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Review Article

Sensor of molecular imbalance in metabolic disorder: Determination of molecular behavior wired in disease utilizing metabolomics

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Abstract

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.

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. There are several different sources of metabolic disorders that exist, such as thyroid hormonal imbalances and unregulated cholesterol levels, and the specific types of these metabolic disorders can directly affect possible treatment methods. The type of diabetes a person has, such as a type 1 or type 2, will dictate a specific treatment algorithm. Metabolic diseases are not usually caused by any direct act, but rather arise due to a plethora of issues that can make a person or group of people more susceptible to certain diseases. Environmental factors often influence disease development within populations, and this concept is known as the sociological construct of food deserts [1]. Additionally, access to healthcare, the means to be able to go to a healthier supermarket that sells more organic, or less processed foods, or have education necessary to understand why eating the right types of foods, are important when considering disease causes. Physicians and healthcare providers can use many advanced detection methods and assessment skills in order to determine causation and to better patient outcomes. For example, healthcare providers can use nutrigenomics, which looks at the interaction between inherited factors like genes and how that can affect nutrition

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in regards to the disease. Metabolomics serve as an analytical tool to look at the possible metabolites byproduct within an organism to help digest food and other products. Detection for metabolic endpoints combined with analytic tools (i.e., quadrupole time-of-flight mass spectrometry (UHPLC-Q-TOF-MS)) is available in urine or in simple blood tests which can also be used to predict disease progress or patterns using biological markers: glycated hemoglobin (HbA1C) levels and C-peptide in diabetics, Chem 20, troponin, CMB-K in cardiac conditions, hormonal levels of TSH, thyroxine, and iodine for thyroid conditions [2,3].

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. For instance, proteomics can be used for diabetic treatments, metabolomics can be used to help fight against obesity, and genomics can be used for cardiovascular injury or disease. Each one of these diseases works on different sub-structures or uses macromolecules to communicate with the cells or surrounding tissue. In diabetics, the main hormone that is not being used appropriately is insulin. Insulin is a small peptide hormone that is secreted by pancreatic beta cells when it senses high blood glucose levels. Molecular structure such as the difference between configuration and conformation or allosteric structure, stress-driven imbalance of hormonal secretion, and interaction with environmental factors like chemical ingredients in food, could change the sensitivity or binding activity of insulin and its receptor. Currently, precision medicine using selective medications has proved the difference between drug sensitivity to target, for example insulin receptor and/or insulin, in organ and binding efficacy such as functional aspects rooted from blueprint of genomics study.

A pattern of molecular behavior changes during onset of disease in personal care

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. Furthermore, utilizing MALDI-TOF-MS and LC-MS/MS analysis followed by target-specific detection technologies like proteomics and metabolomics, can potentially decrease the harmful effects of systemic pharmaceutical interventions, and help reduce any unnecessary risks and improve a patient's overall health. There are a plethora of different proteomic materials both at the macro and macromolecular levels, including CKD273 (urinary peptide) and albuminemia in diabetes, actinin-2, desmin (cytoskeletal structure), the chaperone function of TCP-1 in obesity and diabetes type 2, Galectin-1 in type 2 diabetes(T2D), Platelet basic proteins/ C1 inhibitor in type 1 diabetes(T1D), and Tamm-Horsfall urinary the glycoprotein, apolipoprotein A-1, apolipoprotein E, alpha2-thiol proteinase inhibitor, CD 59, and another glycol -and retinol-binding proteins [4-8].

In a previous study, there was evidence that different metabolic pathways could be associated with obesity utilized

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-methyland 4-PY (N1-methyl-4-2-pyridone-5-carboxamide) 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, adiposederived 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 cells 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-acetyl 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

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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

- Cooksey-Stowers K, Schwartz MB, Brownell KD (2017) Food Swamps Predict Obesity Rates Better Than Food Deserts in the United States. Int J Environ Res Public Health 14: 1366. Link: https://bit.ly/2CIpFdN
- Koehn J, Krapfenbauer K (2010) Advanced proteomics procedure as a detection tool for predictive screening in type 2 pre-Diabetes. EPMA J 1: 19-31. Link: https://bit.ly/3eFrtkX
- Men L, Pi Z, Zhou Y, Wei M, Liu Y, et al. (2017) Urine metabolomics of high-fat diet induced obesity using UHPLC-Q-TOF-MS. J Pharm Biomed Anal 132: 258-266. Link: https://bit.ly/3ezo0o0
- Lindhardt M, Persson F, Oxlund C, Jacobsen IA, Zürbig P, et al. (2018) Predicting albuminuria response to spironolactone treatment with urinary proteomics in patients with type 2 diabetes and hypertension. Nephrol Dial Transplant 33: 296-303. Link: https://bit.ly/30ma6R4
- Hwang H, Bowen BP, Lefort N, Flynn CR, De Filippis EA, et al. (2010) Proteomics analysis of human skeletal muscle reveals novel abnormalities in obesity and type 2 diabetes. Diabetes 59: 33-42. Link: https://bit.ly/3hd6qYB

- Fryk E, Sundelin JP, Strindberg L, Pereira MJ, Federici M, et al. (2016) Microdialysis and proteomics of subcutaneous interstitial fluid reveals increased galectin-1 in type 2 diabetes patients. Metabolism 65: 998-1006. Link: https://bit.ly/3eEXWYw
- Zhang Q, Fillmore TL, Schepmoes AA, Clauss TR, Gritsenko MA, et al. (2013) Serum proteomics reveals systemic dysregulation of innate immunity in type 1 diabetes. J Exp Med 210: 191-203. Link: https://bit.ly/396uSrX
- Soggiu A, Piras C, Bonizzi L, Hussein HA, Pisanu S, et al. (2012) A discoveryphase urine proteomics investigation in type 1 diabetes. Acta Diabetol 49: 453-464. Link: https://bit.ly/3hdCvQ8
- Pražienková V, Holubová M, Pelantová H, Bugáňová M, Pirník Z, et al. (2017) Impact of novel palmitoylated prolactin-releasing peptide analogs on metabolic changes in mice with diet-induced obesity. PLoS One 12: e0183449. Link: https://bit.ly/2ZGq617
- Mastrangelo A, Panadero MI, Pérez LM, Gálvez BG, García A, et al. (2016) New insight on obesity and adipose-derived stem cells using comprehensive metabolomics. Biochem J 473: 2187-203. Link: https://bit.ly/2CNxajm
- 11. Cowan E, Kumar P, Burch KJ, Grieve DJ, Green BD, et al. (2016) Treatment of lean and diet-induced obesity (DIO) mice with a novel stable obestatin analogue alters plasma metabolite levels as detected by untargeted LC-MS metabolomics. Metabolomics 12: 124. Link: https://bit.ly/2WA3QUO
- Lawler PR, Akinkuolie AO, Chandler PD, Moorthy MV, Vandenburgh MJ, et al. (2016) Circulating N-Linked Glycoprotein Acetyls and Longitudinal Mortality Risk. Circ Res 118: 1106-1115. Link: https://bit.ly/2DLGbtN

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