Obesity and Carcinogenesis

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Obesity is currently reaching epidemic levels worldwide and increases the risk of cardiovascular diseases and type 2 diabetes. Recently, it has also been suggested to be linked with cancer. It accounts for a substantial proportion of global morbidity and mortality. Obesity is strongly associated with changes in the physiological function of adipose tissue, leading to insulin resistance, chronic inflammation, and altered secretion of adipokines. Several of these factors, such as insulin resistance, increased levels of leptin, plasminogen activator inhibitor-1, and endogenous sex steroids, decreased levels of adiponectin, and chronic inflammation, are involved in carcinogenesis and cancer progression. On the other hand, adipose tissue also has been hypothesized to act as a reservoir for lipophilic, liposoluble environmental carcinogens, so that chemical pollution may in fact generate both overweight/obesity and cancer. More precisely, it is suggested that many carcinogens stored in the adipose tissue could be released at convenient dose in the blood circulation and therefore target peripheral tissues to induce carcinogenesis. Such carcinogens mainly include organochlorine pesticides and polychlorinated biphenyls (PCBs). Their association with an increased risk of cancer seems to be demonstrated for breast and prostate carcinoma, as well as for lymphoma. Here the relevant evidence focusing on adipose tissue dysfunction as a unifying causal factor as well as the hypothesis of chemical pollutes in the link of obesity and carcinogenesis will be reviewed.