Obesity as the sequel of childhood stunting: Ghrelin and GHSR Gene Polymorphism explained / Harry F. L. Muhammad

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Abstrak

ABSTRACT
Stunting or short stature in children is a significant nutritional problem in developing and underdeveloped countries. Stunting during childhood might affect brain development and impair development cognitive function. Additionally, this condition associated with the increased risk for obesity during adulthood. Several studies have shown that the increment risk of obesity and overweight in children with a short stature was due to their metabolic efficiency. Children with stunting have lower resting energy expenditure compared to non stunting children. Additionally, stunted children has higher respiratory quotient and carbohydrate oxidation but lower fat oxidation compared to non-stunting children. These results might explain why stunted children easily become obese, which is due to lower fat oxidation and leading to tendency to store fat.

This review discussed the current status on studies in the nutrigenetic aspects of the relationship between stunting in the childhood and obesity in adulthood. I hypothesized that stunted children are more likely to become obese in their later life because they have lower metabolic rate and higher tendency of fat storage. There are several candidate genes and pathway involved in obesity and I suspected that ghrelin and its receptor growth hormone secretague receptor (GHSR) were responsible.