Epidemiologic Merit of Obese-Years, the Combination of Degree and Duration of Obesity

Asnawi Abdullah*, Rory Wolfe, Haider Mannan, Johannes U. Stoelwinder, Christopher Stevenson, and Anna Peeters

* Correspondence to Dr. Asnawi Abdullah, Department of Biostatistics and Population Health, Faculty of Public Health, University of Muhammadiyah Aceh, Jln. Leung Bata, Batoh, Banda Aceh, Indonesia 23245 (e-mail: Asnawi_Abdullah@yahoo.com).

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This study aims to test the effect of combining the degree and the duration of obesity into a single variable—obese-years—and to examine whether obese-years is a better predictor of the risk of diabetes than simply body mass index (BMI) or duration of obesity. Of the original cohort of the Framingham Heart Study, 5,036 participants were followed up every 2 years for up to 48 years (from 1948). The variable, obese-years, was defined by multiplying for each participant the number of BMI units above 30 kg/m² by the number of years lived at that BMI. Associations with diabetes were analyzed by using time-dependent Cox proportional hazards regression models adjusted for potential confounders. The incidence of type-2 diabetes increased as the number of obese-years increased, with adjusted hazard ratios of 1.07 (95% confidence interval: 1.06, 1.09) per additional 10 obese-years. The dose-response relation between diabetes incidence and obese-years varied by sex and smoking status. The Akaike Information Criterion was lowest in the model containing obese-years compared with models containing either the degree or duration of obesity alone. A construct of obese-years is strongly associated with risk of diabetes and could be a better indicator of the health risks associated with increasing body weight than BMI or duration of obesity alone.

body mass index; duration of obesity; obese-years; obesity; total exposure; type-2 diabetes

Obesity is strongly associated with an increased risk of a number of chronic conditions, including type-2 diabetes (1, 2), cardiovascular disease (3–5), and mortality (6–9). In addition, we have recently demonstrated that the duration of obesity is a risk factor for type-2 diabetes (10) and mortality (11), independent of the level of body mass index (BMI). However, there has been no analysis of the optimal construct for capturing the health risks associated with obesity.

We hypothesized that combining the total effect of the number of years lived with obesity and the level of obesity, using a product of the 2 variables, that is, “obese-years,” would more concisely provide an alternative estimation of the association between obesity and its health outcomes. An implication of this approach is that a person with mild obesity for a long period of time would have an accumulative health risk similar to those with more severe obesity for a shorter period of time.

For smoking, the adverse effects of the combined number of cigarettes or packs smoked per day (degree) and duration have been analyzed as cigarette-years or pack-years (12–14). In contrast, the use of a similar summary measure of body weight (i.e., obese-years) on the risk of morbidity and mortality has not been investigated.

The main objective of this study was to examine the association between total exposure to obesity in terms of obese-years and the risk of type-2 diabetes. The secondary objective was to examine whether the combined variable of obese-years is a better predictor of the risk of type-2 diabetes than current BMI and/or the duration of obesity.
We investigated these objectives using a long-term prospective cohort study—the Framingham Heart Study—in which BMI, type-2 diabetes, and other covariates were measured regularly in 24 biennial examinations, spanning 48 years (15).

**MATERIALS AND METHODS**

**Data source**

We used data from the original cohort of the Framingham Heart Study (16). This cohort study followed up 5,209 participants (aged 28–62 years at the time of enrollment) for approximately 48 years from 1948 with examinations at 2-year intervals. The current study included only participants who were free from diabetes at baseline (n = 5,036).

**Measurement of variables, missing values, and imputation**

In the Framingham Heart Study, body weight, height, demographic variables, health behaviors, physiologic variables, history of illness, and the occurrence of certain chronic diseases were measured regularly, as has been described in detail previously (15). A participant was considered obese if his/her BMI was greater than or equal to 30 kg/m². The demographic and health behavior variables included in the analysis were age, educational level, country of birth, marital status, smoking status, number of cigarettes smoked per day for those who smoked, alcohol consumption (ounces/month), physical activity, and family history of diabetes. Methods of measurement of these variables have been described in detail elsewhere (10).

Most variables were measured at each of the 24 examinations. However, some values are missing. There are 2 different circumstances under which variables may be missing, as has been described elsewhere (11). The first is by design, where variables were not collected at every examination. For example, blood glucose was not measured at examinations 5, 7, and 11; current smoking status was not recorded at examinations 2, 3, 6, and 17; cholesterol was not measured at examinations 11, 12, 17–20, and 22; alcohol consumption was measured at only examinations 2, 4, 7, 19, and 20–23; and physical activity was measured only at examinations 4, 11, 12, and 19. These missing covariate values can reasonably be expected to be missing at random. For this circumstance, the nearest measured value of the covariate was used in our analysis.

In the second circumstance, the missing values occur intermittently, for example, body weight and hence BMI. During 24 examinations, 12% of the participants with missing values had only 1 missing value, 6% had 2 missing values, 5% had 3 missing values, and 3% had 4 or more missing values. In this case, missing values for BMI were imputed with a conditional mean estimated by a multiple linear regression model using age at prior examination, sex, and several transformations of the previously measured BMI (BMI, log BMI, BMI squared, and BMI as a categorical variable).

**Measurement of obese-years**

The term, “obese-years,” was used to indicate a variable combining the duration and the degree of obesity. The duration of obesity was defined by using a definition similar to that described previously (10). The duration of obesity was calculated for those individuals with at least 2 consecutive occurrences of obesity (which implies at least 2 years of being continuously obese), to avoid the potential misclassification of body weight, due to either measurement error or fluctuations between the borderline of the “overweight” BMI category and the “obese” category. For those individuals without 2 consecutive obesity occurrences, duration was considered to be zero at all examinations. For those individuals with 2 consecutive obesity occurrences, the beginning of their obesity duration interval was defined as the date of the first of these 2 examinations and, from that time, the individual was considered to be continuously obese until either the first of 2 consecutive nonobese examinations, death, or the end of follow-up at examination 24. The duration increased incrementally at each examination according to the time (in years) between the current examination and the first obesity examination. Individuals could have multiple periods of obesity duration during follow-up.

The degree of obesity was defined as follows: 1) If BMI < 30 kg/m², the degree was zero; and 2) if BMI ≥ 30 kg/m², the degree was BMI minus 29 kg/m². For example, if BMI was 35 kg/m², the degree was 6 (35 minus 29) obese units. Obese-years were calculated at each examination as the defined degree of obesity (in “obese units”) multiplied by the defined duration of obesity (in years). The cumulative number of obese-years was calculated at each examination as a sum of all obese-year “exposures” up to and including that examination.

Table 1 illustrates the calculation of an obese-years construct for a single individual. This participant first had a measurement of obesity at examination 3 and was assigned obesity duration of zero at this examination. At examination 4, this participant was assumed to have lived with obesity for 1 year (the interval between examination 3 and examination 4) with a degree of obesity of 2 kg/m². This approach assumed that an individual’s BMI was carried forward from a given examination (i.e., examination 3) and did not change until a different BMI value at a subsequent examination (i.e., examination 4). The number of obese-years at examination 4 was therefore 2 obese-years.

This method implies that those with 40 obese-years, for example, could have been obese with BMI of 30 kg/m² for 21 examinations (approximately 40 years multiplied by a degree of 1 kg/m²) or could have been obese with BMI of 34 kg/m² for 5 examinations (approximately 8 years multiplied by a degree of 5 kg/m²) or, indeed, many other combinations.

**Measurement of the outcome and time to event**

A person was defined as having type-2 diabetes if the participant had taken insulin and/or an oral hypoglycemic agent, or if an individual’s casual plasma glucose was >200 mg/dL at a given examination. This was the definition.
that the Framingham Heart Study used to categorize participants with diabetes, and it is in line with the criteria for type-2 diabetes from the World Health Organization (17). We defined the date of diabetes incidence as the midpoint between the day of the first examination when a participant was recorded as diagnosed with type-2 diabetes and the day of the previous attended examination. Individuals who died or reached the end of the follow-up (examination 24) before developing diabetes were censored at date of death or examination 24. All deaths were adjudicated by a panel of 3 investigators using previously described criteria (18).

Data analysis

To model the relation between the various measures of obesity and the risk of type-2 diabetes, we used a dynamic survival model (19), the time-dependent Cox proportional hazards regression model (20, 21). Most variables included in the model were time-varying, except for sex, age at baseline, and ethnicity.

The obese-years construct was analyzed as both a continuous and a categorical variable. The variable was grouped into intervals of 1–24.9, 25–49.9, 50–74.9, and ≥75 obese-years. Survival model parameter estimates were presented as both crude hazard ratios and multivariate-adjusted hazard ratios, for the total sample and separately by sex and age at baseline. Model 1 adjusted for age at baseline. Model 2 adjusted for the demographic variables of sex, marital status, educational level, country of birth, and age at baseline. Model 3 adjusted for the variables in model 2 and additionally adjusted for smoking status, alcohol consumption, and family history of type-2 diabetes and was considered as the primary model in this analysis. For analysis of obese-years as a continuous variable, hazard ratios were presented per 10 obese-years.

For comparing the predictive value of the degree of obesity, the duration of obesity, and the combined obese-years variable, each of these 3 variables was divided into an equal number of categories. One category for each variable was “not obese” (BMI < 30 kg/m²), and this category was used as reference. For those with obesity, 10 categories were created on the basis of deciles. The goodness of fit of competing models was compared by using the Akaike Information Criterion (AIC), computed as –2(log-likelihood) + 2 (number of estimated parameters), with a lower AIC indicating a better fit (22). All analyses were performed by using the Stata statistical software package, version 10.0 (23).

Sensitivity analyses

A sensitivity analysis was performed to examine whether the association between the duration of obesity and the risk of type-2 diabetes might be influenced by the imputation method for missing BMI; specifically, an analysis was performed that included only participants with no missing values of BMI in any examinations (n = 2,530). The effect of the duration of obesity prior to baseline was tested by performing extra analyses that excluded those who were obese at baseline.

RESULTS

Of the 5,036 eligible study participants, 75% were not obese in any of the 24 examinations. Mean BMI at baseline was 25.5 kg/m² (range, 16.2–46.3 kg/m²). For those participants who were obese for at least 2 consecutive examinations during the study follow-up (n = 1,256), the mean cumulative duration of obesity was approximately 16 years (range, 2–46 years), and the mean cumulative obese-years variable was 63 (range, 2–556). During 160,604 person-years of follow-up, 623 (12%) participants were diagnosed with type-2 diabetes (Table 2).

The incidence rates of type-2 diabetes increased with an increase in the number of obese-years. The incidence rate of type-2 diabetes per 1,000 person-years for the categories 0, 1–24.9, 25–49.9, 50–74.9, and ≥75 obese-years was 2.6, 6.0, 9.8, 13.9, and 17.6, respectively (Table 3). In the primary model (model 3), the adjusted hazard ratios of type-2 diabetes for the categories 1–24.9, 25–49.9, 50–74.9, and ≥75 obese-years were, respectively, 2.09 (95% confidence interval [CI]: 1.64, 2.67), 4.03 (95% CI: 3.04, 5.35), 4.26 (95% CI: 2.99, 6.07), and 4.94 (95% CI: 3.88, 6.28) compared with zero obese-years. A dose-response relation (across the 5 categories) was significant (P = 0.001).

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Table 1. Illustration of the Calculation of an Obese-Years Construct for a Single Hypothetical Individual

<table>
<thead>
<tr>
<th>Examination Interval Between Examinations, years</th>
<th>Body Mass Index</th>
<th>Degree of Obesity</th>
<th>Duration of Obesity, years</th>
<th>Obese-Years Lived</th>
<th>Cumulative Obese-Years</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (baseline) 0.0</td>
<td>25.5</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>2</td>
<td>25.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>3</td>
<td>31.0</td>
<td>2.0</td>
<td>2.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>4</td>
<td>32.5</td>
<td>3.5</td>
<td>3.5</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>5</td>
<td>34.0</td>
<td>5.0</td>
<td>5.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>6</td>
<td>33.5</td>
<td>4.5</td>
<td>4.5</td>
<td>0.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

a All intervals refer to the interval between the current examination and the prior examination.

b Body mass index: weight (kg)/height (m)².

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There were significant interactions between the obese-years category and sex \( (P = 0.001) \) and smoking status \( (P = 0.001) \). A dose-response was clear for males and non-smokers but not for females or smokers. For males, the risk of type-2 diabetes increased as the category of obese-years increased \( (P_{\text{trend}} = 0.001) \). A similar relation was found in nonsmokers \( (P_{\text{trend}} = 0.001) \). When obese-years was analyzed as a continuous variable, the adjusted hazard ratio
This analysis found that a new construct of obesity, obese-years, combining both degree and duration of obesity into a single variable, is a strong predictor of type-2 diabetes risk. A clear dose-response relation between obese-years and the risk of type-2 diabetes was observed. The relation was stronger for males and nonsmokers than for females and smokers. Although it is not possible to perform a statistical comparison of discrimination among the 3 models due to the fact that they are nonnested, the AIC does tell us that combining obesity and duration into a single construct provides us with more discriminative power than a model with BMI or obesity duration alone. The sensitivity analyses showed that the association between obese-years and risk of type-2 diabetes was similar between a complete case analysis and the analysis using imputed missing values for BMI. Exclusion of those who were obese at baseline produced slightly higher hazard ratios, for example, in the primary model (model 3), going from 1.07 (95% CI: 1.05, 1.09) per additional 10 obese-years (Table 3). There was also evidence of an interaction between obese-years as a continuous variable and each of sex ($P=0.001$) and smoking status ($P=0.02$).

Table 5 shows the hazard ratios for each of 3 different constructs of obesity on the risk of type-2 diabetes. Each of the degree of obesity (based on BMI), the duration of obesity, and the combination of these 2 variables into the single variable of obese-years was analyzed in a separate model, and the strength of association was compared. In addition, the AIC was compared for each of these 3 models plus a model containing the 2 variables of degree and duration of obesity as additive effects (AIC was 4,303 for males and 4,587 for females). The AIC was lowest in the model containing obese-years.

The sensitivity analyses showed that the association between obese-years and risk of type-2 diabetes was similar between a complete case analysis and the analysis using imputed missing values for BMI. Exclusion of those who were obese at baseline produced slightly higher hazard ratios, for example, in the primary model (model 3), going from 1.07 (95% CI: 1.05, 1.09) to 1.10 (95% CI: 1.08, 1.13) per additional 10 obese-years.

**DISCUSSION**

This analysis found that a new construct of obesity, obese-years, combining both degree and duration of obesity into a single variable, is a strong predictor of type-2 diabetes risk. A clear dose-response relation between obese-years and the risk of type-2 diabetes was observed. The relation was stronger for males and nonsmokers than for females and smokers. Although it is not possible to perform a statistical comparison of discrimination among the 3 models due to the fact that they are nonnested, the AIC does tell us that combining obesity and duration into a single construct provides us with more discriminative power than a model with BMI or obesity duration alone. This finding has important implications for future studies of the health impact of obesity and obesity trends. These need to take into account the impact of duration and the changes in duration of obesity.

In this analysis, it was observed that, for males, the risk of type-2 diabetes increased with additional obese-years more strongly than for females. Our previous work analyzing the association between the duration of obesity and the risk of type-2 diabetes also showed that the effect of duration of obesity was slightly higher in males than in females (10). This contrasts with the effect of the degree of obesity on the risk of type-2 diabetes, which is slightly higher in females than in males (1). The reason for this is as yet unknown and requires further investigation. Gender differences might arise from differences in body composition, insulin resistance, energy balance (24), and fat metabolism (25).

Variation in the diabetes association with increasing obese-years was also observed between smokers and nonsmokers. In most obesity studies, cigarette smoking has been shown to be a confounder or an effect modifier (9). Smokers tend to be leaner and, among smokers, weight loss appears to signal a greater amount of smoking and often ill health. In addition, smoking appears to be associated with increased abdominal fatness even without an increase in BMI (26). Generally, analysis of the effect of obesity in smokers may lead to underestimation of the exposure effect, and we have found a similar effect in the analysis of obese-years.

The strength of this study, demonstrating for the first time the potential benefit of using an obesity construct that combines both degree and duration of obesity, is its use of the Framingham Heart Study. This long-term study, with biennial measured weight and diabetes status, enabled us to analyze changes in body weight and obesity duration over a lifetime, and to take into account consequent variation in covariates. The analysis adjusted for a large number of potential confounding variables, including smoking status. However, some potential confounders, such as diet, were not collected consistently in the Framingham Heart Study and therefore have not been adjusted for in this study.
Table 4. The Hazard Ratio of Type-2 Diabetes per 10 Obese-Years or According to Obese-Years Category in the Original Framingham Heart Study That Commenced in 1948.\textsuperscript{a,b}

<table>
<thead>
<tr>
<th>Continuous obese-years\textsuperscript{b}</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>All sample</td>
<td>1.08</td>
<td>1.07, 1.10</td>
<td>1.08</td>
</tr>
<tr>
<td>Males</td>
<td>1.12</td>
<td>1.09, 1.15</td>
<td>1.12</td>
</tr>
<tr>
<td>Females</td>
<td>1.08</td>
<td>1.06, 1.10</td>
<td>1.08</td>
</tr>
<tr>
<td>Nonsmokers</td>
<td>1.10</td>
<td>1.08, 1.12</td>
<td>1.10</td>
</tr>
<tr>
<td>Smokers</td>
<td>1.07</td>
<td>1.06, 1.09</td>
<td>1.08</td>
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</table>

<table>
<thead>
<tr>
<th>Categorical obese-years</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
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<tbody>
<tr>
<td>All sample</td>
<td>1.10</td>
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<td>1.10</td>
</tr>
<tr>
<td>Males</td>
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<td>1.09, 1.14</td>
<td>1.12</td>
</tr>
<tr>
<td>Females</td>
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</tr>
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<td>1.08</td>
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<table>
<thead>
<tr>
<th>Dose-response P value</th>
<th>0.001</th>
<th>0.001</th>
<th>0.001</th>
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<tbody>
<tr>
<td>Males</td>
<td>1.10</td>
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<tr>
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</table>

Abbreviations: CI, confidence interval; HR, hazard ratio.

\textsuperscript{a} Model 1 was adjusted for age. Model 2 was adjusted for age, sex, marital status, educational level, and country of birth. Model 3 was adjusted for age, sex, marital status, educational level, country of birth, alcohol consumption, physical activity, and family history of diabetes.

\textsuperscript{b} Hazard ratios refer to the increase in type-2 diabetes risk associated with each additional 10 obese-years.

Table 5. Obese-Years Compared With Body Mass Index or Obesity Duration for Prediction of the Risk of Incident Type-2 Diabetes in the Original Framingham Heart Study That Commenced in 1948a,b

<table>
<thead>
<tr>
<th>Models</th>
<th>Males and Females</th>
<th>Males</th>
<th>Females</th>
<th>Minimum AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR</td>
<td>95% CI</td>
<td>HR</td>
<td>95% CI</td>
</tr>
<tr>
<td>Model A: body mass index</td>
<td>1.19</td>
<td>1.16, 1.21</td>
<td>1.19</td>
<td>1.15, 1.24</td>
</tr>
<tr>
<td>Model B: the duration of obesity</td>
<td>1.18</td>
<td>1.15, 1.20</td>
<td>1.18</td>
<td>1.14, 1.22</td>
</tr>
<tr>
<td>Model C: obese-years</td>
<td>1.19</td>
<td>1.17, 1.22</td>
<td>1.20</td>
<td>1.16, 1.24</td>
</tr>
</tbody>
</table>

Abbreviations: AIC, Akaike Information Criterion; CI, confidence interval; HR, hazard ratio.

a Each model analyzes the obesity exposure measure categorized into 11 categories: category 1 = “not obese,” and categories 2–11 are deciles of obesity exposure.

b Models were adjusted for sex, age at baseline, marital status, education, country of birth, smoking status, physical activity, and family history of type-2 diabetes.

The key limitation of this analysis is its use of a cohort who experienced a different environment relating to obesity in their adulthood compared with adults today. The original Framingham cohort study began in 1948, and the prevalence rates of obesity and type 2 diabetes were relatively low at that time. The prevalence of obesity in the 1950s was below 10% (27). It could be argued that the results of this study might not reflect the current population, where the prevalence of obesity is markedly higher than 50 years ago. In 2008, the prevalence of obesity among adults in the United States was approximately 30% (28). Yet the contemporary obesity epidemic is also characterized by a much earlier onset of obesity, which should result in even longer exposure time when today’s obese generation of children reach the age of our studied cohort. In our study, the average age at onset of obesity was approximately 50 years, and the average number of years lived with obesity was approximately 13 years but, in today’s society, the average age at onset of obesity is likely to be more than 10 years earlier than in previous decades (29).

To minimize the potential bias of the impact of age onset of obesity on the number of years lived with obesity in the Framingham cohort study compared with the current population, in the analysis, age at the onset of obesity was adjusted for, and the association was analyzed per additional obese-year by using time-dependent analysis. By minimizing this potential limitation, we believe the results of this study are also applicable to the current context.

In this study, the obese-years construct was based on the measurement of BMI. It was not possible to compare a construct using another anthropometric measure, such as waist circumference, as the Framingham Heart Study does not include such data. It has been argued that another anthropometric measurement, such as waist circumference, might be a better indicator of obesity and a stronger predictor of type-2 diabetes than BMI. This argument was supported by a number of studies (30, 31), but a recent meta-analysis study has found BMI, waist circumference, and the waist/hip ratio to each estimate a similar risk of type-2 diabetes (32). Consequently, it is unclear whether using an anthropometric measure other than BMI would produce different results. This hypothesis needs further analysis and testing using other similar prospective cohort studies where waist circumference was measured regularly for a long period of time.

Another potential limitation of this approach is that it does not count people with short periods of obesity as obese. In particular, in those who experience many body weight fluctuations, this will lead to an underestimation of the accumulated duration of obesity. Such misclassification would lead to an underestimation of the association of obese-years and type 2 diabetes found in this study.

The obese-years construct was calculated as a product, implying that intensity and duration have the same impact, a concept similar to pack-years in smoking studies. However, some smoking-related studies highlighted that, for some health outcomes, the effect of the duration was more important than the intensity (33), while in some others, duration was not significant after adjustment for intensity (34). The concept of obese-years needs further exploration. The notion that obese-years are “switched on” only when BMI reaches 30 kg/m² is crude; however, it does find a parallel in pack-years for smoking which is a construct that ignores any effects of passive smoking.

There are different ways to operationalize the impact of excess weight over time. This study confirms that analyzing the combined effect of the duration and the severity of obesity as a predictor of the risk of type-2 diabetes is worthwhile. Such approaches add information over and above simply the degree of excess weight or length of time with obesity. Other potential further additions could be aspects of body weight trajectory, such as rate of weight gain over time or degree of weight fluctuation over time (35, 36).

Our results demonstrate that it is important to take into account both the degree and duration of increasing body weight when analyzing the association between obesity and the risk of type-2 diabetes. This effect is likely to be because account is taken of the cumulative damage of obesity on body systems and, consequently, is likely to be
applicable to other chronic diseases. However, it is recommended that further investigations be undertaken of other possible intensity/duration constructs of obesity, such as considering the effect of overweight and of the relations between obese-years and other chronic diseases and mortality.

In conclusion, the combination of the degree and the duration of obesity, referred to as the variable, “obese-years,” yields a strong predictor of the risk of type-2 diabetes and a more effective explanation of diabetes incidence than does BMI or duration of obesity alone. It will be important to take into account both the duration of obesity and the levels of BMI attained when estimating the population health burden of current obesity trends.

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Author affiliations: Department of Biostatistics and Population Health, Faculty of Public Health, University Muhammadiyah Aceh, Banda Aceh, Indonesia (Asnawi Abdullah); and Department of Epidemiology and Preventive Medicine, School of Public Health and Preventive Medicine, Monash University, Melbourne, Victoria, Australia (Rory Wolfe, Haider Mannan, Johannes U. Stoelwinder, Christopher Stevenson, Anna Peeters).

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REFERENCES