Obesity, Metabolic Hormones, and Tumors

Living large can mean dying large. We are continually reminded about a relationship between obesity, cardiovascular disease, and diabetes; however, those warnings often overshadow another threat from obesity: the risk of cancer. Researchers estimate that excess weight may account for 14 percent of cancer deaths in men and 20 percent in women. Among all preventable risk factors for cancer, only smoking claims more lives.

Obesity’s link to cancer should come as no surprise. Signs of that relationship began to emerge two decades ago. In the late 1980’s, a connection was discovered between cancer and insulin—one of the major hormones that responds to obesity.

Although this finding received little attention then, today at least a half-dozen companies are developing cancer drugs that interfere with the hormone’s cousin—insulin-like growth factor I (IGF-1). If clinical trials find that dampening IGF-1 shrinks tumors in cancer patients, scientists would have a new kind of cancer drug and a new source of insight into the interplay between body weight, metabolism, and cancer. If the population in the United States were of a healthier weight, hundreds of thousands of deaths associated with cancer could be prevented each year.

Lower weight and more physical activity can affect the production of insulin, the hormone that allows the body to soak up fuel. After a meal, food is broken down into glucose, the body’s main source of energy. Insulin triggers cells to take up and use glucose. As a person gains excess weight, the cells can become resistant to insulin’s actions. To compensate, the pancreas begins to produce more insulin, but it cannot do so indefinitely. Eventually, insulin production falls and blood glucose levels rise in some people.

The potent hormone IGF-1 and a related hormone, IGF-2, are similar to insulin; they support rapidly dividing cells, especially during childhood and adolescence. The link between these insulin-like hormones and obesity is less clear than the connection between insulin and obesity.

Unlike cancer genes that encode other proteins and start down the path to cancer after mutating, the IGF-1 receptor gene is not altered in tumors. IGF-1 receptors show up in normal tissues throughout the body. The hormone itself is such a basic substance for animal life that even flies produce it. It was difficult to imagine that a normal receptor found in normal cells could have anything to do with cancer.

Scientists hypothesized that malignant cells may be overly dependent on IGF-1 receptors, on a scale far surpassing their dependence of normal cells. Epidemiological studies have linked cancer and the insulin-IGF axis in people, a discovery that caused the entire field of cancer treatment to undergo a transformation.

For example, in 1998, the risk of prostate cancer among men with the highest circulating levels of IGF-1 was four times as great as the risk among men with the lowest IGF-1 levels. Similar findings quickly followed in patients with breast, colon, and other cancers.

So far, colon cancer has the most consistent association with insulin and IGF-1 levels. Rates of colon cancer were more than twice as high among men with the highest levels of IGF-1 as in men with the lowest IGF-1 levels. Such findings fit with global patterns of the disease. Physical activity and reduced calorie intake can lower insulin levels. In contrast, populations with more sedentary jobs and calorie-dense diets have higher rates of obesity and higher insulin levels. In addition, diabetes is a risk factor for colon cancer.

Higher insulin levels are not the only chemical change that can occur with obesity. Concentrations of hormones that cause inflammation may also escalate, as do sex hormones, which can be produced in fat tissue. These and other modifications in the body could likewise set cancer in motion. Any of these fluctuations may work together to initiate carcinogenesis.

(Source: International Conference on Molecular Targets and Cancer Therapeutics, October 2008.)

Growth Hormone Affected by Starvation

Starvation blocks the effects of growth hormone (GH) via a mechanism that may have implications in treating diabetes and extending our life span, according to a study from the University of Texas in Dallas.

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THE AGING PROCESS

In the upcoming issue of Nutrition Health Review, the process of aging in humans will be examined. Aging is an unavoidable aspect of life. We will look at some of the reasons why we age and become slower. Aging is the result of a genetic program, such as a multidimensional process of physical, psychological, and social changes. Some dimensions of aging expand over time, whereas others decline. Reaction time may slow with age, whereas knowledge of world events and wisdom may expand. Biological and chronological changes occur in aging. Quality of life and environmental factors also play a role in the aging process.

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Can Light at Night Cause Cancer?

Women who are unable to sleep, consistently, nine or more hours a night had more than one-third the risk of developing a breast tumor than women who slept only seven to eight hours a night. Researchers in Finland found that breast cancer seems to be less common in women who slept more than nine hours a night than women who slept less.

Harvard researchers found that women who happen to have above-average concentrations of the hormone melatonin seem relatively unlikely to develop breast cancer.

(Source: Journal of the National Cancer Institute, July 20, 2005.)