Biomedical Reviews 2: 79-80 (1993) Published by the Bulgarian - American Center, Varna, Bulgaria

EDITORIAL

INTRACELLULAR FUSOGENS AND ANTIFUSOGENS IN HIV-1-CD4 MEDIATED MEMBRANE FUSION?

• The work of Dimitrov (1) is likely to be highly significant in elucidating the mechanism of HIV-1 envelope glycoprotein-mediated membrane fusion, together with the "in press" data of requirements of calcium ions and cell component(s) for HTV-1-CD4 fusion (1, his Ref 5, 14).

There are at least two HIV-1 envelope glycoprotein-mediated fusion events: (i) the heterotypic HIV-1-CD4 fusion leading to the virus cell entry, and (ii) the homotypic HTV-1-infected cell's HIV-1 envelope glycoprotein-mediated membrane fusion leading to the syncytia formation (1).

Recent knowledge of molecular dissection of membrane fusion in both biosecretion and endocytosis pathways (2-7 and Refs therein) prompts me to suggest that if it works in the fusion events of HIV-1 entry and syncytia formation, then the known cellular fusogens and antifusogens (Table 1) might be explored in HIV-1-CD4 fusion studies.

Table 1.

Fusogens and antifusogens proposed as an approach in HTV-1-CD4 fusion research

Fusogens	Refs
annexin I	4
annexin II	5
clathrin assembly protein (AP-2)	3
small G-binding proteins	2,6
NEM (N-ethylmaleimide)-sensitive fusion protein	2,7
SNAP (soluble NSF-attachment protein[s])	2
Antifusogens	
NEM	2
GTPγS (nonhydrolyzable GTP analogue)	2

Therefore I conclude this comment with two related questions: (i) might antifusogens such as NEM and GTP"^S be effective in "dissection" of HIV-1 cell entry and/ or syncytia formation, and (ii) does calcium ions requirement for efficient fusion (1) require some calcium-dependent phospholipid-binding proteins such as annexins? Last not least, recent experience in studying the exocytotic fusion pore formation (7) may also be applied to HIV-1-CD4 fusion research.

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