

VIRAL PROZONE IN MACROPHAGES

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The arboviruses West Nile and Batai when serially diluted and added to the macrophage cell line P388D 1, produce cytopathic effect (CPE) and viral plaques to titres of 10^5 - 10^6 pfu/ml. However, when high virus inputs 10^4 - 10^5 pfu are added to 2×10^5 cells there is no CPE or plaque formation viz. a virus "prozone". A comparable phenomenon is not seen in non-macrophage cells e.g. Vero. When P388D 1 cells are infected with CPE producing (10-100 pfu) and

"prozone" producing ($10^4 - 10^5$ pfu) virus inputs (per 2×10^5 cells), and virus replication monitored by daily titration of virus yield in supernatant fluid, virus replication in both cultures were comparable. Thus high virus inputs do not result in inhibition of viral replication, but convert a cytolytic infection to a nonlytic one.

When P388D 1 cells are exposed to high virus input for a short time (e.g. 2 h) followed by removal of unabsorbed virus, the CPE prozone does not occur. Heating at 58°C for 45 min or storage at 4°C for 3-4 days does not destroy the "Prozone inducing factor" in virus preparation though heating at 80°C for 1 hour does so. "Prozone inducing factor" is not destroyed by pH 2 treatment. When "prozone" inducing virus inputs are mixed with antiviral antibody at high or low dilutions, the prozone is lost.

Viral prozone has been recognised for many years, and is thought to be due to Defective Interfering particles in the virus preparation. However, the phenomenon described here differs in many respects from the classical virus prozone and is unlikely to be produced by Defective particles because (a) it does not occur in non-macrophage cells, (b) viral replication is not inhibited, only viral cytolysis is, (c) the inducing factor is pH 2 stable and heat 58°C stable, (d) the "prozone" is abolished by virus specific antibody.