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Trabecular gel network capable of phase transition may exist in brain neurons

B Kovács*, F Gallyas

Department of Neurosurgery, University of Pécs, Pécs, Hungary

In the course of many neurological diseases, individual non-apoptotic neurons randomly distributed among undamaged neurons become dramatically shrunken, hyperbasophilic and argyrophilic („dark" neurons). The shrinkage is caused by a potentially reversible, striking reduction of all distances between undamaged ultrastructural elements (compaction), which is accompanied by the escape of the excess water through visibly intact plasma membrane into adjacent astrocytes. The same ultrastructural compaction can also be produced by momentary physical forces, such as head injury and electric shock, even under post-mortem circumstances that are extremely unfavorable for enzyme-mediated biochemical processes. From this, we (Gallyas et al. 2004) concluded that the ultrastructural compaction consists in a pure physical phenomenon.

It is known from polymer chemistry (Annaka and Tanaka, 1992) that certain synthetic gels can have two or more metastable phases, each having a discrete minimum of non-covalent free energy, a discrete set of conformations of the macromolecules constituting the gel matrix, a discrete water content (and therefore a discrete volume), and also a discrete set of physical and chemical properties. Initiated at a single point, transition from one gel phase to another, the essence of which is a cooperative conformational change in the macromolecules constituting the gel matrix, can spread throughout the whole gel volume, propelled on the domino principle by the difference in non-covalent free energy. The initiation can be brought about by a subtle change around a critical concentration of various chemical substances or a critical degree of various physical forces, and was assumed to comprise the central mechanism in most cell functions in which mechanical work is involved (reviewed by Pollack 2001).

By analogy with the above physicochemical phenomenon, we (Gallyas et al. 2004) deduced from several enigmatic phenomena concerning the "dark" neurons that the morphological substratum of the dramatic compaction of the ultrastructural elements is a ubiquitous intracellular gel structure capable of spreading phase transition at the expense of stored non-covalent free energy. In a recent paper we (Kovács et al. 2007) raised the idea that this gel structure does not fill completely and evenly the spaces between the ultrastructural elements, but exists in the form of an unbroken network of interconnecting trabeculae embedded in a confluent system of lacunae filled with fluid cytoplasm. Through these lacunae water molecules can rapidly reach the plasma membrane from any point of the cell, resulting in the same degree of compaction both in the core and the periphery of the affected neurons. Furthermore, this idea can reconcile the membrane-derived properties (e.g. ion channels) with gel-derived properties (see Pollack 2001). Finally, this trabecular gel network might be the *in-vivo* (unfixed) equivalent of the strongly criticized microtrabecular lattice demonstrated in aldehyde-fixed and subsequently freeze-dried cells in the high-voltage stereo electron microscope, and interpreted as a "solid" component of the cytoskeleton (reviewed by Porter 1989). By gradually forming covalent bonds, aldehyde fixation stabilizes the energy-storing phase of the trabecular gel structure.

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*Corresponding author
E-mail: fiona55@freemail.hu