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Impact of dietary and lifestyle factors on the prevalence of hypertension in Western populations

JOHANNA M. GELEIJNSE, FRANS J. KOK, DIEDERICK E. GROBBEE *

Background: Data on the impact of the many dietary and lifestyle factors on the prevalence of hypertension in Western societies are lacking. This study quantified the contributions of body weight, physical inactivity and dietary factors to the prevalence of hypertension in Finland, Italy, the Netherlands, United Kingdom (UK) and USA. Methods: Distributions of blood pressure (BP) and risk factors in Western populations were obtained from nationwide surveys. The effect of risk factors on BP was assessed by meta-regression analysis of randomized trials, published between 1966 and March 2001. Population attributable risk percentages (PAR%) for hypertension (i.e. systolic BP ≥140 mmHg) were computed for all risk factors in the five countries. Results: Being overweight made the largest contribution to hypertension, with PAR% between 11% (Italy) and 25% (USA). PAR% were 5–13% for physical inactivity, 9–17% for high sodium intake, 4–17% for low potassium intake and 4–8% for low magnesium intake. The impact of alcohol was small (2–3%) in all populations. PAR% varied among populations for inadequate intake of calcium (2–8%), magnesium (4–8%), coffee (0–9%) and fish fatty acids (3–16%). Conclusions: Diet and lifestyle have a substantial impact on hypertension in Western societies, with being overweight, physical inactivity, high sodium intake and low potassium intake being the main contributors. The relative significance of different risk factors varies among populations, which is important for preventative strategies.

Keywords: dietary and lifestyle factors, hypertension, population attributable risk, prevention

Even modest reductions in blood pressure (BP) could have a large impact on cardiovascular disease morbidity and mortality in Western populations.[1] Many dietary and lifestyle factors have been implicated in the etiology of hypertension. However, an objective scientific understanding of their relative significance in the general population is lacking. The population attributable risk percentage (PAR%) may provide insight into this complex matter, as it is based not only on the strength of the risk factor–BP association but also on BP and risk factor distributions in the population.[2] This study quantified the impact of dietary and lifestyle factors on the prevalence of hypertension in Finland, Italy, the Netherlands, the UK and the USA. PAR% were computed for the following modifiable risk factors: body weight, physical activity, and intake of alcohol, coffee, sodium, potassium, magnesium, calcium and fish oil (containing eicosapentaenoic acid and docosahexaenoic acid, subsequently referred to as fish fatty acids). Findings from this study may indicate priorities for preventative strategies to reduce the burden of hypertension in Western populations.

METHODS

Estimation of BP effects of dietary and lifestyle factors

Meta-analyses and quantitative reviews of non-pharmacological BP trials were identified from MEDLINE and the Cochrane Library database up to March 2001, using appropriate MESH-terms (search strategy and results available from the authors). BP trials were identified from the reference lists of these papers. An additional MEDLINE search was done for trials published after 1990.

Trials were eligible for meta-regression analysis if they met the following criteria: i) randomized controlled design; ii) mean age of study population ≥18 years; and iii) published after 1966. A total of 646 out of 887 publications satisfied these criteria, 393 of which were excluded for the following reasons: i) overlap with other publication (n=72); ii) lack of BP data (n=32); iii) co-intervention from which the effect of the risk factor could not be separated (n=93); iv) pregnant or diseased subjects (n=64); v) non-placebo control group (n=31); vi) pharmacological intervention (n=10); vii) duration less than 2 weeks (n=89); and viii) BP reductions >30 mmHg (n=2).

In total, 253 trials were selected (overview available from the authors) for which original reports were retrieved for data abstraction on trial design (parallel versus cross-over), duration, number of participants, size of intervention and BP changes. Data were also collected on age, gender (% males), and initial BP and risk factor level of the trial population. Whenever possible, stratified data were obtained (e.g. men and women, treatment doses). A database was created with trials (or trial strata) as the units of observation. Pooled BP estimates with 95% confidence intervals were obtained for each risk factor using meta-regression analysis, as described in detail elsewhere.[3,4] BP estimates were weighted for trial sample sizes, and these estimates were used for calculation of PAR% (see below). SPSS 11.0.1 for Windows was used for statistical analyses.

Data collection on BP and risk factors in Western populations

Population-specific BP distributions were obtained from the MONICA project (ages 25–64 years)[5,6] or, for the Netherlands, from the MORGEN project (ages 20–59 years)[7] combined with the Rotterdam Study (ages 55 years and over).[8] Mean systolic BP was 139 ± 20 mmHg for Finland, 133 ± 19 mmHg for Italy, 127 ± 19 mmHg for the Netherlands, 130 ± 20 mmHg for the UK and 125 ± 20 mmHg for the USA. Under the assumption of a normal distribution, population proportions with systolic BP ≥140 mmHg were estimated at 48% for Finland, 36% for Italy, 25% for the Netherlands, 31% for the UK and 23% for the USA.

Next, we defined risk categories for exposure to dietary and lifestyle factors (see *table 2*). If possible, these were based on recommended dietary allowances or dietary guidelines for adults.[9–11] For coffee, widely accepted criteria were lacking and four or more cups per day were arbitrarily defined as inadequate. For intake of fish fatty acids there are different recommended

* J.M. Geleijnse[1,2], F.J. Kok[1], D.E. Grobbee[2]
1 Division of Human Nutrition (www.humannutrition.wur.nl), Wageningen University, Wageningen, The Netherlands
2 Julius Center for Health Sciences and Primary Care (www.juliuscenter.nl), University Medical Center Utrecht, Utrecht, The Netherlands
Correspondence: Johanna M. Geleijnse, Division of Human Nutrition, Wageningen University, PO Box 8129, 6700 EV Wageningen, The Netherlands, tel. +31 317 482574, fax +31 317 483342, e-mail: marianne.geleijnse@wur.nl

levels[12,13] and we used <200 mg per day for risk group definition. Physical activity was not uniformly assessed in population-based surveys, and we therefore chose a general risk group definition of 'no exercise during leisure time, exclusive of walking'.

Distributions of dietary and lifestyle factors were obtained from relevant age strata in large nationwide databases. For Finland, prevalence of overweight was obtained from FINRISK 1997 (age stratum: 25–64 years)[14] and data on physical inactivity from the Annual Health Behavior Surveillance System 2000 (25–64 years).[15] Dietary intakes were estimated from the 1992 Dietary Survey of Finnish Adults (25–64 years).[16] Body weight in Italy was estimated from the Italian Household Multipurpose Survey (Cycle V; 25–74 years), conducted by the Italian National Institute of Statistics (ISTAT) in 1990–1991,[17] and alcohol use from the 3rd National Survey on Italians and Alcohol, 1997 (>25 years).[18] Other Italian data were obtained from the Nationwide Nutritional Survey of Food Behaviour of the Italian Population (INN-CA) 1994–1996 (18–64 years).[19,20] Physical inactivity in this study was defined as 'zero hours of sports per week', and coffee use was estimated from coffee powder (6 g equals 1 cup). For the Netherlands, overweight and physical inactivity (i.e. 'no participation in sporting activities or heavy exercise during leisure time') were estimated from the MORGEN project[7] and dietary data from the 3rd Dutch Nutrition Surveillance System 1997–1998 (19–75 years).[21] For the UK, data on physical inactivity (i.e. 'no single occasion of sports for 15 min or more during the past 4 weeks') was obtained from the Health Survey for England 1998 (>18 years)[22,23] and other data from the Dietary and Nutritional Survey of British Adults 1986–1987 (18–64 years).[24,25] For the USA, data were obtained from the Third National Health and Nutrition Examination Survey (NHANES III) 1988–1994 (18–79 years).[26] Physical inactivity in NHANES III was defined as 'no single occasion of sports during past month'.

Population-based data on sodium intake, based on 24 h urine collections, were obtained from the worldwide INTERSALT study (20–59 years).[27] For the USA, we disregarded the outlying sodium excretion in the 'Goodman–Black' sample. Mean intake of fish oil could not be obtained for the UK. We therefore used data on fatty fish intake from the Dietary and Nutrition Survey of British Adults,[24,25] and assessed the proportion of adults with a daily intake below 15 g (roughly equivalent to 200 mg of fish fatty acids).[28] Similarly for Italy, we estimated fish fatty acid intake from total fish (data from INN-CA survey),[27] 14% of which was assumed to be fatty fish.[29]

Calculation of population attributable risk percentages (PAR%)

Hypertension was defined on the basis of systolic BP only (≥140 mmHg) because the prevalence of isolated diastolic hypertension is very low. Pooled systolic BP estimates from randomized trials, as presented in *table 1*, were taken as the BP reduction that may be achieved after elimination of the risk factor. Calculation of the expected change in the prevalence of hypertension after risk factor negation was performed using a JAVA™ applet for the standard normal distribution.[30] PAR% were obtained after multiplying this figure with the proportion of the population exposed to the risk factor, divided by the prevalence of hypertension in that population.[2]

RESULTS

Findings from meta-regression analysis of BP trials are presented in *table 1*. Mean systolic BP was ≥140 mmHg in 45% of trial populations. The unweighted, unadjusted change in systolic and diastolic BP ranged from –1.5/–0.7 mmHg for calcium

Table 1 Blood pressure response to dietary and lifestyle changes in randomized trials

	No. of trials	Duration (wk)[a]	Age (year)	Men (%)	Initial risk factor level	Size of intervention	Initial BP (mmHg)		BP change (mmHg)[b]		Weighted BP change (mmHg)[c]	
							Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
Body weight	25	24 (2–156)	48 ± 7	55 ± 33	89 ± 10 kg	–6.5 ± 2.4 kg	139 ± 13	88 ± 9	–5.3 (–7.0, –3.5)	–3.6 (–4.9, –2.4)	–4.8 (–6.5, –3.1)	–3.4 (–4.7, –2.2)
Physical activity	49	16 (4–52)	45 ± 14	59 ± 40	67% inactive[d]	2.5 ± 1.1 h/wk	130 ± 14	82 ± 11	–4.2 (–5.5, –2.9)	–2.5 (–3.5, –1.6)	–2.8 (–3.9, –1.7)	–1.8 (–2.6, –1.1)
Alcohol	13	6 (2–52)	45 ± 6	100	66 ± 7 ml/d[e]	–41 ± 17 ml/d[e]	139 ± 10	85 ± 9	–3.5 (–4.6, –2.3)	–2.0 (–2.8, –1.3)	–2.6 (–3.7, –1.4)	–1.4 (–2.0, –0.7)
Coffee	10	8 (2–11)	43 ± 17	55 ± 24	5.1 ± 0.9 cups/d	–4.9 ± 0.9 cups/d	125 ± 8	76 ± 6	–1.7 (–3.3, –0.1)	–1.0 (–2.1, 0.1)	–2.2 (–3.9, –0.6)	–1.0 (–2.1, 0.0)
Sodium	40	4 (2–156)	48 ± 15	61 ± 23	3.5 ± 0.8 g/d[f]	–2.1 ± 1.2 g/d[f]	144 ± 17	88 ± 12	–4.1 (–5.3, –2.9)	–2.5 (–3.3, –1.6)	–2.5 (–3.4, –1.6)	–2.0 (–2.6, –1.4)
Potassium	27	6 (2–114)	45 ± 12	60 ± 35	2.4 ± 0.4 g/d[g]	2.0 ± 1.0 g/d[g]	143 ± 21	89 ± 14	–3.3 (–4.8, –1.8)	–2.1 (–3.4, –0.8)	–2.4 (–3.7, –1.2)	–1.6 (–2.6, –0.6)
Magnesium	16	8 (3–26)	52 ± 11	54 ± 28	277 ± 56 mg/d	483 ± 216 mg/d	147 ± 13	93 ± 9	–1.7 (–3.8, 0.5)	–1.8 (–3.1, –0.6)	–1.3 (–2.9, 0.3)	–0.9 (–1.9, 0.1)
Calcium	36	10 (2–208)	45 ± 13	54 ± 35	0.80 ± 0.18 g/d	1.2 ± 0.4 g/d	133 ± 16	83 ± 11	–1.5 (–2.9, –0.0)	–0.7 (–1.7, 0.2)	–1.5 (–2.8, –0.3)	–0.7 (–1.6, 0.1)
Fish oil[h]	36	8 (3–52)	46 ± 11	85 ± 22	i	4.1 ± 2.7 g/d	136 ± 13	85 ± 11	–2.1 (–3.4, –0.9)	–1.5 (–2.2, –0.7)	–2.1 (–3.2, –1.0)	–1.6 (–2.2, –1.0)

Values given as unweighted mean ± standard deviation, unless stated otherwise.
a: Median, with range in parentheses.
b: Mean change in blood pressure during intervention in pooled set of trials for given risk factor, with 95% confidence interval.
c: BP estimate with 95% confidence interval, weighted by trial sample sizes.
d: Indicates the proportion of trial populations that were sedentary at baseline, according to the trial reports.
e: Absolute level of alcohol (ethanol) intake.
f: 1 g sodium equals 43 mmol (median reduction: 77 mmol/24 h).
g: 1 g potassium equals 26 mmol (median increase: 44 mmol/24 h).
h: Fish oil supplements in different trials contain varying amounts of eicosapentaenoic acid and docosahexaenoic acid.
i: Data on initial intake of fish fatty acids were lacking in 89% of the trials and can therefore not be reported.
In Western populations, intake of fish fatty acids is <500 mg per day.

supplementation to –5.3/–3.6 mmHg for weight reduction. BP estimates for physical activity, alcohol, sodium, potassium and magnesium were attenuated after weighing for trial sample sizes. Weighted BP estimates were statistically significant, except for systolic and diastolic BP in magnesium trials and diastolic BP in calcium trials and coffee trials (*table 1*).

Table 2 shows population proportions in risk categories of dietary and lifestyle factors in five Western populations. These figures, together with BP estimates presented in *table 1*, were used to obtain PAR% for hypertension. To illustrate, the impact of being overweight on hypertension in Finland is explained in more detail. For a decrease of 4.8 mmHg in systolic BP due to weight loss (*table 1*), population BP in Finland would decline from 139.0 to 134.2 mmHg and the prevalence of hypertension (defined as systolic BP ≥140 mmHg) from 48.0% to 38.5%. If 6 kg of weight loss (average reduction in trials) was achieved in 60% of Finnish adults who are overweight (*table 2*), hypertension in Finland would decline by 5.7% (i.e. 0.60 × 9.4%). Dividing this figure by the prevalence of hypertension yields a PAR% of 12% (calculations for all risk factors in the five populations are available from the authors).

PAR% for hypertension are given in *table 3*. Being overweight made a substantial contribution to the prevalence of hypertension in all populations, with PAR% between 11% (Italy) and 25% (USA). PAR% were 5–13% for physical inactivity, 9–17% for high sodium intake, 4–17% for low potassium intake and 4–8% for low magnesium intake. The impact of alcohol was small (2–3%) in all populations. PAR% for low calcium intake were also small (<5%), except for the USA (8%). PAR% varied among populations for inadequate intake of magnesium (4–8%), fish fatty acids (3–16%) and coffee (0–9%).

DISCUSSION

This study shows that diet and lifestyle have a large effect on the prevalence of hypertension in Western societies, with different rankings of risk factors within populations. If one assumes risk factors to have additive effects and preventative strategies to be effective, a minimum decline of 40% in the prevalence of hypertension may be achieved, depending on the population. Being overweight, physical inactivity, high salt intake and low potassium intake appeared to be the major contributors to hypertension in Western populations. With regard to fish fatty acids, better data on dietary intake in the population and more BP trials with intake of fish fatty acids in the normal dietary range (<500 mg per day) are needed before drawing conclusions.

Table 2 Exposure to risk factors for hypertension in five Western populations

		Exposed proportion of the population[a]				
Dietary or lifestyle factor	Risk group definition	Finland	Italy	The Netherlands	UK	USA
Body mass index	≥25 kg/m^2	0.60	0.40	0.48	0.41	0.60
Physical activity[b]	Inactive	0.41	0.58	0.42	0.63	0.52
Alcohol[c]	≥3 drinks/d	0.18	0.19	0.15	0.19	0.13
Coffee	≥4 cups/d	0.48	0.03	0.48	0.27	0.05
Sodium[d]	≥2.4 g/d	0.84	0.88	0.81	0.82	0.78
Potassium[e]	<3.5 g/d	0.36	0.74	0.46	0.80	0.79
Magnesium	<350 mg/d	0.36	0.98	0.62	0.80	0.74
Calcium	<500 mg/d	0.25	0.35	0.28	0.43	0.60
Fish fatty acids[f]	<200 mg/d	0.39	0.75	0.85	0.83	0.85

a: Prevalences of exposure have been derived from nationwide surveys (details given in text).
b: Roughly defined as 'no regular exercise during leisure time, exclusive of walking'. The precise definition of physical inactivity may vary among populations (details given in text).
c: 3 drinks equal 25–50 g ethanol, depending on the type of alcoholic beverage consumed.
d: 1 g sodium equals 43 mmol of sodium and 6 g of salt (NaCl).
e: 1 g potassium equals 26 mmol.
f: Total of eicosapentaenoic acid and docosahexaenoic acid; for Italy and UK, population-based intakes of fish fatty acids were not available and proportions have been derived from intake of fatty fish (details given in text); exposure to inadequate intake of fish fatty acids is likely to be overestimated if based on single 24-h dietary recalls or dietary records.

Table 3 PAR% for hypertension for dietary and lifestyle factors in five Western populations

	PAR% for hypertension[b]				
Risk factor[a]	Finland %	Italy %	The Netherlands %	UK %	USA %
Overweight	12	11	19	13	25
Physical inactivity	5	10	10	11	13
High alcohol intake	2	3	3	3	3
High coffee intake	4	<1	9	4	1
High sodium intake	9	13	17	13	17
Low potassium intake	4	10	9	12	17
Low magnesium intake	4	8	7	7	8
Low calcium intake	2	3	4	4	8
Low intake of fish fatty acids[c]	3	9	15	11	16

a: Risk factor definitions are given in *table 2*.
b: PAR%: the percentage of hypertension in a population that is caused by exposure to a risk factor and thus that could be eliminated if the risk factor were negated. The PAR% depends on the blood pressure effect of the risk factor (*table 1*), the prevalence of risk factor exposure in the population (*table 2*) and the prevalence of hypertension in the population (see text).
c: Must be interpreted with caution as PAR% are based on the blood pressure effect of high doses of fish oil (*table 1*).

Several methodological issues in this study warrant further consideration. BP estimates in this study were obtained from randomized trials, excluding multifactorial interventions, and are likely to be causal and fully attributable to the specific risk factors. Although short-term trials were excluded to eliminate acute BP responses, BP effects (and consequently PAR%) may be underestimated because true long-term effects (>1 year) were not accounted for. As in other meta-analyses, our BP estimates should be interpreted against the background of the pooled trial populations from which the estimates have been derived. Subject characteristics may influence BP response to intervention, e.g. BP sensitivity to sodium is probably larger in populations with elevated BP. Calculating changes in the prevalence of hypertension after a uniform shift of the population BP distribution to a lower level, as we did, is therefore a simplified model. Also, BP estimates for some risk factors (e.g. magnesium and coffee) have wide confidence intervals due to the small number of trials in meta-regression analysis and PAR% should therefore be interpreted with caution. Furthermore, PAR% may be too conservative since they were based on risk groups only, whereas others may also benefit from prevention. To illustrate, weight loss is known to also reduce BP in normal-weight subjects. Finally, we did not examine risk factor interactions.

If we assume BP effects of all risk factors to be additive, population BP may decline by more than 20 mmHg. Cardiovascular risk strongly increases with BP, even in the 'normal' range.[31,32] In the large MRFIT study, 20 mmHg decrease in population systolic BP was associated with a 26% reduction in mortality, meaning 56,479 lives saved in the US male population.[33] We need to point out that prevalences of hypertension in our study are overestimated due to non-repeated BP measurements in epidemiological surveys.[34] However, this is unlikely to have had a large effect on PAR% and the ranking of risk factors in the different populations.

We conclude that effective dietary and lifestyle interventions could substantially reduce population BP in Western societies. For several risk factors, the impact on hypertension varied among populations, which is important for setting priorities in preventative strategies. Hypertension itself, however, is not the outcome of primary interest. More research is needed to assess the impact of diet and lifestyle on the total cardiovascular risk profile, including serum lipids and oxidative stress, and (cardiovascular) mortality. For Europe, a standardized core population database for dietary and lifestyle exposures would be extremely useful to facilitate public health research in this field.

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REFERENCES

1 Collins R, Peto R, MacMahon S, et al. Blood pressure, stroke, and coronary heart disease. Part 2, short-term reductions in blood pressure: overview of randomized drug trials in their epidemiological context. Lancet 1990;335:827-38.

2 Jekel JF, Elmore JG, Katz DL. Assessment of risk in epidemiologic studies. In: Epidemiology, Biostatistics, and Preventive Medicine. 2nd ed. Philadelphia, PA: W.B. Saunders Company, 1996:74-82.

3 Geleijnse JM, Giltay EJ, Grobbee DE, Donders ART, Kok FJ. Blood pressure response to fish oil supplementation: metaregression analysis of randomized trials. J Hypertens 2002;20:1493-9.

4 Geleijnse JM, Kok FJ, Grobbee DE. Blood pressure response to changes in sodium and potassium intake: a meta-regression analysis of randomised trials. J Hum Hypertens 2003;17:471-80.

5 Keil U, Kuulasmaa K, for the WHO MONICA Project. WHO MONICA Project: Risk factors. Int J Epidemiol 1989;18(Suppl.1):S46-55. http://www.ktl.fi/monica/ (last accessed 20 January 2004).

6 Kuulasmaa K, Hense HW, Tolonen H, for the WHO MONICA Project. Quality Assessment of Data on Blood Pressure in the WHO MONICA Project, 1998. http://www.ktl.fi/publications/monica/bp/table8.htm (last accessed 20 January 2004).

7 Blokstra A, Seidell JC, Smit HA, Bueno de Mesquita HB, Verschuren WMM. Het Project Monitoring Risicofactoren en Gezondheid Nederland (MORGEN-project): jaarverslag 1997 [The Monitoring Project on Risk Factors for Chronic Diseases (MORGEN project): annual report 1997]. Report 263200007. Bilthoven, The Netherlands: National Institute of Public Health and the Environment (RIVM), 1998.

8 Grobbee DE, Bom JG van der, Bots ML, Bruijne MC de, Mosterd A, Hoes AW. Coronaire hartziekten bij ouderen: het ERGO-onderzoek [Coronary heart disease in the elderly: the Rotterdam Study]. Ned Tijdschr Geneeskd 1995;139:1978-82.

9 Subcommittee on the Tenth Edition of the Recommended Dietary Allowances. Recommended Dietary Allowances: 10th Edition. Washington, DC: National Research Council, National Academy Press, 1989.

10 Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D and fluoride. Food and Nutrition Board, Institute of Medicine, National Academy of Sciences. Washington, DC: National Academy Press, 1997.

11 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;102:2284-99.

12 De Deckere EAM, Korver O, Verschuren PM, Katan MB. Health aspects of fish and N-3 polyunsaturated fatty acids from plant and animal origin. Eur J Clin Nutr 1998;52:749-53.

13 Simopoulos AP, Leaf A, Salem N. Essentiality of and recommended dietary intakes for omega-6 and omega-3 fatty acids. Ann Nutr Metab 1999;43:127-30.

14 Lahti-Koski M, Vartiainen E, Männistö S, Pietinen P. Age, education and occupation as determinants of trends in body mass index in Finland from 1982 to 1997. Int J Obes Relat Metab Disord 2000;24:1669-76.

15 Helakorpi S, Uutela A, Prättälä R, Puska P. Suomalaisen Aikuisväestön Terveyskäyttäytyminen ja Terveys, Kevät 2000 [Health Behaviour and Health among Finnish Adult Population, Spring 2000]. National Public Health Institute, Finland. Helsinki: Department of Epidemiology and Health Promotion, Health Promotion Research Unit, 2000. http://www.ktl.fi/publications/avtk/avtk2000.pdf (last accessed 20 January 2004).

16 Kleemola P, Virtanen M, Pietinen P. The 1992 Dietary Survey of Finnish Adults. National Public Health Institute, Department of Nutrition, Finland. Yliopistopaino, Pikapaino, Helsinki, 1994.

17 Pagano R, La Vecchia C. Overweight and obesity in Italy, 1990-91. Int J Obes Relat Metab Disord 1994;18:665-9.

18 The 3rd National Survey on Italians and Alcohol. Osservatorio Permanente sui Giovani e l'Alcool - Doxa, 1998. http://www.alcol.net/Inglese/home.htm (last accessed 20 January 2004).

19 INN-CA Study 1994-96 Data Bank - Istituto Nazionale di Ricerca per gli Alimenti e la Nutrizione [National Italian Dietary Survey 1994-1996]. Rome, Italy: National Institute for Food and Nutrition Research. (Personal communication with Dr A. Turrini, Istituto Nazionale della Nutrizione, Rome.)

20 Turrini A, Saba A, Cialfa E, D'Amicis A. Food consumption patterns in Italy: the INN-CA Study 1994-1996. Eur J Clin Nutr 2001;55:571-88.

21 Hulshof KFAM, Kistemaker C, Bouman M. De inname van energie en voedingsstoffen door Nederlandse Bevolkingsgroepen: voedselconsumptiepeiling 1997-1998 [The third Dutch Nutrition Surveillance System, 1997-1998]. Reports V98.805, V98.806, V98.813, V98.815. TNO, Zeist, 1998; Report V99.516. TNO, Zeist, 1999.

22 Erens B, Primatesta P, editors. Health Survey for England: cardiovascular disease '98, volume I and II. London: HMSO, 1998. ISBN 0113223072. http://www.official-documents.co.uk/document/doh/survey98/hse98.htm (last accessed 20 January 2004).

23 National Centre for Social Research. Health Survey for England 1998 [computer file]. Colchester, Essex: The Data Archive [distributor], 14 December 2000. SN:4150.

24 Gregory J, Foster K, Tyler H, Wiseman M. The Dietary and Nutritional Survey of British Adults. Office of Population Censuses and Surveys, Social Service Division. London: HMSO, 1990. ISBN 0116013002.

25 Ministry of Agriculture, Fisheries and Food. The Dietary and Nutritional Survey of British Adults 1986-1987 [computer file]. Colchester, Essex: The Data Archive [distributor], 18 September 1991. SN:2836.

26 US Department of Health and Human Services (DHHS). National Center for Health Statistics. Third National Health and Nutrition Examination Survey, 1988-1994, NHANES III Examination Data File and Total Nutrient Intake File. Hyattsville, MD: Centers for Disease Control and Prevention, 1996. Public use data files, available from National Technical Information Service (NTIS), Springfield, VA. http://www.cdc.gov/nchs/about/major/nhanes/nh3data.htm (last accessed 20 January 2003).

27 Elliott P, Dyer A, Stamler R. The INTERSALT study: results for 24 hour sodium and potassium, by age and sex. INTERSALT Co-operative Research Group. J Hum Hypertens 1989;3:323-407.

28 Kris-Etherton PM, Taylor DS, Yu-Poth S, et al. Polyunsaturated fatty acids in the food chain in the United States. Am J Clin Nutr 2000;71(Suppl.1):S179-88.

29 Oomen CM, Feskens EJ, Rasanen L, et al. Fish consumption and coronary heart disease mortality in Finland, Italy, and The Netherlands. Am J Epidemiol 2000;151:999-1006.

30 McClelland GH. Stanford University, Department of Statistics, Stanford, CA, USA. Seeing Statistics. Duxbury Press, 1999. http://psych.colorado.edu/~mcclella/java/normal/accurateNormal.html (last accessed 20 January 2004).

31 Stamler J, Stamler R, Neaton JD. Blood presure, systolic and diastolic, and cardiovascular risks: US population data. Arch Intern Med 1993;153:598-615.

32 Van den Hoogen PCW, Feskens EJM, Nagelkerke NJD, et al. The relation between blood pressure and mortality due to coronary heart disease among men in different parts of the world. N Engl J Med 2000;342:1-8.

33 Psaty BM, Furberg CD, Kuller LH, et al. Association between blood pressure level and the risk of myocardial infarction, stroke, and total mortality: the cardiovascular health study. Arch Intern Med 2001;161:1183-92.

34 Klungel OH, de Boer A, Paes AH, Nagelkerke NJ, Seidell JC, Bakker A. Estimating the prevalence of hypertension corrected for the effect of within-person variability in blood pressure. J Clin Epidemiol 2000;53:1158-63.

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