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Placenta-on-a-Chip Technology in Teratogenicity Research

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Abstract: Teratogenicity remains a major concern in maternal and fetal health, as exposure to drugs, chemicals, environmental pollutants, infectious agents and other harmful factors during pregnancy can adversely affect normal embryonic and fetal development. Such exposure may result in congenital malformations, growth restriction, functional abnormalities and even fetal loss. For many years, animal models have been the primary approach for evaluating developmental toxicity. However, differences in placental structure and function between humans and animals often limit their ability to accurately predict human fetal responses. In recent years, advances in microfluidic technology have led to the development of placenta-on-chip, an innovative in vitro platform that closely mimics the human placental barrier and maternal–fetal interface. This technology provides a more physiologically relevant system for investigating placental transport, drug transfer and the effects of teratogenic agents. This review presents an overview of the principles of placenta-on-chip technology, its applications in developmental toxicity and teratogenicity research, along with its advantages, current challenges, and future perspectives. By offering a more human relevant alternative to conventional models, placenta-on-chip has the potential to improve developmental toxicity assessment while supporting efforts to reduce reliance on animal testing.

Key words: placenta-on-a-chip; organ-on-chip; teratogenicity; developmental toxicity; microfluidics; feto-maternal barrier; trophoblast; new approach methodologies

1. Introduction

Teratology is the science of studying and investigating the birth defects and their etiologies Teratology is the science of studying and investigating the birth defects and their etiologies Teratology is the science of studying and investigating the birth defects and their etiologies Teratology is the science of studying and investigating the birth defects and their etiologies Teratology is the science of studying and investigating the birth defects and their etiologie Teratogenicity can be defined as the ability of any drug, chemical substance, environmental toxicant, infectious agent, or physical factor to interfere with normal embryonic/fetal development, leading to fetal malformations, growth retardation, functional defects, or even fetal death. Teratology is the science of studying and investigating birth defects and their causes. Exposure to teratogens during pregnancy can lead to congenital anomalies, growth restrictions, pregnancy loss and fetal deaths. Since directly experimenting on pregnant women is both unethical and unfeasible, researches conduct the experiments on animal subjects such as rats, rabbits and mice according to the international

safety guidelines such as OECD Test Guideline 414 for prenatal developmental toxicity. OECD is the guideline for the internationally prenatal developmental toxicity studies and it is abbreviated as organization for economic co-operation and development test, guideline 414 for prenatal developmental toxicity. It helps to determine the toxicity to the embryo and foetus. The tragedy caused by thalidomide use in pregnant women during late 1950's and early 1960's demonstrated the limitations of animal studies. The thalidomide drug was considered absolutely safe in animal subjects but it caused phocomelia in thousands of babies when taken by pregnant women. This occurred because of humans and animals are different in placental structure and function.

Earlier, the placenta was considered a passive organ that separated the maternal and fetal compartments. Researchers recognized that the placenta is a dynamic organ which not only supplies the oxygen and nutrients, produce hormones, facilitate waste products, regulates maternal-fetal exchange and acts as a selective barrier. In addition, the placenta metabolizes drugs by using metabolic enzymes and transports them to the foetus, some of which lead to harm to the foetus. Although these mechanisms provide partial protection, some harmful substances cross the placental barrier and effect fetal development. Although the animals are used as the primary approach for conducting of experiments and evaluating developmental toxicity, there are significant differences between the human and animal placenta with respect to structure, function, metabolic activity transporter proteins, metabolic enzymes and physiology. Apparently, drug may also behave differently in animals and humans, which may lead to differences in humans and animals. To overcome these limitations, researchers developed a human placental model known as placenta-on-a-chip, which mimics the structure and functions of the human placenta.

To overcome these limitations of conventional animal and static in vitro models, placenta-on-a-chip technology has emerged by performing same like human placenta. The placenta- on- a -chip consists of two chambers one representing maternal and other representing fetal compartment, which are used to evaluate the toxicity of a drugs and other xenobiotic. The placenta- on- a -chip has gained increasing popularity because of its potential as a novel approach methodology for improving development toxicity assessment while reducing dependence on animal subjects.

This review summarizes the principles of placenta-on-chip technology and discusses its applications in studying placental transport, teratogenicity, and developmental toxicity, highlighting its potential as a novel approach methodology for reducing reliance on animal testing.

2. Teratogenicity: Definition and Concept

Teratogenicity is the ability of a drug or other substance to cause congenital abnormalities or developmental defects in the developing embryo or fetus. These abnormalities may be structural (physical malformations) or functional (intellectual or developmental disorders). The process through which these abnormalities develop is known as teratogenesis. The term teratogenesis is derived from Greek words "Terato" meaning "monster" and "genesis" meaning "formation" and describes the development of structural or functional abnormalities during embryonic or fetal development. The agents that are responsible for causing these defects are known as teratogens.

The teratogenic effect of a substance depends on several factors such as the nature of the teratogen, genetic background of the embryo and most importantly, the timing of exposure during pregnancy. The highest risk of teratogenicity is observed during organogenesis, which occurs in the first two months of pregnancy (up to approximately 10 weeks of amenorrhea), when the developing embryo is particularly susceptible to structural abnormalities due to active organ formation. Depending on the extent of exposure, teratogenic effects may range from no observable changes to structural malformations, functional impairments, intrauterine growth restriction, fetal or embryonic death, stillbirth and transplacental carcinogenesis.

3. Historical Perspective on Teratogenicity

The understanding of teratogenicity has evolved considerably over centuries, progressing from mythological and super natural interpretations to a well-established discipline known as teratology. In ancient civilizations, congenital malformations were commonly attributed to supernatural forces, divine

punishment or mystical influences because their actual causes were not yet understood. However, advances in embryology, pathology and developmental biology gradually replaced these traditional beliefs with scientific evidence leading to the emergence of teratology, the branch of science dedicated to the study of congenital anomalies and their underlying causes.

The foundations of experimental teratology were established during the early 20th century through studies demonstrating that environmental factors could adversely affect embryonic development. One of the earliest experimental observations reported that exposure to X-rays during pregnancy resulted in developmental abnormalities including microcephaly. Studies conducted during the 1930s demonstrated that maternal vitamin A deficiency in pregnant pigs resulted in congenital malformations, particularly ocular defects. These findings provided convincing evidence that environmental factors, in addition to genetic influences could disrupt normal embryonic and fetal development. During this period, Josef Warkany recognizes as the founder of experimental teratology, demonstrated that exogenous agents were capable of inducing congenital developmental defects in mammals, thereby establishing the scientific foundation of modern teratology.

A major turning point in the history of teratology occurred during the early 1960s following the thalidomide tragedy. Thalidomide initially introduced as a sedative and prescribed for the management of nausea and morning sickness during pregnancy, was subsequently linked to severe congenital malformations in thousands of infants worldwide. Prenatal exposure to thalidomide was found to cause limb reduction defects (Phocomelia), as well as abnormalities involving the ears, eyes, heart and other organs. This unprecedented event established a definitive relationship between maternal drug exposure and fetal developmental abnormalities, fundamentally transforming the field of teratology. It led to the implementation of stringent regulatory guidelines, mandatory developmental toxicity testing and preclinical safety evaluations before the approval of pharmaceuticals for use during pregnancy.

Subsequent research expanded the understanding of developmental toxicity by identifying the roles of nutritional deficiencies, radiation exposure, infectious agents, environmental chemicals and pharmaceuticals in disrupting normal embryogenesis. Advances in molecular biology have significantly enhanced the understanding of mechanisms underlying teratogenicity. These studies have demonstrated that teratogens can disrupt normal embryonic development by altering cellular proliferation, differentiation, apoptosis, gene expression, enzymatic activity, membrane function and fetal growth. Collectively, these scientific advancements have transformed teratology into a multidisciplinary field, providing the foundation for improved developmental toxicity assessment, safer drug development and evidence based clinical decision-making during pregnancy.

4. Teratogens

A teratogen is any agent that adversely affects embryonic or fetal development during pregnancy and results in congenital abnormalities, growth restriction, functional impairment or fetal death. The teratogenic effects of teratogens are influenced by factors such as the timing, dose, duration of exposure and genetic susceptibility of both the mother and the developing fetus.

Teratogens are broadly classified into physical, chemical, infectious and maternal disease related teratogens based on their origin and mechanism of action. Understanding the classification of teratogens is essential for identifying potential risk factors, implementing preventive strategies and minimizing adverse effects during pregnancy.

4.1 Physical Teratogens

Physical teratogens consist of physical agents capable of disrupting embryonic and fetal development. They primarily include ionizing radiation and maternal hyperthermia. Ionizing radiation is one of the most studied physical teratogens. Exposure during pregnancy, particularly throughout the critical period of organogenesis, is associated with an increased risk of growth restriction, neurodevelopmental impairment and fetal death. The severity of these effects depends on the radiation dose and the stage of pregnancy at which exposure occurs.

4.2 Chemical Teratogens

Chemical teratogens comprise a diverse group of substances capable of crossing the placental barrier and adversely affecting normal embryonic and fetal development. They include pharmaceutical agents, recreational substances and environmental chemicals, all of which have the potential to induce structural, functional, or developmental abnormalities following prenatal exposure. The teratogenic effects of these agents are influenced by several factors, including the chemical properties of the substance, the dose and duration of exposure and the stage of fetal development at the time of exposure. Depending on these factors, chemical teratogens may interfere with critical cellular and molecular processes involved in embryogenesis, thereby increasing the risk of congenital malformations, growth restriction, neurodevelopmental impairment and fetal loss.

4.3 Infectious Teratogens

Infectious teratogens comprise pathogenic microorganisms capable of crossing the placental barrier and disrupting normal embryonic and fetal development. They include viral, bacterial and parasitic infections, which can result in a wide spectrum of adverse pregnancy results including congenital malformations, fetal growth restriction, neurodevelopmental abnormalities and pregnancy loss, depending on the type of pathogen and the timing of maternal infection during pregnancy.

4.4 Maternal Disease-Related Teratogens

Maternal disease-related teratogens refer to pre-existing or pregnancy-associated maternal medical conditions that adversely affect normal embryonic and fetal development. Conditions such as diabetes mellitus and maternal phenylketonuria are associated with an increased risk of congenital abnormalities when they are not controlled during pregnancy. The teratogenic effects of these conditions are influenced by the severity of the maternal disease and the timing of exposure during fetal development.

5. Significance and Importance of Teratogenicity Testing

The use of drugs during pregnancy has increased considerably over the years. However, the extent of placental drug transfer and the potential effects of maternally administered drugs on fetal development remain largely unknown. Therefore, there is a need for reliable and innovative approaches to evaluate fetal drug exposure, particularly during the first trimester of pregnancy. This period is the most critical for teratogenicity because organogenesis occurs during early fetal development, making the fetus highly susceptible to congenital abnormalities caused by drug exposure.

Recent advances in placenta-on-a-chip technology have provided a promising platform for improving our understanding of placental biology and drug transport mechanisms. This systematic review highlights the potential applications of placenta-on-a-chip models in studying the transfer of drugs and other compounds across the placental barrier. Studies have demonstrated that these models can be used to investigate glucose transport, evaluate the transfer of various therapeutic agents, understand the mechanisms of placental development, simulate the function of placental efflux transporters and assess the effects of nanoparticles on placental barrier integrity.

Furthermore, placenta-on-a-chip technology has been successfully employed to recreate pathological conditions affecting the placenta, including bacterial infections, placental disorders and preeclampsia. These disease models provide valuable insights into placental dysfunction and its influence on fetal drug exposure and development.

Overall, placenta-on-a-chip technology represents a promising alternative to conventional in vivo and ex vivo models for teratogenicity testing. By closely mimicking maternal blood flow and the physiological microenvironment of the human placenta, it offers a more accurate representation of placental function. In addition, this technology reduces the need for invasive procedures or direct fetal tissue sampling, thereby minimizing risks to both the mother and the developing fetus. Consequently, placenta-on-a-chip systems have significant potential to improve the prediction of drug safety during pregnancy and support the development of safer therapeutic strategies for pregnant women**.**

6. The Human Placental Barrier: Structure and Relevance to Teratogenicity

Only eutherian mammals possess a placenta, which is discoid because of its shape haemochorial. The placenta is attached to the uterine wall and generates between the mother and fetus through the umbilical cord. The mature human placenta is a discoid organ 20-25cm in diameter, 3cm thick and weighing 400-600g. During the establishment of the placenta the mother and foetal tissues come in direct contact without any immunological rejections.

The placental barrier is a biological interface that separates the maternal and fetal compartments, while permitting the exchange of gases, nutrients, hormones and drugs for fetal development. The placenta is located within the chorionic villi. Each chorionic villus consists of a core containing fetal capillaries which are surrounded by trophoblast layers. By the fusion of cytotrophoblast cells, the syncytiotrophoblast cells are formed and it is a continuous multinucleated epithelial layer. The syncytiotrophoblast layer is directly exposed to maternal blood and serves as a primary site for the exchange of essential substances from maternal to fetal and vice versa.

Under the syncytiotrophoblast lies discontinuous cytotrophoblast cells, followed by the villous stroma. The villous stroma contains extracellular matrix, immune cells, connective tissue cells, fetal capillaries together with fetal endothelium are called as structure of placental barrier. The essential substances transported from the mother to foetus they must travel through all these successive layers. The structure of the placental barrier changes significantly throughout the pregnancy.

The placental barrier plays a crucial role in determining fetal exposure to teratogenic agents. Even though it functions as a selective interface between the maternal and fetal circulations, it does not completely prevent the transfer of harmful substances. Many chemical teratogens, including prescription medications, non-medical drugs, alcohol, nicotine, and environmental pollutants, are capable of crossing the placental barrier and directly affecting developing fetal tissues. Similarly, infectious agents such as rubella virus, cytomegalovirus, Toxoplasma gondii and Treponema pallidum can cross the placenta, resulting in congenital infections and developmental abnormalities. The severity of teratogenic effects depends not only on the ability of these agents to cross the placental barrier but also on the timing, duration, and dose of exposure. By crossing the placental barrier, these teratogens are causing functional impairment, growth restriction, disorders, fetal loss in the fetus.

7. Placenta-on-Chip: Design Principles and Technological Evolution

The placenta-on-chip concept was introduced in the mid of 2010_s. The placenta-on-chip design is made of polydimethylsiloxane and consists of two chambers. They are maternal and fetal compartments which are separated by a thin semi-permeable membrane. The trophoblast cells are seeded on the maternal side membrane. The endothelial cells are seeded on the fetal side. The trophoblast cells are called as BeWo b30 cells and endothelial cells are called as HUVEC cells. The chambers are separated by extracellular matrix membrane.

The placenta-on-chip design is based on several principles that help to produce a maternal placental environment in vitro. The principles are:

7.1 Biometric layers

The first principle is biometric layers. This is achieved by introducing trophoblast cells and fetal endothelial cells on the porous extracellular matrix membrane.

7.2 Dynamic Flow

The second principle is dynamic flow. This principle generates an environment which applies continuous fluid shear stress, supplies nutrients, oxygen and facilitates waste removal between the maternal and fetal compartments.

7.3 Selective transportation across the placental barrier

The third principle is Selective transportation across the placental barrier. The combination of thin porous semipermeable extracellular matrix membrane, trophoblast cells and endothelial cells facilitates selective transportation between the maternal and fetal compartments. This regulates the study of multiple transportation like passive transportation, active transportation and facilitated transportation to

exchange of gases, drugs, hormones, xenobiotics, pathogens, and nutrients between the maternal and fetal compartments.

8. Applications of Placenta-on-Chip in Teratogenicity Research

8.1 Pharmaceutical Transfer and Fetal Drug Safety

The isolated human placental lobule perfused in vitro is the best model for predicting drug transfer and transport from the mother to the fetus. Data from a perfused intact tissue. Where structural integrity and cell-cell organization are preserved, more closely resembles the in vivo situation than subcellular preparations or tissue homogenates. Additionally, the experimental conditions can be controlled and confounding metabolic and physiological influences are reduced.it is crucial to keep in mind that there may be variations in the transplacental transfer of medications due to notable variations in the placenta`s function during the first two months of pregnancy (histotrophic nutrition) and later in the pregnancy (hemotrophic nutrition). Studies on young placentas that function during the organogenesis phase are necessary, even the majority of our knowledge comes from research on term placenta

The ability of drugs to pass through the placenta is also influenced by the activity of placental phase I I and II Drug metabolising enzymes, which vary in concentration during pregnancy. Cytochrome P450 (CYP) enzymes, in particular, have been extensively studied in the placenta, with their presence confirmed at the levels of mRNA, protein, and enzyme function. Several CYP enzymes, including CYP1A1,2E1,3A4,3A5,3A7, and 4B1, have been identified in the mature placenta. Although less is known about phase II enzymes in the placenta, some such as uridine diphosphate glucuronosyltransferases, have been found to have specific activity against known substrate markers, indicating an important role for these enzymes in the detoxification of drugs in the placenta

8.2 Environmental Toxicants and Endocrine-Disrupting Chemicals

The endocrine system consists of a variety of glands that release hormones to regulate metabolism, growth, tissue development, reproductive and sexual functions, as well as sleep and mood, among other physiological processes. The use of synthetic chemicals by humans has significantly increased since their introduction. Endocrine-disrupting chemicals (EDCs) are a diverse group of synthetic and natural substances that can interfere with different functions of the endocrine system, potentially leading to negative health effects for those exposed. Recent reports indicate that approximately 800 chemicals commonly used in everyday life exhibit endocrine-disrupting properties. Of the EDCs available, only a limited number have been thoroughly studied. These substances are linked to several chronic health conditions sych as Cardiovascular problems, diabetes, obesity reproductive abnormalities.

In 2009, The Endocrine Society written the first Scientific Statement on EDCs sending the concerns to community health, established evidence from animal models, dispassionate remarks and epidemiological studies.6 The report involves evidence of the belongings of endocrine disrupters on male and female duplication, conscience growth, prostate and conscience malignancy, neuroendocrinology, thyroid, absorption and corpulence, and cardiovascular endocrinology, and entails an raised understanding of the belongings of EDCs, accompanying the difficulty of individual and controlled humankind collaborators in writing and executing changes honestly procedure and knowledge

8.3 Nanoparticle Risk Assessment

Nanotechnology continues to be an emerging multidisciplinary science platform that is often defined as creating products and applications based primarily upon the synthesis of molecules in the nanoscale (10^{-9} meter) size range. The term “nano” arises from the Greek term, referring to “dwarf”. What makes the technology intriguing is that particle properties may often become altered as they are reduced below 100 nm in size. For example, gold particles may transform to blue or red pigments. Electrical insulating functions of certain particle types may become conductors at the nanoscale. In short, particle properties can be modified to promote different and more flexible applications, resulting in consumer benefits, particularly in the areas of medical and industrial applications. From a material science perspective, this represents an exciting challenge because as the particulate material size decreases in the direction of the

nanoscale (particularly below 100 nm), the physical properties of particles are known to be altered and can be harnessed to create new products and applications. Because of the economic potential, commercializing various types of engineered nanomaterials for a range of applications, including industrial, consumer, medical, and diagnostic, clearly presents a challenge for companies and regulators to ensure the development of safe and effective products for consumers. Therefore, assessments of potential hazards associated with this technology and corresponding products have become emerging areas for health risk assessments. This understanding represents an element of the broad engagement process with stakeholders and potential customers with regard to environmental health and safety.

8.4 Infection, Inflammation, and Placental Injury

The histological pattern known as acute chorioamnionitis (ACA) is defined by maternal neutrophilic inflammation of the placental membranes with or without a fetal neutrophilic co-response. ACA is caused by bacteria, mycoplasma, or fungi which gains access to either the membranous decidua or the amnionic epithelium. Meticulous microbiological studies have confirmed that micro-organisms can be isolated in virtually all cases of ACA.30, 31, 32 The spectrum of organisms isolated from the placenta.

9. Limitations and Challenges

Although placenta-on-chip technology has emerged as a promising alternative to conventional models, there are several limitations. several challenges still limit its broader application. Most current platforms depends on immortalized trophoblast cell lines rather than primary human placental cells, which may not fully mimic the physiological characteristics of the human placenta. Another limitation is many researchers uses different types of chip design, membrane materials, cell sources, and experimental protocols. As a result it is difficult to compare findings across studies and establish standardized methods. Most of the current using models donot involve all the cell types in human placenta such as immune, stromal and decidual cells are often absent, making difficult for creating micro environment like placenta. The use of placenta-on-chip technology also requires advanced equipment, technical expertise, and considerable cost, making it difficult to implement on a large scale. Therefore, further improvements and validation studies are required before placenta-on-chip can be routinely adopted for developmental toxicity assessment.

11. Future Directions

Although placenta-on-chip technology has shown considerable potential in teratogenicity research, further improvements are required to enhance its application in developmental toxicity studies. Future research should focus on the use of primary trophoblasts or human induced pluripotent stem cell (hiPSC)-derived trophoblasts, as these may better represent the characteristics of the human placenta. The incorporation of additional cell types, including decidual, immune and stromal cells, may further improve the ability of the model to mimic the in vivo placental environment. In addition, integrating placenta-on-chip models with other fetal organ-on-chip systems may provide a better understanding of the transfer of teratogens across the placenta and their effects on fetal organs. Further studies are also needed to establish standardized protocols and validation criteria to improve the reproducibility and wider acceptance of these models. Moreover, the application of advanced imaging techniques and artificial intelligence-based data analysis may improve the evaluation and interpretation of experimental findings, thereby supporting future developments in placenta-on-chip technology.

12. Conclusion

Placenta-on-a-chip is an in vitro model that mimics the structure and functions of the human placental barrier. Compared with animal models, placenta-on-a-chip provides more accurate and physiologically relevant information for evaluating maternal-fetal transport and assessing the developmental toxicity of drugs, chemicals and other harmful substances. The placenta-on-a-chip also has certain limitations, as it cannot completely replicate like human placental microenvironment. However, with continued research and technological advancements, placsenta-on-a-chip has the potential to become an important Novel Approach Methodology (NAM) for developmental toxicity testing.

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