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Commentary

Oncovirus and its viral mechanism

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DESCRIPTION

Oncovirus or oncogenic virus is a virus that can cause cancer. The term came from a study of mutant retroviruses in the 1950-60s, when the term "oncornaviruses" was used to describe the origin of the RNA virus (Rusyn I, 2014). Since the letters "RNA" has been removed, it now refers to any virus that contains DNA or RNA genome that causes cancer and is similar to the word "tumour virus" or "cancer virus" (Chang Y, 2017). Most viruses in humans and animals do not cause cancer, possibly because of the long-term evolution of the virus and its host. Oncoviruses have been important not only in epidemiology, but also in research into cell cycle control mechanisms such as retinoblastoma protein (Valladares Y, 1960).

The World Health Organization for Research on Cancer estimates that in 2002, the virus caused 17.8% of human cancers, 11.9% of the virus in one of seven cases. A 2020 study of 2,658 samples of 38 types of cancer found that 16% were associated with the virus. These cancers can be easily prevented by vaccination (e.g., papillomavirus vaccine), obtained by simple blood tests, and treated with antimicrobial compounds that are less toxic (Parkin DM, 2006).

Oncogenic viral mechanism

• The precise mechanism of the oncogenic viral mechanism involves either the insertion of additional viral oncogenic genes into the host cell or the development of existing oncogenic genes (proto-oncogene) in the genome. For example, it has been shown that vFLIP and vCyclin interfere with the TGF-β indirect expression method by inserting a collection of oncogenic host mir17-92 (Zapatka M, 2020).

• Indirect viral oncogenicity involves chronic, indirect inflammation that occurs over decades, as is the case with HCV liver cancer. These two mechanisms vary in their biology and epidemiology: specific viral pathogens must have at least one copy of each tumor cell producing at least one protein or RNA that causes the cell to become cancerous (Fredericks DN, 1996). Because of the foreign antigens that come from these tumors, people who are not immune, such as AIDS or transplant patients, are at greater risk for these cancers.

• On the other hand, indirect tumor bacteria can be lost (at least in theory) from a mature tumor that has accumulated enough mutations and growth conditions (hyperplasia) from chronic inflammation of the virus (Mantovani F, 2001). In the latter case, it is controversial but at least from the point of view that it is possible for an indirect tumour virus to get into a "hit-and-run" and therefore the virus could be lost to a clinically found tumour. Materially, this is a rare occurrence.

DNA oncoviruses

DNA oncoviruses usually damage two families of protein-suppressing proteins: tumour p53 protein and retinoblastoma (Rb) protein. It is beneficial from nature

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that the viruses do not activate p53 because p53 can cause cell cycle arrest or apoptosis in infected cells when the virus tries to replicate its DNA. Similarly, Rb proteins regulate many important cell functions, including but not limited to a critical cell cycle, making them the target of viruses that try to disrupt normal cell function.

Of the many DNA oncoviruses that were discovered, three have been studied extensively. Adenoviruses can lead to tumors in rodent models but do not cause cancer in humans; however, they have been exploited as a vehicle for genetic treatment for diseases such as cystic fibrosis and cancer. Simian virus 40 (SV40), polyomavirus, can cause tumours in rodent models but is not oncogenic to humans (Felsani A, 2006). This situation has been one of the major controversies of oncogenesis in the 20th century because an estimated 100 million people are unknowingly exposed to SV40 with polio vaccines. Human papillomavirus-16 (HPV-16) has been shown to lead to cervical cancer and other cancers including the head and neck. These three viruses have similar processes, forming the archetype of DNA oncoviruses. All three of these DNA oncoviruses are able to bind their DNA to host cells, and use this to record and modify cells through the G1 / S test site cycle.

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