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Molecular and Metabolic Pathways Underlying Parthenogenetic Activation and Early Embryonic Development in Mammals

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Abstract *Parthenogenesis involves initiating embryonic development from an unfertilised oocyte. In mammals, experimentally induced parthenogenetic activation provides a valuable model for investigating the mechanisms that regulate the oocyte-to-embryo transition. However, early developmental competence must be distinguished from the ability to produce viable offspring. This review examines the molecular signalling pathways and metabolic processes involved in mammalian parthenogenetic activation, with particular attention to factors that support or limit subsequent embryonic development. The molecular component focuses on intracellular calcium signalling and its role in releasing the oocyte from meiotic arrest. The paper discusses calcium-dependent activation of calmodulin-dependent protein kinase II, changes in M-phase-promoting factor activity, and the regulation of cell-cycle progression in relation to meiotic resumption, pronuclear formation, and the initiation of cleavage. Electrical and chemical activation approaches are considered as experimental tools for triggering these events in the absence of fertilisation. The metabolic component addresses mitochondrial function, ATP production, carbohydrate metabolism and redox balance as determinants of developmental competence. It focuses on the coordination between calcium signalling and energy metabolism, including the potential contribution of AMPK–mTOR signalling to the oocyte-to-embryo transition. We examine oxidative stress and antioxidant defence in relation to embryo survival and progression towards the blastocyst stage. The review also considers genomic imprinting as a major barrier to mammalian parthenogenetic development, highlighting the consequences of an exclusively maternal genome for embryonic and extraembryonic development. By connecting activation signals, metabolic requirements and epigenetic constraints, this work provides an integrated framework for understanding mammalian parthenogenesis and its applications in reproductive biology and experimental embryology.*

• Introduction

Parthenogenesis is a particular form of reproduction in which an embryo develops without the fusion of the oocyte and the sperm cell. It occurs naturally in many invertebrates (aphids, insects, water flies) but can also be observed in some vertebrates, including lizards, snakes, fish. In mammals, parthenogenesis does not occur spontaneously. The oocyte can be experimentally activated and thus develop into a parthnote, which only contains maternal genetic material. Studies have shown that these embryos can develop until a certain stage, but the development is limited. In mice, parthenotes developed until the forelimbs became visible. Parthenogenesis involves three stages: oocyte activation, cleavage and formation of the blastocyst. The activation triggers an increase in intracellular calicium. Pathenogenesis also has a practical use in human reproductive medicine. In IVF and ICSI cases when normal oocyte activation fails, artificial oocyte activation can be very useful.

• Discussion

During normal fertilisation, the mature mammalian oocyte is arrested in metaphase II by MPF(maturation-promoting factor) and CSF (cytostatic factor). The sperm then, during fertilization, triggers calcium signalling , which leads to the completion of meiosis and thus to the beginning of the embryonic development. Calcium plays a vital role in this process. An increase in calcium insides the cell activates CaMKII (calmodulin-dependent protein kinase II), which then leads to changes in MPF activity, allowing the cell cycle to continue.

Mammalian oocytes can be experimentally activated through electrical and chemical stimuli. Frequently used chemical stimuli include: calcium ionophores, for example Ionomycin; ethanol and strontium chloride. These methods mimic what a sperm cell would under normal circumstances do. These stimuli induce an calcium oscillation that will lead to cycle resumption. However, this activation does not guarantee a succesful embryonic development.

The early development is conditioned by the metabolism. An essential role plays the mitochondria, that produces ATP through oxidative phosphorylation by using pyruvate, fatty acids and glutamine. When ATP decreases, AMPK (AMP kinase) is activated and it regulates ATP synthesis. Furthermore, mTOR (mechanistic target of rapamycin) controls cell growth, protein synthesis and metabolism.

Nontheless, there is a major barrier: genomic imprinting. Parthenotes contain exclusively maternal genetic material, which can lead to atypical gene expression. This affects directly the embryonic developement, explaining why parthenogenetic embryos usually do not develop into viable offsprings.

•Conclusions

Parthenogenesis is a complex process, that depends on many components, such as: activation, metabolism and genomic imprinting. Understanding the mechanism that lies behind parthenogenesis provides us a new way to look at reproductive biology, assisted reproduction, as well as regenreative medicine.