Dietary Reference Intakes for Japanese 2010: Fat

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Summary In the Dietary Reference Intakes (DRIs) for fat, adequate intake (AI) and tentative dietary goal for preventing lifestyle-related disease (DGs) were used. AIs were set for *n*-6 and *n*-3 polyunsaturated fatty acids, which are essential fatty acids because they are not produced by the human body and their deficiency leads to dermatitis. DGs have been set for total fat, saturated fat, *n*-6 fatty acids, *n*-3 fatty acids, and cholesterol, whose consumption levels affect risk of lifestyle-related disease, including obesity, diabetes mellitus, cardiovascular disease, and stroke. As AI for *n*-6 and *n*-3 polyunsaturated fatty acids, the 50th percentile of *n*-6 and *n*-3 fatty acid intake was set. In the Japanese population, 98% of dietary *n*-6 fatty acids come from linoleic acid; therefore the amount of *n*-6 fatty acid intake is considered to be that of linoleic acid. Both α -linolenic (60% of total *n*-3 fatty acids) acid and fish oils are considered essential fatty acids because it has been difficult to conclude that only α -linolenic acid is essential for humans. The prevention of diabetes mellitus and stroke was emphasized. For example, an increase in saturated fatty acids intake leads to increased incidences in obesity, diabetes, and myocardial infarction, whereas a decrease of saturated fatty acids intake is associated with increased incidence in brain hemorrhage. Therefore, DG of saturated fatty acids in those more than 18 y of age was set between 4.5 and 7% energy.

Key Words total fat, saturated fat, monounsaturated fat, *n*-6 fatty acids, *n*-3 fatty acids, cholesterol, trans fatty acids

Background Information

In the Dietary Reference Intakes for Japanese (DRIs-J) 2010 for fat, the adequate intakes (AIs) and tentative dietary goal for preventing lifestyle-related disease (DGs) for fat were determined. Specifically, AIs were set for *n*-6 and *n*-3 polyunsaturated fatty acids, which are essential fatty acids because they are not produced by the human body and their deficiency leads to disease. DGs have been set for total fat, saturated fat, *n*-6 fatty acids, *n*-3 fatty acids, and cholesterol, whose consumption levels affect risk of lifestyle-related disease, including obesity, diabetes mellitus, cardiovascular disease, and stroke.

Total fatty acids, saturated fat, and *n*-6 fatty acids are major fuels that supply energy to humans. Therefore, they are expressed as percentage of energy (%en) from total energy intake. Essential fatty acids, including metabolites of α -linolenic acid are expressed as absolute values (g/d) but not relative values (en% of total energy) due to their essentiality.

To estimate the average amount of fatty acid intake in the Japanese which was used for DRIs, it was calculated using the original data that had been collected by the 2005 and 2006 NHNS. The 50th percentiles of the

major fatty acids and cholesterol are presented in the original Japanese DRIs. For the determination of DGs in the DRIs-J 2010, systematic reviews were conducted by using appropriate key words in PubMed. From these publications, 437 related to DRIs were selected for careful reading and, along with those that had been used for the DRIs-J 2005, were used for a review of the DRIs-J 2010.

In this paper, the original version of the Japanese DRIs has been summarized and only selected sections discussed for the sake of brevity.

Determining DRIs

1. Total fat

1-1. DG (lower boundary). A low fat/high carbohydrate diet leads to increased postprandial glucose and fasting triacylglycerol (TG) concentrations and decreased fasting high-density lipoprotein (HDL)-cholesterol concentration (1). Although there is no definite evidence that average daily fat intake in a low fat/high carbohydrate diet increases risk of obesity and diabetes mellitus, unfavorable metabolite profiles in low fat/high carbohydrate diets indicate that a lower boundary of adequate total fat intake exists.

As described in the following sections, the AI of *n*-6

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Table 1. Dietary Reference Intakes for total fat [Ratio of total fat to total energy (percentage of fat energy): % energy].

Sex	Males		Females	
Age	AI	DG (range)	AI	DG (range)
0–5 mo	50	—	50	—
6–11 mo	40	—	40	—
1–2 y	—	20≤, <30	—	20≤, <30
3–5 y	—	20≤, <30	—	20≤, <30
6–7 y	—	20≤, <30	—	20≤, <30
8–9 y	—	20≤, <30	—	20≤, <30
10–11 y	—	20≤, <30	—	20≤, <30
12–14 y	—	20≤, <30	—	20≤, <30
15–17 y	—	20≤, <30	—	20≤, <30
18–29 y	—	20≤, <30	—	20≤, <30
30–49 y	—	20≤, <25	—	20≤, <25
50–69 y	—	20≤, <25	—	20≤, <25
≥70 y	—	20≤, <25	—	20≤, <25
Pregnant women			—	—
Lactating women			—	—

AI, adequate intake; DG, tentative dietary goal for preventing lifestyle-related diseases.

fatty acids was set at approximately 5 en%, the AI (or DG) of *n*-3 fatty acids at approximately 1 en%, and the lower DG (lower boundary) of saturated fat at approximately 5 en%. The 50th percentile value for monounsaturated fat was found to be approximately 6 en% and the total fatty acid level was 17 en% (=5+1+5+6). Considering the glycerol portion of TG (approximately 10% of total fat), approximately 20 en% was set as the lower boundary for total fat (Table 1).

1-2. DG (upper boundary). The prevention of obesity, which leads to diabetes and other diseases, is a major concern for public health. There might be an optimal dietary fat to carbohydrate ratio for prevention and treatment of obesity. In a meta-analysis of general populations under free-living conditions, a reduction in the percentage of energy as fat was found to be positively and independently associated with weight loss (2). Another meta-analysis of intervention studies provided support for this conclusion (3). However, obese subjects with hyperinsulinemia (or insulin resistance) lost more weight on a moderately low-carbohydrate (or low-glycemic load) diet consisting of 40 en% carbohydrates and 30 to 35 en% fat than on a low-fat diet consisting of 55 to 60 en% carbohydrate and 20% fat, whereas those without hyperinsulinemia lost more weight on the low-fat diet than the moderately low-carbohydrate diet (4–6). The optimal dietary fat to carbohydrate ratio may differ in populations depending on the prevalence of obesity.

Considering the lower prevalence of obesity in the Japanese population, the upper boundary of total fat was set as the 50th percentile of fat en% of Japanese nationwide survey, which is 30 en% for individuals aged

Table 2. Dietary Reference Intakes for saturated fatty acids (% energy).

Sex	Males	Females
Age	AI (range)	AI (range)
0–5 mo	—	—
6–11 mo	—	—
1–2 y	—	—
3–5 y	—	—
6–7 y	—	—
8–9 y	—	—
10–11 y	—	—
12–14 y	—	—
15–17 y	—	—
18–29 y	4.5≤, <7.0	4.5≤, <7.0
30–49 y	4.5≤, <7.0	4.5≤, <7.0
50–69 y	4.5≤, <7.0	4.5≤, <7.0
≥70 y	4.5≤, <7.0	4.5≤, <7.0
Pregnant women		—
Lactating women		—

AI, adequate intake.

1 to 29 y and 25 en% for individuals aged 30 y and over (Table 1).

2. Saturated fat

2-1. DG (lower boundary). In 3 Japanese cohort studies, subjects who ate less saturated fat showed an increased risk of hemorrhagic stroke (7–9). First, in the Ni-Hon-San Study, which followed males aged 45 to 69 y ($n=1,366$) in Hiroshima and Nagasaki for 4 y (1972 to 1976), subjects who ate less than 5 g/d of saturated fat showed an increased incidence of intracranial hemorrhage (9). Second, in the Honolulu Heart Program, a 10-y cohort study of male Hawaiians of Japanese descent that examined the relationship between dietary fat and cholesterol and mortality, subjects who ate less than 10 g/d of saturated fat showed a 2-fold increase in the incidence of stroke (bleeding and infarction were not identified separately) than subjects who ate more than 10 g/d of saturated fat (8). Third, in a 14-y prospective study (1983 to 1997) of 4,775 Japanese aged 40 to 69 y who participated in a single 24-h dietary recall survey, a low intake of saturated fat (approximately <10 g/d) was found to be associated with increased risk of intraparenchymal hemorrhage after adjusting for known cardiovascular risk factors (7). No study found an association between saturated fat intake and risk of brain infarction (10).

To determine the lower DG boundary for saturated fat, the results of 2 studies were examined. In a cohort study in Hawaii, subjects who ate less than 10 g/d (=3.9 en%) of saturated fat showed an increase in total mortality and mortality due to cancer, coronary heart disease, and stroke relative to subjects who ate more than 10 g/d of saturated fat (8). In a cohort study of Japanese subjects, the multivariate relative risk was found to be 3.37 for the lowest quartile (5.0 g/d), 2.60 for the second

lowest quartile (8.5 g/d), and 2.21 for the third lowest quartile (11.9 g/d=5.3 en%) compared to the highest quartile (18.3 g/d) (7). As these findings indicate that individuals who eat less than 4.6 en% ($= (3.9 + 5.3)/2$) saturated fat may have an increased risk of death and lifestyle-related diseases, the rounded value of 4.5 en% was set as the lower boundary of the DG for saturated fat for adults aged 18 y and over (Table 2). Because the amount of animal protein was not adjusted for further examination in these 2 studies, it is possible that the increase in hemorrhagic stroke observed had been due to a shortage of animal protein rather than a shortage of saturated fat. Therefore, to prevent hemorrhagic stroke, consumption of saturated fat from dairy products and animal meat is recommended.

2-2. DG (upper boundary). An increased intake of saturated fat has been hypothesized to elevate low-density lipoprotein (LDL)-cholesterol concentration and, ultimately, promote the development of atherosclerosis. However, cohort studies in the United States have not supported this hypothesis. In the Nurses' Health Study, the significantly positive association that had been found between saturated fat intake and mortality due to coronary heart disease (CHD) disappeared after adjusting for confounding factors (11). In a cohort of US males, the positive association that had been found between intake of saturated fat and incidence of myocardial infarction disappeared after adjusting for dietary fiber intake (12). However, age may affect these associations. Two studies found a positive association between intake of saturated fat and incidence of CHD for adults aged 60 y and over but not for adults aged under 60 y (13, 14). In contrast, several intervention studies demonstrated that reduction of saturated fat intake led to reduced incidence of ischemic heart disease, degree of atherosclerosis, and LDL-cholesterol concentration (15–17). In a meta-analysis to examine the effects of dietary changes on blood lipid profile, intake of less than 10 en% (National Cholesterol Education Program Step I diet) or less than 7 en% (National Cholesterol Education Program Step II diet) of saturated fat resulted in significant reductions in blood LDL-cholesterol concentrations over a period of 1 mo to 2 y (3).

Several cross-sectional studies showed a positive association between intake of saturated fat and prevalence of obesity (18). Observational studies have reported a positive association between saturated fat intake and the prevalence of diabetes, but these positive associations disappeared after adjusting for body mass index (BMI) (19–21). However, cross-sectional studies have reported a positive association between saturated fat intake and prevalence of insulin resistance (a cause of Type 2 diabetes) even after adjusting for BMI (22–24). Furthermore, intervention studies have observed a positive association between dietary saturated fat intake and insulin resistance (25, 26). These results indicate that increased intake of saturated fat may increase body weight and insulin resistance (independent of obesity) and eventually lead to the development of diabetes mellitus.

In summary, saturated fat intake has been associated

with increased incidence of myocardial infarction, obesity, and diabetes mellitus in a dose-dependent manner. Thus, although it is not clear that increased intake of saturated fat is a cause of these diseases due to a lack of large scale intervention study, research suggests that a diet high in saturated fat may promote these diseases. A meta-analysis of intervention studies in the United States and Europe indicates that a diet of 10 en% or less saturated fat decreases LDL-cholesterol concentration by 12% while a diet of 7 en% or less saturated fat decreases in LDL-cholesterol concentration by 16% (3). These data indicate that lower intake of saturated fat leads to lower incidence of myocardial infarction, obesity, and diabetes mellitus.

In the Japanese population, the 50th percentile value of dietary saturated fat, which is approximately 7 en%, was set as the upper boundary of the saturated fat DG for adults (Table 2). In younger individuals, the associations between saturated fat and lifestyle-related diseases are unclear, but it has been reported that subjects whose total blood cholesterol concentrations were high at age 22 y experienced high incidence of cardiovascular disease 27 to 42 y later (27). Therefore, 7 en% was also set as the upper boundary for saturated fat intake for subjects aged 18 to 19 y.

3. Monounsaturated fat

3-1. DG (lower and upper boundaries). In intervention studies conducted over relatively short periods, metabolic markers (LDL-cholesterol or insulin resistance) in subjects fed a high-monounsaturated fat diet were found to be better than those fed a high-saturated fat diet or a high-carbohydrate diet. However, in diabetic subjects, a high-monounsaturated fat diet (25 en%) resulted in a greater increase in body weight than a high-carbohydrate diet (28). The results of long-term cohort studies are mixed, with some finding a negative association (29), others no association (11), and yet others a positive association (13, 14, 30, 31) between monounsaturated fat intake and incidence of CHD.

Increasing dietary monounsaturated fat may lead to obesity and atherosclerosis when total energy intake is not restricted. However, when total fat intake is below 25 to 30 en% and the lower boundary of saturated fat, *n*-6, and *n*-3 fatty acids is maintained, intake of monounsaturated fat will be below 15 to 20 en% and overconsumption of monounsaturated fat will be avoided. Therefore, lower and upper boundaries of monounsaturated fat were not set.

4. *n*-6 fatty acids

4-1. AI. As the human body is unable to synthesize *n*-6 fatty acids, they are classified as essential fatty acids, thus requiring that an AI be set for these lipids. However, there are no data available to elucidate the appropriate AI value in healthy Japanese. In the Japanese population, 98% of dietary *n*-6 fatty acids come from linoleic acid. Patients deficient in *n*-6 fatty acids develop dermatitis, which can be improved by supplementation of 7.4 to 8.0 g/d or 2 en% of linoleic acid. Considering that most Japanese do not suffer from diseases due to *n*-6 fatty acid deficiency, the 50th percentile

Table 3. Dietary Reference Intakes for *n*-6 fatty acids.

Sex	Males		Females	
Age	AI (g/d)	DG (% energy)	AI (g/d)	DG (% energy)
0–5 mo	4	—	4	—
6–11 mo	5	—	5	—
1–2 y	5	—	5	—
3–5 y	7	—	6	—
6–7 y	8	—	7	—
8–9 y	9	—	8	—
10–11 y	10	—	9	—
12–14 y	11	—	10	—
15–17 y	13	—	11	—
18–29 y	11	<10	9	<10
30–49 y	10	<10	9	<10
50–69 y	10	<10	8	<10
≥70 y	8	<10	7	<10
Pregnant women (amount to be added)			+1	—
Lactating women (amount to be added)			+0	—

AI, adequate intake; DG, tentative dietary goal for preventing lifestyle-related diseases.

of *n*-6 fatty acid intake was set as the AI for *n*-6 fatty acids (Table 3).

4-2. DG (lower boundary). As there is no strong evidence that low intake of *n*-6 fatty acids increases risk of disease, a DG (lower boundary) was not set.

4-3. DG (upper boundary). Despite some concern that excessive intake of *n*-6 fatty acids may lead to increased incidence of cancer (32), recent meta-analyses do not support this concern (33, 34). Because delta-6 desaturase competitively acts on both linoleic acid and α -linolenic acid, increased intake of linoleic acid may decrease production of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), the metabolites of α -linolenic acid. However, adequate intake of EPA and DHA could counteract this unfavorable effect.

The effects of high intake of *n*-6 fatty acids (more than 10 en%) on mortality and mobility have not been studied in detail. Because linoleic acid produces inflammatory fat, such as prostaglandin and leukotriene (35), high intake of *n*-6 fatty acids could be a risk to health. Indeed, a recent Japanese cross-sectional study of school children found that the odds ratio of the prevalence of wheezing for the highest quintile of intake (14.5 g/d) was 1.2 (95% CI, 1.06 to 1.37) relative to the lowest quintile (5.7 g/d) (36).

Although there is no definite evidence that high intake of *n*-6 fatty acids is a risk factor, an upper boundary was set at 10 en% for adults in recognition of the possible association between high intake and chronic inflammation (Table 3).

5. *n*-3 fatty acids

5-1. Background information. Dietary *n*-3 fatty

Table 4. Dietary Reference Intakes for *n*-3 fatty acids (g/d).

Sex	Males		Females	
Age	AI	DG	AI	DG
0–5 mo	0.9	—	0.9	—
6–11 mo	0.9	—	0.9	—
1–2 y	0.9	—	0.9	—
3–5 y	1.2	—	1.2	—
6–7 y	1.6	—	1.3	—
8–9 y	1.7	—	1.5	—
10–11 y	1.8	—	1.7	—
12–14 y	2.1	—	2.1	—
15–17 y	2.5	—	2.1	—
18–29 y	—	2.1≤	—	1.8≤
30–49 y	—	2.2≤	—	1.8≤
50–69 y	—	2.4≤	—	2.1≤
≥70 y	—	2.2≤	—	1.8≤
Pregnant women			1.9	—
Lactating women			1.7	—

AI, adequate intake; DG, tentative dietary goal for preventing lifestyle-related diseases.

Note: In the DG, it is advised to have more than 1 g/d of EPA+DHA.

acids are primarily found in 2 sources: vegetable oil, which contains α -linolenic acid, and fish oil, which contains EPA, DHA, and docosahexaenoic acid (DPA). A portion of α -linolenic acid is metabolized to EPA and DHA in humans and 59% of total *n*-3 fatty acid in diet is in the form of α -linolenic acid, as well as that DHA intake is 1.8-fold larger than EPA intake and that DPA intake is only 30% of EPA intake. Moreover, according to a Japanese nationwide survey, there are marked differences between the 50th percentile median and mean values of EPA, DPA, and DHA intake, with the former approximately half the latter (data not shown). Therefore, it is uncertain whether the 50th percentile values of fish oil intake are a good index of the average amount of fish oil intake by a population.

Because the beneficial physiological effects of *n*-3 fatty acids might be due to the direct effects of *n*-3 fatty acids rather than their metabolic competition with *n*-6 fatty acids, the ratio of *n*-3/*n*-6 fatty acids was not used to set the DRIs for *n*-3 fatty acids. Epidemiologic observations support this notion. In the Nurses' Health Study, the inverse association that had been found between α -linolenic acid and risk of coronary artery disease (CAD) was not affected by linoleic acid intake (37). In the Health Professional Study, the inverse association that had been found between α -linolenic acid or EPA and DHA intake and risk of coronary artery disease was not confounded by linoleic acid intake (38).

5-2. AI. Since *n*-3 fatty acids are essential fatty acids, an AI for *n*-3 fatty acid intake should be set. Because administering both α -linolenic acid and fish oil to patients deficient in *n*-3 fatty acids has been found

to result in improvement of dermatitis and increase in body weight (39), it has been difficult to conclude that only α -linolenic acid is essential for humans. Therefore, all *n*-3 fatty acids, including both α -linolenic acid and fish oils, are considered essential fatty acids. Although there are no data with which to elucidate the appropriate AI value for healthy Japanese, the 50th percentile of *n*-3 fatty acid intake was set as the AI in consideration of the fact that most Japanese do not suffer from diseases due to *n*-3 fatty acid deficiency (Table 4).

5-3. DG (lower boundary) of α -linolenic acid. Intervention studies in France and India identified 1.8 g/d as the intake of α -linolenic acid that reduces the mortality of patients with CHD (40, 41). The Iowa Women's Health Study, a prospective cohort study of postmenopausal women, found an inverse association between intake of α -linolenic acid and total mortality (42). Several cohort studies have shown an inverse association between intake of α -linolenic acid and incidence of CHD in the United States (12, 37, 43). Recognizing that these favorable effects may apply to the Japanese population, intake of α -linolenic acid for adults aged 18 y and over is advised to be equal to or higher than the current 50th percentile values of the Japanese population (in men, 50th percentile values of α -linolenic acid are 1.49 (in 18–29 y old), 1.42 (30–49 y old), 1.32 (50–69 y old) and 1.06 g/d (70 y old and over), respectively, and in women, 1.24 (in 18–29 y old), 1.19 (30–49 y old), 1.14 (50–69 y old) and 0.96 g/d (70 y old and over), respectively).

5-4. DG (upper boundary) of α -linolenic acid. A long-term intervention study in Japanese elderly subjects showed that an increase of 3.0 g/d of α -linolenic acid (total intake of α -linolenic acid of 4.8 g/d) had no adverse effects on lipid profiles or major metabolites in blood (44). Although the DG (upper boundary) of α -linolenic acid was not set, large habitual intake of α -linolenic acid in males should be avoided due to concern that it may increase the incidence of prostate cancer (45).

5-5. DG (lower boundary) of EPA and DHA. Many studies have found a positive association between intake of *n*-3 fatty acids and reduced risk of CAD (46). A recent review that examined the association between the intake of EPA and DHA and mortality due to CAD identified a threshold of EPA and DHA intake—0.5 g/d—above which no further reduction in CAD mortality resulted (47). Likewise, clinical studies have identified a threshold of 0.75 g/d for reducing blood pressure and risk of arrhythmia (47). However, no threshold regarding intake and nonfatal coronary events has been identified in Japanese subjects. In a Japanese cohort study (the JPHC Study), the multivariable hazard ratio of nonfatal coronary events of the highest quintile (EPA and DHA intake of 2.1 g/d) was found to be 67% lower than that of the lowest quintile (EPA and DHA intake of 0.3 g/d) (48), while the hazard ratio of the middle quintile (EPA and DHA intake of 0.9 g/d) was found to decrease significantly (39%). In the Japan Eicosapentaenoic Acid Lipid Intervention Study (the JELIS), in which 18,645 patients with a total cholesterol of 250 mg/dL or greater

were randomly assigned to receive 1.8 g/d EPA with statins or statins only, a 19% relative reduction in major coronary events was observed in the EPA with statins group over a 5-y follow-up period (49). However, this reduction was only observed regarding unstable angina, not coronary death.

The findings of other studies indicate that EPA and DHA intake may reduce the incidence of heart failure. In a Japanese cohort study (the JACC Study), the hazard ratio for the highest quintile (EPA, DHA, and DPA intake of 2.11 to 5.06 g/d) was found to be 0.58 (95% CI, 0.36 to 0.93) relative to the lowest quintile (EPA, DHA, and DPA intake of 0.05 to 1.18 g/d) (50). In an intervention study in Italy, supplementation of 1 g/d of EPA and DHA significantly reduced risk of death and rate of hospital re-admission for heart failure patients (51), while several US studies have found an inverse association between fish intake and the incidence of brain infarction (52–54). The JELIS found that supplementation of 1.8 g/d of EPA decreased the relative risk of stroke recurrence by 20% (55). Other studies have found an inverse association between EPA and DHA intake and incidence of age-related macular degeneration (56–58), as well as that high EPA+DHA intake has favorable effects on allergic rhinitis (59), peak bone mineral density (60), and aged-induced cognitive decline (61, 62).

These findings indicate that high EPA and DHA intake could reduce the incidence of CAD, stroke, and age-related macular degeneration. One study found that Japanese subjects whose average intake of EPA and DHA was 0.9 g/d showed a significant reduction in hazard ratio (0.61; 95% CI, 0.38 to 0.98) for nonfatal cardiac events compared subjects whose intake was 0.3 g/d (48). Rounding this value (0.9 g/d), the DG for the lower boundary of EPA and DHA was set at 1 g/d, which is equivalent to approximately 90 g/d of fish (Table 4).

5-6. DG (upper boundary) of EPA and DHA. The possible adverse effects of EPA and DHA intake on bleeding time, LDL-cholesterol concentration, blood glucose level, immune functions, lipid peroxide level, and plasminogen activator inhibitor-1 (PAI-1) have been reviewed systematically (46). Intake at typical daily levels has not been found to result in increased occurrence of clinically significant adverse effects (46). In the JELIS, administration of 1.8 g/d EPA did not increase hemorrhagic stroke, stomach cancer, lung cancer, colon cancer, breast cancer, or LDL-cholesterol concentration (49). Therefore, a DG (upper boundary) of EPA and DHA was not set.

In setting the DRIs, the safety of incidental intake of heavy metals, such as mercury, cadmium, lead, and tin, and of chemical environmental pollutants, such as dioxins and polychlorinated biphenyls (PCBs), which are generally present in fish, was not considered because other regulations apply to these compounds. In addition, the amount of toxic compounds varies between fish species and the areas where fish are caught. Guidelines for the safety of toxic compounds in food have been issued by the Japanese Government and should also be referred to.

Table 5. Dietary Reference Intakes for cholesterol (mg/d).

Sex	Males	Females
Age	DG	DG
0–5 mo	—	—
6–11 mo	—	—
1–2 y	—	—
3–5 y	—	—
6–7 y	—	—
8–9 y	—	—
10–11 y	—	—
12–14 y	—	—
15–17 y	—	—
18–29 y	<750	<600
30–49 y	<750	<600
50–69 y	<750	<600
≥70 y	<750	<600
Pregnant women		—
Lactating women		—

DG, tentative dietary goal for preventing lifestyle-related diseases.

5-7. DG (lower and upper boundary) of *n*-3 fatty acids.

Questions such as “If sufficient amounts of EPA and DHA are consumed, is it unnecessary to consume α -linolenic acid?” and “When very low amounts of EPA and DHA are consumed, should intake of α -linolenic acid be increased?” are difficult to answer because of insufficient data regarding the optimal ratio of α -linolenic acid to EPA and DHA intake. Therefore, the DG (lower boundary) of total *n*-3 fatty acid intake (including α -linolenic acid, EPA, and DHA) for adults aged 18 y and over was set at the 50th percentile value of the dietary intake of the Japanese population. However, as both the JPHC study and the JELIS observed beneficial effects of fish oil intake on CAD (albeit without considering basal intake of α -linolenic acid), more than 1 g/d intake of EPA and DHA is advised, regardless of intake of α -linolenic acid. A DG for the upper boundary of total *n*-3 fatty acids was not set because the values for α -linolenic acid and fish oils were not set (Table 4).

6. Dietary cholesterol

6-1. DG (lower boundary). Either increased or decreased blood cholesterol concentration has been associated with elevated mortality from stroke in a U-shaped-curve manner (63). The increased mortality from ischemic stroke observed in subjects with high blood cholesterol concentrations was due in part to increased LDL-cholesterol concentration, which promotes atherosclerosis. Observation of elevated mortality from intracerebral hemorrhage in patients with lower blood cholesterol concentrations does not confirm that low blood cholesterol concentration is a cause of hemorrhagic stroke (64, 65). Japanese cohort studies have found no association between dietary cholesterol intake and incidence of stroke, including hemorrhagic stroke (7, 8, 10, 66). Interestingly, one study that had

identified an inverse association between dietary cholesterol intake and incidence of stroke found that this association disappeared after adjusting for intake of animal protein and fat (66). As a meta-analysis found that treatment to reduce blood cholesterol concentration did not increase incidence of stroke (67), a DG (lower boundary) for cholesterol was not set.

6-2. DG (upper boundary). In cohort studies in the United States, no association was found between intake of cholesterol (or egg consumption) and incidence of CAD (12, 68–70). However, in the Honolulu Heart Program Study, Japanese whose intake of cholesterol was more than 325 mg/1,000 kcal (747 mg/d expressed on a daily basis), showed a significant increase in mortality from CHD (8). In one of the NIPPON DATA 80 studies, a series of cohort studies conducted in Japan, no association was found between egg consumption and death due to ischemic heart disease in subjects who had undergone dietary assessment in 1980 and been followed up to 1994 (71). In a study in which subjects underwent dietary assessment between 1990 and 1994 and were followed up to 2001, those who ate fewer eggs were found to have increased incidence of CHD (72). However, this finding could be attributed to reverse causation; that is, the subjects with high blood cholesterol tended to reduce egg consumption due to exposure to a public campaign advising them to do so to lower their blood cholesterol. Therefore, it is difficult to interpret the results of recent studies that examined the association between cholesterol intake and cardiovascular disease. In the NIPPON DATA 80 study, women who ate more than 2 eggs per day were found to have a 2-fold higher risk of mortality from cancer compared with women who seldom ate eggs (71). Recent studies have supported this finding, having found a positive association between intake of cholesterol and incidence of ovarian and endometrial cancer (73, 74) as well as lung, pancreatic, and colon/rectal cancer (75). Thus, a high intake of cholesterol is not recommended for the public at large. Using the data from the Honolulu Heart Program Study (8), the DG for the upper boundary of cholesterol intake was set at 750 mg/d for men and 600 mg/d for women, with these different values reflecting adjustment by differences in daily energy intake (Table 5).

7. Trans fatty acids

7-1. Background information. Trans fatty acids are mostly derived from 3 sources: 1) partially hydrogenated foods, such as margarine; 2) geometrical isomers of linoleic and α -linolenic acid resulting from the deodorization process; and 3) naturally occurring trans fatty acids from beef, lamb, and dairy fat resulting from biohydrogenation in ruminants. In humans, high intake of partially hydrogenated vegetable oils has been associated with increased incidence of CHD, obesity, allergies, lower birth weight, and fetal loss (76). As high intake of trans fatty acids derived from ruminants has not been associated with CHD, obesity, or diabetes, it is considered less harmful than high intake of other forms of trans fatty acids (77–80).

7-2. DG (upper boundary). High intake of trans

fatty acids leads to an increase in blood LDL-cholesterol and a decrease in HDL-cholesterol concentration, resulting in an increase in the LDL-cholesterol/HDL-cholesterol and total cholesterol/HDL-cholesterol ratios in a dose-dependent manner (81). High intake of trans fatty acids has also been associated with increased risk of CHD in a dose-dependent manner (11). However, it is unclear whether the incidence of CHD is significantly higher among average Japanese adults, who consume a low amount of trans fatty acids, than it is among Japanese adults who consume no trans fatty acids at all. Nevertheless, it is conceivable that in individuals with multiple risk factors for CHD, such as smoking, hypertension, diabetes mellitus, and dyslipidemia, increased intake of trans fatty acids may promote atherosclerosis to a greater degree than in individuals without these risk factors. Increased intake of trans fatty acids may increase the incidence of several diseases, such as CHD, obesity, and allergies and result in lower birth weight and increased risk of fetal loss, especially in individuals with other risk factors. Therefore, it is recommended that we eat less trans fatty acids at all ages.

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