

ORIGINAL ARTICLE

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

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The impact of dietary and lifestyle factors on the prevalence of hypertension was quantified for Finland, Italy, The Netherlands, UK and USA. For this purpose, we combined data of blood pressure (BP) and risk factors distributions in these five countries with BP estimates from randomized controlled trials of dietary and lifestyle factors to obtain population attributable risk percentages (PAR%) for hypertension. Overweight made a substantial contribution to hypertension (PAR%: 11–17%), as was the case for excessive sodium intake (9–17%), low potassium intake (4–17%), physical in-

activity (5–13%), and low intake of fish oil (3–16%). PAR% were smaller for low calcium intake (2–8%), low magnesium intake (4–8%), excessive coffee consumption (1–9%) and excessive alcohol intake (2–3%). We conclude that diet and lifestyle have a major impact on hypertension in Western societies. The relative significance of different risk factors varies among populations, which is important for preventive strategies.

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Introduction

Many dietary and lifestyle factors have been implicated in the aetiology of hypertension. However, an objective scientific understanding of their relative significance in the general population is lacking. The population attributable risk percentage (PAR%) provides insight into this complex matter, as it is based not only on the strength of the risk factor–blood pressure (BP) association but also on BP and risk factor distributions in the population.¹ The impact of dietary and lifestyle factors on the prevalence of hypertension was quantified for Finland, Italy, the Netherlands, the UK and the USA. PAR% was computed for the following modifiable risk factors: body weight, physical activity, and intake of alcohol, coffee, sodium, potassium, magnesium, calcium and fish oil (eicosapentaenoic and docosahexaenoic acid). Findings from this study may indicate priorities for preventive strategies to reduce the burden of hypertension in Western populations.

Methods

First, we obtained BP estimates for each dietary and lifestyle factor by performing metaregression analysis of randomised controlled trials, as described in detail elsewhere.^{2–5}

Second, BP distributions (mean \pm s.d.) were obtained for five Western populations using data from population-based surveys, such as the MONICA project.⁶ 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.

Third, we defined risk categories for exposure to dietary and lifestyle factors. If possible, these were based on recommended dietary allowances or dietary guidelines for adults. Overweight was defined as a body mass index ≥ 25 kg/m². For physical inactivity, we chose a general risk group definition of ‘no exercise during leisure time, exclusive of walking’. Heavy alcohol consumption was defined as ≥ 3 drinks per day and heavy coffee consumption as ≥ 4 cups per day (one cup ≈ 125 ml). High sodium intake was defined as ≥ 2.4 g/day (equals 6 g of salt). Low intake of potassium was defined as < 3.5 g, low

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intake of magnesium as <350 mg and low intake of calcium as <750 mg per day. For fish fatty-acids (eicosapentaenoic acid and docosahexaenoic acid), low intake was defined as <200 mg per day. Distributions of dietary and lifestyle factors in the five populations studied were obtained from nationwide dietary surveys, such as NHANES,⁷ and salt intake from the worldwide INTERSALT study,⁸ as described in detail elsewhere.⁵

Finally, PAR% was calculated for each dietary and lifestyle factor within the five populations. The PAR% is the percentage of hypertension in a population that is caused by exposure to a risk factor and thus that can be eliminated if the risk factors are negated.¹ PAR% was based on only systolic BP (cutoff: 140 mmHg) because the proportion of population with isolated diastolic hypertension is negligible. Pooled systolic BP estimates from randomised trials (Table 1) were taken as the BP reduction that may be achieved after risk factor elimination. Calculation of the expected change in the prevalence of hypertension after risk factor elimination was

performed using a JAVA™ applet for the standard normal distribution.⁹ PAR% was obtained after multiplying this figure with the proper population exposed to the risk factor, divided by the prevalence of hypertension in that population (detailed calculations available from the authors).

Results

Weighted BP estimates for dietary and lifestyle factors, obtained by metaregression analysis of randomised controlled trials, are presented in Table 1. Overweight had the strongest effect on BP with an average decrease of 5/3 mmHg in weight loss trials (~1 mmHg/kg). BP decreases were 2–3 mmHg systolic and 1–2 mmHg diastolic in intervention studies of physical activity, alcohol reduction, coffee reduction, sodium reduction, potassium supplementation and fish oil supplementation. The BP effects of calcium and magnesium supplementation were smaller and only borderline statistically significant.

Table 1 BP response to dietary and lifestyle interventions: metaregression analysis of randomized controlled trials

Dietary of lifestyle factor	No. of trials	Size of intervention ^b	BP change (mmHg) ^a	
			Systolic BP	Diastolic BP
Body weight	25	-6.5 ± 2.4 kg	-4.8 (-6.5, -3.1)	-3.4 (-4.7, -2.2)
Physical activity	49	+2.5 ± 1.1 h/wk	-2.8 (-3.9, -1.7)	-1.8 (-2.6, -1.1)
Alcohol	13	-41 ± 17 ml	-2.6 (-3.7, -1.4)	-1.4 (-2.0, -0.7)
Coffee	10	-4.9 ± 0.9 cups	-2.2 (-3.9, -0.6)	-1.0 (-2.1, 0.0)
Sodium	40	-2.1 ± 1.2 g	-2.5 (-3.4, -1.6)	-2.0 (-2.6, -1.4)
Potassium	27	+2.0 ± 1.0 g	-2.4 (-3.7, -1.2)	-1.6 (-2.6, -0.6)
Magnesium	16	+483 ± 216 mg	-1.3 (-2.9, 0.3)	-0.9 (-1.9, 0.1)
Calcium	36	+1.2 ± 0.4 g	-1.5 (-2.8, -0.3)	-0.7 (-1.6, 0.1)
Fish oil ^c	36	+4.1 ± 2.7 g	-2.1 (-3.2, -1.0)	-1.6 (-2.2, -1.0)

^aPooled BP estimate obtained from randomized controlled trials, weighted by trial sample size, with 95% confidence interval.

^bAverage change in dietary intake (per day) or lifestyle factor in trials.

^cSupplementation of fish fatty-acids (eicosapentaenoic acid and docosahexaenoic acid).

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

Risk factor ^a	Finland		Italy		The Netherlands		UK		USA	
	Exposed ^b	PAR% ^c	Exposed	PAR%	Exposed	PAR%	Exposed	PAR%	Exposed	PAR%
Overweight	0.60	12%	0.40	11%	0.48	19%	0.41	13%	0.60	25%
Physical inactivity	0.41	5%	0.58	10%	0.42	10%	0.63	11%	0.52	13%
High alcohol intake	0.18	2%	0.19	3%	0.15	3%	0.19	3%	0.13	3%
High coffee intake	0.48	4%	0.03	<1% ^d	0.48	9%	0.27	4%	0.05	1%
High sodium intake	0.84	9%	0.88	13%	0.81	17%	0.82	13%	0.78	17%
Low potassium intake	0.36	4%	0.74	10%	0.46	9%	0.80	12%	0.79	17%
Low magnesium intake	0.36	4%	0.98	8%	0.62	7%	0.80	7%	0.74	8%
Low calcium intake	0.25	2%	0.35	3%	0.28	4%	0.43	4%	0.60	8%
Low intake of fish fatty-acids ^e	0.39	3%	0.75	9%	0.85	15%	0.83	11%	0.85	16%

^aRisk group definitions for dietary and lifestyle factors are given in the text.

^bProportion of the general adult population in risk category, estimated from nationwide surveys.

^cThe percentage of hypertension in a population that is caused by exposure to a risk factor and thus that could be eliminated if the risk factors are 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 (given in text).

^dPrevalence of heavy coffee consumption in Italy may be underestimated (details given in text).

^eAccurate population-based data are lacking and prevalence figures should be interpreted with caution.

Table 2 shows population proportions with unfavourable levels of risk factors in the five populations which, combined with BP estimates from randomised trials, were used to obtain PAR% for hypertension. PAR% calculation for overweight in Finland is explained as an example. For a 4.8 mmHg decrease in systolic BP (ie 139.0–134.2 mmHg), the prevalence of hypertension (systolic BP \geq 140 mmHg) would decline from 48.0 to 38.5%. It is estimated that 60% of Finnish adults are overweight (Table 2). If 6 kg of weight loss (average reduction in trials) were achieved in this segment of the population, hypertension in Finland would decline by 5.7% (ie $0.60 \times 9.4\%$). Dividing this figure by the prevalence of hypertension yields a PAR% of 12%. Overweight made a substantial contribution to the prevalence of hypertension in all populations, with PAR% between 11% (Italy) and 25% (USA). PAR% was 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 was also small (<5%), except for the USA (8%). PAR% varied among populations with respect to inadequate intake of magnesium (2–8%), fish fatty-acids (3–16%) and coffee (0–9%).

Discussion

This study shows that diet and lifestyle have a substantial impact on the prevalence of hypertension in Western societies, with different ranking of risk factors within populations. Overweight, physical inactivity, high salt intake and low potassium intake appeared to be the major contributors to hypertension in Western societies.

BP estimates in this study were obtained from randomised trials and are likely to be causal and fully attributable to the specific risk factors. We calculated expected changes in the prevalence of hypertension after a uniform shift of the population BP distribution to a lower level. However, it should be noted that this is a simplified model. Subject characteristics may influence BP response to intervention; for example, BP sensitivity to sodium is probably larger in populations with elevated BP, which we did not take into account in our study. Also, PAR% should be interpreted with caution when based on BP estimates with wide confidence intervals, for example for magnesium, for which the number of trials in meta-regression analysis was small. PAR% in our study may be too conservative since they were calculated only for risk groups, whereas other people may also benefit from prevention. To illustrate, weight loss is known to also reduce BP in normal-weight subjects. Furthermore, we would like to emphasise that PAR% for different risk factors may not be fully additive owing to, for example, risk factor interactions. In case of hypertension,

which has a multifactorial aetiology, the sum of PAR% for all risk factors combined could easily exceed 100%.

The total BP effect of all dietary and lifestyle interventions in this study was more than 20 mmHg. Cardiovascular risk strongly increases with BP, even in the 'normal' range.¹⁰ 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.¹¹ We need to point out that prevalences of hypertension in our study are overestimated due to single BP measurements in epidemiological surveys.¹² However, this is unlikely to have had a large effect on PAR% and the ranking of risk factors in the different populations. Also, there are several uncertainties with regard to uniform assessment of risk factor prevalences in different populations. Data on intake of fish fatty-acids (eicosapentaenoic acid and docosahexaenoic acid) on a population-based level were scanty and the number of people consuming less than 200 mg/day could only roughly be estimated. For coffee, uniform assessment was hampered by differences in cup size among populations. We considered a cup of coffee to be equal to 125 ml, but for Italy cup size may be only 50 ml. A study by Ferraroni *et al*¹³ among 395 Italian adults (median age of 50 years) showed an average coffee intake of 100–110 ml per day, which is in line with the low intake that we report. In a case-control study by Tavani *et al*¹⁵ in Northern Italian women, however, over 10% consumed four or more cups of coffee per day,¹⁴ and in another case-control study in this population¹⁵ this prevalence exceeded 20% (cup size in these studies was not reported).

From this study, we conclude that effective dietary and lifestyle interventions could make a major contribution to the prevention of hypertension in Western societies. For several risk factors, the impact on hypertension varied among populations, which is important for setting priorities in preventive strategies. Hypertension itself, however, is not the outcome of primary interest. More research is needed to assess the total impact of diet and lifestyle on (cardiovascular) morbidity and mortality. Public health research in this field may be facilitated by standardised data collection on dietary and lifestyle factors in different countries.

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References

- 1 Jekel JF, Elmore JG, Katz DL (eds). Assessment of risk in epidemiologic studies. In: *Epidemiology, Biostatistics, and Preventive Medicine*, 2nd edn. WB Saunders Company: Philadelphia, 1996, pp 74–82.
- 2 Geleijnse JM *et al*. Blood pressure response to fish oil supplementation: metaregression analysis of randomized trials. *J Hypertens* 2002; **20**: 1493–1499.
- 3 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**: 471–480.
- 4 Neter JE *et al*. Influence of weight reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension* 2003; **42**: 878–884.
- 5 Geleijnse JM, Grobbee DE, Kok FJ. Impact of dietary and lifestyle factors on the prevalence of hypertension in Western populations. *Eur J Public Health* 2004; **14**: 235–239.
- 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 October 2, 2005].
- 7 U.S. 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 October 2, 2005].
- 8 Elliott P, Dyer A, Stamler R. The INTERSALT study: results for 24 h sodium and potassium, by age and sex. INTERSALT Co-operative Research Group. *J Hum Hypertens* 1989; **3**: 323–407.
- 9 McClelland GH Stanford University, Dept of Statistics Seeing Statistics, ©Duxbury Press: Stanford, CA, USA, 1999 [<http://psych.colorado.edu/~mcclella/java/normal/accurateNormal.html>; last accessed October 2, 2005].
- 10 Psaty BM *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**: 1183–1192.
- 11 Stamler J, Stamler R, Neaton JD. Blood pressure, systolic and diastolic, and cardiovascular risks. US population data. *Arch Intern Med* 1993; **153**: 598–615.
- 12 Kungel OH *et al*. Estimating the prevalence of hypertension corrected for the effect of within-person variability in blood pressure. *J Clin Epidemiol* 2000; **53**: 1158–1163.
- 13 Ferraroni M *et al*. Reproducibility and validity of coffee and tea consumption in Italy. *Eur J Clin Nutr* 2004; **58**: 674–680.
- 14 Tavani A, Negri E, La Vecchia C. Coffee intake and risk of hip fracture in women in northern Italy. *Prev Med* 1995; **24**: 396–400.
- 15 Tavani A *et al*. Risk factors for non-fatal acute myocardial infarction in Italian women. *Prev Med* 2004; **39**: 128–134.