Role of Inflammation in Obesity and Diabetes

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the adipocytes have a real commanding role in the insulin mechanism so it clears the link between obesity and diabetes.

Immune system activation in adipose tissue during obesity

Adipocytes store excessive nutrient load and progressively become hypertrophic. Cell hypertrophy leads to a pro-inflammatory response mainly through hypoxia and endoplasmic reticulum (ER) stress-related mechanisms. Eventually, this may lead to adipocyte death. Furthermore, stressed adipocytes produce a wide range of cytokines and chemokine's, including TNF-α, that in turn promote immune cell accumulation and activation in adipose tissue. Therein, numerous macrophages create a local pro-inflammatory loop with adipocytes. Other immune cells, such as T cells, might also contribute to inflammation. In parallel, circulating FFAs and mLDL particles may directly bind to TLR2 and TLR4, inducing NF-κB activation and production of various pro-inflammatory factors including pro-IL-1β. In the meantime, hyperglycaemia promotes the activation of the NLRP3 inflammasome through the binding of TXNIP in macrophages. Lipid species such as ceramides may directly activate the inflammasome. The NLRP3-caspase-1 complex promotes IL-1β secretion through cleavage of the proform. IL-1β strongly contributes to adipose tissue inflammation through auto amplification and paracrine activation during obesity.

Tackling diabetes and obesity

The basis of therapeutic interventions in inflammation and insulin resistance is to ameliorate obesity by physical exercise and diet control. The significance of chronic inflammation and its molecular mechanisms when the development of type 2 diabetes is demonstrated in mice, suppression of inflammation-related molecules has successfully improved glucose intolerance. The contribution of exercise and diet is generally admitted to be effective to attenuate obesity and sustain health. Also Clinical applications of anti-inflammatory drugs such as Aspirin/salsa late, IL-1β and TNFα can reduce the activity of inflammasome by blocking the inflammatory response in different ways.

A well-established drug Metformin enhances the oxidation of fat and glucose presumably by activating adenosine monophosphate kinase [16]. A newer class of insulin-sensitizing drugs used are thiazolidinedione's. These drugs are consistent with the theory that obesity-induced adipose tissue inflammation is a pivotal mediator of insulin resistance and provide additional scientific basis for therapy with PPAR-γ agonists. Additional approaches that could be used to treat obesity and its effects on hyperglycaemia include drugs that attenuate appetite and enhance energy expenditure [17-19].

Epidemiology

Type 2 diabetes which was since thought to be a metabolic disorder exclusively of adulthood has become increasingly more frequent in obese adolescents in the past few decades.

Type 2 diabetes occurs in all races even though a very high prevalence of type 2 diabetes has been observed in non-Caucasian groups (African Americans, Native Americans, Hispanics [20-24]. In recent studies diabetes is in the highest rates among youths aged 15–19 years in minority populations with incidence rate per 100,000 person-year. In particular, the reported incidence rate was 49.4 for Native Americans, 22.7 for Asian/Pacific Islanders, 19.4 for African Americans, 17 for Hispanics, and 5.6 for non-Hispanic whites. Type 2 diabetes in youth is reported worldwide.

As there is an increase in the prevalence of type 2 diabetes in the obese paediatric population there is also an increase in the prevalence of the pre-diabetes conditions. There is a drastic growth in the number of obese children and adolescents affected by type 2 diabetes. In addition to this there is also an upraise in the deregulation of glucose homeostasis. This explains the link between both obesity and diabetes as well as points out why type 2 diabetes is becoming one of the most important public health problems. Therefore, identifying the factors that causes obesity is of primary importance in order to interrupt its progression and the diabetes-related cardiovascular complications.

Discussion and Conclusion

The alarming increase in obesity rate makes it more widely discussed field of research. Obesity which is associate with adipocyte dysfunction, results in releasing and altering of adipokine production and signalling. Along with the systemic effects on metabolic regulation, these changes also promote infiltration of a wide range of immune cells into adipose tissue. The activation state and signalling of these immune cells is further varied by these factors, leading to initiation of metabolically driven, pro-inflammatory signalling cascades that inhibit insulin signalling in adipocytes. It also further enhances pro-inflammatory signalling in immune cells. As an outcome, adipocyte function is disrupted, they become insensitive to insulin and a vicious inflammatory cycle is engaged. This internal inflammation precedes the development of diabetes.

In a nut shell, the increased concentrations of TNF-α and IL-6, associated with obesity and type 2 diabetes, might interfere with insulin action by suppressing insulin signal transduction. This might interfere with the anti-inflammatory effect of insulin, which in turn might promote inflammation. Body mass index has a strong

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