

Researchers redefine hypothesis on holes in the brain

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Researchers at University of Copenhagen have studied access conditions at brain cell level. A new study explodes existing paradigm that huge channels uncritically perforate cell membranes.

Over the years, researchers have described how some of the body's <u>cells</u> have giant channels – a kind of holes that completely uncritically allow both small and large molecules to penetrate into and out of the cell. The hypothesis is that these normally closed gatekeeper proteins in the <u>cell membrane</u> allow unrestricted access in the event of diseases such as <u>myocardial infarction</u>, stroke or Alzheimer's. If the hypothesis was correct, the obvious choice would be to look for novel drugs to block the relevant membrane proteins and in this way cure or prevent disease.

New research findings published in the Journal of Biological Chemistry



unfortunately show that the hypothesis is a bit too optimistic. A new study conducted by researchers from the Department of Cellular and Molecular Medicine and the Department of Biomedical Sciences at the University of Copenhagen indicates a more complicated picture. The holes apparently do not act as unrestricted pathways.

Nanna MacAulay, the researcher behind the new results that disprove an existing paradigm within cell biology:

– If there are huge channels in the cells that open, e.g. in response to hypoxia or inflammation of the brain, it would be very interesting to focus on these channels as targets for new drugs. Over the years, many researchers have formulated how such channels could open in the cell membrane in connection with various diseases. Laboratory tests now show that the channels in question behave differently than previously described, being far more restrictive and behaving differently depending on the type of <u>channel</u>.

The researchers have studied the <u>membrane</u> proteins connexin and pannexin and their ability to lead various chemical compounds into and out of the cells.

More information: *Journal of Biological Chemistry*, www.jbc.org/content/289/38/26058.full.pdf+html

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