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MULTINUCLEATED CELL: VIRUS-CELL FUSION AND ITS ROUTES.

Oral Pathology

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ABSTRACT

Macrophages are the mononucleated cell which turns out into multinucleated cell. The process of multinucleation enhances the phagocytic action of the cell efficiently. The virulent virus at small doses while the temperate virus at high doses can cause cell fusion of both infected and uninfected cells producing multinucleation. There are two routes of cell-virus fusion namely endoplasmic and ectoplasmic. The present article aims to review the process of cell-virus fusion and its endoplasmic and ectoplasmic routes.

KEYWORDS

Multinucleated cell, Macrophages, Virus, Cell Fusion, Giant cell

INTRODUCTION

Macrophages, the peripatetic mononucleated cell, and the ardent phagocytes are the fundamental governor of the homeostasis. Their phenotypic form and function are influenced by the environmental influence.[1,2,3]

Monocytes individualize to form macrophages; the specially designed cell having the proficiency of fusion with each other to turn out into multinucleated cell.[4] Multinucleation escalates the phagocytic performance of the cell in response to the foreign constituents and inflammation.[5]

CELL FUSION

The cell membrane is a lipid bilayer that is sturdy and secured by the presence of integral proteins on both sides.[6] The presence of any foreign particle imparts instability to the membrane by altering its proteins. This reduces the gap between the adjacent cells, resulting in constancy between opposing membranes inducing fusion.[5,6]

VIRAL INDUCED CELL FUSION

The virulent virus at small doses can cause cell fusion via endocytosis preceding alterations in integral proteins of the cell membrane. It results in the fusion of both infected and adjoining dis-infected cells producing multinucleation.[1,6,7,8]

The temperate virus can induce cell fusion at a high dose. The viral envelope gets adhere to the lipid bilayer membrane causing abatement in cell membrane thickness. This process is followed by the fusion of two or more cells in close contact with the virus.[5,8]

MECHANISM OF VIRUS-CELL FUSION

Virus-cell fusion is a feature of all enveloped virus to cause infection.[7] It is mediated by both viral antigens and cell surface molecules which act as viral receptors.[5] The viral antigen present in its envelope gets incorporated into cell membrane of macrophage.[6] Fusion process occurs both endoplasmic and ectoplasmic depending upon whether the interaction between antigen and cell surface molecule (viral receptors) is extracellular or intracellular respectively.[7,8]

Two routes of virus-cell fusion were described by Hernandez et al, 1996 as follows:-

I. ENDOPLASMIC FUSION

Enveloped virus penetrate the cell via endocytosis reaches the endoplasmic reticulum (ER) transport to RER –SER, leaves it behind in a clathrin-coated vesicle to enter the cis phase of golgi apparatus (GA). It leaves behind the GA again in clathrin coated vesicle to enter the endosome. The clathrin coated vesicle acts as protective cover as the pH from ER to endosome is declined. Within the endosome, the low pH enables the viral membrane to get fused with the endosomal membrane while releasing the nucleus producing multinucleation.[7,8] **(Fig. 1)**

II. ECTOPLASMIC FUSION

The viral antigens bind to its receptors producing destabilization to the integral proteins in the bilipid layer. It fuses directly with the cell membrane with the help of specific fusion glycoproteins at neutral pH. Nucleus enters inside the cell causing multinucleation.[7,8] **(Fig.2)**

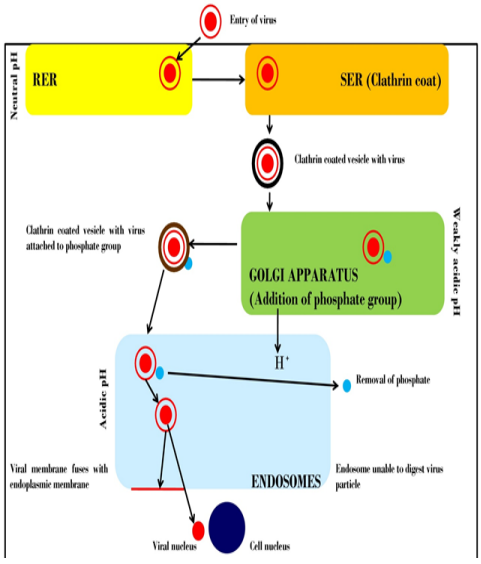

Fig1: Endoplasmic Route

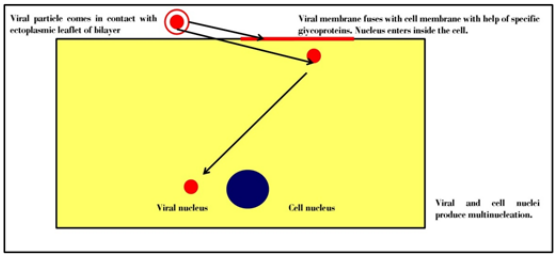

Fig2: Ectoplasmic Route

CONCLUSION

Virus is able to cause multinucleation at both high and low doses respectively based on their ability to infect the cell. The virus-cell fusion occurs both intracellularly and extracellularly depending upon the first area of contact with the lipid bilayer. Once infected, the cell has altered glycoproteins which enable it to bind with adjacent uninfected cell to initiate fusion furtherly.

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