Human Obesity Management, Pathways and Therapeutics
Beyond Metabolic Limitation

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Abstract

Obesity is a prevalence metabolic phenotype caused by a manifestation of gene-environmental interactions. Despite a series of preventive and therapeutic options, diverse therapeutic outcomes are still presence in different backgrounds of obese people. A small proportion of obesity persons are ineffective by lifestyle and therapeutic convention (metabolic homeostasis controls and limitations). In order to improve obese treatments in future, further genetic and molecular basis of pathological and therapeutic exploration is required, especially in the area of host genetic/molecular abnormality.

Keywords: Obesity; Endocrinology; Human Genome; Inflammatory Factors; Neural Disorder; Mental Disorder; Obese Treatment; Metabolic Disease

Backgrounds

Epidemiological condition and current limitations

Obesity is a prevalence metabolic and physiological disorder caused by a sequence of host-environmental interactions. Obese people commonly face a lot of personal trouble in the society and a number of pathophysiologic damages [1-6]. Many types of preventive and therapeutic options have been widely sought after. However, most of these medications are not predictable-mostly item of metabolic targeting-energy imbalance and glucose homeostasis disorder. Since different patterns of therapeutic interventions by metabolic limitation are often temporary and commonly bounce back [7], a number of genetic/molecular exploration should be emphasized.

Molecular basis for human obesity

Different types of counteractive measures are suitable for different individuals. Apart from life-style and energy limitation, cellular and molecular etiologic/pathological mechanism study may be other ways for obese therapeutics in patients resistance to energy control.

Possible pathological targets

• Pathologic factorials (endocrinological factors)-leptin, thyroxine, insulin and many other hormonal dysfunction.
• Brain-visual-appetite axis (hypothalamic).
• Psychiatric burden and disorder.
• Drug-induced (hormonal drugs, antibiotics or other drugs associated with human liver dysfunction).

• Inflammatory factors (TNF secretion).
• Tumor-induced (pituitary tumors and others).
• Physiological change (adipose cells or tissues).
• Genetic alleles and loci (loss-of-function or copy number changes of key genes and molecules) [8-21].

Therapeutic combination

Therapeutic combinations (drugs plus life-style) are widely recommended for obese patients over the past two decades. These kinds of therapeutic paradigms are very useful for many other chronic diseases, such as HIV/AIDS and neoplasm metastasis [22-27]. These therapeutic paradigms may be lend into anti-obese therapeutics-combination of metabolic limitation and pathological molecules. Nonetheless, these therapeutic systems are usually based on doctor’s experience rather than scientific-supportive formats.

Conclusion

Human obesity is a strong risk factor for human morbidity and mortality. New insights into human obese molecules may help human beings with overweight problem. After these genetic/molecular study, all obese people can be fully targeted. We need to promote these researches in the future.

Conflict of Interests

None.

Bibliography


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