Immunity and cancer are not connected

> *A surprising argument used in some of the reviews dealing with immune surveillance is based on the assumption that in any information transfer system, such as somatic cell replication, there are inevitable errors, and neoplastic transformation therefore must be frequent. The argument is made that immunological surveillance must be efficacious or overt clinical neoplasia would necessarily be more frequent than it actually is. This circular argument also includes the assumption that frequent accidents of somatic cell replication produce neoplastic variants that are invariably antigenic and thus can be rapidly eliminated by the immune system.*
>
> *Osias Stutman* [1]

The idea that the clinical course of cancer depends on whether or not a tumor's potential for unrestricted growth wins out over inherent host defenses is 200-years-old [2]. A modern formulation of this view known as the immune surveillance hypothesis of cancer was advanced by Burnet [3] and Thomas [4]. The main assertions of the immune surveillance hypothesis are: (1) most tumors are antigenic, and (2) such antigenic differences can "under appropriate conditions" provoke an immune response [5].

Based on this thinking, in the late 1950s Jonas Salk attempted to stimulate the immune systems of terminally ill cancer patients by injecting them with what he thought were monkey heart cells. He had hoped that the patients' activated immune systems would attack the cancer cells. However, in 1978 Salk revealed that he had not injected the cancer patients with monkey heart cells but mistakenly with HeLa cancer cells [6]. The cancer patients' immune systems did indeed become activated and functioned well enough to eliminate the small tumors formed at the sites of injection of the HeLa cells within three weeks, never to return. Yet the activated immune systems of these same cancer patients were not effective against their natural tumors.

It is not the purpose here to rehash the exhaustive analysis of, and compelling arguments against, the immune surveillance hypothesis [1,7,8] but simply to add that the aneuploidy theory provides additional support for the view that there is no significant connection between cancer and immunity.

Cancer is us because it is derived from our very own genome. What makes cancer cells not us is that they have rearranged our genome to differ from their diploid predecessors in both the number of chromosomes and the dosage of thousands of genes. Since there are no new genes, and no cancer-specific mutant genes, and no new chromosomes (except hybrid or marker chromosomes) in cancer cells [9], there is little or nothing for immune surveillance to monitor. This is especially true for the earliest stages of carcinogenesis where the immune surveillance mechanism is supposed to be most effective but the aneuploid cells are least abnormal.

Even if an aberrant antigenic cell happened to result from the chaotic scrambling of the genome, the immune system could be expected to eliminate it, while the vast majority of aneuploid cells remained invisible to the immune system. Therefore, even in principle, there is no possibility of an immune surveillance system guarding against the appearance of cancer cells.

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