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Cancer's Progression that Influence by Diets

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Abstract

Diet is just one of the lifestyle factors that influence the risk of developing cancer. Smoking, obesity, alcohol, sun exposure and physical activity levels are also important. Although some foods can affect cancer risk, there is no evidence that specific foods can cause or cure cancer.

Keywords: Cancer; Smoking; Obesity; Sun exposure; Alcohol

Introduction

The protein p53 has a solid reputation. It functions as a typical tumour suppressor, controlling cell development and eliminating cells that have turned rogue by proliferating excessively [1].

Case studies

But researchers discovered approximately 15 years ago that when cells are starved of glucose, the protein switches allegiances and instead of killing tumour cells, it aids in their survival. Nutrients ingested through food could affect the p53 metabolic pathway [2].

They discovered that depriving tumour cells lacking p53 of serine and glycine reduced their growth whereas a control diet rich in these nutrients sped it up. It was unclear whether eliminating serine and glycine from the diet would have any impact because they are nonessential amino acids, meaning that the body can synthesise them from other molecules. To their amazement, however, the experiment also functioned in the same manner with mice that had been given injections of these tumour cells and were given diets deficient in serine and glycine. "The fundamental finding was that altering one's diet can decrease tumour growth [3].

The idea that a person's food can affect cancer has long been hypothesised by researchers, but up until recently, the majority of the evidence for this theory came from surveys of big populations' diets rather than studies that examined the underlying physiological processes at play. The discovery established the first direct biochemical connection between nutrition and cancer [4]. The number of connections has increased since then, according to scientists. Recent research has begun to shed light on the potential role that certain nutrients, including carbohydrates, amino acids, and fats, may play in either promoting or inhibiting the growth of various cancers.

Only a few clinical trials have been launched to investigate food-based methods in people, despite the fact that researches in animals have so far shown promise. It's a pipe dream to imagine that diet [alone] is going to cure cancer." But we need to conduct the studies to see whether or not it makes a difference. Many cautions are issued by researchers looking at the biochemical relationship between nutrition and cancer [5]. The most significant is that doctors cannot yet advise patients on what to eat if they have cancer. There is almost certainly no single food or dietary fix that would help address all types of cancer, the role of amino acids and other nutrients in cancer. It's also highly unlikely that dietary interventions will completely replace cancer-killing medicines as therapies, he adds.

However, as scientists understand more about the underlying biochemistry, they foresee developing a kind of matrix that would pair specific nutritional strategies that work in conjunction with cancer medications with genetic abnormalities that cause cancer or specific metabolic pathway segments. Many cancer medications prolong a patient's life by only a few months. And even though their tumour types indicate they should, some patients never benefit from certain medications for unknown reasons [6]. The appropriate nutrient punch could increase the effectiveness of cancer medications and increase the number of people who respond to them. It is not novel to think that certain foods can directly feed cancer cells. Tumour cells consume a significant amount of glucose almost a century ago. The subject of cancer cell metabolism was founded and for many years, researchers studied cancer using that framework.

In fact, the impacts of cell metabolism provided the basis for the first effective cancer medications. Utilising folate (vitamin B9) could cause the leukaemia to develop shockingly swiftly, a disease brought on by their leukaemia, in the late 1940s. Today, methotrexate is still often used to treat cancer and a number of other illnesses.

However, in the 1980s, cancer researchers started to focus less on metabolism and more on the genetic features of cancer. A number of significant cancer medicines were developed as a result of the quest for tiny compounds that could treat these specific mutations, but intriguingly, this research led researchers back to cell metabolism. Because they regulate metabolism, we always find ourselves back where we started [7]. That change has been fuelled by research on the phosphoinositide 3-kinase (PI3K), an enzyme first identified in the middle of the 1980s. PI3K is a glutton for glucose: given the correct circumstances, PI3K will drive the growth of tumours as long as the sugar is accessible. Additionally, a huge range of tumours have mutations in it. For the treatment of cancer, a number of PI3K inhibitors have been licenced, although their effectiveness is frequently subpar.

Current research may have neglected the effect that nutrition can have on the suppression of PI3K and, in particular, that minimising insulin spikes may be one key to bolstering some cancer therapy. The

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most effective activator of PI3 kinase, by far, is insulin, he explains. It follows that if a person's blood insulin levels are too high, PI3K-shutdown medications could not be sufficient to eradicate a tumour [8]. He and his associates validated this suspicion in a study published in 2018 PI3K inhibitor effectiveness was increased by an insulin-lowering diet, at least in mice. Because tumours use glucose, glucose may have an unusually extensive connection to malignancies, claims Cantley. There are a lot more nutrients to investigate, though. Serine and glycine depletion may be beneficial for treating tumours by going beyond p53 to other components of cancer communication.

Researchers can learn more about other metabolic processes by studying how cells utilise amino acids. High levels of histidine increased the sensitivity of cancer cells to methotrexate, probably as a result of the drug's one-two punch of blocking folate activity and the cells using up their supply of folate as they break down the histidine. It is unclear exactly how and in what tumour types folate impacts cancer metabolism [9]. It is also unclear whether adding extra histidine to the diet could boost the potency of this medication and possibly other comparable ones under development.

Added nutrients like fats and broader view of diets. According to numerous studies, both diets that drastically reduce calorie consumption overall and ketogenic diets, which strictly restrict carbohydrates while containing high quantities of fat, may inhibit the growth of cancer cells in a dish and in mice. Although the two diets appear to function differently, researchers have speculated that this may be due of the decreased glucose and insulin levels the diets cause. Why so foods vary [10]. Although both diets do limit the amount of glucose tumours can access, they also have an adverse effect on the production of an enzyme that turns food into fatty acids. The two diets, however, actually alter the playing field of this enzyme in two different ways. Tumor cells don't need the enzyme because the ketogenic diet is heavy in fats, thus they can simply use those nutrients. But because the caloric restriction diet generally results in decreased lipid levels, tumours might become increasingly reliant on the enzyme. Due to their inability to generate fatty acids or obtain them from their environment, cancer cells grow more slowly when following that diet.

Conclusion

Finally, the genetics of the cancer will ultimately determine how any dietary intervention would impact a tumour. We don't fully understand why various tumour forms with different genetic alterations might respond to the same diet in different ways. Researchers still need to learn a lot more about the underlying biochemistry regulating the metabolism of particular nutrients in order to match them with dietary interventions that might stop some cancers from growing but let others proliferate better.

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Conflict of Interest

The Author declares no conflict of interest in this study.

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