

## Obesity-Cancer Relationship: Emerging Challenges and Opportunities in Healthcare

Ximena Paredes-Gonzalez<sup>1,2</sup> and Francisco Fuentes<sup>3\*</sup>

<sup>1</sup>Department of Pharmaceutics, Ernest Mario School of Pharmacy, The State University of New Jersey, USA

<sup>2</sup>Chile's National Health Fund (Fondo Nacional de Salud), Chile

<sup>3</sup>Faculty of Agriculture and Forestry, Faculty of Engineering and Faculty of Medicine, Catholic University of Chile, Chile

**\*Corresponding Author:** Francisco Fuentes, Faculty of Agriculture and Forestry, Faculty of Engineering and Faculty of Medicine, Catholic University of Chile, Chile.

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Obesity is considered a worldwide epidemic and one of the leading preventable causes of cancer [1,2]. It is usually defined as an excess of body adiposity that is evaluated according to the body weight by using the body mass index (BMI), which correlates the weight in kilograms (kg) and the square of height in meters (m<sup>2</sup>) (where BMI = kg/m<sup>2</sup>) [3]. The BMI is considered the standard measure for evaluating whether a person is obese. Thus, it can be considered that an individual with a BMI between 25 and 29.9 is overweight or pre-obese, while an individual with a BMI ≥ 30 is obese [3]. Considerable body of evidence suggests that the risk of developing and dying from many common cancers is increased in obese individuals being especially important in breast, colon and prostate cancers and acute leukemia [1,4,5]. In this context, obesity not only interferes with the effective delivery of systemic cancer therapy, but also contributes to morbidity from treatments which are strongly related with poor cancer outcomes and increased risk of recurrence and mortality [1,4,5].

More recently, multiple mechanisms involved in the association between obesity and increased risk of certain cancers have been described. Factors such as increased levels of insulin, insulin-like growth factor-1 (IGF-1) and leptin as well as decreased levels of adiponectin, excess of fat tissue, that produces excess amounts of estrogen -particularly important in increased risk of breast and endometrial cancers- have been correlated with the deregulation of target pathways such as mammalian target of rapamycin, AMP-activated protein kinase, enhanced oxidative stress, impaired immune responses and chronic inflammation [3,6,7]. Although further basic and clinical research are ongoing in this field aiming at elucidation of specific molecular mechanisms that can be targeted in the obesity-cancer relationship, it is clear that lifestyle patterns have a large impact in this system. Thus, dietary and physical activity habits are considered the major risk factors although obesity may be influenced by genetic, biological, behavioral and cultural patterns, pharmacological therapies among many others factors [3,6,7].

It is believed that maintaining a healthy weight, keeping active and changing dietary habits may reduce the burden of cancer [3,4,7]. Therefore, strategies to increase public awareness regarding the importance of healthy lives style are increasing over the past few years. Strategies such as healthy diet, increased physical activity, behavioral therapy and also, pharmacotherapy and bariatric surgery may be considered in obese cancer survivors in order to reduce the impact of obesity on cancer risk and cancer-related mortality [3,4]. Specifically, pharmacological strategies to decrease obesity may be considered in cases of BMI ≥ 27, since a history of cancer and obesity may represent a "dual-risk" profile [3,4]. In this context, collaborative preventive health-care strategies may play an important role decreasing the undesirable effects of obesity in cancer outcomes by encouraging life style changes and monitoring the effects of the pharmacological therapies, since majority of people are unaware that obesity can increase cancer risk and worsen cancer outcomes. Furthermore, these strategies might also include collaborative efforts in monitoring drug-drug, drug-food and drug-herbs interactions, side effects and dose adjustment related with specific drugs to prevent undesirable outcomes and maximize the benefits of cancer treatments.

### Bibliography

1. Calle EE, *et al.* "Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults". *The New England Journal of Medicine* 348.17 (2003): 1625-1638.
2. Caballero B. "The global epidemic of obesity: an overview". *Epidemiologic Reviews* 29 (2007): 1-5.
3. Paredes-Gonzalez X, *et al.* "Overview of Obesity, Inflammation, and Cancer, in Inflammation, Oxidative Stress, and Cancer: Dietary Approaches for Cancer Prevention". *ANT Kong. Editor CRC Press* (2014): 21-39.
4. Ligibel JA, *et al.* "American society of clinical oncology position statement on obesity and cancer". *Journal of clinical oncology* 32.31 (2014): 3568-3574.
5. Calle EE, *et al.* "Body-mass index and mortality in a prospective cohort of U.S. adults". *The New England Journal of Medicine* 341.15 (1999): 1097-1105.
6. Khandekar MJ, *et al.* "Molecular mechanisms of cancer development in obesity". *Nature Reviews Cancer* 11.12 (2011): 886-895.
7. Calle EE and R Kaaks. "Overweight, obesity and cancer: epidemiological evidence and proposed mechanisms". *Nature Reviews Cancer* 4.8 (2004): 579-591.

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