Introduction

Mounting evidence indicates metabolic disorders are a major public health burden in the world. Medical cost has gradually increased due to a lack of diagnostics and efficacy of medications focused on targeting a molecule. Metabolic disorders are known as one of the largest obesity epidemics and associated with health-related problems such as cardiovascular disease, diabetes, hypertension and hyperlipidemias in the US. Obesity related to an imbalance of metabolism from environmental stressors impacting metabolic rate, reflects the multidimensional molecular network. Obesity is also associated with the genetic predisposition of a build-up of adipose tissue dynamics, which is unable to properly undergo lipolysis and breakdown at the tissue level, or it can be a hormonal issue, where the patient is producing too much ghrelin or diminishing supply of leptin. Omics, a detection platform for macromolecules (i.e., DNA, RNA, and protein level), can be developed for use in many different types of illnesses based on the pathophysiology behind the ailment or disease, including obesity. Personalized care focused on molecule assessment can help decrease the need for synthetic insulin, increase the body’s own ability to use it’s already producing pancreatic beta cells of insulin, and decrease the likelihood of other comorbidities from progressing by adapting part of Omics metabolomics. Metabolomics is more advantageous than determining the pathologic structure of molecular behavior in ghrelin and leptin. In the future, metabolomics has strong potential to be considered as an alternative preventive tool to fight against obesity, hyperlipidemia, or secondary health complications, including cardiovascular and cancer mortality.
metabolomics, with a platform using NMR spectroscopy in C57BL/6 following glucagon–like peptide-1 receptor agonists (GLP-1RAs), liraglutide treatment comparison with dipeptidyl peptidase-4 (DPP-4) inhibitor (vildagliptin) as animal mice model for T2D and Obesity. They found that metabolic profiling unfolds from decreased levels of 2-PY (N1-methyl-2-pyridone–5–carboxamide) and 4-PY (N1-methyl-4-pyridone–3–carboxamide) in liraglutide treatment for two weeks. The finding suggests that both 2-PY and 4-PY as an end product of nicotinamide adenine dinucleotide metabolism could be associated with diabetic metabolism, compared to another compensatory metabolic cascade such as tryptophan metabolism, phenylalanine and tyrosine metabolism, gut microbiota metabolism, insulin related metabolism, adipose-derived stem cell metabolism, and cysteine metabolism [9].

Impact of omics approach on metabolic disorder

Obesity related to an imbalance of metabolism from environmental stressors impacting metabolic rate, reflects the multi-dimensional molecular network. Obesity is also associated with the genetic predisposition of a build-up of adipose tissue dynamics, which is unable to properly undergo lipolysis and breakdown at the tissue level, or it can be a hormonal issue, where the patient is producing too much ghrelin or diminishing supply of leptin. Ghrelin is a hormone that is produced to encourage appetite by increasing parasympathetic tone and shunting blood towards the digestive tract in preparation for metabolic processing of the food. Ghrelin increases hunger, while leptin increases the feeling of satiety. In addition, the axis of immune and neuronal circuits plays a role to regulate metabolic disorders. Molecular features of neural networks in the brain regulate hormonal signals from a build-up of blood sugars and other metabolic by-products depending on the type of nutrients under the microenvironment niche. Because it could be very hard to properly manage obesity at a hormonal level, focus will not be on that aspect. Patients who are more obese are more likely to have other comorbidities like improper systemic circulation thus that will impair the ability for this type of omics to effectively work. Therefore, metabolomics is more advantageous than determining the pathologic structure of molecular behavior in ghrelin and leptin. Also, it can apply preventive benefits using microbiome behavioral interaction with adipose status, including an aspect of proliferation and differentiation by monitoring molecular-based cell imaging and by comparison detail between metabolites inside and outside cells using in adipose-derived stem cells or diet-induced obesity mice model with biomarker, a stable obestatin analog (PEG–OB(Cys10, Cys13)) [10,11].

In the future, metabolomics has strong potential to be considered as an alternative preventive tool to fight against food addiction like obesity, hyperlipidemia, or secondary health complications, including cardiovascular, hypertension, and cancer mortality which could be preventive by detecting circulating glycoprotein N–acyctyl glucosamine residues in urine or blood [12]. Using an advanced detection system coupled with multi-dimensional omics under the systemic biology platform, metabolic disorders are able to mitigate and manipulate the impact of imbalance in the molecular level and its phenotypic
consequence through the animal model by victualing the difference of metabolic features and imaging pattern of reward circuit prior to translational medicine, creating new functional food or replenishing its already natural products to greatly increase the patient’s overall quality of life.

References


