II
Energy and Nutrients

Energy

1. Background Information

The energy obtained by the body through external sources is used for the maintenance of vital functions, and performance of physical activity. Most of this energy is ultimately released from the body in the form of heat. Energy intake, expenditure, and accumulation in the body, therefore, are expressed in their equivalent calorific values. The unit for energy in the International System of Units is joule (J), although calorie (cal) is often used in nutrition science. Since “1 J” is an extremely small unit, it is more practical to use kJ (or MJ) and kcal. The unit “kcal” is used in the current *Dietary Reference Intakes* (DRIs) 2015. In accordance with the Joint Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Special Technical Committee Report 1, 1 kcal = 4.184 kJ.

Energy intake is the sum of each energy conversion factor (amount of energy used per g of each component) calculated for the fats, proteins, and carbohydrates contained in foods. Energy expenditure, meanwhile, is classified into three categories, namely basal metabolism, postprandial thermogenesis, and physical activity. Physical activity is further divided into exercise (intentionally performed to improve physical fitness), activities of daily living, and spontaneous activity (such as postural and muscle tone maintenance).

Energy balance is defined as the balance between energy intake and energy expenditure. In adults, changes in energy balance results in changes in body weight and body mass index (BMI). If individuals’ energy intake continues to exceed their energy expenditure (positive energy balance), their body weight increases; however, if their energy expenditure exceeds their energy intake (negative energy balance), their body weight decreases. Short-term energy imbalance, therefore, can be assessed through changes in body weight. Moreover, energy imbalance can be adjusted on a long-term basis through reciprocal changes in energy intake, energy expenditure, and body weight. For example, if excessive energy intake continues over a long period of time, the energy expenditure increases due to changes in the exercise efficiency associated with weight gain. Weight gain eventually levels off at a certain amount, and the body transitions to a new state in which the energy balance is maintained. In many adults, maintaining a relatively constant body weight and body composition over a long period of time can result in a state in which the energy balance is maintained at almost zero. Energy intake and expenditure are almost equal even in individuals with obesity or malnourishment if no changes in body weight or composition occur. It is, therefore, insufficient, from the perspective of maintaining and promoting health and preventing lifestyle-related diseases (LRDs), to simply satisfy the required intake of energy without excess or deficiency. It is important to consume the amount of energy sufficient to maintain a desirable BMI. This is why BMI has been adopted in the current DRIs as an indicator of the maintenance of energy intake and expenditure balance.

2. Energy Intake and Expenditure

2-1. Factors Involved in Energy Intake and Expenditure

Various factors, and their interactions, influence energy intake, such as the nutrient composition of meals (energy density[1,2]), the energy intake ratio from fats[3,4], amount of proteins[5] and dietary fiber[6], and other characteristics of food (taste, color, texture, and palatability),[7,8] as well as eating patterns (portion sizes[9], eating speed[10], meal time zone[11], and number of foods[7,12]).

Food selection and dietary patterns are influenced by various external and social factors, in modern society: the convenience of obtaining food[13], snack intake[14], communal dining[12], TV viewing[15], food advertising on television[16], food prices[17], and internal and subjective factors such as stress[18] are also involved, in addition to the intentional control of intake by individuals.

The mechanisms regulating appetite and satiety *in vivo*[19,20] involve the transmission of satiety signals from the liver to the hypothalamus via the vagus nerve, and various appetite-related hormones associated with dietary intake derived from the gastrointestinal tract and pancreas. Various external and internal factors also ultimately control intake by transmitting signals to the hypothalamus via the cerebral cortex. Furthermore, hormones secreted from adipocytes act on the hypothalamus to adjust intake and to maintain a certain amount of body fat (lipostat theory)[21]. Factors such as lack of sleep[22], physical activity[23,24], sex[25], menstrual cycle[26], and genetics[27] also influence the amount of intake.

Energy expenditure is composed of parts that vary intentionally (exercise, and activities of daily living), and those that are biologically defined (basal metabolism, postprandial thermogenesis, and spontaneous activity). Energy expenditure during exercise and activities of daily living is determined based on the body weight and degree of obesity. Basal metabolism is determined by factors such as body weight, body composition, age, and sex, and is also affected by energy balance. Postprandial thermogenesis is equivalent to approximately 10% of the calorific value of energy intake, and is also affected by the nutrient composition of foods such as proteins[28]. Expenditure for both activities of daily living and spontaneous activity is referred to as non-exercise activity thermogenesis (NEAT). NEAT is affected by energy balance[29,30], and the degree of obesity[31].

Energy intake and energy expenditure, therefore, constitute factors that are influenced by individual biological and external factors, and those that can be controlled intentionally; in addition, these factors are interrelated. When strategically managing energy intake for the maintenance and promotion of health, and prevention of LRDs, it is best to consider facilitation of energy intake control after acquiring a full understanding of the effect of these factors.

2-2. Relationship between Energy Intake, Energy Expenditure, and Estimated Energy Requirement

The methods of estimating energy requirement are broadly divided into those that estimate energy intake under constant weight conditions, and those that measure energy expenditure. Energy intake measurement includes various dietary assessments, while energy expenditure measurement involves the doubly labeled water method, and calculation methods using sex, age, height, body weight, and measured values for basal metabolism and physical activity level (PAL). The doubly labeled water method directly measures energy expenditure. Any types of dietary assessment methods can produce large measurement errors for energy intake. It is, therefore, very difficult to estimate energy requirement from energy intake estimation methods. For this reason, the method in which the energy requirement is estimated closer to the energy expenditure than the energy intake is widely used (Figure 1). The doubly labeled water method, in particular, can directly measure (somewhat habitual) energy expenditure over approximately 2 weeks, and has high measurement accuracy. Therefore, it provides useful basic data for the estimation of energy requirement(32). Energy requirement can be estimated according to PAL, as well as sex and age group. However, individual differences in energy requirements, that cannot be estimated using these methods but cannot be ignored, exist(33). Therefore, it is difficult to estimate energy requirement, at an individual level, even when the energy requirement is estimated taking into account PAL, using the energy expenditure obtained from the doubly labeled water method. This also includes estimation formulas using factors such as basal metabolism and PAL(34). Moreover, different methods are used to measure energy intake and consumption, and each method has its own measurement errors. Thus, there is little sense in comparing measured energy intake, and measured energy expenditure.

The results of energy balance are expressed as BMI, and changes in body weight. Therefore, it is possible to acquire an overview of energy balance, if the BMI and changes in body weight are known. However, it should be noted that, while BMI and changes in body weight merely indicate one of the results of energy balance, they do not indicate energy requirements.

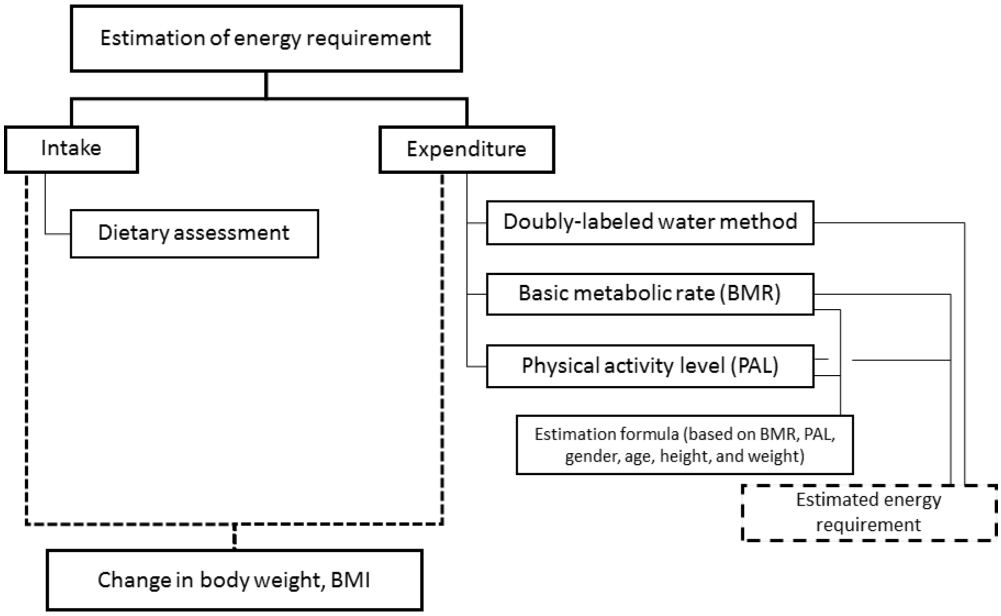

Figure 1 Measurement methods for estimation of energy requirement and association with change in body weight, BMI, or estimated energy requirement

3. Body Weight Control

3-1. Basic Concept

If the level of physical activity remains unchanged, energy intake control is almost the same as that of body size. It is, therefore, best to assess and change energy intake and expenditure by calculating the results of measurements of body size. In doing so, measurements of estimated energy requirements, using an estimation formula, or measurements of energy intake and supply should not be used. To make these changes, the desired body size must be determined in advance.

As no significant changes in height occur after an individual enters adulthood, body size is primarily controlled by body weight. To control body weight in adults, taking into account differences in height, BMI is primarily used as a body size index. Under normal circumstances, fat--including subcutaneous and visceral fat--and tissues other than fat (primarily muscle) need to be considered. One of the ways to do so is measuring waist circumference. Some reports state that waist circumference or its ratio to height is more strongly correlated with the incidence of diabetes and cardiovascular disease and total mortality than BMI[35,36]. However, due to the many accumulated research outcomes, and BMI being the most basic body size index, body weight and BMI are used as the body size indices here. It is recommended to take into account waist circumference as well, when preventing the development and progression of diabetes and cardiovascular disease.

Distribution curves (growth curves) for the height and body weight of Japanese people of a relevant sex and age are used for infants and children.

A high level of physical activity is an effective means of preventing and improving obesity[37], and a PAL of 1.7 or higher is recommended to prevent unhealthy weight gain[38]. A high level of physical activity has also been found to be related to a decrease in total mortality, independent of body weight(39,40). From the standpoint of preventing the development and progression of the LRDs associated with weight gain, having a low PAL (level I) is not advisable; energy needs to be balanced by increasing the level of physical activity.

3-2. Prevention

3-2-1. Basic Concept

To determine a healthy body weight using BMI, in adults, it is necessary to define what is considered “most healthy” in advance, and to examine the effect of BMI on health. A BMI state in which the all-cause mortality (total mortality) was the lowest was considered “healthy” in the current DRIs. Another possible concept is the BMI, at which individuals have the least number of diseases and adverse health effects, at a certain point in time (prevalence), being considered “healthy”. However, the mortality is not necessarily high for diseases or highly prevalent adverse health effects. Therefore, care must be taken, considering that mortality and disease prevalence are not always the same, and that BMI showing lowest rates mortality does not necessarily show corresponding decrease of the latter.

It is also inappropriate to use total mortality in the case of infants and children, and body weight control during pregnancy.

3-2-2. Methods Using Total Mortality as an Indicator

According to a meta-analysis that summarized the correlation between BMI, at the start of follow-up, and subsequent total mortality, using data from 57 cohort studies in 35–89-year-old individuals (conducted in Europe and the United States of America; total sample size: 894,576 individuals), the lowest mortality was seen in the group with a BMI of 22.5–25.0 kg/m^2 (in both men and women) after adjusting for age(41). However, an analysis of non-smokers alone, conducted with the aim of eliminating the effect of increased mortality and weight loss due to smoking, found that having a slightly lower BMI resulted in the lowest mortality(42). The results of studies in Japan and neighboring East Asian countries need to be referenced in addition to the results of studies in Europe and America. The correlation between BMI (kg/m^2), at the start of follow-up, and subsequent mortality, in a pooled analysis of two representative cohort studies and seven other cohort studies, in healthy individuals in Japan, is presented in Figure 2(43–45). Representative reports from neighboring East Asian countries are also summarized in Figure 3(46–48).

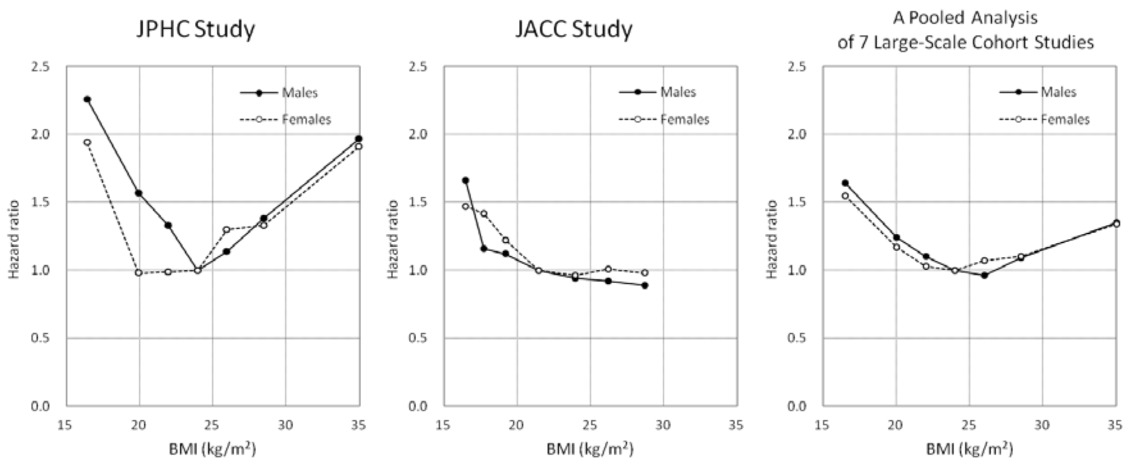

Figure 2. The association between BMI at baseline and mortality among normal subjects in JPHC study, JACC study, and a pooled analysis of 7 large-scale cohort studies(43-45)

The intermediate values of the BMI ranges studied were shown as plots. The result was not shown if the maximum or minimum value of the highest or lowest BMI categories was not available.

JPHC study: The reference category was BMI=23.0-24.9kg/m^2. The range of age at baseline was 40-59 years, the mean follow-up duration was 10 years, the analytic sample was 19,500 men and 21,315 women, and the number of death was 943 men and 483 women. The analysis was adjusted for residential area, age, body weight change around 20 years old, alcohol drink, leisure physical activity and educational background.

JACC study: The reference category was BMI=20.0-22.9kg/m^2. The range of age at baseline was 65-79 years, the mean follow-up duration was 11.2 years, the analytic sample was 11,230 men and 15,517 women, and the number of death was 5,292 men and 3,964 women. The analysis was adjusted for smoking status, alcohol drinking, physical activity, sleep duration, stress, educational background, marital status, green-vegetable intake, history of stroke, history of myocardial infarction and history of cancer.

A pooled analysis of 7 large-scale cohort studies: The reference category was BMI=23.0-24.9kg/m^2. The range of age at baseline was 40-103 years, the mean follow-up duration was 12.5 years, the analytic sample was 162,092 men and 191,330 women, and the number of death was 25,944 men and 16,036 women. The analysis was adjusted for age, smoking status, alcohol drinking, history of hypertension, leisure or physical activity, and other variables (depending on each cohort). This analysis excluded early follow-up (less than 5 years).

Of the studies presented in Figures 2 and 3, a tendency toward lower mortality with a higher BMI is only seen in the Japan Collaborative Cohort (JACC) study, which limited its analysis to a group of participants aged 65–79 years (at the start of follow-up). As this study demonstrated, the correlation between BMI and total mortality differs depending on age and BMI, with the lowest total mortality showing a tendency to increase as the age at the start of follow-up increases, in both men and women. The South Korean study presented in Figure 3 too did not reveal a clear increase in total mortality, even when the BMI exceeded 30.0 kg/m^2, in a sub-analysis of individuals aged 65 years and older(48). Moreover, according to a Japanese study that examined BMI with the lowest total mortality, according to age at the start of follow-up, the BMI values in men and women, respectively, were 23.6 kg/m^2 and 21.6 kg/m^2 for those aged 40–49 years, 23.4 kg/m^2 and 21.6 kg/m^2 for those aged 50–59 years, 25.1 kg/m^2 and 22.8 kg/m^2 for those aged 60–69 years, and 25.5 kg/m^2 and 24.1 kg/m^2 for those aged 70–79 years(49). Furthermore, the results of a pooled analysis (results of lifetime non-smokers), summarizing data from 19 cohort studies in American Caucasian individuals (total: 1.46 million individuals),

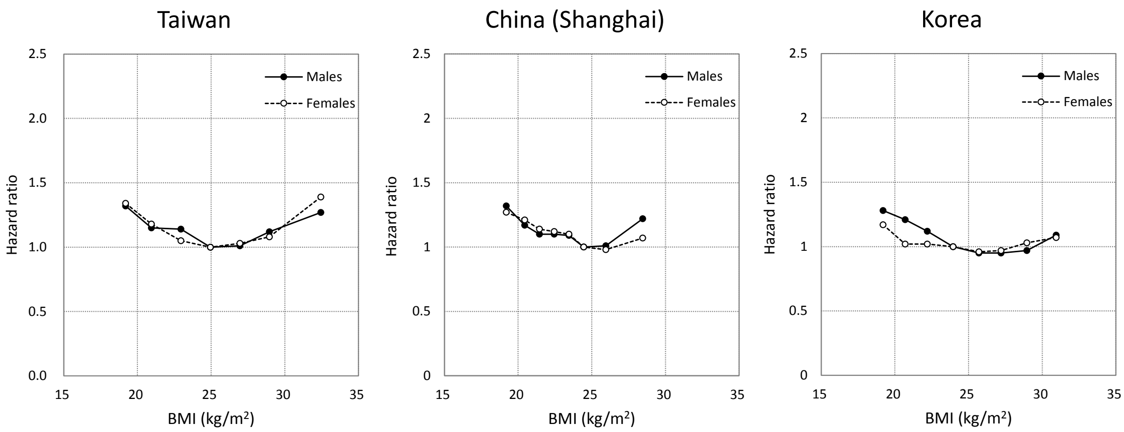

Figure 3. The association between BMI at baseline and mortality among normal subjects in East Asian representative 3 cohort studies(46-48)

The intermediate values of the BMI ranges studied were shown as plots. The result was not shown if the maximum or minimum value of the highest or lowest BMI categories was not available.

Taiwan: The reference category was BMI=24.0-25.9 kg/m^2. The range of age at baseline was 20 years and higher, the mean follow-up duration was 10 years, the analytic sample was 58,738 men and 65,718 women, and the number of death was 3,947 men and 1,549 women. The analysis was adjusted for age, alcohol drink, educational background, smoking status, income and use of betal nuts.

China (Shanghai): The reference category was BMI=24.0-24.9 kg/m^2. The range of age at baseline was 40 years and higher, mean follow-up duration was 8.3 years, the analytic sample was 158,666 men and women, and the number of death was 10,047 men and 7,640 women. The analysis was adjusted for age, alcohol drink, physical activity, residential area and urbanization of the residential area.

Korea: The reference category was BMI=23.0-24.9 kg/m^2. The range of age at baseline was 30-95 years, mean follow-up duration was 12 years, the analytic sample was 770,556 men and 443,273 women, and the number of death was 58,312 men and 24,060 women. The analysis was adjusted for age, smoking status, alcohol drinking, exercise participation, fasting plasma glucose, systolic blood pressure and serum cholesterol levels.

are presented in Figure 4. These results revealed that the BMI producing a supposed hazard ratio lower than ±0.1, with 22.5–24.9 kg/m^2 as the standard, was 18.5–24.9 kg/m^2 in those aged 20–49 years, 20.0–24.9 kg/m^2 in those aged 50–59 years, and 20.0–27.4 kg/m^2 in those aged 60–69 and 70–84 years[42]. Incidentally, the above-mentioned studies inevitably included individuals who had already experienced weight loss due to preexisting latent diseases, or adverse health effects at the time of the baseline survey; this may have led to some kind of "reversal causality". The possibility of a phenomenon, whereby total mortality is lowest for a slightly higher BMI, which contradicts the true correlation, cannot be ruled out. Some schools of thought question the existence of this phenomenon and its effect on results; however, no consensus has yet been reached[50,51].

Another report states that weight gain or a loss of 5 kg or more, over a 5-year period, is correlated with an increase in mortality, irrespective of BMI[52]. However, the effects of weight gain or loss on health are thought to differ, depending on whether the gain or loss is intentional or unintentional. One report found that the mortality in a group of obese individuals, who intentionally lost weight, was significantly lower than that of a group of individuals whose weight did not change[53]; however, a meta-analysis found that the effect of intentional weight

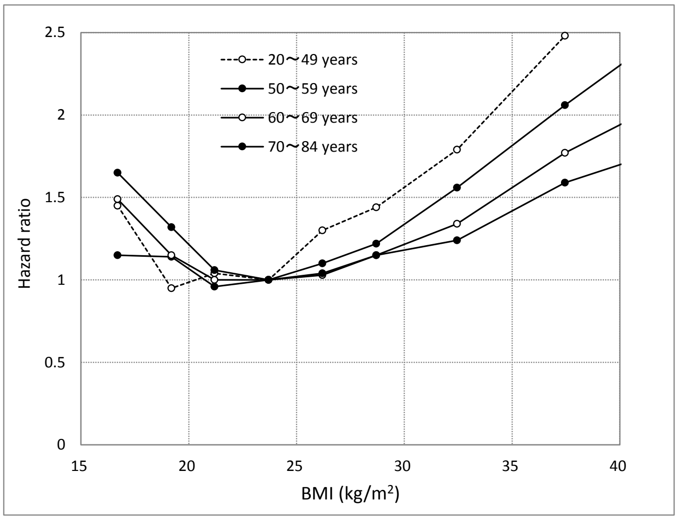

Figure 4. The hazard ratio for mortality by age categories from the results of a pooled analysis of 19 cohort studies (1,460,000 white Americans): the analysis of never smokers[(42)]

The intermediate values of the BMI ranges studied were shown as plots. The reference category was BMI=22.5-24.9 kg/m^2. The range of age at baseline was 19-84 years (median 58), and the mean follow-up duration was 10 years (range: 5-28). The analysis was adjusted for sex, alcohol drink, educational background, marital status and physical activity.

loss on decreased mortality is not always clear[(54)], and conclusions are yet to be drawn.

Additionally, according to a study that observed the correlation of cause-specific mortality with BMI, the BMI with the lowest mortality for cardiovascular diseases, particularly cardiac diseases, was lower than the BMI with the lowest total mortality; however, the BMI

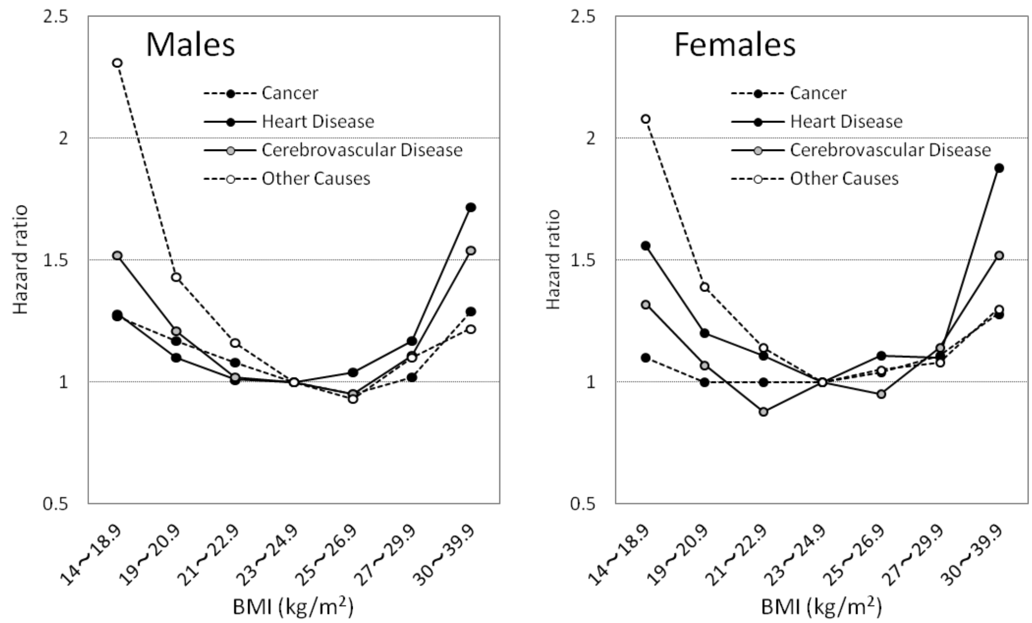

Figure 5 The association between cause-specific mortality and baseline BMI: the hazard ratio compared to BMI=23-24.9kg/m^2 from a pooled analysis of 7 Japanese cohort studies

The range of age at baseline was 40-103 years, the mean follow-up duration was 12.5 years, the analytic sample was 162,092 men and 191,330 women, and the number of death was 25,944 men and 16,036 women. The analysis was adjusted for age, smoking status, alcohol drinking, history of hypertension, leisure or physical activity, and other variables (depending on each cohort). This analysis excluded early follow-up (less than 5 years).

with the lowest mortality for other diseases, particularly respiratory diseases, tended to be higher(41,43,45). The results of a pooled analysis of seven Japanese cohort studies are presented in Figure 5 as an example. Furthermore, a study found that the incidence of diabetes decreased as the BMI decreased(55,56), and the correlation between the two differed greatly from the correlation observed between BMI and total mortality.

Thus, a summary of the range of BMIs with the lowest total mortality reported in observational epidemiological studies is presented in Table 1.

Table 1. The range of BMI which showed the lowest all-cause mortality in observational studies (18 years and older)[1]

Age (years)	BMI (kg/m^2) which showed the lowest all-cause mortality
18-49	18.5-24.9
50-69	20.0-24.9
70+	22.5-27.4

[1] For both males and females.

However, as presented in Table 2, on studying the proportion of Japanese individuals whose BMI falls below, within, and above the range with the lowest total mortality, a large discrepancy was observed, in real-world settings, for those aged 18–49 years (10.1% above, 68.4% within, and 21.5% below), 50–69 years (15.8% above, 56.5% within, and 27.7% below), and 70 years or older (45.0% above, 45.5% within, and 9.5% below).

Table 2. BMI distribution by gender and age groups

<table>
<tr><th colspan="2">Age (years)</th><th colspan="7">Distribution of BMI (%)</th></tr>
<tr><td rowspan="7">18-49</td><td>Range of BMI</td><td><18.5</td><td>18.5-19.9</td><td colspan="2">20.0-22.4</td><td>22.5-24.9</td><td>25.0-27.4</td><td>27.5≦</td></tr>
<tr><td rowspan="2">Total</td><td>10.1</td><td>17.3</td><td colspan="2">29.8</td><td>21.3</td><td>11.6</td><td>9.8</td></tr>
<tr><td>10.1</td><td colspan="4">68.4</td><td colspan="2">21.5</td></tr>
<tr><td rowspan="2">Males</td><td>4.7</td><td>11.2</td><td>16.2</td><td>11.4</td><td>26.9</td><td>15.7</td><td>14.0</td></tr>
<tr><td>4.7</td><td colspan="4">65.7</td><td colspan="2">29.7</td></tr>
<tr><td rowspan="2">Females</td><td>14.7</td><td>22.5</td><td>20.7</td><td>11.0</td><td>16.6</td><td>8.1</td><td>6.4</td></tr>
<tr><td>14.7</td><td colspan="4">70.8</td><td colspan="2">14.5</td></tr>
<tr><td rowspan="7">50-69</td><td>Range of BMI</td><td><18.5</td><td>18.5-19.9</td><td colspan="2">20.0-22.4</td><td>22.5-24.9</td><td>25.0-27.4</td><td>27.5≦</td></tr>
<tr><td rowspan="2">Total</td><td>5.7</td><td>10.1</td><td colspan="2">28.0</td><td>28.5</td><td>17.3</td><td>10.3</td></tr>
<tr><td colspan="2">15.8</td><td colspan="3">56.5</td><td colspan="2">27.7</td></tr>
<tr><td rowspan="2">Males</td><td>2.9</td><td>7.2</td><td>12.2</td><td>12.7</td><td>32.3</td><td>21.7</td><td>11.0</td></tr>
<tr><td colspan="2">10.1</td><td colspan="3">57.2</td><td colspan="2">32.7</td></tr>
<tr><td rowspan="2">Females</td><td>8.1</td><td>12.5</td><td>18.0</td><td>12.6</td><td>25.4</td><td>13.7</td><td>9.8</td></tr>
<tr><td colspan="2">20.6</td><td colspan="3">56.0</td><td colspan="2">23.5</td></tr>
<tr><td rowspan="10">70+</td><td>Range of BMI</td><td><18.5</td><td>18.5-19.9</td><td colspan="2">20.0-22.4</td><td>22.5-24.9</td><td>25.0-27.4</td><td>27.5≦</td></tr>
<tr><td rowspan="3">Total</td><td>8.7</td><td>9.9</td><td>14.4</td><td>12.0</td><td>28.6</td><td>16.9</td><td>9.5</td></tr>
<tr><td colspan="4">45.0</td><td colspan="2">45.5</td><td>9.5</td></tr>
<tr><td colspan="3">33.0</td><td colspan="2">40.6</td><td colspan="2">26.4</td></tr>
<tr><td rowspan="3">Males</td><td>7.2</td><td>8.9</td><td>13.4</td><td>11.8</td><td>31.9</td><td>18.3</td><td>8.6</td></tr>
<tr><td colspan="4">41.3</td><td colspan="2">50.2</td><td>8.6</td></tr>
<tr><td colspan="3">29.5</td><td colspan="2">43.7</td><td colspan="2">26.9</td></tr>
<tr><td rowspan="3">Females</td><td>9.9</td><td>10.7</td><td>15.2</td><td>12.2</td><td>26.0</td><td>15.9</td><td>10.2</td></tr>
<tr><td colspan="4">48.0</td><td colspan="2">41.9</td><td>10.2</td></tr>
<tr><td colspan="3">35.8</td><td colspan="2">38.2</td><td colspan="2">26.1</td></tr>
</table>

Data source: National Health and Nutrition Survey 2010, 2011

3-2-3. Target BMI Range

The current target BMI range, comprehensively determined taking into account mortality, incidence rates of each of the diseases, and their correlation with BMI, the correlation between cause of death and BMI, and the BMI state in Japanese people, as obtained from the results of observational epidemiological studies, is presented in Table 3. In those aged 70 years or older, in particular, a discrepancy was observed between reality and the BMI with the lowest total mortality; therefore, the current target BMI range was set at 21.5–24.9 kg/m^2, based on the need to consider the prevention of both frailty and LRDs. However, many factors (including genetic factors and environmental factors, such as living habits) contribute to total mortality, and there is little sense in strictly managing only BMI to control body weight. Furthermore, a high level of physical activity is effective in preventing and reducing obesity(37), and reportedly correlates with a decrease in total mortality, independent of body weight(39,40). Therefore, the use of BMI should be limited to the maintenance of health and prevention of LRDs. The correlation of BMI with malnutrition and disease prevention (including stroke prevention) is important from the perspective of preventative care, particularly in those aged 70 years or older, in order to avoid frailty due to advanced old age. However, it is best to manage BMI, taking into consideration the characteristics of each individual.

Table 3. Target BMI range (18 years and older)[1, 2]

Age (years)	Target BMI
18-49	18.5-24.9
50-69	20.0-24.9
70+	21.5-24.9[3]

1 For both males and females. These values shall be used merely as a reference.

2 Target range is defined through comprehensive consideration on the association between incidence rate for each disease and BMI, the association between causes of death and BMI, and actual BMI of Japanese people, based on BMI with the lowest all-cause mortality reported in epidemiological observational studies.

3 For people 70 years and over, the actual BMI deviates from the BMI with the lowest all-cause mortality. The tentative target BMI range is determined to be 21.5-24.9, considering the necessity to take into account both the prevention of frailty and prevention of LRDs.

For example, energy requirements calculated for a normal PAL (level II), using reference values for basal metabolism and reference height, as explained later in this text, in men and women aged 18–29 years, 30–49 years, 50–69 years, and 70 years or older, respectively, are 2,300–3,000 and 1,800–2,400, 2,100–2,800 and 1,800–2,400, 2,100–2,600 and 1,700–2,100, and 2,000–2,400 kcal/day and 1,700–1,900 kcal/day, demonstrating a wide range of requirements. Moreover, it should be noted that there are considerable inter-individual differences in energy requirement, even among those with the same BMI or body weight.

(Supplemental Statement) Estimated Energy Requirement

Method Used to Calculate the Estimated Energy Requirement

1. Basic Concept behind the Calculation Methods

If both body weight and body composition remain unchanged, energy intake is equal to energy expenditure, and total energy expenditure can be assessed using the doubly labeled water method. However, as explained earlier, various dietary assessments are typically affected by underreporting in the form of systematic errors, as well as random errors caused by diurnal variations. The estimated energy requirement is, therefore, calculated from the estimated value for total energy expenditure without using the energy intakes obtained from dietary assessments.

The estimated energy requirement for adults (excluding pregnant and lactating women) is calculated as follows:

Estimated energy requirement = basal metabolism reference value (kcal/kg, body weight/day) × reference body weight (kg) × physical activity level

In addition, to calculate the estimated energy requirements of infants, children, pregnant women, and lactating women, the amount of energy necessary for growth, the continuation of pregnancy, or lactation is added.

The estimated energy requirement for each sex, age group, and PAL was calculated as shown in Table 5. The factors used in the calculation are described below.

2. Basal Metabolism Reference Values

The basal metabolism reference values are presented in Table 4, based on those for adults, as measured in 13 Japanese studies (Figure 6)[57–69], and a study on individuals aged 6–17 years[70].

The basal metabolism reference value is determined such that the estimated value and the actual measured value in the reference body size match. This is why the estimation errors are greater for body sizes and greatly deviate from the standard. Basal metabolism is overestimated in Japanese people too, when the basal metabolism reference values are used for obese individuals[71]. Conversely, basal metabolism is underestimated in slim individuals. The estimated energy requirement, obtained by multiplying this over- or underestimated basal metabolism and the PAL, is likely to be greater than the true energy requirement of obese individuals and lower than that of slim individuals. Therefore, if this estimated energy intake is used to plan the energy intake, the body weight may increase in obese individuals and decrease in slim individuals.

Table 4. The basal metabolism reference values for the reference bodyweight (BW)

Gender	Male			Female		
Age (years)	Basal Metabolic Reference value (kcal/kg BW/day)	Reference BW (kg)	Basic Metabolic Rate (kcal/day)	Basal Metabolic Reference value (kcal/kg BW/day)	Reference BW (kg)	Basic Metabolic Rate (kcal/day)
1-2	61	12	700	60	11	660
3-5	55	17	900	52	16	840
6-7	44	22	980	42	22	920
8-9	41	28	1,140	38	27	1,050
10-11	37	36	1,330	35	36	1,260
12-14	31	49	1,520	30	48	1,410
15-17	27	60	1,610	25	52	1,310
18-29	24	63	1,520	22	50	1,110
30-49	22	69	1,530	22	53	1,150
50-69	22	65	1,400	21	53	1,100
70+	22	60	1,290	21	50	1,020

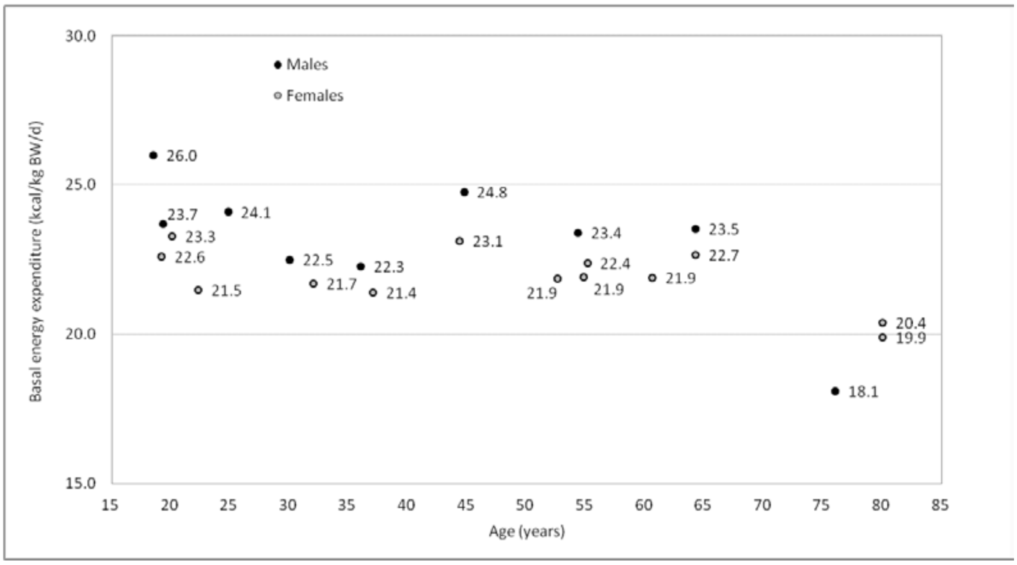

Figure 6. Reported basal metabolic rates in Japanese adults (13 studies)

The following estimation formula, for the basal metabolic rate of Japanese people, using age, sex, height, and body weight(65), has been shown not to produce systematic errors due to body weight, up to a BMI of approximately 30 kg/m^2(34), and can estimate the basal metabolic rate in obese individuals with a BMI of 25–29.9 kg/m^2.

Basal metabolism (kcal/day) = [0.0481 × body weight (kg) + 0.0234 × height (cm) − 0.0138 × age (years) − constant (men: 0.4235, women: 0.9708)] × 1000/4.186

Basal metabolic rate was found to be more strongly correlated with fat-free mass than body weight[62,65,68,72]. In the future, it may be possible to estimate basal metabolic rate with a higher degree of accuracy through the appropriate assessment of physical composition.

Incidentally, many reports claim that the basal metabolic rate of diabetes patients is either no different from that of healthy individuals or approximately 5%–7% higher, when corrected for body composition (this is thought to be due to the energy consumed during gluconeogenesis in the liver, among other factors.)[73–80]. Few studies have focused on the outcomes of hyperglycemic individuals receiving health guidance; however, a cross-sectional study found that the metabolic rate during sleep exhibited normal glucose tolerance < impaired glucose tolerance (IGT) < diabetes, and changes in the basal metabolism, over time, in the same individual resulted in normal glucose tolerance < IGT (+4%) < diabetes (+3%)[81]. Those with hyperglycemia, requiring just health guidance (fasting blood glucose levels: 100–125 mg/dL), therefore, have a basal metabolism that is not very different from that of those with euglycemia. Furthermore, only a few studies have examined the total energy expenditure in diabetes patients using the doubly labeled water method, and no differences in the PAL and total energy expenditure have been observed between diabetes patients and individuals with normal glucose tolerance[73,75].

3. Physical Activity Level

3-1. Adults

The PALs of adults, calculated from the measured energy expenditure and estimated basal metabolic rate of healthy Japanese adults (aged 20–59 years; 150 individuals)[82], were used. In other words, the overall PAL derived from the PALs of men and women was 1.72 ± 0.26 (mean ± standard deviation), and the overall PAL of 63 individuals with level II physical activity was 1.74 ± 0.26. Three types of PAL were established based on these data (Table 5).

Table 5. Daily activities and time of physical activity by PAL

PAL[1]	Low (I)	Moderate (II)	High (III)
	1.50 (1.40-1.60)	1.75 (1.60-1.90)	2.00 (1.90-2.20)
Daily activities[2]	Corresponds to sedentary lifestyle	Corresponds to sedentary work, however, includes movement and housework such as commuting and shopping, and sports with light intensity.	Individuals who involved in works with high-intensity physical activity or high-intensity leisure-time physical activity such as regular sports habits
Total time per day on physical activity of a moderate intensity (hour/day)[3]	1.65	2.06	2.53
Total length of walking at work (hour/day)[3]	0.25	0.54	1.00

1 Representative values (approximate range).

2 Based on reports of Black, et al.[83] and Ishikawa-Takata, et al.[82] and considered that PAL is largely influenced by physical activity during work

3 The data was based on Ishikawa-Takata, et al.[84]

Metabolic equivalent (MET; indicator of the intensity of each physical activity, expressed as a multiple of metabolic rate at rest, in the sitting position), and activity factor (Af; indicator of the intensity of each physical activity, expressed as a multiple of basal metabolic rate) are the indicators of the intensity of physical activity. As the metabolic rate at rest, in the sitting position, is approximately 10% higher while fasting than the basal metabolic rate measured in the supine position(85,86), the following relational equation was established: MET × 1.1 = Af. The METs of various physical activities, in healthy adults, have been summarized by Ainsworth *et al*.(87).

A study of a population of Japanese adults (mean age: 50.4 ± 17.1 years), including a relatively large number of individuals with a high PAL, found a difference between the three PALs, for the total time per day spent on physical activity of a moderate intensity (3–5.9 METs) and walking to work (Table 5)(84). Level II physical activity (normal) corresponds to sedentary work; however, a total of 2 hours/day are spent on movement and housework such as commuting and shopping, and a total of 30 minutes/day are spent on movement within the workplace.

The above study found, however, that the time spent on physical activity during leisure time was almost zero for all three PALs. A more accurate method of estimating PAL will need to be developed in the future, taking into account the time spent on each physical activity and exercise intensity, with a particular focus on work, traveling (commuting, shopping, etc.), and housework.

In addition, when the energy expenditure from physical activity is estimated in the activity logs in the US-Canada DRIs(33,85), excess post-exercise oxygen consumption (EPOC) from hypermetabolism after physical activity is included in the calculations for estimated energy requirement, assuming it accounts for 15% of the energy expenditure during the physical activity in question. However, the EPOC in activities of daily living is, in fact, very low(86).

3-2. Elderly Individuals

Elderly individuals are more likely to have PALs that differ from those of other age groups. The typical PAL was set at 1.70, on the basis of reports that measured the PALs in healthy, independent elderly individuals (Table 6)(88–97). Levels I, II, and III were also determined based on a study in which participants were divided into three groups based on their level of physical activity (Table 7)(98). The mean age of the participants in a majority of those reports was 70–75 years; there is a lack of data on those aged 80 years and older. A study that re-assessed 75-year-old participants when they reached age 82 years found that the levels dropped only in men who previously had a high PAL, and that the overall PAL of both men and women was approximately 1.68(99).

Table 6. Reports that measured the PALs in healthy, independent elderly individuals

Reference	Subjects	Age (years) mean ± standard deviation	Sex (number)	BMI (kg/m²) mean ± standard deviation	PAL mean ± standard deviation/inter quartile range
87	Healthy subjects	73	males (3), females (9)	25 ± 3	1.73 ± 0.25
88	Healthy subjects	74 ± 6	males (14) females (18)	22.5 ± 2.5	1.66 ± 0.24
89	Dependently-living individuals	72.8 ± 6.1	males (8)	22.4 ± 2.5	1.4 ± 0.1
90	Retired subjects	74.0 ± 4.4	females (10)	24.1 ± 2.8	1.59 ± 0.19
91	Healthy subjects	73 ± 3	females (10)	none	1.80 ± 0.19
92	Healthy subjects	73.4 ± 4.1	males (19)	none	1.71 ± 0.32
93	Black	74.6 ± 3.2	females (67)	28.6 ± 5.9	1.69 ± 0.24
	White	74.6 ± 3.2	females (77)	26.2 ± 5.3	1.65 ± 0.21
	Black	74.8 ± 2.9	males (72)	27.1 ± 4.5	1.71 ± 0.24
	White	75.1 ± 3.2	males (72)	27.6 ± 4.2	1.74 ± 0.22
94	Relatively-healthy subjects	78	males (2) females (9)	24.3 ± 2.6	1.74 ± 0.25
95	Home-living subjects	82 ± 3*	males (17)	24.8 ± 3.8	1.6 ± 0.2
96	Disease-free and walkable subjects	74.7 ± 6.5	males (12) females (44)	25.8 ± 4.2	1.72 (1.63-1.92)
98	Follow-up study of reference 93	74.7	males (47)	27.0 ± 4.3	1.77 ± 0.23
		82.2		27.1 ± 4.8	1.68 ± 0.21
		74.5	females (40)	28.4 ± 4.5	1.68 ± 0.19
		82.0		28.0 ± 4.3	1.67 ± 0.31

* The value for age and BMI is the mean value of 17 ± 6 individuals (total 23).

3-3. Children

Studies that measured the PALs of children, using the doubly labeled water method, were systematically reviewed, and the average, weighted by the number of participants, was set as the PAL. As a general rule, only reports that measured basal metabolism were included in the review(100–132); however, reports that estimated the PAL using the basal metabolic rate in children aged less than 5 years were also included(133–139). The resulting PALs were 1.36 for ages 1–2 years, 1.48 for ages 3–5 years, 1.57 for ages 6–7 years, 1.62 for ages 8–9 years, 1.63 for ages 10–11 years, 1.74 for ages 12–14 years, and 1.81 for ages 15–17 years, demonstrating that the PAL had a tendency to increase with age (Figure 7). A separate meta-analysis, summarizing the results of 17 studies on the relationship between age and PAL in children, also found that PAL increased with age(140). The typical PAL for children was determined based on those reports (Table 7). The typical PALs of those aged 12–14 years and 15–17 years were set at just 0.05 lower than the weighted average. Some reports found that the PAL exceeded "normal; level II" in these age groups. Moreover, the 2012 Physical Fitness and Athletic Ability Survey found that the ratio of individuals who spent many hours, per day, on exercise and sports was high in this age group, because the typical level II PAL was assumed to be lower than the average level. From the age of 6 years, children were divided into the same three categories as adults, considering individual differences in the PAL. The standard deviations of the averages, weighted by the number of participants in each age group, extracted from the literature, varied, with widths between 0.17 and 0.27, according to the age group, and a mean width of 0.23. The PAL of each category, among children was, therefore, set at just 0.20 higher or lower than the "normal" value for each age group.

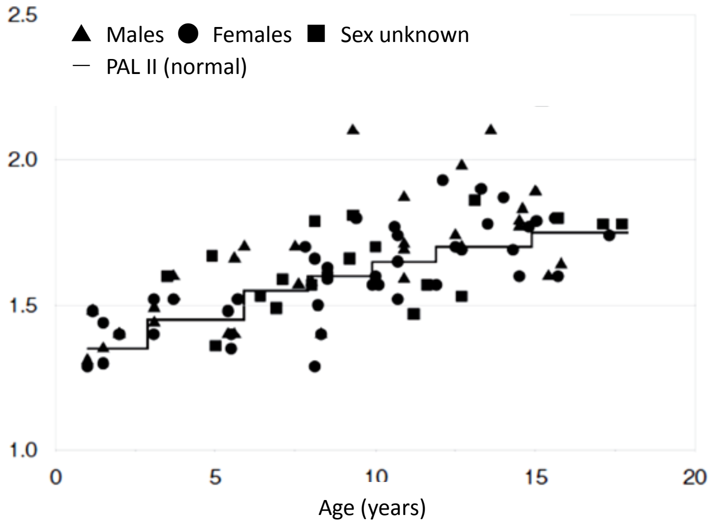

Figure 8. PAL of children by age

Table 7. PAL classification according to age categories

PAL	Low (I)	Moderate (II)	High (III)
1-2 (years)	-	1.35	-
3-5 (years)	-	1.45	-
6-7 (years)	1.35	1.55	1.75
8-9 (years)	1.40	1.60	1.80
10-11 (years)	1.45	1.65	1.85
12-14 (years)	1.50	1.70	1.90
15-17 (years)	1.55	1.75	1.95
18-29 (years)	1.50	1.75	2.00
30-49 (years)	1.50	1.75	2.00
50-69 (years)	1.50	1.75	2.00
70 years and older	1.45	1.70	1.95

3-4. For Overweight and Underweight Individuals

In obese individuals, the PAL assessed with a motion sensor, such as an accelerometer, is generally low, and it has been pointed out that obesity can be a cause of decreased activity[141]. However, PAL does not correlate with BMI up to a BMI of approximately 30 kg/m[142,143]. Moreover, the PAL does not change after weight loss, in obese individuals[144,145]. This is likely because obese individuals have poor exercise efficiency, and require more energy to perform certain external tasks[146,147]. In conclusion, the use of the same PAL values as those of normal individuals is allowed for obese individuals with a BMI of 25–29.9 kg/m^2.

4. Estimated Energy Requirement

4-1. Adults

The estimated energy requirement (kcal/day) in adults (aged 18 years and older) was calculated as follows:

Estimated energy requirement (kcal/day) = basal metabolic rate (kcal/day) × PAL

4-2. Children

Children in their growth phase (age 1–17 years) must consume extra energy for tissue synthesis and to build tissue (energy deposition), in addition to the energy required for their physical activity. The energy consumed during tissue synthesis is included in the total energy expenditure, so the estimated energy requirement (kcal/day) can be calculated as follows:

Estimated energy requirement (kcal/day) = basal metabolic rate (kcal/day) × PAL + stored energy storage (kcal/day)

The energy deposition for the increased tissue mass is calculated as the product of the sum of the daily weight gained in addition to the reference weight, and the energy deposition density[85]. See Table 8 for more information on the method of calculation.

Table 8. Energy deposition accompanied with growth

Gender	Males				Females			
Age	Reference BW (kg)	BW Increase (kg/year)	For building tissue		Reference BW (kg)	BW Increase (kg/year)	For building tissue	
			Energy Density (kcal/g)	Energy Deposition (kcal/day)			Energy Density (kcal/g)	Energy Deposition (kcal/day)
0-5 (months)	6.3	9.4	4.4	115	5.9	8.4	5.0	115
6-8 (months)	8.4	4.2	1.5	15	7.8	3.7	1.8	20
9-11 (months)	9.1	2.5	2.7	20	8.4	2.4	2.3	15
1-2 (years)	11.5	2.1	3.5	20	11.0	2.2	2.4	15
3-5 (years)	16.5	2.1	1.5	10	16.1	2.2	2.0	10
6-7 (years)	22.2	2.6	2.1	15	21.9	2.5	2.8	20
8-9 (years)	28.0	3.4	2.5	25	27.4	3.6	3.2	30
10-11 (years)	35.6	4.6	3.0	40	36.3	4.5	2.6	30
12-14 (years)	49.0	4.5	1.5	20	47.5	3.0	3.0	25
15-17 (years)	59.7	2.0	1.9	10	51.9	0.6	4.7	10

BW, body weight

BW increase was calculated as following:

Ex) BW increase amount for female children aged 9-11 months,

X = [(reference BW of 9-11 months old (at 10.5 months) – (reference BW of 6-8 months old (at 7.5 months)) / [0.875 (year old)-0.625(year old)] + [(reference BW of 1-2 years old) – (reference BW of 9-11 months old)] / [2 (years old)-0.875 (years old)]

BW increase=X/2 = [(8.4 – 7.8) / 0.25 + (11.0 - 8.4) / 1.125]] /2 ≒ 2.4

Energy density for building tissue was calculated according to the US/Canada DRIs[85].

Energy deposition for building tissue was calculated as the product of BW increase and energy density for building tissue.

Ex) Energy deposition for female children aged 9-11 months (kcal/day) = [(2.4 (kg/year)* 1000/365day)] * 2.3 (kcal/g) = 14.8 ≒ 15

4-3. Infants

Much like children, infants must consume energy for tissue synthesis and energy deposition, in addition to the energy required for physical activity. The energy consumed during tissue synthesis is included in the total energy expenditure, so the estimated energy requirement (kcal/day) can be calculated as follows:

Estimated energy requirement (kcal/day) = total energy expenditure (kcal/day) + stored energy storage (kcal/day)

With regards to the total energy expenditure of infants, the FAO/WHO/UNU conducted various studies on the relationship between sex, age (in months), body weight, height, and total energy expenditure, based on the results of previous studies that used the doubly labeled water method. Consequently, it was reported that the total energy expenditure of breastfed infants during infancy can be calculated using the following regression formula, in which body weight is the only independent variable[148,149]:

Total energy expenditure (kcal/day) = 92.8 × reference weight (kg) − 152.0

No reports have measured total energy expenditure using the doubly labeled water method, in Japanese infants. Therefore, the total energy expenditure per day (kcal/day) was calculated by substituting the reference weight of Japanese people into these formulas.

Much like in the case of children, the energy deposition in infants was calculated as the product of the sum of the daily weight gained, in addition to the reference weight, and the energy density for the increased tissue (Table 8)[133].

The estimated energy requirement in infants is shown according to age in months (0–5 months, 6–8 months, and 9–11 months). Furthermore, it should be noted that during the ages of 0–5 months, when an infant's body weight varies greatly, large differences arise in the energy requirement, between the first and second half of that period.

Attention must also be paid to the fact that artificially fed infants generally have a larger total energy expenditure than breastfed infants[148]. Moreover, the FAO/WHO/UNU state that the total energy expenditure of artificially fed infants can be estimated as follows[148,149]:

Total energy expenditure (kcal/day) = 82.6 × body weight (kg) − 29.0

4-4. Pregnant Women

The estimated energy requirement of pregnant women is calculated as follows:

Estimated energy requirement of pregnant women (kcal/day) = estimated energy requirement before pregnancy (kcal/day) + additional energy required for pregnancy (kcal/day)

Considering the childbearing age of women covers several age groups, for the estimated energy requirement, the amount of energy that should be additionally consumed compared to the amount consumed before pregnancy needs to be shown as the additional amount of energy required for each trimester to ensure a normal delivery and the maintenance of an appropriate nutritional status during pregnancy.

A longitudinal study using the doubly labeled water method found that the PAL during pregnancy decreased in the first and third trimesters; however, the basal metabolic rate greatly increased in the late stage due to pregnancy-related weight gain[148–154]. As a result, the rate of increase in the total energy expenditure was almost the same as the rate of increase in the body weight, across all trimesters, and there was almost no difference in the total energy expenditure per body weight between the trimesters. Therefore, when the amount of change in the total energy expenditure in each pregnancy trimester, compared to the total energy expenditure before pregnancy (estimated energy requirement), was corrected so as to correspond to the final weight gain from pregnancy (11 kg), changes of +19 kcal/day, +77 kcal/day and +285 kcal/day were obtained for the first, second and third trimesters, respectively[148,149,155].

Energy depositions, in the form of protein and fat, corresponding to a final weight gain of 11 kg were calculated from the protein and fat depositions in each pregnancy trimester, and the sum of both was used to obtain the total energy deposition[148,149]. As a result, the energy depositions were 44 kcal/day, 167 kcal/day, and 170 kcal/day in the first, second and third trimesters, respectively.

Thus, the additional energy in each pregnancy trimester is ultimately calculated as follows.

Additional energy during pregnancy (kcal/day) = amount of change in total energy

expenditure due to pregnancy (kcal/day) + energy deposition (kcal/day)
When rounded to the closest 50 kcal, the additional energy values were calculated to be 50 kcal/day, 250 kcal/day and 450 kcal/day in the first, second and third trimesters, respectively.

4-5. Lactating Women

The estimated energy requirement of lactating women is calculated as follows:
Estimated energy requirement of lactating women (kcal/day) = estimated energy requirement before pregnancy (kcal/day) + additional energy for lactation (kcal/day)

A woman's body weight, directly after childbirth, is greater than her weight before pregnancy, and the need for the energy consumed to synthesize breast milk is a factor that increases the basal metabolic rate. However, no clear increase in basal metabolic rate is actually seen(149). In one of four studies that longitudinally examined basal metabolic rate using the doubly labeled water method, the energy used for physical activity was found to decrease significantly(151); however, in the other three studies, the difference was not significant, despite an approximate decrease of 10% in the absolute amount of energy(152,153,156). These studies showed that the total energy expenditure during the lactation period is the same as that before pregnancy(149,152,153,156), and that no specific additional amount of energy needs to be set for lactating women, from the standpoint of changes in total energy expenditure. However, total energy expenditure does not include the amount of energy required to synthesize breast milk, so lactating women need to consume the energy required for this.

The amount of energy required to synthesize breast milk is calculated as follows, assuming the lactated amount is the same as the milk volume (0.78 L/day), and the energy content of breast milk is 663 kcal/L(157–159):
Amount of energy required to synthesize breast milk (kcal/day) = 0.78 L/day × 663 kcal/L = 517 kcal/day

In contrast, energy is gained as a result of weight loss (decomposition of body tissue) after delivery (childbirth), but the required energy intake decreases that much more for it. Given an energy decrease of 6,500 kcal per 1 kg of body weight, from weight loss, and a weight loss of 0.8 kg/month(148,149), the amount of energy required can be calculated as follows:
Energy decrease from weight loss (kcal/day) = 6,500 kcal/kg body weight × 0.8 kg/month ÷ 30 days = 173 kcal/day

Therefore, if the extra energy that lactating women who experience a normal pregnancy and delivery should consume during the lactation period compared to the energy consumed before pregnancy is assumed to be the amount of additional energy needed for lactation, this amount can be calculated as follows:
Additional amount of energy for lactation (kcal/day) = amount of energy required to synthesize breast milk (kcal/day)–energy decrease from weight loss (kcal/day)
Thus, an additional 517–173 = 344 kcal/day of energy was obtained, which was rounded to 350 kcal/day.

Table appendix Estimated Energy Requirement (kcal/day)

Gender	Males			Females		
PAL [1]	I	II	III	I	II	III
0-5 months	-	550	-	-	500	-
6-8 months	-	650	-	-	600	-
9-11 months	-	700	-	-	650	-
1-2 years	-	950	-	-	900	-
3-5 years	-	1,300	-	-	1,250	-
6-7 years	1,350	1,550	1,750	1,250	1,450	1,650
8-9 years	1,600	1,850	2,100	1,500	1,700	1,900
10-11 years	1,950	2,250	2,500	1,850	2,100	2,350
12-14 years	2,300	2,600	2,900	2,150	2,400	2,700
15-17 years	2,500	2,850	3,150	2,050	2,300	2,550
18-29 years	2,300	2,650	3,050	1,650	1,950	2,200
30-49 years	2,300	2,650	3,050	1,750	2,000	2,300
50-69 years	2,100	2,450	2,800	1,650	1,900	2,200
70+ years [2]	1,850	2,200	2,500	1,500	1,750	2,000
Pregnant women (additional) [3]						
Early-stage				+50	+50	+50
Mid-stage				+250	+250	+250
Late-stage				+450	+450	+450
Lactating women (additional)				+350	+350	+350

[1] PALs (physical activity levels) of I, II, and III indicate low, medium and high activity levels, respectively.

[2] Calculated mainly from reports made on healthy independent subject persons 70-75 years old.

[3] It is important to assess the physique of individual pregnant women, weight increase during pregnancy, and fetal growth.

Note 1: On application of the present table, ensure to conduct assessment of dietary intake, measurement of the body weight and calculation of BMI. Excess energy or inadequate energy shall be evaluated according to change in body weight or BMI.

Note 2: If a subject falls under the category of PAL I, the energy intake may have to be maintained low level to match the low energy consumption level. Such subject needs to increase the level of physical activities from the prospect of health maintenance and promotion.

References

1. Prentice AM (1998) Manipulation of dietary fat and energy density and subsequent effects on substrate flux and food intake. *Am J Clin Nutr* **67**, 535S–41S.
2. Drewnowski A, Almiron-Roig E, Marmonier C, et al. (2004) Dietary energy density and body weight: Is there a relationship? *Nutr Rev* **62**, 403–413.
3. Shikany JM, Vaughan LK, Baskin ML, et al. (2010) Is dietary fat 'fattening'? a comprehensive research synthesis. *Crit Rev Food Sci Nutr* **50**, 699–715.
4. Astrup A, Grunwald GK, Melanson EL, et al. (2000) The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *Int J Obes Relat Metab Disord* **24**, 1545–52.
5. Fromentin G, Darcel N, Chaumontet C, et al. (2012) Peripheral and central mechanisms involved in the control of food intake by dietary amino acids and proteins. *Nutr Res Rev* **25**, 29–39.
6. Howarth NC, Saltzman E & Roberts SB (2001) Dietary fiber and weight regulation. *Nutr Rev* **59**, 129–39.
7. McCrory MA, Burke A & Roberts SB (2012) Dietary (sensory) variety and energy balance. *Physiol Behav* **107**, 576–83.
8. Stubbs RJ & Whybrow S (2004) Energy density, diet composition and palatability: Influences on overall food energy intake in humans. *Physiol Behav* **81**, 755–764.
9. Ello-Martin JA, Ledikwe JH & Rolls BJ (2005) The influence of food portion size and energy density on energy intake: implications for weight management. *Am J Clin Nutr* **82**, 236S–241S.
10. Karl JP, Young AJ, Rood JC, et al. (2013) Independent and combined effects of eating rate and energy density on energy intake, appetite, and gut hormones. *Obesity* **21**, 244–252.
11. Wilborn C (2011) The impact of nutrient timing considerations on weight loss and body composition. In *Nutrient Timing: Metabolic Optimization for Health, Performance, and Recovery*. pp. 237–287 [Kerksick C, editor]. Boca Raton: CRC Press.
12. Levisky D (2008) The control of food intake and the regulation of body weight in humans. In *A ppetite and food intake: behavioral and physiological considerations*. pp. 21–42 [Harris R, Mattes R, editors]. Boca Raton: CRC Press.
13. Cohen DA (2008) Neurophysiological pathways to obesity: Below awareness and beyond individual control. *Diabetes* **57**, 1768–1773.
14. Larson N & Story M (2013) A review of snacking patterns among children and adolescents: What are the implications of snacking for weight status? *Child Obes* **9**, 104–115.
15. Crespo CJ, Smit E, Troiano RP, et al. (2001) Television watching, energy intake, and obesity in US children: results from the third National Health and Nutrition

Examination Survey, 1988-1994. *Arch Pediatr Adolesc Med* **155**, 360–365.

16. Levitsky DA & Pacanowski CR (2012) Free will and the obesity epidemic. *Public Health Nutr* **15**, 126–41.
17. Eyles H, Ni Mhurchu C, Nghiem N, et al. (2012) Food pricing strategies, population diets, and non-communicable disease: a systematic review of simulation studies. *PLoS Med* **9**, e1001353.
18. Torres SJ & Nowson CA (2007) Relationship between stress, eating behavior, and obesity. *Nutrition* **23**, 887–94.
19. Dulloo AG (2010) Energy balance and body weight homeostasis. In: *Clinical obesity in adults and children.* pp. 67–81 [Kopelman PG, Caterson ID, Dietz WH, editors.] 3rd ed. Chichester: Wiley-Blackwel,.
20. Woods SC, Lutz TA, Geary N, et al. (2006) Pancreatic signals controlling food intake; insulin, glucagon and amylin. *Philos Trans R Soc B Biol Sci* **361**, 1219–1235.
21. Kadowaki T, Yamauchi T & Kubota N (2008) The physiological and pathophysiological role of adiponectin and adiponectin receptors in the peripheral tissues and CNS. *FEBS Lett* **582**, 74–80.
22. Pannain S, Beccuti G & Van Cauter E (2012) The connection between sleep loss, obesity, and type 2 diabetes. In *Sleep loss and obesity: intersecting epidemics.* pp. 133–168 [Shiromani P, Horvath T, Redline S, et al., editors]. New York: Springer.
23. King NA, Caudwell P, Hopkins M, et al. (2007) Metabolic and behavioral compensatory responses to exercise interventions: Barriers to weight loss. *Obesity* **15**, 1373–1383.
24. Blundell JE, Stubbs RJ, Hughes DA, et al. (2003) Cross talk between physical activity and appetite control: does physical activity stimulate appetite? *Proc Nutr Soc* **62**, 651–661.
25. Westerterp KR, Meijer GA, Janssen EM, et al. (1992) Long-term effect of physical activity on energy balance and body composition. *Br J Nutr* **68**, 21–30.
26. McNeil J & Doucet É (2012) Possible factors for altered energy balance across the menstrual cycle: A closer look at the severity of PMS, reward driven behaviors and leptin variations. *Eur J Obstet Gynecol Reprod Biol* **163**, 5–10.
27. Bouchard C (2007) The biological predisposition to obesity: Beyond the thrifty genotype scenario. *Int J Obes* **31**, 1337–1339.
28. Westerterp-Plantenga MS, Nieuwenhuizen A, Tomé D, et al. (2009) Dietary protein, weight loss, and weight maintenance. *Annu Rev Nutr* **29**, 21–41.
29. Levine JA, Eberhardt NL & Jensen MD (1999) Role of nonexercise activity thermogenesis in resistance to fat gain in humans. *Science* **283**, 212–214.
30. Weyer C, Walford RL, Harper IT, et al. (2000) Energy metabolism after 2 y of energy restriction: the biosphere 2 experiment. *Am J Clin Nutr* **72**, 946–53.
31. Levine JA, Vander Weg MW, Hill JO, et al. (2006) Non-exercise activity

thermogenesis: The crouching tiger hidden dragon of societal weight gain. *Arterioscler Thromb Vasc Biol* **26**, 729–736.

32. Tanaka S (2009) Methodology for evaluation of total energy expenditure. (in Japanese). *J Japanese Soc Parenter Enter Nutr* **24**, 1013–1019.
33. Brooks GA, Butte NF, Rand WM, et al. (2004) Chronicle of the Institute of Medicine physical activity recommendation: how a physical activity recommendation came to be among dietary recommendations. *Am J Clin Nutr* **79**, 921S–30S.
34. Miyake R, Tanaka S, Ohkawara K, et al. (2011) Validity of predictive equations for basal metabolic rate in Japanese adults. *J Nutr Sci Vitaminol* **57**, 224–232.
35. Kodama S, Horikawa C, Fujihara K, et al. (2012) Comparisons of the strength of associations with future type 2 diabetes risk among anthropometric obesity indicators, including waist-to-height ratio: A meta-analysis. *Am J Epidemiol* **176**, 959–969.
36. Savva SC, Lamnisos D & Kafatos AG (2013) Predicting cardiometabolic risk: Waist-to-height ratio or BMI. A meta-analysis. *Diabetes, Metab Syndr Obes Targets Ther* **6**, 403–419.
37. Donnelly JE, Blair SN, Jakicic JM, et al. (2009) Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci Sports Exerc* **41**, 459–471.
38. Saris WHM, Blair SN, Van Baak MA, et al. (2003) How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st stock conference and consensus statement. *Obes Rev* **4**, 101–114.
39. Samitz G, Egger M & Zwahlen M (2011) Domains of physical activity and all-cause mortality: Systematic review and dose-response meta-analysis of cohort studies. *Int J Epidemiol* **40**, 1382–1400.
40. Inoue M, Iso H, Yamamoto S, et al. (2008) Daily Total Physical Activity Level and Premature Death in Men and Women: Results From a Large-Scale Population-Based Cohort Study in Japan (JPHC Study). *Ann Epidemiol* **18**, 522–530.
41. Prospective Studies Collaboration, Whitlock G, Lewington S, et al. (2009) Body-mass index and cause-specific mortality in 900 000 adults: Collaborative analyses of 57 prospective studies. *Lancet* **373**, 1083–1096.
42. Berrington de Gonzalez A, Hartge P, Cerhan JR, et al. (2010) Body-mass index and mortality among 1.46 million White adults. *N Engl J Med* **363**, 2211–2219.
43. Tsugane S, Sasaki S, Tsubono Y, et al. (2002) Under- and overweight impact on mortality among middle-aged Japanese men and women : a 10-y follow-up of JPHC Study cohort I. *Int J Obes* **26**, 529–537.
44. Tamakoshi A, Yatsuya H, Lin Y, et al. (2010) BMI and all-cause mortality among Japanese older adults: Findings from the Japan collaborative cohort study. *Obesity* **18**, 362–369.
45. Sasazuki S, Inoue M, Tsuji I, et al. (2011) Body Mass Index and Mortality From All

Causes and Major Causes in Japanese: Results of a Pooled Analysis of 7 Large-Scale Cohort Studies. *J Epidemiol* **21**, 417–430.
46. Lin W-Y, Tsai S-L, Albu JB, et al. (2011) Body mass index and all-cause mortality in a large Chinese cohort. *Can Med Assoc J* **183**, E329–E336.
47. Gu D, He J, Duan X, et al. (2006) Body Weight and Mortality Among Men and Women in China. *JAMA* **295**, 776–83.
48. Jee SH, Sull JW, Park J, et al. (2006) Body-Mass Index and Mortality in Korean Men and Women. *N Engl J Med* **355**, 779–787.
49. Matsuo T, Sairenchi T, Iso H, et al. (2008) Age- and gender-specific BMI in terms of the lowest mortality in Japanese general population. *Obesity* **16**, 2348–2355.
50. Hainer V & Aldhoon-Hainerov I (2013) Obesity Paradox Does Exist. *Diabetes Care* **36 Suppl 2**, S276-81.
51. Standl E, Erbach M & Schnell O (2013) Defending the con side: Obesity paradox does not exist. *Diabetes Care* **36**, S282-6.
52. Nanri A, Mizoue T, Takahashi Y, et al. (2010) Weight change and all-cause, cancer and cardiovascular disease mortality in Japanese men and women: The Japan Public Health Center-Based Prospective Study. *Int J Obes* **34**, 348–356.
53. Wannamethee SG, Shaper AG & Lennon L (2005) Reasons for intentional weight loss, unintentional weight loss, and mortality in older men. *Arch Intern Med* **165**, 1035–1040.
54. Harrington M, Gibson S & Cottrell RC (2009) A review and meta-analysis of the effect of weight loss on all-cause mortality risk. *Nutr Res Rev* **22**, 93–108.
55. Asia Pacific Cohort Studies Collaboration, Ni Mhurchu C, Parag V, et al. (2006) Body mass index and risk of diabetes mellitus in the Asia-Pacific region. *Asia Pac J Clin Nutr* **15**, 127–33.
56. Narayan KMV, Boyle JP, Thompson TJ, et al. (2007) Effect of BMI on lifetime risk for diabetes in the U.S. *Diabetes Care* **30**, 1562–1566.
57. Yanai R, Masuda T, Kitagawa S, et al. (2006) Relationship between under and overestimation of energy intake and physical and psychological factors and lifestyle characteristics in young Japanese men and women. (in Japanese) *Kawasaki Med Welf J* **16**, 109–119.
58. Shimada M, Nishimuta M, Kodama N, et al. (2006) Existence in subjects of low plasma triiodothyronine correlated with post-absorptive resting metabolism measurement of T3 is essential for determining standard basal metabolic rate (in Japanese). *Japanese J Phys Fit Sport Med* **55**, 295–305.
59. Yokozeki T (1993) Basal metabolic rate and energy requirement of bed-ridden elderly women. (in Japanese) *J Japan Soc Nutr Food Sci* **46**, 459–466.
60. Tahara Y (1983) Seasonal variation of heat production by body composition in basal metabolic condition and cold exposure. (in Japanese) *J Japanese Soc Nutr Food Sci* **36**,

255–263.
61. Maeda T, Fukushima T, Ishibashi K, et al. (2007) Involvement of basal metabolic rate in determination of type of cold tolerance. *J Physiol Anthropol* **26**, 415–418.
62. Taguchi M, Higuchi M, Oka J, et al. (2001) Basal metabolic rate in Japanese female endurance athletes. (in Japanese) *Japanese J Nutr Diet* **59**, 127–134.
63. Sui CU, Akahashi ET, Ando YG, et al. (2007) Relationship between blood adipocytokines and resting energy expenditure in young and elderly women. *J Nutr Sci Vitaminol* **53**, 529–535.
64. Yamamura C, Tanaka S, Futami J, et al. (2003) Activity diary method for predicting energy expenditure as evaluated by a whole-body indirect human calorimeter.*J Nutr Sci Vitaminol* **49**, 262–269.
65. Ganpule AA, Tanaka S, Ishikawa-Takata K, et al. (2007) Interindividual variability in sleeping metabolic rate in Japanese subjects. *Eur J Clin Nutr* **61**, 1256–1261.
66. Hirose M (1989) Studies on the basal metabolic rates of the today's middle-aged and elder Japanese. *Ehime Med J* **8**, 192–210.
67. Hioki C & Arai M (2007) Bofutsushosan use for obesity with IGT: search for scientific basis and development of effective therapy with Kampo medicine. *J Tradit Med* **24**, 115–127.
68. Usui, C, Oka J, Yamakawa J, et al. (2003) Basal metabolic rate and its determinants in postmenopausal women. (in Japanese) *Japanese J Phys Fit Sport Med* **52**, 189–198.
69. Yokozeki T (1993) Basal metabolic rate and physical activity in the elderly. (in Japanese) *J Japanese Soc Nutr Food Sci* **46**, 451–458.
70. Kaneko K, Ito C, Koizumi K, et al. (2013) Resting energy expenditure (REE) in six- to seventeen-year-old Japanese children and adolescents. *J Nutr Sci Vitaminol* **59**, 299–309.
71. Tanaka S, Ohkawara K, Ishikawa-Takata K, et al. (2008) Accuracy of predictive equations for basal metabolic rate and contribution of abdominal fat distribution to basal metabolic rate in obese Japanese people. *Anti-Aging Med* **5**, 17–21.
72. Takahashi E, Higuchi M, Hosokawa Y, et al. (2007) Basal metabolic rate and body composition of Japanese young adult females. (in Japanese) *Japanese J Nutr Diet* **65**, 241–247.
73. Chong PKK, Jung RT, Rennie MJ, et al. (1993) Energy expenditure in lean and obese diabetic patients using the doubly labelled water method. *Diabet Med* **10**, 729–735.
74. Chong PKK, Jung RT, Rennie MJ, et al. (1995) Energy expenditure in type 2 diabetic patients on metformin and sulphonylurea therapy. *Diabet Med* **12**, 401–408.
75. Sallè A, Ryan M & Ritz P (2006) Underreporting of food intake in obese diabetic and nondiabetic patients. *Diabetes Care* **29**, 2726–2727.
76. Fontvieille AM, Lillioja S, Ferraro RT, et al. (1992) Twenty-four-hour energy expenditure in Pima Indians with type 2 (non-insulin-dependent) diabetes mellitus.

Diabetologia **35**, 753–759.

77. Bitz C, Toubro S, Larsen TM, et al. (2004) Increased 24-h energy expenditure in type 2 diabetes. *Diabetes Care* **27**, 2416–2421.
78. Bogardus C, Taskinen MR, Zawadzki J, et al. (1986) Increased resting metabolic rates in obese subjects with non-insulin- dependent diabetes mellitus and the effect of sulfonylurea therapy. *Diabetes* **35**, 1–5.
79. Nair KS, Webster J & Garrow JS (1986) Effect of impaired glucose tolerance and type II diabetes on resting metabolic rate and thermic response to a glucose meal in obese women. *Metabolism* **35**, 640–4.
80. Miyake R, Ohkawara K, Ishikawa-Takata K, et al. (2011) Obese Japanese adults with type 2 diabetes have higher basal metabolic rates than non-diabetic adults. *J Nutr Sci Vitaminol* **57**, 348–54.
81. Weyer C, Bogardus C & Pratley RE (1999) Metabolic factors contributing to increased resting metabolic rate and decreased insulin-induced thermogenesis during the development of type 2 diabetes. *Diabetes* **48**, 1607–1614.
82. Ishikawa-Takata K, Tabata I, Sasaki S, et al. (2008) Physical activity level in healthy free-living Japanese estimated by doubly labelled water method and International Physical Activity Questionnaire. *Eur J Clin Nutr* **62**, 885–891.
83. Black AE, Coward WA, Cole TJ, et al. (1996) Human energy expenditure in affluent societies: an analysis of 574 doubly-labelled water measurements. *Eur J Cinical Nutr* **50**, 72–92.
84. Ishikawa-Takata K, Naito Y, Tanaka S, et al. (2011) Use of doubly labeled water to validate a physical activity questionnaire developed for the Japanese population. *J Epidemiol* **21**, 114–121.
85. Food and Nutrition Board, Institute of Medicine. (2005) *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids*. Washington DC: National Academies Press.
86. Ohkawara K, Tanaka S, Ishikawa-Takata K, et al. (2008) Twenty-four-hour analysis of elevated energy expenditure after physical activity in a metabolic chamber: Models of daily total energy expenditure. *Am J Clin Nutr* **87**, 1268–1276.
87. Ainsworth BE, Haskell WL, Whitt MC, et al. (2000) Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sport Exerc* **32**, S498-504.
88. Rothenberg E, Bosaeus I, Lernfelt B, et al. (1998) Energy intake and expenditure: Validation of a diet history by heart rate monitoring, activity diary and doubly labeled water. *Eur J Clin Nutr* **52**, 832–8.
89. Yamada Y, Yokoyama K, Noriyasu R, et al. (2009) Light-intensity activities are important for estimating physical activity energy expenditure using uniaxial and triaxial accelerometers. *Eur J Appl Physiol* **105**, 141–152.

90. Baarends EM, Schols AM & Pannemans DL (1997) Total free living energy expenditure in patients with severe chronic obstructive pulmonary disease. *Am J Res Crit Care Med* **155**, 549–554.
91. Sawaya A, Saltzman E, Fuss P, et al. (1995) Dietary energy requirements of young and older women determined by using the doubly labeled water method. *Am J Cardiol* **62**, 338–44.
92. Reilly JJ, Lord A, Bunker VW, et al. (1993) Energy balance in healthy elderly women. *Br J Nutr* **69**, 21–7.
93. Bonnefoy M, Normand S, Pachiaudi C, et al. (2001) Simultaneous validation of ten physical activity questionnaires in older men: A doubly labeled water study. *J Am Geriatr Soc* **49**, 28–35.
94. Blanc S, Schoeller DA, Bauer D, et al. (2004) Energy requirements in the eighth decade of life. *Am J Clin Nutr* **79**, 303–310.
95. Rothenberg EM, Bosaeus IG & Steen BC (2003) Energy expenditure at age 73 and 78--a five year follow-up. *Acta Diabetol* **40 Suppl 1**, S134-8.
96. Fuller NJ, Sawyer MB, Coward WA, et al. (1996) Components of total energy expenditure in free-living elderly men (over 75 years of age): measurement, predictability and relationship to quality-of-life indices. *Br J Nutr* **75**, 161–173.
97. Colbert LH, Matthews CE, Havighurst TC, et al. (2011) Comparative validity of physical activity measures in older adults. *Med Sci Sports Exerc* **43**, 867–876.
98. Manini TM, Everhart JE, Patel KV, et al. (2009) Activity energy expenditure and mobility limitation in older adults: Differential associations by sex. *Am J Epidemiol* **169**, 1507–1516.
99. Cooper JA, Manini TM, Paton CM, et al. (2013) Longitudinal change in energy expenditure and effects on energy requirements of the elderly. *Nutr J* **12**, 73.
100. Bratteby LE, Sandhagen B, Fan H, et al. (1998) Total energy expenditure and physical activity as assessed by the doubly labeled water method in Swedish adolescents in whom energy intake was underestimated by 7-d diet records. *Am J Clin Nutr* **67**, 905–911.
101. Fontvieille AM, Harper IT, Ferraro RT, et al. (1993) Daily energy expenditure by five-year-old children, measured by doubly labeled water. *J Pediatr* **123**, 200–207.
102. Treuth MS, Butte NF & Wong WW (2000) Effects of familial predisposition to obesity on energy expenditure in multiethnic prepubertal girls. *Am J Clin Nutr* **71**, 893–900.
103. Maffeis C, Pinelli L, Zaffanello M, et al. (1995) Daily energy expenditure in free-living conditions in obese and non-obese children: comparison of doubly labelled water (2H2(18)O) method and heart-rate monitoring. *Int J Obes Relat Metab Disord* **19**, 671–7.
104. JL S, LG B, Must A, et al. (2005) Longitudinal changes in energy expenditure in girls from late childhood through midadolescence. *Am J Clin Nutr* **81**, 1102–1109.

105. Anderson SE, Bandini LG, Dietz WH, et al. (2004) Relationship between temperament, nonresting energy expenditure, body composition, and physical activity in girls. *Int J Obes Relat Metab Disord* **28**, 300–6.
106. Delany JP, Bray GA, Harsha DW, et al. (2006) Energy expenditure and substrate oxidation predict changes in body fat in children. *Am J Clin Nutr* **84**, 862–70.
107. DeLany JP, Bray GA, Harsha DW, et al. (2002) Energy expenditure in preadolescent African American and white boys and girls: the Baton Rouge Children's Study. *Am J Clin Nutr* **75**, 705–13.
108. Adachi M, Sasayama K, Hikihara Y, et al. (2007) Assessing daily physical activity in elementary school students used by accelerometer: A validation study against doubly labeled water method. (in Japanese). *Japanese J Phys Fit Sport Med* **56**, 347–356.
109. Perks SM, Roemmich JN, Sandow-Pajewski M, et al. (2000) Alterations in growth and body composition during puberty. IV. Energy intake estimated by the Youth-Adolescent Food-Frequency Questionnaire: Validation by the doubly labeled water method. *Am J Clin Nutr* **72**, 1455–1460.
110. Delany JP, Bray GA, Harsha DW, et al. (2004) Energy expenditure in African American and white boys and girls in a 2-y follow-up of the Baton Rouge Children's Study. *Am J Clin Nutr* **79**, 268–73.
111. Bandini LG, Schoeller DA & Dietz WH (1990) Energy expenditure in obese and nonobese adolescents. *Pediatr Res* **27**, 198–202.
112. Bunt JC, Salbe AD, Harper IT, et al. (2003) Weight, adiposity, and physical activity as determinants, of an insulin sensitivity index in Pima Indian children. *Diabetes Care* **26**, 2524–2530.
113. Arvidsson D, Slinde F & Hulthèn L (2005) Physical activity questionnaire for adolescents validated against doubly labelled water. *Eur J Clin Nutr* **59**, 376–83.
114. Slinde F, Arvidsson D, Sjöberg A, et al. (2003) Minnesota leisure time activity questionnaire and doubly labeled water in adolescents. *Med Sci Sports Exerc* **35**, 1923–8.
115. Ekelund U, Åman J, Yngve A, et al. (2002) Physical activity but not energy expenditure is reduced in obese adolescents: a case-control study. *Am J Clin Nutr* **76**, 935–41.
116. Eriksson B, Henriksson H, Löf M, et al. (2012) Body-composition development during early childhood and energy expenditure in response to physical activity in 1.5-y-old children. *Am J Clin Nutr* **96**, 567–573.
117. Sijtsma A, Schierbeek H, Goris AHC, et al. (2013) Validation of the TracmorD triaxial accelerometer to assess physical activity in preschool children. *Obesity* **21**, 1877–1883.
118. Corder K, Van Sluijs EMF, Wright A, et al. (2009) Is it possible to assess free-living physical activity and energy expenditure in young people by self-report? *Am J Clin Nutr* **89**, 862–870.

119. Bell KL & Davies PS (2010) Energy expenditure and physical activity of ambulatory children with cerebral palsy and of typically developing children. *Am J Clin Nutr* **92**, 313–319.
120. J Zinkel SR, Moe M, Stern EA, et al. (2013) Comparison of total energy expenditure between school and summer months. *Pediatr Obes* **8**, 404–10.
121. Bandini LG, Lividini K, Phillips SM, et al. (2013) Accuracy of Dietary Reference Intakes for determining energy requirements in girls. *Am J Clin Nutr* **98**, 700–704.
122. Butte NF, Ekelund U & Westerterp KR (2012) Assessing physical activity using wearable monitors: Measures of physical activity. *Med Sci Sports Exerc* **44**, 5–12.
123. Franks PW, Ravussin E, Hanson RL, et al. (2005) Habitual physical activity in children: the role of genes and the environment. *Am J Clin Nutr* **82**, 901–8.
124. Ishikawa-Takata K, Kaneko K, Koizumi K, et al. (2013) Comparison of physical activity energy expenditure in Japanese adolescents assessed by EW4800P triaxial accelerometry and the doubly labelled water method. *Br J Nutr* **110**, 1347–1355.
125. Foley LS, Maddison R, Rush E, et al. (2013) Doubly labeled water validation of a computerized use-of-time recall in active young people. *Metabolism* **62**, 163–169.
126. Arvidsson D, Slinde F & Hulthén L (2009) Free-living energy expenditure in children using multi-sensor activity monitors. *Clin Nutr* **28**, 305–12.
127. Hoos MB, Plasqui G, Gerver WJM, et al. (2003) Physical activity level measured by doubly labeled water and accelerometry in children. *Eur J Appl Physiol* **89**, 624–626.
128. Livingstone M, Coward W, Prentice A, et al. (1992) Daily energy expenditure in free-living children : comparison of heart-rate monitoring with the doubly labeled water (2H2(18)O) method. *Am J Clin Nutr* **56**, 343–52.
129. Dugas LR, Ebersole K, Schoeller D, et al. (2008) Very low levels of energy expenditure among pre-adolescent Mexican-American girls. *Int J Pediatr Obes* **3**, 123–6.
130. Luke A, Roizen NJ, Sutton M, et al. (1994) Energy expenditure in children with Down syndrome: Correcting metabolic rate for movement. *J Pediatr* **125**, 829–838.
131. Ramírez-Marrero FA, Smith BA, Sherman WM, et al. (2005) Comparison of methods to estimate physical activity and energy expenditure in African American children. *Int J Sports Med* **26**, 363–71.
132. Treuth M, Figueroa-Colon R, Hunter G, et al. (1998) Energy expenditure and physical fitness in overweight vs non-overweight prepubertal girls. *Int J Obes Relat Metab Disord* **22**, 440–447.
133. Butte NF, Wong WW, Hopkinson JM, et al. (2000) Energy requirements derived from total energy expenditure and energy deposition during the first 2 y of life. *Am J Clin Nutr* **72**, 1558–69.
134. Tennefors C, Coward WA, Hernell O, et al. (2003) Total energy expenditure and physical activity level in healthy young Swedish children 9 or 14 months of age. *Eur J*

Clin Nutr **57**, 647–653.

135. Davies PS, Gregory J & White A (1995) Physical activity and body fatness in pre-school children. *Int J Obes Relat Metab Disord* **19**, 6–10.
136. Atkin LM & Davies PS (2000) Diet composition and body composition in preschool children. *Am J Clin Nutr* **72**, 15–21.
137. Reilly JJ, Jackson DM, Montgomery C, et al. (2004) Total energy expenditure and physical activity in young Scottish children: Mixed longitudinal study. *Lancet* **363**, 211–212.
138. Salbe AD, Weyer C, Lindsay RS, et al. (2002) Assessing risk factors for obesity between childhood and adolescence: I. Birth weight, childhood adiposity, parental obesity, insulin, and leptin. *Pediatrics* **110**, 299–306.
139. Montgomery C, Reilly JJ, Jackson DM, et al. (2004) Relation between physical activity and energy expenditure in a representative sample of young children. *Am J Clin Nutr* **80**, 591–596.
140. Hoos MB, Gerver WJM, Kester AD, et al. (2003) Physical activity levels in children and adolescents. *Int J Obes* **27**, 605–609.
141. Tucker JM, Tucker LA, Lecheminant J, et al. (2013) Obesity increases risk of declining physical activity over time in women: A prospective cohort study. *Obesity* **21**, E715-20.
142. Park J, Ishikawa-Takata K, Tanaka S, et al. (2011) Relation of body composition to daily physical activity in free-living Japanese adult women. *Br J Nutr* **106**, 1117–1127.
143. Park J, Ishikawa-Takata K, Tanaka S, et al. (2014) The relationship of body composition to daily physical activity in free-living Japanese adult men. *Br J Nutr* **111**, 182–188.
144. Amatruda JM, Statt MC & Welle SL (1993) Total and resting energy expenditure in obese women reduced to ideal body weight. *J Clin Invest* **92**, 1236–1242.
145. Weinsier RL, Hunter GR, Zuckerman PA, et al. (2000) Energy expenditure and free-living physical activity in black and white women: comparison before and after weight loss. *Am J Clin Nutr* **71**, 1138–46.
146. Salvadori A, Fanari P, Mazza P, et al. (1992) Work capacity and cardiopulmonary adaptation of the obese subject during exercise testing. *Chest* **101**, 674–9.
147. Hulens M, Vansant G, Lysens R, et al. (2001) Exercise capacity in lean versus obese women. *Scand J Med Sci Sport* **11**, 305–309.
148. FAO. (2004) *Human energy requirements. Report of a Joint FAO/WHO/UNU Expert Consultation. FAO Food and Nutrition Technical Report Series No 1*. Rome: .
149. Butte NF & King JC (2005) Energy requirements during pregnancy and lactation. *Public Heal Nutr* **8**, 1010–1027.
150. Goldberg GR, Prentice AM, Coward WA, et al. (1993) Longitudinal assessment of energy expenditure in pregnancy by the doubly labeled water method. *Am J Clin Nutr*

57, 494–505.

151. Goldberg GR, Prentice AM, Coward WA, et al. (1991) Longitudinal assessment of the components of energy balance in well- nourished lactating women. *Am J Clin Nutr* **54**, 788–798.
152. Forsum E, Kabir N, Sadurskis A, et al. (1992) Total energy expenditure of healthy Swedish women during pregnancy and lactation. *Am J Clin Nutr* **56**, 334–42.
153. Kopp-Hoolihan LE, van Loan MD, Wong WW, et al. (1999) Longitudinal assessment of energy balance in well-nourished, pregnant women. *Am J Clin Nutr* **69**, 697–704.
154. Butte NF, Wong WW, Treuth MS, et al. (2004) Energy requirements during pregnancy based on total energy expenditure and energy deposition. *Am J Clin Nutr* **79**, 1078–87.
155. Takimoto H, Sugiyama T, Fukuoka H, et al. (2006) Maternal weight gain ranges for optimal fetal growth in Japanese women. *Int J Gynecol Obstet* **92**, 272–278.
156. Butte NF, Wong WW & Hopkinson JM (2001) Energy requirements of lactating women derived from doubly labeled water and milk energy output. *J Nutr* **131**, 53–58.
157. Suzuki K, Sasaki S, Shizawa K, et al. (2004) Milk intake by breast-fed infants before weaning. (in Japanese). *Japanese J Nutr* **62**, 369–372.
158. Hirose J, Endo M, Shibata K, et al. (2008) Amount of breast milk sucked by Japanese breast feeding infants. (in Japanese). *J Japanese Soc Breastfeed Res* **2**, 23–28.
159. Yamawaki N, Yamada M, Kan-no T, et al. (2005) Macronutrient, mineral and trace element composition of breast milk from Japanese women. *J Trace Elem Med Biol* **19**, 171–181.