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Influence of Age on the Association between Lifestyle Factors and Risk of Hypertension

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Abstract

BACKGROUND—Although hypertension is a highly prevalent disease in older populations, risk factors for developing hypertension have been studied primarily in younger cohorts. We sought to determine whether the strength of traditional hypertensive risk factors varied with age.

METHODS—We analyzed the prospective association between five modifiable risk factors and hypertension incidence among 78,590 initially non-hypertensive women of different ages in the Nurses' Health Study I cohort over 26 years.

RESULTS—Older age attenuated the association between incident hypertension and four of five risk factors associated with hypertension in younger women. Persons aged 50 years and under who were low risk for all five risk factor behaviors had a hazard ratio (HR) for incident hypertension of 0.13 (95% CI, 0.03-0.52), compared with others in this age group. In women 61 and older, the HR was 0.62 (95% CI, 0.51-0.75). However, the hypothetical number needed to treat (the number of women needed to join the low risk factor group for a 10 year period to prevent one hypertension case) was similar between the age groups.

CONCLUSION—The fraction of incident hypertension attributable to modifiable lifestyle factors decreases with age. Because the incidence of hypertension is higher in older persons, however, lifestyle modification would hypothetically prevent similar numbers of hypertension cases in younger and older populations.

Introduction

The public health impact of hypertension is staggering, contributing to more cardiovascular deaths in women than any other preventable disease[1]. This is especially true for women past childbearing age: incidence rates for hypertension in women increase sharply in the post-menopausal period, and ultimately the prevalence of hypertension in older women exceeds the prevalence in men[2]. While traditional lifestyle risk factors for incident hypertension have been examined in younger and middle-aged women[3], they have not been studied extensively

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in older cohorts. We performed a prospective analysis of the Nurses' Health Study I cohort to examine the association between previously established modifiable lifestyle risk factors and hypertension in older age groups, and to determine whether associations and hypothetical effects vary with age.

Methods

Source Population

The Nurses' Health Study I (NHS I) is a prospective cohort study begun in 1976, when 121,700 nurses, aged 30-55, in eleven states returned initial questionnaires surveying lifestyle and health history. Participants are followed via biennial mailed questionnaires that ascertain information on health-related behaviors and medical events. Extensive dietary information using a food-frequency questionnaire (FFQ) was collected in 1984, then 1986, and every four years thereafter. The follow-up rate has been >90%. The institutional review board at Brigham and Women's Hospital reviewed and approved this study, including that participants provided implied consent by virtue of voluntarily returning their questionnaires.

Study Population

Although the NHS I began in 1976, we defined 1984 as the baseline year because that was the first year in which an expanded dietary history was obtained. Of the original 121,700 women included in the cohort, we limited our study population to women who, by self-report, did not have the following conditions at baseline in 1984: hypertension; angina or history of myocardial infarction; diabetes mellitus; or previous malignancy, except non-melanoma skin cancer. Women who reported taking antihypertensive medications at baseline were excluded from the study. To reduce potential bias, we additionally excluded women from analysis in a questionnaire cycle if, on the questionnaire, they did not report undergoing physical or screening exams during which blood pressure was likely to have been measured. The final study population was 78,590 women.

Assessment of Non-dietary Risk Factors

All information on non-dietary risk factors was updated every two years, or whenever new data on risk factors were available. Weight was ascertained on each biennial questionnaire; height was queried on the initial 1976 survey. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Self-reported weight by participants of the NHS I correlated highly with measured weight ($r=0.97$)[4]. To assess physical activity, participants were asked about the amount of time per week (minutes to 11+ hours) they spent engaged in vigorous exercise. Total energy expenditure was calculated by multiplying time spent on exercise and the standard metabolic equivalent value (MET) assigned to each exercise type. Self-reported activity on the biennial questionnaires was compared to activity diaries recorded by a subset of participants in the NHS II study, a study of younger women that utilizes a similar questionnaire, and was found to be valid ($r=0.79$)[5]. Use (vs. non-use) of non-narcotic analgesics (aspirin, acetaminophen, and non-steroidal anti-inflammatory drugs, or NSAIDs) was assessed beginning with the 1980 questionnaire. Race and family history of hypertension were both ascertained on the 1992 questionnaire.

Assessment of Diet

Detailed FFQs were included in the 1984, 1986, 1990, 1994, 1998, and 2002 surveys. The questionnaires asked about intake of over 130 food types, cooking preparation, and portion sizes. The Harvard University food consumption database was then used to determine the nutrient content of each food, which was multiplied by portion size and consumption

frequency to obtain the total nutrient intake. The validity of the FFQ and analysis method has been established previously[6]. In addition to nutrient content in food, the intake of supplements was calculated from reported use of multivitamins or individual vitamin formulations. Alcohol consumption in grams per week was calculated based on reported weekly intake of alcoholic beverages (beer, wine, or liquor).

In assessing the association between diet and hypertension risk, we used a dietary score based on eight components (high intake of fruits, vegetables, nuts and legumes, low-fat dairy products, whole grains, low intake of sodium, sweetened beverages, and red and processed meat) that was modeled on the diet followed in the DASH (Dietary Approaches to Stop Hypertension) trial[7]. Each participant's DASH score was calculated at baseline using the 1984 FFQ, and then the DASH score was updated as participants completed subsequent FFQs during the period of follow-up. For each of the eight dietary components, participants were classified into quintiles according to their intake, and a score was assigned. The eight component scores were then summed to obtain an overall DASH score ranging from 8 to 40.

Ascertainment of Hypertension

On the initial 1976 questionnaire, women were asked if they had previously been diagnosed with hypertension by a physician. Each biennial follow-up survey asked if there had been a new diagnosis of hypertension made since the last questionnaire was returned. Self-reporting of hypertension in the NHS I cohort has been validated previously[8]: a random sample of 100 women who reported hypertension on the 1982 questionnaire were contacted, and of the 84 who confirmed a diagnosis of high blood pressure, medical records were obtained from 51 women. A blood pressure greater than 140/90 was confirmed in all 51 cases after medical record review. To assess the likelihood of false-negative reports, blood pressure in 161 women without a diagnosis of hypertension was measured. Only 7% of those women had measured blood pressure over 140/90. In the current analysis, women who reported a diagnosis of hypertension on any questionnaire up to and including the 1984 questionnaire were excluded so that the study would be prospective.

Statistical Analysis

The person-time for each participant was measured from the date of return of the 1984 questionnaire and was truncated at the date when hypertension was reported, the date of death, or June 2004, whichever came first. Person-time was grouped into three age categories: 50 years and under, 51-60 years, and 61+ years. These age groups were chosen to coincide temporally with age-related changes in blood pressure phenotypes observed in other cohorts, specifically the plateau and eventual decline in average diastolic blood pressures contrasted with steady age-related increases in systolic blood pressure[9]. Participants were censored if they died or developed incident hypertension, cardiovascular disease, malignancy or diabetes. When a woman who had not previously been censored due to hypertension or death moved into a new age category, her subsequent person-time was counted in the new category. We analyzed the association with hypertension of five modifiable risk factors that have been previously established in younger and middle-aged individuals. These included BMI, physical activity, DASH diet, use of non-narcotic analgesics, menopause, and alcohol intake. Physical activity and DASH diet scores were grouped into quintiles; BMI and analgesic intake frequency were analyzed in five categories, while six groups were used to characterize amount of alcohol consumption. These factors were included into regression models as time-varying variables; specifically, information on all five of these factors was initially available at baseline in 1984, and was updated every two years thereafter to reflect the newest available information. Cox proportional hazards regression methods were used to examine the relation between the quintiles of modifiable risk factors and incident hypertension. Hazard ratios (HRs) and 95% confidence intervals

(CIs) were computed. In addition to the five risk factors tested, the multivariable models also included adjustment for race/ethnicity, smoking status (current, never or former), categories of supplemental folic acid intake, menopause status, and family history of hypertension. The interactions between age and risk factors were tested in unstratified multivariable models that included appropriate interaction terms between age and each of the five factors.

The five modifiable lifestyle factors were then grouped together to explore their hypothetical contributions to hypertension incidence in each of the age groups. In order to assemble these risk factors together for the purposes of calculating hypothetical attributable fractions, we dichotomized each of them into "high risk" and "low risk" groups. BMI was dichotomized according to the accepted definitions of normal weight (a BMI of <25 kg/m^2 was defined as "low risk"). For physical activity, women in the highest quintile of activity were defined as the "low risk group"; similarly, women in the highest quintile of DASH scores were considered "low risk". For non-narcotic analgesics, women who used these medications less frequently than once per week were considered "low risk" for hypertension[10]. For alcohol, daily consumption of less than 15 g/d, or slightly over one drink daily, was considered low risk[3]. Women were then grouped according to combinations of two (BMI and physical activity), three (BMI, physical activity, and DASH diet), four (BMI, physical activity, DASH diet, and abstention from non-narcotic analgesics), and five (all) low risk factors, and the age-specific HRs and 95% CIs were calculated for each of these combinations using multivariable-adjusted Cox proportional hazards regression. The population attributable fractions (AFs), estimates of the percentage of new hypertension cases occurring in this population that could have hypothetically been prevented if all women had been in the low-risk groups (assuming a causal and independent relationship between the risk factor and incident hypertension) were then calculated. The standard equation used to compute this hypothesized attributable fraction, where P_e is the exposed proportion, is: $AF = \{(HR-1) \times P_e\} \div \{([HR-1] \times P_e) + 1\}$[11]. Finally, using the incidence rate of hypertension in the reference groups and the adjusted HRs for the various combinations of low risk factors, we calculated hypothetical absolute risk differences (ARD) and number needed to treat (NNT) for 10 years. The NNT for 10 years signifies the number of high-risk women that would hypothetically have to move into the low risk group for a period of 10 years to prevent one case of hypertension.

Results

Characteristics of NHS I

Baseline characteristics of the NHS I cohort in 1984 are displayed in Table 1. During a total of 915,765 person-years of follow-up over 20 years, there were 29,858 new diagnoses of hypertension recorded across all age groups (Table 2). The crude incidence of hypertension was more than 3-fold higher in the 61+ age group, compared to the 50-and-under group, a finding consistent with previous assessments of hypertension prevalence in older cohorts[9].

Risk factors for hypertension by age

The associations between the five modifiable risk factors and incident hypertension are shown in Table 2 according to age group. The magnitude of the association for the strongest risk factor in the 50-and-under group, BMI, was markedly attenuated in the older age groups (p-value for interaction with age < 0.001); for women aged 50 and under, the HR for hypertension associated with a BMI ≥ 30 kg/m^2 compared with a BMI < 21 kg/kg/m^2 was 5.07 (95% CI, 4.48-5.74), more than twice the HR seen in the 61-and-over cohort (2.35, 95% CI, 2.20-2.51). For physical activity, the association with incident hypertension also varied significantly by age (p-value for interaction < 0.001); comparing extreme quintiles

yielded HRs of 0.87 (95% CI 0.78-0.97) for the 50 years and under age group and 0.95 (95% CI 0.90-1.01) for the 61 years and over group. Women who followed a DASH-style diet had a decreased risk of new-onset hypertension compared with women who did not, and a marginal interaction with age was observed (HR for age 50 and under=0.85, 95% CI 0.76-0.95; HR for age 61+=0.89, 95% CI 0.84-0.95; p-value for interaction = 0.05). Regular NSAID, acetaminophen or aspirin use at least once per week was associated with an increased risk of incident hypertension compared with avoidance of these medications. For all three analgesic classes, the magnitude of the association was greater in younger compared with older women (see Table 2). The large percentage of missing data for analgesic consumption is noted, and is due to study participants leaving that part of the questionnaire blank. Alcohol consumption of 15 g/d or more was significantly associated with an increased risk of hypertension, but unlike the other lifestyle factors, the association did not differ significantly among age groups.

Age-specific attributable fractions of risk-factor combinations

The attributable fractions (AFs) of incident hypertension that could hypothetically be prevented by combining a lower BMI, higher level of physical activity, a DASH style diet, avoidance of non-narcotic analgesics and modest alcohol intake were then calculated (Table 3). In women aged 50 years and under, the HR for the combination of a BMI < 25 and higher physical activity was 0.52 (95% CI, 0.46-0.59) compared with women who did not possess both of these low risk factors. This translated into a hypothetical AF of 45% (95% CI, 38-51%). The same comparisons yielded AFs of 36% (95% CI, 31-40%) in women between 50 and 60 years, and 21% (95% CI, 18-25%) in women aged 61+ years. In the older age groups, despite the higher incidence of hypertension, the AFs associated with combinations of low risk factors were considerably lower. Combinations of three low risk lifestyle factors (a lower BMI, high level of physical activity, and adherence to a DASH-style diet) yielded AFs of 53%, 43%, and 24% in women 50 and under, between 51 and 60, and 61+ years, respectively. When a fourth low-risk factor, avoidance of non-narcotic analgesics, was incorporated into the model, AFs of 83% in women 50 years and younger, 67% in women aged 51-60 years, and 37% in women 61 years and older were observed. Finally, when moderate alcohol use (a factor not significantly associated with age) was added as a fifth low-risk factor, AFs of 87%, 65%, and 38% were observed in the different age categories. The NNT over 10 years for women 50 years and under possessing three, four or five low-risk factors were 9.6, 6.3 and 6.2; for women 61 years and above, the NNT were 8.7, 4.5 and 5.6. The NNTs were not significantly different between age groups despite the differences in hypothetical attributable fractions.

Discussion

Our results suggest that in older, non-hypertensive women, lower BMI, high levels of physical activity, adherence to a DASH-style diet, and avoidance of non-narcotic analgesics are more weakly associated with hypertensive risk than in younger cohorts of women. As a result, the hypothetical fraction of hypertension developing in older women that is due to a less healthy lifestyle is considerably smaller than the corresponding fraction in younger women. However, it is important to note that although modifiable risk factors accounted for a smaller hypothetical fraction of hypertension in older women, the hypothetical NNT to prevent one case of hypertension was similar among age groups. This observation is due to the much higher incidence of hypertension in older individuals. Thus, a key finding in this study is that even though lifestyle modification may have less benefit in any given older woman, the overall public health benefit in reducing hypertension prevalence is similar. This study provides compelling evidence that lifestyle and diet modification should be prime

targets in effort to promote healthy aging. Health care practitioners should see our results as further motivation to impress the importance of a healthy lifestyle on patients of all ages.

Our findings also raise important questions about the physiologic differences that occur with advancing age. Higher BMI in older women may reflect preservation of lean muscle mass, which typically decreases with age[12]. Obesity is also associated with increased sympathetic nervous system activation and increased renin release, which are thought to contribute to hypertension development in obese individuals. The sympathetic nervous system becomes less responsive to stimuli with age[13], and thus obese older persons may display less sympathetic nervous system upregulation than is seen in obese younger individuals[14]. The observed attenuation of the relation between physical activity and hypertensive risk may result from an age-related increase in arterial stiffness. While the mechanisms are unclear, current theories include remodeling of the extracellular matrix, elastin fragmentation, and increases in fibronectin as well as collagen[15]. These changes may impair vasodilation in response to exercise-induced local vasodilatory mediators, such as nitric oxide. Because older persons exhibit blunted responsiveness to autonomic nervous system stimuli[13,16], they may be less sensitive to the hypertensive effects of salt, and thus may not derive as much benefit from a DASH-style diet as younger persons. Although analgesic use has previously been associated with new-onset hypertension in cohorts of younger women[3], older women[17], and men[10], the mechanisms remain uncertain. Endothelin-1 dysfunction[18], renal sodium retention[19], and inhibition of vasodilatory prostaglandin production[20] have all been proposed as potential etiologies. Since endothelial dysfunction has been identified as an age-related process[21], the attenuated relation between analgesic use and hypertension may represent a limited effect of analgesic-related inhibition of vasodilation in an environment already subject to age-related vasoconstrictive forces. Although we adjusted for menopause in our models, several studies suggest that it has no association with blood pressure elevation [22,23].

There are several limitations to our study. The Nurses' Health Study I cohort is comprised of 94% Caucasian female nurses, and thus the results may not be generalizable to other ethnicities or to men. There is also the potential for misclassification bias in the reporting of dietary intake and lifestyle factors, since accuracy is dependent on a person's memory and observation. However, this type of misclassification is likely to be random, and therefore would produce an underestimate of the true associations. As with all observational studies, our findings could result from residual confounding. Moreover, calculations of attributable fraction and NNT are usually reserved for randomized controlled trials; however, a trial of this magnitude and duration would not be feasible, and ample trial data suggest that the associations between weight, physical activity, diet, analgesics, and alcohol with hypertension or increased blood pressure are likely to be causal. The large amount of missing non-narcotic analgesic data, owing to low response to this question on the questionnaire, could cause a confounding bias in our analysis of this risk factor. Finally, the decreased association between hypertensive risk factors and incident hypertension in older participants may be due to a survivorship bias. There may be certain genetic or environmental factors that protect women from hypertension, despite possessing hypertensive risk factors. Such individuals would be enriched in an older, non-hypertensive, cohort, and their resistance to adverse lifestyle factors such as obesity or physical inactivity would reduce the strength of risk factor associations with incident hypertension compared with younger, non-hypertensive women.

Conclusion

Lower BMI, high levels of physical activity, adherence to a DASH-style diet, and the avoidance of regular aspirin, acetaminophen, and NSAIDs are associated with weaker

relationships with hypertension in older compared with younger women. However, the hypothetical NNT to prevent one case of hypertension is similar among age groups. Our observations suggest that physiologic changes of aging may play a role in the weakened association between lifestyle and dietary behaviors and hypertensive risk. Given the increased incidence of new-onset hypertension with advancing age, however, these modifiable risk factors continue to bear enormous influence in efforts to prevent hypertension and its complications in all age groups. Women of all ages should be encouraged to follow a healthy diet and lifestyle in order to prevent the development of high blood pressure. Further analyses examining hypertensive risk factors in older minority populations as well as in older men are warranted.

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Table 1
Baseline Characteristics of NHS I Cohort Participants in 1984

Age of participants (years)	49 (44-56)
BMI (kg/m^2)	23.4 (21.4-26.2)
Physical Activity (mets/wk)	7.9 (2.9-36.5)
Frequency of Aspirin Use on the 1990 Survey	
<1 day/week	17,975 (32.9)
1 d/week	10,222 (18.7)
2-3 d/wk	5528 (10.1)
4-5 d/wk	2019 (3.7)
6+ d/wk	4600 (8.4)
Missing	14,327 (26.2)
Frequency of Acetaminophen Use on the 1990 Survey	
<1 day/week	18,124 (33.2)
1 d/week	12,766 (23.4)
2-3 d/wk	3718 (6.8)
4-5 d/wk	1001 (1.8)
6+ d/wk	1432 (2.6)
Missing	17,630 (32.2)
Frequency of Ibuprofen Use on the 1990 Survey	
<1 day/week	20,637 (37.7)
1 d/week	8557 (15.7)
2-3 d/wk	3505 (6.4)
4-5 d/wk	1039 (1.9)
6+ d/wk	3328 (6.1)
Missing	17,605 (32.2)
Alcohol consumption (g/wk)	2.1 (0-9.2)

Median and interquartile ranges are given for age, BMI, physical activity and alcohol. The percent of participants in each non-narcotic analgesic group is listed in parentheses.

Table 2

Unadjusted Hazard Ratios For Incident Hypertension Development According To Age And Modifiable Risk Factor

Age Category	**Age ≥ 50**	**Age 51-60**	**Age 61+**	
Cases	4,392	11,389	14,077	
Person-Years	36,245	366,073	313,447	
Variable	Hazard Ratio (95% CI)	Hazard Ratio (95% CI)	Hazard Ratio (95% CI)	p-interaction
BMI (kg/m^2)				<.0001
<21	1.0	1.0	1.0	
21.0-22.9	1.36 (1.19-1.55)	1.22 (1.12-1.32)	1.21 (1.13-1.29)	
23.0-24.9	1.79 (1.57-2.03)	1.52 (1.40-1.64)	1.44 (1.35-1.53)	
25.0-29.9	2.95 (2.62-3.33)	2.11 (1.96-2.28)	1.72 (1.62-1.82)	
≥30.0	5.07 (4.48-5.74)	3.06 (2.83-3.31)	2.35 (2.20-2.51)	
Physical activity quintiles				<.0001
1	1.0	1.0	1.0	
2	1.00 (0.91-1.11)	0.94 (0.88-1.00)	1.03 (0.97-1.09)	
3	1.03 (0.94-1.14)	0.94 (0.88-1.00)	0.98 (0.93-1.04)	
4	1.01 (0.91-1.12)	0.91 (0.85-0.97)	0.99 (0.93-1.05)	
5	0.87 (0.78-0.97)	0.86 (0.80-0.92)	0.95 (0.90-1.01)	
DASH diet adherence quintiles				.05
1	1.0	1.0	1.0	
2	0.90 (0.82-0.98)	0.97 (0.92-1.03)	0.96 (0.90-1.01)	
3	0.90 (0.82-.99)	0.95 (0.90-1.01)	0.94 (0.89-1.00)	
4	0.86 (0.78-.96)	0.88 (0.83-0.95)	0.93 (0.88-0.98)	
5	0.85 (0.76-0.95)	0.89 (0.83-0.95)	0.89 (0.84-0.95)	
Frequency of Aspirin use				<.001
<1 day/week	1.0	1.0	1.0	
1 day/week	1.05 (0.97-1.14)	1.04 (0.99-1.10)	1.06 (1.00-1.13)	
2-3 d/wk	1.27 (1.15-1.41)	1.17 (1.09-1.25)	1.13 (1.06-1.21)	
4-5 d/wk	1.36 (1.17-1.58)	1.27 (1.16-1.38)	1.18 (1.10-1.28)	
6+ d/wk	1.40 (1.24-1.58)	1.30 (1.22-1.37)	1.18 (1.13-1.23)	
Frequency of acetaminophen use				<.001
<1 day/week	1.0	1.0	1.0	
1-3 d/mo	1.05 (0.96-1.14)	1.01 (0.95-1.07)	1.10 (1.03-1.17)	
1-2 d/wk	1.25 (1.11-1.40)	1.20 (1.12-1.30)	1.14 (1.06-1.22)	
3-4 d/wk	1.41 (1.17-1.70)	1.24 (1.10-1.40)	1.20 (1.08-1.33)	
5+ d/wk	1.38 (1.17-1.63)	1.24 (1.13-1.37)	1.23 (1.15-1.31)	
Frequency of ibuprofen use				<.001
<1 day/week	1.0	1.0	1.0	
1-3 d/mo	1.04 (0.95-1.13)	1.08 (1.01-1.14)	1.05 (0.97-1.13)	
1-2 d/wk	1.16 (1.04-1.30)	1.09 (1.01-1.18)	1.10 (1.02-1.19)	

Age Category	**Age ≥ 50**	**Age 51-60**	**Age 61+**	
3-4 d/wk	1.07 (0.88-1.31)	1.18 (1.05-1.32)	1.28 (1.15-1.43)	
5+ d/wk	1.43 (1.27-1.62)	1.28 (1.20-1.38)	1.23 (1.14-1.33)	
Alchohol consumption				0.5
< 1 g/d	1.0	1.0	1.0	
1-4 g/d	0.96 (0.89-1.05)	0.97 (0.92-1.02)	1.00 (0.95-1.04)	
5-9 g/d	0.99 (0.87-1.11)	0.99 (0.92-1.06)	1.03 (0.97-1.10)	
10-14 g/d	1.11 (0.97-1.27)	0.95 (0.88-1.04)	1.09 (1.01-1.16)	
15-29 g/d	1.20 (1.03-1.39)	1.10 (1.00-1.20)	1.11 (1.03-1.19)	
30+	1.62 (1.37-1.91)	1.34 (1.21-1.49)	1.23 (1.12-1.35)	

models were adjusted for race/ethnicity, smoking status (current, never or former), categories of supplemental folic acid intake, menopause status, and family history of hypertension.

Table 3

Multivariate Hazard Ratios and Attributable Fraction by Age Group

Number of low-risk factors =2: BMI < 25, highest quintile of physical activity
Number of low-risk factors=3: above low-risk factors plus highest quintile of DASH diet
Number of low-risk factors=4: above low-risk factors plus no history of regular analgesic use
Number of low-risk factors=5: above low-risk factors plus moderate alcohol intake (<15 g/d)

Age Category	Number of low-risk factors	Exposed population (%)	Multivariate Hazard Ratio (95% CI)	Absolute Risk Difference (ARD), Cases per 1000 Person-Years	Number Needed to Treat (NNT) for 10y	Attributable Fraction (AF) (%) (95% CI)
Age ≤ 50	2	12	0.52 (0.46-0.59)	9.7	10.3	45 (38-51)
	3	3	0.46 (0.35-0.60)	10.4	9.6	53 (39-64)
	4	0.4	0.16 (0.05-0.50)	15.8	6.3	83 (50-95)
	5	0.3	0.13 (0.03-0.52)	16.2	6.2	87 (48-97)
Age 51-60	2	12	0.61 (0.57-0.66)	11.8	8.5	36 (31-40)
	3	4	0.56 (0.49-0.63)	14.1	7.1	43 (36-50)
	4	0.9	0.33 (0.24-0.46)	21.3	4.7	67 (54-76)
	5	0.8	0.35 (0.25-0.48)	20.6	4.9	65 (52-75)
Age 61+	2	15	0.76 (0.72-0.80)	11.4	8.7	21 (18-25)
	3	5	0.75 (0.69-0.82)	11.6	8.6	24 (18-30)
	4	1.3	0.63 (0.53-0.75)	22.0	4.5	37 (25-47)
	5	1.1	0.62 (0.51-0.75)	17.7	5.6	38 (25-49)

Regression models were adjusted for race/ethnicity, smoking status (current, never or former), categories of supplemental folic acid intake, menopause status, and family history of hypertension.