

Table 1 Factor-loading matrix for the major dietary pattern identified by factor analysis

	Japanese pattern	'Animal food' pattern	DFA ^a pattern
Rice			-0.59
Miso soup	0.25		-0.39
Beef		0.48	
Pork (excluding ham, sausage)		0.55	
Ham, sausage		0.56	
Chicken		0.49	
Liver		0.43	
Egg	0.34	0.32	
Milk	0.26		0.28
Yoghurt			0.50
Cheeses		0.44	
Butter		0.50	
Margarine		0.37	0.40
Deep fried-dishes, tempura	0.28	0.39	
Fried vegetable	0.43		
Raw fish, fish boiled with soy, roast fish	0.51		
Boiled fish paste	0.39	0.32	
Dried fish	0.37		
Green vegetables	0.64		
Carrot, pumpkin	0.59		0.36
Tomato	0.45		0.32
Cabbage, lettuce	0.59		
Chinese cabbage	0.62		
Wild plant	0.27		
Mushrooms (shiitake, enokitake)	0.42		
Potato	0.61		
Seaweeds	0.59		0.26
Pickles (radish, chinese cabbage)	0.41		
Food boiled with soy			
Boiled beans			
Soybean (tofu, fermented soybeans)	0.57		
Orange	0.50		0.42
Other fruits	0.49		0.47
Fresh juice			
Confectioneries	0.27		
Green tea	0.29		
Black tea			
Coffee		0.29	
Chinese tea			
Alcoholic beverages		0.27	-0.50
Variance explained (%)	15.1	6.4	4.8

^a DFA means high-dairy, high-fruit-and-vegetable, low-alcohol. Absolute values <0.25 were not listed for simplicity.

with a higher Japanese dietary pattern score tended to be older, were more likely to walk and have a history of hypertension, and less likely to be current drinkers and smokers. Participants with a higher 'animal food' dietary pattern score tended to be younger and male, were more likely to be current smokers and drinkers, and less likely to have a history of hypertension. Participants with a higher DFA dietary pattern score tended to be female, and were similar to those with a higher Japanese dietary pattern score except for walking duration, education and history of hypertension.

Table 3 shows total energy-adjusted daily nutrient and food intakes according to dietary pattern score quartiles. Although the fat and protein intakes in any given quintile of dietary patterns were almost equivalent, their sources varied among dietary patterns. Participants with a higher Japanese dietary pattern score consumed more fish and soybean, a higher 'animal food' dietary pattern score was associated with higher meat and fat intake, and a higher DFA dietary pattern was associated with a higher intake of dairy products. Although the Japanese and DFA dietary patterns were similar in terms of high vegetable and fruit intake, the Japanese dietary pattern differed from the DFA pattern in terms of high intakes of sodium, fish, soybean, seaweeds and green tea.

During 7 years of follow-up (252 647 person-years), we documented 801 deaths from CVD. These deaths included 181 CHDs, 432 total strokes (163 cerebral infarctions, 129 ICHs). Table 4 shows the association between the three dietary pattern score quartiles and CVD mortality. The Japanese dietary pattern was associated with a reduced risk of CVD mortality. On the other hand, the 'animal food' dietary pattern was associated with an increased risk of CVD mortality, and the DFA dietary pattern was not associated with CVD mortality.

After adjustment for age, sex, smoking status, walking duration, education and total energy intake, the multivariate HRs (95% CI) of CVD mortality across increasing quartiles of the Japanese dietary pattern score were 1.00, 0.76 (0.63–0.93), 0.71 (0.58–0.87) and 0.73 (0.59–0.90) (*P* for trend = 0.003), whereas for the 'animal food' dietary pattern, the corresponding multivariate HRs (95% CI) of CVD mortality were 1.00, 0.93 (0.76–1.13), 1.13 (0.92–1.38) and 1.22 (0.99–1.51) (*P* for trend = 0.03). With additional adjustment for body mass index and history of hypertension, the results were essentially unchanged. Furthermore, the results of analysis using body mass index as a continuous variable and history of hypertension remained similar.

As the Japanese and 'animal food' dietary patterns were associated with CVD mortality, their associations with CHD and stroke were further investigated (Table 5). After adjusting for potential confounders, the point estimate of the HR for CHD mortality of participants with the highest quartile of the Japanese dietary pattern score was 20% lower than the lowest, whereas those with the highest quartile of the 'animal food' dietary pattern score was 49% higher.

The multivariate HRs (95% CI) of CHD mortality across increasing quartiles of the Japanese dietary pattern score were 1.00, 0.84 (0.56–1.26), 0.70 (0.45–1.08) and 0.80 (0.51–1.25) (*P* for trend = 0.24), and the corresponding multivariate HRs (95% CI) of the 'animal food' dietary pattern score were 1.00,

Table 2 Baseline characteristics of the participants according to dietary pattern score quartiles

Characteristic	Dietary pattern score quartiles			
	1 (lowest)	2	3	4 (highest)
Age (years), mean (SD)				
Japanese pattern	57.7 (11.1)	59.3 (10.5)	60.6 (9.9)	61.9 (9.3)
'Animal food' pattern	63.8 (8.9)	60.6 (10.1)	58.2 (10.4)	57.0 (10.6)
DFA ^a pattern	58.5 (10.1)	60.5 (10.4)	60.5 (10.4)	60.1 (10.4)
Male (%)				
Japanese pattern	55.3	48.3	43.4	41.3
'Animal food' pattern	23.5	42.2	53.4	69.1
DFA ^a pattern	89.8	53.0	28.8	16.7
Body mass index (kg/m²), mean (SD)				
Japanese pattern	23.4 (3.3)	23.5 (3.2)	23.5 (3.1)	23.6 (3.1)
'Animal food' pattern	23.8 (3.4)	23.6 (3.2)	23.5 (3.1)	23.3 (3.0)
DFA ^a pattern	23.5 (3.0)	23.5 (3.3)	23.7 (3.3)	23.5 (3.2)
Current smoker (%)				
Japanese pattern	44.8	35.1	28.6	24.4
'Animal food' pattern	18.1	30.0	36.1	46.9
DFA ^a pattern	58.0	35.7	21.3	14.8
Walking duration ≥1 hour/day (%)				
Japanese pattern	41.2	45.0	47.7	52.1
'Animal food' pattern	43.9	44.8	47.7	49.5
DFA ^a pattern	54.8	47.3	44.0	40.0
Current drinker (%)				
Japanese pattern	55.6	51.1	46.9	43.5
'Animal food' pattern	29.6	45.9	54.6	64.8
DFA ^a pattern	80.0	49.6	34.4	29.8
Education until age ≥19 years (%)				
Japanese pattern	8.3	7.7	7.6	7.8
'Animal food' pattern	5.8	7.3	8.9	9.4
DFA ^a pattern	4.4	5.8	7.8	13.4
History of hypertension (%)				
Japanese pattern	22.2	23.4	25.7	27.0
'Animal food' pattern	31.6	26.2	21.7	18.9
DFA ^a pattern	22.3	25.0	25.7	25.4

^a DFA means high-dairy, high-fruit-and-vegetable, low-alcohol. SD, standard deviation.

1.12 (0.73–1.72), 1.38 (0.89–2.15) and 1.49 (0.94–2.34) (*P* for trend = 0.06).

The Japanese dietary pattern was associated with a decreased risk of total stroke, cerebral infarction and ICH mortality. The multivariate HRs (95% CI) of total stroke mortality across increasing quartiles of the Japanese dietary pattern score were 1.00, 0.70 (0.54–0.92), 0.66 (0.50–0.87) and 0.64 (0.47–0.85) (*P* for trend = 0.003). The 'animal food' dietary pattern was not positively associated with the risk of total stroke.

We analysed the association between the Japanese dietary pattern derived from participants who left no blanks for food items (*n* = 17010) and CVD mortality, and the results were similar. The multivariate HR (95% CI) of CVD mortality for the

highest quartile of the Japanese dietary pattern score vs the lowest was 0.74 (0.51–1.07) (*P* for trend = 0.04). After exclusion of participants who reported daily energy intakes at the extreme 2.5% instead of the extreme 0.5% of the upper or lower ends of the range, we analysed the association between the Japanese dietary pattern derived and CVD mortality (*n* = 38 937), and the results were similar. The multivariate HR (95% CI) of CVD mortality for the highest quartile of the Japanese dietary pattern score vs the lowest was 0.75 (0.60–0.93) (*P* for trend = 0.005).

After excluding the 287 participants who died from CVD in the first 3 years of follow-up, the multivariate HR (95% CI) of CVD mortality for the highest quartile of the Japanese dietary pattern score vs the lowest was 0.70 (0.54–0.92)

Table 3 Daily nutrient and food intakes of the participants according to dietary pattern score quartiles

Variable	Dietary pattern score quartiles			
	1 (lowest)	2	3	4 (highest)
Daily nutrient and food intakes^a (median)				
Energy (kJ)				
Japanese pattern	5046	5793	6227	6861
'Animal food' pattern	5128	5826	6430	7458
DFA ^b pattern	8346	5956	5537	5466
Fat (g)				
Japanese pattern	31	35	38	41
'Animal food' pattern	34	35	37	41
DFA ^b pattern	32	35	37	41
Protein (g)				
Japanese pattern	61	66	70	74
'Animal food' pattern	65	66	68	72
DFA ^b pattern	68	67	67	69
Sodium (mg)				
Japanese pattern	2418	2762	2925	3152
'Animal food' pattern	2909	2824	2768	2822
DFA ^b pattern	2684	2865	2897	2878
Rice (g)				
Japanese pattern	814	684	620	567
'Animal food' pattern	617	647	734	744
DFA ^b pattern	885	739	601	524
Total meats (g)				
Japanese pattern	20	21	22	22
'Animal food' pattern	14	19	24	34
DFA ^b pattern	21	21	21	22
Dairy products (g)				
Japanese pattern	127	191	217	223
'Animal food' pattern	192	198	201	206
DFA ^b pattern	86	142	226	268
Total Fish (g)				
Japanese pattern	50	64	73	96
'Animal food' pattern	68	67	66	69
DFA ^b pattern	69	67	66	68
Total vegetables (g)				
Japanese pattern	59	79	99	138
'Animal food' pattern	101	89	86	87
DFA ^b pattern	66	82	96	118
Soybean (g)				
Japanese pattern	64	82	98	101
'Animal food' pattern	95	88	85	87
DFA ^b pattern	87	86	89	92
Seaweeds (g)				
Japanese pattern	3	5	6	9
'Animal food' pattern	6	5	5	5
DFA ^b pattern	4	5	6	7

Variable	Dietary pattern score quartiles			
	1 (lowest)	2	3	4 (highest)
Total fruits (g)				
Japanese pattern	111	145	173	204
'Animal food' pattern	178	155	147	147
DFA ^b pattern	79	132	183	238
Green tea consumption ≥ 5 cups/day (%)				
Japanese pattern	16	26	33	44
'Animal food' pattern	39	31	26	25
DFA ^b pattern	29	30	30	32

^a Nutrient and food intakes presented in this table are adjusted for total energy intake.

^b DFA means high-dairy, high-fruit-and-vegetable, low-alcohol.

(P for trend = 0.009). We performed additional analysis restricted to participants who performed vigorous activity and with a well self-perceived health status ($n = 27\,239$, 312 deaths from CVD), and a similar result was obtained. The multivariate HR (95% CI) of CVD mortality for the highest quartile of the Japanese dietary pattern score vs the lowest was 0.71 (0.50–1.01) (P for trend = 0.07). When we used the model adjusted for total energy intake by the residual method, the multivariate HR (95% CI) of CVD mortality for the highest quartile of the Japanese dietary pattern score vs the lowest was 0.78 (0.64–0.96) (P for trend = 0.03).

The results of further analysis stratified by smoking status on the association between the Japanese dietary pattern and CVD mortality are shown in Table 6. Although the point estimates of the HR for CVD mortality were consistently below unity compared with the reference category, the inverse association was less pronounced among current smokers than among never or past smokers ($P = 0.07$ for interaction with smoking). No interaction between the other covariates and quartiles of the Japanese dietary pattern score was observed (data not shown).

Discussion

We identified three dietary patterns among the Japanese population: the Japanese, 'animal food' and DFA dietary patterns. The Japanese dietary pattern was associated with a decreased risk of CVD mortality. In contrast, the 'animal food' dietary pattern was associated with an increased risk of CVD mortality, and the DFA dietary pattern was not.

Three dietary patterns we identified were consistent with previously reported patterns observed among different Japanese populations.^{20–23} Corresponding to the (i) Japanese, (ii) 'animal food' and (iii) DFA dietary patterns, the previous studies reported dietary patterns that were correlated with (i) vegetables, fruits, seaweeds, soy products and fish,^{20–23} (ii) meat and fat^{20–23} and (iii) rice (negatively), miso soup (negatively) or bread and dairy products.^{20,22,23} Although our FFQ is short, it includes these key foods, especially Japanese foods.

Table 4 Age, sex-adjusted and multivariate-adjusted hazard ratio for cardiovascular disease mortality according to dietary pattern score quartiles

Dietary pattern	Dietary pattern score quartiles				P for Trend
	1 (low)	2	3	4 (high)	
Japanese pattern					
No. of deaths	243	189	179	190	
Person-years	62 529	63 035	63 316	63 767	
Age, sex-adjusted HR (95% CI)	1	0.73 (0.60–0.88)	0.64 (0.53–0.78)	0.63 (0.52–0.77)	<0.001
Multivariate-adjusted HR ^{1a} (95% CI)	1	0.76 (0.63–0.93)	0.71 (0.58–0.87)	0.73 (0.59–0.90)	0.003
Multivariate-adjusted HR ^{2b} (95% CI)	1	0.77 (0.63–0.94)	0.71 (0.58–0.88)	0.74 (0.59–0.91)	0.004
'Animal food' pattern					
No. of deaths	244	188	184	185	
Person-years	63 113	63 396	63 143	62 996	
Age, sex-adjusted HR (95% CI)	1	0.90 (0.74–1.09)	1.04 (0.85–1.27)	1.07 (0.88–1.32)	0.31
Multivariate-adjusted HR ^{1a} (95% CI)	1	0.93 (0.76–1.13)	1.13 (0.92–1.38)	1.22 (0.99–1.51)	0.03
Multivariate-adjusted HR ^{2b} (95% CI)	1	0.93 (0.76–1.13)	1.14 (0.93–1.39)	1.24 (1.00–1.54)	0.02
DFA^c pattern					
No. of deaths	180	251	211	159	
Person-years	63 303	62 878	62 848	63 618	
Age, sex-adjusted HR (95% CI)	1	1.27 (1.04–1.55)	1.17 (0.94–1.45)	0.94 (0.74–1.19)	0.38
Multivariate-adjusted HR ^{1a} (95% CI)	1	1.18 (0.96–1.45)	1.07 (0.85–1.35)	0.88 (0.68–1.13)	0.14
Multivariate-adjusted HR ^{2b} (95% CI)	1	1.18 (0.96–1.45)	1.10 (0.87–1.38)	0.89 (0.69–1.14)	0.19

^a Multivariate-adjusted HR1 was adjusted for age (in years), sex, smoking status (never, former, currently smoking <20 cigarettes/day and currently smoking 20 cigarettes/day), walking duration (<1 hour/day and 1 hour/day), education (until age up to 15 years, until age 16–18 years, and until age 19 years) and total energy intake (continuous).

^b Multivariate-adjusted HR2 was adjusted for the same as HR1, body mass index (<18.5 kg/m², 18.5–24.9 kg/m² and 25 kg/m²), and history of hypertension (yes or no).

^c DFA means high-dairy, high-fruit-and-vegetable, low-alcohol.

HR, hazard ratio; CI, confidence interval.

The Japanese dietary pattern has partly similar characteristics to 'healthy' dietary patterns reported previously among Western populations that were inversely associated with CVD mortality. High consumption of vegetables and fruits is a common component of 'healthy' dietary patterns in Western populations.^{13–19} These components might partly explain the possible protective effect of the Japanese dietary pattern against CVD mortality including CHD and stroke, although we might have failed to detect associations with CHD mortality due to insufficient statistical power. However, compared with 'healthy' dietary patterns among Western populations, the Japanese dietary pattern also has unique characteristics.

The Japanese diet has so far been considered to increase the risk of CVD because it includes a large amount of salt.³⁵ In the present study, the Japanese dietary pattern was related to higher sodium consumption (Table 3) and higher prevalence of hypertension (Table 2). In spite of these risk factors, the Japanese dietary pattern was associated with lower CVD mortality. Although some components of the Japanese diet (i.e. salt) increase the risk of hypertension, other components may compensate for this, and decrease the risk of CVD. Components unique to the Japanese diet would include items such as soybeans, seaweeds and green tea. The effect of those foods upon CVD risk has yet to be clarified.

Factor analysis has both strengths and limitations. On the one hand, it can overcome multicollinearity of various dietary variables, because it is a statistical dimension-reduction technique that exploits the correlation of each variable. However, factor analysis requires several decisions about the methods used for extracting initial factors and rotation.³⁶ Even after multiple sensitivity analyses using these methods and various groupings by age and sex, the dietary patterns were essentially unchanged.

Our study also had other limitations. First, healthy behaviour in adhering to a Japanese dietary pattern could have confounded the association between dietary patterns and mortality. Although we adjusted our data using measured potential confounders including non-dietary variables, we could not completely exclude the effects of unmeasured confounders. Second, 12% of the participants were lost to follow-up. This proportion did not vary across the quartiles of each dietary pattern score (13%, 13%, 12% and 11% of participants from the lowest to highest Japanese dietary pattern score quartiles, respectively). Therefore, we consider it unlikely that the association between each dietary pattern and CVD mortality was substantially distorted by the effect of loss to follow-up.

Third, our FFQ did not ask about individual portion size, and preparation of foods including added oil. Some misclassification of food consumption could have arisen in deriving dietary pattern scores and estimating the effect of the patterns on

Table 5 Multivariate-adjusted hazard ratio for coronary heart disease and stroke mortality according to dietary pattern score quartiles

Variable	Dietary pattern score quartiles				P for Trend
	1 (low)	2	3	4 (high)	
Coronary heart disease					
Japanese pattern					
No. of deaths	54	45	38	44	
Multivariate-adjusted HR1 ^a (95% CI)	1	0.84 (0.56–1.26)	0.70 (0.45–1.08)	0.80 (0.51–1.25)	0.24
Multivariate-adjusted HR2 ^b (95% CI)	1	0.86 (0.57–1.29)	0.71 (0.46–1.11)	0.82 (0.52–1.29)	0.29
'Animal food' pattern					
No. of deaths	42	44	46	49	
Multivariate-adjusted HR1 ^a (95% CI)	1	1.12 (0.73–1.72)	1.38 (0.89–2.15)	1.49 (0.94–2.34)	0.06
Multivariate-adjusted HR2 ^b (95% CI)	1	1.10 (0.72–1.70)	1.39 (0.89–2.16)	1.50 (0.95–2.37)	0.05
Total stroke					
Japanese pattern					
No. of deaths	138	100	97	97	
Multivariate-adjusted HR1 ^a (95% CI)	1	0.70 (0.54–0.92)	0.66 (0.50–0.87)	0.64 (0.47–0.85)	0.003
Multivariate-adjusted HR2 ^b (95% CI)	1	0.71 (0.54–0.92)	0.67 (0.51–0.88)	0.64 (0.48–0.86)	0.004
'Animal food' pattern					
No. of deaths	142	103	103	84	
Multivariate-adjusted HR1 ^a (95% CI)	1	0.89 (0.68–1.15)	1.11 (0.84–1.43)	0.97 (0.72–1.31)	0.79
Multivariate-adjusted HR2 ^b (95% CI)	1	0.89 (0.69–1.15)	1.11 (0.85–1.45)	1.00 (0.74–1.35)	0.66
Cerebral infarction					
Japanese pattern					
No. of deaths	48	44	38	33	
Multivariate-adjusted HR1 ^a (95% CI)	1	0.89 (0.59–1.36)	0.73 (0.47–1.15)	0.60 (0.37–0.99)	0.03
Multivariate-adjusted HR2 ^b (95% CI)	1	0.89 (0.59–1.36)	0.73 (0.47–1.15)	0.60 (0.37–0.99)	0.03
'Animal food' pattern					
No. of deaths	51	43	35	34	
Multivariate-adjusted HR1 ^a (95% CI)	1	1.04 (0.68–1.57)	1.08 (0.69–1.70)	1.11 (0.69–1.78)	0.66
Multivariate-adjusted HR2 ^b (95% CI)	1	1.03 (0.68–1.56)	1.09 (0.69–1.71)	1.14 (0.71–1.85)	0.57
Intracerebral hemorrhage					
Japanese pattern					
No. of deaths	45	29	25	30	0.04
Multivariate-adjusted HR1 ^a (95% CI)	1	0.62 (0.38–1.00)	0.52 (0.31–0.87)	0.60 (0.35–1.01)	0.04
Multivariate-adjusted HR2 ^b (95% CI)	1	0.63 (0.39–1.02)	0.52 (0.31–0.88)	0.60 (0.36–1.03)	
'Animal food' pattern					
No. of deaths	44	28	30	27	
Multivariate-adjusted HR1 ^a (95% CI)	1	0.71 (0.44–1.15)	0.87 (0.53–1.43)	0.82 (0.48–1.41)	0.59
Multivariate-adjusted HR2 ^b (95% CI)	1	0.71 (0.44–1.16)	0.89 (0.54–1.46)	0.86 (0.50–1.47)	0.71

^a Multivariate-adjusted HR1 was adjusted for age (in years), sex, smoking status (never, former, currently smoking <20 cigarettes/day and currently smoking 20 cigarettes/day), walking duration (<1 hour/day and 1 hour/day), education (until age up to 15 years, until age 16–18 years, and until age 19 years), and total energy intake (continuous).

^b Multivariate-adjusted HR2 was adjusted for the same as HR1, body mass index (<18.5 kg/m², 18.5–24.9 kg/m², and 25 kg/m²) and history of hypertension (yes or no).

HR, hazard ratio; CI, confidence interval.

Table 6 Multivariate-adjusted hazard ratio for cardiovascular disease mortality according to the Japanese dietary pattern score quartiles stratified by smoking status

Variable	Dietary pattern score quartiles				P for Trend
	1 (low)	2	3	4 (high)	
Never smoker					
No. of deaths	82	71	76	73	
Multivariate-adjusted HR1 ^a (95% CI)	1	0.70 (0.50–0.97)	0.68 (0.48–0.95)	0.54 (0.38–0.78)	0.002
Multivariate-adjusted HR2 ^b (95% CI)	1	0.70 (0.50–0.97)	0.68 (0.48–0.95)	0.54 (0.37–0.78)	0.002
Past smoker					
No. of deaths	53	28	30	27	
Multivariate-adjusted HR1 ^a (95% CI)	1	0.53 (0.33–0.85)	0.53 (0.33–0.85)	0.53 (0.32–0.89)	0.01
Multivariate-adjusted HR2 ^b (95% CI)	1	0.53 (0.33–0.85)	0.53 (0.33–0.86)	0.53 (0.32–0.89)	0.01
Current smoker					
No. of deaths	87	62	45	61	
Multivariate-adjusted HR1 ^a (95% CI)	1	0.79 (0.57–1.10)	0.66 (0.46–0.97)	0.92 (0.64–1.33)	0.41
Multivariate-adjusted HR2 ^b (95% CI)	1	0.79 (0.57–1.11)	0.66 (0.46–0.97)	0.93 (0.65–1.34)	0.44

HR, hazard ratio; CI, confidence interval.

^a Multivariate-adjusted HR1 was adjusted for age (in years), sex, walking duration (<1 hour/day and ≥ 1 hour/day), education (until age up to 15 years, until age 16–18 years, and until age ≥ 19 years), and total energy intake (continuous).

^b Multivariate-adjusted HR2 was adjusted for the same as HR1, body mass index (<18.5kg/m², 18.5–24.9kg/m², and ≥ 25kg/m²), and history of hypertension (yes or no).

CVD mortality. However, this misclassification may be non-differential and would tend to result in underestimation of the impact of the dietary patterns.

In conclusion, we have found that the Japanese dietary pattern is associated with lower CVD mortality, despite the fact that the Japanese dietary pattern appeared to be related to higher sodium intake and high prevalence of hypertension.

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Conflict of interest: None declared.

KEY MESSAGES

- Although ecological observations suggest that the Japanese diet may reduce the risk of cardiovascular disease, the impact of a Japanese dietary pattern upon mortality due to CVD is unclear.
- The association between dietary patterns among 40 547 Japanese aged 40–79 years and CVD mortality was examined in a 7-year prospective cohort study.
- The Japanese dietary pattern was associated with a lower risk of CVD mortality, despite its relation to sodium intake.

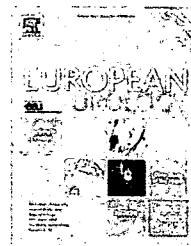
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Incontinence

Association between Physical Activity and Urinary Incontinence in a Community-Based Elderly Population Aged 70 Years and Over

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Abstract

Objectives: The objective of the present study was to evaluate the association between physical activity (PA) levels and urinary incontinence (UI) in a community-based elderly population aged ≥ 70 yr.

Methods: This population-based cross-sectional survey was conducted in 2003 using an extensive health interview for each participant. A self-reported single-item questionnaire was used to estimate different levels of PA in each subject. The prevalence of UI was estimated by the self-administered International Consultation on Incontinence Questionnaire. The study population included 676 Japanese men and women.

Results: The prevalence of UI was 25% (34% in women and 16% in men). After adjustment for potential confounding factors, the odds ratio (95% confidence interval) of UI compared with the lowest PA group was 0.71 (0.47–1.09) and 0.58 (0.35–0.96) in subjects exhibiting middle and high levels of PA, respectively (p for trend = 0.02).

Conclusions: High PA level was independently related to a lower self-reported prevalence of UI in a community-dwelling elderly population aged ≥ 70 yr. Although this cross-sectional study cannot demonstrate a temporal relationship between PA and the onset of UI, the findings suggest that PA may have a potentially beneficial effect on the prevention of UI. A prospective study or randomized trials are required to clarify the causality.

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1. Introduction

Urinary incontinence (UI) is a frequent and bothersome symptom that is common in the elderly population [1]. Population-based estimates of the prevalence of UI among elderly subjects (age ≥ 60 yr) is 47.6% for women and 14.5% for men in Japan [2]. UI is increasingly recognized as a health and economic problem that affects the physical, psychological, social, and economic well-being of individuals and their families and poses a substantial economic burden on health and social services [3,4].

Understanding the risk factors of UI is an important step toward developing direct treatment resources and providing preventive care for UI. Based on many epidemiologic studies on UI in various populations, a number of risk factors have been identified. Some chronic medical conditions, such as diabetes and Hypertension, have also been found to be associated with the occurrence of UI [2,5], and obesity is an especially well-established factor that can cause UI or contribute to the severity of the condition [6]. Regular physical activity (PA) was confirmed to be effective for the primary and secondary prevention of these chronic diseases or conditions (eg, diabetes, hypertension, and obesity) [7]. Furthermore, pelvic floor muscle dysfunction is also a risk factor for UI. Burgio et al have shown that pelvic floor exercise is an effective treatment for both stress and urge incontinence [8]. Because PA is a determinant of muscle strength including abdominal muscles [9], and abdominal muscle training indirectly strengthens the pelvic floor muscles [10], PA may prevent UI through an improvement in abdominal muscle strength.

Therefore, it is conceivable that PA may have a potentially beneficial effect on the prevention of UI. However, to our knowledge, only a few studies that have investigated the relationship between PA and UI [11–16], and their results have not suggested any beneficial effect of PA on the prevention of UI. Moreover, no studies fully assessed the relationship between PA and UI in a community-dwelling elderly population. Thus, it is still unclear how PA relates to UI in community-dwelling elderly adults aged ≥ 70 yr among whom this condition is highly prevalent.

Thus, we designed a cross-sectional study to investigate the relationship between PA and UI in community-dwelling elderly aged ≥ 70 yr.

2. Methods

2.1. Subjects

Our study population comprised subjects aged ≥ 70 yr who were living in the Tsurugaya area of Sendai, one of the major

cities in the Tohoku area of Japan. The Tsurugaya investigators conducted two cross-sectional surveys in 2002 and 2003 [17]. We used 2003 data in this study because the 2002 survey did not collect information on UI. In 2003, 2625 persons aged ≥ 70 yr lived in Tsurugaya. We invited all of them to participate in a comprehensive geriatric assessment of medical status, physical function, cognitive function, severity of UI, and dental status, and 948 (36.1%) of them did so, giving their informed consent for analysis of the data. All assessments were carried out in nonclinical public facility. The protocol of this study was approved by the Institutional Review Board of the Tohoku University Graduate School of Medicine.

We excluded subjects who did not complete the questionnaire items regarding PA ($n = 73$). We also excluded participants who had the potential for PA restriction, that is, those who reported they were incapable of walking 50 m independently ($n = 77$) or who had a history of stroke ($n = 28$) or depression (Geriatric Depression Scale scores of ≥ 14 ; $n = 94$). As a result, we analyzed 346 women and 330 men (mean age \pm standard deviation, 75.5 ± 4.4 yr).

2.2. Assessment of UI

The International Consultation Incontinence Questionnaire (ICIQ) was used to assess the prevalence and types of UI and to determine UI scores [18]. The ICIQ is a new subjective measure for evaluating the frequency, amount of leakage, and quality-of-life (QOL) impact of UI using four questions. This questionnaire was developed under the auspices of the International Consultation on Incontinence and the content validity, reproducibility, and responsiveness of the test have been investigated [18]. Linguistic validation of the Japanese translation of ICIQ was also confirmed [19].

2.3. PA questionnaire

A self-reported single-item questionnaire was used to estimate different levels of PA in each subject. The question asked whether the subject had performed any activities from the following categories in the previous 12 mo: walking, brisk walking, or sports (eg, aerobics, tennis, swimming, jogging, etc). If they had participated in a given activity, the frequency and duration spent in the activity were ascertained using the following categories: for frequency, (1) 1–2 times/mo, (2) 1–2 times/wk, (3) 3–4 times/wk, or (4) almost every day; and for duration (per walk or workout), (1) 0–30 min, (2) 0.5–1 h, (3) 1–2 h, (4) 2–3 h, (5) 3–4 h, or (6) ≥ 4 h. Among the levels of exercise intensity, sports were considered the highest, followed in order by brisk walking and walking. Each of the three types was further classified into three subcategories according to the frequency and duration of the walks or workouts as follows [20]: (1) high, at least 3–4 times/wk for at least 30 min each time; (2) low, reporting some activity in the past year, but not enough to meet high levels; and (3) none, no PA. Finally, we used these categories and subcategories to define the following three levels of PA: (1) “low,” no walking or low walking, no brisk walking, no sports; (2) “middle,” high walking, low brisk walking, no sports; and (3) “high,” any walking, high brisk walking, low or high sports.

2.4. Assessment of other variables

Anthropometrics (height, body weight) were recorded by a standardized protocol. Body mass index (BMI) was calculated as weight (kg)/height² (m²). Blood pressure was measured at the left upper arm two times using an automatic device (HEM747IC; Omron Life Science, Tokyo, Japan) following 5 min rest in the resting position.

Blood samples were drawn from the antecubital vein of the seated subject with minimal tourniquet use. Specimens were collected in siliconized vacuum glass tubes containing sodium fluoride for blood glucose and no additives for lipids analyses.

Blood glucose concentration was measured by enzymatic methods (Shino-Test, Tokyo, Japan). Information on smoking status, drinking status, medication, delivery, and histories of diseases were obtained from the questionnaire survey. The drug information was confirmed by a well-trained pharmacist.

2.5. Statistical analysis

On the basis of the positive answers to question 4 in the ICIQ, “leaks when you cough or sneeze” and/or “leaks when you are physically active/exercising” were defined as stress incontinence, “leaks before you can get to the toilet” as urge incontinence, and the combinations as mixed incontinence. Other positive answers, such as to “leaks when you are asleep,” “when you have finished urinating and are dressed,” “for no obvious reason,” and “all the time,” were regarded as other incontinence, irrespective of the presence of stress, urge, or mixed incontinence. If the participants claimed one of these symptoms, we treated them as having UI. A single summed score associated with the degree of UI was also calculated based on the ICIQ [18]. The score ranged from 0 to 21 with greater values indicating increased severity. Because the distribution of the ICIQ scores was rather skewed,

log-transformed ICIQ scores were used to assess the relationship between PA levels and ICIQ score [21]. When we calculated log-transformed ICIQ scores, 1.0 was added (ICIQ score + 1) before transformation.

Hypertension was defined as a systolic blood pressure of ≥ 140 mm Hg and/or a diastolic blood pressure of ≥ 90 mm Hg or use of nondiuretic antihypertensive agents. Diabetes was defined as a casual blood glucose concentration of ≥ 200 mg/dl or current use of antidiabetic medication.

Descriptive data are presented as means \pm standard error or percentages. The age- and sex-adjusted variable differences according to continence status were examined by analysis of covariance (ANCOVA) for continuous variables or by the multiple logistic regression analysis for variables of proportion. The odds ratio (OR) and 95% confidence interval (CI) of UI (0: no incontinence; 1: urinary incontinence) compared with the lowest PA level were calculated using multiple logistic regression analysis. Model fit was evaluated using the Hosmer-Lemeshow goodness-of-fit statistic. For all models, the test was not significant ($p \geq 0.54$). ANCOVA was used to examine the adjusted relation of PA with log-transformed ICIQ score. We used age, sex, BMI, diabetes, hypertension (nondiuretic drugs), history of coronary heart disease (CHD) or nephropathy, alcohol consumption, use of tranquilizers, use of hypnotics, use of diuretics, and history of delivery as covariates for multiple adjustments. For sex-specific analyses, we used age, BMI, diabetes, hypertension (including diuretic drugs), history of CHD or nephropathy, use of tranquilizers, use of hypnotics, and history of delivery in women and adjustment for age, BMI, hypertension (including diuretic drugs), history of nephropathy in men as covariates. When men and women were analyzed separately, the amount of variables was limited to \leq (amount of cases)/10 (ie, ≤ 11 in women and ≤ 5 in men) in multiple regression models [22]. All p values for linear trend were calculated by using the category

Table 1 – Characteristics of the population according to urinary continence status

	No incontinence (n = 507)	Urinary incontinence (n = 169)	<i>p</i>
Age, yr [†]	75.17 \pm 0.19	76.34 \pm 0.34	<0.01
Sex (women, %)	45.0	69.8	<0.01
BMI, kg/m ^{2†}	24.03 \pm 0.15	24.13 \pm 0.26	0.74
Diabetes, %	7.9	11.2	0.10
Hypertension (nondiuretic drugs; %)	67.7	70.4	0.34
Drinking status			
Current drinker, %	49.8	44.2	0.06
Ex-drinker, %	11.9	9.7	0.64
Nondrinker, %	38.4	46.1	0.12
Self-reported illness			
Renal, %	5.1	7.7	0.43
CHD, %	8.7	10.1	0.33
Use of tranquilizers, %	9.5	11.8	0.59
Use of hypnotics, %	7.1	4.1	0.16
Use of diuretics, %	5.1	4.7	0.48
History of delivery, % in women	89.5	92.5	0.45

BMI = body mass index; CHD = coronary heart disease.

* Analysis of covariance (age and BMI) or multiple logistic regression adjusted for age and sex where appropriate.

† Adjusted least squares mean \pm standard error.

of the PA levels (low: 1; median: 2; high: 3). A significant difference was defined as $p < 0.05$. SAS software (version 9.1) was used for analyses.

3. Results

In this study, the overall prevalence of UI was 30.5% (289 of 948). Among 676 subjects who were available to be analyzed, 169 (25.0%) had self-reported UI, including 118 women (34.2%) women and 51 men (15.5%). Among them, 61 (36.1%; 56 women and 5 men) had stress UI, 62 (36.7%; 33 women and 29 men) had urge incontinence, 31 (18.3%; 25 women and 6 men) had mixed UI, and 15 (8.9%; 4 women and 11 men) had other UI.

3.1. Age- and sex-adjusted baseline characteristics according to continence status

The mean age was higher in the UI groups than in the non-UI groups ($p < 0.01$) (Table 1). The proportion of women was significantly greater in the UI groups ($p < 0.01$). Although not statistically significant, the proportions of diabetics appeared to be

higher among the UI groups ($p = 0.10$). Otherwise, no significant difference was observed between the non-UI groups and the UI groups.

3.2. Relationships between PA levels and UI prevalence

In a crude model, the ORs of occurrence of UI across the PA levels low, middle, and high were 1.00 (reference), 0.63 (95%CI, 0.42–0.94), and 0.43 (95%CI, 0.26–0.68), respectively (Table 2). These results were unchanged when we adjusted for age, sex, or multiple confounding factors. Although not statistically significant, the positive relation of BMI (OR = 1.003; 95%CI, 0.95–1.06) and diabetes (OR = 1.60; 95%CI, 0.84–2.94) with UI were observed in multiple model. Furthermore, although p for trend was statistically significant only in women, similar relations were also observed in men (p for interaction = 0.66).

3.3. Relationships between PA levels and log-transformed ICIQ score

In this study population, the median (interquartile range) ICIQ score was 0 (0–3) in low PA level, 0 (0–3) in

Table 2 – Adjusted relationships of PA levels to the occurrence of urinary incontinence

	PA levels			p for trend
	Low level	Middle level	High level	
All, n = 676				
No. of participants	276	232	168	—
No. of urinary incontinence*	88	53	28	—
Odds ratio (95% CI)				
Crude	1.00	0.63 (0.42–0.94)	0.43 (0.26–0.68)	<0.01
Age- and sex-adjusted	1.00	0.71 (0.47–1.07)	0.56 (0.33–0.91)	0.01
Multiple adjusted†	1.00	0.71 (0.47–1.09)	0.58 (0.35–0.96)	0.02
Women, n = 346				
No. of participants	168	108	70	—
No. of urinary incontinence*	64	41	13	—
Odds ratio (95% CI)				
Crude	1.00	0.99 (0.60–1.63)	0.37 (0.18–0.71)	0.01
Age-adjusted	1.00	0.99 (0.60–1.63)	0.43 (0.21–0.84)	0.04
Multiple adjusted†	1.00	0.98 (0.58–1.64)	0.44 (0.21–0.87)	0.046
Men, n = 330				
No. of participants	108	124	98	—
No. of urinary incontinence*	24	12	15	—
Odds ratio (95% CI)				
Crude	1.00	0.38 (0.17–0.78)	0.63 (0.31–1.28)	0.15
Age-adjusted	1.00	0.38 (0.17–0.79)	0.66 (0.31–1.34)	0.18
Multiple adjusted‡	1.00	0.38 (0.18–0.80)	0.66 (0.31–1.35)	0.18

PA, physical activity; CI = confidence interval.

* Had a self-reported urinary incontinence including stress urinary incontinence, urge incontinence, mixed urinary incontinence, and other urinary incontinence.

† Adjusted for potential confounding factors (see Methods section).

‡ Adjusted for potential confounding factors (see Methods section).

§ Adjusted for potential confounding factors (see Methods section).

Table 3 – Adjusted PA level in relation to log-transformed ICIQ score among the subjects

	PA levels			p for trend
	Low level	Middle level	High level	
All, n = 676				
No. of participants	276	232	168	
Crude log ICIQ score [†]	0.50 ± 0.04	0.37 ± 0.05	0.27 ± 0.06	<0.01
Age-adjusted log ICIQ score [‡]	0.47 ± 0.04	0.38 ± 0.05	0.32 ± 0.06	0.03
Multiple adjusted log ICIQ score ^{‡,§}	0.46 ± 0.04	0.38 ± 0.05	0.33 ± 0.06	0.06
Women, n = 346				
No. of participants	168	108	70	
Crude log ICIQ score [†]	0.60 ± 0.06	0.60 ± 0.07	0.28 ± 0.09	<0.01
Age-adjusted log ICIQ score [‡]	0.59 ± 0.06	0.59 ± 0.08	0.34 ± 0.10	0.02
Multiple adjusted log ICIQ score ^{‡,§}	0.59 ± 0.06	0.59 ± 0.08	0.34 ± 0.10	0.02
Men, n = 330				
No. of participants	108	124	98	
Crude log ICIQ score [†]	0.35 ± 0.06	0.17 ± 0.05	0.27 ± 0.06	0.35
Age-adjusted log ICIQ score ^{‡,*}	0.35 ± 0.06	0.17 ± 0.05	0.27 ± 0.06	0.40
Multiple adjusted log ICIQ score ^{‡,¶}	0.34 ± 0.06	0.18 ± 0.06	0.27 ± 0.06	0.40

PA = physical activity; ICIQ = International Consultation Incontinence Questionnaire.

* Before log transformation, 1.0 was added to the ICIQ score.

† Variables are presented as least squares means ± standard error.

‡ Adjusted for potential confounding factors (see Methods section).

§ Adjusted for potential confounding factors (see Methods section).

¶ Adjusted for potential confounding factors (see Methods section).

middle PA level, and 0 (0–0) in high PA level. In all subjects, in a crude, age-adjusted model, increasing PA levels showed a significantly inverse relationship with mean log ICIQ score (p for trend <0.01 and 0.03, respectively; Table 3). In a multiple model, although not statistically significant, increasing PA levels were inversely related to mean log ICIQ score (p for trend = 0.06). In the multivariate model, although increasing PA levels showed a significantly inverse relationship with mean log ICIQ score in women only (p for trend 0.02 in women and 0.40 in men), no significant interaction between sex and PA for UI was observed.

4. Discussion

In this cross-sectional study, we examined the relationship between PA and UI among community-dwelling elderly population aged ≥ 70 yr, among whom this condition is highly prevalent. These results suggested that high PA was independently related to a lower self-reported prevalence of UI and lower degree of UI in community-dwelling elderly population aged ≥ 70 yr.

Although regular PA was confirmed to be effective in the primary and secondary prevention of factors in UI such as diabetes, hypertension, obesity, and pelvic floor muscle dysfunctions [7,9], it was also considered that high-impact PA may be a potential risk factor associated with UI

because high-impact PA (eg, jogging, tennis) also increases abdominal pressure, which is an important factor associated with the occurrence of stress incontinence [16,23]. In this study, the highest PA level including sports was not related to a higher prevalence of UI. Moreover, we also assessed the relationship between ICIQ score, which is an indicator of the degree of UI. The results did not indicate that higher PA levels increased the degree of UI. However, because information about high-impact PA such as jogging and tennis was not derived specifically from our questionnaire [20], we cannot establish that any specific high-impact PA had an effect on UI.

In this study, we have hypothesized that PA may have a potentially beneficial effect on the prevention of UI. Although several studies [6,11,12,14–16] investigated the relationship between PA and UI, only one study [13] used a community-based population including older (73–79 yr) women. However, they did not adjust for any confounding factors when they analyzed the relationship between PA and UI. Incidentally, these studies have not suggested any beneficial effect of PA on UI in contrast to our hypothesis and our results. Although the reason for this discrepancy is unclear, because high-impact PA is a risk factor for UI [16,23] and age was associated with a decline in jump peak performance according to peak leg muscle power [24], the difference in the age of the subjects may partly explain this discrepancy.

UI prevalence rises in elderly adults regardless of sex [25]. In this study, women had a higher prevalence of UI (34.2%) compared with men (15.5%) as seen in other studies; for example, the prevalence of UI in community-dwelling elderly ≥ 60 yr of age in Europe is 19.3% in women and 10.4% in men [26]. Although interaction between sex and PA for UI was not significant, *p* for trend of the relation between PA and UI was statistically significant only in women. Prostate diseases may explain the weaker association observed in men [27]. However, since we did not have information on prostate diseases, we could not answer the issue. Further study is required to clarify the discrepancy.

UI is a major elderly health problem. Gasquet et al [3] indicated that stress UI is frequent in French women, causing embarrassment and negatively affecting their QOL. A recent study also indicated that UI severity was the single most important predictor of QOL and bother in a large group of European women, regardless of type of UI [28]. In this study, we found that PA was independently related to IGIQ score, which is an indicator of the QOL. This result suggested that PA may have a potentially beneficial effect on the QOL associated with UI.

This study had several limitations. First, because all assessment was carried out in a public facility, participants were sufficiently active and healthy. In addition, we have excluded the subjects who had a potential for PA restriction. Moreover, because depressive symptoms might influence the frequency and degree of PA [29] and UI can be a reason for depression, we also excluded participants with depression. Therefore, our results may not represent an elderly general population. However, we believe that these exclusions were necessary to investigate the relation of PA with UI. Second, because this study was a cross-sectional study, we could not conclude that PA decreases the occurrence of UI or that UI leads to impairment of PA among subjects aged ≥ 70 yr. Therefore, a prospective study or trial should be undertaken to confirm the relationship between PA and UI. Third, because the question of PA is focused on some activities such as walking, brisk walking, and sports, we could not infer the influence of other sorts of exercise on UI. Fourth, although we adjusted for several confounders related to UI, we could not adjust some factors associated with UI, such as genitourinary surgery or prostate disease status because of lack of information. Finally, a self-reported single-item question and corresponding response sets were used to estimate different levels (not a quantitative) of PA. Moreover, we did not directly measure the exercise intensities of walking, brisk walking, and sports by our subjects. Still, one

may easily discriminate one's own "brisk walking" from ordinary walking. We therefore believe that the categorization of relative walking intensity based on the subjects' own perceptions was reliable. It is well known that ratings of perceived exertion correspond well to exercise intensity as measured by oxygen uptake [30].

5. Conclusions

A high PA level was independently related to a lower self-reported prevalence of UI in community-dwelling elderly population aged ≥ 70 yr. Although this cross-sectional study cannot demonstrate temporal relationship between PA and the onset of UI, the findings suggest that PA may have a potentially beneficial effect on the prevention of UI. A prospective study or randomized trials are required to clarify the causality.

Conflicts of interest

The authors have no conflicts of interest to disclose.

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Editorial Comment on: Association between Physical Activity and Urinary Incontinence in a Community-Based Elderly Population Aged 70 Years and Over

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Because urinary incontinence among the elderly consumes a considerable part of health care costs and often leads to personal suffering and lowered quality of life, it is essential to use preventive solutions that are cost effective but also have other positive effects, such as increased physical functioning and improved quality of life. Interventions that may prolong time without health care or time

to be able to live independently on one's own are extremely important from a personal viewpoint but also from the perspective of the whole society. It is time to test interventions in a wide health economic overview, in multicentre studies focusing on different groups of elderly, to prove that staff-intensive prophylactic physical training is a good investment in the care of older people.

Urinary incontinence among the elderly is complex; it has several causative factors and a potential impact on daily life for the individual and also for relatives. Additionally, urinary incontinence is related to nursing home admittance or increased need for health care and help in performing daily activities. Older persons performing regular physical activities reported lower prevalence of urinary incontinence episodes [1]. Several other studies have pointed out similar findings. For instance, impaired mobility, such as difficulties in walking or moving the arms and hands, as well as oedema in the legs were factors strongly related to urinary incontinence and faecal incontinence in older women and men [2]. Furthermore, studies have shown the positive effects of physical training on the frequency of leakage among nursing home populations [3,4] but not on cost reduction [5]. Improving mobility function has many other effects apart from urinary incontinence, such as reducing the incidence of falls and improving balance, coordination, cardiorespiratory fitness, and quality

of life [6]. These positive outcomes have to be considered as well.

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世界各国の健康寿命

POINT

■日本人の健康寿命は世界で最も長い。 ■平均寿命の長い国は健康寿命も長い。 ■平均寿命の長い国は不健康期間が短い。

大森 芳
辻 一郎

はじめに

世界保健機関(WHO)では2000年より死亡状況と疾病や障害の保有状況に基づく複合的な健康指標である健康寿命(Healthy Life Expectancy)を算出している¹⁾。健康寿命の算出には様々な方法が提案されているが、WHOでは健康な状態での生存期間に加えて様々な疾病を抱えて生きる期間をその重症度に応じて換算したものを合計した指標を用いている^{1,2)}。これは、各国の性・年齢別の死亡率、健康状況や障害度で重みづけした有病率からSullivan法により算出したものである。2004年にWHOが当時の加盟国192カ国の2002年時点での健康寿命を推計したものが現在最新のデータであり³⁾、以下このデータを基に検討していくこととする。

世界各国の健康寿命

健康寿命の上位・下位10カ国の値を表1、2に示す。2002年の日本人の健康寿命は、男性72.3年、女性77.7年で、男女ともに第1位である。男女とも上位10カ国は日本、オーストラリア、イスラエルを除くとすべて西ヨーロッパの国々である。アメリカは男性67.2年(28位、平均寿命は74.6年で33位)、女性71.3年(29位、平均寿命は79.8年で29位)である。一方、健康寿命の最も短い国は、2002年まで10年にわたり内戦が続いていたアフリカのシエラレオネで、男性27.2年、女性29.9年であった。シエラレオネは平均寿命も192カ国中最も短か

った。日本は、健康寿命はシエラレオネの男性2.7倍・女性2.6倍、平均寿命は男女とも2.4倍に相当する。下位10カ国はアフガニスタンを除くと、サハラ以南のアフリカの国々である。

健康寿命の性差

同じ国では男女の健康寿命は正の関連があり、女性の健康寿命は男性より5%長い(図1)。これを上回る性差は、性特異的な健康問題が存在する可能性がある。健康寿命の女性/男性比は高い順に、ロシア(1.20)、エストニア(1.17)、ラトビア(1.16)である。これらは、いずれも旧ソビエト連邦の国々であり、1990年代に男性のアルコール中毒が蔓延しそれに関連して事故や暴力や心血管疾患が増加して、男性の死亡率が急増したことが一因であると指摘されている^{1,4,5)}。一方、女性/男性比が低い国は、カタール、モルディブ、バングラデシュ(いずれも0.96)の順であり、女性の健康寿命の方が男性よりも短く、特に女性の健康に問題があることが示唆される。

平均寿命と健康寿命

x軸を平均寿命、y軸を健康寿命として各国の値をプロットすると、直線関係になる(図2)。健康寿命の割合が低い国は、男性ではイエメン(平均寿命に占める健康寿命の割合が81.7%)、イラク(82.5%)、アンゴラ(83.4%)、女性ではイエメン(81.5%)、イラク(81.6%)、ソマリア

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表1 男性の健康寿命 上位10カ国と下位10カ国(2002年)

上位10カ国				下位10カ国			
順位	国名	健康寿命 (年)	平均寿命 (年)(順位)	順位	国名	健康寿命 (年)	平均寿命 (年)(順位)
1	日本	72.3	78.4(1)	192	シエラレオネ共和国	27.2	32.4(192)
2	アイスランド共和国	72.1	78.4(1)	191	レソト王国	29.6	32.9(191)
3	スウェーデン王国	71.9	78.0(3)	190	アンゴラ共和国	31.6	37.9(188)
4	スイス連邦	71.1	77.7(6)	189	スワジランド王国	33.2	36.9(190)
5	オーストラリア連邦	70.9	77.9(4)	188	ブルンジ共和国	33.4	38.7(187)
5	サンマリノ共和国	70.9	77.2(9)	187	リベリア共和国	33.6	40.1(184)
7	イタリア共和国	70.7	76.8(11)	186	ジンバブエ共和国	33.8	37.7(189)
7	モナコ公国	70.7	77.8(5)	185	ザンビア共和国	34.8	39.1(186)
9	イスラエル国	70.5	77.3(8)	184	ブルキナファソ	34.9	40.6(182)
10	ノルウェー王国	70.4	76.4(14)	183	マラウイ共和国	35.0	39.8(185)

(文献3より作成)

表2 女性の健康寿命 上位10カ国と下位10カ国(2002年)

上位10カ国				下位10カ国			
順位	国名	健康寿命 (年)	平均寿命 (年)(順位)	順位	国名	健康寿命 (年)	平均寿命 (年)(順位)
1	日本	77.7	85.3(1)	192	シエラレオネ共和国	29.9	35.7(192)
2	サンマリノ共和国	75.9	84.0(3)	191	レソト王国	33.2	38.2(190)
3	スペイン	75.3	83.0(7)	190	ジンバブエ共和国	33.3	38.0(191)
3	スイス連邦	75.3	83.3(6)	189	マラウイ共和国	34.8	40.6(186)
5	モナコ公国	75.2	84.5(2)	188	ザンビア共和国	35.0	40.2(189)
6	スウェーデン王国	74.8	82.6(9)	187	アンゴラ共和国	35.1	42.0(185)
7	フランス共和国	74.7	83.6(5)	185	スワジランド王国	35.2	40.4(188)
7	イタリア共和国	74.7	82.5(10)	185	ニジェール共和国	35.2	42.7(183)
9	アンドラ公国	74.6	83.7(4)	184	ボツワナ共和国	35.4	40.6(186)
10	オーストラリア連邦	74.3	83.0(7)	183	アフガニスタン・イスラム国	35.8	43.4(181)

(文献3より作成)

とアフガニスタン(82.3%)である。なお、健康寿命の推定はイラク戦争の前である。健康寿命の割合が高い国は、男性ではノルウェー、日本、ドイツ(いずれも92.2%)、女性では日本(91.1%)、ドイツとスペイン(90.7%)の順であった。

平均寿命と不健康期間

平均寿命と健康寿命の差は不健康な状態での生存期間(不健康期間: expectation of lost

healthy years)となる^{5,6)}。x軸を平均寿命、y軸を不健康期間としたグラフを図3に示す。日本の不健康期間は男性6.1年、女性7.5年(いずれも短い方から30位)であった。不健康期間が長い国は男性ではイエメン(10.8年)、イラン(10.4年)、イラク(10.3年)、女性ではイラン(12.5年)、セーシェル(12.3年)、モロッコ(11.9年)の順である。これらの国々の平均寿命は、男性は58~67年、女性では71~78年であり、不健康期間の長さは平均寿命と必ずしも比例し

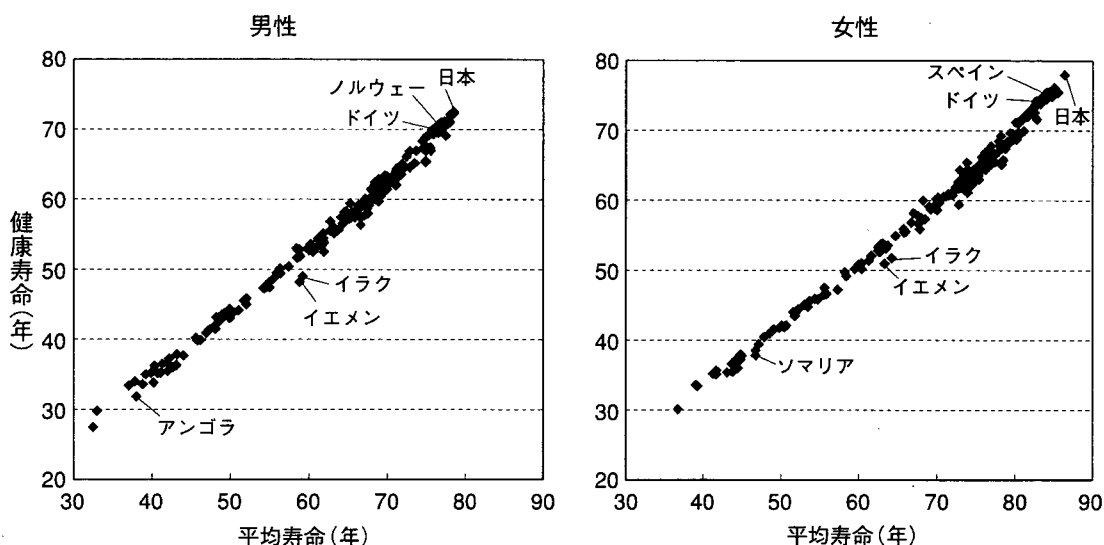


図2 平均寿命と健康寿命(文献3より作成)

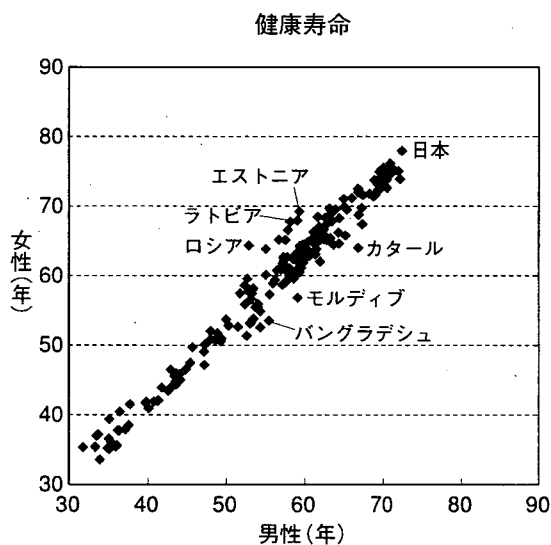


図1 健康寿命の男女比較(文献3より作成)

ない。グラフからは平均寿命が70年くらいまでは平均寿命とともに不健康期間は緩やかに増加する傾向にあるが、それ以上では反対に平均寿命が長いほど不健康期間は短くなるという傾向が読み取れる⁵⁾。ある程度以上の健康水準を達成した国々では、不健康期間の短縮を伴う健康寿命の延伸(compression of morbidity)^{6,7)}が実現しつつあるのかもしれない。

おわりに

日本は平均寿命・健康寿命ともにWHO加盟国中最も長く、不健康期間の年数は短く、平均寿命に占める健康寿命の割合が高く、日本人の健康状況は非常によい。2000年のWHOのプレスリリースでは、日本の健康寿命が長いことの理由として、伝統的な低脂肪食のために心疾患が少ないこと、過去の喫煙率が低かったために肺癌発生率が比較的低いことが関係していることを挙げている⁴⁾。一方で、同報告では、戦後に喫煙率が急上昇し赤身肉などの脂肪食の食事が増えたことなどが影響して、将来、特に男性の健康寿命は影響を受けるおそれがあると指摘している。世界最高水準にある日本の健康寿命をこれからも維持していくために、われわれ医療関係者の果たすべき責任は大きい。

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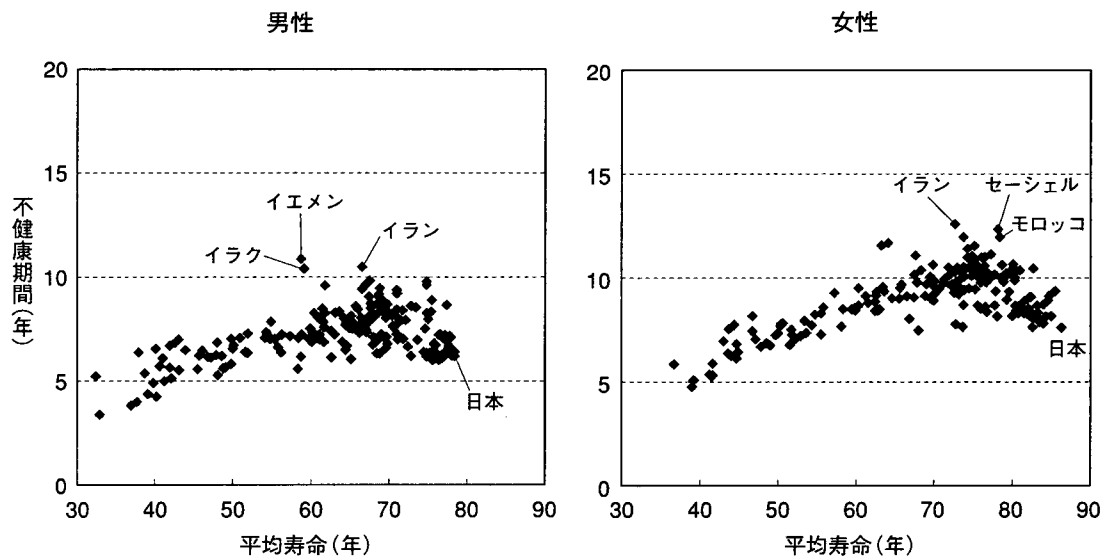


図3 平均寿命と不健康期間(文献3より作成)

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Effect of coaching on psychological adjustment in patients with spinocerebellar degeneration: a pilot study

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Objective: To examine effect of coaching intervention on psychological adjustment to illness and health-related QOL (HRQOL) in patients with spinocerebellar degeneration.

Design: Randomized controlled trial.

Subjects: Twelve independently living patients with spinocerebellar degeneration aged 20–65 years old, without cognitive impairment or psychiatric disorder received coaching intervention, which was postponed in another 12 (control).

Interventions: Three physician coaches telephoned assigned patients for 15–30 minutes in each of 10 weekly coaching sessions over three months.

Main outcome measures: Primary endpoints were HRQOL (SF-36) and psychological adjustment to illness (Nottingham Adjustment Scale, Japanese version; NAS-J).

Results: Two-way analysis of variance (group \times time) showed statistically significant main effects of time for vitality ($F=5.00$; $P=0.036$), anxiety/depression ($F=5.15$; $P=0.033$), and locus of control ($F=5.58$; $P=0.027$), indicating improvement of scores over time in both coaching and control groups. No main effect of group or interaction was seen. However analysis of covariance with baseline scores as the covariate showed the coaching group to have better self-efficacy scores than controls at follow-up (least-square mean, experimental group, 65.1; control group, 52.7; $P=0.037$).

Conclusion: Carefully structured telephone coaching can improve self-efficacy in patients with spinocerebellar degeneration.

Introduction

Neurodegenerative diseases such as spinocerebellar degeneration follow a chronic course characterized by disabilities requiring long-term

care in the absence of an established drug treatment. Rehabilitative intervention includes therapeutic exercises, walking aids, orthotics, or environmental modification to ameliorate various kinds of disability.

Patients with neurodegenerative diseases are burdened mentally as well as physically. Patients with spinocerebellar degeneration have shown affective disturbances when assessed with a depression scale,¹ which is not surprising considering that progressive impairment in carrying out

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