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Editorial

Cell, calcium and psychiatry

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Calcium plays an important role in regulating neuronal function. One of the most important features in this regulation is the alteration of the intracellular calcium concentration $[Ca^{2+}]_i$, since many hormones, neurotransmitter or electrical stimulation exert their effect in part by raising $[Ca^{2+}]_i$, thus activating calcium-dependent enzymes that direct the cellular response. This several thousandfold increase in $[Ca^{2+}]_i$ from extremely low resting levels ($-10^{-9}M$) is tightly controlled by calcium membrane channels, calcium pumps, and release or uptake of calcium from membrane surfaces related to the second messenger system of the cell such as IP_3 or G-proteins. It was a tempting idea that this highly regulated functional apparatus could be altered in a specific way in psychic disorders.

The use of this concept became possible after calcium-dependent mechanisms, similar to those in neuronal cells, had been discovered in non-excitable, peripheral cells. For example, some membrane- or receptor-like mechanisms in lymphocytes are very similar to neuronal ones, but the functional context, in which these cells become activated is immunological and, as yet, only speculatively related to neurobiological models.

However, in this early phase of the psychoimmunological approach, emphasis on common features might be more fruitful than focusing on opposing facts.

This issue summarizes the present state of the art in using non-excitable, peripheral cells as models of altered cellular function in psychic disorders, such as depression or dementia. The methodological part of most papers is quite large, reflecting the need for careful exploration of the experimental possibilities in the present phase.

Although these models may suggest to use them as "markers" in the context of "state or trait", we would like to primarily use them as indicators of physiological functions, which we should understand far better before generating marker concepts.