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Physics and chemical links of obesity to human diseases: The role of hyperbarism

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The abnormal accumulation of fatty acids within and around the peritoneal organs increases physics pressure at both organ and cellular levels. Sensing and responding these physic stimuli, cellular synthesis and/or secretion of a variety of chemical mediators are altered, by which diabetes, renal diseases, coronary artery disease, adipose liver, and preeclampsia are developed or augmented. The cure of preeclampsia by delivery and the substantial cure of type-2 diabetes by bariatric surgery can be considered as *in vivo* human studies, which suggest that physic pressure on abdominal organs arising from the obesity is the cause that results in functional and/or structural injuries of the abdominal organs. Exposed to the increased abdominal pressure in obese individuals, the increased pressure works as a cause, interacting with the susceptible factors of various genotypes, to alter the expression of susceptible genes and other functional genes required for homeostasis. The cellular illness featured by the endophenotypes of abnormal autocrine, paracrine, endocrine, and exocrine of chemical mediators. Either altered chemical mediators, or responses to these chemicals, eventually lead to the variety of exophenotypes of divergent types of diseases in obese individuals. For all clinical complications of the abdominal hyperbarism syndrome, obesity is an environmental factor and is necessitated to initiate and trigger pathological processes. Based on the severity and the number of organs/systems involved, abdominal hyperbarism syndromes are clinically divergent in some individuals only have obesity with one abdominal organ dysfunction, others with 2, and severe patients with more than 2 organs including gastrointestinal cancers.

Biography

Andy Zhang began his biomedical research at Genotheramics Inc., San Diego and is a visiting student of Soochow University. He is interested in identifying the mechanisms by which environmental factors act on gene regulation. Currently, he is focusing on the effects of chronically increased abdominal pressure on the development of human diseases. His other research interests include the more efficient interference of gene regulation through gene editing technology. He has been involved in the development of a TAL effector library and now participates in the development of assays for off-targeting analysis of engineered nucleases.

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