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Perspective

Cell-to-Cell Signaling Using Tunneling Nanotube or Membrane Nanotube

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DESCRIPTION

The term "Tunnelling Nanotube" (TNT) or "membrane nanotube" has been used to describe protrusions that protrude from the plasma membrane and allow various animal cells to contact over considerable distances, occasionally over 100 m between T cells. Nanotubes are two different categories of structures. The first kind has a diameter of less than 0.7 micrometers, contains actin, and transports sections of plasma membrane back and forth between cells. The second kind may transport cytoplasmic elements like vesicles and organelles across cells, including complete mitochondria, and are larger (>0.7 m), contain both actin and microtubules. TNTs can reach lengths of several cell diameters and have a diameter of 50 to 200 nm. The transmission of diseases or poisons like HIV and prions, as well as cell-to-cell communication, nucleic acid transfer within a tissue, and pathogen-to-tissue communication are all possible functions of these structures. Several proteins have been linked to the generation or suppression of TNTs, which have documented lifetimes ranging from a few minutes to several hours.

Formation

The production of nanotubes may be influenced by a number of factors. Cell-to-cell interactions and molecular controls are examples of this.

There have been two main methods put out for TNT creation. First, there are cytoplasmic protrusions that move from one cell to another before joining the membrane of the target cell. The second is that TNTs continue to act as bridges connecting the two cells as they move apart from one another.

Induction: When subjected to bacterial or mechanical stimuli, certain dendritic cells and THP-1 monocytes have been demonstrated to join *via* tunneling nanotubes and exhibit calcium flux. According to research, TNT-mediated signaling causes spreading in target cells, which is comparable to the lamellipodia created when bacterial products are exposed to dendritic cells. The TNTs that were found to work in this study spread at an initial pace of 35 micrometers per second and connected THP-1 monocytes to nanotubes that were up to 100 micrometers long.

It has been found that cytonemes form in response to a BnL-FGF gradient, indicating that chemotactic controls may trigger the production of TNT-like structures. Phosphatidylserine exposure directed TNT synthesis from Mesenchymal Stem Cells (MSCs) to a population of damaged cells, which is a supporting finding. As p53 causes caspase 3 to cleave the protein S100A4 in the beginning cell, creating a gradient in which the target cell has higher amounts of the protein, it has been demonstrated that the protein S100A4 and its receptor control the direction of TNT growth.

According to one study, the development of nanotube bridges between T cells required cell-to-cell interaction. The downstream genes up-regulated by p53 (EGFR, Akt, PI3K, and mTOR) were discovered to be involved in nanotube creation after hydrogen peroxide treatment and serum deprivation, further pointing to p53 activation as a required mechanism for the growth of TNTs. According to research, connexin-43 helps connect bone marrow stromal cells (BMSCs) with alveolar epithelial cells, which creates nanotubes.

It has also been demonstrated that cellular stress caused by rotenone or TNF- causes TNT to develop between epithelial cells. It has been demonstrated that lipopolysaccharide or interferon-induced inflammation increases the expression of proteins involved in the production of TNT.

Inhibition: A different study utilizing cytochalasin B discovered influenced TNT production without the destruction of preexisting TNTs. Cytochalasin D, an F-actin depolymerizing chemical, inhibited the formation of TNT-like structures known as streamers. It was discovered that latrunculin-B, another F-actin depolymerizing substance, entirely prevents TNT production. Blocking CD38, which has been connected to astrocytes releasing mitochondria, also markedly reduced TNT production. TNT synthesis is known to be mediated by TNFAIP2, also known as M-Sec, and shRNA reduction of this protein decreased TNT development in epithelial cells by around two-thirds.

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CONCLUSION

Whole mitochondria have been suggested as a possible method of transmission across cells using tunneling nanotubes. Nanotechnology claims that cancer cells can physically tunnel nanotubes to take over immune cells' mitochondria. Although the precise level of damage required to cause TNT creation is still unknown, it appears that mitochondrial DNA damage is the primary catalyst for the formation of TNTs in order to transport complete mitochondria. The greatest speed of mitochondria over TNTs was discovered to be about 80 nm/s, which is less than the observed speed of 100-1400 nm/s of mitochondria being transported along axons. This could be because the TNTs' smaller diameter prevents mitochondria from migrating over them.

In one investigation, researcher employed four lines of mesenchymal stem cells, each of which expressed the Rho-GTPase Miro1 with a different phenotype. A higher level of Miro1 was connected to more effective mitochondrial translocation *via* TNTs. Through the specific inhibition of TNT synthesis, numerous investigations have demonstrated that TNTs constitute a key mechanism for the transport of whole mitochondria between diverse cells.