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...[To] develop, implement and monitor evidence based programmes to address the impact of tobacco use, diet and physical activity, targeted at the most disadvantaged communities in Wales.

National Service Framework

National Assembly for Wales. *Tackling CHD in Wales: Implementing Through Evidence*. Cardiff: National Assembly for Wales, July 2001

The average annual European age-standardised MI mortality rates for all ages by Welsh unitary authority area, over the six-year period 1990-1995 …show a considerable difference between unitary authority areas with the age-standardised mortality rate varying from 87 to 177. The Standard Morbidity Rate for Wales as a whole is 108. [paragraph 2.8]

What are the current mortality rates for Wales?

The *statements*

The *evidence*

1.1 Mortality rates for coronary heart disease

1.1a. In 1999 there were 7.7 thousand deaths from **ischaemic heart disease** in Wales, compared to 8.7 thousand deaths in 1995 and 9.5 thousand in 1991[i]. The figures for all **heart diseases** (in thousands) were 9.0 in 2001, 9.1 in 2000, 9.9 in 1999 and 9.7 in 1998[ii]. In 1998 20.8% of adults had ever been treated for heart disease (19.8% males and 21.7% females)[ii].

The European standardised death rates in Wales from ischaemic heart disease were reduced from 163.4 in 1995 to 133.0 in 1999[iii]. The age-adjusted mortality rates from ischaemic heart disease per 100,000 population in 1998 were 230 in Wales compared to 218 in England and 266 in Scotland[iv].

i. *Health Statistics Wales 2003*. Chapter 1 – population and vital statistics. Cardiff: National Assembly for Wales, 2003 **http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/health/2002/hsw2003/hsw2003-ch1/hsw2003-ch1.htm** [accessed 17.12.03] (Type IV evidence – statistics)

ii. *Key Health Statistics for Wales 2002*. Cardiff: National Assembly for Wales, 2003 **http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/health/2003/key-health-stats-02-e.pdf** [accessed 17.12.03] (Type IV evidence – statistics)

iii. *Health Statistics Wales 2001*. Chapter 1 – Population and vital statistics. Cardiff: National Assembly for Wales, March 2002 **http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/health/2002/hsw2001/hsw2001-ch1/hsw2001-ch1.htm** [accessed 17.12.03] (Type IV evidence – statistics)

iv. *Comparative Indicators 2000. Wales England Scotland*. Cardiff: National Assembly for Wales, November 2001. **http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/compendia/2001/Comparative_Indicators_2000.pdf** [accessed 17.12.03] (Type IV evidence – statistics)

National Service Framework

National Assembly for Wales. *Tackling CHD in Wales: Implementing Through Evidence.* Cardiff: National Assembly for Wales, July 2001

There are high numbers of people who are overweight and who smoke in Wales. Conversely, the consumption of fruit and vegetables and the taking of exercise are low. [paragraph 4.2]

What is the latest epidemiological information regarding the levels of obesity, fruit and vegetable consumption, smoking and exercise in the population?

The *statements* | The *evidence*

1.2 Lifestyle measures in Wales

Obesity

1.2a. The percentage of the adult Welsh population (aged 18-64) who are **overweight or obese** rose from 43.2% in 1985 (44.4% in males & 42.0% in females) to 52.5% (53.2% & 51.2%) in 1996[i].

Definition: A person is classed as **overweight** if their Body Mass Index (BMI) (weight in kg)/(height in m) is ≥ 25 for men and ≥ 24 for women. They are classed as **obese** if their BMI is over 30.

Fieldwork for the new Welsh Health Survey starts in October 2003[ii].

i. *Health Statistics Wales 2001*. Chapter 2 – Lifestyle. Cardiff: National Assembly for Wales, March 2002 **http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/health/2002/hsw2001/hsw2001-ch2/hsw2001-ch2.htm** [accessed 17.12.03]
(Type IV evidence – statistics)

ii. Personal communication, Health Promotion Division, Welsh Assembly Government **http://www.hpw.wales.gov.uk/** [accessed 17.12.03]

1.2b. The percentage of **overweight children**, aged 4-11, increased between 1984 and 1994 from 5.4% to 9.0% in English boys (an increase of 3.6%, 95% CI 2.3%-5.0%) and from 6.4% to 10.0% in Scottish boys (3.6%, 1.9%-5.4%). Values for girls were 9.3% to 13.5% (4.1%, 2.4%-5.9%) and 10.4% to 15.8% (5.4%, 3.2%-7.6%)[i].

The most recent estimates for England are that 8.5% of six-year olds and 15% of 15-year olds are obese[ii].

The international HBSC (Health Behaviour in School-aged Children) report, due to be published by the World Health Organisation in February 2004, will present BMI data for 35 countries including Wales[iii] and pre-publication data suggest that Wales will be in the top-third of countries[iv].

i. Chinn S, Rona RJ. Prevalence and trends in overweight and obesity in three cross sectional studies of British children, 1974-94. *British Medical Journal* 2001; **322(7277)**: 24-26 **http://bmj.bmjjournals.com/cgi/content/full/322/7277/24** [accessed 17.12.03]
(Type IV evidence – results from three cross-sectional surveys measuring prevalence and change in prevalence of overweight and obesity, as defined by the national obesity task force, in 1974, 1984 and 1994 for each sex and for England and Scotland. Participants were 10,414 boys and 9,737 girls in England and 5,385 boys and 5,219 girls in Scotland aged 4-11 years)

ii. *Annual Report of the Chief Medical Officer 2002. 5. Obesity. Defusing the Health Time Bomb*. London: Department of Health, 2003 **http://www.doh.gov.uk/cmo/annualreport2002/obesity.htm** [accessed 17.12.03]
(Type IV evidence – statistics)

iii. **http://www.hbsc.org** [accessed 17.12.03]

iv. Personal communication, Health Promotion Division, Welsh Assembly Government **http://www.hpw.wales.gov.uk/** [accessed 17.12.03]

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1.2c. **Socio-economic** gradients in cholesterol intake in boys and physical inactivity and total energy intake in girls were present at 12 and 15 years of age. Differences in fat and fruit intake and smoking behaviour (in boys and girls) became established at the age of 15 years, with the least favourable levels occurring in subjects in the manual socio-economic group[i].

i. Van Lenthe FJ, Boreham CA, Twisk JW, Strain JJ, Savage JM, Smith GD. Socio-economic position and coronary heart disease risk factors in youth. Findings from the Young Hearts Project in Northern Ireland. *European Journal of Public Health* 2001; **11(1)**: 43-50
(Type IV evidence – school based prospective study of 251 boys and 258 girls examined at age 12 (78% response) in 1989/1990 and age 15 (87% response) in 1992/1993)

Fruit and vegetable consumption

1.2d. The consumption of **vegetables or salad** (on six or seven days per week) by adults aged 18-64 in Wales has increased from 24.7% (20.3% in males & 29.2% in females) in 1985 to 33.2% (26.9% & 39.6%) in 1996 but levels of consumption are still low compared to other parts of the UK[i].

The consumption of **fruit** (on six or seven days per week) by adults aged 18-64 in Wales has increased from 34.6% (27.0% in males & 42.1% in females) in 1985 to 46.4% (39.2% & 53.8%) in 1996[i].

i. *Health Statistics Wales 2001*. Chapter 2 – Lifestyle. Cardiff: National Assembly for Wales, March 2002
http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/health/2002/hsw2001/hsw2001-ch2/hsw2001-ch2.htm [accessed 17.12.03]
(Type IV evidence – statistics)

Smoking

1.2e. The number of **adults** aged 18-64 in Wales who **smoke** at least occasionally fell from 37.0% (40.8% in males & 33.2% in females) in 1985 to 31.5% (33.5% & 29.4%) in 1996. The decrease among men has been more marked than in women[i].

There is a **socio-economic gradient**. Cigarette smoking is more prevalent among manual social groups than among non-manual groups. In 1998 in Great Britain, 36% of men and 31% of women in manual groups smoked compared to 21% of men and women in non-manual groups[ii]. In the updated statistics for 2003 it is estimated that 35% of men and 30% of women in manual occupations smoke compared with 23% of men and 22% of women in non-manual occupations[iii]. In the updated statistics for 2003 it is estimated that 35% of men and 30% of women in manual occupations smoke compared with 23% of men and 22% of women in non-manual occupations[iii].

i. *Health Statistics Wales 2001*. Chapter 2 – Lifestyle. Cardiff: National Assembly for Wales, March 2002
http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/health/2002/hsw2001/hsw2001-ch2/hsw2001-ch2.htm [accessed 17.12.03]
(Type IV evidence – statistics)

ii. *British Heart Foundation Statistics Database 2002. Annual Compendium: 2002 edition*. Smoking.
http://www.heartstats.org/uploads/documents/2003%20Smoking%20chapter.pdf [accessed 17.12.03]
(Type IV evidence – statistics)

iii. British Heart Foundation. *Coronary Heart Disease Statistics in British Heart Foundation Statistics Database 2003*. London: British Heart Foundation, 2003.
http://www.bhf.org.uk/professionals/uploaded/bhf%20heartstats%202003%20-%204-page.pdf
[accessed 19.01.04]

The *statements*

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1.2f. The proportion of children in Wales who **smoke regularly** rose between 1986 and 2000, particularly for older girls. In 1986, 20% of girls and 16% of boys aged 15-16 reported smoking weekly; by 2000 these proportions were 29% and 20%. However it can be seen from the most recent surveys that the prevalence of weekly smoking has stabilised and in some cases fallen (eg older boys)[i]

i. Roberts C, Kingdon A, Parry-Langdon N, Bunce J. *Young People in Wales: Findings from the Health Behaviour in School-aged Children (HSBC) Study 1986-2000.* Cardiff: Health Promotion Division, Welsh Assembly Government, August 2002.
http://www.hpw.wales.gov.uk/English/resources/reportsandpapers/HBSC02_final_e.pdf [accessed 17.12.03]
(Type IV evidence – cross-national research study conducted in collaboration with the World Health Organisation Regional Office for Europe. Surveys are conducted every four years. In 1997/8, 120,000 young people from 26 European countries, Canada and the United States participated. Wales also undertakes interim surveys on a biennial basis, the most recent being in 2000)

Physical activity (updated September 2004)

1.2g. The number of adults aged 18-64 in Wales who take regular **physical activity** has increased slightly from 22.4% (33.0% in males & 11.9% in females) in 1985 to 25.5% (34.9% & 16.0%) in 1998[i].

Regular exercise was defined as undertaking at least 20 minutes moderate or strenuous physical activity in leisure time on three or more occasions per week[i].

The Department of Health recommends that in order to benefit health, adults should aim to take at least 30 minutes of at least moderate intensity activity on five or more days a week[ii] (confirming their recommendation in 1996). A person who is doing moderate intensity activity will usually experience:

- an increase in breathing rate
- an increase in heart rate (the pulse can be felt during this level of activity)
- a feeling of increased warmth, possibly accompanied by sweating[ii]

The American College of Disease Prevention and the World Health Organisation recommend at least 30 minutes moderate-intensity activity on most days of the week [iii,iv]. These recommendations are arbitary points on a continuum

(see statements 1.9a and 1.9e)

i. *Health Statistics Wales 2001*. Chapter 2 – Lifestyle. Cardiff: National Assembly for Wales, March 2002
http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/health/2002/hsw2001/hsw2001-ch2/hsw2001-ch2.htm [accessed 17.12.03]
(Type IV evidence – statistics)

ii. Department of Health, Physical Activity, Health Improvement and Prevention. At Least Five a Week, Evidence on the Impact of Physical Activity and its Relationship to Health. Department of Health: London, 2004
(Type V evidence - expert opinion)

iii. Pate RR, Pratt M, Blair SN et al. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *Journal of the American Medical Association* 1995; **273(5)**: 402-407
(Type V evidence - expert opinion)

iv. Joint WHO/FAO Expert Consultation. Diet, Nutrition and the Prevention of Chronic Disease. WHO Technical Report Series 916. Geneva: World Health Organisation, 2003
(Type V evidence - expert opinion)

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1.2h. Boys **exercise** more than girls[i,ii] across most countries and this declines with age, especially for girls[ii]. In Wales the proportion of young people taking part in physical activity for at least four hours per week (outside school) increased between 1986 and 2000 for boys and girls in all age groups. 2000 data show that 34% of boys and 18% of girls aged 11-12 reported exercising four or more times a week. Unlike girls, where weekly activity was similar among all three age groups, the proportion of boys reporting to exercise at this level increased with age[i].

The international HBSC (Health Behaviour in School-aged Children) report, due to be published by WHO in February 2004, will present updated physical activity data for 35 countries including Wales[iii].

The measure used in Wales is 'physical activity is any activity that increases your heart rate and makes you get out of breath some of the time… running, brisk walking, rollerblading, biking, dancing, skateboarding, swimming, netball, basketball, football and rugby'. For the data covering 1996-2000 the definitions used for times/hours of exercise per week was '[How many hours per week/how often] do you usually exercise in your free time so much that you get out of breath or sweat?'[iii]. The measures are based on self report and developed from a validated instrument[iv].

The *evidence*

i. Roberts C, Kingdon A, Parry-Langdon N, Bunce J. *Young People in Wales: Findings from the Health Behaviour in School-aged Children (HSBC) Study 1986-2000*. Cardiff: Health Promotion Division, Welsh Assembly Government, August 2002.
http://www.hpw.wales.gov.uk/English/resources/reportsandpapers/HBSC02_final_e.pdf [accessed 17.12.03]
(Type IV evidence – cross-national research study conducted in collaboration with the World Health Organisation Regional Office for Europe. Surveys are conducted every four years. In 1997/8, 120,000 young people from 26 European countries, Canada and the United States participated. Wales also undertakes interim surveys on a biennial basis, the most recent being in 2000)

ii. Currie C, Hurrelmann K, Settertobulte W, Smith R, Todd J (eds). *Health and Health Behaviour among Young People*. Copenhagen: World Health Organisation Regional Office for Europe, 2000
http://www.hbsc.org/downloads/Int_Report_00.pdf
[accessed 17.12.03]
(Type IV evidence – see i. above]

iii. **http://www.hbsc.org** [accessed 17.12.03]

iv. Prochaska JJ, Sallis JR, Long B. A physical activity screening measure for use with adolescents in primary care. *Archives of Paediatrics and Adolescent Medicine* 2001; **155**: 554-559

1.2i. A small heart monitoring study suggested that British children meet the **UK and US minimum recommendations** of 60 minutes accumulated moderate-intensity physical activity (>120 beats/minute) per day[i]
Caveats: The heart rate monitor may not be an accurate measure of physical activity. In addition, only half the children approached volunteered for the study and data were not available for all of these so there is potential for bias.

In another small study of children from the South of England 67.3% of the boys and 59.5% of the girls accumulated 30 minutes of at least moderate intensity physical activity (≥ 140 beats/minute) on each of the three school days monitored[ii].
Caveat: The heart rate monitor may not be an accurate measure of physical activity.

i. Sleap M, Tolfrey K. Do 9 to 12 yr-old children meet existing physical activity recommendations for health? *Medicine and Science in Sports and Exercise* 2001; **33(4)**: 591-596
(Type IV evidence - a heart rate monitoring study of data from 79 British children (aged 9-12) over four days)

ii. Welsman J, Armstrong N. Physical activity patterns in secondary school children. *European Journal of Physical Education* 2000; **5(2)**: 147-157
(Type IV evidence – a heart rate monitoring study of data covering three school days from 52 boys and 42 girls. Data were collected in 1999 and 2000)

The *statements*

The *evidence*

Alcohol consumption

1.2j. More than one in four men in Wales **drink above sensible limits** compared with one in nine women. There has been a rise in the proportion of women drinking to excess in recent years[i].

The number of adults aged 18-64 in Wales who drink alcohol above the recommended sensible limits was 19.8% (28.8% in males & 9.5% in females) in 1988 compared to 19.7% (27.4% & 11.2%) in 1996[ii].

In 1998 25% of males and 16% of females in Wales drank above recommended limits compared to 27% of males and 14% of females in England, and 25% of males and 11% of females in Scotland[iii].

Definition: The sensible drinking limit for men is 21 units per week and 14 units per week for women. A unit represents 10 grams of alcohol.

i. *A Statistical Focus on Wales*. Chapter 5 – health. Cardiff: National Assembly for Wales, 1999 **http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/compendia/1999/fow/focwalch5.pdf** [accessed 17.12.03]
(Type IV evidence – statistics)

ii. *Health Statistics Wales 2001*. Chapter 2 – Lifestyle. Cardiff: National Assembly for Wales, March 2002 **http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/health/2002/hsw2001/hsw2001-ch2/hsw2001-ch2.htm** [accessed 17.12.03]
(Type IV evidence – statistics)

iii. *Comparative Indicators 2000. Wales England Scotland*. Cardiff: National Assembly for Wales, November 2001. **http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/compendia/2001/Comparative_Indicators_2000.pdf** [accessed 17.12.03]
(Type IV evidence – statistics)

1.2k. The number of **children** aged 11-16 in Wales who **drink alcohol** weekly was 31.0% (35.9% in boys & 25.9% in girls) in 1986 and 30.6% (34.7% & 26.4%) in 1998[i].

i. *Health Statistics Wales 2001*. Chapter 2 – Lifestyle. Cardiff: National Assembly for Wales, March 2002 **http://www.wales.gov.uk/keypubstatisticsforwales/content/publication/health/2002/hsw2001/hsw2001-ch2/hsw2001-ch2.htm** [accessed 17.12.03]
(Type IV evidence – statistics)

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National Service Framework

National Assembly for Wales. *Tackling CHD in Wales: Implementing Through Evidence.* Cardiff: National Assembly for Wales, July 2001

People at high risk of developing CHD are those with one or more of the following risk factors, multiple risks being especially dangerous: [paragraph 1.7]

- Diabetes;
- Hypertension (high blood pressure);
- Hyperlipidaemia (high blood cholesterol and/or triglyceride);
- A family history of CHD;
- A smoking habit;
- Too overweight;
- Too little exercise.

What is the evidence for increased risk/high risk groups?

The *statements*	The *evidence*

Risk factors for coronary heart disease *See Chapter Two for risk assessment tools*

1.3 Diabetes

1.3a. The life expectancy of a patient with **type 2 diabetes** is reduced by 8-10 years. Atherosclerotic vascular disease (especially **coronary artery disease** and stroke) is the principal cause of death in about 70% of these patients[i].

i. Scottish Intercollegiate Guidelines Network. *Management of Diabetes*. SIGN Publication No. 55. Edinburgh: SIGN, November 2001
http://www.sign.ac.uk/guidelines/fulltext/55/index.html
[accessed 17.12.03]
(Type IV evidence – statistics)

1.3b. Results from a meta-analysis suggested that, after combining studies that adjusted for other cardiac risk factors, the relative risk of coronary death from **diabetes** was 2.58 (95% CI 2.05-3.26) for **women** and 1.85 (1.47-2.33) for men. The difference is just statistically significant (p=0.045)[i]. *Further research is required to explain this difference between the sexes*[i].

i. Lee WL, Cheung AM, Cape D, Zinman B. Impact of diabetes on coronary artery disease in women and men: A meta- analysis of prospective studies. *Diabetes Care* 2000; **23(7)**: 962-8.
(Type IV evidence – systematic review, Medline only to January 1999, and meta-analysis of 10 prospective cohort studies)

1.3c. Populations with **lower insulin resistance** are consistently associated with better overall cardiovascular risk profiles (including reduced clustering of risk factors), and improved coronary artery disease outcomes than populations with elevated insulin resistance[i].

i. Smiley T, Shame LG. The relationship of insulin resistance measured by reliable indexes to coronary artery disease risk factors and outcomes - a systematic review. *Canadian Journal of Cardiology* 2001; **17(7)**: 797-805
(Type IV evidence - systematic review and meta-analysis of 28 studies (twenty investigated the relationship of insulin resistance markers with coronary artery disease risk factor profiles only, while eight publications primarily evaluated coronary artery disease outcomes)

The *statements* | The *evidence*

1.4 Hypertension

1.4a. **Hypertension** is a major risk factor for stroke and coronary heart disease[i]. The residual lifetime risk for hypertension in middle-aged and elderly individuals is 90%, indicating a huge public health burden. The residual lifetime risks for developing hypertension and stage 1 high blood pressure or higher (≥ 140/90 mm Hg regardless of treatment) were 90% in both 55 and 65 year-old participants. The lifetime probability of receiving antihypertensive medication was 60%[i].

The risk for hypertension remained unchanged for women, but it was approximately 60% higher for men in the contemporary 1976-1998 period compared with an earlier 1952-1975 period. In contrast, the residual lifetime risk for stage 2 high blood pressure or higher (≥ 160/100 mm Hg regardless of treatment) was considerably lower in both sexes in the recent period (35%-57% in 1952-1975 vs. 35%-44% in 1976-1998), likely due to a marked increase in treatment of individuals with substantially elevated blood pressure[ii]. **Caveat:** As a considerable proportion of individuals with hypertension have onset of the condition before age 55 the actual lifetime risk for hypertension for younger individuals may be different from those found in this study.

i. Dawber TR. *The Framingham Study. The Epidemiology of Atherosclerotic Disease*. Cambridge, MA: Havard University Press, 1980

(Type IV evidence - summary of results from prospective cohort study of 5,127 persons with 24 year follow-up)

ii. Vasan RS, Beiser A, Seshadri S, Larso MG, Kannel WB, D'Agostino RB, Levy D. Residual lifetime risk for developing hypertension in middle-aged women and men: The Framingham Heart Study. *Journal of the American Medical* Association 2002; **287(8)**: 1003-1010

(Type IV evidence – community-based prospective cohort study of 1,298 (589 men and 709 women providing 8,469 person years of observation) participants from the Framingham Heart Study who were aged 55 to 65 years and free of hypertension at baseline (1976-1998)

1.4b. **Opportunistic case finding** of patients at high risk of coronary heart disease is the method of choice in primary care for people aged 45 –64[i].

Primary care teams should target opportunistic screening and treatment at persons aged over 65 years with high blood pressure before assessing younger patients, aged 46 –64 years. No health benefit results from screening persons aged less than 45 years[ii].

i. Temple JMF, Fone DL. Implementing national guidance on prevention of coronary heart disease : Clinical governance and computer simulation modelling. *British Journal of Clinical Governance* 2002; **7(1)**: 27-33

(Type IV evidence – comparison of population screening with opportunistic case finding in a discrete event computer simulation model of the population aged 45-64 in one local health group in Wales)

ii. Marshall T, Rouse A. Meeting the National Service Framework for coronary heart disease: which patients have untreated high blood pressure? *British Journal of General Practice* 2001; **51(468)**: 571-4

(Type IV evidence – modelling exercise to estimate the potential number of patients who are eligible for blood pressure assessment, the number of preventable cardiovascular disease events and the relative efficiency of the strategy in different age groups using a hypothetical population of 100,000; Data from the Health Survey for England, the General Practice Research Database & the Newcastle Heart Project were used, plus trial results for the efficacy of antihypertensive medication)

The *statements*

The *evidence*

1.5 Hyperlipidaemia

1.5a. The relationship between **serum cholesterol** and lifetime CHD mortality is continuous and curvilinear[i,ii]. The strength of this relationship is greatest in younger people: a 10% reduction in serum cholesterol is associated at five year follow-up with a 54% reduction in the incidence of CHD at 40 years, 27% at 60 years and a 19% reduction at 80 years[i].

i. Law MR, Wald NJ, Thompson SG. By how much and how quickly does reduction in serum cholesterol concentration lower risk of Ischemic heart disease? *British Medical Journal* 1994; **308**: 367-72

(Type II evidence - review of 28 randomised controlled trials and 10 prospective cohort studies)

ii. Navas-Nacher EL, Colangelo L, Beam C, Greenland P. Risk factors for coronary heart disease in men 18 to 39 years of age. *Annals of Internal Medicine* 2001; **134**: 433-439

(Type IV evidence – prospective cohort study with 20 years follow-up of 11,016 men aged 18-39 years (mean age 19.7 years) at baseline as part of the Chicago Heart Association Detection Project in Industry. 8,955 men aged 40-59 years at baseline served as the reference group)

1.6 Family history of coronary heart disease

1.6a. In a case-control study the **parents**; especially the fathers of children with coronary risk factors had higher levels of coronary risk factors than those in the control group. There is a familial aggregation of body fat and adverse lipid levels in the families of the children with coronary risk factors. The parents of children recognized as having coronary risk factors should therefore be evaluated for these risk factors too[i].

After adjusting for covariates, the mothers' "childhood-offspring" correlations were consistently higher than mothers' "adulthood-offspring" correlations for body mass index (BMI) [r = 0.45 vs. 0.32], systolic blood pressure (SBP) [r = 0.30 vs. 0.10], diastolic blood pressure (DPB) [r = 0.22 vs. 0.13] and low-density lipoprotein cholesterol (LDLC) [r = 0.20 vs. 0.11]. In contrast, high-density lipoprotein cholesterol (HDLC) and triglycerides did not show such age-specific trends in mother-offspring correlations[ii].

i. Ucar B, Kilic Z, Sonmez HM, Ata N, Ozdamar K. Relationships between the children and the parents for coronary risk factors. *Paediatrics International* 2001; **43(6)**: 611-623

(Type IV evidence – observational study in Turkey of 252 parents of 164 children with two or more coronary risk factors. The control group consisted of 175 parents of 114 children with no risk factors)

ii. Chen W, Srinivasan SR, Bao W, Berenson GS. The magnitude of familial associations of cardiovascular risk factor variables between parents and offspring are influenced by age: The Bogalusa Heart Study. *Annals of Epidemiology* 2001; **11(8)**: 522-528

(Type IV evidence – observational study of 727 unrelated children (mean age: 11.2 years) and their parents who participated in the Bogalusa Heart Study during their childhood (mean age: 11.3 years) and adulthood (mean age: 25.5 years)

The *statements*

The *evidence*

1.6b. **Family history** of coronary heart disease is not only a strong risk factor for myocardial infarction in both sexes, but its effect is synergistic with other cardiovascular risk factors as well. The adjusted odds ratio (OR) for myocardial infarction was 2.0 (95% CI, 1.6-2.6) for men reporting one or more affected parent or sibling, compared with men with no family history of coronary heart disease, and 3.4 (2.1-5.9) for those reporting two or more affected parents or siblings. The corresponding OR for women was 2.1 (1.5-3.0) and 4.4 (2.4-8.1). There was evidence for synergistic interactions in women exposed to a family history of coronary heart disease in combination with current smoking and with a high quotient between low-density lipoprotein and high-density lipoprotein cholesterol (>4.0), respectively, which yielded adjusted synergy index scores of 2.9 (1.2-7.2) and 3.8 (1.5-9.7), respectively. Similarly, in men there was evidence for interaction for the co-exposure of family history of coronary heart disease and diabetes mellitus[i].
Caveat: Fatal myocardial infarction (MI) cases were excluded from this study. It has been suggested that the influence of a family history may be stronger on fatal cases of MI than on non-fatal cases. A further potential limitation of this study is the risk of recall bias. Both underreporting and over reporting of affected relatives are possible.

i. Leander K, Hallqvist J, Reuterwall C, Ahlbom A, De Faire U. Family history of coronary heart disease, a strong risk factor for myocardial infarction interacting with other cardiovascular risk factors: Results from the Stockholm Heart Epidemiology Program (SHEEP). *Epidemiology* 2001; **12(2)**: 215-221

(Type IV evidence – observation study of 1,091 male and 531 female (45-70 years of age) first-time acute myocardial infarction patients who had survived at least 28 days after their infarction)

1.6c. An American study found that physicians do not appear to follow **national recommendations** for the screening of family members of their high-risk patients. Less than 1% of inpatient medical records contained a discharge plan by the physician recommending screening family members of patients younger than age 55[i].

i. Swanson JR, Pearson TA. Screening family members at high risk for coronary disease: Why isn't it done? *American Journal of Preventive Medicine* 2001; **20(1)**: 50-55

(Type IV evidence – observational study using data from the American College of Cardiology Evaluation of Preventive Therapeutics (ACCEPT) study a national survey conducted in 1996-1997, determined if physicians were screening first-degree relatives of patients with early CVD. The ACCEPT study included 5,553 patients with either their first bypass surgery, first angioplasty, an acute myocardial infarction, or myocardial ischemia, admitted to 53 hospitals)

The *statements*

The *evidence*

1.7 Smoking

1.7a. Cigarette smoking is associated with a two to three-fold increase in coronary heart disease[i] (CHD) and peripheral vascular disease[ii] risk in adults or "compared to the adult population" . The risk rises with the number of cigarettes smoked per day. In 1990, smoking accounted for 28% of male and 26% of female all-cause vascular deaths aged 35 to 69[iii].

The Health Education Authority estimated that more than 20% of deaths under age 65 from ischaemic heart disease in the UK were caused by smoking in 1995[iv] and, more recently, the British Heart Foundation has also suggested that about 20% of deaths from CHD in men and 17% of deaths from CHD in women are due to smoking[v].

i. Dawber TR. *The Framingham Study. The Epidemiology of Atherosclerotic Disease*. Cambridge, MA: Havard University Press, 1980

(Type IV evidence - summary of results from prospective cohort study of 5,127 persons with 24 year follow-up)

ii. Kannel WB, McGee DL. Update on some epidemiologic features of intermittent claudication: the Framingham Study. *Journal of the American Geriatric Society* 1985; **33**: 13-18

(Type IV evidence - prospective cohort study of 5,209 subjects with 26 year follow-up)

iii. Peto R, Lopez AC, Boreham J, Thun M, Heath C. *Mortality from Smoking in Developed Countries 1950-2000*. Oxford: Oxford University Press (Oxford Medical Publications), 1994

(Type IV evidence - summary of indirect estimates of mortality from National Vital Statistics)

iv. Callum C. *The UK Smoking Epidemic: Deaths in 1995*. London: Health Education Authority, 1998

(Type IV evidence - statistics)

v. *British Heart Foundation Statistics Database 2002. Annual Compendium: 2002 edition*. Smoking. **http://www.heartstats.org/uploads/documents/2003%20Smoking%20chapter.pdf** [accessed 17.12.03]

(Type IV evidence – statistics)

The *statements*

1.7b. In **non-smokers**, self-reported exposure levels to **environmental tobacco smoke** ("passive smoking") and serum cotinine levels are associated with diagnosed Ischemic heart disease[i]. The best estimate of the reversible (cause and effect) component of the association is a relative risk of 1.23; 95% CI 1.14-1.33[ii, iii]. A recent report concluded that exposure to other peoples' tobacco smoke causes a number of life-threatening health problems and calls on the government to introduce legislation to ban smoking in public places and to introduce public information campaigns[iv].

Conversely, a recently published large prospective cohort study did not support a causal relationship between environmental tobacco smoke and coronary heart disease. The age adjusted relative risk for never smokers married to ever smokers compared with never smokers married to never smokers was 0.94 (95% CI 0.85-1.05)[v]. **Caveats:** In keeping with some earlier studies, the smoking status of the spouse does not equate with environmental tobacco smoke exposure. The exposures to spousal tobacco smoke in a Californian climate may not be generalisable to the more 'indoor' lifestyle in the UK. The study was supported in part by tobacco company funding although the authors (both lifelong non-smokers) claimed unbiased status.

The *evidence*

i. Tunstall-Pedoe H, Brown CA, Woodward M, Tavendale R. Passive smoking by self-report and serum cotinine and the prevalence of respiratory and coronary heart disease in the Scottish heart study. *Journal of Epidemiology and Community Health* 1995; **49**: 139-143

(Type IV evidence - cross-sectional survey of 786 men and 1,492 women aged 40-59 years who reported never having smoked)

ii. Law MR, Morris JK, Wald NJ. Environmental tobacco smoke exposure and ischaemic heart disease: an evaluation of the evidence. *British Medical Journal* 1997; **315**: 973-980 **http://bmj.bmjjournals.com/cgi/content/full/315/7114/973** [accessed 17.12.03]

(Type IV evidence – systematic review and meta-analysis of 19 observational studies)

iii. Poswillo D, Chairman. Department of Health, Department of Health and Social Services, Northern Ireland, The Scottish Office Department of Health, Welsh Office. *Report of the Scientific Committee on Tobacco and Health*. London: The Stationery Office, 1998 **http://www.archive.official-documents.co.uk/document/doh/tobacco/contents.htm** [accessed 17.12.03]

(Type V evidence - expert opinion)

iv. British Medical Association Board of Science and Education and Tobacco. *Towards Smoke-Free Public Places*. London: BMA, 2002

v. Enstrom JE, Kabat GC. Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians, 1960-1998. *British Medical Journal* 2003; **326**: 1057 **http://bmj.bmjjournals.com/cgi/content/full/326/7398/1057** [accessed 17.12.03]

(Type IV evidence – prospective cohort study covering 39 years. 118,094 adult Californians enrolled in late 1,959 in the American Cancer Society cancer prevention study were followed until 1998. 35,561 never smokers had a spouse in the study with known smoking habits)

1 LIFESTYLE

The *statements*

1.7c. A study in Greece suggested that **stopping smoking** is exponentially related to the reduction of coronary risk while passive smoking increases the risk from 48% (when encountered at work) to 112% (a non-smoking woman with a smoking husband). Familial smoking habits are related to current smoking status, increasing the coronary risk four-fold[i].

Caveats: The lifestyle of a Mediterranean population may differ from the British lifestyle. The baseline differences between cases and controls were also significantly different in terms of income, marital status, education and occupation, although these confounders were taken into account in the analysis.

The *evidence*

i. Panagiotakos DB, Pitsavos C, Chrysohoou C, Stefanadis C, Toutouzas P. Risk stratification of coronary heart disease through established and emerging lifestyle factors in a Mediterranean population: cardio2000 epidemiological study. *Journal of Cardiovascular Risk* 2001; **8**: 329-335

(Type IV evidence – case-control study of 535 male and 126 female patients with a first event of an acute coronary syndrome and 661 controls matched by sex, age and region)

1.8 Obesity and overweight

1.8a. Non-diabetic subjects with BMI > 35 kg/m^2 carry a burden of coronary risk factors that appears to increase with greater **obesity**. In comparison with the group of subjects with BMI 30-35 kg/m^2 mean values for all variables were higher in the more obese subjects. All variables measured showed an increase with BMIs over 35 (triglycerides, P=0.04; glucose, P=0.007; urate, P < 0.001; systolic BP, P < 0.001; diastolic BP, P < 0.001). Cholesterol concentration showed no relationship with BMI[i].

Caveat: Blood pressure data were only available for those patients who were not on any relevant drug therapy. Study personnel were unable to distinguish true hypertensive patients from those using potentially anti-hypertensive agents for other reasons.

i. Whitelaw DC, O'Kane M, Wales JK, Barth JH. Risk factors for coronary heart disease in obese non-diabetic subjects. *International Journal of Obesity & Related Metabolic Disorders* 2001; **25(7)**: 1042-1046

(Type IV – observational study of 386 consecutive non-diabetic obese subjects (301 women, 85 men) attending an obesity clinic for the first time (mean BMI 43.3 kg/m2; range 30.6-71.5), aged 17-69y (mean 40.1)

The *statements*

The *evidence*

1.8b. Of **overweight children** (BMI ≥ 95th percentile), 77% remained obese (≥ 30 kg/m^2) as adults. Childhood overweight was related to adverse risk factor levels among adults, but associations were weak (r ~ 0.1-0.3) and were attributable to the strong persistence of weight status between childhood and adulthood. Although obese adults had adverse levels of lipids, insulin and blood pressure, levels of these risk factors did not vary with childhood weight status or with the age (≤ 8 years, 12-17 years, or ≥ 18 years) of obesity onset[i].

In contrast, another longitudinal study found **little tracking from childhood overweight to adulthood obesity** when using a measure of fatness that was independent of build. Only children who were obese at 13 showed an increased risk of obesity as adults. No excess adult health risk from childhood or teenage overweight was found. Being thin in childhood offered no protection against adult fatness, and the thinnest children tended to have the highest adult risk at every level of adult obesity[ii].

Caveat: The subjects were a post-war cohort and findings may not be generalisable to the current situation.

i. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: The Bogalusa heart study. *Pediatrics* 2001; **108(3)**: 712-718

(Type IV evidence – observational study of the longitudinal relationship of childhood body mass index (BMI, kg/m2) to adult levels of lipids, insulin, and blood pressure among 2,617 participants. All participants were initially examined at ages 2 to 17 years and were reexamined at ages 18 to 37 years; the mean follow-up was 17 years)

ii. Wright CM, Parker L, Lamont D, Craft AW. Implications of childhood obesity for adult health: findings from thousand families cohort study. *British Medical Journal* 2001; **323**: 1280-1284 **http://bmj.bmjjournals.com/cgi/content/full/323/7324/1280** [accessed 17.12.03]

(Type IV evidence – prospective cohort study of 932 members of thousand families 1,947 birth cohort (from Newcastle upon Tyne, UK), of whom 412 attended for clinical examination aged 50)

1.8c. **Waist circumference** may be the best anthropometric predictor of cardiovascular risk factors with cut-off points of ≥ 90 cm in men and ≥ 80 cm in women as the most appropriate for prediction of individual and multiple risk factors in the Caucasian population[i].

Caveat: The cross-sectional design weakens the strength of the evidence. There is a lack of clarity in tables and text in this study.

i. Dobbelsteyn CJ, Joffres MR, MacLean DR, Flowerdew G; and the Canadian Heart Health Surveys Research Group. A comparative evaluation of waist circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk factors. The Canadian Heart Health Surveys. *International Journal of Obesity* 2001; **25**: 652-661

(Type IV evidence – population based cross-sectional surveys from 1986-1992 of 9,913 men and women aged 18-74 selected using health insurance registries from five Canadian provinces)

1.8d. **Body mass index** was a **poor discriminator** of women at different levels of coronary heart disease risk although a significant rise in risk for those with a BMI above 22 kg m^{-2} was observed[i]. Based on a multifactor 10-year coronary heart disease risk estimate, odds ratios for being in the highest quintile of risk for each category of body mass index were 1 (<20 kg m^{-2}), 0.91, 1.56, 2.18, 2.97, 3.83 and 4.21 (≥ 30 kg m^{-2})[i].

i. Ashton WD, Nanchahal K, Wood DA. Body mass index and metabolic risk factors for coronary heart disease in women. *European Heart Journal* 2001; **22**: 46-55

(Type IV evidence – cross-sectional survey in the UK of cardiovascular risk factors in 14,077 women, aged 30-64 years, employed by Marks & Spencer)

1 LIFESTYLE

The *statements*

1.8e. **Lifestyle modification** such as weight loss may be effective in long-term primary prevention of hypertension and reduction in blood pressure[i, ii]. From one trial of weight loss or dietary sodium reduction, the odds of hypertension were reduced by 77% (odds ratio 0.23; 95% CI 0.07-0.76; P=0.02) in the weight loss group and by 35% (odds ratio 0.65; 0.25 -1.69; P=0.37) in the sodium reduction group compared with their control groups[i].

In another trial of a weight loss intervention the risk ratio for hypertension in the intervention group was 0.58 (95% CI, 0.36-0.94) at 6 months, 0.78 (0.62 -1.00) at 18 months, and 0.81 (0.70-0.95) at 36 months. In subgroup analyses, intervention participants who lost at least 4.5 kg at 6 months and maintained this weight reduction for the next 30 months had the greatest reduction in blood pressure and a relative risk for hypertension of 0.35 (0.20-0.59)[ii].
Caveat: Follow up rates were more than 92% for weight measurements but an intention to treat analysis was not carried out.

The *evidence*

i. He J, Whelton PK, Appel LJ, Charleston J, Klag MJ. Long-term effects of weight loss and dietary sodium reduction on incidence of hypertension. *Hypertension* 2000; **35**:544-549

(Type II evidence – randomised controlled trial of 181 patients (30-54 years old) followed-up for 6-8 years)

ii. Stevens VJ, Obarazanet E, Cook NR *et al.* Long-term weight loss and changes in blood pressure: results of the Trials of Hypertension Prevention, Phase II. *Annals of Internal Medicine* 2001; **134:1**-11

(Type II evidence – randomised controlled trial of 1,191 patients (30 – 54 years of age) who had non-medicated DBP of 83 to 89 mm Hg and SBP less than 140 mm Hg and were 110% to 165% of their ideal body weight at baseline with follow-up of 6, 18 and 36 months)

1.8f. Slow foetal and infant growth followed by **rapid weight gain** in early childhood are associated with large increases in disease risk. It has been suggested that any effective strategy to prevent coronary heart disease should include:

- Improving body composition and dietary balance among girls and young women
- Protecting the growth of babies in utero and during infancy, and
- Preventing rapid weight gain in young children who had low birthweight[i].

i. Barker DJP. The foetal and infant origins of coronary heart disease. Chapter one in Giles A (ed.); *National Heart Forum. A Lifecourse Approach to Coronary Heart Disease Prevention*. London: The Stationery Office, 2003

(Type V evidence – expert opinion)

The *statements* | The *evidence*

1.9 Physical activity

1.9a. **Exercise** is beneficial in terms of cardiovascular health but authors differ as to whether the law of diminishing returns may apply to increasing amounts of exercise or whether the relationship is linear[i-vi].
Caveat: It is possible that most studies in the past used too broad a baseline reference group to allow conclusions to be drawn about the effect of small amounts of activity undertaken as part of daily living. This makes interpretation of the low activity end of the graph difficult.

The Chief Medical Officer for England has commissioned a major report on the health benefits of physical activity and this should be published early in 2004[vii].

i. Williams PT. Physical fitness and activity as separate heart disease risk factors: A meta-analysis. *Medicine and Science in Sports and Exercise* 2001; **33(5)**: 754-761.
(Type IV evidence – review & meta-analysis of seven fitness and 16 physical activity cohort studies)

ii. Kohl HW. Physical activity and cardiovascular disease: evidence for a dose response. *Medicine and Science in Sports and Exercise* 2001; **33(6)**: S472-S483.
(Type IV evidence – systematic review & meta-analysis, Medline search only to August 2000, of 39 observational studies)

iii. Lee IM, Skerrett PJ. Physical activity and all cause mortality: what is the dose response relationship? *Medicine and Science in Sports and Exercise* 2001; **33(6)**: S459-S471.
(Type IV evidence – systematic review & meta-analysis of 44 observational studies)

iv. US Department of Health and Human Services, *Physical Activity and Health: A Report of the Surgeon General*: Atlanta, GA. US Department of Health and Human Services, Centres for Disease Control & Prevention National Centre for Chronic Disease Prevention and Health Promotion, 1996
http://www.cdc.gov/nccdphp/sgr/pdf/sgrfull.pdf
[accessed 17.12.03]
(Type V evidence – expert opinion)

v. Philips WT, Pruitt LA, King AC. Lifestyle activity - Current Recommendations. *Sports Medicine* 1996; **22(1)**: 1-7.
(Type V evidence – expert opinion)

vi. Pate RR, Pratt M, Blair SN *et al*. Physical Activity and Public Health. *Journal of the American Medical Association* 1995; **273(5)**: 402-407.
(Type V evidence – expert opinion)

vii. **http://www.doh.gov.uk/** [accessed 17.12.03]

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1.9b. Evidence from observational studies suggests that regular **physical activity** attenuates the risk of coronary heart disease associated with overweight or obesity and active obese individuals actually have lower morbidity and mortality than normal weight individuals[i]. In one of the studies included in the review, men with a BMI ≥ 27.0 who regularly participated in vigorous exercise had a heart attack rate of 1.3/1000 man years, and men with a BMI < 24.0 who were inactive had a rate of 5.5/1000 man-years[ii].

The *evidence*

i. Blair S, Brodney S. Effects of physical inactivity and obesity on morbidity and mortality: current evidence and research issues. *Medicine and Science in Sports and Exercise* 1999; **31**(11 Suppl.): S646-S662

(Type IV evidence – systematic review, literature search date not given, of 24 prospective observational studies. The study results cited are from:

ii. Morris JN, Clayton DG, Everitt MG, Semmence AM, Burgess EH. Exercise in leisure time: coronary attack and death rates. *British Heart Journal* 1990; **63**: 325-334)

1.9c. The **cost effectiveness** of exercise is very dependant on the indirect cost assigned to the time spent on exercise. If this time is valued at "no cost", then exercise is cost saving. However, even using half the wage rate as a measure of the "opportunity cost", the cost per QALY rises very rapidly for those who earn more than a very low wage[i].

i Hatziandreu EI, Koplan JP, Weinstein MC *et al*. A cost effectiveness analysis of exercise as a health promotion activity. *American Journal of Public Health* 1988; **78**: 1417-1421.

(Type IV evidence - economic analysis)

1.9d. When advice to **increase walking** is taken up, the beneficial effects are sustained for up to 10 years[i].

Advising walking is more successful in attaining high levels of participation as compared to advising other forms of exercise. Patients exercising alone also complete more exercise sessions than those in groups[ii].

Robust primary care research programmes advising an increase in walking are required in a UK context.

i. Pereira MA, Kriska AM, Day RD, Cauley JA, LaPorte RE, Kuller LH. A randomised walking trial in postmenopausal women. Effects on physical activity and health 10 years later. *Archives of Internal Medicine* 1998; **158(15)**: 1695-1701.

(Type II evidence – 10 year follow-up of a randomised controlled trial of 229 volunteer postmenopausal women randomised to a walking group versus control group intervention)

ii. Hillsdon M, Thorogood M, Anstiss T, Morris J. Randomised controlled trials of physical activity promotion in free living populations: a review. *Journal of Epidemiology and Community Health* 1995; **49**: 448-453

(Type I evidence – systematic review, literature search to 1993, of 10 randomised controlled trials and 1,494 patients)

The *statements*

The *evidence*

1.9e. The **minimum level of exercise** required to provide some reduction in heart disease risk in healthy women aged 45 yrs and over may be as low as 20 - 60 minutes purposeful walking (200-599 kcal/wk) at a gentle pace (<3.2km/hr or 2miles/hr) per week. Age and treatment adjusted relative risks for energy expenditure of 200-599 kcal/wk as opposed to <200kcal/wk was 0.59 (95% CI 0.42-0.81)[i].

The maximum level of exercise that is beneficial may vary between groups. There appears to be increasing benefit from exercise up to a level equivalent to running 80 miles per week for some individuals, at least in terms of lipid levels[ii].

i. Lee I-M, Rexrode KM, Cook NR, Manson JE, Buring JE. Physical activity and coronary heart disease in women: Is 'No Pain, No Gain' passe? *Journal of the American Medical Association* 2001; **285(11)**: 1447-1454.
(Type IV evidence – cohort study of 39,372 health female health professionals aged 45 and older)

ii. Williams PT. Relationship of distance run per week to coronary heart disease risk factors in 8,283 male runners – The national runners' health study. *Archives of Internal Medicine* 1997; **157(2)**: 191-198
(Type IV evidence – cross-sectional survey in the USA of 8,283 male recreational runners)

Other lifestyle factors:

■ Alcohol consumption ■ Antioxidants ■ C-reactive protein ■ Coffee ■ Fibrinogen ■ Folate ■ Fruit and vegetable consumption ■ Gout/uric acid ■ Hormone replacement therapy ■ Hyperhomocysteinemia ■ Lycopene ■ Psychosocial factors

1.10 Alcohol consumption

1.10a. The association between **alcohol consumption** and risk of chronic hypertension in young women follows a J-shaped curve, with light drinkers demonstrating a modest decrease in risk and more regular heavy drinkers demonstrating an increase in risk[i].

Compared with non-drinkers, the risk of developing hypertension according to average number of drinks consumed per day was as follows (standard drink, 12g alcohol): 0.25 or less, 0.96 (95% CI 0.89-1.03); 0.26-0.50, 0.86 (0.75-0.98); 0.51-1.00, 0.92 (0.82-1.04); 1.01-1.50, 1.00 (0.80-1.24); 1.51-2.00, 1.20 (0.92-1.58); more than 2.0 drinks, 1.31 (1.02-1.68)[i].
Caveats: Alcohol consumption and the diagnosis of hypertension were self-reported, although some validity tests were carried out. The nurses included in the study were mostly young and white.

i. Thadhani R, Camargo CA, Stampfer MJ, Curhan CG, Willett WC, Rimm EB. Prospective study of moderate alcohol consumption and risk of hypertension in young women. *Archives of Internal Medicine* 2002; **162(5)**: 569-574
(Type IV evidence – eight year cohort study in the USA of 70,891 women aged 25-42 years; the Nurses Health Study II. A 98% follow-up rate was achieved)

The *statements*

The *evidence*

1.11 Antioxidants

1.11a. There is no evidence of benefit from **betacarotene** supplements and randomised controlled trials suggest they may be harmful. The most recent systematic review (6 RCTs, 86 056 people) found no significant difference between ß carotene and control for cardiovascular disease (OR 1.02, 95% CI 0.96 to 1.08). However, ß carotene was combined with other antioxidants in some treatment groups, and it was not clear whether the systematic review accounted for effects of multiple interventions compared with control[i].

i. Murphy M, Foster C, Sudlow C *et al*. Cardiovascular disorders. Primary prevention. *Clinical Evidence* January 2003. London: BMJ Publications, 2003

(Summary of quality appraised research evidence from a review of the literature completed in March 2002. The most recent systematic review cited, of 17 case-control, 18 cohort and eight randomized controlled studies, was: Asplund K. Antioxidant vitamins in the prevention of cardiovascular disease: a systematic review. *Journal of Internal Medicine* 2002; **251**: 372-392)

Insufficient evidence from randomised controlled trials was found to support the use of **vitamin C, vitamin E, copper, zinc, manganese, flavinoids** in primary prevention[i].

Vitamin E does not prevent cardiovascular events in patients at cardiovascular risk. See statement 2.15b.

1.11b. In 57 trials of mainly Caucasians with normal blood pressure, **low sodium intake** reduced systolic blood pressure by -1.27 mm Hg (95% CI, -1.76 to -0.77, p <0.0001) and diastolic blood pressure by -0.54 mm Hg (-0.94 to -0.14, p = 0.009) as compared to high sodium intake. In 58 trials of mainly Caucasians with elevated blood pressure, low sodium intake reduced SBP by -4.18 mm Hg (-5.08 to -3.27, p < 0.0001) and DBP by -1.98 mm Hg (-2.46 to -1.32, p < 0.0001) as compared to high sodium intake[i].

i. Jürgens G, Graudal NA. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglyceride (Cochrane Review). In: *The Cochrane Library*, Issue 1, 2003. Oxford: Update Software (most recent update 20 November 2002) **http://www.update-software.com/abstracts/ab004022.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search to December 2001 of 57 randomised controlled trials)

1.12 C-reactive protein

1.12a. A series of prospective studies provide consistent data documenting that mild elevation of baseline levels of **c-reactive protein** among apparently healthy individuals is associated with higher long-term risk for future cardiovascular events[i,ii].

i. Ridker PM, Morrow DA. C-reactive protein, inflammation, and coronary risk. *Cardiology Clinics* 2003; **21(3)**: 315-325

(Type V evidence – expert review)

ii. Blake GJ, Ridker PM. C-reactive protein and other inflammatory risk makers in acute coronary syndromes. *Journal of the American College of Cardiology* 2003; **41(4)**: 37S-42S

(Type V evidence – expert review)

The *statements*

The *evidence*

1.13 Coffee

1.13a. **Coffee drinking** in high socio-economic status white men is associated with small increases in blood pressure but appears to play a small role in the development of hypertension. Coffee drinkers had higher rates of hypertension but these were not significant after adjustment for parental incidence of hypertension and time dependent body mass index, cigarette smoking, alcohol drinking and physical activity[i].

i. Klag MJ, Wang N-Y, Meoni LA *et al*. Coffee intake and risk of hypertension. The Johns Hopkins Precursors Study. *Archives of Internal Medicine* 2002; **162**: 657-662

(Type IV evidence – prospective cohort study in the US of 1,017 white male former medical students (mean age 26 years at baseline) with mean follow-up of 33 years)

1.14 Fibrinogen

1.14a. In a population of middle-aged and older men, **high fibrinogen** levels associated with **high lipoprotein** (a) levels significantly increased the risk of coronary heart disease (CHD). Only fibrinogen levels in the upper tertile of the distribution compared with the lower tertiles were associated with a significant risk of CHD (adjusted risk ratio 2.5, 95% CI 1.4-4.2, p=0.0010). Men with high fibrinogen (≥ 4.05 g/L) and high lipoprotein (a) levels (≥ 300 mg/L) levels had a significantly increased risk compared to those with the lower levels (<4.05 g/L fibrinogen and <300 mg/L lipoprotein (a))[i].

i. Cantin B, Després J-P, Lamarche B *et al*. Association of fibrinogen and lipoprotein (a) as a coronary heart disease risk factor in men (the Quebec Cardiovascular Study). *American Journal of Cardiology* 2002; **89**: 661-666

(Type IV evidence – cohort study with five year follow-up of 2,125 men aged 47-76 years)

1.15 Folate

1.15a. Dietary **folate** appears to have a role in the promotion of good cardiovascular health. Men in the highest fifth of folate intake had a relative risk of acute coronary events of 0.45 (95% CI 0.25-0.81, p=0.008) compared with men in the lowest fifth[i].

i. Voutilainen S, Rissanen TH, Virtanen J, Lakka TA, Salonen JT. Low dietary folate intake is associated with an excess incidence of acute coronary events. The Kuopio Ischemic Heart Disease Risk Factor Study. *Circulation* 2001; **103(22)**: 2674-2680

(Type IV evidence – prospective cohort study in Finland of 1,980 men aged 42-60 years old, followed up for an average of 10 years)

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The *statements*

The *evidence*

1.16 Fruit and vegetable consumption

1.16a. Cohort studies have shown that consumption of **fruit and vegetables** reduces ischaemic heart disease and stroke. The size and nature of any real effect is uncertain[i].

i. Murphy M, Foster C, Sudlow C *et al*. Cardiovascular disorders. Primary prevention. *Clinical Evidence* January 2003. London: BMJ Publications, 2003
(Summary of quality appraised research evidence from a review of the literature completed in March 2002)

1.17 Gout/uric acid

1.17a. After adjusting for known risk factors there is no evidence to support **gout** as a risk factor for coronary heart disease in black and white male physicians[i].

i. Bryan E. *Are Gout or Increased Uric Acid Levels Risk Factors for Cardiac Disease?* Center for Clinical Effectiveness Series 2002:Ateiology. Clayton: Monash Institute of Health Services Research, 2002
(Type I evidence - systematic review of 2 prospective longitudinal cohort studies of 371 black men and 1,181 white men with a median follow-up of 30 years. Literature search to 2002)

1.18 Hormone replacement therapy

1.18a. Postmenopausal **oestrogen replacement** is associated with an increased risk for venous thromboembolism. When data from all studies were pooled, current oestrogen use was associated with an increased risk for **venous thromboembolism** (relative risk, 2.14 [95% credible interval, 1.64-2.81]). Estimates did not significantly change when studies were pooled according to study design, quality score, or whether participants had pre-existing coronary artery disease. The absolute rate increase was 1.5 venous thromboembolic events per 10, 000 women in 1 year[i].

The latest estimates from the Committee on Safety of Medicines are that in the age group 50-59 over a five year period three in every thousand non-HRT users will suffer a venous thromboembolism. There will be an additional four cases during the same time period in HRT users. The figures for women aged 60-69 are 8 cases per 1,000 non-HRT users and an additional 9 cases for HRT users[ii]. Detailed analyses of data concerning the cardiovascular effects of HRT are currently being carried out within the Million Women Study[iii].

i. Miller J, Chan BK, Nelson HD. Postmenopausal oestrogen replacement and risk for venous thromboembolism: a systematic review and meta-analysis for the U.S. Preventive Services Task Force. *Annals of Internal Medicine* 2002; **136(9)**: 680-690
(Type I evidence - systematic review and meta-analysis of 12 studies (3 randomised controlled trials; 8 case-control studies; and 1 cohort study). Literature search to 2000)

ii. Medicines and Healthcare Products Regulatory Agency & the Committee on Safety of Medicines. *Hormone Replacement Therapy* (HRT): Latest Safety Update, 7 August 2003.
http://medicines.mhra.gov.uk/ourwork/monitorsafequalmed/safetymessages/hrtupdate803.pdf
[accessed 17.12.03]
(Type V evidence – estimates based on results from recent trials)

iii. The Million Women Study.
http://www.millionwomenstudy.org.uk/index2.html
[accessed 17.12.03]

The *statements*

The *evidence*

1.18b. Overall health **risks** exceeded benefits from use of combined **oestrogen plus progestin** among healthy postmenopausal US women and the results indicate that this regimen should **not** be initiated or continued for the primary prevention of **coronary heart disease** (CHD). The estimated hazard ratio for coronary heart disease was 1.29 (95% CI 1.02-1.63). The absolute excess risk per 10,000 person years were seven more CHD events (37 in the treatment groups versus 30 in the placebo group)[i].

A Cochrane review of **hormone replacement therapy** for preventing cardiovascular disease in post-menopausal women is currently underway[ii].

i. Writing Group for the Women's Health Initiative Investigators. Risks and benefits of oestrogen plus progestin in healthy postmenopausal women. Principal results from the women's health initiative randomised controlled trial. *Journal of the American Medical Association* 2002; **288(3)**: 321-333
http://jama.ama-assn.org/cgi/content/short/288/3/321
(Type II evidence – randomised controlled trial of 16,607 postmenopausal women (aged 50-79 years) with intact uterus at baseline assigned to conjugated equine estrogens, 0.625 mg/day, plus medroxyprogesterone acetate, 2.5 mg/day in one tablet, or placebo. The trial was stopped early, after a mean of 5.2 years of follow-up. See also guidance for women on:
http://jama.amaassn.org/issues/v288n3/fpdf/joc21036.pdf
[accessed 17.12.03]

ii. Gabriel Sanchez R, Roque M, Sanchez Gomez LM, Soares K, Calaf J, Bonfill X. Hormone replacement therapy for preventing cardiovascular disease in post-menopausal women (Protocol for a Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software (most recent update 20 May 2000)
(Type I evidence – systematic review and meta-analysis in progress)
[accessed 17.12.03]

1.19 Hyperhomocysteinemia

1.19a. Prospective studies offer weaker support than case-control studies for an association between homocyst(e)ine concentration and cardiovascular disease[i,ii]. For coronary heart disease, the summary odds ratios (OR) for a 5-micromol/l increase in **homocysteine** concentration were 1.06 (95% CI, 0.99-1.13) for cohort studies, 1.23 (1.07-1.41) for nested case-control studies, and 1.70 (1.50-1.93) for case-control studies. For cerebrovascular disease, the summary OR for a 5-micromol/l increase in homocyst(e)ine concentration were 1.10 (0.94-1.28) for cohort studies, 1.58 (1.35-1.85) for nested case-control studies, and 2.16 (1.65-2.82) for case-control studies[i]. *Although other lines of evidence support a role for homocyst (e) ine in the pathogenesis of cardiovascular disease, more information from prospective epidemiological studies or clinical trials is needed to clarify this role*[i].

Homocysteine may, however, be an indicator for unhealthy lifestyles, and therefore, an important variable for cardiologists to take into account when assessing coronary artery disease[ii].

i. Ford ES, Smith SJ, Stroup DF, Steinberg KK, Mueller PW, Thacker S. Homocyst (e) ine and cardiovascular disease: a systematic review of the evidence with special emphasis on case-control studies and nested case-control studies. *International Journal of Epidemiology* 2002; **31(1)**: 59-70
(Type I evidence - systematic review and meta-analysis of 57 publications (3 cohort studies, 12 nested case-control studies, 42 case-control studies) including 5,518 people with coronary heart disease (11,068 control subjects) and 1,817 people with cerebrovascular disease (4,787 control subjects). Literature search to 1999)

ii. Cleophas TJ, Hornstra N, van Hoogstraten B, van der Meulen J. Homocysteine, a risk factor for coronary artery disease or not? A meta-analysis. *American Journal of Cardiology* 2000; **86(9)**: 1005-9
(Type I evidence - systematic review and meta-analysis of 33 studies published between 1976 and 1999 (22 case-control and 11 cohort) of 16,097 patients. Medline search only)

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1.20 Lycopene

1.20a. In a prospective study, men in the lowest quarter of serum **lycopene** levels (≤ 0.07 micromol/L) had a 3.3 fold (95% CI 1.7-6.4, p<0.001) risk of acute coronary events of stroke compared with the others[i].

i. Rissanen TH, Voutilainen S, Nyyssönen K *et al*. Low serum Lycopene concentration is associated with an excess incidence of acute coronary events and stroke: the Kuopio Ischemic Heart Disease Risk Factor Study. *British Journal of Nutrition* 2001; **85**: 749-754

(Type IV evidence – prospective cohort study in Finland of 725 men aged 46-64 years, followed up for an average of 5.3 years)

1.21 Psychosocial factors

1.21a. **Psychological distress** may confer increased risk of coronary heart disease in men that is not explained by health behaviours, social isolation or work characteristics. Baseline psychological distress (as measured by the General Health Questionnaire, GHQ) was associated with an increased incidence of overall reported coronary heart disease (odds ratio 1.83, 95% CI 1.5-2.3) and ECG abnormalities (OR 1.51, 1.1-2.1), after adjustment for age, employment grade and length of follow-up. This risk was not consistently demonstrated in women[i].

Caveats: Coronary heart disease indicators were self-reported and the authors note that the association with ECG abnormalities was much weaker. There is also a risk that the GHQ may have been measuring sub-clinical coronary heart disease.

i. Stansfeld AS, Fuhrer R, Shipley MJ, Marmot MG. Psychosocial distress as a risk factor for coronary heart disease in the Whitehall II Study. *International Journal of Epidemiology* 2002; **31**: 248-255

(Type IV evidence – prospective cohort study, with five-year follow-up, of 10,308 civil servants aged between 35 and 55 years. The overall response rate was 73%)

1.21b. Prospective observational studies show aetiological roles for **social supports, depression** and **anxiety**, and **work characteristics** and prognostic roles for social supports and depression in coronary heart disease risk. However, conflicting data exist on whether **psychosocial** interventions reduce mortality after myocardial infarction[i].

i. Hemingway H, Marmot M. Psychosocial factors in the aetiology and prognosis of coronary heart disease: systematic review of prospective cohort studies. *British Medical Journal* 1999; **318**:1460-1467 **http://bmj.bmjjournals.com/cgi/content/full/318/7196/1460** [accessed 17.12.03]

(Type I evidence - systematic review, literature search to 1997, of 47 prospective cohort studies)

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1.21c. Among **South Asians**, the distribution of **psychosocial factors** was consistent with ethnic differences in coronary rates. Further research is required to test the extent to which psychosocial factors predict coronary events within ethnic groups and to characterize better psychosocial measures[i].

i. Hemingway H, Whitty CJ, Shipley M *et al*. Psychosocial risk factors for coronary disease in White, South Asian and Afro-Caribbean civil servants: the Whitehall II study. *Ethnicity & Disease* 2001:**11(3)**: 391-400
(Type IV evidence – cross sectional survey of 8,973 participants)

1.21d. The absence of demonstrated blood pressure lowering efficacy in intention-to-treat analyses suggests that **stress management intervention (SMI)** is an **unlikely** candidate for primary prevention of hypertension in a general population. The isolated finding of significant diastolic BP lowering in SMI participants with higher adherence provides tentative evidence of SMI BP lowering efficacy but may be a chance finding. Whether similar or other stress management interventions can produce significant BP lowering in populations selected for higher levels of BP, stress, or intervention adherence remains to be demonstrated[i].

i. Batey DM, Kaufmann PG, Raczynski JM *et al*. Stress management intervention for primary prevention of hypertension: detailed results from phase I of Trials of Hypertension Prevention (TOHP-I). *Annals of Epidemiology* 2000; **10:45**-58
(Type II evidence - randomised controlled trial of 542 healthy participants (30-54 years old) at four clinical centres. The SMI consisted of 37 contact hours in 21 groups and two individual meetings over 18 months and included: training in a number of stress and anger management methods. An intention to treat analysis was used)

1.22 Ongoing risk factor studies

1.22a. The **British Regional Heart Study (BRHS)** is a prospective study in middle-aged men drawn from general practices in 24 British towns, set up to determine the factors responsible for the considerable variation in coronary heart disease, hypertension and stroke in Great Britain. It also seeks to determine the causes of these conditions in order to provide a rational basis for recommendations towards their prevention. The first four phases of this study have been completed and phase 5, the post re-examination follow-up, is currently ongoing until 2004[i].

i. British Regional Heart Study, Department of Primary Care & Population Sciences
Royal Free & University College Medical School, Royal Free Campus
Rowland Hill Street, London NW3 2PF UK Telephone: +44 (0) 207 830 2335
http://www.ucl.ac.uk/primcare-popsci/brhs/index.htm
[accessed 17.12.03]

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1.22b. A study is underway of 14,000 cases of acute myocardial infarction (AMI) and 16,000 matched control patients from 46 countries to determine the association between **risk factors** and acute myocardial infarction within populations defined by ethnicity and/or geographic region[i].

i. Ôunpuu S, Negqassa A, Yusuf S; for the INTER-HEART Investigators. INTER-HEART: A global study of risk factors for acute myocardial infarction. *American Heart Journal* 2001; **141**: 711-721

National Service Framework

National Assembly for Wales. *Tackling CHD in Wales: Implementing Through Evidence.* Cardiff: National Assembly for Wales, July 2001

What is the current evidence for programmes at the community and individual level targeting tobacco use, diet and physical activity?

All NHS bodies and local authorities should have comprehensive tobacco policies that address: [key action 4]

- Smoking on the premises by staff, patients and visitors;
- The recording of the smoking status of all patients;
- The provision of programmes to support people to give up smoking.

What are the most effective anti-smoking policies?

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1.23 Interventions to prevent or reduce tobacco use at society/community level

1.23a. Long-term ecological evidence from many different countries consistently shows that **tobacco-advertising bans** are associated with reduced tobacco consumption. Bans in Norway (in 1975) and Finland were associated with a 9% and 7% reduction respectively, although when bans were introduced in Canada, Australia and New Zealand the reduction was less[i]. In particular, the Norwegian ban led to a substantial reduction in smoking in school students and adult men[ii]. In New Zealand heavy advertising before the ban in December 1990, was closely followed by a peak in smoking among 15 to 19 year olds in 1991 despite several preceding years of sharp decline. The peak declined by the second half of 1991. It is thought that the effect of an advertising ban interacts with concurrent measures on legislation, taxation, health promotion programmes and publicity campaigns[iii].

Department of Health. *Report of the Scientific Committee on Tobacco and Health*. London: The Stationery Office, 1998 **http://www.archive.official-documents.co.uk/document/doh/tobacco/contents.htm** [accessed 17.12.03]

(Type V evidence – expert opinion based on type IV evidence) citing the following publications:

i. Department of Health. *Effect of Tobacco Advertising on Tobacco Consumption: A Discussion Document Reviewing the Evidence*. London: Department of Health, 1992

ii. Kersler DA, Barnett PS, Witt A, Zeller MR, Mande JR, Schultz WB. The legal and scientific basis for the FDA's assertion of jurisdiction over cigarette and smokeless tobacco. *Journal of the American Medical Association* 1997; **277**: 405-9

iii. New Zealand Ministry of Health. *Tobacco Statistics 1996.*

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1.23b. There is some limited support for the effectiveness of **community interventions** in helping prevent the **uptake of smoking in young people.** Of thirteen studies which compared community interventions to no intervention controls, two, which were part of cardiovascular disease prevention programmes, reported lower smoking prevalence.

Of three studies comparing community interventions to school-based programmes only, one found differences in reported smoking prevalence. One study reported a lower rate of increase in prevalence in a community receiving a multi-component intervention compared to a community exposed to a mass media campaign alone. One study reported a significant difference in smoking prevalence between a group receiving a media, school and homework intervention compared to a group receiving the media component only[i].

i. Sowden A, Arblaster L. Community interventions for preventing smoking in young people. (Cochrane Review) In: *The Cochrane Library,* Issue 1 2003. Oxford: Update Software (most recent update 24 September 2002) **http://www.update-software.com/abstracts/ab001291.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search date to September 2002 of 17 studies. All studies used a controlled trial design, with six using random allocation of schools or communities.)

1.23c. **Mass media campaigns** can be effective in **preventing the uptake of smoking** in young people. Campaigns were more effective when they had a theoretical basis, used formative research in designing messages, and were of sufficient intensity over an extended period[i].

Caveats: Studies were in different countries. Media campaigns tend to be culturally sensitive so the response in Wales may be different.

i. Sowden AJ, Arblaster L. Mass media interventions for preventing smoking among young people. (Cochrane Review). In *The Cochrane Library* Issue 1 2003. Oxford: Update software (most recent update 19 August 1998) **http://www.update-software.com/abstracts/ab001006.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search to June 1998, of 6 controlled trials of children and young adults under 25)

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1.23d. **School smoking policies** may help reduce teenage smoking[i,ii].

There is an association between policy strength, policy enforcement and the prevalence of smoking among pupils, having adjusted for pupil level characteristics. The prevalence of daily smoking in schools with a written policy on smoking for pupils, teachers, and other adults, with no pupils or teachers allowed to smoke anywhere on the school premises was 9.5% (95% CI 6.1%-12.9%). In schools with no policy on pupils' or teachers' smoking 30.1% (23.6-36.6%) of pupils reported daily smoking. In schools with an intermediate level of smoking policy, 21.0% (17.8-24.2%) smoked every day[i].

Caveat: An effect from reverse causality (ie no smoking policies may be easier to maintain in schools where smoking is less of a problem) cannot be ruled out but an association between policy strength, enforcement and pupil behaviour is demonstrated.

There is evidence that **state wide tobacco control programmes** in the US, including a variable component of **school based cessation programmes**, increased tax levels and mass media interventions, lead to reductions in teenage smoking. One of the most critical factors was the extent of programme funding[ii].

i. Moore L, Roberts C, Tudor-Smith C. School smoking policies and smoking prevalence among adolescents: multilevel analysis of cross-sectional data from Wales. *Tobacco Control* 2001; **10**: 117-123

(Type IV evidence – cross-sectional data from surveys of 55 secondary schools in Wales, and 1,375 pupils aged 15-16)

ii. Wakefield M, Chaloupka F. Effectiveness of comprehensive tobacco control programmes in reducing teenage smoking in the USA. *Tobacco Control* 2000; **9**: 177-186

(Type IV evidence – review and narrative summary of five statewide comprehensive tobacco reduction programmes in the US)

1.23e. Conclusions from a narrative systematic review of **smoking control programmes at the workplace** were that[i]:

- Group programmes were more effective than minimal treatment programmes although less intensive treatment when combined with high participations rates can influence the total population
- Competitions have the potential to increase recruitment to smoking cessation programmes and possibly to increase cessation rates.
- The evidence is less strong that incentives will increase participation or quit rates.

Caveat: Because of the lack of experimental control, the literature was rated as weak although authors noted a strong consistency in results for reduced cigarette consumption and decreased exposure to environmental tobacco smoke at work.

i. Eriksen MP, Gottlieb NH. A review of the health impact of smoking control at the workplace. *American Journal of Health Promotion* 1998; **13(2)**: 83-104

(Type I evidence – systematic review, literature search to 1994, of 52 smoking cessation programmes (including 23 randomised controlled trials) and 29 worksite smoking policy interventions)

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1.23f. Employees in workplaces with **smoking bans** (all of which were hospitals) had higher rates of smoking cessation than employees where smoking was permitted, but relapse was similar between the two groups. Differences in the post-ban quit ratio were observed between intervention and comparison groups ($p \leq 0.02$). For employees whose bans were implemented at least seven years before survey, the post-ban quit ratio was estimated at 0.256, compared with 0.142 for employees in non-smoke-free workplaces ($p=0.02$). After controlling for a variety of factors, time to quit smoking was shorter for the hospital employees ($p<0.001$), with an overall relative risk of quitting of 2.3. Contrary to expectations, relapse rates were similar between groups[i].

Caveats: There were some baseline differences between hospital and non-hospital employees but attempts were made to control for socio-economic differences. Quit rates were not independently assessed and may have been subject to recall and/or reporting bias.

i. Longo DR, Johnson JC, Kruse RL, Brownson RC, Hewett JE. A prospective investigation of the impact of smoking bans on tobacco cessation and relapse. *Tobacco Control* 2001; **10**: 267-272

(Type IV evidence – natural prospective cohort study of 1,033 current or former smokers employed in smoke-free hospitals (intervention group) and 816 current or former smokers employed in non-smoke-free workplaces (comparison group). Both hospitals and employees were randomly selected from 12 strata based on hospital size and state tobacco regulations. Surveys were carried out in 1994 and each of the following two years. Response rates were lower in the community than the hospital cohorts (response rates at surveys 1, 2 and 3 were 84%, 69% and 59% in the hospital employees compared with 66%, 57% and 48% in the community employees)

1.23g. **Smoke-free workplaces** not only protect non-smokers from passive smoking but also encourage smokers to quit or reduce their consumption. Totally smoke-free workplaces are associated with reductions in prevalence of smoking of 3.8% (95% CI, 2.8% -4.7%) and 3.1 (2.4 -3.8) fewer cigarettes smoked per day per continuing smoker. Combination of the effects of reduced prevalence and lower consumption per continuing smoker yields a mean reduction of 1.3 cigarettes per day per employee, which corresponds to a relative reduction of 29%[i].
Caveat: The lack of unpublished studies may introduce bias but there was no evidence of publication bias from funnel plots.

i. Fichtenberg CM, Glantz SA. Effect of smoke-free workplaces on smoking behaviour; systematic review *British Medical Journal* 2002; **325 (7357)**: 188-194 **http://bmj.bmjjournals.com/cgi/content/full/325/7357/188** [accessed 17.12.03]

(Type IV evidence - systematic review of observational studies. 26 studies reported in 24 papers. Worksite studies measured changes in smoking that accompanied regulations in individual workplaces assessed prospectively, in sequential cross sections, or retrospectively).

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1.23h. **Population-based interventions** generally attract 1-2% of the target population but these rates can be potentially increased by the use of innovative recruitment techniques. No specific type of recruitment strategy was shown to be consistently more effective than others. Quit rates among participants may initially be high (mean quit rate of 34% at one-month follow-up) but these decrease over time (mean rate of 23% at one year)[i].

Caveats: Incentive-based smoking cessation programmes may attract only smokers who are motivated to quit. Methodological differences among existing studies make them difficult to compare and interpret.

i. Bains N, Pickett W, Hoey J. The use and impact of incentives in population-based smoking cessation programs: a review. *American Journal of Health Promotion* 1998; **12(5)**: 307-320

(Type III evidence – systematic review, literature search to Spring 1997, of 17 quasi experimental and observational studies)

1.23i. **Interventions with retailers** can lead to large decreases in the number of outlets selling tobacco to youths. However, few of the communities studied in this review achieved sustained levels of high compliance. In three controlled trials, there was little effect of intervention on youth perceptions of access to, or prevalence of, smoking[i].

i. Stead LF, Lancaster T. Interventions for preventing tobacco sales to minors. (Cochrane Review) In *The Cochrane Library* Issue 1 2003 Oxford: Update Software (most recent update 12 October 2001)

http://www.update-software.com/abstracts/ab001497.htm [accessed 17.12.03]

(Type I evidence – systematic review, literature search to October 2001, of 30 studies of which some were randomised controlled trials)

1.23j. Carefully planned and resourced **multicomponent strategies** effectively reduced smoking within public places. Less comprehensive strategies were less effective[i].

Caveats: All the studies used relatively weak experimental designs. Most studies were done in the USA and there is a need to identify ways in which these strategies can be adopted and used in countries with different attitudes to tobacco use. *Future studies should also consider the use of more rigorous experimental designs*[i].

i. Serra C, Cabezas C, Bonfill X, Pladevall-Vila M. Interventions for preventing tobacco smoking in public places (Cochrane Review). In. *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 27 April 2000)

http://www.update-software.com/abstracts/ab001294.htm [accessed 17.12.03]

(Type III evidence – systematic review, literature search to 1999, of eleven uncontrolled before and after studies)

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1.23k. **Summaries** of systematic reviews and other evidence of interventions to reduce tobacco use at community and individual levels are available[i] or in preparation[ii].

i. *Health Evidence Bulletins Wales: Healthy Living*. Chapter 1. Smoking. Cardiff: National Assembly for Wales, 2000 **http://hebw.uwcm.ac.uk/healthyliving/chapter1.html** [accessed 17.12.03]
(Summaries of quality appraised research evidence from a review of the literature completed in July 1999)

ii. Health Development Agency. *A Review of the Effectiveness of Public Health Interventions to Reduce Smoking Initiation and Increase Smoking Cessation: A Synthesis of the Evidence from Systematic Reviews and Meta-analyses*. London: Health Development Agency. **http://www.hda-online.org.uk/evidence/EBBD.html#pub** [accessed 17.12.03]
(Evidence Briefing Document in preparation)

1.24 Interventions to prevent or reduce tobacco use in hospitals

1.24a. Studies comparing a **nursing intervention** to a control or usual care found intervention to significantly increase the odds of quitting (odds ratio 1.50, 95% CI 1.29-1.73)[i]. **Caveat:** There was heterogeneity between the study results but pooling using a random effects model did not alter the estimate of effect.

i. Rice VH, Stead LF. Nursing interventions for smoking cessation. (Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software (most recent update 15 May 2001) **http://www.update-software.com/abstracts/ab001188.htm** [accessed 17.12.03]
(Type I evidence – systematic review, literature search of the Cochrane Tobacco Addition Group register to April 2000, of 16 randomised trials)

1.25 Interventions to prevent or reduce tobacco use at the individual level

1.25a. All of the commercially available forms of **Nicotine Replacement Therapy** (nicotine gum, transdermal patch, the nicotine nasal spray, nicotine inhaler and nicotine sublingual tablets/lozenges) are effective as part of a strategy to promote smoking cessation. They increase quit rates approximately 1.5 to 2 fold regardless of setting.

The effectiveness of NRT appears to be largely independent of the intensity of additional support provided to the smoker. Provision of more intense levels of support, although beneficial in facilitating the likelihood of quitting, is not essential to the success of NRT[i].

i. Silagy C, Lancaster T, Stead L, Mant D, Fowler G. Nicotine replacement therapy for smoking cessation (Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 20 August 2002) **http://www.update-software.com/abstracts/ab000146.htm** [accessed 17.12.03]
(Type I evidence – systematic review, literature search of the Cochrane Tobacco Addition Group register to July 2002, of 110 trials)

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1.25b. Systematic reviews have found that quit rates are significantly increased by the antidepressant **buproprion (Zyban)**[i]. The odds ratio of quitting with bupropion versus placebo was 2.54, 95% CI 1.90 -3.41; NNT 10). Quit rates are **not** increased by the use of **moclobemide** or **anxiolytics**[i].

i. Thorogood M, Hillsdon M, Summerbell C. Cardiovascular disorders. Changing behaviour. *Clinical Evidence* January 2003. London: BMJ Publishing Group, 2003

(Type I evidence – summary of systematic reviews from a review of the literature completed in May 2002)

1.25c. **Smoking cessation counselling** can assist smokers to quit. Individual counselling was more effective than control (odds ratio for successful smoking cessation = 1.62, 95% CI 1.35-1.94). Reviewers failed to detect a greater difference of intensive counselling compared to brief counselling (OR=0.98, 0.61-1.56)[i].

i. Lancaster T, Stead LF. Individual behavioural counselling for smoking cessation. (Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 8 April 2002) **http://www.update-software.com/abstracts/ab001292.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search of the Cochrane Tobacco Addiction Trials Register to February 2002, of 18 randomised or quasi randomised trials)

1.25d. **Simple physician advice** has a small effect on smoking cessation rates. Pooled data from 16 trials of brief advice versus no advice (or usual care) revealed a small but significant increase in the odds of quitting (odds ratio 1.69, 95% CI 1.45-1.98; absolute difference in the cessation rate ca 2.5%). More intensive interventions are marginally more effective than minimal interventions (odds ratio for intensive versus minimal intervention, 1.44, 95% CI 1.23-1.68)[i].

The community pharmacy-based PAS smoking cessation service can be an effective method of helping people to stop smoking when delivered by pharmacists willing to adopt this approach. Of smokers in the PAS group, 14.3% were abstinent up to 12 months compared with 2.7% in the control group (p<0.001 for the difference)[ii].

i. Silagy C, Stead LF. Physician advice for smoking cessation. (Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 31 January 2001) **http://www.update-software.com/abstracts/ab000165.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search of the Cochrane Tobacco Addition Group register to October 2000, of 34 randomised trials including over 27,000 smokers)

ii. Maguire TA, McElnay JC, Drummond A. A randomized controlled trial of a smoking cessation intervention based in community pharmacies. *Addiction* 2001; **96(2)**: 325-331

(Type II evidence – randomised controlled trial of 484 smokers enrolled by pharmacists and assigned to a structure community pharmacy-based smoking cessation programme (the PAS model) or ad hoc advice only (the control group))

1.25e. The evidence would suggest that in the context of a **smokers' clinic** the use of **buddies** may be of some benefit but the research methodology in many cases was poor. Two of the included studies showed a significant benefit of the intervention in the short term. *This is an important area for future research*[i]

i. May S, West R. Do social support interventions ("buddy systems") aid smoking cessation? A review. *Tobacco Control* 2000; **9**: 415-422

(Type I evidence – systematic review, literature search date not given, of 10 randomised controlled trials)

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1.25f. There is no clear evidence that **acupuncture; acupressure, laser therapy** or **electro-stimulation** is effective for smoking cessation. Acupuncture was not superior to sham acupuncture in smoking cessation at any time point. The odds ratio for early outcomes was 1.22 (95% CI 0.99-1.49), after six months it was 1.22 (95% CI 0.99-1.49) and after 12 months, 1.08 (95% CI 0.77-1.52)[i].

i. White AR, Rampes H, Ernst E. Acupuncture for smoking cessation. (Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 18 February 2002) **http://www.update-software.com/abstracts/ab000009.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search to January 2002, of 22 randomised controlled trials)

1.25g. Standard **self-help** materials may increase quit rates compared to no intervention, but the effect is likely to be small. In 11 trials of self-help compared to no intervention there was a pooled effect that just reached statistical significance (odds ratio 1.24, 95% CI 1.07-1.45). No evidence was found that self-help interventions had an additional benefit when used alongside other interventions such as advice from a health care professional or nicotine replacement therapy. There is evidence that materials that are tailored for individual smokers are more effective when compared to standard materials (OR 1.36, 95% CI 1.13-1.64) or no materials (OR 1.80, 95% CI 1.46-2.23)[i].

i. Lancaster T, Stead LF. Self-help interventions for smoking cessation. (Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 11 April 2002) **http://www.update-software.com/abstracts/ab001118.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search to March 2002, of 51 trials)

1.25h. Only one of eight trials in a systematic review of **exercise interventions** for smoking cessation offered evidence for exercise aiding smoking cessation. All but one of the other trials was too small to exclude reliably an effect of intervention[i]. *Trials are needed with larger sample sizes, equal contact control conditions, tailored and lifestyle exercise programmes and measures of exercise adherence*[i].

i. Ussher MH, West R, Taylor AH, McEwen A. Exercise interventions for smoking cessation (Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 20 May 2002) **http://www.update-software.com/abstracts/ab002295.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search of the Cochrane Tobacco Addition Group register to March 2002, of eight trials, six of which had fewer than 25 people in each treatment arm)

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1.25i. **Proactive telephone counselling** can be effective compared to an intervention without personal contact. Successful interventions generally involve multiple contacts timed around a quit attempt. The available evidence neither confirms nor rules out a benefit of telephone counselling as an adjunct to face to face counselling or pharmacotherapy. Further trials randomising access to helplines are unlikely to be done but indirect evidence suggests they can be a useful part of a smoking cessation service.
Meta-analysis using all less intensive intervention arms as the control removed the heterogeneity and suggests that telephone counselling compared to less intensive intervention increases quit rates (OR 1.56, 95% CI 1.38 - 1.77)[i].

1.25j. **Groups** are better than self-help (odds ratio 1.97, 95% CI 1.57-2.48) and other less intensive interventions for smoking cessation. Compared to no intervention controls the odds ratio was 2.19 (1.42-3.37). There is not enough evidence on their effectiveness, or cost-effectiveness, compared to intensive individual counselling. The inclusion of skills training to help smokers avoid relapse appears to be useful although the evidence is limited. There is not enough evidence to support the use of particular components in a programme beyond the support and skills training normally included[i].

1.25k. The existing studies provide insufficient evidence to determine the efficacy of **rapid smoking** for smoking cessation, or whether there is a dose response to **aversive stimulation**. *Rapid smoking is an unproven method with sufficient indications of promise to warrant evaluation*[i].

The *evidence*

i. Stead LF, Lancaster T, Perera R. Telephone counselling for smoking cessation (Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 15 October 2002)
http://www.update-software.com/abstracts/ab002850.htm [accessed 17.12.03]
(Type I evidence – systematic review, literature search to September 2002, of 23 randomised or quasi randomised trials)

i. Stead LF, Lancaster T. Group behaviour therapy programmes for smoking cessation. (Cochrane Review) In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 1 May 2002)
http://www.update-software.com/abstracts/ab001007.htm [accessed 17.12.03]
(Type I evidence – systematic review, literature search to December 2001, of 52 randomised trials)

i. Hajek P, Stead LF. Aversive smoking for smoking cessation. (Cochrane Review) In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 29 May 2001)
http://www.update-software.com/abstracts/ab000546.htm [accessed 17.12.03]
(Type I evidence – systematic review, literature search to May 2001 of 25 randomised controlled trials)

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1.25l. Two systematic reviews have found that antismoking interventions in **pregnant women** increase abstinence rates. The increase in abstinence with non-nicotine replacement interventions was similar to the increase found in trials of nicotine replacement in men and non-pregnant women[i].

i. Thorogood M, Hillsdon M, Summerbell C. Cardiovascular disorders. Changing behaviour. *Clinical Evidence* January 2003. London: BMJ Publishing Group, 2003
(Type I evidence - summary of systematic reviews from a review of the literature completed in May 2002)

1.26 Guidelines - Interventions to reduce tobacco use

1.26a. **Guidelines** on interventions to reduce tobacco use are available[i,ii,iii,iv].

i. Hopkins DP, Briss PA, Ricard CJ *et al*. Reviews of evidence regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke. *American Journal of Preventive Medicine* 2001; **20(2S)** **http://www.thecommunityguide.org/tobacco/tobac-AJPM-evrev.pdf** [accessed 17.12.03]
(Type I evidence – systematic review, literature search to August 1999 plus articles in 2000 noted by members of the development team)

ii. *Institute for Clinical Systems Improvement (ICSI) Tobacco Use Prevention and Cessation for Adults and Mature Adolescents*. Bloomington: ICSI, August 2001. **http://www.guideline.gov/algorithm/2248/FTNGC-2248.pdf** [accessed 17.12.03]
(Evidence based guidelines, literature search date unknown)

iii. Recommendations regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke. *American Journal of Preventive Medicine* 2001; **20(2 Suppl)**: 10-5 **http://www.thecommunityguide.org/tobacco/tobac-AJPM-recs.pdf** [accessed 17.12.03]
(Evidence based guidelines)

iv. West R, McNeill A, Raw M. Smoking cessation guidelines. *Thorax* 2000; **55(12)**: 987-999
(Expert consensus guidelines. Recommendations are not all directly linked to the evidence and, although recommendations are graded according to the type of supporting evidence, the evidence type is not confirmed)

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1.27 Interventions to reduce overweight and obesity and promote healthy eating at society/community level

1.27a. **Summaries of systematic reviews** and other evidence of effective interventions to reduce overweight and obesity and to promote healthy eating at community and individual levels are available[i,ii].

i. Mulvihill C, Quigley R. *The Management of Obesity and Overweight. An Analysis of the Reviews of Diet, Physical Activity and Behavioural Approaches*. London: Health Development Agency, 2003
http://194.83.94.67/niche_docs/EB_DATABASE_CONTENT/HTML_database_content/EBBD-Obesity.html
[accessed 17.12.03]
(Systematic review of reviews)

ii. NHS Centre for Reviews & Dissemination. The prevention and treatment of childhood obesity. *Effective Healthcare* 2002; **7(6)**
http://www.york.ac.uk/inst/crd/ehc76.pdf
[accessed 17.12.03]
(Summary of two Cochrane reviews and an update literature search; literature date & details not provided but available on request from the NHS CRD)

1.27b. The Food Standards Agency has commissioned the Bangor Food Research Unit to coordinate and evaluate the **Food Dudes** programme in Schools across Wales. The programme adopts three techniques - taste exposure, modelling (to encourage imitation of a well liked person) and rewards to persuade children to increase their consumption of fruit and vegetables. Pilot studies are promising but are, to date, published as abstracts only[i].

A randomised controlled trial of **fruit tuck shops** in primary schools[ii] demonstrated no increase in fruit consumption[iii].

i. Tapper K, Horne PJ, Lowe CF. The Food Dudes to the rescue! *The Psychologist* 2003; **16(1)**: 18-21
http://www.fooddudes.co.uk [accessed 17.12.03]
(Type V evidence – expert summary of the development and evaluation of the Food Dudes programme)

ii. Moore L, Paisly CM, Dennehy A. Are fruit tuck shops in primary schools effective in increasing pupils' fruit consumption? A randomised controlled trial. *Nutrition & Food Science* 2000; **30(1)**: 35-38
(Methodology for a randomised controlled trial carried out in 43 schools in South West England and Wales during 1999/2000)

iii. Moore L. Are fruit tuck shops in primary schools effective in increasing pupils fruit consumption? A randomised controlled trial. 2001
(Abstract only available)
http://www.cf.ac.uk/socsi/whoswho/moore-tuckshop.html
[accessed 17.12.03]

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1.27c. In a survey following the BBC's **'Fighting Fat, Fighting Fit'** campaign 57% of respondents had heard of the campaign and 30% recalled the healthy lifestyle messages, although fewer than 1% registered to participate in the scheme[i].

Of the small percentage that registered in the scheme, participants in a questionnaire study reported significant reductions in weight, and in fat and snack intake, and significant increases in exercise levels and fruit, vegetable and starch intake during the six-months of the campaign[ii].

Caveat: There is potential for bias in this study of self-reported outcomes from a self-selected group.

i. Wardle J, Rapoport L, Miles A, Afuape T, Duman M. Mass education for obesity prevention: the penetration of the BBC's 'Fighting Fat, Fighting Fit' campaign. *Health Education Research* 2001; **16(3)**: 343-355

(Type IV evidence – cross-sectional survey (part of the ONS Omnibus survey) in March 1999 following a seven-week television and radio campaign in late 1998 and early 1999. 1,894 people were interviewed; 70% of the 2,690 eligible addresses from random sampling of the UK population)

ii. Miles A, Rapoport L, Wardle J, Afuape T, Duman M. Using the mass media to target obesity: an analysis of the characteristics and reported behaviour change of participants in the BBC's 'Fighting Fat, Fighting Fit' campaign. *Health Education Research* 2001; **16(3)**: 357-372

(Type IV evidence – questionnaire survey of 6000 adults registering with the campaign, at the start of the campaign and five months later. 3661 respondents completed the baseline questionnaire and 2112 (58%) completed the follow-up evaluation)

1.28 Interventions to reduce overweight and obesity and promote healthy eating at the individual level
See also statement 1.27a

1.28a. Three of the four long-term studies that combined **dietary education and physical activity interventions** resulted in no difference in overweight, whereas one study reported a difference in favour of the intervention group. In two studies of dietary education alone, a multimedia action strategy appeared to be effective but other strategies did not. The one long-term study that only focussed on physical activity resulted in a slightly greater reduction in overweight in favour of the intervention group, as did two short-term studies of physical activity[i].

i. Campbell K, Waters E, O'Meara S, Kelly S, Summerbell C. Interventions for preventing obesity in children. (Cochrane Review). In: *The Cochrane Library* Issue 1 2003. Oxford: Update Software. (most recent update 4 January 2002). **http://www.update-software.com/abstracts/ab001871.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search to July 2001, of 10 randomised and non-randomised studies)

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1.28b. Findings from trials longer than two years of **reduced or modified dietary fat intake** are suggestive of a small but potentially important reduction in cardiovascular risk[i,ii]. There was no significant effect on total mortality (rate ratio=0.98, 95% CI 0.86-1.12), a trend towards protection from cardiovascular mortality (rate ratio=0.91, 95% CI 0.77-1.07), and significant protection from cardiovascular events (rate ratio=0.84, 95% CI 0.72-0.99). The latter became non-significant on sensitivity analysis[i].

Fish consumption is not associated with reduced coronary heart disease mortality in low-risk populations. However, fish consumption at 40-60 g daily is associated with markedly reduced coronary heart disease mortality in high-risk populations. The underlying biochemical mechanism is not known and causal inference premature[iii].

A Cochrane review is underway to look at the effect of **omega-3 fatty acids** (eg fish oil) in the prevention of cardiovascular disease[iv].

The *evidence*

i. Hooper L, Summerbell CD, Higgins JPT *et al*. Reduced or modified dietary fat for preventing cardiovascular disease. (Cochrane Review). In: *The Cochrane Library* Issue 1 2003. Oxford: Update Software. (most recent update 30 May 2001). **http://www.update-software.com/abstracts/ab002137.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search to May 1999, of 27 randomised controlled trials (40 intervention arms, 30,901 person years) comparing interventions to reduce or modify fat or cholesterol intake)

ii. Astrup A, Grunwald GK, Melanson EL, Saris WHM, Hill JO. The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *International Journal of Obesity* 2000; **24**: 1545-1552

(Type I evidence – systematic review, literature search to July 1999, of 16 trials and 1,910 subjects)

iii. Marckmann P, Gronbaek M. Fish consumption and coronary heart disease mortality. A systematic review of prospective cohort studies. *European Journal of Clinical Nutrition* 1999; **53(8)**: 585-590

(Type IV evidence – systematic review of 11 studies and 116,764 individuals in all)

iv. Hooper L, Thompson R, Harrison R *et al*. Omega-3 fatty acids for prevention of cardiovascular disease risk (Protocol for a Cochrane Review). In: *The Cochrane Library* Issue 1 2003. Oxford: Update Software.

(Ongoing systematic review)

1.28c. **Fat-restricted diets** are only as effective as calorie-restricted diets in achieving long-term weight loss in overweight or obese people. There was no significant difference in weight loss between the two groups at 12 and 18 months[i].

i. Pirozzo S, Summerbell C, Cameron C, Glasziou P. Advice on low-fat diets for obesity (Cochrane Review). In: *The Cochrane Library* Issue 1 2003. Oxford: Update Software. (most recent update 25 February 2002). **http://www.update-software.com/abstracts/ab003640.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search to February 2002, of four randomised controlled trials comparing low-fat diets with other weight reducing diets)

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1.28d. At present there are few solid leads about **improving obesity management**, *although reminder systems, brief training interventions, shared care, in-patient care and dietician-led treatments may all be worth further investigation*[i].

i. Harvey EL, Glenny A-M, Kirk SFL, Summerbell CD. Improving health professionals' management and the organisation of care for overweight and obese people (Cochrane Review). In: *The Cochrane Library* Issue 1 2003. Oxford: Update Software. (Latest update 23 July 2001). **http://www.update-software.com/abstracts/ab000984.htm** [accessed 17.12.03]

(Type I evidence – systematic review, literature search to April 2000, of 18 studies, including some randomised controlled, involving 446 providers and 4,158 patients)

1.28e. **Psychological interventions** to prevent weight gain exhibited various degrees of effectiveness and only one of five studies with a randomised controlled design reported a significant effect on weight[i].

i. Hardeman W, Griffin S, Johnston M, Kinmouth AL, Wareham NJ. Interventions to prevent weight gain: a systematic review of psychological models and behaviour change methods. *International Journal of Obesity* 2000; **24**: 131-143

(Type I evidence – systematic review, search date not given, of 11 randomised controlled and other intervention studies)

1.28f. Prospective studies with physical activity measured at baseline gave inconsistent results regarding the effects of **increased physical activity** on weight change. The weighted mean weight regain in randomised studies with or without exercise training was 0.28 and 0.33 kg/month respectively. Based on observational studies, it seemed that an actual increase in energy expenditure of physical activity of approximately 1,500-2,000 kcal/week is associated with improved weight maintenance. This is more than was prescribed in most randomised trials, and certainly more than the participants actually achieved. Adherence to a prescribed exercise programme remains a big challenge[i].

i. Fogelholm M, Kukkonen-Harjula K. Does physical activity prevent weight gain - a systematic review. *Obesity Reviews* 2000; **1**: 95-111

(Type I evidence – systematic review, literature search to early 2000, of 46 interventional and observational studies)

1.28g. A Cochrane review is underway to evaluate the effectiveness of **dietary interventions** in obtaining sustained desirable dietary changes or improved cardiovascular risk profile among healthy free-living adults[i].

i. Brunner EJ, Thorogood M. Dietary interventions for reducing cardiovascular risk (Protocol for a Cochrane Review). In: *The Cochrane Library* Issue 1 2003. Oxford: Update Software.

(Ongoing systematic review)

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1.28h. A Cochrane review is underway to assess the association between **fruit and vegetable intake** and cardiovascular morbidity and mortality, and all cause mortality, using all available cohort studies[i].

i. Ness A, Hooper L, Egger M, Powles JW, Davey Smith G. Fruit and vegetables for cardiovascular disease. (Protocol for a Cochrane Review). In: *The Cochrane Library* Issue 1 2003. Oxford: Update Software.
(Ongoing systematic review)

1.29 Guidelines for the reduction of overweight and obesity and the promotion of healthy eating

1.29a. **Guidelines** are available for the identification, evaluation and treatment of overweight and obesity in adults[i,ii]

i. National Institutes of Health. *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Executive Summary*. Bethesda: National Institutes of Health, 1998
http://www.nhlbi.nih.gov/guidelines/obesity/ob_exsum.pdf [accessed 17.12.03]
(Evidence based guidelines)

ii. *Prodigy Clinical Recommendation - Obesity*
http://www.guidance.prodigy.nhs.uk/Obesity
[accessed 17.12.03]
(Type V evidence – expert guidance based on a review of the evidence)

1.29b. **Guidelines** for the management of obesity in **children** and young people are available[ii].
Caveat: None of the recommendations are graded above level D (evidence from non-analytic studies and expert opinion)

i. Scottish Intercollegiate Guidelines Network. *Management of obesity in children and young people*. Guideline No. 69. Edinburgh: SIGN, April 2003
http://www.sign.ac.uk/pdf/sign69.pdf [accessed 17.12.03]
http://www.show.scot.nhs.uk/sign/pdf/sign69.pdf
[accessed 17.12.03]
(Type IV evidence – systematic review, literature search to December 2001)

1.29c. **Guidelines** on diet and nutrition are available[i,ii]

i. Krauss RM, Eckel RH, Howard B *et al*. AHA dietary Guidelines Revision 2000: A Statement for Healthcare Professionals From the Nutrition Committee of the American Heart Association. *Circulation* 2000; **102**: 2284-2299
http://circ.ahajournals.org/cgi/reprint/102/18/2284.pdf
[accessed 17.12.03]
(Type V evidence – expert guidance based on a review of the evidence)

ii. Stockley L. Towards public health nutrition strategies in the European Union. To implement food based dietary guidelines and to enhance healthier lifestyles. *Public health Nutrition* 2000; **4(2A)**: 307-324
(Type V evidence – expert guidance based on a review of the evidence)

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1.30 Interventions to promote physical activity among the adult population

1.30a. **Primary care based physical activity counselling** is moderately effective in the short term. Studies in which the interventions were tailored to participant characteristics and which offered written material to patients produced stronger results. Questions remain about the consistency of implementation and long-term maintenance of outcomes[i].
Caveat: There was considerable variability across studies.

A review of reviews is in preparation[ii].

i. Eakin EG, Glasgow RE, Riley KM. Review of primary care-based physical activity intervention studies: effectiveness and implications for practice and future research. *Journal of Family Practice* 2000; **49**: 158-68.
(Type I evidence – systematic review, literature search to 1998, of 15 controlled trials including nine randomised controlled trials)

ii. Hillsdon M, Foster C, Naidoo B. *The Evidence on the Effectiveness of Public Health Interventions for Increasing Physical Activity Amongst Adults: A Synthesis of Evidence from Systematic Reviews and Meta-analyses.* London: Health Development Agency.
http://www.hda-online.org.uk/evidence/EBBD.html#pub
[accessed 17.12.03]
(Evidence Briefing Document in preparation)

1.30b. In a systematic review of interventions in adults aged over 40 years a variety of home based, group based or education based interventions were shown to increase **physical activity** but the changes were relatively small and short lived. However, this study did not explore the effect of exercise on health gain outcomes[i].

i. Van der Bij AK, Laurant MGH, Wensing M. Effectiveness of physical activity interventions for older adults - A review. *American Journal of Preventive Medicine* 2002; **22(2)**: 120-133.
(Type I evidence – systematic review of 38 studies)

1.30c. The results of a longitudinal survey **do not** provide any evidence that the ***ACTIVE* for LIFE** campaign improved physical activity, either overall or in any subgroup[i].

38% of those surveyed were aware of the main advertising images, assessed six to eight months after the main television advertisement. The proportion of participants knowledgeable about moderate physical activity recommendations increased by 3.0% (95% CI, 1.4%-4.5%) between waves 1 and 2 and 3.7% (95% CI 2.1%-5.3%) between waves 1 and 3. The change in the proportion of active people between baseline and waves 1 and 2 was –0.02 (95% CI –2.0 to +1.7) and between waves 1 and 3 was –9.8 (-7.9 to –11.7)[i].

Caveats: 48% of participants provided data at all three time points (the 'completers') and there were baseline differences between completers and non-completers. This, and the very large loss to follow-up could have introduced bias.

i. Hillsdon M, Cavill N, Nanchahal K, Diamond A, White IR. National level promotion of physical activity: Results from England's *ACTIVE* for LIFE campaign. *Journal of Epidemiology and Community Health* 2001; **55**: 755-761
(Type IV evidence – prospective longitudinal survey of a nationally representative sample of 3,189 adults aged 16-74 years to assess the impact of a national multi-component campaign. The campaign aimed to increase knowledge and acceptability of the recommendation that *"adults should aim to take part in at least 5 sessions of 30 minutes of moderate intensity physical activity per week"*. Data were collected at baseline (wave 1), one (wave 2) and two-years (wave 3) later)

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1.30d. The typical **work-site intervention** has yet to demonstrate a statistically significant increase in physical activity or fitness. The mean effect size was heterogeneous and small (r=0.11, 95% CI –0.20 to 0.40). Effects were smaller in randomised studies compared with studies using quasi-experimental designs[i].

The *evidence*

i. Dishman RK, Oldenburg B, O'Neal H, Shephard RJ. Worksite physical activity interventions. *American Journal of Preventive Medicine* 1998; **15:**344-361

(Type I evidence – systematic review, literature search to August 1997, of 26 randomised and non randomised studies, & nearly 9,000 subjects)

1.30e. A review summarising studies of physical activity interventions targeting **older adults** is available. No clear practice implications are provided but *there are recommendations for further research*[i].

i. King AC, Rejeski WJ, Buchner DM. Physical activity interventions targeting older adults. A critical review and recommendations. *American Journal of Preventive Medicine* 1998; **15**: 316-33.

(Type I evidence – systematic review, literature search date unclear, of 29 randomised and quasi-randomised studies of interventions in adults aged 50 years and older)

1.30f. Intervention in **health care settings** can increase physical activity for primary prevention. Three of four randomised studies with short-term measurement (four weeks to three months) and two of five randomised studies with long-term measurement (six months) achieved significant effects on physical activity[i].

A more recent systematic review concluded that advice in **routine primary care consultations** is not an effective means of producing sustained increases in physical activity. The two randomised controlled trials had negative short and long-term results[ii].
Caveat: The majority of included trials were in the United States and may not be generalisable to the UK. *Quality research in UK primary care would be valuable.*

i. Simons-Morton DG, Calfas KJ, Oldenburg B, Burton NW. Effects of interventions in health care settings on physical activity or cardio respiratory fitness. *American Journal of Preventive Medicine* 1998; **15**: 413-430.

(Type I evidence – systematic review, literature search to 1997, of 12 studies of primary prevention of which seven were randomised, and 24 randomised studies of secondary prevention)

ii. Lawlor DA, Hanratty B. The effect of physical activity advice given in routine primary care consultations: a systematic review. *Journal of Public Health Medicine* 2001; **23**: 219-226.

(Type I evidence – systematic review, literature search to December 2000, of eight randomised or quasi-experimental trials and 4,747 participants)

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The *evidence*

1.30g. Among women, adherence to lifestyle guidelines involving **diet, exercise**, and abstinence from **smoking** is associated with a very low risk of coronary heart disease[i].

Caveat: This analysis has several important limitations. Despite the large numbers of subjects and the long follow-up, the estimates were somewhat imprecise, largely because there were few cases of coronary heart disease among women in the low-risk categories. This study could not provide reliable estimates on which to base more stringent recommendations because of the small number of cases[i].

i. Stampfer MJ, Hu FB, Manson JE, Rimm EB, Willett WC. Primary prevention of coronary heart disease in women through diet and lifestyle. *New England Journal of Medicine* 2000; **343 (1)**: 16-22

(Type IV evidence - cohort study of 84,129 women participating in the Nurses' Health Study (aged 30-55 years at baseline) with 14 years of follow-up)

National Service Framework

National Assembly for Wales. *Tackling CHD in Wales: Implementing Through Evidence*. Cardiff: National Assembly for Wales, July 2001

It is recognised that it is important to influence young people from an early age to live healthy lives. This means the appropriate encouragement to take exercise which needs to be facilitated by the provision of sports facilities where accessible. [key paragraph 4.3]

What are the best ways of encouraging young people to take more exercise?

Does the provision of more/free sports facilities increase the average exercise level of children?

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1.31 Encouraging young people to be physically active

1.31a. Consensus guidance is available on health enhancing **physical activity** for young people[i].

i. Cavill N, Biddle S, Sallis JF. Health enhancing physical activity for young people: Statement of the United Kingdom expert consensus conference. *Paediatric Exercise Science* 2001; **13(1)**: 12-25

(Type V evidence – expert consensus guidance)

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1.31b. **School based programmes** can reduce cardiovascular risk factors and **increase exercise levels** in the short term although the effects have not yet been shown to be sustained and the effect sizes are small[i,ii,iii].

Two recent systematic reviews of barriers to, and facilitators of, physical activity in children aged 4-10[iv] and in young people aged 11-16[v] suggested that interventions can lead to positive changes.

For 4-10 year olds, education and provision of equipment for monitoring and reducing TV, video-tape and video-game use appear to be promising population based approaches, as do multi-component interventions set in schools, homes and the wider community[iv].

For 11-16 year olds, only four rigorous outcome evaluations were identified but these showed some effect on increasing participation, particularly for young women. A 'whole school' approach (involving all members of the school community) can promote greater involvement in physical activity, as can peer-led initiatives, particularly where peers lobby for environmental changes throughout the school (although these may be more beneficial for promoting healthy eating). Adequate changing rooms and appropriate gym kit make physical education more appealing, particularly to young women[v].
Caveats: Both the above reviews found few rigorously evaluated interventions. Children and young people have clear views on the barriers to, and facilitators of, their participation in physical activity but their views rarely informed the development of interventions.

The *evidence*

i. Nader PR, Stone EJ, Lytle LA *et al*. Three year maintenance of improved diet and physical activity: the CATCH cohort. *Archives of Paediatrics and Adolescent Medicine* 1999; **153(7)**: 695-704.

(Type II evidence - Follow-up of the 4-center, randomized, controlled field trial (CATCH) with 56 intervention and 40 control elementary schools. 3,714 (73%) of the initial CATCH cohort of 5,106 students from ethnically diverse backgrounds in California, Louisiana, Minnesota, and Texas at grades 6, 7, and 8 were studied)

ii. Harrell JS, McMurray RG, Gansky SA, Bangdiwala SI, Bradley CB. A public health vs a risk-based intervention to improve cardiovascular health in elementary school children: The Cardiovascular Health in Children study. *American Journal of Public Health* 1999; **89(10)**: 1529-1535.

(Type II evidence – randomised controlled field trial of 18 schools or school clusters in North Carolina. The stratified schools or clusters were randomised into a classroom-based intervention, a risk-based intervention, given to small groups of children with identified risk factors, or a control group)
also in: Harrel JS, Gansky SA, McMurray RG, Bangdiwala SI, Frauman AC, Bradley CB. School based interventions improve heart health in children with multiple cardiovascular risk disease factors. Paediatrics 1998; 102(2): 371-380.

iii. Burke V, Milligan RAK, Thompson C *et al*. A controlled trial of health promotion programs in 11-year olds using physical activity "enrichment" for higher risk children. *Journal of Paediatrics* 1998; **132(5)**: 840-848.

(Type II evidence – 10 week randomised controlled trial in Perth, Australia including 800 11 year olds of a standard physical activity and nutrition programme in six schools, the standard programme in seven schools with the addition of physical activity enrichment for higher risk children, and no programme in five control schools)

iv. Brunton G, Harden A, Rees R, Kavanagh J, Oliver S, Oakley A. *Children and Physical Activity: A Systematic Review of Barriers and Facilitators*. London: EPPI-Centre, Social Science Research Unit, Institute of Education, University of London, 2003
http://eppi.ioe.ac.uk/EPPIWebContent/hp/reports/physical_activity02/Children_PA.pdf [accessed 17.12.03]

(Type I evidence – systematic review, literature search to November 2001, of 21 outcome evaluations and five qualitative studies examining childrens' views. In all, 90 qualitative and quantitative studies were examined of which 27 were randomised controlled trials)

v. Rees R, Harden A, Shepherd J, Brunton G, Oliver S, Oakley A. *Young People and Physical Activity: A Systematic Review of Barriers and Facilitators*. London: EPPI-Centre, Social Science Research Unit, Institute of Education, University of London, 2001
http://eppi.ioe.ac.uk/EPPIWebContent/hp/reports/physical_activity01/physical_activity.pdf [accessed 17.12.03]

(Type I evidence – systematic review, literature search to September 2000, of 12 outcome evaluations and 16 studies of young peoples' views. In all, 42 interventional studies (including some randomised controlled trials), 41 observational studies and 7 systematic reviews were examined)

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1.31c. Guidelines on **activity promotion in schools** advise that[i]:

- an appropriately designed, delivered and supported physical activity curriculum can enhance current levels of physical activity and can improve physical skill development;
- Young people benefit from access to suitable and accessible facilities and opportunities for physical activity;
- Interventions are likely to be more effective when young people are involved in planning programmes[i].

Further research is needed.

The *evidence*

i. Health Development Agency. *Coronary Heart Disease. Guidance for Implementing the Preventive Aspects of the National Service Framework.* 2nd edition. London: Health Development Agency, 2001
http://www.hda-online.org.uk/documents/chdframework.pdf [accessed 17.12.03]
(Type V evidence – consensus guidance based on a review of the evidence. The articles summarised for this topic area were published between 1988 and 1998)

National Service Framework

National Assembly for Wales. *Tackling CHD in Wales: Implementing Through Evidence.* Cardiff: National Assembly for Wales, July 2001
CHD must be tackled by the concerted effort of a large number of individuals and organisations all of which have a major part to play. These include: [paragraph 1.12]

- Communities and schools;
- Patients and carers;
- Professionals in primary, secondary and tertiary care;
- The ambulance service;
- Rehabilitation services;
- Occupational health services;
- Local health groups and health authorities [now Local Health Boards];
- Local authorities, health alliances;
- Health promotion groups;
- Voluntary sector organisations;
- Employers;
- Employment services;
- The National Assembly for Wales [now the Welsh Assembly Government];
- The media;
- All Wales Health Professional Groups;
- Exercise practitioners.

Which collaborative programmes work to reduce Coronary Heart Disease?

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1.32 Multiple risk factor interventions

1.32a. **Multiple risk factor interventions** comprising **counselling, education and drug therapies** were ineffective in achieving reductions in total or cardiovascular disease mortality when used in general or workforce populations of middle-aged adults. The pooled effects of intervention were statistically insignificant[i].

Net changes in systolic and diastolic blood pressure, smoking prevalence and blood cholesterol were –3.9 mmHg (95% CI –4.2 to –3.6 mm Hg), -2.9 mm Hg (3.1 to –2.7), -4.2% (-4.8 to –3.6) and –0.08 mMol/L (-0.1 to –0.06) respectively. In ten trials with clinical event end-points, the pooled odds ratios for total and coronary heart disease mortality were 0.97 (95% CI 0.92-1.02) and 0.97 (0.88-1.04) respectively[i].

Caveat: There was statistically heterogeneity between the studies. Only four trials were sufficiently large to have adequate power to show meaningful changes in clinical events and a potentially useful benefit of treatment (about a 10% reduction in coronary heart disease mortality) may have been missed[i].

See also chapter 2 for multiple risk factor interventions in high risk and CHD diagnosed groups.

i. Ebrahim S, Davey Smith G. Multiple risk factor interventions for primary prevention of coronary heart disease. (Cochrane Review) In *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 8 May 2001)
http://www.update-software.com/abstracts/ab001561.htm [accessed 17.12.03]

(Type I evidence – systematic review and meta-analysis, literature search to April 1995, of 18 randomised controlled trials of which 10 reported clinical event data. Reviewed in: Gluckman.R. Review: multiple interventions modestly reduce cardiovascular risk factors in primary prevention, but effects on mortality are uncertain. Commentary on Ebrahim, S *et al*. Multiple risk factor interventions for primary prevention of coronary heart disease (Cochrane Review). *ACP Journal Club* 2000; **132**: 43)

1.32b. There was no net intervention effect for the five-year **Heartbeat Wales** programme over and above observed change in the reference area. However, programme positive changes (for health) in behavioural outcomes were observed among the population in Wales, including a reduction in reported smoking prevalence and improvements in dietary choice[i].

Caveat: In the reference area, no additional community heart health promotion was planned although considerable activity did take place.

i. Tudor-Smith C, Nutbeam D, Moore L, Catford J. Effects of the Heartbeat Wales programme over five years on behavioural risks for cardiovascular disease: quasi-experimental comparison of results from Wales and a matched reference area. *British Medical Journal* 1998; **316**: 818-822
http://bmj.bmjjournals.com/cgi/content/full/316/7134/818 [accessed 17.12.03]

(Type IV evidence – quasi-experimental longitudinal study with a before-and-after design. Independent cross-sectional surveys were conducted in 1985 and 1990 within random, stratified, samples of people aged 18-64 years in Wales (18,538 in 1985 and 13,045 in 1990) and in North East England (1,483 and 4,534 respectively))

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1.32c. During the first ten years of a **community intervention programme** in an area of Sweden, where cardiovascular disease was a major public health problem, more than 90% of those invited participated in the individual health screening and counselling. A new food labelling system was introduced in the grocery stores and sales statistics regarding dairy products showed a significant turnover of low fat products. According to public opinion, the health screening and counselling were reported to be the most influential factors supporting lifestyle changes[i].
Caveat: No data on health outcomes were collected during this study.

i. Weinehall, Hellsten G, Boman K, Hallmans G. Prevention of cardiovascular disease in Sweden: the Norsjo community intervention programme motives, methods and intervention components. *Scandinavian Journal of Public Health* 2001; **29 (56)** 13-20

(Type lV evidence - longitudinal study of a local health promotion collaboration using annual cross-sectional survey data from 1985-1994, with control data from a reference area. The collaboration was between healthcare provided, grocery stores, schools, municipal authorities and the public, no hard outcomes i.e. actual effect on lifestyle or healthcare.)

1.32d. A theory based comprehensive **community** strategy in North Karelia, Finland, influenced the diet (particularly fat consumption) of the population and led to a major reduction in serum cholesterol and blood pressure levels. Serum cholesterol levels in men and women decreased by 18% between 1972 and 1997. During the same time period diastolic blood pressure decreased by 5% in men and 13% in women. Ischemic heart disease mortality in the working age population declined by 73% in North Karelia and by 65% in the whole country from 1971 to 1995[i] although the difference may not have been significant[ii].

i. Pekka P, Pirjo P, Ulla U. Influencing public nutrition for non-communicable disease prevention: from community intervention to national programme - experiences from Finland. *Public Health Nutrition* 2002; **5 (1A)** 245-251

(Type lV evidence - longitudinal study, of a community strategy monitored by risk factor, dietary and health behaviour surveys from 1972 –1995. In addition to health information and nutrition counselling, practical skills were taught, social and environmental support was provided and all sectors of the community were involved; health services, schools and social services, health related and other non-government organisations, supermarkets and the food industry, various community leaders and the local media. Full details of the project are available in Puska R, Tuomilehto J, Nissinen A, Vartianen E (eds). The North Karelia Project. 20-Year Results and Experiences. Helsinki: University Press, 1995)

ii. Ebrahim S, Davey Smith G. Multiple risk factor interventions for primary prevention of coronary heart disease. (Cochrane Review) In *The Cochrane Library*, Issue 1 2003. Oxford: Update Software. (most recent update 8 May 2001)
http://www.update-software.com/abstracts/ab001561.htm [accessed 17.12.03]

(Type I evidence - systematic review, Medline only searched to April 1995, of 18 trials)

1 LIFESTYLE

The *statements*

1.32e. **Management training seminars** resulted in increased organisational support for employee heart health in New York companies (as measured by *Heart Check*). A four-fold difference in change for *Heart Check* was observed in the experimental versus comparison groups ($p<0.01$)[i].

Caveats: The comparison group was examined at a different time period (dates not given) and comparison organisations contained a higher proportion of blue-collar workers. No health outcomes were measured.

The *evidence*

i. Golaszewski T, Barr D, Cochran S. An organization-based intervention to improve support for employee heart health. *American Journal of Health Promotion* 1998; **13(1)**: 26-35

(Type III evidence – a quasi-experimental worksite intervention comparing a management-training seminar to delayed treatment comparison group. The subjects were 20 western New York companies matched on size)

1.32f. While many of the **general practice-based lifestyle interventions** show promise in effecting small changes in behaviour, none appears to produce substantial changes[i]. *There is a need for more extensive and rigorous research in this area before substantial public funds are committed to general practice-based health promotion. Furthermore, it is clear that if general practice-based interventions are to be effective in a public health sense, a greater number of GPs will need to become involved in promoting behaviour change that the literature suggests is currently occurring*[i].

i. Ashenden R, Silagy C, Weller D. A systematic review of the effectiveness of promoting lifestyle change in general practice. *Family Practice* 1997; **14(2)**: 160-175

(Type I evidence – systematic review, literature search to May 1995, of 37 randomised controlled trials)

The *statements* | The *evidence*

1.33 Guidelines for the primary prevention of coronary heart disease

1.33a. Evidence-based guidelines are available for the primary prevention of cardiovascular disease[i,Ii,iii].

Guidance for the implementation of the **preventive aspects of the English National Service Framework** for coronary heart disease is available[iv].

i. British Cardiac Society, British Hyperlipidaemia Association, British Hypertension Society, endorsed by the British Diabetic Association. Joint British recommendations on prevention of coronary heart disease in clinical practice. *Heart* 1998; **80(suppl.2)**; S1- S29) (Evidence based guidelines. These guidelines are currently being updated)

Summary published as:
British Cardiac Society, British Hyperlipidaemia Association, British Hypertension Society, British Diabetic Association. Joint British recommendations on prevention of coronary heart disease in clinical practice: summary. *British Medical Journal* 2000; 320: 705-708

http://bmj.bmjjournals.com/cgi/content/full/320/7236/705

Correction on:

http://bmj.bmjjournals.com/cgi/content/full/323/7316/780/a [accessed on 17.12.03]

ii. Pearson TA, Blair SN, Daniels SR, Eckel RH, Fair JM *et al*. AHA Guidelines for Primary Prevention of Cardiovascular Disease and Stroke: 2002 update. *Circulation* 2002; **106**: 388-391

http://circ.ahajournals.org/cgi/reprint/106/3/388.pdf [accessed 17.12.03] (Evidence based guidelines)

iii. Wood D, De Backer G, Faergeman O, Graham I, Mancia G & Pyorala K. Prevention of coronary heart disease in clinical practice. *European Heart Journal* 1998; **19**: 1434-1503

http://www.escardio.org/scinfo/Guidelines/98prevention.pdf [accessed 17.12.03]

(Evidence based guidelines)

iv. Health Development Agency. *Coronary Heart Disease. Guidance for Implementing the Preventive Aspects of the National Service Framework*. 2nd edition. London: Health Development Agency, 2001

http://www.hda-online.org.uk/documents/chdframework.pdf [accessed 17.12.03]

(Type V evidence – consensus guidance based on a review of the evidence)