

# Can a decline in the population means of cardiovascular risk factors reduce the number of people at risk?

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## Abstract

**Objective**—To prove the possibility of shifting distribution of cardiovascular risk factors for a whole population over time and thereby to influence the prevalence of the corresponding disease states, according to the theory stated by Geoffrey Rose in 1985.

**Design**—Examination of standardised data from the German Cardiovascular Prevention Study (GCP), a seven year long, population-based, multicentre intervention trial, concerned with decreasing risk factors for cardiovascular disease.

**Setting and subjects**—three cross sectional surveys of a population 25 to 69 years old in six study regions, and three nationwide cross sectional surveys in the former West Germany in 1984, 1988 and 1991.

**Main outcome measures**—The relation between the population mean for systolic and diastolic blood pressure, total serum cholesterol, body mass index, and alcohol intake, and the prevalence of the corresponding disease states, as are systolic ( $\geq 140$  and  $\geq 160$  mm Hg) and diastolic hypertension ( $\geq 90$  and  $\geq 95$  mm Hg), hypercholesterolaemia ( $\geq 250$  and  $\geq 300$  mg/dl), overweight (body mass index  $\geq 30$  kg/m<sup>2</sup>), and heavy drinking (weekly alcohol intake  $\geq 300$  g/week). Results are expressed as linear regression equations and Pearson correlation coefficients.

**Results**—The correlation between the mean population values and prevalence of disease was close for blood pressures and body mass index. The Pearson coefficients, corrected for the influence of values increased above borderlines, were 0.86 and 0.81 respectively for systolic blood pressure, 0.88 and 0.91 for diastolic blood pressure, 0.28 and 0.52 for cholesterol, and 0.86 for the body mass index. The coefficient for alcohol intake was 0.55.

**Conclusions**—It seems possible to shift the risk distribution of a population for some physiological parameters over time with the effect of changing the disease prevalence. This strategy can be used successfully for specific preventive measures, as was strongly advocated by Geoffrey Rose. (J Epidemiol Community Health 2001;55:179–184)

Arterial hypertension was proved to widely vary among different geographical regions as well as among different cultural settings. While

nomads in Kenya<sup>1</sup> and the Yanomamo Indians in Brazil hardly know this disease, it is quite common in the Western industrialised world.<sup>2</sup>

By looking at the distribution of blood pressure values of these populations, you can see that not only the number of people in the abnormal range differs, but that the whole distribution curve is shifted as well. In this case, the population means of blood pressure and the prevalence of disease states of these—that is, hypertensive blood pressure—will correlate.

In 1990 Geoffrey Rose examined the relation between the population means of several health indicators, like blood pressure, body mass index, alcohol intake and sodium intake, and the corresponding prevalence rates of disease states operationally defined as being above certain values. Using data from a large intercultural epidemiological study (Intersalt) with 52 distinct populations,<sup>2</sup> Rose and Day<sup>3</sup> showed that the population means for physiological as well as for behavioural parameters have an almost linear relation to the prevalence of the corresponding disease states. In this intercultural comparison, populations with higher means also had higher prevalence rates of the diseases. From these results, the authors drew the conclusion that it is possible to predict the number of diseased people in a given population from the mean of the corresponding health indicators in the same population. They stated that the tail of the risk distribution—that is, the high risk people—belongs to its body—that is, to the “normal” ones. Therefore, they emphasised the collective responsibility of the society for the number of its deviant people.

From this concept, two main preventive strategies can be deduced. The commonly used and traditional strategy in clinical medicine that consists of identifying people at high risk to be treated with appropriate means to reduce individual susceptibility. While this offers a favourable benefit to risk ratio to the patient and makes a cost effective use of medical resources, the underlying causes of the incidence rate within a population are not tackled.

Its aim is to cut off the high end tail of a population's risk distribution by treating only those susceptible to a certain disease.

This so called high risk strategy contrasts with the population strategy, where one tries to change the behaviour or characteristics of the whole population. To this aim, the risk distribution of the entire population has to be moved to a lower risk range. This is a radical approach that has an unexpectedly high potential of prevention for the population, but on the

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other hand offers only a small benefit to risk ratio to each person. The immediate benefit of the high risk approach can be appreciated easily by the patient and the doctor. The population strategy, however, lacks a large benefit for most people, who would have been well anyway. Its great advantage and challenge lies in the attempt to change the view of what is normal within a population by accepting the population's responsibility for those with deviant values.<sup>4,5</sup>

So far, the underlying theory has been proved only by intercultural comparison of observational data. This paper attempts to add one more piece of evidence to the relation between induced changes of the population mean and prevalence of disease within a population over time. Therefore, the hypothesis of Rose was tested in an intervention study\*. To this end, we make use of the multiple regional and national surveys in the German Cardiovascular Prevention Study (GCP).<sup>6</sup>

### Methods

The GCP was a seven year long, population-based, multicentre intervention trial aiming at reducing risk factors for cardiovascular diseases. In addition to a high risk approach to prevention, a population strategy was also applied. Broad preventive programmes were implemented in overlapping waves in six regions in West Germany. The programmes consisted of educational, behavioural, and medical advice on eating and drinking habits, early detection of hypertension and hypercholesterolaemia, exercise, non-smoking campaigns, and stress reduction. For further information on the design refer to the GCP Study Group<sup>7</sup> and Forschungsverbund DHP (Hrsg).<sup>8</sup>

\* Our paper follows closely the famous publication of Geoffrey Rose in 1990,<sup>3</sup> we therefore devote it to his memory.

From a total population, aged 25 to 69, three cross sectional representative surveys have been sampled (see the tables in appendices 1 and 2) in each of the six intervened regions (Regional Health Surveys) as well as from the former West Germany as a whole (excluding the intervention regions) providing the reference population (National Health Surveys): at baseline (1984), midpoint (1988), and at the end of the programme (1991). The response rates for the national samples varied between 66.7%, 71.4%, and 69.0% respectively, and for the pooled intervention samples between 74.5%, 73.0%, and 71.6%.<sup>6</sup> For statistical analyses a weighting factor based on the census data of 1987 was applied to adjust for slight deviations in the national samples of the intended age, sex, community size and federal state distribution.<sup>6</sup> Among others, measurements of arterial blood pressure, cholesterol, and body mass were taken. Behavioural items included daily alcohol intake.

The population mean values of each of the three measurements have been calculated for each region and the national survey regarding the following parameters: systolic blood pressure, diastolic blood pressure, total serum cholesterol, body mass index, and weekly alcohol intake.

Abnormal deviance for the purpose of this analysis—also in order to be comparable with Rose and Day<sup>3</sup>—is defined as follows (for comparison see also the centiles in appendix 2): systolic hypertension as systolic blood pressure  $\geq 140$  and  $\geq 160$  mm Hg, diastolic hypertension as diastolic blood pressure  $\geq 90$  and  $\geq 95$  mm Hg, hypercholesterolaemia as total serum cholesterol  $\geq 250$  and  $\geq 300$  mg/dl, overweight as body mass index  $\geq 30$  kg/m<sup>2</sup>, and heavy drinking as alcohol intake  $\geq 300$  g/week.

Table 1 Correlation coefficients and linear regression slopes based on the original GCP data and a corrected dataset (see text)

Independent variables (mean values)	Dependent variables (% prevalence of deviance)	Original GCP data			Corrected GCP dataset			Correlation coefficients according to Rose et al <sup>3</sup>	
		Pearson correlation coefficients	Slopes (linear regression)	95% Confidence intervals	Pearson correlation coefficients	Slopes (linear regression)	95% Confidence intervals		
Systolic blood pressure	Systolic hypertension ( $\geq 140$ mm Hg)	all	0.97	1.71	1.51 / 1.91	0.86	2.90	2.07 / 3.73	0.76
		male	0.95	1.93	1.61 / 2.24	0.78	3.00	1.85 / 4.16	
	female	0.98	1.48	1.33 / 1.64	0.90	2.65	2.03 / 3.26	—	
	Systolic hypertension ( $\geq 160$ mm Hg)	all	0.91	0.67	0.53 / 0.82	0.81	0.80		0.52 / 1.07
		male	0.94	0.63	0.52 / 0.75	0.89	0.78		0.59 / 0.97
	female	0.90	0.77	0.59 / 0.96	0.72	0.87	0.46 / 1.27		
Diastolic blood pressure	Diastolic hypertension ( $\geq 90$ mm Hg)	all	0.97	2.35	2.06 / 2.63	0.88	3.40	2.54 / 4.27	—
		male	0.96	2.61	2.25 / 2.96	0.84	3.55	2.46 / 4.64	
		female	0.96	2.12	1.83 / 2.42	0.87	2.91	2.10 / 3.72	
	Diastolic hypertension ( $\geq 95$ mm Hg)	all	0.96	1.38	1.20 / 1.56	0.91	1.76	1.39 / 2.13	—
		male	0.93	1.56	1.27 / 1.85	0.85	1.88	1.32 / 2.44	
		female	0.95	1.20	1.01 / 1.39	0.88	1.44	1.06 / 1.81	
Cholesterol	Hypercholesterolaemia ( $\geq 250$ mg/dl)	all	0.95	0.89	0.75 / 1.03	0.28	0.49	-0.32 / 1.31	—
		male	0.94	0.84	0.69 / 0.99	0.40	0.62	-0.08 / 1.36	
		female	0.95	0.89	0.74 / 1.04	0.15	0.27	-0.59 / 1.14	
	Hypercholesterolaemia ( $\geq 300$ mg/dl)	all	0.83	0.38	0.26 / 0.50	0.52	0.32	0.07 / 0.58	—
		male	0.68	0.28	0.13 / 0.42	0.36	0.16	-0.07 / 0.40	
		female	0.84	0.42	0.29 / 0.55	0.46	0.33	0.27 / 0.64	
Body mass index	Overweight ( $\geq 30$ kg/m <sup>2</sup> )	all	0.97	5.97	5.21 / 6.74	0.86	9.03	6.44 / 11.62	0.78
		male	0.93	5.71	4.61 / 6.82	0.78	7.29	4.50 / 10.08	
		female	0.95	5.88	4.96 / 6.80	0.75	8.94	5.18 / 12.71	
Alcohol intake	Heavy drinking ( $\geq 300$ g/week)	all	0.95	0.18	0.15 / 0.21	0.55	0.24	0.07 / 0.42	0.64
		male	0.98	0.19	0.17 / 0.21	0.83	0.37	0.25 / 0.49	
		female	0.83	0.14	0.10 / 0.19	0.22	0.06	-0.07 / 0.20	
		all*	0.77	13.44	8.02 / 18.88	0.32	7.17	-2.90 / 17.24	
		male*	0.91	19.39	15.24 / 23.53	0.73	20.70	11.41 / 29.99	
		femal*	0.56	5.39	1.54 / 9.24	0.24	2.53	-2.43 / 7.48	

\*Log(1+units).

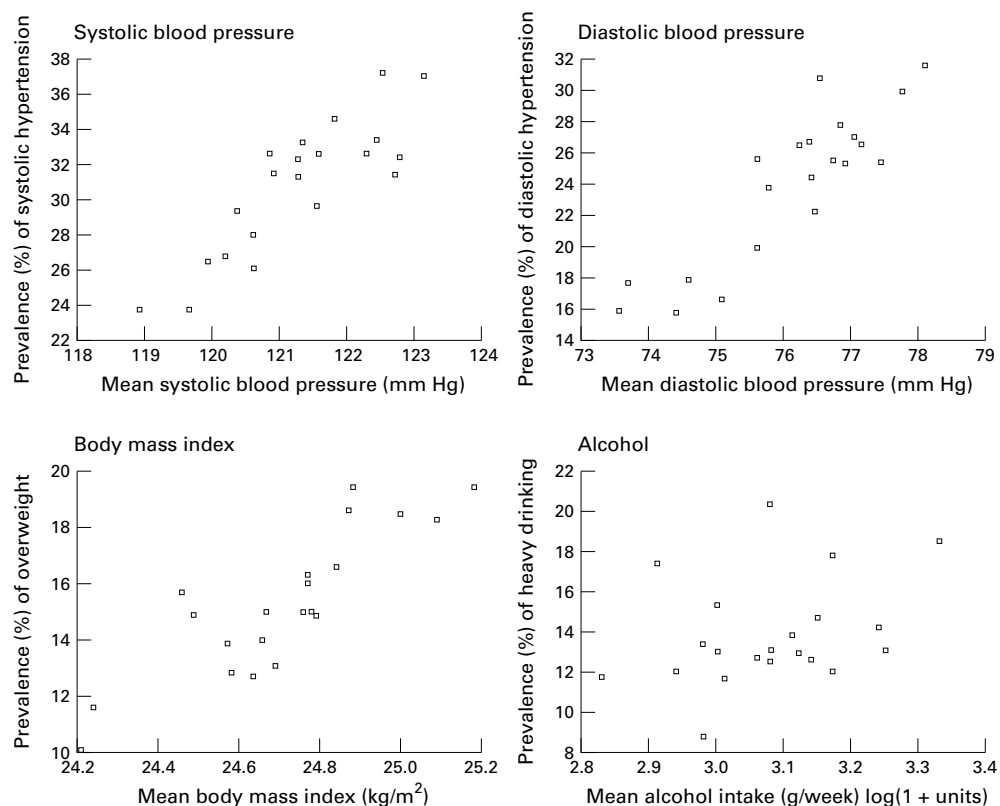


Figure 1 Scatter plots (corrected database) of the population means and the prevalence of deviant (high) values in seven study populations within Germany (1984–1988–1991): systolic hypertension ( $\geq 140$  mm Hg), diastolic hypertension ( $\geq 90$  mm Hg), overweight ( $\geq 30$  kg/m<sup>2</sup>), and heavy drinking ( $\geq 300$  g/week).

Prevalence of the abnormal deviance was calculated in percentages.

Datasets of the corresponding population mean and disease prevalence were formed and the Pearson and Spearman correlation coefficients calculated (as both were very close only the Pearson coefficients are given). For alcohol consumption because of its highly skewed distribution a transformation ( $\log(1+\text{units})$ ) was used in addition (25.6 to 35.6% did not indicate any relevant consumption of alcohol).

As the high values that constitute the prevalence of the disease also contribute to the calculation of the population mean, a certain degree of “autocorrelation” (Rose and Day<sup>3</sup>) enlarges the correlation coefficient. To correct for this effect, we also calculated the correlation coefficients after neglecting the high values (above the cut points as defined for prevalence) in the calculation of the population mean. Linear regression for each dataset was performed to predict the number of deviant people from the population mean. Correlation coefficients and regression lines for each calculation

method (using the original as well as the corrected data) were compared to estimate the size of this effect, following Geoffrey Rose’s approach closely.

## Results

Twenty one datasets for each of the variables were analysed. Table 1 shows firstly, in the original dataset, the Pearson coefficients for the correlation between mean and prevalence. Very close relations can be seen with Pearson correlation coefficients varying from 0.97, for example, for the body mass index to 0.77 for the log transformed alcohol consumption, all of them highly significant ( $p < 0.01$ , two tailed test).

Secondly, the Pearson correlation coefficients and the slopes of the linear regression lines for the corrected database are shown, where the effect of increased values (above defined cut points) has been removed from the calculation of the mean. This leads to a decrease in the size of previously “falsely” high correlation coefficients. At the same time the slopes of the corresponding regression lines

Table 2 Theoretical reduction in prevalence of disease after implementing population based preventive strategies, resulting in reduction of the population mean (corrected data, see text)

Variable	Reduction in mean values	Prevalence (%) of deviance	Theoretical reduction in prevalence (%) according to reduction in mean values	Reduction (%) according to Rose <i>et al.</i> <sup>3</sup> see footnote*
Systolic blood pressure ( $\geq 140$ mm Hg)	5 mm Hg	31.4	14.5	5.0
Systolic blood pressure ( $\geq 160$ mm Hg)	5 mm Hg	9.7	4.0	—
Diastolic blood pressure ( $\geq 90$ mm Hg)	2.5 mm Hg	24.6	8.5	—
Diastolic blood pressure ( $\geq 95$ mm Hg)	2.5 mm Hg	12.2	4.4	—
Body mass index ( $\geq 30$ kg/m <sup>2</sup> )	1 kg/m <sup>2</sup>	15.7	9.0	6.9
Alcohol intake ( $\geq 300$ g/week)	15 g/week	13.8	3.7	4.5

\*Based on table III in Rose *et al.*<sup>3</sup>: for alcohol intake the value used by Rose of 0.24% per ml/week has been increased by 0.8 to obtain g/week.

increase in comparison with the slopes in the original data. This can be explained by the different effects that the treatment of high values in our case has on the independent and dependent variable. While the spread of the independent variable, the population mean, is reduced after neglecting the high values in its calculation, the spread of the dependent variable, the prevalence of disease, remains unchanged. This leads to an increase of the slope. An exemption is the relation of hypercholesterolaemia and cholesterol, and heavy drinking and alcohol consumption (log transformed).

The new correlation coefficients for systolic blood pressure are 0.86 and 0.81 respectively, for diastolic blood pressure 0.88 and 0.91, and for body mass index 0.86. Their size still suggests a close relation between the height of the population mean and the frequency of the corresponding disease states hypertension and overweight. There is only a weak relation between hypercholesterolaemia and cholesterol and between mean alcohol intake and prevalence of heavy drinking. In this case the overall correlation was mainly attributable to the higher values of the distribution leading to a considerable overestimation.

Figure 1 shows the relation between prevalence of selected disease states and means of the population neglecting the high values for each variable as a scatter plot. In the studied range there exists a linear, positive relation between prevalence of hypertension and mean systolic and diastolic pressure, between prevalence of overweight and mean body mass index, however not between heavy drinking and mean alcohol intake per week if the logarithmic transformation of the data is used.

Table 2 shows the theoretical change in prevalence of disease after implementing population based preventive strategies with a defined effect. Reducing the mean systolic blood pressure of the population by 5 mm Hg, a reduction in prevalence of systolic hypertension can be observed that amounts to 14.5% for the lower borderline of  $\geq 140$  mm Hg and to 4.0 for a borderline  $\geq 160$  mm Hg. As can be expected the effect is stronger for lowering the diastolic blood pressure. Therefore we choose a reduction of the population mean only by 2.5 mm Hg leading to a fall in the prevalence of diastolic hypertension of 8.5% (borderline  $\geq 90$  mm Hg) and of 4.4% (borderline  $\geq 95$  mm Hg) respectively. This is a substantial decrease, as, for example, the prevalence of diastolic hypertension  $\geq 95$  mm Hg in our study ranged between 7% and 16% (see appendix 1). A reduction of the same order was found for overweight and body mass index (9.0% for a decrease of 1 kg/m<sup>2</sup> in population mean), and for heavy drinking and weekly alcohol intake (3.7% for a weekly alcohol intake reduced by 15 g).

### Discussion

While we observed high correlation coefficients for all variables in the uncorrected database, results changed after eliminating the contribution of values increased above defined border-

#### KEY POINTS

- It is possible to shift the risk distribution of a population to a lower range thereby reducing the number of people at risk.
- Our data show this relation for physiological parameters such as arterial blood pressure and body mass index.
- These findings support Rose's concept of the great potential of the population strategy.

lines. Yet the relations between blood pressure and hypertension as well as between body mass index and overweight were still very strong and even closer than those found by Rose and Day, whereas the relation between heavy drinking and weekly alcohol intake was somewhat weaker especially after logarithmic transformation (table 1). We could also confirm a weak relation between hypercholesterolaemia (cut point  $\geq 300$  mg/dl) and cholesterol (correlation coefficient 0.52 versus 0.83 at  $p < 0.01$  for the uncorrected data).

Table 2 shows the results found by Rose and Day<sup>3</sup> in comparison with our findings. The percentage reductions they calculated from a very different set of populations across the world compare well for alcohol intake and body mass index but, in the German data, are exceeded clearly for systolic blood pressure. This is probably attributable to the higher age range in the German population (ages 25–69) and therefore much higher prevalence of hypertension—that is, 31.4% for systolic borderline hypertension (see table 2). The relative change of prevalence therefore is reasonably similar, for example, 46.2% in our data compared with 33.3% in the corrected data of Rose.

A reason for observed differences in all variable pairs is the studied range of values and the concomitant precision of the estimates. While Rose and Day examined widely different populations with great variations in population mean and prevalence rates ranging from 0% to 50%, our study populations were relatively homogenous, as they all came from the same cultural background. The range of mean systolic blood pressure in Rose's study varied from 96 to 132 mm Hg. In our study we examined only the range between 124 and 130 mm Hg. This can account for a different slope in the calculation of the regression line. The same applies to the variables "weekly alcohol intake" and "body mass index". Our database covers only 15% respectively 8% of the range of the means of study populations as available to Rose.

A further source of bias is the method of measurement. While blood pressure and body mass index were measured by trained personnel, data on alcohol intake were self reported. As drinking of alcohol is subject to social stigma, there might be a tendency to underestimate your weekly alcohol consumption. This applies especially to heavy drinkers. Another reason why we found a weaker relation between weekly alcohol intake and prevalence of heavy drinking might be the relatively short duration for a radical



Appendix 1 Basic data, German Cardiovascular Prevention Study (GCP)

Region	Phase	Total n=	Systolic blood pressure		Diastolic blood pressure		Cholesterol		Body mass index		Alcohol	
			Prevalence $\geq 160$ mm Hg	Mean (SD)	Prevalence $\geq 95$ mm Hg	Mean (SD)	Prevalence $\geq 250$ mg/dl	Mean (SD)	Prevalence $\geq 30$ kg/m <sup>2</sup>	Mean (SD)	Prevalence $\geq 300$ g/week	Mean (SD)
			mm Hg		mm Hg							log(1+units)
Reference	1984	4790	9.5	131.7 (20.1)	13.6	81.8 (12.4)	34.2	233.5 (49.6)	16.3	26.1 (4.3)	13.8	144.8 (166.7)
	1988	5335	10.6	133.4 (19.6)	11.9	81.4 (12.0)	35.9	235.5 (48.6)	16.0	26.1 (4.4)	12.0	131.8 (160.2)
	1991	5311	10.5	133.2 (19.6)	12.5	81.8 (11.6)	36.2	236.8 (47.1)	18.6	26.5 (4.5)	12.7	137.7 (159.0)
IV 1*	1984	1800	12.3	135.2 (20.0)	12.4	81.0 (12.6)	32.9	231.0 (48.8)	13.1	25.8 (4.0)	12.6	138.8 (163.7)
	1988	1270	9.0	130.2 (19.3)	8.2	77.7 (12.6)	38.4	238.2 (47.3)	15.0	26.0 (4.2)	13.0	129.6 (153.9)
	1991	1282	7.5	128.0 (19.1)	7.7	77.9 (11.7)	32.7	233.7 (44.3)	15.0	26.0 (4.3)	8.8	116.1 (145.9)
IV 2	1984	1836	9.4	131.6 (19.7)	14.8	82.7 (12.1)	37.2	238.0 (50.2)	13.9	25.8 (4.2)	15.3	149.6 (182.0)
	1988	1389	8.1	131.7 (20.0)	14.9	82.5 (12.2)	36.2	236.4 (49.2)	12.7	25.7 (4.0)	13.4	135.0 (166.5)
	1991	1423	7.5	129.7 (19.3)	8.2	78.6 (11.5)	34.0	233.1 (46.2)	14.9	26.1 (4.3)	11.7	126.3 (159.7)
IV 3	1984	1950	6.5	127.2 (19.0)	6.8	77.3 (12.4)	28.7	228.6 (47.3)	15.0	26.0 (4.1)	20.3	172.3 (184.7)
	1988	1572	7.6	129.3 (19.1)	7.6	78.3 (11.6)	33.1	233.5 (46.5)	16.6	26.3 (4.5)	17.8	162.7 (171.9)
	1991	1552	10.4	133.8 (19.5)	11.9	81.9 (11.3)	29.8	231.3 (44.2)	18.3	26.6 (4.2)	12.9	144.7 (163.7)
IV 4	1984	1791	9.2	130.9 (20.6)	14.6	82.6 (12.2)	33.8	231.9 (48.4)	10.1	25.1 (4.0)	18.5	172.9 (183.4)
	1988	1437	10.2	132.4 (20.3)	13.9	82.5 (11.8)	36.3	237.2 (50.1)	11.6	25.2 (4.1)	13.1	145.6 (158.3)
	1991	1313	8.6	129.4 (20.2)	11.6	80.8 (12.2)	30.4	230.5 (45.2)	14.9	25.8 (4.4)	12.0	139.4 (158.7)
IV 5	1984	2003	10.0	132.6 (19.8)	15.1	83.6 (11.7)	32.7	232.6 (48.5)	12.9	25.6 (3.9)	14.7	149.5 (162.4)
	1988	1427	9.7	132.3 (19.7)	11.6	81.0 (11.5)	37.9	239.4 (47.9)	14.0	25.9 (4.5)	12.5	137.5 (160.8)
	1991	1533	9.0	132.8 (19.2)	12.3	82.4 (11.6)	30.8	231.4 (45.3)	15.7	25.9 (4.5)	14.2	153.3 (168.7)
IV 6	1984	2168	12.0	135.0 (20.6)	15.9	84.0 (11.4)	33.2	233.7 (48.5)	19.4	26.4 (4.3)	17.4	157.7 (182.5)
	1988	1648	11.8	133.9 (21.1)	15.0	82.0 (12.3)	35.8	235.7 (46.7)	19.4	26.7 (4.2)	13.1	138.5 (154.4)
	1991	1533	7.6	131.3 (18.9)	9.5	79.7 (11.5)	30.4	230.6 (45.7)	18.5	26.5 (4.3)	11.6	133.0 (154.8)
IV 1-6 pooled	1984	11548	10.0	132.1 (20.1)	13.3	81.9 (12.3)	33.0	232.6 (48.7)	14.2	25.8 (4.1)	16.5	156.9 (177.2)
	1988	8734	9.5	131.7 (20.0)	11.9	80.7 (12.1)	36.2	236.6 (47.9)	15.0	26.0 (4.3)	13.9	142.0 (161.5)
	1991	8636	8.5	131.0 (19.4)	10.2	80.3 (11.8)	31.3	231.7 (45.2)	16.3	26.2 (4.3)	12.0	136.1 (159.5)

\*IV = intervention region.

preventive strategy. The group of heavy drinkers probably comprises a high percentage of addicted alcoholics who cannot quit or reduce alcohol intake easily. In this case the population's risk distribution does not simply shift to the left but also increases skewness to the right. This phenomenon can lead to the observed independence of the studied variables. In this case the incidence rate of alcoholism would be a better estimate of short-term changes.

Despite the possible sources of bias discussed above, our study basically yields the same results as were deduced by Rose, to a degree that is rather astonishing, especially as a mixed intervention strategy—directed to the general population as well as to the high risk people—has been applied in the GCP. Earlier analyses<sup>9,10</sup> even seem to show that the high risk oriented GCP intervention had a stronger impact than the population strategy. For example, using logistic regression coefficients for West European men from the ERICA Study,<sup>11</sup> reductions of the means of systolic blood pressure and total cholesterol by 2%, and of smoking by 7% as achieved in the GCP, led to a lowering of the six year CHD mortality by about 10%. In comparison a simulated but carefully realistic reduction of the prevalence of these risk factors by 10% resulted in a decrease of the CVD mortality between 10% and 20% depending on the borderlines chosen.

We believe to have shown that differences induced artificially through active intervention may behave like differences between free living populations, and follow closely Rose's hypothesis stating that "the tail belongs to the body".<sup>3</sup> It therefore is plausible that multi-factorially determined parameters such as arterial blood pressure and body mass index can be influenced by what is considered "normal" in a society. If most people begin to increase their amount of exercising or reduce their salt and fat intake, this will have an impact even on those who do not actively try to change their lifestyle. As was advocated by Rose, the implications for society and the medical community are great. We have to accept a collective responsibility for disease and abnormality by what we consider normality in our life style. While this might be more readily accepted for behavioural parameters such as aggression, it also holds true for physiological parameters.

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Appendix 2 Basic data, German Cardiovascular Prevention Study (GCP)—centiles (P20, P40, P60, P80)

Region	Phase	Total n =	Systolic blood pressure (mm Hg)				Diastolic blood pressure (mm Hg)				Cholesterol (mg/dl)				Body mass index (kg/m <sup>2</sup> )				Alcohol (g/week)			
			P20	P40	P60	P80	P20	P40	P60	P80	P20	P40	P60	P80	P20	P40	P60	P80	P20	P40	P60	P80
Reference	1984	4790	114	124	134	146	72	78	84	92	196.6	216.9	240.9	271.4	22.6	24.8	26.6	29.2	0	70.0	136.5	266.0
	1988	5335	118	126	136	148	72	78	84	90	193.7	220.4	243.6	273.7	22.6	24.7	26.8	29.3	0	66.5	122.5	220.5
	1991	5311	116	126	134	148	72	78	84	92	196.0	221.2	244.4	273.8	22.7	24.9	27.0	29.7	0	70.0	133.0	245.0
IV 1*	1984	1800	118	128	138	150	68	74	80	88	191.4	215.8	239.5	270.7	22.7	24.6	26.4	28.7	0	70.0	133.0	231.0
	1988	1270	114	124	132	144	68	74	80	88	196.8	223.1	247.6	276.8	22.7	24.7	26.6	29.2	0	66.5	122.5	220.5
	1991	1282	112	120	130	142	68	74	80	86	195.3	220.4	241.3	268.0	22.4	24.5	26.4	29.0	0	52.5	105.0	189.0
IV 2	1984	1836	116	124	134	148	73	80	86	92	197.8	223.5	246.9	278.4	22.5	24.4	26.2	28.9	0	70.0	136.5	245.0
	1988	1389	116	124	134	146	74	80	86	92	197.6	221.1	245.5	274.9	22.5	24.4	26.3	28.6	0	70.0	122.5	220.5
	1991	1423	114	124	132	146	70	76	82	88	195.3	219.6	243.2	271.4	22.6	24.8	26.6	29.2	0	52.5	119.0	220.5
IV 3	1984	1950	110	120	130	142	68	74	80	88	189.9	213.9	236.6	264.5	22.8	24.8	26.5	29.1	0	70.0	175.0	308.0
	1988	1572	114	122	132	144	68	76	81	88	195.2	216.9	242.0	269.9	22.6	24.9	26.9	29.5	0	70.0	140.0	280.0
	1991	1552	118	126	136	150	72	78	84	92	196.0	218.1	238.6	266.4	23.1	25.2	27.1	29.8	0	70.0	122.5	259.0
IV 4	1984	1791	114	124	132	148	72	78	86	92	191.4	219.6	243.2	271.8	21.9	23.8	25.6	28.2	0	70.0	154.0	280.0
	1988	1437	116	126	134	148	72	80	84	92	195.6	220.9	245.3	279.3	21.9	23.9	25.9	28.2	0	70.0	140.0	259.0
	1991	1513	114	122	132	144	70	78	84	92	192.2	213.4	238.6	270.7	22.2	24.3	26.2	28.8	0	70.0	136.5	245.0
IV 5	1984	2003	116	126	136	148	74	80	86	92	192.2	217.3	241.7	270.3	22.4	24.3	26.3	28.7	0	70.0	140.0	273.0
	1988	1427	116	126	136	148	72	78	84	90	199.3	223.5	246.7	277.4	22.4	24.4	26.4	28.9	0	70.0	122.5	238.0
	1991	1533	116	126	134	148	72	78	84	90	193.3	216.5	237.0	266.8	22.0	24.1	26.5	29.2	0	70.0	140.0	248.5
IV 6	1984	2168	118	128	138	150	74	80	86	92	193.3	218.5	240.5	271.8	22.9	24.9	27.0	29.9	0	70.0	140.0	280.0
	1988	1648	116	126	136	150	72	78	84	92	196.4	221.9	246.3	275.3	23.3	25.3	27.4	30.0	0	70.0	140.0	245.0
	1991	1533	116	124	134	146	70	76	84	90	191.0	217.3	238.2	266.4	23.1	25.1	26.9	29.6	0	70.0	136.5	238.0
IV 1–6 pooled	1984	11548	116	124	134	148	72	78	84	92	192.6	217.7	241.3	271.4	22.5	24.5	26.4	28.9	0	70.0	140.0	280.0
	1988	8734	114	124	134	146	70	78	84	90	196.8	221.1	245.5	275.3	22.5	24.6	26.6	29.2	0	70.0	136.5	245.0
	1991	8636	114	124	132	146	70	78	82	90	193.7	217.7	239.3	268.0	22.6	24.7	26.6	29.3	0	70.0	122.5	231.0

\* = intervention region.

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