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# Consumption of energy-dense, nutrient-poor foods and hypertension in obese children

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## Abstract

***Background*** Obesity has become an increasingly important medical problem in children. Obesity-induced hypertension in childhood should be considered as a chronic medical condition that is likely to require long-term management of dietary patterns, especially for energy-dense, nutrient-poor (EDNP) food consumption.

***Objective*** To examine the contribution of EDNP foods to daily energy and macronutrient intakes and to examine the relationship between intake of EDNP foods and the prevalence of hypertension in children.

***Methods*** Four hundred and forty children were randomly selected to participate. Weight and height were measured with precision electronic scales and fixed microtoise, respectively. Blood pressure (BP) measurements were performed by standard procedure. A 24-hour dietary recall was obtained by a trained interviewer to determine the intake of EDNP foods, which were classified to 4 major groups: visible fat, sweeteners, desserts, or salty snacks. The difference in mean was evaluated using paired T-test. Logistic models were fitted to assess for an association between hypertension and the various characteristics.

***Results*** The proportion of children who were overweight (including obese) was 23.2%. The prevalence of elevated BP was 10.5%, similar in boys and girls, with most of them having isolated elevated systolic BP. There was a relationship between BP and body mass index (BMI) in all children. Approximately 27% of total daily energy intake was contributed by all EDNP foods. Of the EDNP food subgroups examined, dessert and sweeteners contributed nearly 20% of total daily energy intake. In the highest one-third of subjects who consumed EDNP food , these foods provided 49% of total daily carbohydrate intake and 34% of total daily fat intake.

***Conclusion*** Eating patterns of EDNP foods provide 49% of total daily carbohydrate intake and 34% of total daily fat intake. This EDNP food is independently associated with hypertension in children. **[Paediatr Indones. 2014;54:236-44.]**.


***Keywords:*** *energy dense nutrient poor foods, obesity hypertension, children*

Obesity has become an increasingly important medical problem in children and adolescents. Once considered rare, primary hypertension in children has become increasingly common in association with obesity and other risk factors, including a family history of hypertension predisposing to hypertensive disease.[1,2] Obese children are at an approximately 3-fold higher risk for hypertension than non-obese children. In addition, the risk of hypertension in children increases across the entire range of body mass index (BMI) values and is not defined by a simple threshold effect. As in adults, a combination of factors, including overactivity of the sympathetic nervous system (SNS), insulin resistance, and abnormalities in vascular structure and function may contribute to obesity-related hypertension in children. The benefits of weight loss for blood pressure reduction in children have been demonstrated in both observational and interventional studies. Obesity-related hypertension in childhood should be considered as a chronic medical condition that is

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likely to require long-term management. Ultimately, prevention of obesity and its complications, including hypertension, is the goal, by managing dietary patterns, especially the consumption of energy-dense, nutrient-poor (EDNP) foods.[3,4]

The rising rate of obesity in the world has been linked to the growing consumption of refined grains, added sugars, and added fats. These refined grains, fats, and sweets are inexpensive, palatable, and convenient. However, they can also be energy-dense and sometimes poor in vitamins, minerals, and other micronutrients. The World Health Organization has found sufficient evidence to link high consumption of energy-dense foods to the global obesity epidemic.[5-10]

Concerns that the American diet has become energy-rich but nutrient-poor have been expressed for ≥3 decades. Energy-dense sweets and fats have long been contrasted, unfavourably, to foods that contained substantial amounts of key nutrition per serving or per unit weight. The terms “energy-dense” and “nutrient-poor” are commonly used to characterize foods perceived as unhealthy and to distinguish them from more nutritious options.[8,10-12] Disparaging terms such as “junk food” or “empty calories” are commonly used in antithesis to such descriptions as “healthful,” “packed with nutrients,” “nutrient-dense,” or “nutrient-rich.”[12,13]

The prevailing dietary guidance for reducing the risk of chronic disease promotes consumption of fruits, vegetables, and whole grains while limiting energy and fat intakes. Therefore, the dietary guidance communicated to the public has advocated reduced consumption of EDNP foods containing fats, oils and sugars. These foods constitute the tip of the food guide pyramid. Data from the second *National Health and Nutrition Examination Survey* (NHANES II) showed that EDNP foods rich in fat, oil and sugar provided one-third of the total daily energy intake for adult Americans.[2,4] In the period since NHANES II, public awareness of diet and health issues has increased and an enormous variety of fat- and sugar-modified food products have appeared in the marketplace, resulting in speculation that the intake of EDNP foods may have declined. However, no recent information is available on consumption patterns for EDNP foods.[3,4]

The purpose of this study was to examine the contribution of EDNP foods to daily energy and macronutrient intakes and to examine the relationship between the intake of EDNP foods and the prevalence of hypertension in children.

## Methods

The study took place in Semarang from July to November 2012. Using a cross-sectional design, 440 children of the ninth grade of the public school were selected by cluster random sampling to participate. This study was approved by the Research Ethics Committee of the University of Diponegoro. Measurements were performed in a quiet setting by a trained clinical officer. Subjects’ weights and heights were measured with precision electronic scales (to the nearest 0.1 kg) and a fixed microtoise (to the nearest 0.1 cm). At each visit, BP and heart rate measurements were taken on the subject’s right arm at 1-minute intervals, after a rest of at least 3 minutes, in a seated position. Readings were obtained with a clinically validated oscillometric device. Each device was checked for accuracy by comparing BP values obtained with a mercury sphygmomanometer using a Y tube connected to the automated device.

Elevated BP was defined according to American reference data, which are generally considered the best available standard. Measurements were performed by the school nurses, who had been trained to perform the standardized BP measurement methods, using the same BP measurement devices.[3.4]

Parents were asked to complete structured questionnaires about their educational level and obesity status. Parents’ educational level was based on the highest degree completed by either the mother or the father (grouped into primary, secondary, or tertiary education). Parents were considered to have hypertension if they reported ever having been told by a physician or a health professional that they had elevated BP.

Subjects’ BMIs were calculated as weight divided by height-squared (kg/m$^2$). Body mass index (BMI) status was classified as not overweight, overweight, or obese and defined as BMI < 85$^{th}$ percentile, BMI 85$^{th}$ to 94$^{th}$ percentile, and BMI $\geq$ 95$^{th}$ percentile, respectively. Based on these US percentiles, we also calculated BMI subcategories (percentiles <25, 25-74, 75-84, 85- 94, and ≥ 95). In addition, we categorized overweight and obese along the sex-specific and age-specific BMI criteria of the *International Obesity Task Force*, for the sake of comparison with studies that have used these criteria.[14]

Elevated BP was defined as systolic or diastolic BP equal to or greater than the 95th sex-specific percentile (or a sex-specific, age-specific and height-specific distribution of BP Z-score ≥ 1.64). Sex-specific and age-specific percentiles of height, which are needed to assess elevated BP, were similarly derived from the *Centers for Disease Control and Prevention* (CDC) growth charts. Children were considered to have hypertension if they had elevated BP based on the average of the last two of the three BP readings, which were obtained at each visit.

A 24-hour dietary recall was obtained by a trained dietary interviewer. The type and amount of each food consumed was recalled with the help of food models, charts, measuring cups, and rules. Interviewers asked probing questions to prompt subjects' recall of commonly forgotten items, such as condiments, accompaniments, fast foods and beverages.[9,14]

To determine the intake of EDNP foods, it was necessary to distinguish foods that belong in this category from other foods reported during the 24-hour dietary recalls. As a first step, the foods reported by subjects were classified as belonging to ≥ of the 5 major food groups (i.e., dairy, fruit, grains, meat/beans or vegetables). Briefly, the assignment of foods into the various groups was dependent of their nutrient content and use in the diet. The EDNP foods were further subcategorized into 4 groups as follows: 1) visible fat (e.g., butter, margarine, oil, dressing, and gravy); 2) sweeteners (e.g., sugar, syrup, candy, and carbonated or noncarbonated sweetened drinks); 3) dessert (e.g., baked goods such as cookies, cakes, pies, and pastries or dairy desserts such as ice cream, pudding, and cheesecake); 4) salty snacks (e.g., potato, corn, and tortilla chips).[8,10,15]

The prevalence and standard error were estimated for boys and girls. The differences in mean BP or mean heart rate were evaluated using a paired T-test or analysis of variance. Logistic models were fitted to assess for an association between hypertension and the various characteristics, i.e., BMI categories and tertiles of heart rate, as well as parental educational level and history of hypertension. We calculated the proportion of hypertension in children that could be attributed to overweight or obesity (CDC criteria).[16,17]

The mean percentage of the daily energy from EDNP food groups (separately and combined) was adjusted for age and sex. The percentage of energy from EDNP foods was categorized into tertiles on the basis of the distribution of this variable in the analytic sample. Mean intakes of energy, macronutrients, and micronutrients by tertiles of the percentage of energy from EDNP foods were obtained after adjusting for age and sex. All statistical analyses were performed by using *SPSS version 1.7*.

The independent associations of EDNP food intake to nutrient intake were examined by regression procedures to adjust for multiple covariates. Linear regression procedures were used for continuous outcome variables. For categorical outcomes, such as meeting the standard for a nutrient intake, logistic regression procedures were used.

## Results

**Table 1** shows selected characteristics of the subjects. Overweight was found in 24.2% (0.7) of boys and 22.2% (0.6) of girls. (The overweight category includes the obese subjects).

Overweight (including obesity; CDC criteria) was inversely associated with parents educational level. The proportions of children who were overweight (including obesity) were 24.3% (1.8), 98% (0.7) for parents with primary, secondary and tertiary as highest educational levels, respectively (P<0.001). The mean heart rates increased with increasing BMI categories: 82.7 beats per min (0.2), 83.4 beats per min (0.5) and 84.2 beats per min (0.9) in lean, overweight (not obese), and obese children, respectively (trend, P=0.048).

**Table 2** shows the prevalence of elevated BP: 10.5% of all subjects, similar between boys and girls. Among children with elevated BP, most had isolated systolic elevated BP. Very few children had elevated diastolic BP.

The contributions to daily energy of all EDNP foods and the EDNP subgroups, by sex and age categories are shown **Table 3.** Approximately 27% of total daily energy intake was contributed by all EDNP foods. Of the EDNP food subgroups examined, dessert and sweeteners contributed nearly 20% of total daily energy intake. In **Table 4**, the energy and micronutrient

**Table 1.** Characteristics of the subjects

| Characteristics | Male n=231 | Female n=209 | Total n=440 |
|---|---|---|---|
| Mean age (SD), years | 11.8 (0.4) | 11.7 (0.4) | 11.8 (0.4) |
| Range | 9.8-13.2 | 9.7-13.1 | 9.7-13.2 |
| Mean parent's education, % (SE) | | | |
| Tertiary | 30.2 (0.8) | 30.1 (0.8) | 30.2 (0.8) |
| Secondary | 58.8 (1.0) | 57.9 (1.1) | 58.5 (1.0) |
| Primary | 11.0 (0.7) | 12.0 (0.7) | 11.8 (0.7) |
| Mean parent's with obesity, % (SE) | | | |
| None | 61.0 (0.9) | 59.8 (0.8) | 60.4 (0.8) |
| Father | 16.8 (0.7) | 1.4 (0.7) | 16.6 (0.7) |
| Mother | 17.2 (0.6) | 17.0 (0.5) | 17.1 (0.5) |
| Both | 5.0 (0.4) | 6.8 (0.5) | 5.9 (0.4) |
| Mean BMI, % (SE) | | | |
| No excess weight (<85$^{th}$ percentile) | 75.0 (0.7) | 79.9 (0.7) | 76.8 (0.7) |
| Overweight (85$^{th}$ to 94$^{th}$ percentile) | 18.1 (0.6) | 16.3 (0.5) | 1.2 (0.5) |
| Obese ($\geq$95$^{th}$ percentile) | 6.1 (0.4) | 5.9 (0.4) | 6.0 (0.4) |

**Table 2.** Mean BP and prevalence of elevated BP

| Gender | n | Mean systolic BP (SD), mmHg | Mean diastolic BP (SD), mmHg | Elevated systolic BP (SE), % | Elevated diastolic BP (SE), % | Elevated BP (SE), % |
|---|---|---|---|---|---|---|
| Male | 231 | 113.0 (10.6) | 68.7 (7.1) | 9.9 (0.6) | 0.8 (0.2) | 10.4 (0.4) |
| Female | 209 | 112.8 (9.2) | 69.2 (7.3) | 10.2 (0.7) | 0.9 (0.2) | 10.6 (0.5) |
| All | 440 | 112.9 (9.8) | 68.9 (7.2) | 10.1 (0.6) | 0.8 (0.2) | 10.5 (0.4) |

**Table 3**. Percentage of total daily energy intake from EDNP food and the EDNP subgroups by sex and age

| | All EDNP foods | Desserts | Sweeteners | Salty snacks | Visible fat |
|---|---|---|---|---|---|
| All subjects | 24.8 $\pm$ 0.30 | 85 $\pm$ 0.13 | 4.2 $\pm$ 0.16 | 10.2 $\pm$ 0.25 | 2.8 $\pm$ 0.10 |
| Gender | | | | | |
| Male | 25.2 $\pm$ 0.40 | 8.7 $\pm$ 0.20 | 4.4 $\pm$ 0.25 | 10.0 $\pm$ 0.20 | 2.6 $\pm$ 0.11 |
| Female | 24.6 $\pm$ 0.35 | 8.4 $\pm$ 0.16 | 4.0 $\pm$ 0.20 | 10.6 $\pm$ 0.17 | 3.0 $\pm$ 0.10 |
| Age by groups | | | | | |
| 9-10 yrs | 20.5 $\pm$ 0.42 | 8.0 $\pm$ 0.16 | 4.0 $\pm$ 0.18 | 10.0 $\pm$ 0.20 | 2.6 $\pm$ 0.10 |
| 11-12 yrs | 24.7 $\pm$ 0.41 | 8.4 $\pm$ 0.14 | 4.2 $\pm$ 0.20 | 10.3 $\pm$ 0.18 | 2.8 $\pm$ 0.10 |
| 13-14 yrs | 28.6 $\pm$ 0.36 | 8.7 $\pm$ 0.15 | 4.4 $\pm$ 0.18 | 10.5 $\pm$ 0.20 | 2.9 $\pm$ 0.12 |

contributions of EDNP foods for each tertile of percentage of daily energy from EDNP foods are shown. In the highest one-third of EDNP food consumption, these foods provided 49% of total daily carbohydrate intake and 34% of total daily fat intake.

**Table 5** shows the univariate and multivariate associations between hypertension and selected characteristics of the children and parents. Hypertension was significantly associated with children's overweight/obese BMI status and higher heart rate. The mean heart rate was 82.7 beats per min (0.2) in normotensive children and 90.3 beats per min (1.3) in hypertensive children ($P<0.001$). Children's hypertension was significantly associated with parents' history of hypertension (in particular, paternal history of hypertension). The proportions of all children with hypertension that were overweight or obese BMI status (CDC criteria) were 37% overall (95%CI 24 to 48), 40% (95%CI 26 to 52) in boys, and 34% (95%CI 21 to 44) in girls.

**Table 4.** Total daily energy, macro nutrient, fiber, sodium, and food group intakes by tertile of percentage of EDNP foods

| Outcome.var | Percentage of daily energy from EDNP foods | | |
|---|---|---|---|
| | 1st tertile (0 – 16.5%) 78% (n = 146) | 2nd tertile (16.6 – 31.3%) 24.0% (n = 147) | 3rd tertile (>31.3%) 40.8% (n = 147) |
| Energy, kJ | 7,812 ± 105 | 9,070 ± 110 | 9,204 ± 101 |
| Carbohydrate, % of E | 44 ± 0.4 | 46 ± 0.4 | 49 ± 0.3 |
| Protein, % of E | 17 ± 0.1 | 14 ± 0.1 | 11 ± 0.1 |
| Fat, % of E | 30 ± 0.4 | 33 ± 0.3 | 34 ± 0.3 |
| Saturated, % of E | 9 ± 1.2 | 11 ± 1.2 | 12 ± 1.1 |
| Fiber, g | 18 ± 0.3 | 16 ± 0.2 | 15 ± 0.1 |
| Sodium, mg | 2,608 ± 280 | 3,110 ± 305 | 3,370 ± 330 |

E=energy

**Table 5.** Factors associated with hypertension in children

| Variables | | Normal BP n | Hyper-tension n | Crude-odds ratio (95% CI) | P value |
|---|---|---|---|---|---|
| BMI categories | - No excess weight | 338 | 3 | 1 | |
| | - Overweight | 75 | 8 | 2.6 (1.5 – 4.6) | <0.01 |
| | - Obese | 27 | 33 | 11.8 (7.2 – 18.2) | <0.01 |
| Heart rate categories | - <77.5 per min | 150 | 18 | 1 | |
| | - 77.5–86.9 per min | 146 | 10 | 1.2 (0.5 – 2.1) | 0.08 |
| | - ≥87 per min | 144 | 26 | 3.1 (1.4 – 7.2) | <0.01 |
| Parents' educational level | - Tertiary | 132 | 14 | 1 | |
| | - Secondary | 257 | 23 | 1.1 (0.8 – 2.0) | 0.22 |
| | - Primary | 51 | 7 | 1.5 (0.8 – 3.0) | 0.11 |
| Parents with hypertension | - None | 264 | 24 | 1 | |
| | - Father | 704 | 13 | 2.4 (1.7 – 4.4) | <0.01 |
| | - Mother | 76 | 5 | 2.7 (1.5 – 4.9) | <0.01 |
| | - Both | 26 | 2 | 2.9 (1.2 – 8.0) | <0.05 |
| EDNP food consumption | - Low | 102 | 2 | 1 | |
| | - Moderate | 208 | 10 | 3.2 (1.4 – 4.8) | <0.01 |
| | - High | 130 | 32 | 12.0 (7.0– 18.1) | <0.01 |

## Discussion

The prevalence of hypertension was 10.5% in our subjects. Most children with hypertension had isolated systolic hypertension (ISH). Hypertension was associated with children's excess weight, children's increased heart rate, and parent's history of hypertension, but not with parent's educational level. One-third of all children with hypertension had excess body weight.

The study had some limitations. First, BP was not reassessed in children with normal BP. As such, the prevalence of hypertension may have been underestimated if some of the children had low BP at the initial visit but generally high BP (i.e., masked hypertension). Only a few studies have assessed the prevalence of masked hypertension in children. Masked hypertension could be a precursor of persistent hypertension and is more frequent among overweight youths, therefore, it may be a potential issue in the context of the worldwide obesity epidemic. The prevalence of overweight/obesity may, therefore, may have been underestimated, and thus the population attributable fraction of hypertension related to excess weight may have been correspondingly underestimated. The population attributable fraction assumes causality between exposure (overweight) and outcome (hypertension). This cross-sectional study does not provide a direct argument for this causation, but reverse causation is unlikely. Furthermore, our estimates have been adjusted for several potentially confounding variables.[1,4,15]

More than 10% of hypertensive children had ISH. This finding was consistent with higher prevalence of elevated ISH than elevated diastolic BP as reported in most other surveys. In Houston, Sorof *et al*. found that 88% of hypertensive children had ISH. In adults, ISH is usually linked to increasing arterial stiffness with age. In children, sympathetic nervous system hyperactivity leads to a hyperdynamic hemodynamic state and may contribute to the pathogenesis of ISH: obese children with isolated systolic BP had increased heart rate and BP variability. We also found that obese children had a higher resting heart rate and that children with hypertension had a higher resting heart rate, independent of BMI. [1,4,15,17]

This association between obesity and hypertension in children has been reported in numerous studies among a variety of ethnic groups, with virtually all studies finding higher blood pressure and/or higher prevalence of hypertension in obese compared with lean children. The most comprehensive study pooled data from 8 large US epidemiological studies involving over 47,000 children to describe blood pressure in relation to body size.[1,4,17] Irrespective of gender or age, the risk of elevated blood pressure was significantly higher for children in the upper compared with the lower decile of BMI, with an odds ratio of systolic hypertension ranging from 2.5 to 3.7. Overweight children in the Bogalusa Heart Study were 4.5 and 2.4 times as likely to have elevated systolic blood pressure and diastolic blood pressure, respectively. Sorof *et al*.[17] reported a 3-times greater prevalence of hypertension in obese than in non-obese adolescents in a school-based hypertension and obesity screening study.

The early clinical course of obesity-related hypertension appears to be characterized by a preponderance of isolated systolic hypertension (systolic hypertension without diastolic hypertension). Data from a recent multicenter trial of an antihypertensive medication in children showed that from 140 subjects who enrolled in the trial, 37% had isolated systolic hypertension alone. The prevalence of isolated systolic hypertension was 50% in obese subjects compared with 30% in nonobese subjects. In the school-based screening for hypertension and obesity by Sorof *et al*.,[17] the prevalence of isolated systolic hypertension among adolescents who were obese and had blood pressure above the 95$^{th}$ percentile on a single set of measurements, was 94%. Because isolated systolic hypertension has been shown to be a major risk factor for cardiovascular morbidity and mortality in adults, further investigation of the causes and interventions for this pattern in children is clearly needed.[14,16,18]

Sorof *et al*.[17] found an increased prevalence of systolic hypertension (based on a single set of measurements) as BMI percentile increased from the 5$^{th}$ to the 95$^{th}$ percentile. Among all demographic and clinical factors analyzed, BMI was most strongly associated with hypertension. Blood pressure is a continuous variable that is positively correlated with cardiovascular risk across the entire blood pressure range. For example, studies of normotensive and hypertensive children have reported that blood pressure and left ventricular mass index are positively associated across a wide range of blood pressure values.[21,17] Furthermore, elevated left ventricular mass may be present even in children whose blood pressure values fall within the so-called "normotensive" range. Although a child's current blood pressure may fall within the population-based range of normality, a previously undetected patterns of relative increases in blood pressure across percentiles over time may still effectively render that patient "hypertensive".[22] This is consistent with the observation that children with high normal blood pressure during adolescence have a greater tendency to develop hypertension during adulthood.[19]

Although the majority of data on the pathophysiology of obesity hypertension are derived from studies of animals and adults, the mechanisms of obesity-related hypertension have been studied in children as well. Most studies in children have focused on the investigation of three main pathophysiological mechanisms: disturbances in autonomic function, insulin resistance and abnormalities in vascular structure and function. Although obesity-induced hypertension is likely due to an overlap or combination of these factors, systemic review of the data consistent with each mechanism is useful for understanding how they may contribute to the early stages of the disease process in children.[2,20]

The link between obesity and hypertension may be mediated in part by sympathetic nervous system

(SNS) hyperactivity. This state of hyperactivity may include cardiovascular manifestations such as increased heart rate and blood pressure variability, neurohumoral manifestations such as increased levels of plasma catecholamines, and neural manifestations such as increased peripheral sympathetic nerve traffic. Consistent with the SNS hyperactivity hypothesis, the Bogalosa Heart Study reported that, in a biracial group of children, resting heart rate was positively correlated with blood pressure and subcapsular skinfold thickness. Also, a hyperdynamic cardiovascular state was positively associated with several measures of obesity. Similarly, Sorof *et al.*[17] reported from a school-based screening for obesity and hypertension that obese hypertensive adolescents had the highest resting heart rate and nonobese normotensive adolescents had the lowest heart rate. When the analysis was restricted to only those who were hypertensive, a higher heart rate was observed in the obese compared to nonobese adolescents. Rocchini *et al.*[3] found that weight loss, with or without exercise, resulted in a significant reduction in heart rate in obese adolescents.

Increased heart rate variability, blood pressure variability altered balance between parasympathetic and sympathetic activity exclusively to increased sympathetic activity. Insulin resistance has been implicated in the pathogenesis of obesity-related hypertension in children. Several studies have reported positive associations between fasting insulin levels and resting blood pressure in obese children and young adults. Nonetheless, this association does not necessarily indicate causation. Chiolero *et al.*[15] studied 350 obese children who were categorized as hypertensive or normotensive. Although insulin levels were significantly higher in hypertensive than in normotensive children, the difference was not clinically relevant. Furthermore, insulin explained only a small amount of systolic and diastolic blood pressure variance, which disappeared after accounting for the confounding effects of age, weight, or other anthropometric dimensions. Weight loss in obese adolescents has also been shown to result in reductions in serum insulin levels and blood pressure,[16-18] and to render previously salt-sensitive individuals insensitive to the hypertensive effect of salt-loading. Based on these data, it has been suggested that the insulin resistance associated with obesity may prevent insulin-induced glucose uptake but leave the renal sodium retention effects of insulin relatively preserved, thereby resulting in chronic volume overload and maintenance of blood pressure elevation. How altered vascular structured and function may also contribute to the pathogenesis of obesity hypertension.

Since there is a higher prevalence of elevated BP among overweight children, a screening strategy could be limited to those who are overweight. However, weight reduction, the primary therapeutic goal for obesity-related hypertension, is advised in obese children irrespective of BP level. As such, screening for hypertension among obese children might further stigmatize these children.

Most interventions for pediatric obesity have focused of behavioral approaches to diet and physical activity to address the main components of energy balance. Dietary counseling suggests hypocaloric dietary modifications for a 3-week diet and exercise program. Although these studies suggest that blood pressure reduction is induced by weight loss in obese in children, each is limited by the absence of a matched control group to show that the blood pressure reduction was directly attributable to weight loss. Nonetheless, the methods used to achieve weight management remain controversial. It is also appropriate to reserve such therapy for those who have failed or have had only modest success with behavioral therapy directed at dietary modification and increased physical activity. The presence of ongoing obesity-related outcomes such as hypertension, diabetes mellitus or impaired glucose tolerance and dyslipidemia may increase the rationale for more aggressive therapy. Ultimately, multiple therapeutic strategies may be necessary to achieve the desired goal.[8,10]

Obesity in childhood should be considered as a chronic medical condition and, thus, likely to require long-term treatment. Public health initiatives to educate community leaders and health care providers may prove instrumental in stemming the evolving epidemic of pediatric obesity and its complications. In addition, the scope and acuity of the problem facing our youth suggest that substantial research is needed to focus on the mechanisms of hypertension related to obesity in the pediatric population. Such research will serve as the basis for future guidelines for prevention and treatment of obesity-related hypertension.[10,13]

Throughout history an organism adapted for a situation in which food was unlimited and confronted an environment in which palatable energy-dense foods are obtained easily with minimal physical activity. Increased modernization and a Western diet and lifestyle are associated with an increased prevalence of overweight in many developing countries. This sometimes has been referred to as the nutrition transition or part of a transition to modernity. Clearly, individual behaviors along with social, cultural, and environmental factors must also play important roles. It is likely that a gene-environment interaction, in which genetically-susceptible individuals respond to an environment with increased availability of palatable, energy-dense foods and reduced opportunities for energy expenditure, contribute to our current high prevalence of obesity.[13,16]

Our results showed that energy-dense foods with relatively modest nutrient contents provided about 27% of the total daily energy intake of children, with one-third of the population consuming an average of 45% of energy from EDNP foods. EDNP foods play an important role in the Indonesian diet. The estimates of EDNP food consumption reported in this study were based on the approach in which whole foods were examined for their energy and nutrient contribution to the diet. The implications of high EDNP food consumption are threefold. The first implication is that greater EDNP food intake increases the risk of marginal nutrient intake. These findings are similar to those reported for NHANES II. The second implication of EDNP food intake is its positive association with energy intake. The increasing adiposity of the population has been recognized as a public health issue, and it is probable that highly palatable EDNP foods play a role in promoting positive energy balance. The third implication of EDNP food intake is that higher consumption of EDNP foods was linked to decreased likelihood of compliance with current dietary guidance for risk reduction. EDNP food consumption was inversely related to the odds of consuming 34% of energy from fat, including 12% of energy from saturated fat, or eating foods from all 5 major food groups. Although the percentage of energy from carbohydrates increased with increasing EDNP food intake, dietary fiber intake did not. Instead, these variables were inversely related. Therefore, higher carbohydrate intake in association with EDNP food intake most likely reflects the addition of refined sugar and is also inconsistent with dietary recommendations.[16,21,22]

The suggested strategy to counter the high degree of consumer preference for EDNP foods includes the need for development of simpler and clearer guidelines about the intake of such foods in conjunction with healthful-eating parents. In particular, a joint study of EDNP food intake and consumption of foods from the major food groups, is also needed.

In conclusion, our study suggests that eating patterns of EDNP food are independently associated with obesity-related hypertension in children, and EDNP foods tend to substitute for, rather than supplement, the more nutrient-dense foods in the Indonesian diet. This pattern leads to increased risk of the following: 1) high energy intake, 2) marginal micronutrient intakes, and 3) poor compliance with current nutrient- and food group-related dietary guidance, resulting in obesity-related hypertension. New strategies are needed to educate consumers on how to moderate their intake of EDNP foods.

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