



Fatty Acid Metabolism in Pre-implantation Embryo Development

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Abstract

During the activation and maturation of follicles, as well as the normal development of oocytes and early embryos, glucose, lipids and amino acids-the three energy substrates-are interconvertible and collectively provide energy for these processes. Abnormal metabolism of fatty acid frequently leads to functional dysregulation, inducing reproductive disorders including ovarian function decline, recurrent spontaneous abortion and endometriosis, as well as abnormalities in oocyte and embryo quality. This review aims to comprehensively summarize the characteristics of fatty acid metabolism during reproductive processes and its relationship with the development of oocytes and early embryos, in order to provide novel insights for further research.

Keywords: Fatty Acid Metabolism; Oocyte quality; Embryo development

Introduction

Lipids are a diverse and complex class of macromolecules. Lipids are stored in the form of triglycerides and play an important role in a variety of biological functions, including biofilm construction, cell signaling, steroid precursor production and metabolism, as well as being an important source of nutrients. Lipids are divided into saturated fatty acids and unsaturated fatty acids. Common saturated fatty acids include palmitic acid, stearic acid, myristic acid and so on. Common unsaturated fatty acids include linoleic acid, oleic acid, linolenic acid, arachidonic acid and so on. Fatty acid concentration in the body is regulated by diet and storage lipid decomposition[1]. Fatty acids are involved in the regulation of mammalian reproduction and embryo development, usually stored in the cytoplasm in the form of triacylglycerol (TG). The cytoplasm of mammalian oocytes and embryos contains a large amount of lipids, and high-quality oocytes often contain more linoleic acid and arachidonic acid [2]. Excess fatty acids are partly stored in triglyceride form, partly in free form, partly in mitochondria via β -oxidation provides energy, partially retains cytoplasm, and may undergo peroxidation and generate reactive oxygen species (ROS). ROS may also affect the endoplasmic reticulum calcium pool and induce endoplasmic reticulum stress. Mitochondria are considered to be the main

subcellular structure from which ROS are derived. Mitochondria have at least 10 sites capable of producing ROS [3]. It is reported that fatty acid concentrations in the body increase in both excess and insufficient nutrients, and are associated with low embryonic development potential in humans, cattle and pigs. In order to reveal the regulatory role of fatty acids in oocytes and early embryo development, this paper reviews the research progress in this field.

Characteristics of lipids in oocytes

Fatty acid is an important energy source for oocyte and embryo development. It is an important energy substance that can be directly utilized by oocytes. Oocytes are rich in lipids, most of which are stored in lipid droplets in the form of triglycerides. Lipid droplet is a spherical organelle capable of storing lipids. Lipid droplet is not a typical phospholipid bilayer. It is surrounded by phospholipid monolayer and hydrophobic core composed of triglyceride, cholesterol ester and other neutral lipids. There are also many proteins distributed on the surface[4]. Lipid droplet is a dynamically distributed organelle. Its number, size and distribution are affected by cell type, nutritional state and metabolic state of the body. Generally, the abundance of energy substances such as glycolipids in the body will increase the number and size

of cellular lipid droplets, and store excessive fatty acids in lipid droplets, reducing the toxic effects of lipids; on the contrary, under energy-limited conditions, in order to maintain cellular lipids and energy balance, lipid droplets will be degraded through lipolysis and/or lipophagy metabolic pathways[5]. It is found that the lipid content of oocytes of different species varies greatly, among which the lipid content of porcine oocytes is higher, that of bovine and ovine oocytes is moderate, and that of mouse or human oocytes is less[6]. It is generally believed that the lipid content of oocytes reflects the different ability of oocytes to rely on β oxidation of fatty acids, and the specific reasons are not very clear.

During the maturation of animal oocytes, the distribution of lipid droplets changes dynamically and is closely connected with mitochondria. Before in vitro maturation of oocytes, lipid droplets are large and distributed in clusters, but after in vitro maturation, lipid droplets are small and distributed in a scattered manner; In addition, after oocyte fertilization and during early embryo development, the content of lipid droplets tends to decrease, and lipid droplets are also small and distributed in a scattered manner, which may be related to changes in the fatty acid composition of triglycerides. The lipid droplets of mature oocytes are clustered, and when the medium lacks pyruvate and glutamine, the clustered lipid droplets disappear and the lipid droplets become uniform again[7]. During the development of animal follicles, oocytes accumulate abundant lipid droplets, and the number of lipid droplets associated with mitochondria and endoplasmic reticulum increases gradually, suggesting that oocytes accumulate lipid droplets during development to store energy for meiosis and maturation, and to accumulate a large amount of energy for fertilization and early embryo development. Lipid droplets act as dynamic lipid storage organelles. Triglycerides in lipid droplets are degraded into glycerol skeleton and fatty acids with different chain lengths and saturations by lipase cleavage or lipophagy metabolic pathway. Then, they enter cells through fatty acid protein transporters on cell surface, including fatty acid translocase, tissue-specific fatty acid transporter and plasma membrane-bound fatty acid binding protein, and then enter cells through fatty acyl carnitine transferase 1. Carnitine acyltransferase (CPT1) crosses the outer membrane of mitochondria, and then enters the inner membrane of mitochondria through carnitine, and then fatty acids enter β oxidation to release acetyl coenzyme (CoA), enter the tricarboxylic acid cycle (TC Cycle) to produce adenosine triphosphate (ATP), providing energy [8]. Follicular fluid is mainly secreted by granulosa cells, and also contains local capillary exudation serum, and also includes some plasma components and some active products secreted by theca cells. Follicular fluid is the microenvironment for oocyte development. The lipid metabolism state of maternal follicular fluid is very important for oocyte development and is an important factor determining oocyte and embryo quality. It participates in regulating oocyte maturation, especially meiosis and epigenetic modification. The content of various metabolites in follicular fluid can reflect the developmental potential of oocyte to a certain extent[9].

Oocyte development depends on granulosa cells, which are divided into cumulus granulosa cells and parietal granulosa cells. Cumulus granulosa cells are the only somatic cells in direct contact with oocytes. Cumulus granulosa cells are rich in genes related to fatty acid metabolism, which can regulate the number of lipid droplets in follicular fluid and oocytes, ensure that oocytes have enough fatty acids for beta oxidation and provide energy for meiosis. When the concentration of fatty acids in follicular fluid increases, excess fatty acids accumulate in cumulus cells, which are able to absorb these non-esterified fatty acids to form lipid droplets, and cumulus cells express fatty acid binding protein 3, which is able to transport fatty acids to oocytes to synthesize lipid droplets. When cumulus cells decompensate, oocytes suffer from lipid toxicity due to excessive lipids. Cumulus cells can alleviate the adverse effects of free fatty acids on oocytes in a short time, but long-term high-level lipid stress will cause lipid toxicity, which will lead to abnormal function of endoplasmic reticulum and mitochondria of cumulus cells and oocytes and induce apoptosis[10]. It is known that oocytes spend more than 99% of their time in follicles from growth to maturity. The lipid droplet content and fatty acid composition of oocytes growing and developing in animal follicles are affected by dietary lipid level and metabolic state of the body. Therefore, the metabolic changes in follicular fluid affect the developmental potential of oocytes, oocytes cannot be completely separated from their follicular environment, and metabolic coupling between oocytes and their companion cumulus cells is essential to maintain their normal functions.

Energy metabolism during oocyte maturation-carbohydrate metabolism

The energy consumed by oocyte maturation comes partly from cumulus cells. Sugar and lipid are decomposed in cumulus cells and provide energy for oocyte development through gap junction. However, the main energy source is produced by mitochondria of oocyte, and the substrates of energy metabolism are glucose, lipid and amino acid. cumulus-oocyte complex There are four pathways of glucose metabolism in cumulus-oocyte complex (COC), namely glycolysis pathway, pentose phosphate pathway (PPP), hexosamine biosynthesis pathway (HBP) and polyol pathway [11]. The main metabolic pathway of cumulus cells is glycolysis pathway[12], and the production efficiency is low in anoxia or hypoxia; Oocytes metabolize glucose mainly through mitochondrial oxidative phosphorylation under aerobic conditions, and the production efficiency is the highest. Pyruvate produced by cumulus cells through glycolysis is absorbed by oocytes and enters their mitochondria, and is then metabolized by tricarboxylic acid cycle (TCA). NADH and reduced flavin adenine dinucleotide (FAD) H₂ molecules produced from TCA are used in electron transport chain to produce ATP by oxidative phosphorylation[13,14]. NADPH and phosphoribose pyrophosphate (PRPP) produced by PPP are transferred to oocytes via gap junctions. NADPH acts in the reduction of antioxidant glutathione to neutralize ROS, while

PRPP is a substrate for the synthesis of nucleotides required for DNA repair and meiotic regulation. Glucose metabolized by HBP produces N-acetylglucosamine, a factor secreted by follicular cells (OSF) that regulates follicular cell glycolysis. Fatty acids are beta-oxidized to CoA in mitochondria, and then converted to ATP through TCA cycle/oxidative phosphorylation metabolism. Oocytes have weak ability to metabolize glucose directly and need pyruvate produced by glycolysis of cumulus cells as a substrate for energy metabolism. Oocyte maturation includes nuclear maturation and cytoplasmic maturation, both of which are energy-dependent processes. In the process of in vitro maturation of oocytes, regulating the mode of carbohydrate metabolism, improving energy metabolism, realizing synchronization of oocyte maturation and nuclear cytoplasmic maturation is beneficial to improving the quality and development potential of oocytes matured in vitro. Metabolism and ATP level of oocytes and cumulus cells are closely related to oocyte quality and healthy development of embryos.

Compared with granulosa cells, mammalian oocyte mitochondria are generally round, with smooth inner membrane, fewer cristae, more number and lower methylation degree. Mitochondrial DNA(mt DNA) copy numbers are high in both oocytes and early embryos, suggesting that oocytes and early embryos are at a low level of energy metabolism, maintaining a low level of electron transport chain to efficiently meet their own energy needs, while minimum capacity can reduce the formation of ROS associated with oxidative phosphorylation, the so-called quiet embryo hypothesis [15]. When glucose transport in oocytes and follicular cells is impaired, metabolic mechanisms such as lipids may be activated to compensate for energy requirements and increase fatty acids as alternative energy sources [16]. Metabolites related to β -oxidation were found in oocytes from germinal vesicle (GV stage) to metaphase II of meiosis, (L-carnitine, palmitoyl-L-carnitine) gradually increased, suggesting that fatty acid decomposition energy supply increased, but also indicates that β -oxidation plays an important role in oocyte maturation process, and provides energy reserve for embryo development after fertilization. Energy metabolism of oocytes depends on mitochondria, and mitochondria of fertilized eggs all come from oocytes, so energy metabolism of oocytes affects energy metabolism of embryos and may also affect energy metabolism of next generation[17].

Energy metabolism during oocyte maturation-lipid metabolism

The essential lipid metabolism pathway in oocytes is mainly β -oxidation. The lipid content and mitochondrial metabolic activity in oocytes are considered as important markers of oocyte developmental potential. Fatty acids are characterized by high energy value and can produce more ATP than carbohydrates or other substances. One molecule of palmitic acid can produce 105 ATP, while one molecule of glucose is about 31 ATP. Therefore,

fatty acid β -oxidation is considered as an important energy source during oocyte maturation. Beta oxidation is a multi-step metabolic process. More specifically, oxidation involves the decomposition of long fatty acids that have been converted into acyl coenzyme A chains into smaller fatty acyl coenzyme A chains. This reaction releases acetyl coenzyme A, FADH₂ and NADH, which then enter another metabolic process called the citric acid cycle or TCA, in which ATP is produced as energy. Oxidation continues until two acetyl CoA molecules are produced and the acyl CoA chain is completely broken down. In eukaryotic cells, oxidation occurs in mitochondria, while in prokaryotic cells, oxidation occurs in the cytoplasm. In order to undergo beta oxidation, fatty acids must first enter the cell through the cell membrane, fatty acids are activated as fatty acyl carnitines, which make it into mitochondria, carnitine palmitoyl transferase II (carnitine palmitoyl transferase II,CPTII) removes carnitine, fatty acyl coenzyme A molecules enter mitochondrial matrix for beta oxidation and TCA cycle to produce ATP. Short chain fatty acids can directly enter mitochondria for beta oxidation in order to maintain lipid homeostasis through lipid synthesis and decomposition balance in cells. In addition to providing energy, lipid cholesterol is an important structural component of cell membranes and a precursor of sterols with important biological functions such as steroids, bile acids and vitamin D. Phospholipids are also important components of bio membranes and signal molecules for many important signaling pathways in the body. They are also important precursors of second messengers such as diacylglycerol, phosphatidic acid, lysophosphatidic acid and arachidonic acid, which trigger activation of various signaling pathways. Oocyte maturation involves a lot of reorganization of organelles and cytoskeleton, which requires a lot of energy. Therefore, mitochondria need to provide sufficient levels of ATP at precise times and locations for energy. Therefore, factors that disturb mitochondrial function can become obstacles to oocyte cytoplasmic maturation. Mitochondrial inheritance is entirely maternal, and impaired mitochondrial function in oocytes has direct effects on early embryos, such as cleavage arrest, abnormal cytokinesis, and blastomere fragmentation, which may be caused by mitochondrial driven apoptosis.

The fatty acid metabolism in the cumulus-oocyte complex is regulated by maternal physiology and the external environment, and has an important impact on the developmental potential of the oocyte. The developmental potential of the oocyte is related to the concentration of oleic acid and stearic acid in follicular fluid., exposure to different free fatty acids Free fatty acid (FFA) can not only affect granulosa cells and cumulus cells indirectly, but also affect oocyte quality directly, thus affecting in vitro fertilization. In vitro fertilization (IVF) has an impact on pregnancy outcome [18]. The quality of high density lipoprotein in follicular fluid is an important factor in determining oocyte quality, and various unsaturated fatty acids in follicular fluid play an important role in follicle development, follicle maturation, follicle quality and oocyte quality through various signaling pathways.

Adding activators or inhibitors of fatty acid β -oxidation to oocyte culture medium can change lipid metabolism in oocytes, which can prove that β -oxidation is necessary for meiosis recovery, maturation and early embryo development of oocytes. In addition, L-carnitine can also regulate glucose metabolism and enhance the activity of enzymes involved in oxidative respiratory chain [19]. However, transcripts involved in L-carnitine biosynthesis pathway do not exist in human oocytes and cumulus cells [20]. After application of fatty acid oxidation inhibitor etomoxir, glucose consumption increases, indicating that there is a complementary relationship between glucose metabolism and lipid metabolism in oocyte energy supply [21]. There is great potential for improving human oocyte quality by supplementing L-carnitine to the culture medium during in vitro culture.

The role of lipid metabolism in oocyte and early embryo development

During preimplantation embryo development, there are two distinct events, cell proliferation and cell differentiation, which occur simultaneously and harmoniously. Cell differentiation is largely determined by transcriptional regulation and epigenetic regulation at molecular level, while cell proliferation is related to metabolism at molecular level. Metabolomics and transcriptomics analysis showed that there were more methionine, polyamine and glutathione metabolism in 2-cell stage of embryo, and higher metabolites related to mitochondrial tricarboxylic acid cycle in blastocyst stage [22], indicating that energy demand increased gradually with embryo development. Oocyte and cleavage stage embryo oxygen consumption is lower than morula and blastocyst, when embryo development to morula and blastocyst, in addition to carbohydrate energy supply, beta oxidation also plastically provide energy to meet the energy requirements of embryo development.

β -oxidation of fatty acids plays an irreplaceable role in preimplantation embryo development. Pyruvate is produced by cumulus granulosa cells through glycolysis and supplied to oocytes, which metabolize pyruvate to generate energy through oxidative phosphorylation [23]. Early embryos are similar to oocytes before genome activation and can support embryo development by low levels of oxidative metabolism of pyruvate, lactic acid and amino acids. Blastocyst stage embryos show high levels of glycolysis and oxygen consumption as embryo genome activation and blastocyst cavity formation occur.

During embryo culture in vitro, fatty acids in culture medium can be absorbed by embryos and transported into mitochondria for β -oxidation to produce acetyl coenzyme A (Acetyl-CoA), acetyl-CoA can enter TCA cycle to supply energy for cells. During the development of embryos from 8-cell stage to blastocyst stage, β -oxidation activity of fatty acids in embryos was significantly increased compared with that before 8-cell stage. Acyl co-A dehydrogenase, a key enzyme in the beta-oxidation process. When LCAD (Long-chainyl-Coa dehydrogenase) was knocked out,

blastocyst formation was obviously blocked. There were a large number of lipid droplets (LDs) during early embryo development [24, 25]. If the autophagy degradation pathway of lipid droplets was forcibly initiated in embryos, blastocyst formation rate decreased, which indicated that the existence of lipid droplets in cells could affect normal embryo development. In addition to their role as energy substrates, lipids also play other roles during early embryonic development-the construction of biological membranes. Phospholipids such as phosphatidylcholine (Phosphatidylcholine), Phosphatidylethanolamine Phosphatidyl ethanol (PE) is an essential component of cell membrane and is essential for embryonic development. Choline kinase, a synthetic enzyme of PC and PE, (Cholinekinase, Chka) knockout conditions, PE, PC synthesis blocked, cell membrane composition disorder, and then lead to embryo development arrest in blastocyst stage. Similarly, knockout of phosphocholine cytidine transferase (PCYT1), a key enzyme in phospholipid synthesis, also causes embryo death. Lipids can also indirectly affect embryonic development, such as phospholipase A2 in the body when glucose-6-phosphate dehydrogenase (G6PD) function is impaired. (Phospholipase A 2, PLA2) activity will increase, so that NADPH content will decrease, lipid peroxidation level will increase, Lys phospholipid content will increase, which will directly lead to changes in cell membrane characteristics, such as changes in membrane structure and osmotic pressure, and then cause embryo development arrest [26].

Fatty acids are beta oxidized in mitochondrial matrix to produce ATP for early embryo development. Pyruvate and free fatty acids are taken up into oocytes from follicular fluid or surrounding media. Free fatty acids are stored in lipid droplets as neutral triglycerides (TAGs) or absorbed by mitochondria for beta oxidation. Under high-fat conditions, such as obesity or a high-fat maternal diet, excess free fatty acids are retained in the cytoplasm and may cause lipid peroxidation, ROS production, fragmentation of human embryos and apoptosis. Free fatty acids may also directly affect endoplasmic reticulum calcium (Ca^{2+}) storage, causing endoplasmic reticulum stress and increasing intracellular Ca^{2+} levels. There was a significant correlation between lipids in human serum and lipids in follicular fluid, especially cholesterol, high density lipoprotein, triglyceride, free fatty acid, apolipoprotein A1 and carnitine. Carnitine synthase is expressed very low in oocytes and highly in cumulus cells; CPT and some enzymes involved in fatty acid beta oxidation are highly expressed in oocytes and some enzymes involved in fatty acid beta oxidation are highly expressed in cumulus cells [27,28].

Abnormal lipid metabolism affects oocyte and early embryo development

Female fertility is reflected in ovarian function and egg quality determined by it. Although the egg matures and fertilizes in vitro, its development in vivo is highly dependent on the microenvironment before fertilization, including follicular cells

and follicular fluid. In follicles, the developing egg is surrounded by cumulus cells, which are connected with parietal granulosa cells; these two types of granulosa cells play a decisive role in maintaining egg quality. The cumulus cells are regarded as "nutrient cells" and support the egg by providing paracrine factors such as pyruvate and metabolic substrates. The parietal granulosa cells exchange metabolites, growth factors and signaling molecules with the cumulus cells through cell junctions. In addition, follicular fluid serves as another substrate source, and its nutritional composition depends on maternal blood, so the composition of follicular fluid and eggs reflects maternal nutritional status. Metabolomic analysis of follicular fluid during ART cycles revealed lipid metabolism-related metabolic intermediates in follicular fluid of women with low ovarian response (POR) compared to normal women. The levels of oleic acid, linoleic acid, arachidonic acid and hexacosahexanoic acid in serum and follicular fluid of PCOS women were significantly increased, while the levels of lactic acid and pyruvate were significantly decreased compared with normal women. Therefore, the balance between carbohydrate metabolism and lipid metabolism in mitochondria of oocytes in POR, elderly and PCOS populations is lost, resulting in the decline of oocyte and embryo quality [29, 30]. Research analysis shows that the quality of oocytes can be affected by changing the oocyte itself and its microenvironment, increasing the number and quality of embryos that can be transferred, and finally improving the outcome of assisted pregnancy.

Changes in Oocyte Lipid Metabolism Affect Embryo Development Outcome

Oocyte β -oxidation-related genes are regulated by LH surge in maternal oocytes, but in vitro oocyte maturation Under the condition of In vitro maturation (IVM), FSH and epidermal growth factor make the gene expression related to lipid metabolism of oocytes malregulated, and the lipid composition in serum of IVM culture medium is different from that in follicular fluid during in vivo maturation, all of which lead to the malregulation of gene expression of lipid metabolism and the defect of β -oxidation during IVM. The addition of L-carnitine to IVM medium increased the first polar body ejection rate of oocytes, increased mitochondrial DNA copy number, decreased ROS, increased ATP, increased steroid hormone production, and thus increased oocyte maturation rate and early embryo development potential. Obese women are prone to infertility and have a high probability of fetal and neonatal problems after pregnancy. Studies have shown that obese women have high expression of genes regulating oxidative stress, lipid metabolism and inflammation, and increased concentrations of linoleic acid and stearic acid. Balancing saturated and unsaturated fatty acids in follicular fluid of obese women and changing lipid composition ratio in IVM culture medium will improve the developmental potential of obese women's oocytes and early embryos. The obese mice model established by high fat diet showed obvious reduction of ovary size, decrease of ovarian reserve, abnormal mitochondrial morphology of oocytes, increase

of meiotic chromosome aneuploidy, decrease of blastocyst formation, increase of early embryo loss rate, and defects appearing before early blastocyst, which led to fetal retardation and abnormal brain development. It may be that higher free fatty acids in GV and MII oocytes led to higher ROS levels in cells and endoplasmic reticulum stress. This results in oocyte maturation disorders and low embryo development potential [31].

Fat synthesis is essential for embryonic development, knockout of fatty acid synthase (FAS) gene died before implantation; adding an inhibitor of fat synthesis, cerulein, to IVM medium could promote meiosis recovery of COC oocytes, but could not promote the maturation of naked eggs; adding C57 to promote fatty acid beta oxidation and inhibit fatty acid synthesis could promote meiosis recovery of COC and naked oocytes. These in vitro experiments demonstrate that fatty acid beta-oxidation plays a greater role than fatty acid synthesis in oocyte meiotic recovery. Cell metabolism can be actively involved in regulating cell pluripotency and early embryonic development, and can even determine cell fate. Mechanisms affecting cell fate decisions focus on metabolism-signaling pathways such as AMPK/mTOR pathway, and the involvement of key metabolic intermediates a-ketoglutarate, S-adenosyl-methionine, and acetyl-coenzyme A in regulating epigenetic modifications. Maternal environmental exposure can affect one or more generations through epigenetic modification, and it is necessary to further explore the mechanism of maternal environmental changes affecting embryo and offspring development, especially the role of oocyte metabolic disorders in it [32].

Summary and outlook

Female infertility or reduced reproductive capacity has become a global health problem, which has attracted increasing attention. To explore the mechanism of lipid metabolism in oocytes and early embryos can provide therapeutic basis for the treatment of infertility caused by premature ovarian failure, poor quality oocytes, poor quality early embryos and abnormal embryonic development, and provide strategies for preventing and controlling infertility, and provide theoretical support for individualized assisted reproductive therapy for infertile women and elderly women. Studying the relationship between fatty acid metabolism and early embryonic development will not only improve our understanding of development, but also lay a theoretical foundation for revealing the molecular mechanism of energy metabolism in the process of embryogenic diseases, and provide new theoretical guidance for clinical treatment of female infertility and female reproductive system diseases, and improve female fertility and pregnancy outcome of assisted reproduction.

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