The anti-cancer effect of aerobic exercise

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Fascinating recent research reports that interleukin-6, a protein released by muscles during vigorous exercise, decreases the growth of colorectal cancer cells by promoting DNA repair.

There is strong epidemiological evidence that regular physical activity has a protective effect against colorectal cancer.

Studies report that in people who are more physically active, the risk of developing this cancer is reduced by about 25% compared to those who are more sedentary. This anticancer effect of exercise is also observed in secondary prevention, i.e. in survivors of colorectal cancer, with a reduction in the risk of recurrence and mortality specific to this cancer, which has been reported by several studies.

SYSTEMIC EFFECTS OF EXERCISE

The mechanisms involved in this anticancer effect of exercise are likely related to the production of different molecules by moving muscles. When they are solicited during an exercise, the muscle cells indeed release a panoply of molecules, which diffuse into the bloodstream and subsequently influence several target organs.

One such muscle factor that is currently attracting a lot of interest is interleukin-6 (IL-6), a multifunctional cytokine that controls several physiological processes.

For example, it has recently been shown that IL-6 produced by muscles during exercise accelerates lipolysis (fat destruction) in abdominal fatty tissue and contributes to the positive effect of regular physical activity on decrease in waist circumference (1).

ANTI-CANCER IL-6

A recent randomized clinical study suggests that IL-6 may also play a key role in colorectal cancer prevention associated with regular physical exercise (2).

In this study, British researchers recruited 16 healthy men, but who presented three risk factors for colorectal cancer, namely age 50 and over, overweight (BMI >25 kg/m^2) and lack of regular physical activity.

Participants were randomly assigned to either perform aerobic exercise (6 x 5 minutes of high-intensity stationary cycling) or remain inactive during this period.

A week later, each of the participants was subjected to the reverse condition (those who had exercised were at rest and vice versa).

The strength of this type of crossover study is that each of the participants is exposed to the two experimental conditions tested and that one can therefore directly compare their effects on the same person.

To determine whether exercise caused the appearance of an anti-cancer factor, the researchers took blood samples before and immediately after the period of physical activity (or rest). The blood sera isolated were then exposed to colorectal cancer cells cultured in the laboratory, to measure their impact on tumor growth.

The results show that compared to serum from sedentary participants, the addition of post-exercise serum significantly decreases cancer cell proliferation and is associated with a significant reduction in cell DNA damage, an event that contributes to tumor progression.

These phenomena are probably a consequence of the production of IL-6 by the muscles, since the researchers observed a significant increase in this cytokine in post-exercise sera and the addition of purified IL-6 to the cells makes it possible to reproduce the effects of these serums on the growth of cancer cells and the reduction of damage in the structure of their DNA.

Overall, these results therefore indicate that IL-6 released during an activity requiring muscle contraction can slow the progression of cancer cells, possibly by stimulating cellular pathways specialized in DNA repair.

Another example that shows how exercise is not only a way to maintain good physical shape, but also an essential activity to prevent cancer.

(1) Wedell-Neergaard AS et al. Exercise-induced changes in visceral adipose tissue mass are regulated by IL-6 signaling: a randomized controlled trial. Cell Metab. 2019 ; 29 : 844-855.e3.

(2) Orange ST et al. Acute aerobic exercise-conditioned serum reduces colon cancer cell proliferation in vitro through interleukin-6-induced regulation of DNA damage. Int. J. Cancer (Online ahead of print, February 25th 2022).

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