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Starving cancer: dietary modifications may enhance cancer therapy

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Our diet strongly impacts our health. It is becoming increasingly clear that our diet is also linked to cancer risk, and thus perhaps may be a useful tool for cancer therapy. Studies shown that dietary modifications can be beneficial for cancer therapy in mice and humans. As we learn more, these modifications may become an integral part of cancer therapy in the future.

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We all obsess about our fitness and health and a critical part of it is how and what we eat. Healthy nutrition is not only important for our look, but also linked to all aspects of physiology and disease. Recent years have seen a surge of interest in the idea of supplementing established anti-cancer therapies with dietary modifications. But is this a good idea and do we know enough to take the next steps?

Epidemiological studies show a link between cancer risk or poor prognosis on one side and over-eating, unhealthy diets, and metabolic diseases such as obesity on the other. For example, high consumption of simple sugars such as fructose and glucose, abundant in the western diet, increases the risk for colorectal and liver cancer. But although epidemiological studies can help us generate hypotheses, we need to critically investigate the specific mechanisms before we can make predictions and use nutritional modifications in the clinic.

Recent metabolic studies using model organisms such as mice have allowed us to gain a deeper understanding of the relationship between cancer progression and supply of specific nutrients. In these studies, dietary restriction of specific nutrients such as glucose, fructose, and some amino acids, as well as in some cases supplementation of specific nutrients, result in changes in tumor growth, cancer cell metabolism, tumor-stimulating signaling pathways, response to therapy, and survival.

These are very encouraging results. However, it is clear that there is no "one size fits all" approach; not

any or all dietary modification will be beneficial against all cancer types. Dietary modifications could only be used therapeutically once their effect on a specific tumor type is well documented and reproducible.

These studies also provided us with a better understanding of the tumors' own nutritional requirements, which allows us to methodically test possible nutritional interventions in order to separate cause and effect. This is a far more promising approach than the retrospective study of cancer risk in correlation to people's diets, such as revealed by epidemiological studies.

Although it is too soon for any clear dietary recommendations in the clinic, our current understanding of cancer metabolism is sufficient to tell us that we cannot overlook the contribution of the diet to tumor growth. What we feed the patient, we feed the tumor, so we must consider diet in the context of the tumor's nutritional demand as well as the patient's.

These data can help focus and direct interest to further research on the nutritional vulnerabilities of different tumor types. This will lead to better therapy protocols that include both the targeted cancer therapy and a targeted diet to complement and perhaps enhance the therapy. We hope researchers and funding agencies will be encouraged to complete more pre-clinical and clinical studies that will pave the path to clear recommendations of diets for cancer patients.