

EDITORIAL

VANHIVAX and the Trojan exosome hypothesis

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HIV, like other retroviruses is an enveloped virus. During the budding process through cell membranes, it acquires several hundred host cell molecules; thus retroviral particles have an array of host cell molecules on their surfaces (1).

It is known that humans and other mammals combat enveloped viruses by robust B and T cell immune responses against viral antigens; however, these responses do not seem to be protective, since the viral load of the patients is not much affected (2), and the host is eventually killed if appropriate anti-retroviral therapy is not initiated. Absence of such protective immunity is reflected in the failure so far of conventional immunotherapeutic approaches aimed at enhancing the immune response against HIV (3).

When the HIV pandemic emerged in Cameroon, Prof. V.A. Ngu, an oncologist and former laureate of the Lasker award for cancer research hypothesized that since HIV is a retrovirus and carries host self antigens acquired during budding, the host immune system does not mount a sufficiently robust response to it because it is hidden from the immune system by these host self antigens; therefore viral core antigens devoid of these self antigens would elicit an effective immune response, and can constitute effective therapeutic and prophylactic vaccines (4, 5).

He has since tested this hypothesis using a “vaccine” he has named “VANHIVAX” (6, 7). It is prepared by incubating the plasma of HIV infected persons (assumed to contain many HIV particles bearing host self antigens on their surfaces) with chloroform or ether in the hope of removing (dissolving) the host self antigens from the surface of the viral particles. The incubated plasma (“vaccine”) is then injected into the donor of the plasma. Booster injections every month after the primary injection contain the “vaccine” to which leucocytes from the donor patient have been added (8). Since the sample from each patient is used to prepare their own “vaccines”, it is referred to as “auto-biotherapy”.

Similar “vaccines” derived from the blood of HIV-positive donors include THIVAC (9, 10), and V-1 Immunotor (11).

The budding of retroviruses has been compared to the formation of exosomes, the small extracellular vesicles that eukaryotes synthesize and release to their extracellular environment (12). The Trojan exosome hypothesis (12) states that retroviral particles and exosomes contain a similar array of host cell lipids and proteins; this explains why retroviruses are relatively resistant to adaptive immune responses directed at viral antigens. Effective antiretroviral immunity needs to be directed against exosomal antigens that are present on the surface of retroviruses and retrovirus-infected cells; these host cell exosomal antigens – alloantigens - are highly polymorphic within the host population and elicit alloimmunity which is a major mechanism of retroviral resistance (3, 12).

Interestingly, Ngu (4, 5) predicts that VANHIVAX is based on exposed viral antigens. However, his chloroform or ether incubation would hardly remove non-lipid exosomal antigens. We think that his “vaccine”, like that of others in the domain (9,10,11), is probably based on immune responses to alloantigens present in the plasma and the leukocytes he injects into the patients, since viral antigens have so far not been shown to produce protective immunity.

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