た高齢者が家事活動やゆっくり散歩、ストレッチングのような低強度の生活活動や運動を含む、座ったり横になったりしていること以外の身体活動を実施する際の強度は概ね1.5〜3メッツ程度、平均すると2.2メッツ程度と思われるため、1日約40分の身体活動の実施と同等と考えられる。このことから65歳以上の高齢者を対象とした基準については「横になったままや座ったままにならなければどんな動きでもよいので、身体活動を毎日40分行う」と表現した。

現状に付加する身体活動量の基準として3メッツ以上の中高強度の身体活動を現状よりも少しでも増やすことを提案した。この目標については「現在の身体活動量を少しでも増やす。今より毎日10分ずつ長く歩くようにする。」と表現した。

- 歩行又はそれと同等以上の強度の身体活動を毎日約60分以上行う。
- 歩数で1日当たり約8,000〜10,000歩
- 息が弾み汗をかく程度の運動を毎週60分行う。
- 65歳以上の高齢者は横になったままや座ったままにならなければどんな動きでも良いので、身体活動を毎日40分行う。
- 現在の身体活動量を少しでも増やす。今より毎日10分ずつ長く歩くようにする。

4. 他国等の身体活動ガイドラインとの比較

世界保健機関（WHO）は、高血圧(13%)、喫煙(9%)、高血糖(6%)に次いで、身体不活動(6%)を全世界の死亡に対する危険因子の第4位と認識し、その対策として「健康のための身体活動に関する国際勧告」を平成22年に発表した(1)。欧米諸国でも、「アメリカ人のための身体活動ガイドライン2008」に代表されるガイドラインがすでに策定されている。WHOや米国では、未成年、成人、高齢者の3つの年代別に基準値を示している。年代により身体活動の状況や目標が異なることから年代別に基準値を示すという考え方は適切なアプローチであると考えられる。

我が国の健康づくりのための運動基準2006では、生活習慣病予防を重視していたため、18歳から69歳までの主に成人を対象とした基準値を定めていた。しかし、急速な高齢化の進行と、健康日本21(第2次)において生活習慣病予防だけでなく社会生活機能の維持を目標としたことにより、今回の運動基準の改定作業において、新たに65歳以上の基準値を提案した。しかし、18歳未満の未成年の基準の策定は見送った。その最大の理由は、未成年の参加者を対象に生活習慣病の発症等をアウトカムとした大規模コホート研究の数が限られていたためである。今後、我が国でも未成年者を長期に追跡する研究を実施し、研究成果を蓄積する必要がある。

我が国では、文部科学省や日本体育協会などが、健康づくりの観点だけではないものの、子どもや未成年を対象とした身体活動・運動のガイドラインや指針を策定している。例えば、未就学児を対象とした「幼児期運動指針」(21)、児童・生徒を対象とした「アクティブ・チャイルド60 min.」(22)などが、健康づくりだけでなく体力向上や発育・発達の促進・運動技能の獲得などを目指して、1日あたり60分の活発な遊びやスポーツを推奨している。今後の基準の改定においては、これらの指針との整合性を取りながら、今後蓄積されるエビデンスをレビューして、18歳未満の未成年の基準を策定していく必要があると考えられる。

WHO、米国とも成人が取り組むべき身体活動の基準値は中強度身体活動を週150分、1日あたり30分としている。WHO、米国、我が国とも基準値策定の根拠となるエビデンスやレビューの手法には違いがないにも関わらず、我が国の身体活動量の基準値は欧米の約2倍の1日60分とした。理由は、我が国の平均的身体活動量がすでにWHOや米国の基準値である1日30分を上回っており、基準値策定の原則「⑤基準値は我が国の現状を下回らない」に基づき、国民全体の身体活動量を増加させる方向に導くために、23メッツ・時／週＝1日60分を身体活動量の基準値とした。他国の基準値は10分以上継続した身体活動や運動の時間を積算しているが、我が国は10分以上の活動や運動に限定していないこと、余暇や移動だけでなく就労や家事などの生活活動などのすべての身体活動を含んでいることなどの理由を挙げることができる。

我が国は、身体活動量や運動量の基準値だけでなく、他国のガイドラインでは類を見ない体力（全身持久力）の基準値を示している。表4・6と表10・11とを比較すると、身体活動量や運動量の基準値の達成者と最も身体活動量・運動量が少ない者との間でのRRの減少は10～20%程度であるが、全身持久力の基準値達成者と最も体力の低い者との間でのRRの減少は約40%と、体力を高めることや維持することの健康利益は大きいことがわかる。したがって、単に身体活動量や運動量の基準を達成するだけでなく、積極的に体力の維持・向上に努めることを推奨するために、体力の基準値を定めている。

E. 結論

平成18年に作成された「健康づくりのための運動基準 2006」の改定を目的として、8名の専門家で構成される研究班で検討を重ねた。改定にあたり、①基準値の変更が必要か検討する、②生活習慣病予防だけでなく、がん予防・社会生活機能の低下予防の観点も重視する、③新しく65歳以上の高齢者のための基準を示す、④簡易な表現でも基準値を示す、⑤全身持久力以外の体力の基準値策定の可能性を探る、⑥量反応関係に基づいた現状に加える身体活動量の基準策定の可能性を探る、を目的とした。これらの観点に基づき、システマティックレビューとメタ解析を用いて検討した結果、以下の5つの基準値あるいは基準を提案する。

① 強度が3メッツ以上の身体活動を23メッツ・時／週行う。（歩行又はそれと同等以上の強度の身体活動を毎日60分以上行う、歩数で1日当たり約8,000～10,000歩）
② 強度が3メッツ以上の運動を4メッツ・時／週行う。（息が弾み汗をかく程度の運動を毎週60分行う）
③ 65歳以上の高齢者に対しては、強度を問わず、身体活動を10メッツ・時／週行う。（横になったままや座ったままにならなければどんな動きでもよいので、身体活動を毎日40分行う）
④ 現在の身体活動量を、少しでも増やす。（今より毎日10分ずつ長く歩くようにする）
⑤ 性・年代別の全身持久力（最大酸素摂取量）の基準値として、男性40歳未満：11.0メッツ、40～59歳：10.0メッツ、60歳以上：9.0メッツ、女性40歳未満：9.5メッツ、40～59歳：8.5メッツ、60歳以上：7.5メッツ
⑥ 65歳以上の高齢者の握力の参照値として、男性38kg重、女性23kg重、また、歩行速度（参照値）：74m／分

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Ⅱ

Adult Mortality Attributable to Preventable Risk Factors for Non-Communicable Diseases and Injuries in Japan: A Comparative Risk Assessment

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Abstract

Background: The population of Japan has achieved the longest life expectancy in the world. To further improve population health, consistent and comparative evidence on mortality attributable to preventable risk factors is necessary for setting priorities for health policies and programs. Although several past studies have quantified the impact of individual risk factors in Japan, to our knowledge no study has assessed and compared the effects of multiple modifiable risk factors for non-communicable diseases and injuries using a standard framework. We estimated the effects of 16 risk factors on cause-specific deaths and life expectancy in Japan.

Methods and Findings: We obtained data on risk factor exposures from the National Health and Nutrition Survey and epidemiological studies, data on the number of cause-specific deaths from vital records adjusted for ill-defined codes, and data on relative risks from epidemiological studies and meta-analyses. We applied a comparative risk assessment framework to estimate effects of excess risks on deaths and life expectancy at age 40 y. In 2007, tobacco smoking and high blood pressure accounted for 129,000 deaths (95% CI: 115,000–154,000) and 104,000 deaths (95% CI: 86,000–119,000), respectively, followed by physical inactivity (52,000 deaths, 95% CI: 47,000–58,000), high blood glucose (34,000 deaths, 95% CI: 26,000–43,000), high dietary salt intake (34,000 deaths, 95% CI: 27,000–39,000), and alcohol use (31,000 deaths, 95% CI: 28,000–35,000). In recent decades, cancer mortality attributable to tobacco smoking has increased in the elderly, while stroke mortality attributable to high blood pressure has declined. Life expectancy at age 40 y in 2007 would have been extended by 1.4 y for both sexes (men, 95% CI: 1.3–1.6; women, 95% CI: 1.2–1.7) if exposures to multiple cardiovascular risk factors had been reduced to their optimal levels as determined by a theoretical-minimum-risk exposure distribution.

Conclusions: Tobacco smoking and high blood pressure are the two major risk factors for adult mortality from non-communicable diseases and injuries in Japan. There is a large potential population health gain if multiple risk factors are jointly controlled.

Please see later in the article for the Editors' Summary.

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Abbreviations: CI, confidence interval; HTLV-1, human T-lymphotropic virus type 1; LDL, low density lipoprotein; NHNS, National Health and Nutrition Survey

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Introduction

Controlling risk factors for non-communicable diseases and external causes is essential for the improvement of adult health. Chronic diseases and injuries are the leading causes of global mortality, accounting for 63% and 9%, respectively, of 57 million deaths in 2008 [1]. The five major risk factors for deaths in the world are high blood pressure, tobacco use, high blood glucose, physical inactivity, and overweight and obesity, which contribute to non-communicable diseases and are modifiable with effective interventions [2]. In such an environment, informed decision-making on priority setting for health policies and programs needs consistent and comparative evidence about how many deaths would be averted by changing profiles of preventable risk factors in a population.

The population of Japan has the longest life expectancy at birth in the world. Life expectancy at birth for Japanese women was 54.0 y in 1947 and rapidly increased until 1986, at which point, at 81.0 y, it became the longest in the world for the first time; female life expectancy at birth also reached its highest ever worldwide figure, 86.4 y, in Japan in 2009 [3]. The continuous extension of longevity was largely explained by a decline in the rate of mortality for communicable diseases among children and young adults during the 1950s and the early 1960s and for stroke since the late 1960s [4]. Current leading causes of death are malignant neoplasm, heart disease, and cerebrovascular disease, accounting for more than 50% of total deaths in 2009 [5]. Accidental injuries and suicide have also ranked in the top ten causes of death for the past 50 y [5], and particularly suicide in the working population is a serious social problem reflecting the prolonged economic recession since the 1990s [4]. To further enhance the health status of the Japanese population, it is therefore crucial to prevent deaths from these major causes.

With the aim of increasing the nation's health through the prevention of premature deaths from lifestyle-related diseases, the Japanese government initiated a 10-y national health promotion campaign called Health Japan 21 in 2000 [6]. In this campaign, 59 indicators were established to monitor and improve the management of risk factors and diseases such as diet, smoking, and diabetes. However, the performance of Health Japan 21 was not necessarily satisfactory: there was progress on 60% of the 59 indicators, including decreasing daily salt intake, while deterioration or no improvement was observed for the remaining 40%, for example, the prevalence of overweight and obesity decreased in women aged 40–60 y but increased in men aged 20–60 y [7]. Success of national health promotion campaigns may partly depend on whether the stewardship of central and local governments exists for coordinating diverse activities and investing resources in priority areas with reference to scientific evidence on the disease burden attributable to modifiable risk factors. Although a number of past studies have quantified population-attributable fractions or impacts on life expectancy for individual risk factors in Japan [8–16], no study to our knowledge has used a single comprehensive framework to assess and compare these impacts across multiple risk factors.

In the present study, we therefore aimed to provide the most comprehensive and comparative assessment of preventable risk factors for mortality from non-communicable diseases and injuries in the Japanese adult population. We employed a comparative risk assessment strategy to quantify contributions of health risks to disease outcomes [17,18]. This standard systematic approach has already been applied to examine the burden of disease and injury across major risk factors in a few other countries [19–22]. Using national data sources on risk exposures and cause-specific mortality, as well as epidemiologic evidence on their causal association from large-scale prospective studies and meta-analyses in Japan, this analysis identifies the most important risk factors for deaths and life expectancy at the population level; the results could inform policymakers of which risk factors need to be prioritized in formulating and revising health policies and programs.

Methods

We estimated the number of deaths that would have been saved in 2007 if multiple risk factors had been controlled at their optimal levels as determined by a theoretical-minimum-risk exposure distribution. To quantify and compare the mortality attributable to excess health risks, we used comparative risk assessment methods that have been described in detail elsewhere [18,20]. To summarize, we first calculated the population-attributable fraction of cause-specific mortality for each risk factor, which measures a proportional reduction in mortality that would be achieved if risk factor exposures of a population shifted to an alternative counterfactual distribution that is more favorable. We used the following formula to calculate population-attributable fractions for continuous exposure variables:

$$\text{Population} - \text{attributable fraction} = \frac{\int_x RR(x)P(x)dx - \int_x RR(x)P'(x)dx}{\int_x RR(x)P(x)dx}, \quad (1)$$

where $P(x)$ and $P'(x)$ are actual and counterfactual distributions of exposure in the population, respectively, and $RR(x)$ is the relative risk of mortality at exposure level x. The first and second terms in the numerator of this equation represent the total risk of mortality weighted by exposures in the population under current and counterfactual distributions, respectively. This approach allowed us to compute effects of all nonoptimal exposures of individuals for all risk factors in a consistent and comparable way [21]. For risks measured in multiple categories, we used the following generalized formula to calculate population-attributable fractions:

$$\text{Population} - \text{attributable fraction} = \frac{\sum_{i=1}^{n} P_i(RR_i - 1)}{\sum_{i=1}^{n} P_i(RR_i - 1) + 1}, \quad (2)$$

where i signifies the level of individual categories ($i = 1, \ldots, n$).

We then multiplied the number of cause-specific deaths by population-attributable fractions to estimate mortality from diseases (causes of death) associated with each risk factor. The number of deaths attributable to a single risk factor was summed across different causes to obtain the total number of deaths attributable to that risk factor. The number of deaths from a single cause, however, could not be added across risk factors, because they may be causally related and we did not account for such relationships in the estimation of population-attributable fractions of individual risk factors.

We conducted all analyses separately by sex, using Stata version 11 (StataCorp). We restricted analyses to individuals aged 30 y and over, because the number of deaths from non-communicable

diseases is small for younger ages. However, we included those aged 20 to 29 y when estimating deaths from external causes attributable to alcohol use, because the burden was assumed to be substantial in this age group.

Mortality Data

We obtained data on the number of cause-specific deaths in 2007 from vital records [23]. We applied algorithms developed for the Global Burden of Disease 2010 Study to redistribute ill-defined codes (e.g., cardiac arrest, heart failure, and senility) on death certificates that were not supposed to be underlying causes of death [24,25]. This method enabled us to obtain valid, reliable, and comparable data on cause-specific mortality by ensuring consistency and resolving changes across revisions of the *International Statistical Classification of Diseases and Related Health Problems*.

Selection of Risk Factors and Diseases

We included 16 risk factors in this analysis (Table 1). In the selection of risk factors paired with their relevant diseases or injuries, we employed the criteria of a previous study: (i) an availability of evidence on causality or association from high-quality epidemiological studies, (ii) an existence of interventions to modify exposures, and (iii) an availability of data on risk exposures from nationally representative surveys or large population studies [20]. We also included infection by several agents—hepatitis B virus, hepatitis C virus, the bacterium *Helicobacter pylori*, human papillomavirus, and human T-lymphotropic virus type 1 (HTLV-1)—because they are important risk factors for cancer deaths in Japan [26,27].

Measures and Data Sources of Risk Factor Exposures

Table 2 lists measurements and data sources for the risk factor exposures used in this analysis, and Table 3 shows their basic statistics by sex and age group in 2007. With the exception of tobacco smoking, infections, and alcohol use related to deaths from traffic road accidents, we used individual records from the National Health and Nutrition Survey (NHNS) in 2007. NHNS was a survey based on a nationally representative probabilistic sample to provide data on the health and nutritional status of the Japanese population. This survey included an in-person interview on medication use and lifestyle-related risk factors, a physical examination by health care professionals, and self-administered questionnaires on diet and lifestyle [28].

We used self-reports to quantify exposures to physical inactivity and alcohol use, while we used measured data for other risk factors. In the physical examination for the 2007 NHNS, a blood test was intended to be conducted more than 4 h after a meal, although a number of blood samples were actually drawn less than 4 h after a meal. Because fasting plasma glucose was the unit for relative risk for high blood glucose adopted in the present study, we applied the following conversion equation proposed by the Committee of the Japan Diabetes Society [29,30] to predict equivalents of fasting plasma glucose from measurements of hemoglobin A1c:

Table 1. Risk factors and disease outcomes included in the study.

Risk Factor	Disease Outcomes
High blood glucose	IHD, stroke, diabetes mellitus
High LDL cholesterol	IHD, ischemic stroke
High blood pressure	IHD, stroke, hypertensive diseases, other cardiovascular diseases[a]
Overweight/obesity	IHD; ischemic stroke; hypertensive disease; postmenopausal breast, colon, corpus uteri, kidney, and pancreatic cancers; diabetes mellitus
Alcohol use	IHD; ischemic stroke; hemorrhagic stroke; hypertensive diseases; cardiac arrhythmias; cancers of breast, colorectal, esophagus, mouth, liver, larynx, pharynx, and selected other sites[b]; diabetes mellitus; liver cirrhosis; acute and chronic pancreatitis; road traffic injuries; falls; homicide and suicide; other injuries
Tobacco smoking	IHD; stroke; aortic aneurysms and dissection; diabetes mellitus; lung, esophagus, mouth, pharynx, stomach, liver, pancreas, cervix, bladder, kidney, and other urinary cancers; leukemia; chronic obstructive pulmonary disease; lower respiratory tract infections; asthma; tuberculosis
Physical inactivity	IHD, ischemic stroke, breast and colon cancers, diabetes mellitus
High dietary trans fatty acids	IHD
Low dietary polyunsaturated fatty acids	IHD
High dietary salt	IHD, stroke, hypertensive disease, other cardiovascular diseases[a], stomach cancer
Low intake of fruit and vegetables	IHD; ischemic stroke; colorectal, esophagus, lung, mouth, pharynx, and stomach cancers
Hepatitis B virus	Liver cancer
Hepatitis C virus	Liver cancer
H. pylori	Stomach cancer
Human papillomavirus	Cervix uteri cancer
HTLV-1	Adult T-cell lymphoma/leukemia

[a]This category includes rheumatic heart disease, endocarditis, cardiomyopathy, aortic aneurysms, peripheral vascular disorders, and other ill-defined cardiovascular diseases.

[b]This category includes *International Statistical Classification of Diseases and Related Health Problems, 10th edition* (ICD-10) codes D00–D24 (except D09.9), D26–D37 (except D37.9), and D38–D48 (except D38.6, D39.9, D40.9, D41.9, and D48.9).

IHD, ischemic heart disease.

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Table 2. Measurements, data sources, and alternative distributions of risk exposures.

Risk Factor, Exposure Metric, Data Source[a]	Optimal	Guidelines/National Goals
High blood glucose		
Fasting plasma glucose (mmol/l)	4.9 (0.3)	5.6 (0.3) [66]
High LDL cholesterol		
LDL cholesterol (mmol/l)	2.0 (0.4)	3.1 (0.7) [54]
High blood pressure		
Systolic blood pressure (mm Hg)	115 (6)	130 (7) [53]
Overweight/obesity		
Body mass index (kg/m^2)	21 (1)	22 (1) [67]
Alcohol use		
Current alcohol consumption volumes and patterns	No alcohol use[b]	
Alcohol-related road traffic accidents, national road accident data, 2004 [40]	No alcohol use	
Tobacco smoking		
Smoking impact ratio, vital statistics 2007 data [23–25], pooled cohort studies [15,35,36]	No smoking	
Physical inactivity		
Intensity of physical activity	Highly active	
High dietary trans fatty acids		
Percent of total calories from dietary trans fatty acids	0.5 (0.05)	
Low dietary polyunsaturated fatty acids		
Percent of total calories from dietary polyunsaturated fatty acids	10 (1)	
High dietary salt		
Dietary sodium adjusted for total calories (g/d)	0.5 (0.05)	10 (1) [7]
Low intake of fruit and vegetables		
Dietary fruit and vegetable intake adjusted for total calories (g/d)	600 (50)	350 (29) [7]
Hepatitis B virus		
Seropositivity for hepatitis B surface antigen, blood donors' cohort, 1991–1993 [37]	No infection	
Hepatitis C virus		
Seropositivity for antibody to hepatitis C, blood donors' cohort, 1991–1993 [37]	No infection	
H. pylori		
Seropositivity for anti-*H. pylori* immunoglobulin G, multi-center study, late 1990s [38]	No infection	

Values are means, with standard deviations in parentheses.
[a]We obtained exposure data from the 2007 National Health and Nutrition Survey [28] unless stated otherwise.
[b]The optimal category for liver cancer and suicide was "occasional drinkers" because previous studies used it as the reference category for estimation of relative risks.
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$$\text{Fasting plasma glucose (mg/dl)} = -9.2 + 21.9 \times \text{hemoglobin A1c}_{\text{JDS}}\ (\%) \quad (3)$$

where hemoglobin $A1c_{JDS}$ is a value standardized by calibrators provided by the Japan Diabetes Society and lower than an internationally used value by around 0.4% [29]. As a minor adjustment, we further deducted from this equation a difference in means between predicted fasting plasma glucose and measured casual plasma glucose among 165 participants in the 2007 NHNS who had fasted for more than 8 h (6.4 mg/dl).

In the NHNS, health care professionals measured the blood pressure of seated persons in their right upper arm after 5 min of rest, using a Riva-Rocci mercury manometer. For a trend analysis of cardiovascular mortality attributable to high blood pressure, which is described below, we used the National Nutrition Surveys for 1980–2002 and the NHNS for 2003–2007. These surveys took only one blood pressure measurement per individual until starting to collect two measurements per individual in the 2000 survey. We therefore used a single measurement for the surveys in 1980–1999 and the second measurement for the 2000–2007 surveys. We excluded pregnant or breastfeeding women from the analysis of blood pressure.

For dietary risk exposure variables, dieticians visited households to distribute questionnaires and explain the survey method for diet and lifestyle. Household representatives weighed and recorded the quantity of each food item consumed for one day (excluding holidays). Dieticians visited households again during the survey period to check and correct completed questionnaires. We estimated intakes of dietary trans fatty acids using conversion factors of food items provided by the Cabinet of Japan Food Safety Committee [31]. Considering that nutrition intakes are correlated with energy intake determined by body size, physical activity, and metabolic efficiency, we adjusted intakes of fruit, vegetables, and dietary sodium for total energy intake with a simple linear regression equation having nutrient intake as a dependent variable and total caloric intake as an

Table 3. Exposure to risk factors by sex and age group in 2007.

Sex, Risk Factor	Age														
	30–44 y			45–59 y			60–69 y			70–79 y			≥80 y		
	n[a]	Mean	SE	*n*[a]	Mean	SE	*n*[a]	Mean	SE	*n*[a]	Mean	SE	*n*[a]	Mean	SE
Men															
Fasting plasma glucose (mmol/l)	300	5.4	0.1	374	5.7	0.0	411	6.0	0.1	339	5.9	0.0	107	5.9	0.1
LDL cholesterol (mmol/l)	300	3.3	0.0	375	3.4	0.0	413	3.1	0.0	340	3.0	0.0	108	2.9	0.1
Systolic blood pressure (mm Hg)	312	124.2	0.8	394	133.9	0.9	427	140.9	0.9	359	142.2	1.0	116	144.1	1.8
Body mass index (kg/m^2)	673	23.9	0.1	777	23.8	0.1	620	23.8	0.1	470	23.6	0.2	155	22.6	0.3
Dietary TFA (% of total calories)	806	0.3	0.0	858	0.3	0.0	664	0.2	0.0	517	0.2	0.0	179	0.3	0.0
Dietary PUFA (% of total calories)	806	5.7	0.1	858	5.6	0.1	664	5.3	0.1	517	5.1	0.1	179	5.0	0.1
Dietary SFA (% of total calories)	806	6.8	0.1	858	6.2	0.1	664	5.7	0.1	517	5.6	0.1	179	5.9	0.2
Dietary salt intake (g/d)	806	11.4	0.2	858	12.3	0.2	664	12.6	0.2	517	12.2	0.2	179	10.9	0.3
Fruit and vegetable intake (g/d)	804	288.6	6.0	856	342.5	6.6	663	432.3	8.8	515	446.8	9.6	178	463.5	15.9
Never or former drinkers (%)[b]	850	26.6	1.5	950	25.9	1.4	699	28.9	1.7	525	40.8	2.1	184	55.4	3.7
Alcohol-related accidents/four-wheel vehicle road traffic accidents, 2004 (%) [40][c]		1.7			1.7			1.7			1.7			1.7	
Have intense physical activity (%)	808	34.8	1.7	869	34.5	1.6	667	28.8	1.8	518	42.1	2.2	179	21.8	3.1
Never or former smokers (%)	850	45.2	1.7	946	53.6	1.6	698	65.9	1.8	524	78.6	1.8	184	82.1	2.8
Smoking impact ratio		0.0			0.6			0.5			0.5			0.7	
Hepatitis B virus (%) [37]		0.9			0.9			0.9			0.6			0.6	
Hepatitis C virus (%) [37]		0.6			1.6			2.6			7.9			7.9	
H. pylori (%) [38]		23.6			47.4			66.1			73.4			72.6	
Women															
Fasting plasma glucose (mmol/l)	563	5.3	0.0	620	5.7	0.0	523	5.9	0.0	408	5.9	0.0	154	5.9	0.1
LDL cholesterol (mmol/l)	565	2.9	0.0	622	3.4	0.0	523	3.5	0.0	410	3.3	0.0	154	3.2	0.1
Systolic blood pressure (mm Hg)	527	112.4	0.6	652	128.1	0.8	560	135.8	0.8	433	138.9	0.8	170	143.2	1.4
Body mass index (kg/m^2)	874	21.4	0.1	905	22.7	0.1	723	23.3	0.1	534	23.1	0.2	248	22.4	0.3
Dietary TFA (% of total calories)	955	0.4	0.0	957	0.3	0.0	762	0.3	0.0	561	0.3	0.0	285	0.2	0.0
Dietary PUFA (% of total calories)	955	5.9	0.1	957	6.0	0.1	762	5.6	0.1	561	5.3	0.1	285	5.3	0.1
Dietary SFA (% of total calories)	955	7.8	0.1	957	7.0	0.1	762	6.2	0.1	561	5.9	0.1	285	5.7	0.2
Dietary salt intake (g/d)	955	9.6	0.1	957	10.7	0.1	762	10.9	0.2	561	10.6	0.2	285	10.0	0.2
Fruit and vegetable intake (g/d)	951	346.0	6.1	957	460.2	7.6	761	541.3	9.0	561	522.8	9.7	284	490.3	13.2
Never or former drinkers (%)[b]	1,014	54.9	1.6	1,047	61.1	1.5	795	75.5	1.5	579	83.4	1.5	310	88.7	1.8
Alcohol-related accidents/four-wheel vehicle road traffic accidents, 2004 (%) [40][c]		1.7			1.7			1.7			1.7			1.7	
Have intense physical activity (%)	958	36.3	1.6	959	40.9	1.6	765	39.0	1.8	562	45.4	2.1	286	21.3	2.4
Never or former smokers (%)	1,014	81.8	1.2	1,047	87.1	1.0	794	92.1	1.0	579	96.5	0.8	310	95.5	1.2
Smoking impact ratio		0.0			0.1			0.2			0.2			0.2	
Hepatitis B virus (%) [37]		0.5			0.5			0.5			0.6			0.6	
Hepatitis C virus (%) [37]		0.4			1.6			3.5			7.0			7.0	
H. pylori (%) [38]		23.6			47.4			66.1			73.4			72.6	

[a]Sample size in the National Health and Nutrition Survey in 2007.
[b]For those aged 20–29 y, the mean (standard error) was 40.4 (2.7) in men ($n = 324$) and 53.9 (2.5) in women ($n = 395$).
[c]Reported for the total age group of both sexes combined.
PUFA, polyunsaturated fatty acids; SE, standard error; SFA, saturated fatty acids; TFA, trans fatty acids.
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independent variable [32]. Calorie-adjusted nutrient intakes were computed as the sum of residuals from the regression model and the expected nutrient intake for a person with mean caloric intake.

We used a smoking impact ratio as a more reliable indicator of accumulated exposure to tobacco smoking than the prevalence of current smokers. The smoking impact ratio was defined as total lung cancer mortality in excess of never-smokers in a study

population relative to the excess lung cancer mortality among current smokers in a reference population [33,34]. We used the following formula to calculate smoking impact ratios by age group and sex:

$$\text{Smoking impact ratio} = \frac{C_{LC} - N_{LC}}{S^*_{LC} - N^*_{LC}} \times \frac{N^*_{LC}}{N_{LC}}, \qquad (4)$$

where C_{LC} and N_{LC} denote lung cancer mortality of the total population and never-smokers, respectively, in a study population (i.e., the Japanese population), and S^*_{LC} and N^*_{LC} signify lung cancer mortality among current smokers and never-smokers, respectively, in a reference population. We obtained total lung cancer mortality from the redistributed data of vital records described above. Our reference population was residents included in a pooled study of three large-scale cohorts in Japan [15,35,36]. Because we also adopted never-smokers' lung cancer mortality in the Japanese population from this pooled study, N_{LC} and N^*_{LC} were equivalent to each other in our analysis.

We obtained data on the prevalence of infections with hepatitis B and C viruses and the bacterium *H. pylori* from epidemiological studies undertaken in Japan in the 1990s [37,38]. Assuming that infection rates do not vary within birth cohorts over time, we applied infection rates by age group in the 1990s to those of corresponding age in 2007. For example, the infection rate for hepatitis B virus in men aged 60–69 y in 2007 was that of men aged 45–54 y in 1991–1993. We considered that all deaths from cervix uteri cancer and adult T-cell lymphoma/leukemia were caused by infections with human papillomavirus and HTLV-1, respectively [26,39].

In order to measure exposure levels of alcohol use related to deaths from road traffic injuries, we employed a proportion of alcohol-impaired driving, which was defined as driving with breath alcohol concentrations above 0 mg/l, to the total number of cases of road traffic accidents involving four-wheeled vehicles and motorcycles in 2004 (1.7%). We obtained this figure from a past study on alcohol concentrations in the breath of drivers, which used a national dataset prepared by the Japan Institute for Traffic Accident Research and Data Analysis [40].

Selection of Relative Risks

Tables S1, S2, S3, S4, S5, S6, S7 provide details of relative risks used in this analysis. We conducted a literature review of prospective studies evaluating effects of risk factors on cause-specific deaths in Japan. Strategies for the database search involved contacting authors of key reports and leading experts in the field, and we critically appraised the identified literature. Our motive for undertaking the literature search was to identify evidence from past studies in the Japanese population to be backed up with pooled evidence establishing causalities or associations from the Global Burden of Disease Study [20]. Criteria for the selection of evidence for the Japanese population were: (i) pooled or individual estimates from large-scale prospective observational studies and (ii) confirming causalities or associations that had been already established in past studies. When there was no study for the Japanese population satisfying these conditions, we sought evidence from the Asia-Pacific Cohort Studies Collaboration. If we could not find evidence from this source, then we adopted relative risks identified in the Global Burden of Disease Study. We considered relative risks to be null if they were statistically insignificant. In addition, we had to restrict the source of evidence on relative risks for tobacco smoking to the pooled analysis of large-scale cohorts in Japan, because we used their estimates of current smokers' and never-smokers' lung cancer mortality of a reference population to calculate smoking impact ratios. We excluded mortality from tuberculosis and diabetes mellitus associated with tobacco smoking, because the studies did not examine these causes.

Counterfactual Distributions of Risk Exposures

As an alternative distribution of risk exposures, we used an optimal distribution in which harmful effects of each risk factor on morbidity and mortality would be minimized in a population (i.e., a theoretical-minimum-risk exposure distribution). With the exception of infections, we obtained information on theoretical-minimum-risk exposure distributions from a previous study in the United States (Table 2) [20].

In the analysis of gains in life expectancy and probabilities of death, we also investigated alternative counterfactual distributions of risk exposures that followed recommendations of clinical guidelines and goals of Health Japan 21. This analysis enabled quantification of potential health gains that would be more realistic than theoretical minimums. We included risk factors in this part of our analysis only if specific control targets were available from these sources and units of measurement corresponded to those of relative risks (Table 2). In order to obtain counterfactual distributions for numerical risks, we used their control threshold as the mean and applied the coefficient of variation to estimate the standard deviation.

The relationship between dietary salt intake and cardiovascular mortality was based on a convincing effect of high dietary salt on systolic blood pressure that was estimated from a meta-analysis of dietary trials (Table S6) [20]. In order to obtain hazards of excess dietary salt intake on cardiovascular death, we first estimated the decrease in systolic blood pressure associated with a reduction in dietary salt intake to individual optimal levels and then applied relative risks of high systolic blood pressure for relevant cardiovascular diseases (Table S1).

Effects on Life Expectancy and Probabilities of Death

We translated mortality changes into gains in life expectancy at 40 y of age to understand the potential impact of the management of risk factors on longevity. We constructed life tables using observed age-specific mortality rates and mortality that would be expected if risk factor exposures were controlled at alternative levels. We took the differences between these values as showing life expectancy gains that would occur when shifting from an actual risk factor exposure to a counterfactual. We also calculated effects on probabilities of dying between the ages of 15 and 60 y ($_{45}q_{15}$) and between 60 and 75 y ($_{15}q_{60}$).

Joint Effects of Multiple Risk Factors for Cardiovascular Mortality

We estimated joint effects of multiple risk factors on excess mortality from cardiovascular diseases and the additional life expectancy at age 40 y that would be achieved under counterfactual distributions. Risk factors included in this part of the analysis were high body mass index, high blood pressure, and high concentrations of blood glucose and low density lipoprotein (LDL) cholesterol. We took account of high dietary sodium intake to compensate for its indirect effect through elevated blood pressure, using the steps described above. We also adopted a 50% reduction of the excess risk of high body mass index on cardiovascular deaths to incorporate a mediation of its associations through other risk factors [21]. We used an additive excess risk scale to correct for correlations of these risk factors and calculate joint relative risks at

the individual level. This approach has been described in detail elsewhere [21]. We summed the combined relative risks for individual records to compute population-attributable fractions for the joint effects of these cardiovascular risks.

Long-Term Trends in Attributable Deaths

To examine contributions of the management of modifiable risk factors to the improvement of life expectancy over time, we estimated the number of deaths from cancers attributable to tobacco smoking and deaths from stroke associated with high blood pressure from 1980 to 2007. We employed the algorithm described above to obtain consistent mortality data throughout this period, from which we used total lung cancer mortality in each year to calculate smoking impact ratios over time. For the analysis of high blood pressure and stroke, we excluded people over 80 y of age because the sample size was insufficient. We also incorporated the above-mentioned mediated effects of dietary sodium intake through raised blood pressure at the individual level.

Uncertainty Analyses

We conducted statistical simulation to deal with the uncertainty that was introduced by using sample estimates for risk exposures and relative risks [41]. To account for sampling variability, we randomly drew 1,000 sets of values of all components based on samples. In each sequential step of the simulation, we drew for each age–sex group: (i) a random sample of participants in the 2007 NHNS with replacement to obtain the original sample size of those who had no missing value for each risk factor, (ii) a relative risk for each risk–disease pair from a log-normal distribution with means and standard deviations reported in epidemiological studies, (iii) coefficients of the regression of hemoglobin A1c on fasting plasma glucose from a normal distribution with standard deviations that we calculated from information given in a past study (1.0 for the constant term and 0.2 for the coefficient of hemoglobin A1c) [30], (iv) the difference in means between predicted fasting plasma glucose and measured casual plasma glucose in the 2007 NHNS from a normal distribution with mean of 6.4 mg/dl and standard deviation of 1.1 mg/dl that we estimated from the survey data, (v) the proportion of the excess risk of body mass index mediated through systolic blood pressure and fasting plasma glucose from a normal distribution with mean of 0.5 and standard deviation of 0.1 [21], and (vi) lung cancer mortality of current smokers and never-smokers from a normal distribution with means and standard deviations estimated from the pooled analysis of Japanese cohorts [15,35]. We used each sampled set of risk exposures and relative risks to compute population-attributable fractions, mortality attributable to each risk factor or a combination of risk factors, and changes in life expectancy under counterfactual distributions. We defined a 95% confidence interval (CI) by a span across the estimates of each outcome at the 2.5th and 97.5th percentiles of the 1,000 simulations.

Results

Contributions of Health Risks to Cause-Specific Mortality in 2007

Tables S8 and S9 provide population-attributable fractions of the 16 modifiable risk factors and a combination of physiological risk factors for mortality from non-communicable diseases and injuries by age group and sex in 2007. These fractions cannot to be summed across risk factors for a single cause of death, because causal relationships between risk factors are not considered in the analysis of individual risk factors.

Under the theoretically minimum counterfactuals listed in Table 2, tobacco smoking and high blood pressure were the two major single contributors to the number of deaths from non-communicable diseases and injuries (Table 4). Among the total of 960,000 deaths from causes included in this study, tobacco smoking was associated with 129,000 deaths (95% CI: 115,000–154,000). Approximately three-quarters of these deaths occurred in men (95,000 deaths, 95% CI: 88,000–103,000), although the attributable mortality was still substantial for women (34,000 deaths, 95% CI: 23,000–57,000). In men, 70% of deaths attributable to this risk factor were caused by cancers and took place among those aged 45–79 y. In women, cardiovascular diseases and cancers accounted for 42% and 36%, respectively, of the mortality attributable to tobacco smoking. By disease subtypes for sexes combined, lung cancer was the leading cause (42,000 deaths, 95% CI: 39,000–45,000), followed by ischemic heart disease (27,000 deaths, 95% CI: 19,000–42,000) and chronic obstructive pulmonary disease (13,000 deaths, 95% CI: 9,000–16,000).

High blood pressure was associated with 104,000 cardiovascular deaths (95% CI: 86,000–119,000) in 2007. This was the greatest risk factor for cardiovascular mortality of all risk factors included in this analysis, and the mortality burden was shared evenly between the sexes. A majority of deaths attributable to high blood pressure occurred among people aged 70 y and over (85,000 deaths) and were caused by stroke (47,000 deaths, 95% CI: 38,000–56,000) or ischemic heart disease (28,000 deaths, 95% CI: 15,000–39,000).

Although the numbers of attributable deaths for other physiological, lifestyle, dietary, and infectious factors were small when compared to those for tobacco smoking and high blood pressure, most of these other factors were associated with tens of thousands of deaths from non-communicable diseases and external causes. Physical inactivity was associated with 52,000 deaths (95% CI: 47,000–58,000), and 75% of them occurred among people aged 70 y and older. Ischemic heart disease was the major cause of mortality attributable to this risk factor (31,000 deaths, 95% CI: 28,000–35,000). High blood glucose was associated with 34,000 deaths (95% CI: 26,000–43,000), of which 75% occurred among people aged 70 y and over and 68% were caused by ischemic heart disease. High dietary salt intake was associated with 19,000 cardiovascular deaths (95% CI: 16,000–22,000), which were included in cardiovascular mortality attributable to high blood pressure, and there were 15,000 deaths from stomach cancer (95% CI: 9,000–20,000). Seventy-six percent of deaths attributable to this risk factor occurred among people aged 70 y and over.

Alcohol use was associated with 31,000 deaths (95% CI: 27,000–35,000) from non-communicable diseases and injuries, 84% of which occurred among men. A major cause of death attributable to this risk factor was liver cirrhosis (11,000 deaths, 95% CI: 10,000–12,000), followed by liver cancer (6,000 deaths, 95% CI: 4,000–8,000), esophagus cancer (5,000 deaths, 95% CI: 4,000–5,000), and colon cancer (4,000 deaths, 95% CI: 4,000–5,000). Alcohol use was associated with 3,000 (95% CI: 2,000–5,000) out of 83,000 deaths of people aged 20 y and over from external causes included in this study. Two thousand deaths were from suicide (95% CI: 1,000–4,000), and there were fewer than 1,000 deaths each attributable to falls, road traffic accidents, homicide, and other injuries. Most of the suicide deaths attributable to alcohol use occurred among men, particularly those aged 30 to 59 y (71%).

Infection with *H. pylori* was associated with 31,000 deaths from gastric cancer in 2007 (95% CI: 27,000–34,000). Seventy-two

Table 4. The number of deaths attributable to risk factors in Japan, 2007 (in thousands).

Sex, Risk Factor	Total		Cardiovascular		Cancer		Diabetes Mellitus		Respiratory		Other NCD		Injuries	
Sexes combined														
High blood glucose	34.1	(26.4, 43.1)	27.2	(19.5, 36.2)			6.9							
High LDL cholesterol	23.9	(16.7, 31.2)	23.9	(16.7, 31.2)										
High blood pressure	103.9	(86.0, 119.1)	103.9	(86.0, 119.1)										
High body mass index	19.0	(16.1, 21.9)	13.8	(11.1, 16.4)	4.1	(3.4, 4.9)	1.1	(0.8, 1.3)						
Alcohol use	30.6	(27.5, 34.7)	−2.0	(−4.0, 0.0)	18.2	(16.2, 20.8)	−0.1	(−0.1, −0.1)			11.6	(10.6, 12.7)	2.9	(1.9, 4.6)
Tobacco smoking	128.9	(115.5, 153.6)	33.4	(25.4, 48.8)	77.4	(72.3, 83.9)			18.1	(12.6, 26.4)				
Physical inactivity	52.2	(46.7, 57.7)	42.2	(36.6, 47.6)	9.3	(8.5, 10.0)	0.7	(0.6, 0.9)						
High TFA intake	0.0	(0.0, 0.0)	0.0	(0.0, 0.0)										
Low PUFA intake	21.2	(8.1, 38.7)	21.2	(8.1, 38.7)										
High dietary sodium intake	34.0	(27.3, 39.4)	19.0	(16.1, 22.3)	14.9	(8.8, 19.6)								
Low fruit and vegetable intake	8.9	(6.7, 10.8)	5.1	(3.3, 6.7)	3.8	(2.5, 4.9)								
Hepatitis B virus	11.6	(9.8, 13.5)			11.6	(9.8, 13.5)								
Hepatitis C virus	23.0	(21.3, 24.5)			23.0	(21.3, 24.5)								
H. pylori	30.6	(27.2, 33.5)			30.6	(27.2, 33.5)								
Human papillomavirus	2.6				2.6									
HTLV-1	1.1				1.1									
Joint risk[a]	157.0	(144.0, 173.4)	157.0	(144.0, 173.4)										
Men														
High blood glucose	17.2	(12.7, 22.2)	14.3	(9.8, 19.3)			2.9							
High LDL cholesterol	12.2	(8.1, 15.9)	12.2	(8.1, 15.9)										
High blood pressure	50.1	(39.9, 58.5)	50.1	(39.9, 58.5)										
High body mass index	12.1	(10.0, 14.3)	9.6	(7.6, 11.6)	2.0	(1.6, 2.6)	0.5	(0.4, 0.7)						
Alcohol use	25.8	(22.6, 29.5)	−1.7	(−3.7, 0.2)	15.9	(13.7, 18.2)	−0.1	(−0.1, −0.1)			9.0	(8.2, 9.7)	2.8	(1.7, 4.4)
Tobacco smoking	94.9	(87.7, 103.4)	19.3	(15.4, 24.5)	66.5	(61.8, 71.2)			9.1	(6.8, 10.9)				
Physical inactivity	25.9	(22.8, 29.4)	21.0	(18.1, 24.4)	4.6	(4.0, 5.1)	0.3	(0.3, 0.4)						
High TFA intake	0.0	(0.0, 0.0)	0.0	(0.0, 0.0)										
Low PUFA intake	12.0	(5.3, 29.3)	12.0	(5.3, 29.3)										
High dietary sodium intake	18.4	(13.0, 22.7)	8.8	(7.3, 10.2)	9.7	(4.4, 13.7)								
Low fruit and vegetable intake	7.6	(5.5, 9.5)	4.3	(2.5, 5.9)	3.3	(2.1, 4.4)								
Hepatitis B virus	8.0	(6.6, 9.6)			8.0	(6.6, 9.6)								
Hepatitis C virus	14.8	(13.4, 16.1)			14.8	(13.4, 16.1)								
H. pylori	20.0	(17.0, 22.4)			20.0	(17.0, 22.4)								
HTLV-1	0.6				0.6									
Joint risk[a]	78.5	(70.8, 87.8)	78.5	(70.8, 87.8)										
Women														
High blood glucose	16.9	(11.6, 23.3)	12.9	(7.6, 19.3)			4.0							
High LDL cholesterol	11.7	(6.5, 18.0)	11.7	(6.5, 18.0)										
High blood pressure	53.9	(40.0, 66.9)	53.9	(40.0, 66.9)										
High body mass index	6.8	(5.0, 8.9)	4.2	(2.5, 5.9)	2.1	(1.6, 2.6)	0.5	(0.3, 0.8)						
Alcohol use	4.8	(3.8, 6.3)	−0.3	(−0.5, −0.1)	2.3	(1.7, 3.3)	−0.0	(−0.1, −0.0)			2.6	(2.1, 3.4)	0.2	(0.1, 0.3)
Tobacco smoking	34.0	(22.9, 56.5)	14.1	(7.3, 28.2)	10.9	(8.3, 15.7)			9.0	(4.0, 17.4)				
Physical inactivity	26.3	(21.6, 30.9)	21.2	(16.5, 25.9)	4.7	(4.2, 5.2)	0.4	(0.3, 0.5)						
High TFA intake	0.0	(0.0, 0.0)	0.0	(0.0, 0.0)										
Low PUFA intake	9.3	(4.0, 16.1)	9.3	(4.0, 16.1)										
High dietary sodium intake	15.6	(11.3, 19.2)	10.3	(7.7, 13.1)	5.3	(1.9, 7.5)								

Table 4. Cont.

Sex, Risk Factor	Total		Cardiovascular		Cancer		Diabetes Mellitus	Respiratory	Other NCD	Injuries
Low fruit and vegetable intake	1.3	(0.9, 1.7)	0.8	(0.4, 1.2)	0.5	(0.3, 0.6)				
Hepatitis B virus	3.6	(2.8, 4.5)			3.6	(2.8, 4.5)				
Hepatitis C virus	8.2	(7.3, 9.0)			8.2	(7.3, 9.0)				
H. pylori	10.6	(8.9, 12.0)			10.6	(8.9, 12.0)				
Human papillomavirus	2.6				2.6					
HTLV-1	0.5				0.5					
Joint risk[a]	78.5	(66.9, 91.1)	78.5	(66.9, 91.1)						

Values in parentheses indicate lower and upper bounds of 95% CI.
[a]A combination of high blood glucose, high LDL cholesterol, high blood pressure (directly, and indirectly through high dietary salt intake), and high body mass index.
NCD, non-communicable disease; PUFA, polyunsaturated fatty acids, TFA, trans fatty acids.
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percent of these deaths occurred among people aged 70 y and older. Infection with hepatitis C virus was associated with 23,000 deaths from liver cancer (95% CI: 21,000–24,000). Forty-five percent of these deaths were in people aged 70–79 y, including those born in the early 1930s. For both *H. pylori* and hepatitis C virus infections, around 65% of the attributable mortality took place in men.

High LDL cholesterol was associated with 24,000 cardiovascular deaths (95% CI: 17,000–31,000), largely from ischemic heart disease (23,000 deaths, 95% CI: 16,000–30,000). Low dietary intake of polyunsaturated fatty acids was associated with 21,000 deaths from ischemic heart disease (95% CI: 8,000–39,000), and 47% of these deaths occurred among people aged 80 y and over. High body mass index was associated with 19,000 deaths (95% CI: 16,000–22,000): 64% of these deaths occurred in men, and ischemic heart disease was the major cause (11,000 deaths, 95% CI: 8,000–13,000).

If systolic blood pressure (directly, and indirectly through dietary salt intake), blood glucose, LDL cholesterol, and body mass index were controlled jointly to their optimal distributions, i.e., theoretical-minimum-risk exposure distributions, 157,000 cardiovascular deaths would have been prevented in 2007 (95% CI: 144,000–173,000). The mortality burden attributable to the combination of these risks was shared equally between the sexes, and a majority of the burden occurred among people aged 70 y and older.

Effects of Risk Factors on Life Expectancy and Probabilities of Death

Japanese life expectancy at age 40 y was 40.4 y for men and 46.8 y for women in 2007 [3]. Figure 1 illustrates gains in life expectancy at age 40 y and percentage changes in probabilities of death that would have been expected in 2007 if risk factors had been controlled to their theoretically minimum distributions individually or jointly with others. For men, tobacco smoking was associated with the largest potential increase in life expectancy at age 40 y (1.8 y, 95% CI: 1.6–1.9), followed by a joint effect of multiple physiological factors (1.4 y, 95% CI: 1.3–1.6) and single effects of high systolic blood pressure (0.9 y, 95% CI: 0.7–1.0) and alcohol use (0.5 y, 95% CI: 0.5–0.6). A considerable part of the smoking effect (1.2 y, 95% CI: 1.1–1.3) was accounted for by an expected associated fall in cancer mortality through a decrease in probabilities of dying between the ages of 15 and 60 (45q15) by 8% (95% CI: 7–10) and between the ages of 60 and 75 (15q60) by 13% (95% CI: 12–14). A drop in cardiovascular mortality through the joint control of multiple risks was associated with an expected percentage decrease of 13% (95% CI: 12–15) in 45q15 and 11% (95% CI: 10–13) in 15q60, while controlling high blood pressure was associated with an expected fall of 7% both in 45q15 (95% CI: 6–8) and 15q60 (95% CI: 5–8). Decreasing alcohol use was associated with an expected percentage decrease of 9% (95% CI: 7–11) in 45q15 and 5% (95% CI: 5–6) in 15q60 for men. A substantial part of the potential change in the probability of death among young and middle-aged men through moderate drinking was explained by a fall in mortality from other non-communicable diseases including liver cirrhosis and liver cancer (4%, 95% CI: 4–4) and injuries (2%, 95% CI: 1–4).

For women, controlling systolic blood pressure and tobacco smoking to optimal counterfactuals would have extended life expectancy at age 40 y by 0.9 y (95% CI: 0.7–1.1) and 0.6 y (95% CI: 0.4–1.0), respectively. The impact of tobacco smoking on a probability of death in older women was estimated to be 8% (95% CI: 5–13), which was comparable to that of high blood pressure (7%, 95% CI: 6–8). A joint effect of cardiovascular risk factors on female life expectancy at age 40 y was estimated to be 1.4 y (95% CI: 1.2–1.7), with a decrease in probability of death of 8% (95% CI: 7–10) for younger adults and 11% (95% CI: 10–12) for older ages.

Table 5 shows changes in life expectancy at age 40 y and probabilities of death under the more practical counterfactuals defined by clinical guideline recommendations and national goals. Overall, the gains were less than half of those under theoretically minimum distributions. In both sexes, life expectancy at age 40 y would have increased by 0.7 y (95% CI: 0.6–0.9) through the joint control of cardiovascular risks and by 0.4 y (95% CI: 0.3–0.5) through reducing systolic blood pressure to the distribution recommended by clinical guidelines.

Trends in Mortality Attributable to Tobacco Smoking and High Blood Pressure in 1980–2007

Figure 2 illustrates trends in the number of deaths from cancers that were attributable to tobacco smoking from 1980 to 2007. A continuous increase has been observed in men over 70 y old and women over 80 y old. A fall after a peak around 1995 among men aged 60–69 y reflected the fact that the lifetime smoking prevalence reached a peak in the birth cohort of the late 1920s and decreased in the cohort of the late 1930s [42]. This effect manifested again as a peak in attributable cancer mortality around 2005, when the birth cohort of the late 1920s was 70–79 y old. A temporary halt in the increase of cancer deaths attributable to

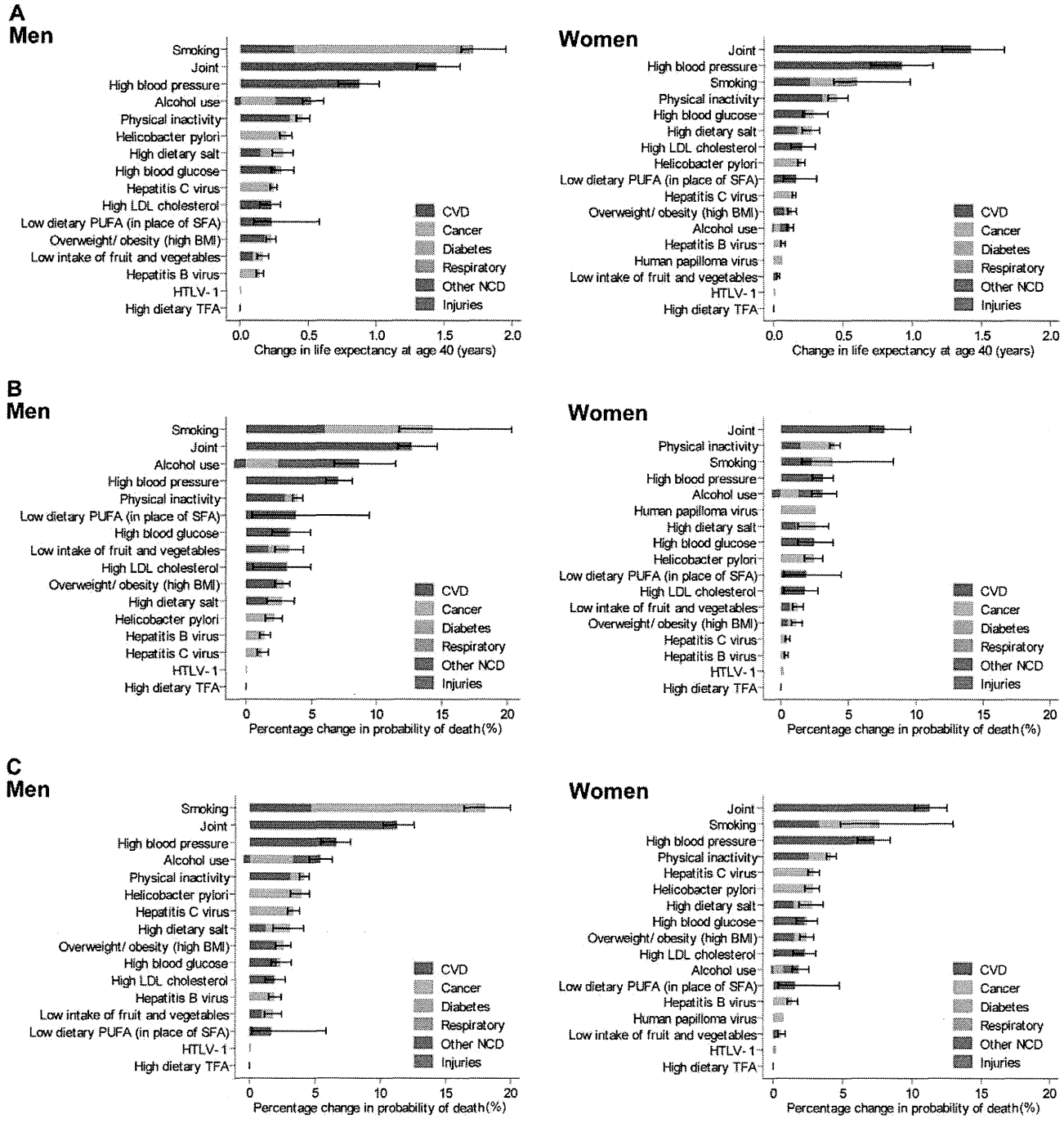

Figure 1. Changes in life expectancy at age 40 y and the probability of death under optimal distributions of risk factors in Japan, 2007. (A) Life expectancy at age 40. (B) Probability of death between 15 and 60 y of age. (C) Probability of death between 60 and 75 y of age. Joint risk is a combination of high blood pressure (directly, or indirectly through high dietary salt intake), high blood glucose, high LDL cholesterol, and high body mass index. BMI, body mass index; CVD, cardiovascular disease; NCD, non-communicable diseases; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids; TFA, trans fatty acids.
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tobacco smoking for men over 80 y old in the early 2000s reflected a reduction in this population group as a result of the 1918 influenza pandemic.

Figure 3 demonstrates trends in the number of deaths from stroke that were attributable to high blood pressure. Stroke deaths associated with this risk factor, either directly or indirectly through high dietary sodium intake, consistently declined for both sexes under 80 y of age. This favorable trend continued in the 2000s for women and for men under the age of 60 y, but it ceased for elderly men by the mid-1990s.