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We all bear tumours

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Some surprising results show that, as we age, over half of the cells in the esophagus accumulate thousands of mutations in their DNA, without actually becoming cancerous. Another important example of the importance of following a healthy lifestyle to prevent these microtumours from developing into mature cancers.

TO ERR IS HUMAN

Throughout our lifetime, the cells which make up our organs must constantly renew themselves to compensate for cells which die or become non-functional. This process is not, however, perfect: during the course of copying the three or so billion letters of DNA, errors (which we call somatic mutations) arise and can provoke the appearance of lesions which have the potential to evolve into cancer. The presence of these mutations means that all people, even those in good health, contain a large number of abnormal cells, which can even in some cases manage to evolve into microscopic tumours. For example, 50% of women in their forties have precancerous lesions within their breasts, a proportion that is much greater than the incidence of this cancer in the population (15%). The same is true for cancer of the pancreas: 75% of the population exhibits precancerous anomalies in this organ while the cancer itself occurs in only 1.4% of the population. These spontaneous mutations which occur by chance thus have the potential to become cancerous, but in most cases they remain in a latent and nonthreatening form. In other words, even if we are biologically predisposed to cancer, it seems that we are also predisposed to prevent the development of these cancers.

THE EXAMPLE OF THE ESOPHAGUS

The concept of mutations which do not succeed in evolving into cancer is well illustrated by the results of a study recently published in the prestigious journal *Science*[1]. A British team lead by Dr. Inigo Martincorena obtained samples of the esophageal wall (epithelium) from 9 donors (20 to 75 years of age) who had died from causes other than cancer and looked for mutations in 74 genes known for their involvement in the development of carcinomas in this organ. While under the microscope these tissues appeared healthy and showed no signs of cancer, the results of DNA analysis showed the opposite: in individuals in their twenties, the healthy cells already contained hundreds of mutations and in older subjects there were over 2000 per cell! These mutant cells multiplied more rapidly than did normal cells, which indicates that at a mature age (50 years and over), more than half of the esophageal wall is formed of mutant clones, containing many thousands of mutations in procancerous genes.

LIFESTYLE INFLUENCE

While it is currently rare, cancer of the esophagus has more than sextupled over the past 40 years, and currently represents one of the cancers whose incidence is increasing fastest[2]. This suggests that there are current lifestyle factors which promote progression of the mutant cells which spontaneously form in this organ.

The strong increase in the number of overweight people is one of the important factors which explain this marked increase in the incidence of esophageal cancer. Obesity is often associated with chronic gastro-esophageal reflux which promotes the development of endobrachyesophagus (Barrett's esophagus), a condition which is associated with a very significant increase (30 to 60-fold) in the risk of adenocarcinoma of the esophagus. The abusive consumption of alcohol (particularly of spirits), smoking as well as the consumption of very hot drinks have also repeatedly been associated with the increased risk of esophageal cancer.

These observations confirm that we are all bearers of precancerous cells, but it is possible to slow their progression by adopting good lifestyle habits such as not smoking, keeping a normal healthy body weight, eating lots of plant-based foods and being physically active. The principal objective in preventing cancer is not simply to prevent the occurrence of cancerous cells (because they form spontaneously) but rather to sufficiently delay their progress so that they are not able to attain mature cancer status during the eight or nine decades of a human lifespan.

(1) Martincorena I et al. Somatic mutant clones colonize the human esophagus with age. Science, 2018, 362(6417): 911-917

(2) Brown LM et al. Incidence of adenocarcinoma of the esophagus among white Americans by sex, stage, and age. J. Natl Cancer Inst. 2008; 100: 1184-1187.

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