

## Human Obesity Therapeutics, Modern Diagnosis and Biomarkers

Da-Yong Lu<sup>1\*</sup>, Jin-Yu Che<sup>1</sup>, Ying Shen<sup>2</sup> and Bin Xu<sup>3</sup>

<sup>1</sup>Shanghai University, Shanghai, China

<sup>2</sup>Medical School, Shanghai Jiao-Tong University, China

<sup>3</sup>Shanghai Institute of Materia Medica, Chinese Academy of Sciences, China

**\*Corresponding Author:** Da-Yong Lu, Shanghai University, Shanghai, China.

**Received:** July 23, 2019; **Published:** August 26, 2019

### Abstract

Obesity is a prevalence metabolic phenotype caused by abnormal metabolic homeostasis and gene-environmental interactions. A small proportion of obesity persons are ineffective by lifestyle and current therapeutics. Genetic and molecular basis of disease diagnosis is required to improve targeted therapy against genetic/molecular abnormality in humans.

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

### Backgrounds

Obesity is a prevalence metabolic and physiological disorder (30 - 35% in common adult worldwide) caused by a sequence of host-environmental interactions [1-6]. Many types of preventive and therapeutic options have been widely sought after. However, most of these medications (life-style-food limitation or high-load of human exercise)-energy imbalance and glucose homeostasis disorder strategies are not always work [7], a number of genetic/molecular exploration should be emphasized in the future.

### New therapeutic convention

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. Following pathways may be new initiatives for obese therapeutics:

- 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].

### Future Directions

To achieve targeted therapeutics for genetic/molecular abnormality, individual therapies and new drug development may be important [22]. Combinations (drugs plus life-style) are widely recommended for obese patients, which are very useful for many other chronic diseases, such as HIV/AIDS and neoplasm metastasis [23-28]. Nonetheless, these therapeutic systems are usually based on doctor's experience rather than scientific-supportive formats. Therapeutic paradigms for genetic/molecular abnormality needs modern diagnosis [29-37] and personalized medicine [38-41]. To achieve better obesity treatments, new drug development is also very useful [42-45].

### Conclusion

Human obesity is a strong risk factor for human morbidity and mortality. Modern genetic/molecular diagnosis in the clinic is indispensable. After these genetic/molecular study, all obese people can be fully controlled.

### Conflict of Interests

None.

### Bibliography

1. World Health Organization. "WHO, Obesity and overweight" (2018).
2. Lu DY, et al. "Obesity, risks and managements". *Metabolomics* 8.1 (2018): e155.
3. Lu DY, et al. "An overview of obesity". *Metabolomics* 8.2 (2018): 200.
4. Jainta N, et al. "Infection diseases and vaccination in patients with diabetes". *EC Diabetes and Metabolic Research* 3.3 (2019): 91-97.
5. Lu DY, et al. "Pathology and treatments of obesity". *Trends in Medicine* 8.5 (2018): 157.
6. Lu DY, et al. "Obese study, keep up the momentum". *International Journal of Endocrinology and Metabolism* 1.1 (2018): 4-8.
7. Brestoff JJR and Artis D. "Immune regulation of metabolic homeostasis in health and disease". *Cell* 161.1 (2015): 146-160.
8. Yanai H. "VLDL is the leading actor in lipid abnormality in patients with diabetes and obesity". *Journal of Endocrinology and Metabolism* 7.4 (2017): 101-102.
9. Steculorum SM, et al. "Hypothalamic UDP increases in obesity and promotes feeding via P2Y6-dependent activation of AgRP neurons". *Cell* 162.6 (2015): 1404-1417.
10. Lee YS, et al. "Increased adipocyte O2 consumption triggers HIF-1 $\alpha$ , causing inflammation and insulin resistance in obesity". *Cell* 157.6 (2014): 1339-1352.
11. Quarta C, et al. "Epigenetic ON/OFF switches for obesity". *Cell* 164.3 (2016): 341-342.
12. Dalgaard K, et al. "Trim28 haploinsufficiency triggers bi-stable epigenetic obesity". *Cell* 164.3 (2016): 353-364.
13. Lu DY, et al. "Mini-review of obesity, etiology progresses and different therapeutics". *EC Diabetes and Metabolic Research* 3.3 (2019): 98-102.
14. Lu DY, et al. "Human obesity, pathological and therapeutic advances". *EC Pharmacology and Toxicology* 7.4 (2019): 231-238.
15. Singh A, et al. "Protective role of Terminalia Chebula in streptozotocin-induced diabetic mice for wound healing activity". *British Journal of Medicine and Medical Research* 22.2 (2017): 1-8.

16. Smith RE., *et al.* "Dietary carbohydrates that modulate the immune system". *Clinical Immunology, Endocrine and Metabolic Drugs* 2.1 (2015): 35-42.
17. Nzuza S., *et al.* "Highly active antiretroviral therapy-associated metabolic syndrome and lipodystrophy: pathophysiology and current therapeutic interventions". *Journal of Endocrinology and Metabolism* 7.4 (2017): 103-116.
18. Correa-Giannella ML and Machado UF. "SLC2A4 gene: a promising target for pharmacogenomics of insulin resistance". *Pharmacogenomics* 14.8 (2013): 847-850.
19. Bretteld C., *et al.* "Micro RNAs responsible for inflammation in obesity". *Journal of Endocrinology and Metabolism* 7.3 (2017): 77-85.
20. Van der Klaauw AA and Farooqi IS. "The hunger genes: pathways to obesity". *Cell* 161.1 (2015): 119-132.
21. Putta S., *et al.* "Diabetes mellitus and male aging, pharmacotherapeutics and clinical implications". *Current Pharmaceutical Design* 23.30 (2017): 4475-4483.
22. Lu DY., *et al.* "Human obesity management, pathways and therapeutics beyond metabolic limitation". *EC Diabetes and Metabolic Research* 3.4 (2019): 106-108.
23. Lu DY., *et al.* "Drug combinations in cancer treatment". *Clinical Experimental Pharmacology* 3.4 (2013): 134.
24. Lu DY., *et al.* "HAART in HIV/AIDS treatments, future trends". *Infectious Disorders-Drug Targets* 18.1 (2018): 15-22.
25. Lu DY., *et al.* "HIV/AIDS curable study, new forms of therapeutic trinity". *Recent Patents on Anti-Infective Drug Discovery* 13.3 (2018): 217-227.
26. Lu DY., *et al.* "Drug combination in clinical cancer treatment". *Reviews on Recent Clinical Trials* 12.3 (2017): 202-211.
27. Lu DY., *et al.* "Anticancer drug combination, how far we can go through?" *Anti-Cancer Agents in Medicinal Chemistry* 17.1 (2017): 21-28.
28. Lu DY., *et al.* "Anticancer drug combinations, studies from different pathways". *Cell and Developmental Biology* 4.3 (2015): 166.
29. Lu DY., *et al.* "Cancer bioinformatics, its impacts on cancer therapy". *Metabolomics* 5.2 (2015): e133.
30. Ocana A and Pandiella A. "Personalized therapies in the cancer "omics" era". *Molecular Cancer* 9 (2010): 202.
31. Stransky B and Galante P. "Application of bioinformatics in cancer research". *An Omics Perspective on Cancer Research* (2010): 211-233.
32. Putta S and Kilari EK. "A review on methods of estimation of advanced glycation end products". *World Journal of Pharmaceutical Research* 4.9 (2015): 689-699.
33. Lu DY., *et al.* "Cancer bioinformatics for update anticancer drug developments and personalized therapeutics". *Reviews on Recent Clinical Trials* 12.2 (2017): 101-110.
34. Garg PK. "Potential of molecular imaging to advance molecular medicine". *Cancer Studies and Molecular Medicine – Open Journal* 3.1 (2017): e3-e4.
35. De Macedo JE. "Knowledge of the molecular signaling pathways improves the chances of treatment of gastro-intestinal stromal tumors". *Cancer Studies and Molecular Medicine* 2.1 (2015): 69-71.

36. Meyer UA. "Pharmacogenetics—five decades of therapeutic lessons from genetic diversity". *Nature Reviews Genetics* 5.9 (2004): 669-676.
37. Lu DY, et al. "Pharmacogenetics of cancer therapy: breakthroughs from beyond?". *Future Science OA* 1.4 (2015): 15-80.
38. Lu DY, et al. "Individualized cancer chemotherapy integrating drug sensitivity tests, pathological profile analysis and computational coordination-an effective strategy to improve clinical treatment". *Medical Hypotheses* 66.1 (2006): 45-51.
39. Lu DY. "Personalized cancer chemotherapy, an effective way for enhancing outcomes in clinics". Woodhead Publishing, Elsevier, UK (2014).
40. Lu DY, et al. "Individualized cancer therapy, what is the next generation?" *EC Cancer* 2.6 (2018): 286-297.
41. Lu DY, et al. "Individualized cancer therapy, future approaches". *Current Pharmacogenomics and Personalized Medicine* 16.2 (2018): 156-163.
42. Putta S, et al. "Anthocyanins: Possible role as multitarget therapeutic agents for prevention and therapy of chronic diseases". *Current Pharmaceutical Design* 23.41 (2017): 6321-6346.
43. Lu DY, et al. "Type 2 diabetes study, introduction and perspective". *The Open Diabetes Journal* 8 (2018): 13-21.
44. Lu DY, et al. "Type 2 diabetes treatment and drug development study". *The Open Diabetes Journal* 8 (2018): 22-33.
45. Lu DY, et al. "Keep up the pace of drug development evolution and expenditure". *Cancer Research Review* 2.5 (2018): 1-8.

**Volume 7 Issue 9 September 2019**

**©All rights reserved by Da-Yong Lu, et al.**