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A conceptual integration of extra-, intra- and gap junctional-intercellular communication in the evolution of multi-cellularity and stem cells: How disrupted cell-cell communication during development can affect diseases later in life?

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An attempt will be made to provide a short conceptual review to integrate, from an evolutionary perspective, how the emergence of gap junctional intercellular communication helped to bring about multi-cellularity and new adaptive phenotypes. This new fundamental biological function of the metazoans was needed to provide homeostatic control of new cellular functions of an interacting society of different cell types existing in a 3-dimensionalunit. Changing paleo-physics- and -chemistry of the earth led to single celled organisms that metabolized sugar via glycolysis and survived via symmetrical cell division and occasional mutations. With the appearance of oxygen-producing phytoplankton, the single cell organism, the mitochondrion, symbiotically- fused with a primitive cell to form the first multi-cellular organism, which could metabolize glucose via oxidative phosphorylation. The new society of adherent cells developed new strategies for adaptive survival. New genes and phenotypes included: growth control, differentiation, programmed cell death; senescence; regulation of gene expression-"epigenesis"; germline and somatic stem cells; asymmetrical cell division; and anoxic stem cell niches. The evolutionary development of the normal human organism, starting from a single "toti-potent" stem cell to the mature, reproductive and self-aware being, consisting of over 100 trillion cells, of which 200 different cell types and having three major functional cells- (organ-specific stem cells; their progenitor derivative cells; and their differentiated daughters), could only come about by a delicate homeostatic integrated feedback system of extra-, intra- and gap junctional inter-cellular communication (GJIC). Since GJIC occurs in all organs, any disruption of the three forms of cell communication mechanisms by genetic or epigenetic factors, particularly during embryonic, fetal and neonatal periods, could lead to alteration of risks to diseases later in life (i.e., the Barker hypothesis). Chronic disruption of these signaling mechanisms in the adult organs could also lead to several kinds of chronic, stem cell-based diseases, diabetes, cancer, atherogenesis and premature aging.

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