Effect of Hypoxia in Uterus on the Expression of MBP and NF-H+L in the White Matter of Old Rats

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Abstract: Hypoxia in utero is a common problem in embryonic development. Early fetal and neonatal periods are periods of rapid brain growth and development, and their development depends on adequate oxygenation. The growth and development of the fetus in the mother's uterus require an adequate supply of oxygen. Conversely, lack of oxygen can adversely affect fetal development. The purpose of this article is to study the effect of intrauterine hypoxia on the expression of MBP and NF-H+L in the white matter of the offspring of aged rats and establish a hypoxia model of SD rats through controlled experiments. Detect fetal rat blood gas and blood ion indexes and use immunohistochemistry to detect the expression of MBP and NF-H+L in the paraventricular white matter of the offspring of the offspring of rats. Parallel image analysis, and then observe the myelin sheath and axis in the offspring of the offspring under electron microscope ultrastructure of protrusions and microvessels. The results show that the main effect of intrauterine hypoxia can reduce the expression of MBP (1666.93, 2179.85, 432.72) and NF-H+L (721.266, 1785.832, 246.512) in the offspring of the white matter in the elderly rats (all P<0.05)), MBP was highly positively correlated with NF-H+L (R=0.64, P<0.01). Observation by electron microscopy showed that compared with the blank control group, the offspring of the intrauterine hypoxia group showed more myelination, axon damage, and microvascular disease in the brain of the elder generation of rats. Therefore, intrauterine hypoxia can affect the expression of MBP and NF-H+L in the white matter of offspring in the offspring of the offspring, resulting in demyelination and damaged axons.

Keywords: Hypoxia in Utero, Fetal Development, White Matter in Old Age, MBP and NF-H+L
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The growth and development of the fetus in the uterus requires an adequate supply of oxygen. Conversely, hypoxia can adversely affect fetal development. Hypoxia in utero is a common problem during embryonic development. If the mother's malnutrition, hypoxia, drug stimulation, or placental function, etc., may lead to reduced blood flow in the uterus and placenta, decreased oxygen supply to the fetus in the womb, and insufficient nutritional intake by the mother. Therefore, the growth and development of the fetus does not conform to the genetically determined growth trajectory, growth retardation and developmental defects make the fetus develop slowly, causing intrauterine growth restriction, resulting in small birth weight, poor health, and reduced survival rates. Early fetal and neonatal periods are periods of rapid brain growth and development and depend on sufficient oxygen.

The malignant damage of hypoxia and ischemic globulin is closely related to the malignant

apoptosis of glial leukocytes with little mutation [1]. However, there are few in-depth studies on the large amount of intrauterine hypoxia and its important role in the control of prenatal fetal procedures and the increase in the prevention of physical and mental diseases in adult later years [2]. There is no clear evidence of clinical anthropology and epidemiological medical research that can prove that prenatal intrauterine hypoxia is associated with an increased risk of cardiovascular disease in later life in adults [3]. The clinical diagnosis rate of various leukocyte diseases such as leukocyte diseases is getting higher and higher, and the clinical importance of various leukocyte diseases related to chronic stroke, dementia, etc. is getting more and more attention.

At present, many scholars have started to study the problem of hypoxia in the womb. The Popovtseva study found that acute hypoxic hypoxia in late pregnancy can cause changes in the biochemical composition of atrial fibrillation. These changes are characterized by high lactate concentrations, and the fetus and uterus may be the source of elevated lactate levels in AF [4]. Carreiro evaluated the effect of resveratrol, a polyphenol in grapes and red wine, on the uterus of non-pregnant rats under hypoxic conditions. The results show that resveratrol can regenerate and protect uterine hypoxia in nonpregnant mice and describe for the first time that these effects are achieved by blocking ATP-sensitive potassium channels [5]. Wu used QRTPCR and WESTERNBLOT methods to detect the expression of HIF-1α. QRT-PCR detected the levels of Bola-A and MICB mRNA. The results showed that the expression of HIF-1α increased in the hypoxic cell model. The expression of Bola-A increased in BEECS, while the level of MICB decreased, which provided a basis for exploring the intrauterine environment during pregnancy [6]. Keenaghan adult IUGR male rats (dam offspring fed on a calorie-restricted diet during pregnancy) were exposed to acute hypoxic stress and cardiac function was assessed by echocardiography. In adult IUGR-born rats, mitochondria from RV are sensitive to hypoxia, which translates into a poorly adapted RV cardiac respons e to acute hypoxia [7]. Richardson reviewed the brain metabolic activity and behavioral status of the entire late fetus, and the methods used to study sheep fetuses for long-term testing. It also outlines methods for studying fetal hypoxia in sheep through placental embolization and umbilical cord occlusion [8].

The research results of this paper are intended to further explore the direct effects of intrauterine hypoxia on the functional expression of white matter F-MBP and white matter NF-H+L in aged mice and mice. Through the experiments of two control groups, the hypoxia response model of intrauterine cells in two groups of ASD rats was established, and each control group of rats was divided into two groups. The results showed that the main side effects of intrauterine hypoxia can be used to reduce the MBP (1666.93, 2179.85, 432.72) and NF-H+L (721.266, 1785.832, 246.512) of white matter cells in aged rats. The clinical innovative features of this article are as follows: First, the expression of MBP and NF-H+L in the central ventricular chronic narcolepsy tissue of elderly male rats can be detected by the rat immunohistochemical detection method. Electron microscopy observation results showed that compared with the early blank control group of mice, the hypoxic reaction group of mice had a significantly higher incidence of benign myelin sheath loss, benign axonal injury and intracranial microvascular benign lesions. high.

THE RELATIONSHIP BETWEEN DIFFUSE HYPOXIA AND WHITE MATTER DAMAGE

Hypoxia and White Matter Damage

White matter has the effect of achieving higher brain function, which can lead to a series of clinical manifestations, including alertness, forgetfulness and personal changes, even dementia and coma and death. Exposure to icy conditions will stimulate short-term and long-term emergency control mechanisms, including inflation and erythropoiesis, at levels sufficient to improve oxygen delivery to vital organs such as the brain, and these compensation mechanisms are insufficient to meet these needs. Oxygen in the system, especially when the brain continues to lack oxygen, will show

structural changes [9].

Myelin sheath is essential for nerve activity. Oligodendrocytes are responsible for the formation of myelin; electrical wires covering myelin cells; (ASTRO) glial cells usually regulate the metabolism and inflammation of structures. It contains myelin sheath nerves in the central nervous system. The myelin sheath in each internal cell is a multinucleated membrane, forming a layer of circular oligodendrocytes circulating in neon axons. Oligodendrocytes can regulate many cellular processes, sometimes up to 40-50. Each cell shape surrounds the next axon, forming part of the myelin sheath and forming the inner body. Therefore, the sex of myelin in liquid substances can be derived from several different oligodendrocytes, and one oligodendrocyte can also provide a part of myelin to several or several nerve fibers [10].

The essence of the human body is from birth to several years after birth, and different brain nerve pathways use different withdrawal periods. From the posterior fossa cerebellum to the brain, the white matter of the brain and myelin sheath, and finally the subcortical white matter, form the occipital bone, parietal lobe, and early and temporal beats. The development of MRI provides a framework for the development of the myelin sheath in the brain (IR). T2W imaging shows the formation of the myelin sheath in the anatomy: 5 months in the white matter of the cerebellum, 4 months in the corpus callosum, 6 months in the corpus callosum knee, and internal capsule forelimb 3 months, frontal white matter 8 months.

Anatomical Features of White Matter

Brain proteins come from two sources of blood, namely the arteries that penetrate the dotted light on the surface of the brain and the terminal branches of the choroidal or striatum arteries. Perforated arteries in the arteries on the surface of the brain originate from right-angled arachnoid blood vessels, pass through the vertical area of the brain surface along the myelinated fibers, and then enter the white matter area. These blood vessels emit short vertical branches to supply white matter, and each short branch of a single perforated artery forms a cylindrical metabolic unit. The blood concentration in the white matter area adjacent to the side wall of the ventricle originates from the subventricular artery of the choroidal release branch or striatum artery. These blood vessels are about 15 mm long, and the anastomosis between them is sparse or missing.

This type of blood vessel causes the arterial boundary (or watershed) of the white diseased area around the blood vessel, and is particularly vulnerable to the reduction of systemic or local blood flow through the brain. The white matter of subcortical structures can be divided into subcortical areas and periventricular areas. The protein in the vicinity of the cortex (3-4 mm wide) is composed of so-called U-fiber dense short circuits, and is also provided by the long-perforated and short-perforated arteries, protein and the adjacent cortex [11]. The area around the ventricle is composed of many long joint fibers that connect subcutaneous structures, such as the striatum and cortex. The damage of white matter around the chamber mainly affects these long fibers. Adult white matter has less blood flow than gray matter and poor blood vessel regulation [12]. Lesions that are destroyed in the white matter area also cause the gap around the small blood vessels to expand, which in turn leads to cortical atrophy.

Hypoxia and Oligodendrocytes

The malignant damage of hypoxia and ischemic globulin is closely related to the malignant apoptosis of glial leukocytes with few mutations. Compared with other oligodendrocyte glial protein cell species, oligodendrocyte cells are more likely to cause local hypoxic and ischemic neuropathy, and may have a local selectivity and vulnerability. Even individual neutral oligodendrocytes located in the brain nerve area may have local transient anaerobic ischemia and are more sensitive to damage than other neurons. In the normal development and growth of the human brain in modern people, between 23 and 32 weeks of gestation, the proteins around the ventricle are often highly ischemic. This is a high risk of infection of hypoxic fetal encephalopathy, which is more likely to directly

cause the protein around the fetal ventricle (PVL) is softened, and the main symptoms are seen in premature babies during pregnancy, especially premature babies born at low age. Protein cell damage is associated with decreased sensitivity of tissue ischemia and cell hypoxia to less mutated glial leukocytes and a line of mature cells. After ischemia and cell hypoxia, different cell types can reduce the performance of synaptic glioblastoma and show different physiological vulnerabilities. Mature future differentiated neutral oligodendrocyte attacks glial leukocytes are more easily exposed to large ischemic neural stress cell attacks than mature non-undifferentiated oligodendrocyte attacks. The four consecutive cycle stages of oligodendrocyte receptor glial protein cell receptor development can be roughly divided into different cell types of receptor surface cell antigens, including early oligodendrocyte receptor glial cell receptor progenitor cells and late oligodendrocyte receptors. Glial cell receptor progenitor cells, mature oligodendrocyte receptor glial protein cells, different oligodendrocyte receptor glial protein cells, different oligodendrocyte receptor glial protein cells. The use of these genetic markers indicates that the progenitor cells in the late oligodendrocyte receptor glial are already the main synthetic cells of proteins belonging to humans: the progenitor cells in the late oligodendrocyte receptor glial are sensitive to this because at this early stage, cells dominate the risk zone of ischemic hypoxic encephalopathy. In contrast, mature oligodendrocytes can tolerate hypoxia.

Repair and regeneration of the myelin sheath associated with residual axons in the lesion area after ischemia. The identification of specific markers provides convincing evidence: in diffuse protein damage, the death of oligodendrocyte precursors is caused by lipid peroxidation (ROS) and protein nitration (NOS). This finding indicates that reactive oxygen clusters and reactive nitrogen clusters attack the pattern of cell destruction. In addition to these direct brain observations, a recent study found that lipid peroxidation levels of premature infants with PVL confirmed by ML were significantly increased and had markers of oxidized protein products in cerebrospinal fluid.

Hypoxia and Pathological Changes of White Matter

The typical response of white matter to various harmful factors is demyelination changes. Less than 2 months of cerebral perfusion can cause myelin sheath and axon damage, astrocyte proliferation, myelin sheath first damages the axon, it may be that the myelin sheath is produced by oligodendrocytes, according to reports, oligodendrocytes are prone to affected by various pressures, and constitute the composition of white matter. Under ischemic and hypoxic conditions, oligodendrocytes are more vulnerable to damage than other cells. It has been confirmed that culturing oligodendrocytes under hypoxic conditions will produce myelin basic protein (MBP), which reduces reversibility. MBP is a specific marker of myelin sheath, therefore, the reduction of oligodendrocyte synthesis is caused by a possible cause of myelin damage.

The second possible cause is extensive or MBP-specific proteolysis. There have been many reports on the activation of calcium-dependent proteases under hypoxic-ischemic conditions. Studies on hypoxic optic nerve have shown that the reduction of extracellular calcium has a protective effect on the axon cytoskeleton. Hypoxic-ischemic white matter injury is closely related to oligodendrocyte apoptosis. Compared with other glial cells, oligodendrocytes are more sensitive to hypoxic-ischemic injury and have the characteristic of easily destroying regional selectivity. Even oligodendrocytes from a single brain region are more sensitive to transient ischemic injury than neurons. During the development of the human brain, within 23 to 32 weeks of pregnancy, the paraventricular white matter is hypoxic or ischemic abnormal. The high-risk period of encephalopathy is that it may cause softening of brain protein cells (PVL) located around the ventricle. This is mainly seen in late preterm birth babies, especially for late preterm birth babies with low preterm birth weight. The damage of leukocyte is related to the effect of tissue ischemia and cell hypoxia on the cell vulnerability of the less mutant glial leukocyte

differentiation lineage. After ischemia and cell hypoxia, different cell types of glial oligodendrocytes and glial leukocytes will have different physiological fragility. Incompletely differentiated mature oligodendrocyte receptor glial protein cells are more likely to receive ischemic and unstressed biological attacks than mature non-undifferentiated oligodendrocyte receptor glial protein cells. Few mutations are the four consecutive cycle stages of glial leukocyte antigen development. We can accurately classify the surface cell antigen types according to the specificity of different antigen types.

EXPERIMENTAL ANIMALS AND EXPERIMENTAL DESIGN OF INTRAUTERINE HYPOXIA MODEL

Materials and Equipment

The Laboratory Animal Center of the Medical College provided 60 pregnant SD rats. 75x65x35cm hypoxia box, S-450 oxygen detection alarm (manufactured by IST-ATM), OlympusBH-2 camera microscope (provided by the Department of Anatomy of Medical University), MBP antibody and NF antibody-H (both provided by Darko, USA), KIT-5001 and KJT-5004, 1.5% paraformaldehyde, 0.1 M PBS (PH7.2) electron microscope binding solution.

Establish an Intrauterine Hypoxia Model

The adaptor placed the female small rats in a male cage at a 2:1 mating ratio and mated again after two weeks. On the morning of the twenty-third day, the inspector injected the suppository vaginally and performed a vaginal smear urine test on the vagina of the female tumor rats. The tester saw the number of sperm present on the day, and was confirmed as successful sperm fertilization, and also recorded the day as the first day of sperm pregnancy. Random controls in late pregnancy rats were divided into normal hypoxia control group and chronic endometrial hypoxia control group. The anti-hypoxia box with perforated plexiglass is used. The hole in the inside of the box is connected to the external atmosphere of the entire box, so that the balance between the indoor air pressure and the external atmospheric pressure in the entire box can be achieved. Chronic intrauterine compression hypoxia or recombination compression chamber should be filled with a small amount of compressed nitrogen and a large amount of compressed oxygen in order to create a low oxygen environment. The built-in outdoor oxygen concentration detector can continue to detect and can ensure the average indoor oxygen concentration (10 ± 1) %.

A large amount of carbon dioxide and its water vapor in the water of the storage tank are fully absorbed by calcium chloride sulfate and sodium lime sulfate. In a preliminary clinical experiment, after 1 hour of hypoxia in rats, 3 acute pregnancy rats in each group were randomly selected to perform blood gas liquefaction analysis of intrauterine aneurysms, and successfully established chronic intrauterine hypoxemia animal model. From the 7th day of pregnancy until the 21st day, a rat body was put into the hypoxic psychological room during pregnancy every day and psychological intervention of hypoxia during pregnancy was carried out for 8 hours. The clinical conditions of other relevant experiments were the same as those in the chronic endometrial hypoxia experimental group. All Chinese rats are free to choose to eat and drink water safely. A total of 30 young pregnant mice with an average gestational age of 15 days were placed in batches in a hypoxic laboratory. Use the air monitor to monitor the indoor oxygen emission concentration in the room in real time, and automatically adjust the average flow of indoor oxygen and nitrogen in the room in real time. Male rats and GT with 17-1.9g within 1-8 days after birth, 2.3-2.5g between 8-17 days, 2.9-3.8g between 19-21 days, and 60 days of body weight. The weight of male rats is 250g, and all physiological characteristics are within normal flower weeks. As shown in Table 1, according to the "random teaching table method" 28 in utero hypoxic offspring mice were selected, and the 28 mice in the control group were divided into completely random groups.

Table 1.
Three-factor two-level orthogonal design table

		Additional hypoxia (SH)	
		+	-
Intrauterine hypoxia	High fat diet	7	7
	Ordinary diet	7	7
No intrauterine hypoxia	High fat diet	7	7
	Ordinary diet	7	7

Under low-fat rat feeding conventional animal feeding and management conditions, the low-fat rats provided to the low-fat rat animals were fed continuously until 12 months old, and daily high-fat, medium-sugar, low-fat rat animals were normal. Diet (main nutrients: 92.3% of rat animals ate basic high-sugar and low-fat animal feed, 2% high-sugar and low-fat animal cholesterol. According to the design of the American rat feeding experiment, the rat feeding management age. Their offspring rats by 12 months of age. Chronic intermittent hypoxia: Set the hypoxic conditions according to his "hypoxia method: 8 to 12 SD rats are placed in a hypoxic tank at a time and enter from two air inlets respectively oxygen, the other two gas inlets enter nitrogen.

Keep a small air intake gap to communicate with the maximum pressure outside the entire box, in order to balance the maximum pressure inside and outside the entire box. The new water tank is equipped with a large amount of alkaline slip lime and calcium chloride hydrochloride, which is used to absorb a large amount of water vapor and release carbon dioxide. The main body of the alarm device of the s-450 oxygen concentration detector is located at the top center of the oxygen alarm box. The average oxygen and nitrogen concentration flow in water can be automatically adjusted at any time to control the average oxygen and nitrogen concentration flow to 10±0.5%. Alkali white lime and calcium chloride hydrochloride can be used to absorb large amounts of carbon dioxide and other water vapor, respectively. The gas inner wall of the engine room is connected with two nitrogen filling bottles to balance the maximum pressure and the atmospheric pressure of the engine room inner wall. The oxygen concentration of the carbon dioxide released in the box is always less than 3%, and the temperature is always kept at 25±2C until the 20th day of pregnancy.

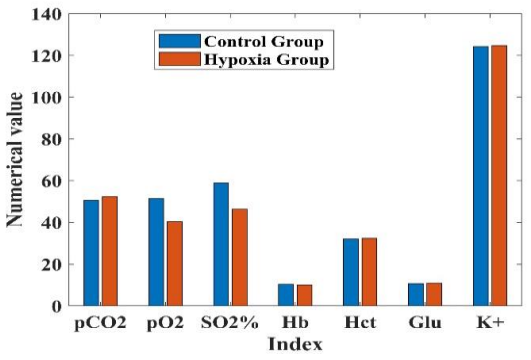
All pregnant rats had normal diet and drinking water. Six pregnant mice in the hypoxia group and the control group were selected for laparotomy at 20 days, and their weight was 0.01 g. Other female rats were born naturally, and male and female offspring rats were divided into cages at 1 month of age. Keep the animal in a cage, eat food and water freely, room temperature 25±2 C, humidity 58%~65%, natural lighting. Select male offspring rats for research. In each group, 6 male offspring rats at 1, 3 and 6 months old were randomly selected and sacrificed. The experimental animals were sacrificed. Sampling: Stability: Ketamine = 1:1. Intraperitoneal injection of 1 mg/500 g dose line after anesthesia. The left ventricular vein was cannulated to the thoracic aortic blood. Emergency thoracotomy, quick dissolution of 0.1 mol/l phosphate buffer solution and 3.5% polyformaldehyde with a large pressure of 200ml to 130cmh2o, individual patients undergo intraperitoneal peritoneal lavage and fixation (both are completed within 30-50 seconds to effectively prevent emergency thoracotomy acute cerebrovascular ischemia caused by hypoxemia in the chest cavity). After 3-5 minutes, remove your brain, cut the 0.4x0.2x0.2cm' brain tissue completely and fix it in the solution under the electron microscope. The rest were fixed in 4% paraformaldehyde-free aqueous solution, dehydrated and embedded, and cut through small ventricle valve plane surgery for postoperative use.

Observation and Determination of Pathological Indicators

Immunohistochemical experiments and staining were performed on rat brain cells MBP and brain F-NF-H+L cells. The specific method is as follows: BS and paraffin defatted rat slice cells were paraffin defatted and hydrated, then washed with PBS and PBS (PH7.4) blocking solution for 3X3 minutes. After removing PBS and PBS using the method, add 50UL selected non-specific blocking antigen hydratase blocking agent to the selected primary antibody in each rat slice. Rinse with PBS (PH7.4) blocking solution for 3X3 minutes. Alkaline limestone and calcium chloride hydrochloride can be used to absorb large amounts of carbon dioxide and other water vapor, respectively. The gas inner wall of the engine room is connected with two nitrogen filling bottles to balance the maximum pressure in the engine room inner wall and the atmospheric pressure of the engine room. The blocking solution was incubated for 30 minutes at each room temperature, and washed with PBS and PBS (PH7.4) blocking solution for 3X3 minutes. After removing the blocking solution of BS and PBS, add 50UL of the selected primary antibody to each rat slice, incubate at room temperature for 60 minutes, and wash with all PBS (PH7.4) staining solutions for 3X3 minutes. Then wash with all PBS (PH7.4) staining solution for 3X3 minutes. Under the microscope, the staining time is controlled according to the color change of the slice. Positive staining is brownish yellow or brownish black, and then washed with distilled water. Balance between the pressure and the external atmospheric pressure. Chronic intrauterine compression hypoxia or recombined compression chambers should be filled with a small amount of compressed nitrogen and a large amount of compressed oxygen in order to create a low-oxygen environment. The built-in outdoor oxygen concentration detector can continue to detect and can ensure the average indoor oxygen concentration (10 ± 1) %. A large amount of carbon dioxide and its water vapor in the storage tank water are fully absorbed by calcium chloride sulfate and sodium lime sulfate.

At 20 days of pregnancy, the fetus was removed by laparotomy, and blood was collected from the amputated fetal head, stored in a heparinized 1 mL centrifuge tube, and the blood of each fetal rat was mixed

Figure 1.
Changes of blood ions and other indexes in cord blood of pregnant rats



for testing. Hydrogen power (pH), oxygen partial pressure (pO_2), carbon dioxide (pCO_2), blood oxygen saturation (SO_2%), hematocrit, hemoglobin (Hit), hemoglobin (Hb), glucose (Glu), lactic acid (Lac) as well as K+ and Na+ levels. At 20 days of pregnancy, an abdominal incision was made and blood was collected from the severed fetus. Blood was collected through the abdominal aorta from the first, third- and sixth-month old offspring of the rats. After centrifugation, the serum was collected and placed in a refrigerator freezer storage tube at -80°C.

Statistical Processing

The experimental data is expressed as mean ± and standard deviation (X ± s), statistical processing is performed with SPSS11.0 software, the umbilical cord blood gas analysis value of pregnant rats is tested by independent sample T, and the cumulative optical density value of MBP and NF-H+L is three. Factor two-level factorial analysis the linear correlation analysis method was used to analyze the cumulative optical density of MBP and NF-H+L. When the test level reaches P<, the difference is significant. Before the analysis, normal test and homogeneity of variance test were conducted.

EFFECT OF HYPOXIA IN UTERUS ON WHITE MATTER OF OLD RATS

Analysis of the Effect of Hypoxia in Uterus on the Indexes of Cord Blood Gas and Blood Ions in

Pregnant Rats

As shown in Table 2, after 1 hour of hypoxia, the pH of pregnant rats dropped to 7.25±0.09, SaO_2 decreased to 0.72±0.20, $PaCO_2$ increased to 7.62±2.27, and PaO_2 decreased significantly to 6.48±2.26.

Table 2.
Mean blood cord blood gas analysis of pregnant rats (x±s)

Group	pH value	PaCO2 (kPa)	PaO2 (kPa)	SaO2
Test group	7.25±0.09	7.62±2.27	6.48±2.26	0.72±0.20
Control group	7.34±0.07	5.53±1.67	10.93±1.83	0.94±0.16

As shown in Figure 1, the blood gas analyzer results showed that compared with the control group, the pO_2 level in fetal rats in the hypoxic group was significantly reduced (40.37±2.06) and the SO_2% level in fetal rat blood (46.33±3.76) was also significantly down. However, it had no significant effect on pH, pCO_2, HCT, Hb, Glu, Lac, K^+, Nat and other indicators in fetal blood (P<0.05). Compared with the plasma ALT and AST of fetal rats in the intrauterine hypoxia group and the control group, the difference in the control group was statistically significant (P<;0.05), while the plasma ALT in the offspring of 1- and 3-months old rats. There was also a significant difference between the level of AST and AST (P<0.05); however, there was no significant difference in ALT and AST plasma levels between the two groups (P<0.05).

Analysis of the Effect of in Utero Hypoxia on the Expression of MBP and NF-H+L in the White Matter of Aging Rats

(1) Observation and analysis of the structure of MBP and NF-H+L in paraventricular white matter

The key expression of MBP is the myelin sheath of the atrium, which is yellowish brown. In the blank test group, the bumps are long and continuous, with excellent straight appearance and neat sorting. In the intrauterine hypoxia group, the hypoxic group and the high-fat diet combined group, white matter MBP showed fracture, intermittent or curved deformation, and the classification was effective. Intrauterine hypoxia fills the hypoxia. In the high-fat diet combination group, the expression of MBP in white matter was further reduced, and it was embellished and fragmented, which was extremely confusing. NF-H+L is expressed in the sympathetic nerves of dark brown anesthetics. The bulge of the blank test group is long flocculent, straight and thin, arranged neatly. In the intrauterine hypoxia group, the hypoxia group and the high-fat diet combined group, the long flocculent expression of NF-HL in the white matter was broken, partially curved, and the order was moderate. As shown in Figure 2, in the intrauterine hypoxia group, the expression of white matter MBP in the additional hypoxia group and the high-fat diet combined with the oxygen group were reduced by 9%, 12%, and 15%, respectively, manifesting as short filamentous disease. The expression of NF-H + L in white matter was further reduced by 18%, 21% and

Figure 2.
White matter MBP and white matter NF-H+L expression

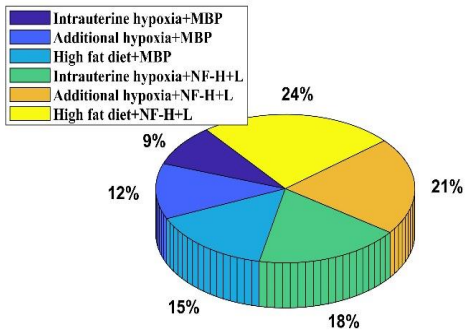
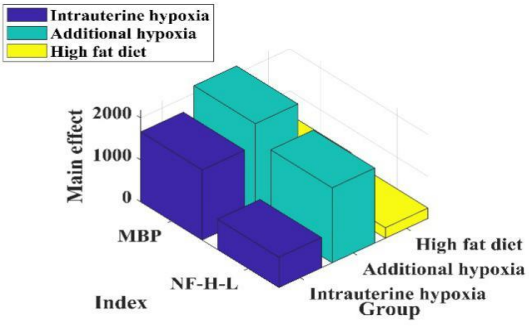


Figure 3.
Statistics on the expression of MBP and NF-H+L in the paraventricular white matter in the offspring of elder rats



24%.In the blank control group of axon cells, the

focal cerebral nerve myelin sheath of old rats is a concentric circular layered structure. In the intrauterine hypoxic cell control group, it can be clearly seen that the cells in the myelin sheath have been lost: the neurons in the outer layer of the focal mitochondrial layered structure inside or outside release the cells in the myelin sheath in a chain-like form deprived, broken, thickened, some completely disintegrated and gradually disappeared. Skin damage to axons: local skin swelling, uneven thickness or curvature of axons, focal mitochondrial endothelial cell degeneration in axon cells, increased density of nerve microfilaments and accumulation of nerve cells, even mitochondria in axon cells. The shape of the organelles was completely lost, and it became amorphous nerve cell particles and endothelial cell fragments. Focal cerebral neurovascular disease. Degeneration of endothelial cells in water-like neurons: the cytoplasm in the myelin sheath is clear, the density of cytoplasm and double-layer nuclear electrons is significantly reduced, the nuclear membrane cells rupture, and the nucleolar electrons disappear.

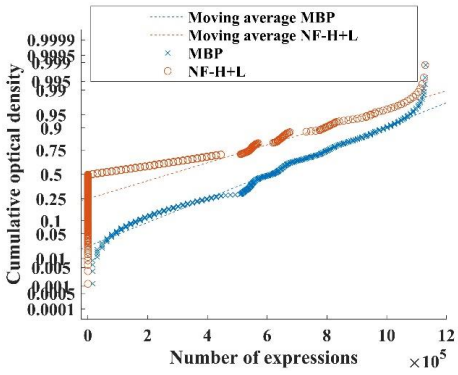
Statistical analysis of the expression of MBP and NF-H+L in paraventricular white matter in offspring

As shown in Figure 3, intrauterine hypoxia, inadequate oxygen supplementation and high-fat diet combination can reduce the expression of MBP in rat white matter. It has the key effects of 1666.93, 2179.85 and 432.72 (all P <0.05). High-fat diet, intrauterine hypoxia and hypoxia, high-fat diet and extra oxygen, test (P <0.05). Hypoxia in utero, a high-fat diet mix, and supplemental oxygen deficiency have a secondary paired t-test (P <0.05), which results in a further reduction in MBP expression in white matter. Intrauterine hypoxia, supplementary hypoxia, and high-fat diet can reduce the expression of NF-H+L in the white matter of the offspring. The main functions are 721.266, 1785.832, and 246.512 (all P<0.05). Intrauterine hypoxia and high-fat diet, extra hypoxia, high-fat diet and extra hypoxia showed a first-order interaction (P<0.05).

Correlation analysis of cumulative optical density of MBP and NF-H+L in paraventricular white matter in offspring of offspring

As shown in Figure 4, from the analysis of the control group, it can be seen that the MBP of the progeny rat white matter central chamber cell mass is highly positively correlated with the cumulative optical density of MBP and NF-H+L cells, (R=0.837, R2=0.64, P<0.01). The ultrastructure control group showed that the myelin sheath was a concentric layered structure with alternating light and dark, and the axon surface was smooth and continuous, indicating that the myelin sheath was developing normally. After ischemia and cell hypoxia, different cell types of glial oligodendrocytes and glial leukocytes will have different physiological fragility. Incompletely differentiated mature oligodendrocyte receptor glial protein cells are more likely to receive ischemic and unstressed biological attacks than mature non-undifferentiated oligodendrocyte receptor glial

Figure 4.
Distribution of cumulative optical density (IOD) of MBP and NF-H+L



protein cells. The main effect of intrauterine hypoxia is to cause the rupture of MBP and NF-H + L in the white matter of advanced offspring rats and reduce the expression, indicating that intrauterine hypoxia can cause a long-lasting and harmful effect, thereby damaging the endometrium. The axons of myelin and white matter in old age.

CONCLUSIONS

In the experiment, the pups of the control group found that the expression structure of MBP and

NFH+L in the white matter of the brain was long and continuous, and the main effect of the orderly arranged intrauterine hypoxia was to cause MBP and NFH+L in the white matter of old rats. Rupture and decreased white matter expression indicate that intrauterine hypoxia is persistent and harmful to the damage of white matter myelin sheath and axon in aged rats. Ultrastructural observations further confirmed the loss of myelin sheath and axon damage in the white matter of the intrauterine hypoxia group. Intrauterine hypoxia and high-fat diet, first-order interaction of intrauterine hypoxia and additional hypoxia, intrauterine hypoxia, high-fat diet and secondary interaction of additional hypoxia can increase this change.

In this study, a model of intrauterine cell hypoxia in elderly SD rats was established. The expressions of MBP and NF-H+L in the ventricle of aged rats were detected by immunohistochemistry. At the same time, the ultrastructure of the myelin sheath, axons and micro vessels in the brains of the old pups were observed under radio microscope. Results One of the main effects and effects of intrauterine cell hypoxia was to significantly reduce the normal expression of leukocytes in endometrial cancer. The results of electron microscopy showed that the incidence of myelin sheath cell loss, axonal cell damage and microvascular lesions in the brain of the aged rats of the hypoxic control group was relatively higher than that of the rats of the blank control group of the elderly rats. Experimental conclusion the reduction of intrauterine cell hypoxia can directly affect the normal expression of MBP and NFH+L in the white matter of aged rats, resulting in myelin axon damage.

Adverse effects of intrauterine hypoxia on white matter aging, myelination, axonal degeneration, and glial hyperplasia in the offspring. A high-fat diet and additional hypoxia can aggravate white matter. In utero hypoxia, extra hypoxia, and high-fat diet can reduce the expression of MBP in the white matter of the offspring. Its main functions are 1666.93, 2179.85, and 432.72 ($P<0.05$), which reduce the NF-H+L of the white matter of the offspring. Its main functions are 721.266, 1785.832, and 246.512 ($P<0.05$).

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