

ORIGINAL INVESTIGATIONS

# Low-Risk Diet and Lifestyle Habits in the Primary Prevention of Myocardial Infarction in Men

## A Population-Based Prospective Cohort Study

Agneta Åkesson, PhD, Susanna C. Larsson, PhD, Andrea Discacciati, MSc, Alicja Wolk, DMSc



### ABSTRACT

**BACKGROUND** Adherence to a combination of healthy dietary and lifestyle practices may have an impressive impact on the primary prevention of myocardial infarction (MI).

**OBJECTIVES** The aim of this study was to examine the benefit of combined low-risk diet and healthy lifestyle practices on the incidence of MI in men.

**METHODS** The population-based, prospective cohort of Swedish men comprised 45- to 79-year-old men who completed a detailed questionnaire on diet and lifestyle at baseline in 1997. In total, 20,721 men with no history of cancer, cardiovascular disease, diabetes, hypertension, or high cholesterol levels were followed through 2009. Low-risk behavior included 5 factors: a healthy diet (top quintile of Recommended Food Score), moderate alcohol consumption (10 to 30 g/day), no smoking, being physically active (walking/bicycling  $\geq 40$  min/day and exercising  $\geq 1$  h/week), and having no abdominal adiposity (waist circumference  $< 95$  cm).

**RESULTS** During 11 years of follow-up, we ascertained 1,361 incident cases of MI. The low-risk dietary choice together with moderate alcohol consumption was associated with a relative risk of 0.65 (95% confidence interval [CI]: 0.48 to 0.87) compared with men having 0 of 5 low-risk factors. Men having all 5 low-risk factors compared with those with 0 low-risk factors had a relative risk of 0.14 (95% CI: 0.04 to 0.43). This combination of healthy behaviors, present in 1% of the men, could prevent 79% (95% CI: 34% to 93%) of the MI events on the basis of the study population.

**CONCLUSIONS** Almost 4 of 5 MIs in men may be preventable with a combined low-risk behavior. (J Am Coll Cardiol 2014;64:1299-306) © 2014 by the American College of Cardiology Foundation.

Coronary heart disease (CHD) incidence and mortality have decreased in many areas of the world, yet the burden of disease remains high (1). Although pharmacological therapies

through lipid-lowering (2), and antihypertensive (3) treatments have proved efficacious in reducing coronary events, adherence to a healthy lifestyle still has an impressive impact (4-16). In contrast to drug

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## ABBREVIATIONS AND ACRONYMS

**CHD** = coronary heart disease

**CI** = confidence interval

**CVD** = cardiovascular disease

**FFQ** = food frequency  
questionnaire

**MI** = myocardial infarction

**RR** = relative risk

therapies, medication-free strategies to help prevent CHD are mostly without the risk of side effects. Because population-wide strategies to shift the entire distribution of risk cannot rely on prescription medication, effective lifestyle-based prevention is essential.

SEE PAGE 1307

For CHD incidence, as much as 77% to 82% of the events were attributed to the lack of adherence to a low-risk lifestyle, consisting of 5 healthy diet and lifestyle choices in women (15,16). In American men, the corresponding attributable risk was 62%, and 57% among those men taking medication for hypertension or hypercholesterolemia (13). Yet, there is limited information available on community-based populations, especially in men. Moreover, the impact of lifestyle in the setting of contemporary guidelines and treatments is less well established.

We examined the benefit of a combined healthy diet and lifestyle on the incidence of myocardial infarction (MI) in a large population-based prospective cohort of healthy Swedish men. We estimated the burden of CHD that could potentially have been avoided had all men adhered to the low-risk practice of a healthy diet, moderate alcohol consumption, no smoking, being physically active, and avoiding abdominal adiposity. A separate analysis was performed among men with hypertension and high cholesterol.

## METHODS

**POPULATION.** In the autumn of 1997, all men born between 1918 and 1952 and residing in 2 counties in central Sweden received a questionnaire that included ~350 items concerning diet and other lifestyle factors (response rate was 49% of the source population). This large population-based cohort is representative of Swedish men 45 to 79 years of age in terms of age distribution, educational level, and prevalence of being overweight (17). Of the 48,850 men who returned the questionnaire, we excluded those with an erroneous or a missing national identification number. We further excluded men from the baseline population with a history of cancer (n = 2,592), ischemic heart disease or stroke (n = 5,405) on the basis of computerized linkage to national cancer and patient registries, as well as men with self-reported hypertension (n = 6,768) and self-reported high cholesterol (n = 2,527) and diabetes (n = 3,173) reported from national diabetes and patient registries and self-reports because these diagnoses may have caused a change in diet and lifestyle. Finally, we excluded those with implausible values for total energy intake (i.e.,

3 SDs from the log<sub>e</sub>-transformed mean energy intake; n = 329) and those with missing information on any of the 5 diet and lifestyle factors (n = 6,975). After these exclusions, 20,721 men remained for the main analyses. A separate analysis was performed of 7,139 men with hypertension and high cholesterol at baseline and with complete information on all exposure variables. The Regional Ethical Review Board at Karolinska Institutet approved the study, and return of the completed questionnaire was considered to imply informed consent.

**ASSESSMENT OF DIET AND LIFESTYLE FACTORS.** Diet was assessed using a self-administered semiquantitative food frequency questionnaire (FFQ) including questions on 96 commonly eaten foods. The validity of the FFQ compared with 14 24-h dietary recall interviews with 248 Swedish men 40 to 74 years of age were (mean Spearman correlation coefficient) 0.65 for macronutrients and 0.62 for total micronutrients (including supplements) (18).

A healthy diet was identified according to the Recommended Food Score, developed in 2000 by Kant et al. (4), for the National Health and Nutrition Examination Survey as a simple way to define diet quality by separating “healthy from less healthy” foods, on the basis of current knowledge and dietary guidelines. The Recommended Food Score is highly predictive of mortality (4,19) and includes foods with a beneficial effect on cardiovascular health (1,20,21), such as, fruits, vegetables, legumes, nuts, reduced-fat dairy products, whole grains, and fish. Adapted for our FFQs (19) (later expanded for the FFQ used in the present study [22]), a food score of 1 (adding up to a maximum of 25) was assigned for ≥1 servings per week of any of 3 reduced-fat dairy products, crisp bread, and whole grain bread, whereas for the remaining food items, the consumed frequency was at least 1 to 3 times per month. We considered those who scored in the highest quintile (scores of 23 to 25) of the Recommended Food Score as having a varied healthy diet (low-risk diet) on the basis of a post-hoc analysis (only the top quintile was associated with a statistically significant decreased risk of MI). The non-Recommended Food Score was based on 21 food items including red and processed meat, fried potatoes, solid fats, full-fat cheese, white bread and refined cereals, and various sweet foods (19).

The low-risk alcohol group comprised those men who consumed moderate amounts of alcohol (10 to 30 g/day) (21). We considered smoking status, level of physical activity, and abdominal adiposity as the 3 major modifiable nondietary low-risk factors (21). The information on smoking included details on the number of years since smoking was stopped. Never

smokers and those who quit smoking  $\geq 20$  years ago were classified as nonsmokers. Physical activity was assessed with a validated questionnaire (23). Participants reported their level of activity at work and home and during leisure time in the year before study enrollment, including questions on time spent walking or bicycling (6 predefined duration categories) and leisure time exercise (5 predefined duration categories). We considered a low-risk physical activity behavior to include both daily nonexercise physical activity (24,25) (walking/bicycling) and a more vigorous weekly exercise, pre-specified according to criteria previously use in women on the basis of the same questionnaire (15). Thus, nonsmoking men who walked or cycled for at least 40 min/day and exercised at least 1 h per week and who had a waist circumference  $< 95$  cm (26) comprised the low-risk group.

We obtained self-reported information on level of education, marital status, family history of MI in parents or siblings before the age of 60 years, and use of aspirin.

**ASCERTAINMENT OF MI.** The cohort was linked to the Swedish National Inpatient and Cause of Death Registers, considered to be  $>99\%$  complete. In a previous validation, the positive predictive value of MI was 98% to 100% (27). All incident cases of nonfatal and fatal MI (International Statistical Classification of Disease, 10th revision code I21) were ascertained from baseline (January 1, 1998 to December 31, 2009). Dates of death were ascertained through the Swedish Death Registry.

**DATA ANALYSIS.** Follow-up was censored at the date of first event of MI, death, or end of follow-up, whichever occurred first. Incident rates of MI were directly age-standardized to the age distribution of the cohort. We used Cox proportional hazards regression models with attained age (1-year units) as the underlying time scale with which to estimate relative risk (RR) with 95% confidence interval (CI), after confirming that the data did not violate the proportional hazard assumption (Schoenfeld's residual test). All associations between low-risk diet and lifestyle factors and MI were adjusted for the following established or proposed risk factors: age, level of education, marital status, family history of MI, use of aspirin, non-Recommended Food Score, and energy intake. Additional models were mutually adjusted for the low-risk behaviors. Assuming a causal relationship between risk factors and MI, we estimated the percent of cases that potentially could be prevented if men adhered to the low-risk practices for all men (the population-attributable

risk:  $p \text{ (RR-1)}/\text{RR}$ , where  $p$  is the prevalence of exposure among the cases) and for a given proportion of men (using generalized impact fraction [28]). All analyses were conducted using Stata software version 12 (StataCorp, College Station, Texas). The statistical tests were 2-sided and considered statistically significant at  $p$  values  $< 0.05$ .

**RESULTS**

During a mean of 11 years with 230,421 person-years of follow-up, we ascertained 1,361 incident cases of primary MI. The characteristics of men classified with a low-risk diet (top quintile of the Recommended Food Score [23 to 25 points]) compared with all other men are shown in Table 1. Overall, men with a low-risk diet were more likely to have attained a higher level of education and to not smoke and were less likely to live alone compared with men with lower scores of recommended foods. Table 2 gives the estimates of the RR of MI for each of the 5 modifiable lifestyle factors included in the low-risk behavior definition and the percent of the cohort in each risk

**TABLE 1 Baseline Age-Standardized Characteristics of 20,721 Men in the Cohort of Swedish Men by Categories of Recommended Food Score\***

	Recommended Food Score	
	Quintiles 1-4	Quintile 5
Mean age, yrs	59	57
Nondietary factors		
Post-secondary education	18	26
Married/cohabitating	83	90
Family history of myocardial infarction	16	15
Aspirin use	28	31
No smoking†	56	67
Walking/bicycling for $\geq 40$ min/day and exercising for $\geq 1$ h/week	29	32
Mean waist circumference, cm	95	94
Mean alcohol consumption, g/day	13	14
Dietary factors, mean		
Mean non-Recommended Food Score‡	15	17
Mean energy intake, kcal/day	2,700	2,900
Food intake/day, mean servings		
Vegetables	2.3	3.2
Fruits	1.2	1.6
Legumes	0.3	0.3
Whole grains	3.7	4.3
Fish/week	1.7	2.3

Values are % unless otherwise indicated. \*The Recommended Food Scores included foods with a beneficial effect on cardiovascular health. A score of 1, adding up to a maximum of 25, was assigned for regular consumption of fruits, vegetables, legumes, nuts, reduced-fat dairy products, whole grains, and fish. †Never smokers and those who quit smoking  $\geq 20$  years ago were classified as nonsmokers. ‡The non-Recommended Food Score included red and processed meat, fried potatoes, solid fats, full-fat cheese, white bread, refined cereals, and various sweet foods. Each of these foods with a regularly consumed frequency was assigned a food score of 1, adding up to a maximum of 21.

**TABLE 2 Relative Risk of Myocardial Infarction Associated With Modifiable Lifestyle Factors in 20,721 Men**

Lifestyle Factors	Low-Risk Group	RR* (95% CI)	
		Model 1	Model 2†
<b>Diet</b>			
Low risk (Recommended Food Score‡ top quintile vs. the rest)	177 (18)	0.76 (0.65-0.90)	0.82 (0.69-0.96)
<b>Alcohol consumption</b>			
Low risk (10-30 g/day vs. the rest)	448 (39)	0.92 (0.82-1.03)	0.89 (0.79-1.00)
<b>Smoking</b>			
Low risk (no smoking vs. the rest)	689 (58)	0.63 (0.57-0.70)	0.64 (0.57-0.71)
<b>Physical activity</b>			
Low risk (40 min/day walking/bicycling and 1 h vs. <1 h weekly exercise)	475 (30)	0.93 (0.83-1.05)	0.97 (0.86-1.09)
<b>Abdominal adiposity</b>			
Low risk (<95 vs. ≥95 cm waist circumference)	632 (52)	0.87 (0.78-0.97)	0.88 (0.78-0.98)

Values are n (%) unless otherwise indicated. \*Estimated from a multivariate Cox proportional hazards model adjusted for age (continuous), educational achievement (≤10, 10 to 12, >12 years), marital status (single, married/cohabiting, divorced, widowed), family history of myocardial infarction (yes/no), use of aspirin (yes/no), non-Recommended Food Score (quintiles), and total energy intake (continuous). †Adjusted for covariates in Model 1 and mutually adjusted for all the other low-risk lifestyle factors. ‡The Recommended Food Scores included foods with a beneficial effect on cardiovascular health. A score of 1, adding up to a maximum of 25, was assigned for regular consumption of fruits, vegetables, legumes, nuts, reduced-fat dairy products, whole grains, and fish.  
CI = confidence interval; RR = relative risk.

category. Each lifestyle factor was inversely and, after mutual adjustment for the other elements of the low-risk profile, independently associated with the risk of coronary events. This reduction in risk corresponded to 18% for the healthy diet, 11% for moderate alcohol

consumption, 36% for no smoking, 3% for being physically active, and 12% for having a low abdominal circumference.

In total, 8.7% of the men combined the low-risk diet with moderate alcohol consumption. This practice was characterized by a mean daily consumption of 5 servings of vegetables and fruits, 4 servings of whole grains, and a weekly consumption of 2.2 servings of fish. The median alcohol intake in this group was 17 g/day. Compared with the high-risk group (who did not fulfill the criteria of any of the 5 low-risk diet and lifestyle factors), this low-risk dietary and moderate alcohol consumption behavior was associated with a 35% (95% CI: 13% to 52%) reduction in the risk of MI (Table 3). The population-attributable risk was 23% (95% CI: 4% to 39%).

In the final analysis, we further investigated the combined effect of the entire low-risk practice by combining the low-risk diet and moderate alcohol consumption with the 3 independent low-risk non-dietary modifiable lifestyle factors: no smoking, high physical activity, and low abdominal adiposity (Table 3). The decrease in age-standardized incidence rates and RRs for the addition of each single low-risk factor where the reference in each category constitutes the remainder of the study population is illustrated in Online Figure 1A. The final comprehensive low-risk profile including all 5 low-risk factors, fulfilled by 1% of the study population, was

**TABLE 3 Effect of Combined Low-Risk Behaviors in Relation to Risk of Myocardial Infarction\***

Group	Low-Risk Group No. of Events (% Men)	Age-Standardized Incidence Rate† (95% CI)	Compared With High-Risk Group‡ RR (95% CI)	Compared With the Remainder of the Study Population RR (95% CI)	Population Attributable Risk§ % (95% CI)
1 low-risk factor: healthy diet (RFS top quintile)	177 (18)	495 (417-572)	0.74 (0.58-0.96)	0.82 (0.69-0.96)	16 (4-35)
2 low-risk factors  : healthy diet (RFS top quintile), alcohol consumption 10-30 g/day	74 (8.7)	429 (321-537)	0.65 (0.48-0.87)	0.75 (0.59-0.95)	23 (4-39)
3 low-risk factors¶: healthy diet (RFS top quintile), alcohol consumption 10-30 g/day, no smoking	36 (5.4)	321 (208-433)	0.36 (0.25-0.53)	0.54 (0.39-0.76)	44 (23-49)
4 low-risk factors#: healthy diet (RFS top quintile), alcohol consumption 10-30 g/day, no smoking, physically active (≥40 min/day of walking/bicycling and ≥1 h/week of exercise)	9 (1.7)	218 (73-363)	0.24 (0.12-0.47)	0.36 (0.19-0.69)	64 (30-81)
5 low-risk factors: healthy diet (RFS top quintile), alcohol consumption 10-30 g/day, no smoking, physically active (≥40 min/day of walking/bicycling and ≥1 h/week of exercise), waist circumference <95 cm	3 (1.0)	131 (0-279)	0.14 (0.04-0.43)	0.21 (0.07-0.66)	79 (34-93)

\*All relative risks were adjusted for age (continuous), educational achievement (≤9, 10 to 12, >12 years), family history of myocardial infarction (yes/no), use of aspirin (yes/no), marital status (unmarried, married, divorced, widowed), non-Recommended Food Score (quintiles), and total energy intake (continuous). †Per 100,000 person-years. ‡The high-risk group (8.3% of the study population and 166 cases of myocardial infarction [age-standardized incidence rate 979 cases per 100,000 person-years]) included men with no low-risk factors and was characterized by the following: median 2.9 servings/day of vegetables and fruit, 3.0 servings/day of whole grains, and 1.4 servings/week of fish; 24 pack-years of tobacco smoking (55% reported to be current smokers); 36% reported neither ≥40 min of daily walking/bicycling nor ≥1 h per week of exercise; and a median waist circumference 101 cm. §Estimated compared with the remainder of the total study population, representing 91.3%, 94.6%, 98.3%, and 99%, respectively, for each additional low-risk factor. ||The model was also adjusted for smoking, physical activity, and waist circumference. ¶The model was also adjusted for physical activity and waist circumference. #The model was also adjusted for waist circumference.  
RFS = Recommended Food Score; other abbreviations as in Table 2.

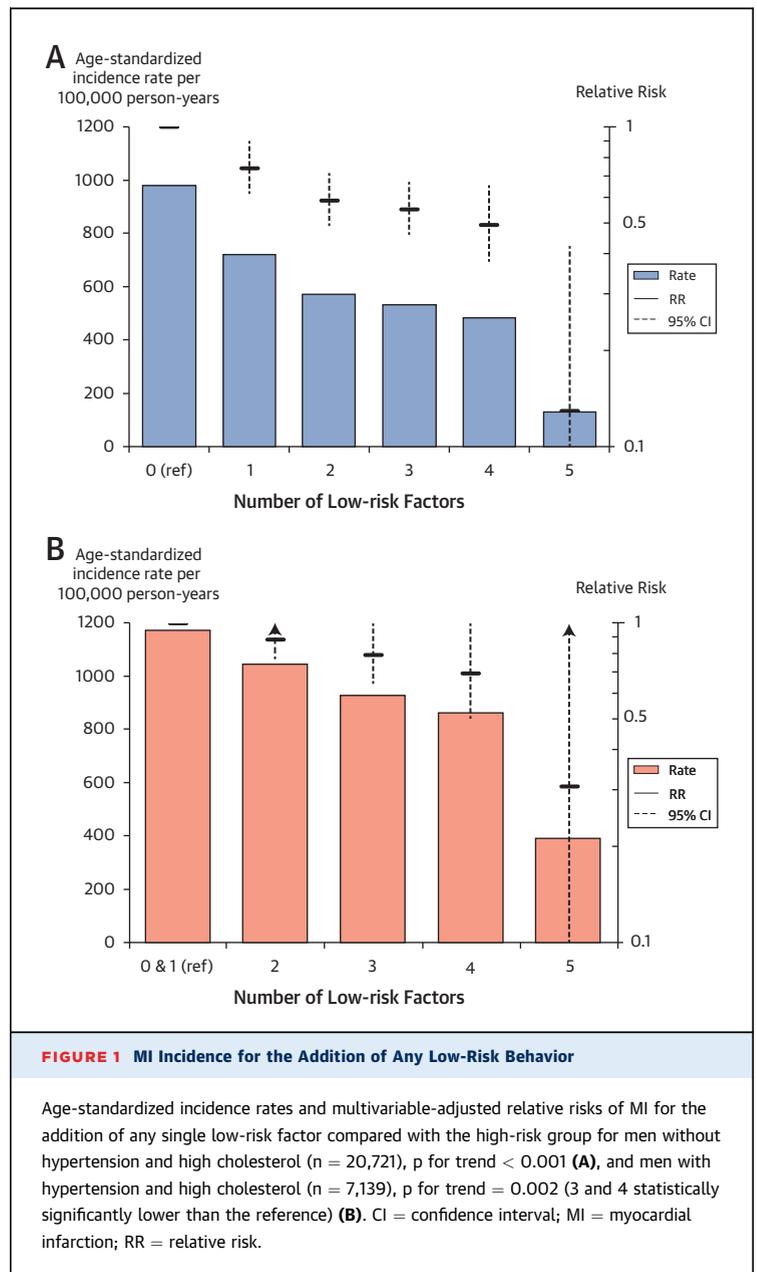
associated with an 86% (95% CI: 67% to 96%) lower risk of MI compared with the high-risk group with no low-risk factors. **Figure 1A** shows the overall decrease in incidence rates and RRs of MI (p for trend <0.001) for the addition of any single low-risk factor compared with the group having no low-risk behaviors. The absolute rate difference between men having no low-risk factors and men having 5 low-risk factors was 848 cases per 100,000 person-years. Excluding alcohol from the low-risk profile (i.e., comparing 4 low-risk factors with no low-risk factors) was associated with a 59% (95% CI: 0.36 to 0.74) lower risk of MI.

The population-attributable risk estimated for the comprehensive low-risk profile compared with the remaining men in the study population was 79% (95% CI: 34% to 93%), suggesting that ~4 of 5 coronary events could potentially have been averted if all men had followed low-risk practices (**Central Illustration**). If instead only 50%, 25%, or 10% of the men followed the low-risk practices, the rates were 39% (95% CI: 26% to 59%), 19% (95% CI: 13% to 24%), and 7% (95% CI: 5% to 9%), respectively.

On additional analyses, we assessed the association between the low-risk practices and MI risk among 7,139 men with hypertension and high cholesterol at baseline with 765 ascertained incident cases of MI. The decrease in age-standardized incidence rates and RRs for the addition of each independent modifiable low-risk lifestyle factor, for which the reference in each category constitutes the remainder of the study population, is illustrated in **Online Figure 1B**. **Figure 1B** shows the overall decrease in incidence rates and RRs of MI (p for trend = 0.002) for the addition of any single low-risk factor compared with the group having no to 1 low-risk behavior. The absolute rate difference between no to 1 versus 5 low-risk factors was 778 cases per 100,000 person-years, similar to that in men without hypertension and high cholesterol.

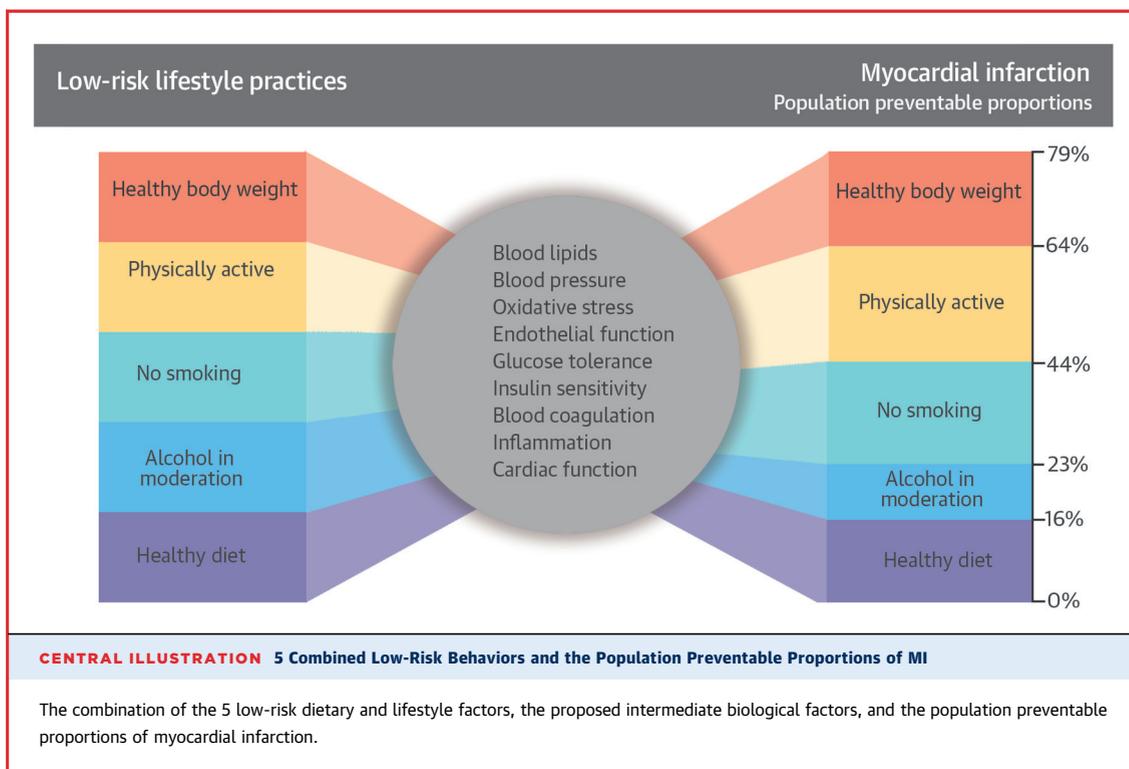
## DISCUSSION

In this prospective cohort of healthy men, we observed that a low-risk diet together with moderate consumption of alcoholic beverages was associated with a 35% risk reduction of primary MI, compared with men in the high-risk group (i.e., men having 0 of 5 low-risk factors). Men who combined this low-risk diet and moderate alcohol consumption with low-risk lifestyle behaviors, including not smoking, being physically active, and avoiding abdominal adiposity had an 86% lower risk. The benefit of



combined diet, lifestyle, and healthy body weight may prevent up to ~4 of 5 cases of MI in this healthy study population (**Central Illustration**). A decrease in risks with increasing adherence to the low-risk behaviors was also observed in men with hypertension and high cholesterol.

We used the Recommended Food Score (19) to identify a healthy diet, a joint measure of the consumption of different healthy foods (fruits, vegetables, legumes, nuts, reduced-fat dairy products, whole grains, and fish) known to have a beneficial



effect on cardiovascular health (1,20,21). In this cohort of men without a history of cardiovascular disease (CVD), hypertension, high cholesterol, or diabetes at baseline, the observed reduction in primary MI incidence associated with a healthy diet together with moderate alcohol consumption was similar to that recently observed in a primary prevention trial in Spain examining a Mediterranean diet supplemented with olive oil or nuts (29). In CVD-free patients at high cardiovascular risk, the combined Mediterranean diet significantly reduced the risk of total CVD by 29% compared with a control diet. The corresponding RR for MI (secondary endpoint) was 0.77 (95% CI: 0.52 to 1.15). This reduction is in the same range as that observed after primary CHD intervention (nonfatal MI) with cholesterol-reducing drugs (statins) in groups at low to intermediate cardiovascular risk (30-32). However, even among those taking medication for CHD risk factors, a previous study observed that the addition of healthy lifestyle behaviors was associated with substantially reduced CHD risk (13). When consumed in moderate amounts, alcohol is consistently associated with reduced risk of CHD (21). For men, this intake should not exceed 30 g/day, according to the recommendation of the Nutrition Committee of the American Heart Association.

Unlike healthy foods, alcohol consumption cannot be recommended for CHD risk reduction without reservation. It is important to note that although it may confer protection from CHD at moderate levels of consumption, alcohol abuse is identified as 1 of 3 leading risk factors for the global burden of all diseases (33).

Men who combined the low-risk diet with moderate alcohol consumption and the 3 low-risk behaviors had a very low risk of MI. The estimated population-attributable risk indicated that 79% of the burden of disease could be reduced by adherence to this combination of healthy lifestyle practices in this group of healthy men without hypertension, high cholesterol, and diabetes (Central Illustration). Similar population-attributable risks have been observed in American (82%) and Swedish (77%) women (15,16) and in American men (62%) (13). In the present study, only 1% of the population comprised the low-risk group and thus followed this set of healthy lifestyle practices of their own accord. Very low prevalence (0.1% to 2%) of "ideal cardiovascular health," according to the American Heart Association's definition (34) has recently been observed in U.S. national (5,35) and community-based (14,36) samples. This definition included 4 (alcohol excluded) of 5 low-risk lifestyle choices together with

favorable metrics of total serum cholesterol, fasting glucose, and blood pressure. Programs targeting men to increase the proportion of those adhering to low-risk behaviors could have a large impact on the burden of disease. Clearly, a strong body of evidence is required before deriving more stringent recommendations on the basis of the results. Randomized trials generally represent the most robust study design to establish causality. However, testing lifestyle interventions in trials has limitations due to lack of blinding, crossover, and noncompliance. Also, testing a combined low-risk behavior in a long-term primary prevention randomized intervention is very complicated and may never be performed. Therefore, well-designed prospective studies have complementary strengths and limitations that are equally robust, and the implementation of prevention strategies should be based on the breadth, depth, and consistency of evidence across multiple research paradigms.

Of great importance is that these lifestyle behaviors are modifiable, and prospective changes from high-risk to low-risk behaviors (adopting at least 2 healthy characteristics) have been associated with 27% reduced incidence of CHD (13). It is, however, also clear that extensive prevention only can be achieved through inhibiting the initiation and establishment of any high-risk behavior (37,38). Preferably, also to control health care expenditures, prevention should focus on ensuring that ideal low-risk behaviors are introduced early and continued throughout life.

Strengths of this study include a prospective population-based design in a contemporary population (begun in 1997), which increases generalizability and relevance to modern guidelines. The availability of detailed information on diet as well as potential risk factors for CHD is also important. The prospective design prevents recall bias with respect to the outcome and the practically complete follow-up of the study population through linkages to various population-based registries minimizes the concern that our findings could be affected by differential loss to follow-up. In contrast to most previous studies that focused on cardiovascular mortality and lacked incident morbidity events (4-11), we assessed the incidence of primary MI. This allows a more complete picture of the preventive effect of these combined lifestyle practices.

**STUDY LIMITATIONS.** Limitations of our study need to be considered. Inevitable measurement error in self-reports can lead to misclassification of the exposure. Because of the prospective design, the

misclassification is most likely nondifferential, which may lead to attenuation of the true association. Despite a large cohort, the study is limited by the small number of subjects and cases of MI in the group with all 5 low-risk behaviors, leading to somewhat imprecise estimates; nevertheless, the power was sufficient to observe statistically significant associations. The gradual decrease in incidence with increasing adherence to low-risk practices was similar to that previously observed in both women (15,16) and men (13) and consistent with findings for cardiovascular mortality (5-10,14,35). Although the cohort is considered representative of Swedish men, the exclusion of men with high cholesterol, hypertension, or missing data may limit the generalizability of our results. The interpretation of the results observed for men with hypertension and high cholesterol, on the other hand, was hampered by the low sample size, leading to unstable and nonsignificant risk estimates. Despite these limitations, our results suggest that important steps can be taken to significantly reduce the incidence of a first MI.

## CONCLUSIONS

Our study indicates that a healthy diet together with low-risk lifestyle practices and absence of abdominal adiposity may prevent the vast majority of MI events in men.

**REPRINT REQUESTS AND CORRESPONDENCE:** Dr. Agneta Åkesson, Institute of Environmental Medicine, Box 210, 171 77 Stockholm, Sweden. E-mail: [Agneta.Akesson@ki.se](mailto:Agneta.Akesson@ki.se).

## PERSPECTIVES

**COMPETENCY IN MEDICAL KNOWLEDGE:** Combining 5 low-risk behaviors: a healthy diet, moderate alcohol consumption, no smoking, being physically active, and a healthy weight may prevent 4 of 5 myocardial infarctions in the population. The incidence of myocardial infarction decreases with the number of positive behaviors in both healthy men and in those with hypertension and high cholesterol.

**TRANSLATIONAL OUTLOOK:** Further studies are needed to develop population-based strategies to promote healthy behaviors that can be introduced early in life and maintained throughout the life span.

## REFERENCES

1. Go AS, Mozaffarian D, Roger VL, et al. Heart disease and stroke statistics-2014 update: a report from the American Heart Association. *Circulation* 2014;129:e28-292.
2. Baigent C, Keech A, Kearney PM, et al. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90,056 participants in 14 randomised trials of statins. *Lancet* 2005;366:1267-78.
3. Law MR, Morris JK, Wald NJ. Use of blood pressure lowering drugs in the prevention of cardiovascular disease: meta-analysis of 147 randomised trials in the context of expectations from prospective epidemiological studies. *BMJ* 2009;338:b1665.
4. Kant AK, Schatzkin A, Graubard BI, Schairer C. A prospective study of diet quality and mortality in women. *JAMA* 2000;283:2109-15.
5. Ford ES, Greenlund KJ, Hong Y. Ideal cardiovascular health and mortality from all causes and diseases of the circulatory system among adults in the United States. *Circulation* 2012;125:987-95.
6. Daviglius ML, Stamler J, Pirzada A, et al. Favorable cardiovascular risk profile in young women and long-term risk of cardiovascular and all-cause mortality. *JAMA* 2004;292:1588-92.
7. Eguchi E, Iso H, Tanabe N, et al. Healthy lifestyle behaviours and cardiovascular mortality among Japanese men and women: the Japan collaborative cohort study. *Eur Heart J* 2012;33:467-77.
8. Knoops KT, de Groot LC, Kromhout D, et al. Mediterranean diet, lifestyle factors, and 10-year mortality in elderly European men and women: the HALE project. *JAMA* 2004;292:1433-9.
9. Odegaard AO, Koh WP, Gross MD, Yuan JM, Pereira MA. Combined lifestyle factors and cardiovascular disease mortality in Chinese men and women: the Singapore Chinese health study. *Circulation* 2011;124:2847-54.
10. Stamler J, Stamler R, Neaton JD, et al. Low risk-factor profile and long-term cardiovascular and noncardiovascular mortality and life expectancy: findings for 5 large cohorts of young adult and middle-aged men and women. *JAMA* 1999;282:2012-8.
11. van Dam RM, Li T, Spiegelman D, Franco OH, Hu FB. Combined impact of lifestyle factors on mortality: prospective cohort study in US women. *BMJ* 2008;337:a1440.
12. Carlsson AC, Wandell PE, Gigante B, Leander K, Hellenius ML, de Faire U. Seven modifiable lifestyle factors predict reduced risk for ischemic cardiovascular disease and all-cause mortality regardless of body mass index: a cohort study. *Int J Cardiol* 2013;168:946-52.
13. Chiuve SE, McCullough ML, Sacks FM, Rimm EB. Healthy lifestyle factors in the primary prevention of coronary heart disease among men: benefits among users and nonusers of lipid-lowering and antihypertensive medications. *Circulation* 2006;114:160-7.
14. Folsom AR, Yatsuya H, Nettleton JA, Lutsey PL, Cushman M, Rosamond WD. Community prevalence of ideal cardiovascular health, by the American Heart Association definition, and relationship with cardiovascular disease incidence. *J Am Coll Cardiol* 2011;57:1690-6.
15. Akesson A, Weismayer C, Newby PK, Wolk A. Combined effect of low-risk dietary and lifestyle behaviors in primary prevention of myocardial infarction in women. *Arch Intern Med* 2007;167:2122-7.
16. Stampfer MJ, Hu FB, Manson JE, Rimm EB, Willett WC. Primary prevention of coronary heart disease in women through diet and lifestyle. *N Engl J Med* 2000;343:16-22.
17. Norman A, Bellocco R, Vaida F, Wolk A. Total physical activity in relation to age, body mass, health and other factors in a cohort of Swedish men. *Int J Obes Relat Metab Disord* 2002;26:670-5.
18. Messerer M, Johansson SE, Wolk A. The validity of questionnaire-based micronutrient intake estimates is increased by including dietary supplement use in Swedish men. *J Nutr* 2004;134:1800-5.
19. Michels KB, Wolk A. A prospective study of variety of healthy foods and mortality in women. *Int J Epidemiol* 2002;31:847-54.
20. World Health Organization. Diet, Nutrition and the Prevention of Chronic Diseases. Geneva, Switzerland: World Health Organization, 2003.
21. Lichtenstein AH, Appel LJ, Brands M, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation* 2006;114:82-96.
22. Messerer M, Hakansson N, Wolk A, Akesson A. Dietary supplement use and mortality in a cohort of Swedish men. *Br J Nutr* 2008;99:626-31.
23. Norman A, Bellocco R, Bergstrom A, Wolk A. Validity and reproducibility of self-reported total physical activity-differences by relative weight. *Int J Obes Relat Metab Disord* 2001;25:682-8.
24. Ekblom-Bak E, Ekblom B, Vikstrom M, de Faire U, Hellenius ML. The importance of non-exercise physical activity for cardiovascular health and longevity. *Br J Sports Med* 2014;48:233-8.
25. Sattelmair J, Pertman J, Ding EL, Kohl HW 3rd, Haskell W, Lee IM. Dose response between physical activity and risk of coronary heart disease: a meta-analysis. *Circulation* 2011;124:789-95.
26. World Health Organization. Waist Circumference and Waist-Hip Ratio. Geneva, Switzerland: 2008.
27. Ludvigsson JF, Andersson E, Ekblom A, et al. External review and validation of the Swedish national inpatient register. *BMC Public Health* 2011;11:450.
28. Loehr LR, Rosamond WD, Poole C, et al. The potentially modifiable burden of incident heart failure due to obesity: the atherosclerosis risk in communities study. *Am J Epidemiol* 2010;172:781-9.
29. Estruch R, Ros E, Salas-Salvado J, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med* 2013;368:1279-90.
30. Thavendiranathan P, Bagai A, Brookhart MA, Choudhry NK. Primary prevention of cardiovascular diseases with statin therapy: a meta-analysis of randomized controlled trials. *Arch Intern Med* 2006;166:2307-13.
31. Gupta AK. The efficacy and cost-effectiveness of statins in low-risk patients. *CMAJ* 2011;183:1821-3.
32. Tonelli M, Lloyd A, Clement F, et al. Efficacy of statins for primary prevention in people at low cardiovascular risk: a meta-analysis. *CMAJ* 2011;183:E1189-202.
33. Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2013;380:2224-60.
34. Lloyd-Jones DM, Hong Y, Labarthe D, et al. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. *Circulation* 2010;121:586-613.
35. Yang Q, Cogswell ME, Flanders WD, et al. Trends in cardiovascular health metrics and associations with all-cause and CVD mortality among US adults. *JAMA* 2012;307:1273-83.
36. Bambs C, Kip KE, Dinga A, Mulukutla SR, Aiyer AN, Reis SE. Low prevalence of "ideal cardiovascular health" in a community-based population: the heart strategies concentrating on risk evaluation (Heart SCORE) study. *Circulation* 2011;123:850-7.
37. Capewell S, Lloyd-Jones DM. Optimal cardiovascular prevention strategies for the 21st century. *JAMA* 2010;304:2057-8.
38. Mozaffarian D, Afshin A, Benowitz NL, et al. Population approaches to improve diet, physical activity, and smoking habits: a scientific statement from the American Heart Association. *Circulation* 2012;126:1514-63.

**KEY WORDS** diet, lifestyle, men, myocardial infarction, population-attributable risk, population based, primary prevention

**APPENDIX** For a supplemental figure, please see the online version of this article.