr>
<td>DM 12</td>
<td>BP&lt;145/85</td>
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<td>12.1</td>
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<td>Smoking 2</td>
<td>Smoking cessation advice/referral</td>
<td>N/A</td>
<td>12.0</td>
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<td>LVD/HF 3</td>
<td>ACE/ARB</td>
<td>11.9</td>
<td>11.9</td>
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<tr>
<td>CHD 6</td>
<td>BP&lt;150/90</td>
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<td>11.5</td>
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<td>Asthma 5</td>
<td>Smoking cessation advice/referral</td>
<td>8.8</td>
<td>N/A</td>
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<tr>
<td>DM 7</td>
<td>HbA1c &lt;10</td>
<td>6.7</td>
<td>6.7</td>
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<td>CHD 11</td>
<td>ACE/ARB</td>
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<td>COPD 5</td>
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<tr>
<td>CHD 4</td>
<td>Smoking cessation advice/referral</td>
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<tr>
<td>DM 4</td>
<td>Smoking cessation advice/referral</td>
<td>2.2</td>
<td>N/A</td>
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<tr>
<td>Stroke 4</td>
<td>Smoking cessation advice/referral</td>
<td>1.0</td>
<td>N/A</td>
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</table>
Table 2: Health gain league table, estimated lives saved per 100,000 populations per year by clinical domain for 2003\(^{(4)}\) and 2006 contract\(^{(5)}\).

<table>
<thead>
<tr>
<th>Clinical domain</th>
<th>Lives saved (2003(^{(4)}))</th>
<th>Lives saved (2006(^{(5)}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary heart disease</td>
<td>163.2</td>
<td>160.9</td>
</tr>
<tr>
<td>Diabetes</td>
<td>105.2</td>
<td>103.0</td>
</tr>
<tr>
<td>Stroke</td>
<td>49.7</td>
<td>48.7</td>
</tr>
<tr>
<td>Hypertension</td>
<td>51.5</td>
<td>46.3</td>
</tr>
<tr>
<td>Chronic obstructive airways disease</td>
<td>27.0</td>
<td>24.4</td>
</tr>
<tr>
<td>Atrial Fibrillation</td>
<td>N/A</td>
<td>21.6</td>
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<td>Chronic kidney disease</td>
<td>N/A</td>
<td>12.8</td>
</tr>
<tr>
<td>Smoking Cessation</td>
<td>N/A</td>
<td>12.0</td>
</tr>
<tr>
<td>Left ventricular dysfunction/ Heart Failure</td>
<td>11.9</td>
<td>11.9</td>
</tr>
<tr>
<td>Asthma</td>
<td>8.8</td>
<td>N/A</td>
</tr>
<tr>
<td>Total including corrections for double counting (range)</td>
<td><strong>415.0 (406.1-423.9)</strong></td>
<td><strong>439.3 (427.2-455.2)</strong></td>
</tr>
</tbody>
</table>
Section 1 - Introduction

1.1 Background to the GMS contract 2003 & 2006

Payment for performance (P4P) scheme was first introduced in the 1990 General Practice (GP) contract, and covered cervical cancer screening, immunization, child health Surveillance and health promotion clinics. The General Medical Services (GMS) contract, signed in February 2003, represents a major change to the funding of UK primary care. The new GMS contract greatly extends these P4P services to cover many clinical areas. The National Health Service (NHS) committed an estimated £8 billion in additional funding over 3 years in support of this contract, leading to both a significant increase in infrastructure to primary care services, and a significant increase in GP principal’s net income. The new contract is an important development in the primary care, introducing a wide range of P4P financial incentives for quality improvement. Points are awarded to general practices in return for reaching target thresholds in indicators of quality care, and points equate to financial payments. These payments are available to all general practices, whether contracted to provide personal medical services (PMS) or general medical services (GMS), and over 99% of practices are participating in the new contract in 2005/6. This has led to a significant increase in a typical general practitioner principal pay, with an average income of a GMS general practitioner principal rising by 32.8% in the first year of the contract from £77,152 in 2003/4 to £102,437 in 2004/5.

1.2 Clinical indicators

In Quality Outcomes Framework there are incentive payments for four components of practice; clinical indicators, organisational indicators, patient experience and additional services. Clinical indicators cover major areas of clinical practice in primary care. Organisational indicators cover records and information about patients, information for patients, education and training, practice management and medicines management. Patient experience indicators cover the services provided, how they are provided and their involvement in service development plans. Additional services cover cervical screening, contraceptive services, vaccinations and immunizations, child health surveillance, maternity services and minor surgery procedures.
In the original version of the new GMS contract (2003\(^4\)) there were 10 clinical domains, coronary heart disease (CHD) with a subset of heart failure (HF), stroke, hypertension (BP), diabetes (DM), chronic obstructive pulmonary disease (COPD), asthma, mental health (MH), epilepsy, cancer and thyroid disorders. This contract was revised (GMS contract 2006\(^5\)) and extended to include a further 8 clinical areas which are smoking, palliative care (PC), depression (DEP), dementia (DEM), obesity (OB), learning disability (LD), chronic kidney disease (CKD) and atrial fibrillation (AF) [Refer to Figure 1].

Over half the maximum points available in the 2003 GMS contract\(^4\) were allocated to clinical performance (550 out of 1050), and many of these indicators were related to areas of health gain in terms of mortality reduction. In the revised 2006 GMS\(^5\) contract, the allocation of points to clinical indicators was increased to 655.

Each clinical domain contains a subset of clinical indicators which can represent “structure”, “process” or “intermediate outcome”. For example the CHD domain contains 12 clinical indicators. These clinical indicators include a disease register (e.g. CHD 1: The practice can produce a register of patients with coronary heart disease) – a “structure” indicator; a diagnostic process (e.g. CHD5: The percentage of patients with coronary heart disease whose notes have a record of blood pressure in the previous 15 months) – a “process” indicator with an intermediate outcome (e.g. BP5: The percentage of patients with hypertension having a recorded blood pressure \(<150/90\) – “outcome indicator”. No indicator contains a “final outcome”, such as decrease in mortality or recurrent cardiovascular events. This is in part because the absolute number of deaths at the GP level is small, and variation in this statistic is largely dependant on factors other than quality practice, such as age, sex, income, education, housing, transport etc.
Figure 1: Flowchart of Indicators

2003 Contract
76 clinical indicators

2006 Contract
80 indicators

Mortality
Health
Gain
N=42

Indirectly
linked to
reduced mortality
N=23

Register
N=11
CHD 1
HF 1
Stroke 1
BP 1
DM 19
COPD 1
Asthma 1
CKD 1
AF 1
Smoking 1
Asthma 3

Diagnostic
N=12
CHD 2
CHD 5
CHD 7
HF 2
Stroke 7
BP 4
DM 5
DM 11
DM 13
DM 22
CKD 2
AF 2

Directly
linked to
reduced mortality
N=19

N=9
Register
Epilepsy 5
Thyroid 1
Cancer 1
PC 1
MH 8
MH 9
DEM 1
OB 1
LD 1

N=11
Diagnostic
Stroke 5
DM16
COPD 9
COPD 10
COPD 11
Thyroid 2
MH 4
MH 5
Asthma 8
DEP 1
DEP 2

Non
Mortality
Health
Gain
N= 38

18
Process/
outcome
Stroke 6
Stroke 8
Stroke 11
DM 2
DM 9
DM 10
DM 17
DM 21
Epilepsy 6
Epilepsy 7
Epilepsy 8
Cancer 3
PC 2
MH 6
MH 7
Asthma 6
DEM 2
CKD 4
The payment for each indicator has been designed to reflect the quality and amount of work anticipated to meet that particular indicator. A proportion of the points scored for each indicator is awarded in a direct linear relationship for achievement between the minimum and maximum percentages set for each indicator. For example if 10 points were available for an indicator with a maximum level of achievement of 85% and the practice had achieved 55%, they would receive 30/60ths of 10 points, i.e. 5 points.

Figure 2: Points gained for indicator with maximum payment of 10 points and minimum and maximum thresholds of 25% and 85% respectively.
1.3 Payments for points

There was a payment of £75 per point achieved for an “average practice” in the first year of the contract, rising to £124.60 from 2005/6, based on a typical practice of 5500 patients. This payment is adjusted directly in proportion to the number of patients in the practice. For example a practice with 11,000 patients will receive £249.20 per point achieved. This is a gross payment to a particular practice, and is not net profit. The practice bears the additional costs of meeting a particular indicator, which includes additional staffing, infrastructure costs and pharmacy costs. Practice expenses which are paid independently of the new GMS contract have also been increasing at a greater rate than inflation.

Table 3: Allocation of points according to the GMS contract 2003 & 2006

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD</td>
<td>101</td>
<td>89</td>
</tr>
<tr>
<td>LVD/ Heart failure</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Stroke and TIA</td>
<td>31</td>
<td>24</td>
</tr>
<tr>
<td>Cancer</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>08</td>
<td>07</td>
</tr>
<tr>
<td>Diabetes</td>
<td>99</td>
<td>93</td>
</tr>
<tr>
<td>Hypertension</td>
<td>105</td>
<td>83</td>
</tr>
<tr>
<td>Mental health</td>
<td>41</td>
<td>39</td>
</tr>
<tr>
<td>Asthma</td>
<td>72</td>
<td>45</td>
</tr>
<tr>
<td>COPD</td>
<td>45</td>
<td>33</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>16</td>
<td>15</td>
</tr>
<tr>
<td>Palliative care</td>
<td>N/A</td>
<td>06</td>
</tr>
<tr>
<td>Dementia</td>
<td>N/A</td>
<td>20</td>
</tr>
<tr>
<td>Depression</td>
<td>N/A</td>
<td>33</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>N/A</td>
<td>27</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>N/A</td>
<td>30</td>
</tr>
<tr>
<td>Obesity</td>
<td>N/A</td>
<td>08</td>
</tr>
<tr>
<td>Learning disability</td>
<td>N/A</td>
<td>04</td>
</tr>
<tr>
<td>Smoking</td>
<td>N/A</td>
<td>68</td>
</tr>
</tbody>
</table>
For the year 2004/05, PMS practice expenses rose by 9.1% per GP to £153,406 and GMS expenses rose by 6.5% per GP to £120,775. These did not include pharmacy costs. (18)

The principal general practitioners have received a substantial increase in net remuneration as a direct consequence of the GMS contract. A general medical service (GMS) contract is a typical contract that a general practitioner will hold with their primary care trust (PCT) (16). The net income of a GMS general practitioner principal before the introduction of the GMS contract was £77,152 in 2003/4, rising after its introduction to £102,437 in 2004/5 (16). This represents an increase of 32.8% in the first year. The personal medical service (PMS) is locally agreed alternative to the GMS contract. (1) The PMS GP principals also had a significant increase in income in the first year of the GMS contract, with income rising from £92,168 in 2003/4 to £116,583 in 2004/5, an increase of 26% (19). However, salaried general practitioners (who represent a growing proportion of all GPs) receive significantly less pay, on a scale of £47,710 to £72,478 for 2004/05 (17). One reason why PMS practices did not receive such a substantive increase as compared with the GMS practices was that many had already received additional remuneration for additional services as part of their PMS contract, and many of these services reflected activity that was also included in the GMS contract. This was adjusted so that PMS practices did not receive a double payment for activity which appears in both the GMS contract and PMS contract.

1.4 Allocation of points to clinical areas

The allocation of points is an important issue as there is possibly a significant financial gain from achievement. It is anticipated that financial incentives will improve performance in those areas targeted, however there is potential for unintended reduction in quality practice in other areas, as those quality areas which do not receive a financial incentive might be marginalised. (20) The current basis for allocation of different points and thresholds to different clinical areas is based on the “workload, costs and importance” of each indicator. The measure of quality is however perceived quality rather than quantified evidence-based quality. The British Medical Association (BMA) has stated that the current basis for allocation for the points system was designed “for rewarding GPs and their staff for the volume and
quality of work done” (21). The GMS contract states that points have been allocated according to workload, costs and importance of each particular indicator. There is however a paucity of information on the amount of work required in general practice to meet the delivery of the clinical indicators in the new GMS contract.

To resolve this dilemma, two groups of general practitioners were formed to estimate the amount of work required to achieve each indicator. The points for each clinical indicator were then allocated based on the recommendations of these groups. There has been no additional information provided as to how the incentive points have been calculated for each particular indicator (14). This approach weighting the incentive points is therefore largely based on the perception of workload that is required to achieve each indicator.

Additionally there is also a paucity of evidence on practice performance in these clinical areas prior to 2004, so there was no accurate baseline to assess performance across many areas prior to implementation of the new contract. Research using data from before the implementation of the GMS contract demonstrates that there was already a significant level of performance in some areas covered by the contract. An observational study of 71 general practices in East Anglia in 2002/3 examined practice performance in top lifesaving prescribing interventions, six of which formed part of the GMS contract 2003 (11). Average performance was already at 47% to 70% in 6 areas covered by the new contract (Table 4) although there was significance variance between practices.
Table 4: Performance in 71 practices in East Anglia in 2002-2003

<table>
<thead>
<tr>
<th>Quality intervention</th>
<th>% patients in target group receiving intervention</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE inhibitors in heart failure</td>
<td>61.4%</td>
<td>16.7</td>
</tr>
<tr>
<td>Influenza immunization over 65yrs</td>
<td>70.4%</td>
<td>5.52</td>
</tr>
<tr>
<td>Smoking cessation advice</td>
<td>59.6%</td>
<td>27.3</td>
</tr>
<tr>
<td>Screening and treatment of hypertension</td>
<td>73.3%</td>
<td>14.2</td>
</tr>
<tr>
<td>Aspirin in heart disease</td>
<td>67.7%</td>
<td>13.4</td>
</tr>
<tr>
<td>Statins in heart disease</td>
<td>59.6%</td>
<td>14.7</td>
</tr>
</tbody>
</table>

Another study examined performance longitudinally in 3 indicators in the GMS contract: heart disease, asthma and diabetes\(^{(13)}\). Performance prior to introduction of the new GMS contract in 2003 was already above 50% for 28 out of 30 clinical indicators similar to those in the GMS contract. Analysis of longitudinal data showed improvement in all 3 clinical domains, though improvement significantly accelerated in asthma and diabetes care with the introduction of the new GMS contract.

The advantage of weighting indicators based on “the volume of work done” is that it will encourage GPs to cover all areas and discourage the GPs from focusing on areas which are easier to achieve. There is however a potential disadvantage.

The weighting of the incentive might not reflect the potential health gain of each indicator, which in turn could lead to equal financial incentives for indicators with very different potential health gain. Activity may be skewed towards areas of high workload rather than areas of greatest health gain, putting patients with non-incentivised conditions at risk of poorer care. Steel et al found that it in a limited range of indicators covering non-incentivised conditions, quality did not appear to improve.\(^{(12)}\)

The new GMS contract could have the perverse effect in encouraging increasing workload that is not directly related to increasing health gain, thus paying GPs for what they do rather than what they achieve in quality for their patients. Additionally the definition of quality practice as an outcome measure for health care may become
synonymous with quality indicators as a process measure in the GP contract. Such a narrow definition of quality may lead to less focus (and lower quality) in areas not covered by the contract, and place excess reliance of process measures as a proxy for health outcomes. A “high” performing practice in terms of the GMS contract may be weak in other areas, including local priorities for patients.

The Institute of Medicine have identified a number of factors that are desirable in implementing a P4P component.\(^{22}\) A model could be developed to award points based on a formula including a number of factors, such as potential health gain, current performance in general practice, and the amount of work needed to achieve indicators chosen for incentive payments. Such a model could also be used for developing new indicators in areas not currently covered in the GMS contract where there is evidence for quality and if current performance is substandard.

The rationale for using lives saved in this study is that this measure of health gain has substantial evidence across the different clinical areas of the new GMS contract, whereas alternative measures such as QALYs\(^{(2)}\) are at present incomplete across the clinical spectrum.
Section 2 - Aims and objectives

2.1 Remit from the Department of Health

The aims of this research were to estimate the potential population health gain of the full implementation of the interventions in the new GMS contract, both in its original form (2003)\(^{(4)}\) and its revised form (2006)\(^{(5)}\). Lives saved were the chosen measure of health gain, and many of the clinical interventions in the new GMS contract have potential to save lives. It also aimed to construct the aggregate of the number of lives saved per 100,000 populations per year at three levels namely indicator, domain and an aggregate of all indicators level. Other measures of quality were explored such as QALYs which is an indication of the benefits gained from medical procedures in terms of quality and life.\(^{(2)}\) However, this was not adopted due to the paucity of information in the literature, which has lead to a companion independent study at the University of York which has explored the cost effectiveness of a subset of the clinical indicators. Our research was conducted in the following manner:

1. A search was made in four sources for the highest level of evidence for lives saved in terms of absolute risk reduction (ARR), these sources being the supporting documentation for the GMS contract\(^{(4, 5)}\), The National Institute of Health and Clinical Excellence\(^{(7)}\), Clinical Evidence\(^{(8)}\) and the Cochrane Library\(^{(9)}\).

2. The risk reduction (RR) in mortality was approximated to a standardised year, with the assumption of a linear relationship between mortality and time. An adjustment was made for the prevalence of each condition to estimate the maximum health gain across a standardized population of 100,000 in terms of number of lives saved.

2.2 Measure of health gain

The measure of population health gain used is the number of potential lives saved per 100,000 people receiving the intervention specified in each clinical indicator, over one year, for each of the clinical indicators where evidence for lives saved can be determined. The disadvantage of lives saved as a quality measure is that it is a narrow
measure of health gain, it does not reflect quality activity that results in quality improvement that does not have a measure of survival.

Lives saved is still a valid and relevant measure of quality in the GMS contract as in its present form the contract contains 42 indicators directly and indirectly linked to interventions which save lives, and these account for a possible 432 points out of 655 points available in the clinical domain. A life saved is a robust indicator, still favoured by the World Health Organization and the DoH as the unit with which to compare health performance in many areas. All cause mortality is the measure that is chosen.

2.3 Prevalence

Prevalence data was taken directly from the QOF data submitted by each practice in England in 2005/6\(^3\). Where this was not available (such as the new GMS contract indicators) then data from the Scottish QOF was used as this was published in advance of the data from English practices\(^{(23)}\). The prevalence data was used as part of the computation of lives saved per 100,000 populations per year, needed to multiply the absolute risk reduction per patient by the estimated number of patients with the specified condition (i.e. prevalence)
Section 3 - Methodology

3.1 Research Question

The main aims of the study were to estimate the maximum potential population health gain from the full implementation of the 2003\(^{(4)}\) and 2006 GMS contract\(^{(5)}\). The study also aimed to construct the aggregate number of lives saved per 100,000 populations per year at domain level and an overall aggregate of all the clinical indicators in the contract adjusted for potential double counting.

3.2 Sources of evidence

To identify the highest level of evidence four sources of evidence were searched; the supporting documentation to the GMS contract 2003 \(^{(4)}\), and revised GMS contract 2006\(^{(5)}\); The National Institute for Health and Clinical Excellence\(^{(7)}\); Clinical Evidence Database\(^{(8)}\); the Cochrane Library Database\(^{(9)}\).

Systematic reviews were not carried out for each indicator as the numbers of clinical indicators are such that systematic reviews were not possible in the timescale of this project. However, the four sources for defining the evidence base were selected as they are regarded as being authoritative and regularly updated sources.

3.3 Strength of evidence

We have used the classic “hierarchy of evidence” grading scale designed by AHCPR (1992)\(^{(10)}\) and adopted by the Cochrane Library Database\(^{(9)}\).

Table 5: Grade of Evidence\(^{(10)}\)

<table>
<thead>
<tr>
<th>Grade of evidence</th>
<th>Type of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia</td>
<td>Evidence from meta-analysis of randomised controlled trials</td>
</tr>
<tr>
<td>Ib</td>
<td>Evidence from at least one randomised controlled trial</td>
</tr>
<tr>
<td>IIA</td>
<td>Evidence from at least one controlled study without randomisation</td>
</tr>
<tr>
<td>IIb</td>
<td>Evidence format least one other type of quasi-experimental study</td>
</tr>
<tr>
<td>III</td>
<td>Evidence from observational studies</td>
</tr>
<tr>
<td>IV</td>
<td>Evidence from expert committee reports or experts</td>
</tr>
</tbody>
</table>

The highest level of evidence was sought for each clinical indicator. Where more than one study was found then all studies of equal quality were included in the results. When the evidence for each indicator in the health league table was selected and there
was more than one study of the highest and equal level of evidence, then the study selected was the one that most closely fitted the indicator in terms of patient characteristics.

3.4 Search Strategies

Searches were conducted independently by two authors (RF, SP) following the search strategies mentioned in Appendix A. Searches were made online of the three electronic databases, namely the Cochrane Library\(^9\), NICE\(^7\) and CE\(^8\). The supporting documentation for the new GMS contract\(^4,5\) was searched manually and references was followed up to the source document for evidence for each indicator. A common search strategy was used at the domain level for each clinical condition, and a specific search strategy was developed for each indicator as described in detail in Appendix A. Studies selected were of the highest grade of evidence and are listed in the detailed summary for each indicator. In the second stage of the search process, two researchers (RF, SP) reviewed all the studies that had been selected in the initial stage of the search process as outlined above. These studies were ranked in quality against the criteria defined by AHCPR\(^10\). The highest quality study was selected, and the studies of a lower level of strength were excluded and are summarized in Appendix C.

3.5 Data Extraction

Data from the selected studies included the study characteristics (author, year), population characteristic (age, number of participants), intervention characteristic (type of intervention, duration) and outcome characteristic (all cause mortality). Measures for mortality reduction in terms of relative risk, odds ratio, absolute risk reduction including 95% confidence intervals, and control event rate were extracted from each study and these data are presented in a tabular form under each individual clinical indicator.

3.6 Calculating risk reduction

ARR was used when reported in the meta-analysis and when the trial participants’ characteristics closely matched to those in the GMS clinical indicator. Linearity was
assumed regarding the effectiveness of the treatment over time. For e.g. If we assumed that when a risk was given for say, 5 year period, then it was annualized by dividing by 5. Therefore, “the reported event rates are annualized by dividing by the number of years of exposure”. This method will slightly understate the true annual rate in mortality if the rate is assumed to remain constant each year. This is because at the end of year 1 only the survivors can die during year 2. Therefore this would require the mortality rate to gradually increase year by year in order to include all the deaths in the study. Consequently ARR is also understated leading to a conservative measure of lives saved. Where the ARR was not stated in a meta-analysis for a particular indicator, then it was calculated from the relative risk reduction (RRR) quoted in the meta-analysis. Subsequently, the ARR was calculated using control event rates from a study identified in the indicator where the patients’ characteristics most closely matched the GMS contract indicator. This was calculated using the following formula:

\[
\text{ARR (1 year)} = \frac{\text{RRR} \times P_{CT}}{T}
\]

Where \( P_{CT} \) was the baseline risk of dying over \( T \) years in control patients, the control event rate

\[
\text{ARR (T)} \quad \text{Absolute risk reduction for a given period of time}
\]

\[
T \quad \text{Length of study (years)}
\]

Where a RCT was the best level of evidence, the ARR was calculated directly from the data provided in the study, and approximated to the effect in one year using the following calculation:

\[
\text{Control group mortality – experimental group mortality} \div \text{Length of study (years)}
\]

There were both significant advantages and disadvantages with using ARR. The advantage that ARR had over relative risk (RR) and odds ratios (OR) was that readers can judge the clinical significance of the risk, and this was recommended as the optimum way of reporting trial conclusions.\(^{(24)}\) The disadvantage was that the ARR was critically dependant on the control event rate (CER), or absolute risk (AR) of an
event happening in the control group. This issue is discussed in the control event rate section that follows.

**Converting odds ratios into population absolute risk reduction**

When used as an approximation to relative risk OR overestimated the potential lives saved. The proportion of lives saved by which they overstate depended solely on the intervention event rate (IER), and is:

\[
\text{IER} \over (1 - \text{IER})
\]

Thus if lives saved was calculated using the OR instead of the RR, then this was multiplied by \(1 - \text{IER}\) to obtain the “true” lives saved.

The intervention event rate was not always available, and in this circumstance the control event rate was used as the proxy measure, which was always larger than the IER in treatments that had shown to confer benefit and hence understates the ARR.

3.7 **Calculating prevalence data**

Prevalence data was obtained from the QOF returns from each general practice in England for the year 2005/6\(^{(19)}\). This was determined from the denominator for each particular indicator (which represented the number of patients eligible for that intervention). These were summated, and expressed as a percentage of the total number of patients registered with the 8410 practices in England. The limitations of this as a tool were inaccuracies of identifying cases, and exception reporting. Both of these have potentially incorrectly reduced the prevalence for that particular indicator. However, it remained a true reflection of the numbers of cases identified in each English general practice as eligible for the intervention of that particular indicator. Where English data on QOF was not available, then data from Scotland was used as the closest estimation, as Scottish QOF data was published in advance of English QOF data.\(^{(23)}\)
3.8 Control event rate

ARR was calculated from a RCT for a particular indicator only when the patients in that RCT closely matched the patients within that particular indicator. It is of importance that the control event rate is used from clinical trials including patients with similar baseline risk as the particular indicator which was higher in patients with increased baseline risk. When ARR was calculated using RRR from meta-analysis, the most suitable RCT identified in this literature review which reflected the patient characteristics of the indicator was used to estimate the control event rate. If no suitable RCT was identified, then a further literature search was made to identify estimates of the control event rate for that particular indicator. If more than one well matched RCT was identified, then all suitably matched trials were included and each trial weighted by the number of patient years. This only applied when RRR from meta-analysis was used.

3.9 Sensitivity analysis and confidence intervals

Sensitivity analysis was performed when possible, to estimate the ranges of expected population health gain for each intervention. The maximum health gain was calculated for the expected health gain, with 95% confidence intervals where possible for individual studies. Where several studies were identified with differing risk reductions for a particular clinical indicator, then the upper and lower risk reductions were used to estimate an upper and lower limit to the population health gain. As the relationship between different studies and their risk reduction estimates was not known, it was not robust to use an “average” to estimate a midpoint value for expected health gain for a particular indicator. In this case, results from the study that best fitted a clinical indicator as specified in the search strategy was used as a midpoint value for that indicator. The most recent study was selected in case of studies with similar clinical profiles of patients. When health gains from all the indicators were combined, the lower and upper values of estimated health gain were used as the upper and lower limits of expected health gain.
3.10  Calculating population health gain (PHG)

A method for calculating population health gain originally used by Mant and Hicks\(^{(25)}\) and further developed by McColl et al\(^{(26)}\) was used in estimating the likely health gain of full implementation of an intervention in a typical primary care trust population of 100,000 people. The reason for this approach was that the number needed to treat (NNT) is a meaningful way of expressing the benefit of an active treatment over a control, and is a format which doctors can understand.\(^{(27)}\)

The ARR was converted to population health gain (PHG) in a typical population of 100,000 using the following formula

\[
\text{PHG} = N \times \text{ARR}_T \text{ (1 year)}
\]

Or alternatively

\[
\text{PHG} = \frac{N \times \text{RRR} \times \text{Pc}_T}{\text{T}}
\]

Where:

- \(N\)  Number of eligible patients in 100,000 populations
- \(T\)  Number of years over which the original study data was collected
- \(\text{Pc}_T\)  Baseline risk of dying over the next \(T\) years in control patients
- \(\text{RRR}\)  Relative risk reduction in risk of dying
- \(\text{ARR}_T\)  Absolute risk reduction in risk of dying over next \(T\) years

The assumptions and limitations of this method was that the benefit of an intervention in terms of mortality was a constant linear relationship with time.

3.11  Potential double counting

Double counting becomes an issue when estimating the maximum potential health gain of the new GMS contract, where achievement in all of the indicators is considered together in a composite calculation. There are several main issues with potential double counting with some of the indicators of care in the new GMS contract.
3.11a  One clinical disorder, multiple interventions

Some patients with a sole pathology may have different interventions for which an evidence base for effectiveness exists for each intervention individually. For example, a patient with heart disease will benefit from multiple interventions such as smoking cessation, treatment to control hypertension and taking aspirin and beta blockers. However, what is not widely known is the effectiveness of each intervention incrementally, and whether the order of adding additional interventions makes a difference. Clinical trials have identified the benefits for each individual intervention, but the evidence is sparse for particular combinations of interventions.

The currently accepted hypothesis is that when 2 different drugs are effective in the same patient and their effects are independent, the relative risk observed with the combination of drugs is the product of the relative risks observed with each drug.\(^{(27)}\)

A recent study by Hippisley-Cox\(^{(28)}\) has attempted to test this hypothesis of the potential benefit of additional drugs in heart disease. This was an open prospective cohort study of patients with ischaemic heart disease whose notes are included in the QResearch database\(^{(29)}\). Drugs examined were statins, aspirin, ACE inhibitors and beta blockers. As further drugs were added the risk of all cause mortality fell confirming a synergistic effect. The precise mechanism of the relationship between the falling mortality with the addition of further drugs was not clear. This was an observational study, and hence is at risk of bias and confounding.

Other researchers have approached this dilemma of how to estimate combined benefits of more than one drug in different ways. McColl et al identified the possibility of complex interactions between conditions and treatments, though only considered the benefits of the interventions in isolation\(^{(26)}\). Unal et al makes the assumption that benefit in terms of absolute risk reduction was additive across the population and for specified individuals\(^{(30)}\). Capewell et al provides evidence that in the case of beta blockers and aspirin that the effect on ARR approximates to additive: that is that the ARR for aspirin post myocardial infarction was 0.7% per year, beta blocker 2.3%, actual combined aspirin and beta blocker 2.9% (expected 3%)\(^{(31)}\). Wald
et al assumes the RRR were a factor of multiples in their calculation of the anticipated risk reduction for the polypill\(^{(32)}\).

In an ideal situation one would have chosen to combine all indicators with multiple treatments by using a multiple of the RR. However what is unknown was the overlap of patients with different pathologies in the new GMS contract. We have therefore taken the pragmatic approach in both of these conditions to add the ARR when combining health gain across the indicators. The addition of ARR in estimating the benefit of combination therapy will differ marginally from using multiples of RRs that is ARR if a function of 100% of patients, whereas combinations of RRs take into account losses due to mortality in the control group. This difference only becomes significant when there are high mortality reductions from interventions. It may however lead to a slight overestimate of the combined health gain across all indicators when comparing health gain calculated from multiples of RRR.

3.11b Multiple disorders, one intervention.

Some patients have multiple pathologies such as diabetes with microalbuminuria and heart disease for which one intervention may be of multiple benefits. For this specific example, a patient appeared on the diabetic indicator for diabetes and proteinuria with the intervention taking and angiotensin converting enzyme (ACE) inhibitor drug (DM 15). Likewise the same patient also appeared in the heart disease indicator for taking an ACE inhibitor drug (CHD 11).

The extent of benefit from a drug intervention is dependant on the baseline risk of the patient, and a patient with multiple pathologies has a greater baseline risk. It is therefore plausible that patients with more than one disease would be expected to have a greater potential benefit from a particular drug intervention due to their increased baseline risk. There were no studies in the literature quantifying the increased ARR in mortality in patients with combinations of diseases in the GMS clinical indicators. We therefore proposed to take the same approach to handling this data as in the previous example that is to regard the benefits additive in terms of ARR.
### Table 6: Drugs with multiple benefit in GMS indicators

<table>
<thead>
<tr>
<th>Drug</th>
<th>GMS indicator</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statin</td>
<td>CHD 8 Stroke 8 DM 17</td>
</tr>
<tr>
<td>Antiplatelet</td>
<td>CHD 9 Stroke 12</td>
</tr>
<tr>
<td>ACE</td>
<td>CHD 11 DM 15 CKD 4</td>
</tr>
<tr>
<td>Influenza immunization</td>
<td>CHD 12 Stroke 10 DM 18COPD 8</td>
</tr>
<tr>
<td>Smoking cessation</td>
<td>CHD Stroke BP DM AsthmaSmoking 1 Smoking 2</td>
</tr>
</tbody>
</table>

### 3.11c Other exceptional circumstances where double counting may be an issue

There are 2 further specific instances where double counting could be an issue. Patients may be on both the primary and secondary hypertension registers. For example if a patient with hypertension also has heart disease (CHD 6), or diabetes (DM12), they appeared on both the respective secondary disease prevention register for their condition, and on the primary disease prevention register for screening and treatment of hypertension (BP5). This problem was handled in the following way.

Since the combined prevalence of all combinations of hypertension was unknown, an assumption was made that these conditions were independent of one another. The overlap in double counting of patients with CHD and hypertension was a product of their combined prevalence, so we expected a 12 % of patients with CHD to be double counted for health gain in respect of hypertension. Likewise in Diabetes we expected
12% of patients on DM to also be double counted. To correct this, we removed those numbers of patients from the health gain for the primary hypertension register.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Prevalence (condition x other condition)</th>
<th>Calculation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension + CHD</td>
<td>12% x 3.6%</td>
<td>0.4%</td>
</tr>
<tr>
<td>Hypertension + Diabetes</td>
<td>12% x 3.3%</td>
<td>0.39%</td>
</tr>
</tbody>
</table>

Correction for reduction in hypertension registers for primary prevention in combining health gain

\[
12\% - (0.4\% + 0.39\%) = \text{Effective prevalence of } 11.2\%
\]

This reduced the expected maximum health gain in indicator BP 5 by 3.1 lives saved per 100,000 populations per year when all indicators were combined. The formula for this calculation is:

\[
\text{Effective prevalence} \times \text{PHG} = \frac{11.2}{12.0} \times 46.3 = 3.1 \text{ lives per 100,000 per year}
\]

In diabetes there were 2 different targets for HbA1c control (DM 7, DM 20), each having a different potential health gain. We proposed to handle this data in the following manner.

Patients who had achieved DM 6/20 (HbA1c < 7.4) already had achieved DM 7 (HbA1c < 10). In estimating the maximum health gain only the health gains from DM 6/20 were used to avoid double counting.
Section 4 - Results
The analysis and synthesis for each clinical indicator was done individually.

4.1 Evidence and Data Synthesis for Each Indicator

Clinical Indicators

4.1a Secondary Prevention in Coronary Heart Disease (CHD)

i. CHD 1: The practice can produce a register of patients with coronary heart disease.

This indicator was a disease register, and had no direct effect on lives saved. A register was however a prerequisite for identifying and treating patients with heart disease.

ii. CHD 2: The percentage of patients with newly diagnosed angina (diagnosed after 1 April 2003) who are referred for exercise testing and / or specialist assessment.

This indicator was both a diagnostic tool of ischaemic heart disease, and a screening tool for selecting patients with ischaemic heart disease for angiography, to identify those patients who might benefit from surgical intervention with coronary artery bypass grafting. Coronary artery bypass grafting has benefit in terms of lives saved and quality of life, though this is not an intervention within the new GMS contract.

iii. CHD 3: The percentage of patients with coronary heart disease whose notes record smoking status in the past 15 months, except those who have never smoked where smoking status need to be recorded only once.

This indicator was a record and on its own did not have direct impact for lives saved. However, it was a prerequisite for CHD 4 that did have evidence for lives saved.

iv. CHD 4: The percentage of patients with coronary heart disease, who smoke, whose notes contain a record that smoking cessation advice or referral to a specialist service, has been offered within the last 15 months.
Table 7: Evidence for population health gain for CHD 4

<table>
<thead>
<tr>
<th>Study</th>
<th>Critchley et al, 200334 - IIb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Systematic review</td>
</tr>
<tr>
<td>Participants</td>
<td>12603 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Prospective cohort trials, previous angina/MI, smokers at baseline and smoking status recorded during the study</td>
</tr>
<tr>
<td>Exclusions</td>
<td>&lt; 2 years follow-up</td>
</tr>
<tr>
<td>Intervention</td>
<td>Observational: continued to smoke / stopped smoking</td>
</tr>
<tr>
<td>Duration</td>
<td>&gt; 2 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Mortality (continued smokers)</td>
<td>26.8% in 6.57 years. Annual adjusted rate = 4.08%</td>
</tr>
<tr>
<td>Mortality (stopped smoking)</td>
<td>18.4% in 6.57 years. Annual adjusted rate = 1.58%</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>0.64</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.58 – 0.71</td>
</tr>
</tbody>
</table>

**Data Analysis:**

**Control Event Rate**

This rate was taken directly from the control arm data of the trials identified by this meta-analysis.

**Data Synthesis**

There were no RCT’s identified that compared mortality between smoking cessation interventions and placebo in patient with coronary heart disease. Therefore the expected health gain of smoking cessation was modelled using the technique previously described by McColl et al26. For prevalence the smoking rates in the adult population are used, which are taken from the Office of National Statistics 200534. This rate was used in preference to the GMS contract data on current smoking rates in coronary heart disease as this represents the outcome of this indicator, rather than the baseline.

*Prevalence of CHD 3500 per 100,000
Prevalence of smoking 24%
*Annual mortality of current CHD smokers 4.08%
RRR mortality by smoking cessation 0.36
**Expected cessation rates on NRT/Bupropion 19%
RRR mortality by full implementation of smoking cessation = 0.36 x 19% = 0.0684
PHG (Population Health Gain) \[ N \times RRR \times \frac{Pc_T}{T} \]

\[ 840 \times 0.0684 \times 0.0408 \]

\[ \frac{1}{1} \]

**2.34 lives per 100,000 per year**

*GP Contract data 2006

**NICE TAG 39*^{35}**

**Caveats**

The likely benefit was taken from cohort studies which may have been susceptible to selection bias. When patients were diagnosed with CHD, the prevalence of smoking may have been higher than that in the general population, as this was a major risk factor for the condition. Smoking prevalence data was not taken from the GMS contract. This likely reflected the current success of smoking cessation programmes and hence underestimated the maximum health gain for this indicator. This model assumed full uptake of the smoking cessation programme.

**v. CHD 5:** The percentage of patients with coronary heart disease whose notes have a record of blood pressure in the previous 15 months.

This indicator was a diagnostic tool and on its own did not have a direct impact on the lives saved. However it was a prerequisite for CHD 6 that did have evidence for lives saved.

**vi. CHD 6:** The percentage of patients with coronary heart disease in whom the last blood pressure reading (measured in the previous 15 months) is 150/90 or less.
Data Analysis:
No studies comparing antihypertensive treatment versus placebo in people with coronary heart disease were identified.

Data Synthesis
Data used for this indicator was extrapolated from studies relating to primary prevention (refer BP 5)

\[
\text{ARR} \quad 0.32\%
\]
\[
\text{Prevalence} \quad 3600
\]
\[
\text{T} \quad 1 \text{ year}
\]
\[
\text{PHG (Population Health Gain)} = \frac{N \times \text{ARR}_T}{T}
\]
\[
3600 \times 0.0032
\]
\[
= 11.52 \text{ lives per 100,000 per year}
\]

Caveats
The control event rate reported in primary prevention was likely to be significantly lower than in secondary prevention as the baseline risk was lower. Therefore, this data analysis was likely to underestimate lives saved for this indicator. Furthermore, patients with CHD should be taking 2 drugs (CHD 10 beta-blocker, CHD 11 ACE inhibitors) both of which also are antihypertensive in their mode of action. It was therefore possible that the main benefit of lowering blood pressure was accounted for in indicators CHD 10 and CHD 11.

vii. CHD 7: The percentage of patients with coronary heart disease whose notes have a record of total cholesterol in the previous 15 months.

This indicator was a diagnostic tool and on its own did not have direct impact on the lives saved. However it was a prerequisite for CHD 8 that did have evidence for lives saved.
viii. CHD 8: The percentage of patients with CHD whose last measured total cholesterol (measured in the last 15 months) is 5 mmol/l or less.

**Table 8: Evidence for population health gain for CHD 8**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Systematic review</td>
<td>Systematic review</td>
<td>Meta-analysis</td>
</tr>
<tr>
<td>Participants</td>
<td>17617 patients</td>
<td>18727 patients</td>
<td>22595 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Randomized controlled trials versus placebo. Patients with prior myocardial infarction</td>
<td>Randomized controlled trials versus placebo. Patients with prior myocardial infarction</td>
<td>All coronary heart disease, CHD + hypercholestrolaemia. Post PTCA, Acute myocardial infarction</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Primary prevention, lack of survival data</td>
<td>Primary prevention</td>
<td>Primary prevention</td>
</tr>
<tr>
<td>Intervention</td>
<td>Pravastatin Simvastatin versus placebo</td>
<td>Pravastatin Simvastatin Lovastatin</td>
<td>Fluvastatin Simvastatin Lovastatin</td>
</tr>
<tr>
<td>Duration</td>
<td>5 – 6.1 years</td>
<td>2 – 6.1 years</td>
<td>0.5 – 6.1 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
<td>All cause mortality</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>Not stated</td>
<td>Not stated</td>
<td>Not stated</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>0.79</td>
<td>0.76</td>
<td>0.79</td>
</tr>
<tr>
<td>95% CIs</td>
<td>0.73 – 0.86</td>
<td>0.60 – 0.95</td>
<td>0.70 – 0.90</td>
</tr>
</tbody>
</table>

**Data Analysis:**

**Control Event Rate**

The best matched RCT in the above meta-analyses was the 4S study.[^39] This included patients aged 35-70 years, with a past history of angina or myocardial infarction, fasting cholesterol > 5.5 and with dietary advice. The annual mortality was 2.13%.

**Data Synthesis**

All the 3 systematic reviews had similar findings, and estimations were made using RRR, RR and the control event rate from the average of the trials included in these meta-analysis.


| RR   | 0.79 |
| RRR  | 0.21 |
| P_CT | 2.13%|
Prevalence 3600/100,000

PHG (Population Health Gain) \[ N \times RRR \times P_{CT} \]

\[ \frac{3600 \times 0.21 \times 0.0213}{1} \]

16.1 lives per 100,000 per year

North of England (2001)\(^{(37)}\)

RR 0.76
RRR 0.21
P_{CT} 2.73%
Prevalence 3600/100,000

PHG (Population Health Gain) \[ N \times RRR \times P_{CT} \]

\[ \frac{3600 \times 0.24 \times 0.273}{1} \]

18.4 lives per 100,000 per year

Caveats

These studies examined the effect of taking a statin in patients with prior myocardial infarction; though did not satisfy a particular target level for cholesterol, or a specific reduction in cholesterol.

This assumption was based on the evidence from the studies included in these meta-analyses that examined statin therapy in stable coronary disease, most closely fitting this GMS clinical indicator. The 4S trial\(^{(39)}\) had cholesterol levels of 5.5-8.0 mmol/litre without treatment, and in those who received a statin 72% had cholesterol levels <5.2 mmol/litre. Likewise, in the LIPID trial\(^{(40)}\), those patients not treated with
a statin had average cholesterol of 5.6 mmol/litre, and those treated with a statin had a cholesterol level of 4.5 mmol/litre.

The control event rate was likely to be comprised of patients taking other interventions to reduce the risk of a fatal event, thus is likely to be an underestimate of the actual potential health gain.

ix. CHD 9: The percentage of patients with coronary heart disease with a record in the last 15 months that aspirin, an alternative anti-platelet therapy, or an anti-coagulant is being taken (unless a contraindication or side effects are recorded).

Table 9: Evidence for population health gain for CHD 9

<table>
<thead>
<tr>
<th>Study type</th>
<th>Antithrombotic Trialists, 2002&lt;sup&gt;(41)&lt;/sup&gt; – Ia</th>
<th>North of England, 2001&lt;sup&gt;(37)&lt;/sup&gt; - Ia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participants</td>
<td>20006 patients</td>
<td>18574 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Previous myocardial infarction (subgroup analysis)</td>
<td>Previous myocardial infarction</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Non-randomised controlled trials</td>
<td>Not stated</td>
</tr>
<tr>
<td>Intervention</td>
<td>Antiplatelet drug</td>
<td>Aspirin as antiplatelet</td>
</tr>
<tr>
<td>Duration</td>
<td>Mean 2 years</td>
<td>Not stated</td>
</tr>
<tr>
<td>Control event rate</td>
<td>All cause mortality</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>0.89</td>
<td>Not stated (OR 0.86, 0.73 – 0.98)</td>
</tr>
<tr>
<td>95% CIs</td>
<td>SE5</td>
<td>0.60 – 0.95</td>
</tr>
<tr>
<td>Reported ARR/year</td>
<td>0.6%</td>
<td>0.7% (0.1 – 1.3) Random effects</td>
</tr>
</tbody>
</table>

Data Analysis:
Control Event Rate

This was taken from the aggregated figures published in the Antithrombotic Trialists Collaboration<sup>(41)</sup>. The caveat was that the data was aggregated and details were not stated as to how this was calculated. It was not known if adjustments were made for the different length of patient/years treatment in each study or whether this was a simple average.
Data Synthesis
The Antithrombotic Trialists Collaboration\(^{(41)}\) meta-analysis was the study that fitted the disease indicator most closely, including patients with a previous myocardial infarction, though excluding those with acute myocardial infarction.

Calculating lives saved per 100,000 Antithrombotic Trialists Collaboration 2002\(^{(41)}\)

\[
\text{PHG (Population Health Gain)} = \frac{N \times \text{ARR}_T}{T} \\
3600 \times 0.006 \\
1 \\
\text{21.6 lives per 100,000 per year}
\]

Calculating lives saved per 100,000 North of England 2001\(^{(37)}\)

Prevalence \(3600 \times \text{ARR}\)

\[
3600 \times 0.007 \\
25.2
\]

\[
\text{PHG (Population Health Gain)} = \frac{N \times \text{ARR}_T}{T} \\
3600 \times 0.7\% \\
1 \\
\text{25.2 lives per 100,000 per year}
\]

Caveats
The control event rate was reported from the selected meta-analysis. The patient group of the studies for this meta-analysis however reflected precisely the patients included in the disease indicator in the GMS contract.
**x. CHD 10:** The percentage of patients with coronary heart disease who are currently treated with a beta blocker (unless a contraindication or side effects are recorded).

**Table 10: Evidence for population health gain for CHD 10**

<table>
<thead>
<tr>
<th>Study</th>
<th>Held et al 1993(42) - Ia</th>
<th>Teo et al, 1993(43) - Ia</th>
<th>Freemantle et al(44), 1999(46) - Ia</th>
<th>North of England, 2001(37) - Ia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Study type</strong></td>
<td>Meta-analysis of placebo controlled RCT’s</td>
<td>Meta-analysis of placebo controlled RCT’s</td>
<td>Meta-analysis of placebo controlled RCT’s</td>
<td>Meta-analysis of placebo controlled RCT’s</td>
</tr>
<tr>
<td><strong>Participants</strong></td>
<td>23298 patients</td>
<td>53268 patients</td>
<td>54234 patients</td>
<td>24974 patients</td>
</tr>
<tr>
<td><strong>Inclusions</strong></td>
<td>Not stated</td>
<td>Acute myocardial infarction 9 within days or weeks</td>
<td>Any stage acute or past myocardial infarction. All trials including intravenous beta blockers</td>
<td>If treatment lasted more than one day</td>
</tr>
<tr>
<td><strong>Exclusions</strong></td>
<td>Not stated</td>
<td>Not stated</td>
<td>Trials less than one day</td>
<td>Not stated</td>
</tr>
<tr>
<td><strong>Intervention</strong></td>
<td>All beta blockers</td>
<td>All beta blockers</td>
<td>Beta blockers</td>
<td>All beta blockers</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>2 – 3 years</td>
<td>Average 1 year</td>
<td>6 – 48 months</td>
<td>6 – 36 months</td>
</tr>
<tr>
<td><strong>Outcomes</strong></td>
<td>All cause mortality</td>
<td>All cause mortality</td>
<td>Death</td>
<td>Death</td>
</tr>
<tr>
<td><strong>Control event rate</strong></td>
<td>3.8% (not adjusted)</td>
<td>6.56% (not adjusted)</td>
<td>8.2%</td>
<td>Not stated</td>
</tr>
<tr>
<td><strong>Effect Size (mortality)</strong></td>
<td>Not stated</td>
<td>OR 0.81</td>
<td>OR 0.77</td>
<td>Relative risk 0.76</td>
</tr>
<tr>
<td><strong>95% CIs</strong></td>
<td>Not stated</td>
<td>0.75 – 0.87</td>
<td>0.69 – 0.85</td>
<td>0.67 – 0.85</td>
</tr>
<tr>
<td><strong>ARR / year</strong></td>
<td>Not stated</td>
<td>Not stated</td>
<td>1.19</td>
<td>1.3 (0.7 – 1.8)</td>
</tr>
</tbody>
</table>

**Data Analysis:**

**Control Event Rate**

Control event rate for CHD can be complicated by a dual benefit from beta-blockers, firstly for all coronary heart disease, and specifically for heart failure. The control events rate for mortality was high in heart failure and affected the control event rate for CHD as a domain. Heart failure was a subcategory of CHD as almost all cases are due to CHD in the UK. As part of the QOF, heart failure accounts for just over 10% of all CHD. Therefore to identify the best estimate of control event rate, studies with heart failure rate of around 10% were selected. These were taken from the meta-analysis by Freemantle et al(44) (Refer to Table 31 – Appendix B)
Data Synthesis
The North of England evidence based guidelines development project (2001)\(^{(37)}\) was commissioned by NICE\(^{(7)}\) to update previous meta-analyses, was the most current study and the study of choice. The study itself reported absolute risk reduction.

Freemantle et al\(^{(44)}\)

PHG (Population Health Gain) \(\begin{align*} N \times \text{ARR}_T \\ T \end{align*}\)

\[
3600 \times 0.019
\]

\[
1
\]

42.8 lives per 100,000 per year

North of England 2001\(^{(37)}\) (ARR)

PHG (Population Health Gain) \(\begin{align*} N \times \text{ARR}_T \\ T \end{align*}\)

\[
3600 \times 0.013
\]

\[
1
\]

46.8 lives per 100,000 per year

From North of England, from RRR and using Control Event Rate

PHG (Population Health Gain) \(\begin{align*} N \times \text{RR} \times \text{PC}_T \\ T \end{align*}\)

\[
3600 \times 0.24 \times 0.052
\]

\[
1
\]

44.9 lives per 100,000 per year
Caveats

The benefit of beta blockers in coronary heart disease varied with a patient who had recently had a myocardial infarction, so the benefit over time was not constant. The results of the benefit identified in these meta-analyses suggested increasing benefit with increasing duration of beta blocker therapy.

There were several types of beta blockers, and they did not all have the same magnitude of treatment effect.

xi. CHD 11: The percentage of patients with history of myocardial infarction (diagnosed after 1 April 2003) who are currently treated with ACE inhibitors.

Table 11: Evidence for population health gain for CHD 11

<table>
<thead>
<tr>
<th>Study Type</th>
<th>ISIS 4, 1995⁴⁵ - Ib</th>
<th>Yusuf et al, 2000⁴⁶ - Ib</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Placebo controlled RCT</td>
<td>Placebo controlled RCT</td>
</tr>
<tr>
<td>Participants</td>
<td>58050 patients</td>
<td>9541 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Upto 24 hour post MI</td>
<td>Cardiovascular disease or diabetes in addition to hypertension, high cholesterol, smoker, microalbuminuria</td>
</tr>
<tr>
<td>Exclusions</td>
<td>&gt; 24 hours post MI</td>
<td>Heart failure, renal failure, recent stroke or MI</td>
</tr>
<tr>
<td>Intervention</td>
<td>ACE (captopril, low dose) versus placebo</td>
<td>ACE (ramipril) versus placebo</td>
</tr>
<tr>
<td>Duration</td>
<td>1 year</td>
<td>5 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>12.53% per year</td>
<td>12.2% in 5 years</td>
</tr>
<tr>
<td>Effect size (mortality)</td>
<td>Relative risk 0.96</td>
<td>Relative risk 0.84</td>
</tr>
<tr>
<td>95% CIs</td>
<td>0.92 – 1.00</td>
<td>0.75 – 0.95</td>
</tr>
<tr>
<td>ARR/year</td>
<td>0.54</td>
<td>0.37</td>
</tr>
</tbody>
</table>

Data Analysis:

Control Event Rate

The control event rate was taken from the control arm of the HOPE study by Yusuf et al⁴⁵, which has a mortality rate of 12.2% in 5 years, which approximated to 2.44% per year.
Data Synthesis
Patients included in the HOPE study\(^{(46)}\) were thought to most closely represented to those included within this disease indicator.

**ISIS 4\(^{(45)}\)**

\[
\text{PHG (Population Health Gain)} \quad \frac{N \times \text{ARR}_T}{T} \\
320 \times 0.0054 \\
1 \\
1.72 \text{ lives per 100,000 per year}
\]

**Yusuf et al (2000)\(^{(46)}\)**

\[
\text{PHG (Population Health Gain)} \quad \frac{N \times \text{ARR}_T}{T} \\
320 \times 0.0037 \\
1 \\
1.18 \text{ lives per 100,000 per year}
\]

Caveats
The HOPE study\(^{(46)}\) also included patients without a previous myocardial infarction, although the patients included all had a similar risk of cardiovascular mortality. ISIS 4\(^{(45)}\) was a trial of immediate ACE inhibitors in myocardial infarction. The effectiveness of ACE was less in this patient group (North of England guideline development group 2001\(^{(37)}\)), and the different patient characteristics may explain the small relative risk reduction and the high control event rate. Captopril had been superseded by newer ACE inhibitors which had better tolerability at higher doses with regard to renal function\(^{(47)}\).
xii. CHD 12: The percentage of patients with coronary heart disease who have a record of influenza vaccination in the preceding 1 September to 31 March.

Table 12: Evidence for population health gain for CHD 12

<table>
<thead>
<tr>
<th>Study</th>
<th>Rivetti, 2006 - Ic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Meta-analysis cohort trials only</td>
</tr>
<tr>
<td>Participants</td>
<td>68032 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>high risk patients with lung, heart, renal, diabetes, immunodeficiency, cancer, stroke, vasculitis, rheumatic disease</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Epidemic years</td>
</tr>
<tr>
<td>Intervention</td>
<td>Immunization versus nothing</td>
</tr>
<tr>
<td>Duration</td>
<td>1 year</td>
</tr>
<tr>
<td>Control event rate</td>
<td>2.86%</td>
</tr>
<tr>
<td>Effect size (Mortality)</td>
<td>RR 0.39</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.16 – 0.97</td>
</tr>
<tr>
<td>ARR/year</td>
<td>Not stated</td>
</tr>
</tbody>
</table>

Data Analysis:

Control Event Rate

Control event rates were taken from the total mortality from the control arm of the 3 trials in the systematic review which were not reported separately.

Data Synthesis

PHG (Population Health Gain) \[ N \times RRR \times Pct_T \]

\[ \frac{3600 \times 0.61 \times 0.286}{1} \]

62.8 lives per 100,000 per year

Caveats

The assumptions that were made:

- The treatment had a similar efficacy in all high risk groups in these studies.
- The control event rate was representative.
- The vaccine was well matched to the circulating virus.
- Bias may have been introduced by herd immunity, selection of patients in these cohort studies.
- Patients may have appeared in more than one domain for the influenza.
This model identified a similar RRR in mortality for influenza immunization to that of McColl et al\textsuperscript{(26)} (RRR 68%) and McElduff et al\textsuperscript{(49)} (RRR 50%). These 2 models also used similar methodology based on Mant and Hicks\textsuperscript{(25)}.

4.1b Left Ventricular Dysfunction

i. LVD / HF 1: The practice can produce a register of patients with CHD and left ventricular dysfunction.

This indicator was a disease register and on its own did not have a direct impact for lives saved. However, it was a prerequisite for LVD3 that did have evidence for lives saved.

ii. LVD / HF 2: The percentage of patients with a diagnosis of CHD and left ventricular dysfunction (diagnosed after 1 April 2003) which has been confirmed by an echocardiogram.

This indicator was a diagnostic test and on its own and did not have a direct impact for lives saved. However, it was a prerequisite for identifying patients for indicator LVD3 that did have evidence for lives saved.

iii. LVD / HF 3: The percentage of patients with a current diagnosis of heart failure due to LVD who are currently treated with an ACE inhibitor or Angiotensin Receptor Blocker who can tolerate therapy and for whom there is no contra-indication.

Table 13: Evidence for population health gain for LVD / HF 3

<table>
<thead>
<tr>
<th>Study</th>
<th>Garg et al 1995\textsuperscript{(30)} - Ia</th>
<th>Flather et al, 2000\textsuperscript{(31)} - Ia</th>
<th>North of England, 2001\textsuperscript{(37)} – Ia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Meta-analysis</td>
<td>Meta-analysis</td>
<td>Meta-analysis</td>
</tr>
<tr>
<td>Participants</td>
<td>7145 patients</td>
<td>12763 patients</td>
<td>8818 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>All heart failure</td>
<td>All heart failure</td>
<td>All heart failure</td>
</tr>
<tr>
<td>Exclusions</td>
<td>&lt; 8 weeks</td>
<td>&lt; 1 year</td>
<td>Not stated</td>
</tr>
<tr>
<td>Intervention</td>
<td>Any ACE versus placebo</td>
<td>ACE versus placebo</td>
<td>ACE versus placebo</td>
</tr>
<tr>
<td>Duration</td>
<td>3 – 42/12</td>
<td>Avg 35 months (15 – 42/12)</td>
<td>Not stated</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
<td>All cause mortality</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>Not stated</td>
<td>Not stated</td>
<td>10.75 (SOLVD)</td>
</tr>
<tr>
<td>Effect (Mortality) Size</td>
<td>OR 0.77</td>
<td>0.80</td>
<td>OR 0.69</td>
</tr>
<tr>
<td>95% CIs</td>
<td>0.67 – 0.88</td>
<td>0.74 – 0.97</td>
<td>0.53 – 0.82</td>
</tr>
</tbody>
</table>
Data Analysis:

Control Event Rate

The baseline values for mortality rates in untreated heart failure from the control arm of the SOLVD trial\(^{(52)}\) which assessed the effect of the ACE inhibitor enalapril on mortality in patients with mild to moderate heart failure, with echocardiogram diagnosis. The survival without treatment over 4 years was 57%, which was an annual mortality of 10.75%. 2 other trials in this meta-analysis were less well matched. Gilbert et al\(^{(53)}\) study was limited to 3 months including only 14 patients.

Data Synthesis

For the North of England study \(^{(37)}\)

\[
\text{PHG} = (1-\text{OR}) \times \text{CER} \times \text{prevalence} \times (1-\text{CER})
\]

\[
(1 - 0.69) \times 0.1075 \times 400 \times (1- 0.1075)
\]

\text{11.9 lives per 100,000 per year}

For Garg et al\(^{(50)}\)

\[
\text{PHG} = (1-\text{OR}) \times \text{CER} \times \text{prevalence} \times (1-\text{CER})
\]

\[
(1 - 0.77) \times 0.1075 \times 400 \times (1- 0.1075)
\]

\text{10.3 lives per 100,000 per year}

For Flather et al\(^{(51)}\)

\[
\text{PHG} = (1-\text{RR}) \times \text{CER} \times \text{prevalence}
\]

\[
0.2 \times 0.1075 \times 400
\]

\text{8.6 lives per 100,000 per year}

Caveats

The severity of heart failure had an impact on the control event rate and thus the benefit of the intervention. The assumption made was that the patients in these studies had similar severity of heart failure compared to UK patients included in this clinical indicator.
4.1c Stroke and Transient Ischaemic Attacks

i. **Stroke 1:** The practice can produce a register of patients with Stroke or TIA.

This indicator was a disease register and on its own did not have direct impact for lives saved. However it was a prerequisite for other Stroke indicators that did have evidence for lives saved.

ii. **Stroke 2:** The percentage of new patients with presumptive stroke (presenting after 1 April 2003) who have been referred for confirmation of the diagnosis by CT or MRI scan.

This indicator was a diagnostic process and on its own did not have direct impact for lives saved. However it was a prerequisite for other Stroke indicators that did have evidence for lives saved.

iii. **Stroke 3:** The percentage of patients with TIA or stroke who have a record of smoking status in the last 15 months, except those who have never smoked where smoking status need be recorded only once since diagnosis.

This indicator was a disease register and on its own did not have direct impact for lives saved. However it was a prerequisite for stroke 4 that did have evidence for lives saved.

iv. **Stroke 4:** The percentage of new patients with a history of TIA or stroke who smoke and whose notes contain a record that smoking cessation advice or referral to a specialist service, if available, has been offered in the last 15 months.

**Data Analysis:**
No trials were identified that specifically studied the mortality from smoking in patients who had suffered a previous stroke or TIA.
Data Synthesis:
The assumption was made that the risks of smoking and benefits of smoking cessation was similar to patients who had a coronary heart disease (CHD 4). For prevalence the smoking rates in the adult population were used, taken from the Office of National Statistics\(^{(34)}\). This rate was used in preference to the GMS contract data on current smoking rates in stroke, as this represented the outcome of this indicator, rather than the baseline.

\[
\text{PHG (Population Health Gain)} = \frac{N \times \text{RRR} \times \text{P}_\text{CT}}{\text{T}}
\]

\[
\frac{1500 \times 24\% \times 0.0684 \times 0.0408}{1}
\]

1.0 lives per 100,000 per year

Caveats

Modelling made the assumptions discussed in indicator CHD 3. In support of the benefit of smoking cessation after stroke, one study identified in Medline by Kammersgaard et al\(^{(54)}\) reported a significant reduction in mortality in those who did not smoke over 5 years\(^{(54)}\). (Hazard ratio was the sole summary statistic, HR 1.2; 95% CI 1.0 – 1.4)

v. Stroke 5: The percentage of patients with TIA or stroke who have a record of blood pressure in the notes in the preceding 15 months.

This indicator was a diagnostic process and on its own did not have direct impact for lives saved. However it was a prerequisite for stroke 6 that did have evidence for lives saved

vi. Stroke 6: The percentage of patients with a history of TIA or stroke in whom the last blood pressure reading (measured in last 15 months) is 150/90 or less.
Table 14: Evidence for population health gain for Stroke 6

<table>
<thead>
<tr>
<th>Study</th>
<th>Rashid et al, 2003[55] - Ia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Study type</strong></td>
<td>Meta-analysis</td>
</tr>
<tr>
<td><strong>Participants</strong></td>
<td>15527 patients</td>
</tr>
<tr>
<td><strong>Inclusions</strong></td>
<td>Ischaemic stroke, haemorrhagic stroke, TIA, Hypertension - BP level not stated</td>
</tr>
<tr>
<td><strong>Exclusions</strong></td>
<td>Acute stroke, acute TIA (less than 3 months)</td>
</tr>
<tr>
<td><strong>Intervention</strong></td>
<td>Antihypertensive treatment versus nothing</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>Not stated</td>
</tr>
<tr>
<td><strong>Outcomes</strong></td>
<td>Mortality</td>
</tr>
<tr>
<td><strong>Control event rate</strong></td>
<td>Not stated</td>
</tr>
<tr>
<td><strong>Effect size (mortality)</strong></td>
<td>OR 0.91</td>
</tr>
<tr>
<td><strong>95% CI</strong></td>
<td>0.79 – 1.05</td>
</tr>
<tr>
<td><strong>ARR/ year</strong></td>
<td>Not stated</td>
</tr>
</tbody>
</table>

**Data Analysis:**

**Control event rate**
Not applicable.

**Data Synthesis**
This study did not demonstrate evidence for lives saved from blood pressure reduction after a stroke. However, there was evidence for a significant non mortality quality gain, in reductions of myocardial infarction and recurrent stroke.

**Caveats**
This study examined the effect of blood pressure reduction on outcomes rather than the effect of a blood pressure target.

vii. **Stroke 7:** The percentage of patients with TIA or stroke who have a record of total cholesterol in the last 15 months.

This indicator was a diagnostic process and on its own did not have direct impact for lives saved. However, it was a prerequisite for Stroke 8.

viii. **Stroke 8:** The percentage of patients with TIA or stroke whose last measured total cholesterol (measured in last 15 months) is 5 mmol/l or less.
Table 15: Evidence for population health gain for Stroke 8

<table>
<thead>
<tr>
<th>Study</th>
<th>Manktelow et al, 2002&lt;sup&gt;(56)&lt;/sup&gt; - Ia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Meta-analysis</td>
</tr>
<tr>
<td>Participants</td>
<td>5 Studies</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Stroke, TIA, &gt; 18 years old</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Coronary heart disease</td>
</tr>
<tr>
<td>Intervention</td>
<td>Lipid lowering treatment</td>
</tr>
<tr>
<td>Duration</td>
<td>17 months to 4 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>Not stated</td>
</tr>
<tr>
<td>Effect size (mortality)</td>
<td>OR 0.87</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.55 – 1.39</td>
</tr>
<tr>
<td>ARR/ year</td>
<td>Not stated</td>
</tr>
</tbody>
</table>

Data Analysis:

Control event rate

Not applicable.

Data Synthesis

There was no significant benefit in lowering serum lipids (of mortality or any other endpoint).

ix. Stroke 9: The percentage of patients with a stroke shown to be non-haemorrhagic, or a history of TIA, who have a record that aspirin, an alternative anti-platelet therapy, or an anti-coagulant is being taken.

Table 16: Evidence for population health gain for Stroke 9

<table>
<thead>
<tr>
<th>Study</th>
<th>Antithrombotic Trialists Collaboration 2002&lt;sup&gt;(41)&lt;/sup&gt; - Ia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Meta-analysis of double blinded RCT’s</td>
</tr>
<tr>
<td>Participants</td>
<td>23020 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Previous stroke or TIA</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Acute stroke (though not defined in time)</td>
</tr>
<tr>
<td>Intervention</td>
<td>Aspirin, dipyridamole</td>
</tr>
<tr>
<td>Duration</td>
<td>3 years (average)</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>4.3% p.a. (pooled)</td>
</tr>
<tr>
<td>Effect size (mortality)</td>
<td>Not stated</td>
</tr>
<tr>
<td>95% CI</td>
<td>Not stated</td>
</tr>
<tr>
<td>ARR/ year</td>
<td>1.5% p = 0.002</td>
</tr>
</tbody>
</table>
Data Analysis:

Control Event Rate
The control event rate was taken from pooled data of the control arms of the studies included in this meta-analysis. The inclusion criteria for patients in this meta-analysis precisely matched with the clinical status of the patients in clinical indicator Stroke 9, and thus were assumed to be representative.

Data Synthesis
The meta-analysis reported ARR alone.

\[
\text{PHG (Population Health Gain)} = \frac{N \times \text{ARR}_T}{T}
\]

\[
1500 \times 0.015 \quad 1 \\
22.5 \text{ lives per 100,000 per year}
\]

Caveats
The assumption was that the study patients represented those in the GMS clinical indicator.

x. Stroke 10: The percentage of patients with TIA or stroke who have had influenza immunisation in the preceding 1 September to 31 March.

Data Synthesis
See CHD 12 for sources of data

\[
\text{PHG (Population Health Gain)} = \frac{N \times \text{RRR} \times \text{Pc}_T}{T}
\]

\[
1500 \times 0.61 \times 0.286 \quad 1 \\
26.2 \text{ lives per 100,000 per year}
\]
**Caveats**

This evidence was from cohort studies which are subject to bias in patient selection. Evidence exists for influenza immunization in high risk patients including stroke, TIA, coronary heart disease and diabetes. All data was aggregated and subgroup analysis was performed in individual disease groups. The assumption was made that the benefit is constant across the disease areas included in this meta-analysis.

**xi. Stroke 11:** The percentage of new patients with a stroke who have been referred for further investigation.

This indicator was a referral process.

**xii. Stroke 12:** The percentage of patients with a stroke shown to be non-haemorrhagic or a history of TIA who have a record that antiplatelet agent (aspirin, clopidogrel, dipyridamole or a combination) or an anti-coagulant is being taken (unless a contraindication or side-effects are recorded. *(This is identical to Stroke 9)*

**4.1d Hypertension**

**i. BP 1:** The practice can produce a register of patients with established hypertension.

This was a disease register and on its own did not have direct impact for lives saved. However it was a prerequisite for other BP indicators that did have evidence for lives saved.

**ii. BP 2:** The percentage of patients with hypertension whose notes record smoking status at least once since diagnosis.

This indicator was a process that is essential for implementation of BP3.

**iii. BP 3:** The percentage of patients with hypertension who smoke, whose notes contain a record that smoking cessation advice or referral to a specialist service, if available, has been offered atleast once.
**Data Analysis:**
No studies were identified that examined smoking cessation and mortality in people with hypertension. Therefore data was manipulated using the technique previously described by McColl et al\(^{(26)}\).

**Data Synthesis**
A prospective observational study by Doll et al observing the UK doctors demonstrated a RR of 0.70 between current smokers and past smokers\(^{(57)}\).

Effectiveness of smoking intervention = 0.2\% \(^{(35)}\)

The annual mortality (CER) in the same observational study\(^{(57)}\) was 3.0\%

Number of smokers = 24\% population \(^{(34)}\)

Number of smokers in hypertension register = 12000 x 24\% = 2880

\[
\text{PHG (Population Health Gain)} = \frac{N \times RRR \times 0.2 \times PCT}{T}
\]

\[
2880 \times 0.30 \times 0.03 \\
1
\]

**5.2 lives per 100,000 per year**

**Caveats**
This data came from smoking cessation from primary prevention so could be an underestimate of effect.

**iv. BP 4:** The percentage of patients with hypertension in whom there is a record of the blood pressure in the past 9 months.

This indicator was a diagnostic measurement essential for the implementation of BP5.

**v. BP 5:** The percentage of patients with hypertension in whom the last blood pressure (measured in last 9 months) is 150/90 or less.
Table 17: Evidence for population health gain for BP5

<table>
<thead>
<tr>
<th>Study</th>
<th>Murlow et al 2000[58] - Ia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Systematic review</td>
</tr>
<tr>
<td>Participants</td>
<td>21908 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Primary prevention, &gt; 60 years</td>
</tr>
<tr>
<td>Exclusions</td>
<td>&lt; 1 year duration</td>
</tr>
<tr>
<td>Intervention</td>
<td>Antihypertensive mainly beta blockers, diuretics</td>
</tr>
<tr>
<td>Duration</td>
<td>&gt; 1 year</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Total mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>Not stated</td>
</tr>
<tr>
<td>Effect Size (mortality)</td>
<td>RR 0.84</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.73 – 0.96</td>
</tr>
<tr>
<td>ARR/ year</td>
<td>0.32%</td>
</tr>
</tbody>
</table>

**Data Analysis:*

**Control Event Rate**

The control event rate was taken from the MRC trial[59]. This trial was set in the 226 general practices in the UK with 4396 patients aged 65-74. This was a primary prevention trial based in 226 practices in England; selected 4396 patients aged 65-74, with a mean BP at entry of 184/91. The trial lasted for 5.8 years and the untreated mortality rate was 2.41% per annum.

**Data Synthesis**

Using RRR from the meta-analysis (16%), and the annual control event rate from the best matched study MRC trial[59] was 2.41%).

\[
\text{ARR} = \text{Control event rate} \times RRR \\
2.41\% \times 16\% = 0.39\% \text{ per year}
\]

\[
\text{PHG (Population Health Gain)} = N \times \text{ARR} \times \text{Pc}_T \\
12000 \times 0.16 \times 0.0241 = 1
\]

\[
46.3 \text{ lives per 100,000 per year}
\]
Caveats
The evidence base was drawn from older populations which had overestimated the benefit for a younger population. However, hypertension was largely distributed amongst the older population therefore; this confounding factor was likely to be small. The evidence base was largely based on diuretics and beta blockers. This was unlikely to influence the estimation of health gain, as a systematic review comparing older and new hypertensive agents by Staessen et al found no significant effect in efficacy.(60)

4.1e Diabetes Mellitus (DM)

i. DM 1: The practice can produce a register of all patients with diabetes mellitus.

This indicator was a disease register.

ii. DM 2: The percentage of patients with diabetes whose notes record BMI in the previous 15 months.

No studies were identified with all cause mortality as an endpoint.

iii. DM 3: The percentage of patients with diabetes in whom there is a record of smoking status in the previous 15 months, except those who have never smoked where smoking status need be recorded only once since diagnosis.

This indicator was a clinical record essential for DM 4.

iv. DM 4: The percentage of patients with diabetes, who smoke and whose notes contain a record that smoking cessation advice or referral to a specialist service, where available, has been offered in the last 15 months.

Data Analysis:
No systematic reviews or RCT’s on smoking cessation specifically in people with diabetes were found. Clinical evidence stated the risk was similar to persons with coronary heart disease (though no references are given).
Data Synthesis

Health gain was assumed to be similar to coronary heart disease and data was extrapolated from this indicator (CHD 4)

*Prevalence of diabetes 3.3%
Assume smoking prevalence is that of population 24%
CER from CHD 4 current smokers 4.08%
RRR mortality by smoking cessation 0.36
** Expected cessation rates on NRT/Bupropion 19%
RRR mortality by full implementation of smoking cessation 0.36 x 19%
0.0684
RR 0.93%

PHG (Population Health Gain) \( \frac{N \times RRR \times PCT}{T} \)

\( (3300 \times 24\%) \times 0.0684 \times 0.0408 \)

1

2.21 lives per 100,000 per year

*GP contract data 2006

Caveats

This data came from smoking cessation from primary prevention so could have been an underestimate of effect.

v. DM 5: The percentage of diabetic patients who have a record of HbA1c or equivalent in the previous 15 months.

This indicator was a diagnostic test, essential for DM 6.
vi. DM 6/20: The percentage of patients with diabetes in whom the last HbA1c is 7.5 or less (or equivalent test/reference range depending on local laboratory) in the previous 15 months.

Table 18: Evidence for population health gain for DM 6/20

<table>
<thead>
<tr>
<th>Study</th>
<th>UKPDS Group 34(^{(61)}) - Ib</th>
<th>Stratton et al (UKPDS 35)(^{(62)}) - IIIa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>RCT</td>
<td>Prospective observational study (mortality and HbA1c levels)</td>
</tr>
<tr>
<td>Participants</td>
<td>753 patients</td>
<td>3642 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Newly diagnosed type II DM</td>
<td>Newly diagnosed type II DM</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Incomplete data</td>
<td>Incomplete data</td>
</tr>
<tr>
<td>Intervention</td>
<td>Metformin versus diet</td>
<td>None (observational)</td>
</tr>
<tr>
<td>Duration</td>
<td>10.7 year</td>
<td>10 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Mortality</td>
<td>Mortality HbA1c 7-8 vs HbA1c &gt; 10</td>
</tr>
<tr>
<td>Control event rate</td>
<td>2.02% per year</td>
<td>Not stated</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>0.64</td>
<td>0.737</td>
</tr>
<tr>
<td>95% CIs</td>
<td>0.45 – 0.91</td>
<td>Not stated</td>
</tr>
</tbody>
</table>

Data Analysis:

Control Event Rate

The most closely matched study was the UKPDS 34\(^{(61)}\), with the conventional arm receiving diet alone. Mortality in this arm was 21.65% in 10.7 years which was an average annual mortality of 2.02%.

Data Synthesis

PHG (Population Health Gain) \(\frac{N \times RRR \times PC_T}{T}\)

For the upper estimate UKPDS 34\(^{(61)}\):

\[
\frac{3300 \times 36\% \times 2.02\%}{1} = 24 \text{ lives per 100,000 per year}
\]
For the lower estimate UKPDS 35(62):

\[
\frac{3300 \times 26.7\% \times 2.02\%}{1} = 17.8 \text{ lives per 100,000 per year}
\]

**Caveats**

These studies were based in the secondary care outpatient clinics recruited in the late 1980s and it is possible that these patients were at had a different baseline risk than those solely managed in primary care. The difference was unlikely to be significant though as the management of the majority of patients with diabetes had been transferred from the secondary sector to primary care over the past decade.

**vii. DM 7:** The percentage of patients with diabetes in whom the last HbA1c is 10 or less (or equivalent test/reference range depending on local laboratory) in last 15 months.

**Table 19: Evidence for population health gain for DM 7**

<table>
<thead>
<tr>
<th>Study</th>
<th>Stratton et al (UKPDS 35)(62) - III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Prospective observational study (mortality and HbA1c levels)</td>
</tr>
<tr>
<td>Participants</td>
<td>3642 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Newly diagnosed type II DM</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Incomplete data</td>
</tr>
<tr>
<td>Intervention</td>
<td>None (observational)</td>
</tr>
<tr>
<td>Duration</td>
<td>10 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Mortality HbA1c 9-10 vs HbA1c &gt; 10</td>
</tr>
<tr>
<td>Control event rate</td>
<td>Not stated</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>0.90</td>
</tr>
<tr>
<td>95% CIs</td>
<td>Not stated</td>
</tr>
</tbody>
</table>

**Data Analysis**

**Control Event Rate:**

The most closely matched study was the UKPDS 34(61), with the conventional arm receiving diet alone. Mortality in this arm was 21.65% in 10.7 years, which is an average annual mortality of 2.02%.
Data Synthesis

*Stratton et al* analyzed relative risk for total mortality comparing different levels of achievement of HbA1c levels.\(^{(62)}\) There were 2 subgroup analyses relevant to this clinical indicator. Comparing HbA1c > 10 with both HbA1c 9-10 (RR 0.90), and HbA1c > 10 with HbA1c 8-9 (RR 0.78)

\[
\text{PHG (Population Health Gain)} = \frac{N \times \text{RRR} \times \text{Pc}_T}{T}
\]

For upper estimate of health gain:

\[
\text{PHG} = \frac{3300 \times 0.22 \times 2.02\%}{1}
\]

**14.7 lives per 100,000 per year**

For lower estimate of health gain:

\[
\text{PHG} = \frac{3300 \times 0.10 \times 2.02\%}{1}
\]

**6.7 lives per 100,000 per year**

Caveats

The UKPDS 35\(^{(62)}\) was an observational study so there was potential for selection bias of patients. The relative risk could have been an underestimate of the actual gain. The actual level of HbA1c in patients achieving this indicator was not known.

**viii. DM 8:** The percentage of patients with diabetes who have a record of retinal screening in the previous months.

No studies were found with evidence for mortality benefit.
ix. DM 9: The percentage of patients with diabetes with a record of the presence or absence of peripheral pulses in the previous 15 months.

No studies were found with evidence for mortality benefit.

x. DM 10: The percentage of patients with diabetes with a record of neuropathy testing in the previous 15 months.

No studies were found with evidence for mortality benefit.

xi. DM 11: The percentage of patients with diabetes who have a record of the blood pressure in the past 15 months.

This indicator was a diagnostic procedure, an essential process to indicator DM 12.

xii. DM 12: The percentage of patients with diabetes in whom the last blood pressure is 145/85 or less.

**Table 20: Evidence for population health impact for DM 12**

<table>
<thead>
<tr>
<th>Study</th>
<th>Adler et al (UKPDS 36)(43), 2000 - III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Prospective observational study</td>
</tr>
<tr>
<td>Participants</td>
<td>4801 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Type II Diabetes and patients aged 25-65 years</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Vascular disease, myocardial infarction or stroke within previous year. Major systematic illness</td>
</tr>
<tr>
<td>Intervention</td>
<td>Randomized to tight control of target blood pressure vs less tight control. BP&gt;160 and BP 140-149</td>
</tr>
<tr>
<td>Duration</td>
<td>8.4 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>2.94%</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>0.65</td>
</tr>
<tr>
<td>95% CIs</td>
<td>Not reported 0.52 – 0.81</td>
</tr>
<tr>
<td>ARR / year</td>
<td>Not stated</td>
</tr>
</tbody>
</table>
Data Synthesis
From UKPDS 36\textsuperscript{(63)} CER 2.94%

Prevalence data on hypertension in a diabetic population is taken from UKPDS 38\textsuperscript{(64)}, hypertension occurs in 36% diabetics

\begin{align*}
\text{Prevalence of hypertensive diabetes} & \quad \text{Prevalence diabetes} \times 36\% \\
& \quad 3.3\% \times 36\% \\
& \quad 1.18\%
\end{align*}

\begin{align*}
\text{PHG (Population Health Gain)} & \quad N \times \text{RRR} \times \text{PcT} \\
& \quad \frac{T}{1} \\
& \quad 1180 \times 0.35 \times 2.94\% \\
& \quad 12.1 \text{ lives per 100,000 per year}
\end{align*}

Caveats

There was no QOF data on the numbers of diabetic patients who had a blood pressure < 145/85. The prevalence of hypertension amongst diabetic patients was taken from UKPDS 38\textsuperscript{(64)} where a 36% of patients had a blood pressure >160 / >90. This would have been an underestimate of the population health impact (the indicator defines >145 / >185)

\textbf{xiii. DM 13:} The percentage of patients with diabetes who have a record of microalbuminuria testing in the previous 15 months (exception reporting for patients with proteinuria).

This indicator was a diagnostic test essential for DM 15.

\textbf{xiv. DM 14:} The percentage of patients with diabetes who have a record of serum creatinine testing in the previous 15 months.

This indicator was a diagnostic test.

\textbf{xv. DM 15:} The percentage of patients with diabetes with proteinuria or microalbuminuria who are treated with ACE-inhibitors (or A2 antagonists).
Table 21: Evidence for population health gain for DM 15

<table>
<thead>
<tr>
<th>Study type</th>
<th>Strippoli et al, 2006&lt;sup&gt;(45)&lt;/sup&gt; – Ia</th>
<th>Yusuf et al (HOPE), 2000&lt;sup&gt;(46)&lt;/sup&gt; – Ib</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Systematic review with subgroups</td>
<td>RCT</td>
</tr>
<tr>
<td>Q</td>
<td>2034 patients</td>
<td>3577 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>RCT’s, full dose ACE all stages DKD</td>
<td>Diabetes &gt; 55 years old, plus another risk factor</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Not stated</td>
<td>Not stated</td>
</tr>
<tr>
<td>Intervention</td>
<td>ACE versus placebo</td>
<td>ACE (ramipril) versus placebo</td>
</tr>
<tr>
<td>Duration</td>
<td>Not stated</td>
<td>4.5 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>Not stated</td>
<td>18.6% in 4.5 years</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>0.78</td>
<td>0.76</td>
</tr>
<tr>
<td>95% CIs</td>
<td>0.61 – 0.98</td>
<td>0.63 – 0.92</td>
</tr>
<tr>
<td>ARR / year</td>
<td>Not stated</td>
<td>Not stated</td>
</tr>
</tbody>
</table>

Data Analysis:

We identified one systematic review. The evidence base was drawn from the older populations and this may have overestimated the benefit for a younger population.

Control Event Rate

This was taken from the HOPE<sup>(46)</sup> study, which had an event rate in controls of 18.6% mortality in 4.5 years; averaging to 4.13% p.a.

Data Synthesis

Using the RRR from the meta-analysis (22%), and the control event rate was 0.0413. Prevalence of microalbuminuria in diabetes (type 2) was 30.9% of the diabetic population i.e. 1.02% of total population.<sup>(66)</sup>

\[
PHG \text{ (Population Health Gain)} = \frac{N \times RRR \times P_{\text{c}}}{T}
\]

\[
\begin{align*}
1020 \times 0.22 \times 0.0413 \\
1
\end{align*}
\]

9.26 lives per 100,000 per year
Caveats
The evidence base for this level of risk reduction only applies to patients treated with therapeutic doses of ACE inhibitor drugs, which was a subgroup analysis of the systematic review identified\(^{(65)}\). It did not apply to A2RA drugs. The current British National Formulary\(^{(47)}\) (BNF 53; 2:5:5:1) did not specify a dose for the use of ACE inhibitors in diabetic nephropathy.

xvi. DM 16: The percentage of patients with diabetes who have a record of total cholesterol in the previous 15 months.
This was a diagnostic procedure essential to DM 17.

xvii. DM 17: The percentage of patients with diabetes whose last measured total cholesterol within the previous 15 months is 5 mmol/l or less.
No studies were identified that reported significant mortality benefit.

Caveats
One study which reported a non significant mortality benefit for atorvastatin versus placebo in type 2 diabetes was terminated early due to other absolute benefits. It was possible that this study would have reached a significant mortality gain if it had not been prematurely terminated.

xviii. DM 18: The percentage of patients with diabetes who have had influenza immunization in the preceding 1 September to 31 March.

Table 22: Evidence for population health gain for DM 18

<table>
<thead>
<tr>
<th>Study</th>
<th>Rivetti et al, 2006(^{(48)}) - Ic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Meta-analysis of cohort trials only</td>
</tr>
<tr>
<td>Participants</td>
<td>68032 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>high risk patients</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Epidemic years</td>
</tr>
<tr>
<td>Intervention</td>
<td>Immunization versus nothing</td>
</tr>
<tr>
<td>Duration</td>
<td>1 year</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>2.86%</td>
</tr>
<tr>
<td>Effect size (mortality)</td>
<td>RR 0.39</td>
</tr>
<tr>
<td>95% CIs</td>
<td>0.16 – 0.97</td>
</tr>
<tr>
<td>ARR / year</td>
<td>Not stated</td>
</tr>
</tbody>
</table>
Lung, heart, renal, diabetes, immunodeficiency, cancer, stroke, vasculitis, rheumatic disease.

Data Analysis:
We identified one meta-analysis. We have assumed that the treatment had similar efficacy in all high risk groups in these studies.

Control Event Rate
Control event rates were taken from the total mortality from the control arm of the 3 trials in the systematic review which were not reported separately.

Data Synthesis
PHG (Population Health Gain) \[ \frac{N \times ARR \times PCT}{T} \]

\[ 3300 \times 0.61 \times 0.0286 \]

\[ 1 \]

57.6 lives per 100,000 per year

Caveats
The assumptions that were made are:

- The treatment had similar efficacy in all high risk groups in these studies.
- The control event rate was representative.
- The vaccine was well matched to the circulating virus.
- Bias may have been introduced by herd immunity, selection of patients in these cohort studies.
- Patients may have appeared in more than one domain for the influenza.

xix. DM 19: The practice can produce a register of all patients aged 17 years and over with diabetes mellitus, which specified whether the patient has Type 1 or Type 2 diabetes.

This indicator was a disease register.
xx. DM 21: The percentage of patients with diabetes who have a record of retinal screening in the previous 15 months

This indicator was same as DM 8.

xxi. DM 22: The percentage of patients with diabetes who have a record of estimated glomerular filtration rate (eGFR) or serum creatinine testing in the previous 15 months.

This indicator was a diagnostic test.

4.1f Chronic Obstructive Pulmonary Disease (COPD)

i. COPD 1: The practice can produce a register of patients with COPD.

This indicator was a disease register.

ii. COPD 2: The percentage of patients in whom diagnosis has been confirmed by spirometry including reversibility testing for newly diagnosed patients with effect from 1 April 2003.

This indicator was a diagnostic procedure.

iii. COPD 3: The percentage of all patients with COPD in whom diagnosis has been confirmed by spirometry including reversibility testing.

This indicator was a diagnostic procedure.

iv. COPD 4: The percentage of patients with COPD in whom there is a record of smoking status in the previous 15 months, except those who have never smoked where smoking status need be recorded only once since diagnosis.

This was a medial record essential for COPD 5.
v. COPD 5: The percentage of patients with COPD, who smoke, whose notes contain a record that smoking cessation advice or referral to a specialist service, where available, has been offered in the past 15 months.

Table 23: Evidence for population health gain for COPD 5

<table>
<thead>
<tr>
<th>Study</th>
<th>Anthonisen et al 2005&lt;sup&gt;67&lt;/sup&gt; - Ib</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>RCT</td>
</tr>
<tr>
<td>Participants</td>
<td>5887 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Smokers, 35 – 60 years of age</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Serious illness e.g. cancer, IHD, hypertension</td>
</tr>
<tr>
<td>Intervention</td>
<td>Smoking cessation program versus usual care</td>
</tr>
<tr>
<td>Duration</td>
<td>14.5 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>1.08 p.a.</td>
</tr>
<tr>
<td>Effect size (mortality)</td>
<td>RR 0.815</td>
</tr>
<tr>
<td>95% CIs</td>
<td>Not reported (p=0.03)</td>
</tr>
<tr>
<td>ARR / year</td>
<td>Not stated</td>
</tr>
</tbody>
</table>

Data Analysis:

Control Event Rate

The control event rate was taken from the above study.

Data Synthesis

Clinical evidence quoted smoking prevalence rates of 90% in patients who had developed COPD. The absolute prevalence rates were the product of smoking rates and prevalence of COPD, 90% x 1.4% = 1260 per 100,000

\[
\text{PHG (Population Health Gain)} = \frac{N \times RRR \times P_c \times \text{RRR} \times P_e}{T}
\]

\[
1260 \times 0.19 \times 0.0108 = 2.58 \text{ lives per 100,000 per year}
\]
Caveats
Mortality data used was taken from the USA and Canada. Patients with cancer and ischaemic heart disease were excluded, which could lead to an underestimate of treatment benefits.

vi. COPD 6: The percentage of patients with COPD with a record of FeV1 in the previous 27 months.
This was a clinical measurement.

vii. COPD 7: The percentage of patients with COPD receiving inhaled treatment in whom there is a record that inhaler technique has been checked in the preceding 27 months.
This was a management procedure.

viii. COPD 8: The percentage of patients with COPD who have had influenza immunisation in the preceding 1 September to 31 March.

Table 24: Evidence for population health gain for COPD 8

<table>
<thead>
<tr>
<th>Study</th>
<th>Rivetti et al, 2006[^1^] - Ic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Meta-analysis of cohort trials only</td>
</tr>
<tr>
<td>Participants</td>
<td>68032 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>high risk patients</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Epidemic years</td>
</tr>
<tr>
<td>Intervention</td>
<td>Immunization versus nothing</td>
</tr>
<tr>
<td>Duration</td>
<td>1 year</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>2.86%</td>
</tr>
<tr>
<td>Effect size (mortality)</td>
<td>RR 0.39</td>
</tr>
<tr>
<td>95% CIs</td>
<td>0.16 – 0.97</td>
</tr>
<tr>
<td>ARR / year</td>
<td>Not stated</td>
</tr>
</tbody>
</table>

[^1^]: Lung, heart, renal, diabetes, immunodeficiency, cancer, stroke, vasculitis, rheumatic disease.
Data Analysis:

Control Event Rate

Control event rates were taken from the total mortality from the control arm of the 3 trials in the systematic review which were not reported separately.

Data Synthesis

PHG (Population Health Gain) \[ \frac{N \times ARR \times P_{CTR}}{T} \]

\[
\frac{1400 \times 0.61 \times 0.0286}{1}
\]

24.4 lives per 100,000 per year

Caveats

The assumptions that were made are:

- The treatment had similar efficacy in all high risk groups in these studies.
- The control event rate was representative.
- The vaccine was well matched to the circulating virus.
- Bias may have been introduced by herd immunity, selection of patients in these cohort studies.
- Patients may have appeared in more than one domain for the influenza.

ix. COPD 9: The percentage of all patients with COPD in whom diagnosis has been confirmed by spirometry including reversibility testing.

This indicator was a diagnostic test.

x. COPD 10: The percentage of patients with COPD with a record of FeV1 in the previous 15 months.

This indicator was a clinical measurement.

xi. COPD 11: The percentage of patients with COPD receiving inhaled treatment in whom there is a record that inhaler technique has been checked in the previous 15 months.

This indicator was a clinical procedure, without evidence for benefit in terms of mortality.
4.1g Epilepsy

i. Epilepsy 1: The practice can produce a register of patients receiving drug treatment for epilepsy.

This was a disease register.

ii. Epilepsy 2: The percentage of patients aged 16 and over on drug treatment for epilepsy who have a record of seizure frequency in the previous 15 months.

This was a clinical record.

iii. Epilepsy 3: The percentage of patients aged 16 and over on drug treatment for epilepsy who have a record of medication review in the previous 15 months.

This was a clinical record.

iv. Epilepsy 4: The percentage of patients aged 16 and over on drug treatment for epilepsy who have been seizure free for the last 12 months recorded in the last 15 months.

This was a clinical record.

v. Epilepsy 5: The practice can produce a register of patients aged 18 and over receiving drug treatment for epilepsy.

This indicator was a disease register.

vi. Epilepsy 6: The percentage of patients age 18 and over on drug treatment for epilepsy who have a record of seizure frequency in the previous 15 months.

This indicator was a medical record.
vii. Epilepsy 7: The percentage of patients age 18 and over on drug treatment for epilepsy who have a record of medication review involving the patient and/or carer in the previous 15 months.

No studies were identified with mortality benefit.

viii. Epilepsy 8: The percentage of patients age 18 and over on drug treatment for epilepsy who have been seizure free for the last 12 months recorded in the previous 15 months.

This indicator was a medical record.

4.1h Hypothyroidism

i. Thyroid 1: The practice can produce a register of patients with hypothyroidism.

This was a disease register.

ii. Thyroid 2: The percentage of patients with hypothyroidism with thyroid function tests recorded in the previous 15 months.

No studies were identified with evidence for mortality benefit.

4.1i Cancer

i. Cancer 1: The practice can produce a register of all cancer patients diagnosed after 1 April 2003.

This was a disease register.

ii. Cancer 2: The percentage of patients with cancer diagnosed from 1 April 2003 with a review by the practice recorded within six months of confirmed diagnosis. This should include an assessment of support needs, if any, and a review of co-ordination arrangements with secondary care.
No studies were identified that reported mortality benefit.

**iii. Cancer 3:** The percentage of patients with cancer, diagnosed within the last 18 months who have a patient review recorded as occurring within six months of the practice receiving confirmation of the diagnosis.

No studies were identified with all cause mortality as endpoint.

**4.1j Mental Health (MH)**

**i. MH 1:** The practice can produce a register of people with severe long-term mental health problems who require and have agreed to regular follow-up.

This was a disease indicator.

**ii. MH 2:** The percentage of patients with severe long-term mental health problems with a review recorded in the preceding 15 months. This review includes a check on the accuracy of prescribed medication, a review of physical health and a review of co-ordination arrangements with secondary care.

No studies were identified with all cause mortality as endpoint.

**iii. MH 3:** The percentage of patients on lithium therapy with a record of lithium levels checked within the previous 6 months.

This was a clinical measurement.

**iv. MH 4:** The percentage of patients on lithium therapy with a record of serum creatinine and TSH in the preceding 15 months.

No studies were identified with all cause mortality as endpoint.

**v. MH 5:** The percentage of patients on lithium therapy with a record of lithium levels in the therapeutic range within the previous 6 months.
No studies were identified with all cause mortality as endpoint.

**vi. MH 6:** The percentage of patients on the register who have a comprehensive care plan documented in the records agreed between individuals, their family and/or carers as appropriate.

No studies were identified with all cause mortality as endpoint.

**vii. MH 7:** The percentage of patients with schizophrenia, bipolar affective disorder and other psychoses who do not attend the practice for their annual review who are identified and followed up by the practice team within 14 days of non-attendance.

This indicator was a management procedure.

**viii. MH 8:** The practice can produce a register of people with schizophrenia, bipolar disorder and other psychoses.

This was a disease register.

**ix. MH 9:** The percentage of patients with schizophrenia, bipolar affective disorder and other psychoses with a review recorded in the preceding 15 months. In the review there should be evidence that the patient has offered routine health promotion and prevention advice appropriate to their age, gender and health status.

No studies were identified that report mortality benefit.

**4.1k Asthma**

**i. Asthma 1:** The practice can produce a register of patients with asthma, excluding patients with asthma who have been prescribed no asthma-related drugs in the last twelve months.

This was a disease register.
ii. Asthma 2: The percentage of patients aged 8 or over diagnosed as having asthma from 1 April 2003 where the diagnosis has been confirmed by spirometry or peak flow measurement.

This was a diagnostic procedure.

iii. Asthma 3: The percentage of patients with asthma between the ages of 14 and 19 in whom there is record of smoking status in the previous 15 months.

This was a record essential for Asthma 5.

iv. Asthma 4: The percentage of patients aged 20 or over with asthma whose notes record smoking status in the past 15 months, except those who have never smoked where smoking status need be recorded only once since diagnosis.

This was a record essential for Asthma 5.

v. Asthma 5: The percentage of patients with asthma who smoke, and whose notes contain a record that smoking cessation advice or referral to a specialist service, where available, has been offered within the last 15 months.

No studies that reported smoking cessation and mortality specifically to asthma were identified.

Data Synthesis
The assumption was made that smoking cessation in asthma patients would have benefits in terms of mortality which were at least as great as smoking cessation in the general population. This data had been described previously in BP4. Prevalence for asthmatics who smoke is taken directly from QOF data.\(^{(3)}\)

<table>
<thead>
<tr>
<th>RRR</th>
<th>0.30</th>
</tr>
</thead>
<tbody>
<tr>
<td>P(_{CT})</td>
<td>0.03</td>
</tr>
<tr>
<td>Prevalence of asthmatics who smoke (from QOF 2006)(^{(3)})</td>
<td>(523007)</td>
</tr>
<tr>
<td></td>
<td>(53211253)</td>
</tr>
</tbody>
</table>
PHG (Population Health Gain) \[
\frac{N \times RRR \times \text{Pce}_T}{T}
\]

\[
980 \times 0.30 \times 0.03
\]

\[
1
\]

8.82 lives per 100,000 per year

Caveats
The assumptions were that mortality and benefit from smoking cessation were similar to the general population. The asthma population is younger, hence would be expected to have a lower control event rate, though greater life long benefit in terms of lives saved.

vi. Asthma 6: The percentage of patients with asthma who have had an asthma review in the last 15 months.

This was a clinical review.

vii. Asthma 7: The percentage of patients aged 16 and over with asthma who have had influenza immunization in the preceding 1 September to 31 March.

No studies were identified that reported benefit in patients solely with Asthma. A meta-analysis was found that reported benefit in high risk patients, though this also included malignancy, coronary artery disease, renal failure, diabetes and stroke. This was not thought to be representative for this clinical indicator which was largely composed of low risk patients.

viii. Asthma 8: The percentage of patients aged 8 or over diagnosed as having asthma for 1 April 2006 with measures of variability or reversibility.

This was a diagnostic procedure.
4.1i Dementia

i. DEM 1: The practice can produce a register of patients diagnosed with dementia.

This indicator was a disease register.

ii. DEM 2: The percentage of patients diagnosed with dementia whose care has been reviewed in the previous 15 months.

No studies were identified with all cause mortality as endpoint.

4.1m Depression

i. DEP 1: The percentage of patients on the diabetes register and/or the CHD register for whom case finding for depression has been undertaken on one occasion during the previous 15 months using two standard screening questions.

No studies were identified with all cause mortality as endpoint.

ii. DEP 2: In those patients with a new diagnosis of depression, recorded between the preceding 1 April to 31 March, the percentage of patients who have had an assessment of severity at the outset of treatment using an assessment tool validated for use in primary care.

No studies were identified with all cause mortality as endpoint.

4.1n Chronic Kidney Disease

i. CKD 1: The practice can produce a register of patients aged 18 years and over with CKD (US National Kidney Foundation: Stage 3 to 5 CKD)

This indicator was a disease register.
ii. CKD 2: The percentage of patients on the CKD register whose notes have a record of blood pressure in the previous 15 months.

This indicator was a clinical measurement essential for CKD 3.

iii. CKD 3: The percentage of patients on the CKD register in whom the last blood pressure reading, measured in the previous 15 months, is 140/85 or less.

Table 25: Evidence for population health gain for CKD 3

<table>
<thead>
<tr>
<th>Study</th>
<th>Ogden et al, 2000&lt;sup&gt;68&lt;/sup&gt; - IIb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Modelling of prospective cohort study</td>
</tr>
<tr>
<td>Participants</td>
<td>Not stated</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Renal, cardiovascular or diabetic disease with hypertension. Stratified systolic blood pressure low/medium/high</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Not stated</td>
</tr>
<tr>
<td>Intervention</td>
<td>Comparison of survival in risk groups</td>
</tr>
<tr>
<td>Duration</td>
<td>10 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Mortality with modelling of impact of 12 mm Hg reduction</td>
</tr>
<tr>
<td>Control event rate</td>
<td>Not stated</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>NNT 8-10 in 10 year</td>
</tr>
<tr>
<td>95% CIs</td>
<td>Not stated</td>
</tr>
<tr>
<td>ARR / year</td>
<td>0.71% baseline BP 130-139/85-89&lt;sup&gt;*&lt;/sup&gt; 1.11% baseline BP &gt;160/&gt;100</td>
</tr>
</tbody>
</table>

Data Synthesis
ARR was reported as an outcome in this study. A range of ARR was reported, depending on the degree of blood pressure reduction, so calculations were made using the upper and lower ranges. The returns for QOF in Scotland for 2006-7 gave a prevalence of 1.8%.<sup>(23)</sup>

At higher BP levels:
NNT 90 p.a. to save 1 life
ARR p.a. = 0.011

\[
\text{PHG (Population Health Gain)} = \frac{N \times ARR_T}{T}
\]
1800 x 0.011

1

19.8 lives per 100,000 per year

At lower BP levels:

PHG (Population Health Gain) = \( \frac{N \times ARR_T}{T} \)

1800 x 0.0071

1

12.78 lives per 100,000 per year

Caveats

There were 3 significant assumptions made in using data from Ogden et al\(^{68}\). Firstly, the patients included in this model were not exclusive to CKD. Patients with diabetes, heart attack, heart failure and stroke were also included in analysis. In the case of diabetes and heart attack, there was a separate evidence base for effectiveness of reducing blood pressure that led to an overestimate of the effect of reducing blood pressure in CKD in this model.

Secondly, it assumed a reduction in systolic blood pressure of 12 mm of Hg. This study was relevant as a 12 mm Hg reduction in systolic blood pressure is the average BP reduction in the use of antihypertensive agents in renal disease.\(^{69}\) Thirdly, Ogden et al demonstrated a similar effect in risk reduction irrespective of initial blood pressure level, whereas this indicator had a cut off level for blood pressure below 140/85 mm Hg.\(^{68}\)

The prevalence of CKD stages III and IV was estimated to be 4.9% in the UK population.\(^{70}\), which is much higher than the 1.8% prevalence figure given in the QOF returns for Scotland.\(^{23}\)
iv. **CKD 4**: The percentage of patients on the CKD register who are treated with an angiotensin converting enzyme inhibitor (ACE-I) or angiotensin receptor blocker (ARB) (Unless a contraindication or side-effects are recorded).

**Table 26: Evidence for population health gain for CKD 4**

<table>
<thead>
<tr>
<th>Study</th>
<th>Jafar et al, 2003&lt;sup&gt;71&lt;/sup&gt; - 1a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Meta-analysis</td>
</tr>
<tr>
<td>Participants</td>
<td>Not stated</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Non-diabetic kidney disease</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Acute renal failure, type I diabetes, transplant, congestive heart failure, obstructive uropathy</td>
</tr>
<tr>
<td>Intervention</td>
<td>ACE versus non ACE antihypertensive drugs</td>
</tr>
<tr>
<td>Duration</td>
<td>Mean 2.2 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Progression of renal disease with ACE, in patients with hypertension and proteinuria &gt; 1g/d</td>
</tr>
<tr>
<td>Control event rate</td>
<td>Not stated</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>0.67</td>
</tr>
<tr>
<td>95% CIs</td>
<td>0.53 – 0.84</td>
</tr>
<tr>
<td>ARR / year</td>
<td>Not stated</td>
</tr>
</tbody>
</table>

**Data Synthesis**

Mortality was not reported as the endpoint in this meta-analysis. The outcome was the progression of renal disease which was significantly lower in the group receiving ACE inhibitors. (RR 0.67, CI 0.53 – 0.84)

**4.1o Atrial Fibrillation (AF)**

i. **AF 1**: The practice can produce a register of patients with atrial fibrillation.

This indicator was a disease register.

ii. **AF 2**: The percentage of patients with atrial fibrillation diagnosed after 1 April 2006 with ECG or specialist confirmed diagnosis.

This indicator was a diagnostic procedure.

iii. **AF 3**: The percentage of patients with atrial fibrillation who are currently treated with anti-coagulant drug therapy or an anti-platelet therapy.
Table 27: Evidence for population health gain for AF 3

<table>
<thead>
<tr>
<th>Study</th>
<th>Aguilar M. I. &amp; Hart R. 2005(^{72}) - Ia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study type</td>
<td>Meta-analysis of RCT's</td>
</tr>
<tr>
<td>Participants</td>
<td>2313 patients</td>
</tr>
<tr>
<td>Inclusions</td>
<td>RCT's comparing oral anti-coagulants with control in patients with chronic non-valvular atrial fibrillation</td>
</tr>
<tr>
<td>Exclusions</td>
<td>Prior stroke or TIA, mitral stenosis or prosthetic cardiac valves</td>
</tr>
<tr>
<td>Intervention</td>
<td>Warfarin</td>
</tr>
<tr>
<td>Duration</td>
<td>1.5 years</td>
</tr>
<tr>
<td>Outcomes</td>
<td>All cause mortality</td>
</tr>
<tr>
<td>Control event rate</td>
<td>Not stated</td>
</tr>
<tr>
<td>Relative risk (mortality)</td>
<td>0.69 (OR)</td>
</tr>
<tr>
<td>95% CIs</td>
<td>Not stated</td>
</tr>
</tbody>
</table>

Data Analysis:

Control Event Rate

The trials included in the updated meta-analysis\(^{72}\) (Hart 2005) were all similar in patient characteristics with this indicator. Data from one trial (SPAF)\(^{73}\) was not available therefore was excluded from analysis. The control event rate was calculated from the remaining trials, weighting each trial by the number of patient years. (Refer to Table 32 – Appendix B)

Data Synthesis

There were no England prevalence figures for AF as the QOF 2006/7 has not yet been published. Data was taken from Scotland QOF 2006/7\(^{23}\) with a prevalence of 1.3%.

PHG (Population Health Gain) = \((1-\text{OR}) \times \text{CER} \times \text{prevalence} \times (1-\text{CER})\)

\[0.31 \times 0.0568 \times 1300 \times (1-0.0568)\]

**21.6 lives per 100,000 per year**

Caveats

Mortality gain was related to age, the trials included have typical average ages of 65+. Younger patients would have a lower control event rate and thus less health gain potential from anticoagulation.
4.1p Obesity (OB)

i OB 1: The practice can produce a register of patients aged 16 and over with a BMI greater than or equal to 30 in the previous 15 months.

This indicator was a disease register.

4.1q Learning Disabilities (LD)

i. LD 1: The practice can produce a register of patients with learning disabilities.

This indicator was a disease register.

4.1r Smoking Indicators

i. Smoking 1: The percentage of patients with any or any combination of the following conditions: coronary heart disease, stroke or TIA, hypertension, diabetes, COPD or asthma whose notes record smoking status in the previous 15 months. Except those who have never smoked where smoking status need only be recorded once since diagnosis.

This indicator is a record essential for Smoking 2.

ii. Smoking 2: The percentage of patients with any or any combination of the following conditions: CHD, stroke or TIA, hypertension, diabetes, COPD or asthma who smoke whose notes contain a record that smoking cessation advice or referral to a specialist service, where available has been offered within the previous 15 months.

Data Analysis:

RCTs were not possible as persons cannot be randomized to “stopping smoking”. Therefore data was handled using the technique previously described by McColl et al.\(^{(26)}\).
**Control Event Rate**

Control event rates available for CHD were 4.08% p.a. – Critchley et al 2003\(^{(33)}\) and the typical background population was 3% p.a – Doll et al 1994\(^{(57)}\). These were used as the higher and lower estimates of effectiveness.

**Data Synthesis**

At a control event rate of 3% p.a.

RR for smoking cessation in smokers was 0.70\(^{(57)}\)

Total prevalence for diseases: DM + CHD + Asthma + COPD + Stroke = 20.4% \(^{(23)}\)

Prevalence of smoking assumed to be that of the general population. 24% \(^{(34)}\)

Eligible population: 20400 x 24% = 4896

Effectiveness of intervention for smoking cessation = 0.20\(^{(35)}\)

\[
\text{PHG (Population Health Gain)} = \frac{N \times RRR \times \text{Effectiveness} \times \text{Pc}_T}{T}
\]

\[
4896 \times 0.30 \times 0.2 \times 0.03
\]

1

8.81 lives per 100,000 per year

At a CER of 4.08

\[
\text{PHG (Population Health Gain)} = \frac{N \times RRR \times \text{Effectiveness} \times \text{Pc}_T}{T}
\]

\[
4896 \times 0.30 \times 0.2 \times 0.0408
\]

1

11.99 lives per 100,000 per year

**Caveats**

Patients with co-morbidity in this indicator may have been expected to have a greater control event rate and therefore greater potential health gain, although no research in this area was identified to substantiate or refute this.
Summary of Results

Table 28: Results for the 2003 and 2006 GMS contract indicators by clinical domain

<table>
<thead>
<tr>
<th>Clinical domain</th>
<th>2003 contract: lives saved</th>
<th>2006 contract: lives saved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary heart disease</td>
<td>163.2</td>
<td>160.9</td>
</tr>
<tr>
<td>Diabetes</td>
<td>105.2</td>
<td>103.0</td>
</tr>
<tr>
<td>Hypertension</td>
<td>51.5</td>
<td>46.3</td>
</tr>
<tr>
<td>Stroke</td>
<td>49.7</td>
<td>48.7</td>
</tr>
<tr>
<td>Chronic obstructive airways disease</td>
<td>27.0</td>
<td>24.4</td>
</tr>
<tr>
<td>Asthma</td>
<td>8.8</td>
<td>-</td>
</tr>
<tr>
<td>Heart failure</td>
<td>11.9</td>
<td>11.9</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>-</td>
<td>21.6</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>-</td>
<td>12.8</td>
</tr>
<tr>
<td>Smoking</td>
<td>-</td>
<td>12.0</td>
</tr>
<tr>
<td><strong>Total including corrections for double counting (range)</strong></td>
<td><strong>415.0</strong> (406.1-423.9)</td>
<td><strong>439.3</strong> (427.2-455.2)</td>
</tr>
</tbody>
</table>
Highest level of evidence by indicator

Table 29: 2003 GMS contract lives saved by indicator

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Intervention</th>
<th>Lives saved</th>
<th>Lower Estimate</th>
<th>Upper Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD 12</td>
<td>Influenza immunization</td>
<td>62.8</td>
<td>62.8</td>
<td>62.8</td>
</tr>
<tr>
<td>DM 18</td>
<td>Influenza immunization</td>
<td>57.6</td>
<td>57.6</td>
<td>57.6</td>
</tr>
<tr>
<td>BP 5</td>
<td>Hypertension, BP,150/90 in past 9 months</td>
<td>46.3</td>
<td>46.3</td>
<td>46.3</td>
</tr>
<tr>
<td>CHD 10</td>
<td>Beta blocker</td>
<td>44.9</td>
<td>44.9</td>
<td>44.9</td>
</tr>
<tr>
<td>Stroke 10</td>
<td>Influenza immunization</td>
<td>26.2</td>
<td>26.2</td>
<td>26.2</td>
</tr>
<tr>
<td>CHD 9</td>
<td>Aspirin</td>
<td>25.2</td>
<td>21.6</td>
<td>25.2</td>
</tr>
<tr>
<td>DM 6</td>
<td>HbA1c&lt;7.4</td>
<td>24.0</td>
<td>17.8</td>
<td>24.0</td>
</tr>
<tr>
<td>COPD 8</td>
<td>Influenza immunization</td>
<td>24.4</td>
<td>24.4</td>
<td>24.4</td>
</tr>
<tr>
<td>Stroke 9</td>
<td>Antiplatelet/ anticoagulant</td>
<td>22.5</td>
<td>22.5</td>
<td>22.5</td>
</tr>
<tr>
<td>CHD 8</td>
<td>Cholesterol &lt; 5 mmol</td>
<td>16.1</td>
<td>16.1</td>
<td>23.6</td>
</tr>
<tr>
<td>DM 12</td>
<td>BP&lt;145/85</td>
<td>12.1</td>
<td>12.1</td>
<td>12.1</td>
</tr>
<tr>
<td>LVD 3</td>
<td>ACE/ARB</td>
<td>11.9</td>
<td>11.9</td>
<td>11.9</td>
</tr>
<tr>
<td>CHD 6</td>
<td>BP&lt;150/90</td>
<td>11.5</td>
<td>11.5</td>
<td>11.5</td>
</tr>
<tr>
<td>DM 15</td>
<td>Proteinuria/microalbuminuria on ACE</td>
<td>9.3</td>
<td>9.3</td>
<td>9.3</td>
</tr>
<tr>
<td>Asthma 5</td>
<td>Smoking cessation advice/referral</td>
<td>8.8</td>
<td>8.8</td>
<td>8.8</td>
</tr>
<tr>
<td>DM 7</td>
<td>HbA1c &lt;10</td>
<td>6.7</td>
<td>6.7</td>
<td>14.7</td>
</tr>
<tr>
<td>BP 3</td>
<td>Smoking cessation advice/referral</td>
<td>5.2</td>
<td>5.2</td>
<td>5.2</td>
</tr>
<tr>
<td>COPD 5</td>
<td>Smoking cessation advice/referral</td>
<td>2.6</td>
<td>2.6</td>
<td>2.6</td>
</tr>
<tr>
<td>CHD 4</td>
<td>Smoking cessation advice/referral</td>
<td>2.3</td>
<td>2.3</td>
<td>2.3</td>
</tr>
<tr>
<td>DM 4</td>
<td>Smoking cessation advice/referral</td>
<td>2.2</td>
<td>2.2</td>
<td>2.2</td>
</tr>
<tr>
<td>CHD 11</td>
<td>ACE/ARB</td>
<td>1.2</td>
<td>1.2</td>
<td>1.7</td>
</tr>
<tr>
<td>Stroke 4</td>
<td>Smoking cessation advice/referral</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td><strong>425.7</strong></td>
<td><strong>415.0</strong></td>
<td><strong>441.7</strong></td>
</tr>
<tr>
<td>Adjustment for double counting in hypertension, minus 3.1</td>
<td></td>
<td><strong>422.6</strong></td>
<td><strong>413.0</strong></td>
<td><strong>438.6</strong></td>
</tr>
<tr>
<td>Adjustment for double counting in diabetes, minus 6.7 to minus 14.7</td>
<td></td>
<td><strong>415.0</strong></td>
<td><strong>406.1</strong></td>
<td><strong>423.9</strong></td>
</tr>
</tbody>
</table>
### Table 30: 2006 GMS contract lives saved by indicator

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Intervention</th>
<th>Lives saved</th>
<th>Lower Estimate</th>
<th>Upper Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD 12</td>
<td>Influenza immunization</td>
<td>62.8</td>
<td>62.8</td>
<td>62.8</td>
</tr>
<tr>
<td>DM 18</td>
<td>Influenza immunization</td>
<td>57.6</td>
<td>57.6</td>
<td>57.6</td>
</tr>
<tr>
<td>BP 5</td>
<td>Hypertension, BP,150/90 in past 9 months</td>
<td>46.3</td>
<td>46.3</td>
<td>46.3</td>
</tr>
<tr>
<td>CHD 10</td>
<td>Beta blocker</td>
<td>44.9</td>
<td>44.9</td>
<td>44.9</td>
</tr>
<tr>
<td>Stroke 10</td>
<td>Influenza immunization</td>
<td>26.2</td>
<td>26.2</td>
<td>26.2</td>
</tr>
<tr>
<td>CHD 9</td>
<td>Aspirin</td>
<td>25.2</td>
<td>21.6</td>
<td>25.2</td>
</tr>
<tr>
<td>DM 20</td>
<td>HbA1c&lt;7.4</td>
<td>24.0</td>
<td>17.8</td>
<td>24.0</td>
</tr>
<tr>
<td>COPD 8</td>
<td>Influenza immunization</td>
<td>24.4</td>
<td>24.4</td>
<td>24.4</td>
</tr>
<tr>
<td>Stroke 12</td>
<td>Antiplatelet/ anticoagulant</td>
<td>22.5</td>
<td>22.5</td>
<td>22.5</td>
</tr>
<tr>
<td>AF 3</td>
<td>Anticoagulation</td>
<td>21.6</td>
<td>21.6</td>
<td>21.6</td>
</tr>
<tr>
<td>CHD 8</td>
<td>Cholesterol &lt; 5 mmol</td>
<td>16.1</td>
<td>16.1</td>
<td>23.6</td>
</tr>
<tr>
<td>CKD3/CKD4</td>
<td>BP&lt;140/85</td>
<td>12.8</td>
<td>12.8</td>
<td>19.8</td>
</tr>
<tr>
<td>DM 12</td>
<td>BP&lt;145/85</td>
<td>12.1</td>
<td>12.1</td>
<td>12.1</td>
</tr>
<tr>
<td>Smoking 2</td>
<td>Smoking cessation advice/referral</td>
<td>12.0</td>
<td>8.8</td>
<td>12.0</td>
</tr>
<tr>
<td>HF3</td>
<td>ACE/ARB</td>
<td>11.9</td>
<td>11.9</td>
<td>11.9</td>
</tr>
<tr>
<td>CHD 6</td>
<td>BP&lt;150/90</td>
<td>11.5</td>
<td>11.5</td>
<td>11.5</td>
</tr>
<tr>
<td>DM 15</td>
<td>Proteinuria/microalbuminuria on ACE</td>
<td>9.3</td>
<td>9.3</td>
<td>9.3</td>
</tr>
<tr>
<td>DM 7</td>
<td>HbA1c &lt;10</td>
<td>6.7</td>
<td>6.7</td>
<td>14.7</td>
</tr>
<tr>
<td>CHD 11</td>
<td>ACE/ARB</td>
<td>1.2</td>
<td>1.2</td>
<td>1.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td><strong>450.0</strong></td>
<td><strong>437.0</strong></td>
<td><strong>473.0</strong></td>
</tr>
<tr>
<td>Adjustment for double counting in hypertension, minus 3.1</td>
<td></td>
<td><strong>446.9</strong></td>
<td><strong>433.9</strong></td>
<td><strong>470.7</strong></td>
</tr>
<tr>
<td>Adjustment for double counting in diabetes, minus 6.7 to minus 14.7</td>
<td></td>
<td><strong>439.3</strong></td>
<td><strong>427.2</strong></td>
<td><strong>455.2</strong></td>
</tr>
</tbody>
</table>
Evidence for lives saved was found for 19 indicators in the 2006 version of the GMS contract. A further 23 indicators were indirectly linked to a reduction in mortality. In the 2003 contract there was potential for 415.0 lives saved in one year (405.4-423.1) aggregated across all clinical domains. In the 2006 contract this increased to a potential for 439.3 lives saved (427.2-455.2) aggregated across all clinical domains. The number of potential lives saved for these indicators ranged from 1.0 to 62.8 per 100,000 people per year. With a UK population of 59,835,000 in 2004, this equated to 248,315 lives potentially saved in the 2003 GMS contract clinical indicators, rising to 262,855 potential lives saved in the 2006 GMS contract. However, the actual number of lives saved would be lower for two reasons. Firstly, there was significant baseline activity in primary care before implementation of the new GMS contract. Secondly, less than 100% of the target population would have received the intervention due to exclusions and less than full implementation of the contract.

In the 2003 GMS contract the potential lives saved per year by domain was 163.2 lives in coronary heart disease, 105.2 lives in diabetes, 51.5 lives in hypertension, 49.7 lives in stroke, 27.0 lives in chronic obstructive pulmonary disease, 11.9 lives in left ventricular dysfunction and 8.8 lives in asthma.

In the 2006 revised GMS contract the potential lives saved per year by domain were 160.9 lives in coronary heart disease, 103 lives in diabetes, 48.7 lives in stroke, 46.3 lives in hypertension, 24.4 lives in chronic obstructive pulmonary disease, 19.2 lives in atrial fibrillation, 12.8 lives in chronic kidney disease, 12 lives in smoking cessation and 11.9 lives in heart failure. The greatest potential for health gain was in 2 primary prevention interventions, influenza immunization and control of hypertension. Influenza immunization alone accounts for 38% of potential lives saved in the 2006 contract.

There were 3 indicators for which the evidence base was less than expected. A systematic review found no evidence of benefit for cholesterol lowering after a stroke - Stroke 8. No evidence was found in a meta-analysis for controlling blood pressure after a stroke - Stroke 6, although there was evidence for a reduction in associated vascular morbidity. Only one RCT was identified examining the effect of cholesterol in diabetes (DM17) by Colhoun et al. This study demonstrated a
reduction in cardiovascular events but not in all cause mortality, although this study was terminated prematurely due to improvements in morbidity.
Section 5 Discussion

The clinical indicators in the new GMS contract have potential for significant health gain. In the original form of the contract (2003) there was potential for 415.0 lives being saved per 100,000 populations in one year. In the revised contract (2006) this raised to 439.3, an increase of 24.3. This difference in the potential to save lives from the original GMS contract to the revised GMS contract 2006/07 was largely due to the inclusion of the Kidney Disease and the Atrial Fibrillation indicators. The greatest number of lives saved by domain was in the CHD and the DM indicators, accounting for more than half of all lives saved across all indicators. These diseases were more common and have a number of clinical interventions that are effective. By indicator, influenza immunization carries the greatest potential for lives saved, followed by treatment for primary prevention for hypertension. Smoking had less health gain than expected by the authors. There are at least two reasons for this: firstly, smoking cessation in the GMS contract had been targeted only at patients with other clinical conditions - thus the large pool of patients who would benefit from primary prevention was not included in the new GMS contract indicators. Secondly, patients who had suffered a stroke, heart attack or diabetes had a low prevalence of smoking in the contract data - presumably as many would have already stopped smoking. Smoking cessation for primary prevention would have significantly increased the potential for lives saved if it were included in the new GMS contract.

Lives saved were only one measure of primary care quality, however it was an important one and currently is more widely available than other measures such as QALYs. Health gain represented a possible additional criterion to be used when allocating points to indicators and conditions in future revisions of the QOF.

5.1 Strengths of the Study

Our study reviewed 4 robust sources of evidence of which three are regularly updated online. These sources were well known sources of evidence. The evidence was independently searched by two researchers. (RF and SP) The independent search increased the robustness of the evidence sought. The included studies were then quality assessed using the quality grading scale designed by AHCPR[10] which has
been adopted by the Cochrane Library\(^9\). Wherever possible we selected the highest level of evidence. Secondly, the measure of health gain chosen was all cause mortality. This measure of health gain was widely available and was a measure of quality also used by the World Health Organization and the Department of Health in performance indicators. Mortality was a relevant and important outcome for many interventions in the new GMS contract.

5.2 Limitations and weaknesses

The limitations specific to each individual indicator have been mentioned in each individual caveats section. There were other limitations general to all indicators in the new GMS contract. Firstly the measure of quality used (lives saved in one year) was narrow. There were many aspects of primary care quality which cannot be captured by lives saved, for example access to services and palliative care. Unfortunately there was as yet sufficient evidence in terms of QALYs across the clinical spectrum covered by the new GMS contract. Secondly trial participants often did not represent the characteristic of patients who were present in general practice. This was most marked in the evidence cited for CKD3, where patients from the evidence base included some patients with other chronic diseases. Thirdly the level of evidence for some indicators was less than grade 1a. For example evidence for the effectiveness for influenza immunization was based on the evidence from cohort studies, which was subject to selection bias. However it would not have been possible to design RCTs effectively for interventions such as influenza immunization, due to ethical issues of the existing evidence and the beneficial effect of herd immunity on patients who were not immunized. There was a lack of research into important areas for some of the indicators, such as cholesterol and diabetes. We identified only one study for this indicator which was terminated early and therefore the study did not have appropriate power to detect a mortality difference if one existed. Finally, many diseases are undergoing a change in prevalence, with for example coronary heart disease falling and diabetes increasing. Changes in disease prevalence would alter the potential population health gain for particular indicators and the nature and pathogenicity of the diseases themselves may change over time. For example some of the increase in prevalence of type 2 diabetes may be due to early diagnosis (and therefore a lower baseline risk in terms of morbidity and mortality).
5.3 Further Implications for Research

Further research is needed in areas of the new GMS contract indicators to complete the evidence base for lives saved. This specific study addressed only the indicators mentioned in the GMS contract, and a systematic approach to evaluating evidence for lives saved across the whole spectrum of primary care will give a more complete picture of potential for lives saved. More research is needed across the spectrum of primary care to evaluate other measures of health gain, such as QALYs. This will enable the development of GP contract to include the new indicators to maximize health gain. Quality can be used as one of a number of factors (including baseline performance and difficulty of clinical tasks) in determining the ideal financial incentive for each indicator.

5.4 Conclusions

Full implementation of the original GMS contract (2003) can be expected to result in 415.0 lives being saved per 100,000 populations per year. Full implementation of the revision of the GMS contract (2006) increases the potential lives saved to 439.3, which equates to 262855 lives in one year in the UK. Lives saved were a direct outcome of 19 of the 2006 GMS contract indicators, and an indirect outcome of a further 23 indicators. Lives saved were only one measure of primary care quality; however they were an important one and currently the most widely available than other measures such as QALYs. Health gain represented a possible additional criterion to be used when allocating points to indicators and conditions in future revisions of the QOF. A limitation of this study was that evidence was of different strength for different indicators Influenza was one of the highest areas for potential health gain, although the evidence was based on cohort studies. Further research includes developing other measures of quality such as QALYs for as many clinical indicators as possible to inform development and weighting of both current and new indicators in future revisions of the GMS contract.
References


22. Committee on Redesigning Health Insurance Performance Measures, Payments, and Performance Improvement Programs. Rewarding Provider Performance. 2007: Aligning Incentives in Medicare (Pathways to Quality Health


105. Law M, Wald NJ, Morris J. 2003; Health Technology Assessment Programme, National Co-ordinating Centre for HTA (Great Britain), Great Britain. Standing Group on Health Technology. Lowering blood pressure to prevent myocardial infarction and stroke: a new preventive strategy. Tunbridge Wells: Gray on behalf of the NCCHTA.


7.1 Detailed Search Strategy

The GMS contract can be divided into clinical indicators with subgroups for each indicator. General and specific search terms were applied to each clinical indicator. Applying these strategies the highest level of evidence was identified. The excluded studies are listed in Appendix B.

Common Search Strategy

There were 4 databases to which the common search strategy was applied for each individual clinical indicator. They are mentioned as follows:

GMS Contract (Supporting Documentation)

The contract was examined at each individual indicator level to identify the papers that were cited and that contained evidence of lives saved that were specific to the patient type as defined by the particular indicator. If the identified paper cited further references then these were also followed-up.

Clinical Evidence

This database was searched in 3 ways:

Section: Domain: Indicator: Intervention
Full Review List: Domain: Indicator: Intervention
Free Text Search: Domain: Indicator: Intervention

Cochrane

This database was searched in 3 ways:

Topic Index: Domain: Indicator: Intervention
A-Z Index: Domain: Indicator
Advanced Search: Indicator: Intervention

NICE

This database was searched in 5 ways as follows:

Our guidance: Type: Clinical Guidelines: Published
Our guidance: Type: Public Health Intervention: Published
Our guidance: Type: Technology Appraisals: Published
Our guidance: Topic: Clinical Domain: Intervention
Free Text Search: Clinical Domain: Intervention: Indicator

Common Search Terms

Common Search Terms for Cardiovascular domain

- Ischaemic Heart Disease
- CHD
- CVD
- Myocardial infarction
- Heart disease
- Death
- Mortality
- Survival

Common Search Terms for Heart Failure domain

- Heart Failure
- Left Ventricular Failure
- Left Ventricular Dysfunction
- Death
- Mortality
• Survival

Common Search Terms for Stroke domain

• Stroke
• TIA
• Transient ischaemic attack
• Death
• Mortality
• Survival

Common Search Terms for Hypertension domain

• BP
• Hypertension
• High Blood Pressure
• Death
• Mortality
• Survival

Common Search Terms for Stroke domain

• Stroke
• TIA
• Transient ischaemic attack
• Death
• Mortality
• Survival

Common Search Terms for Diabetes domain

• Diabetes
• Type I diabetes
• Death
• Mortality
• Survival

Common Search Terms for COPD domain

• COPD
• Chronic bronchitis
• Chronic obstructive pulmonary disease
• Airways
• Death
• Mortality
• Survival

Common Search Terms for Epilepsy domain

• Epilepsy
• Seizures
• Tonic/Clonic
• Death
• Mortality
• Survival

Common Search Terms for Thyroid domain

• Thyroid
• Hypothyroidism
• Myxoedema
• Death
• Mortality
• Survival

Common Search Terms for Cancer domain
• Cancer
• Malignancy
• Neoplasia
• Death
• Mortality
• Survival

Common Search Terms for Mental Health domain

• Mental Health
• Depression
• Schizophrenia
• Bipolar
• Death
• Mortality
• Survival

Common Search Terms for Asthma domain

• Asthma
• Airways
• Death
• Mortality
• Survival

Common Search Terms for Palliative Care

• Palliative
• Terminal
• Death
• Mortality
• Survival
Common Search Terms for Dementia

- Dementia
- Alzheimer’s
- Death
- Mortality
- Survival

Common Search Terms for Depression

- Depression
- Bipolar
- Death
- Mortality
- Survival

Common Search Terms for Chronic Kidney Disease

- Chronic kidney disease
- Renal impairment/failure
- Death
- Mortality
- Survival

Common Search Terms for Atrial Fibrillation

- Atrial fibrillation
- Death
- Mortality
- Survival
• Obesity
• Death
• Mortality
• Survival

Common Search Terms for Learning Difficulties

• Learning difficulties
• Educational delay
• Death
• Mortality
• Survival

Common Search Terms for Smoking

• Smoking
• Tobacco
• Death
• Mortality
• Survival

Specific Search Terms

The specific search terms were used in addition to the common search terms used for each indicator.

Specific Search Terms for Cardiovascular Disease 8

• Cardiovascular disease and statins
• Secondary prevention of ischaemic cardiac events

Specific Search Terms for Cardiovascular Disease 9
• Antiplatelet or anticoagulant therapy and cardiovascular disease

Specific Search Terms for Cardiovascular Disease 10
• Cardiovascular disease and beta-blockers

Specific Search Terms for Cardiovascular Disease 11
• Heart Failure
• Angiotensin converting enzyme (ACE) inhibitors

Specific Search Terms for Cardiovascular Disease 12
• Influenza
• Influenza vaccination

Specific Search Terms for Heart Failure 3
• Angiotensin converting enzyme (ACE) inhibitors

Specific Search Terms for Stroke/TIA 12
• Stroke Prevention
• Antiplatelet therapy

Specific Search Terms for Stroke/TIA 10
• Influenza
• Infectious Diseases

Specific Search Terms for Hypertension 5

No additional specific search terms were used.
Specific Search Terms for Diabetes Mellitus 15

- Diabetic nephropathy
- Angiotensin converting enzyme inhibitors
- Renal disease
- Metabolic and endocrine disorders

Specific Search Terms for Diabetes Mellitus 18

- Influenza
- Influenza vaccination

Specific Search Terms for Diabetes Mellitus 20

- Hb1AC
- Diabetes mellitus and related disorders
- Metabolic and endocrine disorders

Specific Search Terms for Diabetes Mellitus 21/8

- Diabetes mellitus and related disorders
- Metabolic and endocrine disorders
- Retinopathy

Specific Search Terms for COPD 8

- Influenza
- Influenza Vaccination
8.1 Control Event Rates Table

8.1a CHD 10: The percentage of patients with coronary heart disease who are currently treated with a beta blocker.

Table 31: Control event rates for CHD 10

<table>
<thead>
<tr>
<th>Study</th>
<th>% with Heart Failure</th>
<th>Control Deaths</th>
<th>Total Control</th>
<th>Duration in yrs</th>
<th>Annual Event Rate</th>
<th>Patient Years</th>
<th>Weight</th>
<th>Annual Event x Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>BHAT</td>
<td>9.2</td>
<td>188</td>
<td>1921</td>
<td>2.08</td>
<td>0.047051</td>
<td>3996</td>
<td>0.6035342</td>
<td>0.02839689</td>
</tr>
<tr>
<td>EIS</td>
<td>7.7</td>
<td>45</td>
<td>883</td>
<td>1</td>
<td>0.050963</td>
<td>883</td>
<td>0.1333635</td>
<td>0.00679661</td>
</tr>
<tr>
<td>Hjalmanson</td>
<td>10</td>
<td>62</td>
<td>697</td>
<td>2</td>
<td>0.04447</td>
<td>1394</td>
<td>0.2105422</td>
<td>0.00936281</td>
</tr>
<tr>
<td>Salathia</td>
<td>10</td>
<td>52</td>
<td>348</td>
<td>1</td>
<td>0.149425</td>
<td>348</td>
<td>0.0525600</td>
<td>0.00785378</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td>3831</td>
<td>6621</td>
<td>0.0524</td>
<td></td>
<td>0.0524</td>
<td></td>
</tr>
</tbody>
</table>

Annual weighted control event rate = 5.2%

8.1b AF 3: The percentage of patients with atrial fibrillation who are currently treated with anti-coagulation drug therapy or an anti-platelet therapy.

Table 32: Control event rates for AF3

<table>
<thead>
<tr>
<th>Study</th>
<th>Duration</th>
<th>Control Mortality</th>
<th>Total Control</th>
<th>CER</th>
<th>CER/Year</th>
<th>Life Years</th>
<th>Weight</th>
<th>CER x Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>AFASAK 1</td>
<td>1.2</td>
<td>28</td>
<td>336</td>
<td>0.083333</td>
<td>0.069444</td>
<td>403.2</td>
<td>0.25167</td>
<td>0.01747</td>
</tr>
<tr>
<td>BAATAF 2.2</td>
<td>26</td>
<td>208</td>
<td>0.125000</td>
<td>0.056818</td>
<td>457.6</td>
<td>0.28562</td>
<td>0.01622</td>
<td>0.00499</td>
</tr>
<tr>
<td>CAFA 1.3</td>
<td>08</td>
<td>191</td>
<td>0.014885</td>
<td>0.032219</td>
<td>248.3</td>
<td>0.15498</td>
<td>0.00499</td>
<td>0.0004993</td>
</tr>
<tr>
<td>SPAF1 N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>SPINAF 1.7</td>
<td>29</td>
<td>290</td>
<td>0.100000</td>
<td>0.058824</td>
<td>493.0</td>
<td>0.30772</td>
<td>0.01810</td>
<td>0.0018101</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td>1025</td>
<td>1602.1</td>
<td>1</td>
<td>0.05680</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Average CER = 5.68%
9.1 Summary of Excluded Studies

**CHD 8:** The percentage of patients with CHD whose last measured total cholesterol (measured in the last 15 months) is 5 mmol/l or less.

**LaRosa et al (1999)** (76)
This study includes a significant number of patients from a different population.

**Bucher et al (1999)** (77)
This study includes a significant number of patients from a different population.

**LIPID Study (1998)** (40)
This is a randomised controlled trial, a higher level of evidence is given preference.

**Cannon et al (2004)** (78)
The study does not have all cause mortality as the endpoint.

**MRC/BHF Heart Protection Study (2002)** (79)
This is a randomised controlled trial, a higher level of evidence is given preference.

**Shepherd et al (2002)** (80)
This is a randomised controlled trial, a higher level of evidence is given preference.

**Gould et al (1998)** (81)
This study does not directly compare treatment.

**Baigent et al (2005)** (82)
This is a randomised controlled trial, a higher level of evidence is given preference.

**Durazzo et al (2004)** (83)
This is a randomised controlled trial, a higher level of evidence is given preference.
Weisman et al (2002)\(^{(84)}\)

The study includes high risk patients of other vascular disease like stroke and TIA.

CHD 9: The percentage of patients with CHD with a record in the last 5 months that aspirin, an alternative anti-platelet therapy, or an anti-coagulant is being taken (unless a contraindication or side effects are recorded)

Antiplatelet Trialists Collaboration (1994)\(^{(85)}\)

This study is superseded by Antithrombotic Trialists Collaboration, 2002.\(^{(41)}\)

CAPRIE (1996)\(^{(86)}\)

The study does not compare treatment versus placebo.

MRC General Practice Research Framework (1998)\(^{(87)}\)

This study includes a significant number of patients from a different population.

CHD 10: The percentage of patients with CHD who are currently treated with a beta blocker (unless a contraindication or side effects are recorded)

Domanski et al (2003)\(^{(88)}\)

The study studies heart failure.

CHD 11: The percentage of patients with history of myocardial infarction (diagnosed after 1 April 2003) who are currently treated with ACE inhibitors.

Psaty et al (2003)\(^{(89)}\)

The study includes hypertensive patients.

Turnbull et al (2003)\(^{(90)}\)

The study compares the treatment group with different treatments and not with placebo.

CHD 12: The percentage of patients with CHD who have a record of influenza vaccination in the preceding 1 September to 31 March
Vu et al (2002)(91)
The target population is different to that in the indicator.

Voordouw et al (2003)(92)
The target population is different to that in the indicator

Keller et al (2004)(93)
This is a protocol.

LVD/HF 3: The percentage of patients with a current diagnosis of heart failure due to LVD who are currently treated with an ACE inhibitor or Angiotensin Receptor Blocker who can tolerate therapy and for whom there is no contradiction.

Fox et al (2003)(94)
The study population included all causes of coronary heart disease and was not limited to left ventricular dysfunction or heart failure.

Stroke 6: The percentage of patients with a history of TIA or stroke in whom the last blood pressure reading (measured in last 15 months) is 150/90 or less.

The intervention was thrombolysis for acute stroke.

Stroke 8: The percentage of patients with TIA or stroke whose last measured total cholesterol (measured in last 15 months) is 5 mmol/l or less

Amarenco et al (2004)(95)
The study includes primary prevention studies.

Di Mascio et al (2000)(96)
The study did not report all cause mortality as the endpoint.
**Stroke 9:** The percentage of patients with a stroke shown to be non-haemorrhagic, or a history of TIA, who have a record that aspirin, an alternative anti-platelet therapy, or an anti-coagulant is being taken.

*Antithrombotic Trialists Collaboration (1994)*[^85]
This study is superseded by Antithrombotic Trialists Collaboration, 2002.[^41]

**Stroke 10:** The percentage of patients with TIA or stroke who have had influenza immunisation in the preceding 1 September to 31 March.

*Nichol et al (2003)*[^97]
The study does not report all cause mortality as the endpoint.

*Lávallee et al (2002)*[^98]
The study does not report all cause mortality as the endpoint.

**Stroke 12:** The percentage of patients with a stroke shown to be non-haemorrhagic or a history of TIA who have a record that antiplatelet agent (aspirin, clopidogrel, dipyridamole or a combination) or an anti-coagulant is being taken (unless a contraindication or side-effects are recorded

*Antiplatelet Trialists Collaboration (1994)*[^85]
This study is superseded by Antithrombotic Trialists Collaboration, 2002.[^41]

*IST Collaborative Group (1997)*[^99]
This is a randomised controlled trial, a higher level of evidence is given preference.

*Bhatt et al (2000)*[^100]
This study compares different treatments not with placebo.

*Hart et al (1999)*[^101]
This is a randomised controlled trial, a higher level of evidence is given preference.
BP 3: The percentage of patients with hypertension who smoke, whose notes contain a record that smoking cessation advice or referral to specialist service, if available, has been offered atleast once.

Lancaster et al (2000)\(^{(102)}\)

The study does not report all cause mortality as the endpoint.

BP 4: The percentage of patients with hypertension in whom there is a record of the blood pressure in the past 9 months.

Ramsay et al (1999)\(^{(103)}\)

This is a review article.

Alderman (1993)\(^{(104)}\)

The study does not report all cause mortality as the endpoint.

BP 5: The percentage of patients with hypertension in whom the last blood pressure (measured in last 9 months) is 150/90 or less.

Law et al (2003)\(^{(105)}\)

The study does not report all cause mortality as the endpoint.

Staessen et al (2000)\(^{(60)}\)

The study focuses on isolated systolic hypertension only.

DM 2: The percentage of patients with diabetes whose notes record BMI in the previous 15 months.

Norris et al (2005)\(^{(106)}\)

The study does not report all cause mortality as the endpoint.

DM 6/20: The percentage of patients with diabetes in whom the last HbA1c is 7.5 or less (or equivalent test/reference range depending on local laboratory) in the previous 15 months.
**Diabetes Control and Complications Trial Research Group (1993)**\(^{(107)}\)

The study does not report all cause mortality as the endpoint.

**DM 15:** The percentage of patients with diabetes with proteinuria or microalbuminuria who are treated with ACE inhibitors (or A2 antagonists).

**Mathiesen et al (1991)**\(^{(108)}\)

The study includes Type I diabetes.

**Lewis et al (1993)**\(^{(109)}\)

The study includes Type I diabetes.

**EUCLID Study Group (1997)**\(^{(110)}\)

This is a randomised controlled trial, a higher level of evidence is given preference.

**Diabetic Nephropathy Trialists Group (2001)**\(^{(111)}\)

The study includes Type I diabetes.

**DM 18:** The percentage of patients with diabetes who have had influenza immunization in the preceding 1 September to 31 March.

**Jefferson et al (2005)**\(^{(112)}\)

The target population includes only elderly individuals.

**DM 21:** The percentage of patients with diabetes who have a record of retinal screening in the previous 15 months.

**Norris et al (2005)**\(^{(113)}\)

This is a protocol.

**COPD 5:** The percentage of patients with COPD in whom there is a record of smoking status in the previous 15 months, except those who have never smoked where smoking status need be recorded only once since diagnosis.
Connett et al (1993)\textsuperscript{(114)}
This is a trial protocol.

Anthonisen et al (2002)\textsuperscript{(115)}
The study includes other respiratory treatments as intervention.

Tashkin et al (2001)\textsuperscript{(116)}
This is a short term trial of smoking cessation and the study does not report all cause mortality as the endpoint.

COPD 8: The percentage of patients with COPD who have had influenza immunisation in the preceding 1 September to 31 March.

Poole et al (2006)\textsuperscript{(117)}
The study includes patients with other pathologies and the patient number is underpowered for mortality.

MH 5: The percentage of patients on lithium therapy with a record of lithium levels in the therapeutic range within the previous 6 months.

Burgess et al (2001)\textsuperscript{(118)}
The study includes a significant number of patients from a different population.

DEP 1: The percentage of patients on the diabetes register and/or the CHD register for whom case finding for depression has been undertaken on one occasion during the previous 15 months using two standard screening questions.

Barth et al (2004)\textsuperscript{(119)}
This is an observational study without intervention.

CKD 4: The percentage of patients on the CKD register who are treated with an angiotensin converting enzyme inhibitor (ACE-I) or angiotensin receptor blocker (ARB) (Unless a contraindication or side-effects are recorded).
Guidi et al (2001)\textsuperscript{(120)}
This study is a protocol.

Brenner et al (2001)\textsuperscript{(121)}
The study does not report all cause mortality as the endpoint.

Ruggenenti et al (1999)\textsuperscript{(122)}
The study does not report all cause mortality as the endpoint.

AF 3: The percentage of patients with atrial fibrillation who are currently treated with anti-coagulant drug therapy or an anti-platelet therapy.

Van Walraven et al (2002)\textsuperscript{(123)}
The study compares with different treatment not with placebo.