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The gap junction as a biological rosetta stone: Implications of evolution stem cells to homeostatic regulation of health and disease in the barker hypothesis

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The discovery of the gap junction structure, its functions and the family of the connexin genes, has been basically ignored by the major biological disciplines. These connexin genes code for proteins that organize to form membrane-associated hemichannels, connexons, co-join with the connexons of neighboring cells to form gap junctions. Gap junctions appeared in the early evolution of the metazoan. Their fundamental functions, (e.g., to synchronize electrotonic and metabolic functions of societies of cells and to regulate cell proliferation, cell differentiation and apoptosis), were accomplished via integrating the extra-cellular triggering of intra-cellular signaling and therefore, regulating gene expression. These functions have been documented by genetic mutations of the connexin genes and by chemical modulation of gap junctions. Via genetic alteration of connexins in knock-out and transgenic mice, as well as inherited connexin

mutations in various human syndromes, the gap junction has been shown to be directly linked to many normal cell functions and multiple diseases, such as birth defects, reproductive, neurological disorders, immune dysfunction and cancer. Specifically, the modulation of gap junctional intercellular communication (GJIC), either by increasing or decreasing its functions by non-mutagenic chemicals or by oncogenes or tumor suppressor genes in normal or initiated stem cells and their progenitor cells, can have a major impact on tumor promotion or cancer chemoprevention and chemotherapy. The overview of the roles of the gap junction in the evolution of the metazoan and its potential in understanding a system view of human health and aging and the diseases of aging will be attempted.

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