

The Bridge between Obesity and Non-Communicable Diseases Strengthened By Oxidative Stress and Inflammation: Unconventional Systematic Measures Needed To Abate These Health Disorders

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Editorial

The abatement of the non-communicable age-related diseases (NCDs) such as heart disease, dyslipidaemia, stroke, type 2 diabetes and fatty liver diseases is a global challenge assigned high priority by the World Health Organization. Tobacco smoking, physical inactivity and the revolution in food industry, resulting in obesity, are established risk factors for many NCDs. The pathogenesis of NCDs is complex. Moreover, each chronic illness of NCD type can't be considered in isolation as they share common, usually related risk factors. This observation indicates that integrated strategies can be effective for many different conditions.

One common factor that initiates or hastens progression of almost all NCDs is a systemic low-grade inflammation. Both chronic inflammation and reactive oxygen species (ROS) are also key features of ageing. In overweight and obese populations, the subclinical inflammation propagates by the excessive adipocytic production of the pro-inflammatory cytokines compounded by the infiltration of adipose by pro-inflammatory macrophages. In turn, the cytokine production by adipocytes and macrophages contributes to obesity-related insulin resistance, thus, propagating the greatest vicious circle of NCDs.

The conventional ways to manage chronic diseases are disease-specific and usually aimed at the target organs. There is a critical need to develop an intervention strategy for suppressing the root and the confounding factor for most NCDs; a vicious circle propagated by systemic low-grade inflammation. In our review we describe and promote examples and challenges for the standard "one disease-one treatment" paradigm by discussing the developing of an integral strategy for the prevention or delay of NCD onset by counteracting low-grade inflammation at the site of its origin: the visceral adipose.

From the recent discovery that the melanin biosynthesis pathway is functional in adipose, and the well-known anti-oxidative and anti-inflammatory properties of melanin, this biological phenomena worth exploration as one of the non-standard approach. This review argues the idea of further investigations for adipose ectopic melanogenesis. This is needed for providing a proof of principle for the therapeutic potential use of small molecule stimulators targeting melanogenesis in adipose and thereby abating the systemic inflammation and consequently, delaying the development of NCDs in an aging population.

The approach we discussed to systemic inflammation abatement is non-standard in more than one way.

First, instead of aiming at relatively short-lived effects typical for conventional drug therapy, we support strategy to generate long-term changes within the target organ as melanin is essentially insoluble and, once synthesized, cannot be removed from the visceral adipose compartment. Extracellular melanin will continue to exert its beneficial effects through inactivation of ROS even after the death of a particular melanin-synthesizing adipocyte.

Second, this approach may lead to a dramatic shift in the paradigm of NCDs research. So far, the efforts towards reducing healthcare costs have been focused largely on lifestyle modification, e.g. weight loss and cessation of smoking. While these are the desired outcomes, in most cases they are not immediate and in a significant number of people remain simply unobtainable. Our suggested approach may lead to the development of a preventive medication that will allow the postponement of the development of NCDs in predisposed populations and, consequently, a decrease of lifestyle-associated healthcare burden.