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Abstract

most e ective 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 su cient to eradicate a tumour [8]. He and his associates validated this suspicion in a study published in 2018 PI3K inhibitor e ectiveness 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. ere are a lot more nutrients to investigate, though. Serine and glycine depletion may be bene cial 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 di erently, 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 e ect on the production of an enzyme that turns food into fatty acids. e two diets, however, actually alter the playing eld of this enzyme in two di erent 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.

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 di erent genetic alterations might respond to the same diet in di erent 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.

None

e Author declares no con ict of interest in this study.

References

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