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Weight Control, Endocrine Hormones, and Cancer Prevention

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Obesity is well recognized to increase the risk of several types of cancer [1]. It has been suggested that those of 25% over normal weight have a 33% greater cancer risk than those who maintain a normal body weight [2]. There is ample evidence that weight control via decreasing calorie intake and/or increasing physical activity reduces cancer risk. For almost a century, Dietary Calorie Restriction (DCR) has been shown to inhibit the development of almost all types of cancer in animal models [3]. Alternatively, exercise has also been shown to reduce some types of cancer [4]. When compared with DCR, however, the impact of exercise may demonstrate a modest but intricate result for both weight maintenance and cancer prevention. Our previous studies conducted in an animal model, for example, showed that treadmill exercise alone with ad libitum feeding was not sufficient to decrease body weight due to, at least in part, the corresponding increase in dietary intake [5]. If the food intake of the exercised mice was limited by iso-caloric intake with sedentary counterpart, then body weight and body fat were significantly reduced [6]. Indeed, a negative calorie homeostasis appears to be the most potent for preventing cancer, which may be even extrapolated from the greatest of weight control diets to many dietary cancer preventive factors such as whole grain, fiber, and resistant starch, as well as vegetables and/or fruits, etc.

The mechanism by which weight control may reduce cancer risk is not well defined. Recently the National Cancer Institute called for research proposals regarding 24 Provocative Questions (http://provocativequestions.nci.nih.gov). The number one question is how obesity contributes to cancer risk. Previous studies by us and others have found that weight loss is associated with a reduction of many plasma levels of hormones such as IGF-1, leptin, insulin, and thyroid hormones [7,8]. It is possible that the reduction of these hormones accounts for cancer prevention by weight loss due to a mitogenic role, especially by IGF-1 and leptin, in promoting cellular growth and metabolic complications [9].

As a mitogen, IGF-1 is well known to stimulate cell proliferation, inhibit apoptosis, and enhance angiogenesis. Chronically high levels of IGF-1 observed in obese subjects have been linked with an increased risk of developing several types of cancer in case-control and prospective cohort studies [10,11]. It has been suggested that the role of IGF-1 in mitogenesis is triggered through cellular signaling cascades leading to the activation of both IGF-1 dependent MAPK-proliferation and PI3K-anti-apoptosis pathways for cancer promotion [12]. Furthermore, the role of adipocytes on malignance has been suggested via tumor-stroma interaction required during metastases or early micrometastases [13]. Recent data also suggest that adipocytes not only respond to hormonal signals, but also produce hormone-like factors such as pro-inflammatory leptin that may enhance oncological risk [14]. Although it is not clear how leptin interacts in IGF-1 mitogenic signaling, our previous studies conducted in TPA-promoted mouse skin cancer model have demonstrated that the reduction of both IGF-1 and leptin levels in response to weight loss corresponded to a concomitant inhibition of IGF-1-dependent mitogenic cascades and thus diminution of TPA-promoted signaling [5,6,15].

In addition to fundamentally advancing in the fields of obesity and cancer research, better understanding of the relationship between weight control, endocrine hormones, and hormone-dependent signal pathways may lead to a novel approach for cancer prevention.

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