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Is Control of Obesity Hidden in Catecholamine Metabolizing Enzyme Renalase?

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Editorial

Obesity is an underlying situation for both cardiovascular diseases and metabolic diseases such as diabetes, and it is a serious public health problem with an increasing incidence worldwide [1,2]. When genetic factors are eliminated, one of the important causes of obesity becomes uncontrolled appetite [2]. Control of appetite is possible with the cooperative work between many appetizing (*orexigenic*, or appetite stimulant) and non-appetizing (anorexigenic) peptide molecules. As an example, leptin molecule is non-appetizing, whereas ghrelin molecule is appetizing [3]. Impaired balance between these molecules would lead to obesity or puniness. Studies have demonstrated low levels of ghrelin and high levels of leptin hormones in obese individuals. The underlying reason of low appetizing hormone level in the circulation among obese is to restrict food intake via decreasing appetite [4]. Excluding leptin resistance, the mechanism suggested for ghrelin for decreased appetite is the opposite that is suggested for leptin. However, control of appetite is not dependent to these two hormones only. Appetite is controlled by hypothalamus [5]. That means it is balance by both the central nervous system (molecules such as neuropeptide-Y or dopamine) and many appetizing (such as galanine or ghrelin) and non-appetizing (such as nesfatin-1, leptin, insulin or amylin) molecules synthesized by peripheral organs [4,6,7]. In this editorial paper, the relationship between dopamine, which plays a role in the control of appetite [6], and the enzyme renalase that catabolizes dopamine [8], as well as possible effects of the levels of these molecules on obesity, were investigated. Low levels of dopamine have been reported in obese and craving individuals. Dopamine is synthesized naturally in the brain and mediates the transmission of electrical impulses. Dopamine deficiency has been related to factors such as poor nutrition (tyrosine deficiency), stress, insomnia or antidepressant intake [6]. Additionally, excessive amount of renalase enzyme which metabolizes dopamine and other catecholamines (epinephrine and norepinephrine) might be counted as a more effective factor than the factors listed above. In the presence of excessive renalase, a larger amount of dopamine will be catabolized in unit time which will result in

dopamine deficiency. Renalase is commonly synthesized in the kidneys, whereas synthesis from various biological tissues including brain has been reported [9-11]. Deficient dopamine in the brain may be due to excessively synthesized renalase (hypertrophy of renalase synthesizing cells). Renalase is a 3.8 kDa molecule and is large to pass the blood-brain barrier [11]. Therefore, it has been suggested that excessive amount of peripheral renalase would not contribute to dopamine catabolization at an important extent. If dopamine was reduced in relation to excessive peripheral renalase in the brain, all obese individuals would be expected to have hypotension. Because renalase catabolizes dopamine, epinephrine and catecholamines [9]. However, no such situation is observed in obese individuals; in the contrary, they are generally hypertensive.

Dopamine deficiency observed via the mechanism mentioned above (excessive amount of renalase) would reduce the feeling of pleasure and the emotions of the individuals may rapidly change from positive to negative [6]. In order to remove this depressive mood and lack of energy, they may be addicted to excitants and especially caffeine. A desire for caffeine exceeding the self-control of the individual may be a sign of dopamine deficiency. In case of dopamine deficiency, with any cause, the individual would experience situations such as reduced motivation, unwillingness or loss of libido. Increased appetite and subsequent consumption of alcohol and saturated fats are observed, and a rapid gaining of weight and obesity may take place. By partial reduction of renalase in the brain (operation of this site of the brain) dopamine catabolism may be prevented and the symptoms mentioned above may be removed. One day in the future it may be possible to administrate L-DOPA in order to increase dopamine synthesis, which would in turn suppress appetite [11]. Moreover, surgical removal of the cerebral region that accelerates dopamine metabolism in order to provide weight control, which may be an alternative to gastric operations. As a conclusion, it has been proposed that obesity may be avoided by loss of appetite through the control of renalase metabolism observed as a result of the reduction in dopamine levels.

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