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The molecular mechanism of the transcriptional activator SWI regulating gene ARID1B affecting swallowing dysfunction after stroke in rats

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Background: After a stroke, more than 50% of patients are suffering from dysphagia. Because the swallowing dysfunction is closely related to some neural pathways, the probing of the neuro-molecular mechanism of dysphagia is very important for future diagnosis and treatment. *Methods:* Our study is a typical causal study with the purpose of exploring molecular mechanisms. In this study, a rat model of dysphagia after stroke was constructed, and ARID1B overexpression plasmid was injected into the rat body through tail vein injection. The number of swallows and the swallowing response time induced by distilled water in each group of rats on the 7th and 14th day after modeling were detected. After 14 days of successful model establishment, the rat brain tissues were collected, part of the brainstem nucleus tractus solitarius and nucleus suspicious tissues were analyzed with a Ca^{2+} fluorescent indicator to analyze the intracellular concentration of Ca^{2+}. For a part of the brainstem nucleus tractus solitarius and suspected nucleus tissues, immunohistochemistry was used to analyze the expression characteristics of genes ARID1B and TACR1 related proteins. The cerebrospinal fluid of brain tissue was collected, and the expression of gene TAC1 related protein in cerebrospinal fluid was analyzed by ELISA. For a part of the brainstem nucleus tractus solitarius and suspicious nucleus tissues, western blot was used to analyze the expression of gene SMARCA1 related protein, protein UNC80 and NALCN. *Results:* The detection of swallowing characteristics and the detection of intracellular Ca^{2+} concentration indicate the serious impact of stroke on swallowing function. The protein expression showed a consistent trend, which also showed that the overexpression of gene ARID1B can improve swallowing function to a certain extent. *Conclusion:* Due to our experiments, the molecular mechanism related to dysphagia was explored to a certain extent. At the same time, we found that the overexpression of the gene ARID1B can improve the swallowing function.

1. Introduction

Stroke is an acute cerebrovascular disease. Brain tissue damage or dysfunction caused by the sudden rupture or obstruction of blood vessels in the brain causes blood to flow into the brain (Kim et al. 2017). The data show that not only the incidence of stroke is high, but also the disability rate is high, which seriously affects the health and quality of life of patients. Stroke is one of the most common causes of dysphagia. After a stroke, more than 50% of patients show this symptom (Bath et al. 2018).

The pharyngeal phase is the process of the food bolus passing through the pharyngeal cavity. It mainly includes two processes, namely the contraction and extrusion of the pharyngeal muscles to deliver the food bolus and the throat to close the airway to protect the airway (Unluer et al. 20199. When the food bolus is delivered to the root of the tongue, the tactile receptors at the root of the tongue and bilateral palatal arches are activated, and nerve impulses are transferred to the basic swallowing center of the medulla oblongata. The efferent nerves are the glossopharyngeal nerve and the vagus nerve, and the effectors are the pharynx, palate, larynx, and vocal cords. When the brainstem swallowing center reaches the subliminal level of excitement, it can trigger the swallowing reflex (Warnecke et al. 2017).

ARID1B is a gene located on chromosome 6. This site encodes an AT-rich DNA interaction domain protein. The encoded protein is a component of the SWI/SNF chromatin remodeling complex and may play a role in cell cycle activation (Celen et al. 2017). In the brain, insufficient ARID1B haploid function leads to SWI/SNF regulation of gene expression and neuropsychiatric diseases. The mammalian SWI/SNF (BAF) chromatin remodeling complex can use the energy released by ATP hydrolysis to drive the movement of nucleosomes and regulate the structure of chromatin (Mashtalir et al. 2018). Recent whole-exome sequencing studies have shown that genes encoding subunits of the mSWI/SNF complex are mutated in more than 20% of cancers, covering a wide range of tissue types. Most mutations result in the loss of subunit protein expression, suggesting that mSWI/SNF subunit is a tumor suppressor (St. Pierre and Kadoch 2017).

The SMARCA1 gene encodes a member of the SWI/SNF protein family. The encoded protein is an ATPase expressed in a variety of tissues, which contributes to the chromatin remodeling complex involved in transcription. The protein may also play a role in the DNA damage, growth inhibition and apoptosis of cancer cells. Alternative splicing leads to multiple transcript variants (Homann et al. 2016).

The TAC1 gene encodes four products of the tachykinin peptide hormone family, substance P and neurokinin A, and related peptides, neuropeptide K and neuropeptide γ. These hormones are thought to be neurotransmitters that interact with nerve receptors and smooth muscle cells. They are known to induce behavioral responses and act as vasodilators and secretagogues (Gao et al. 2019).

The TACR1 gene belongs to the gene family of tachykinin receptors. These tachykinin receptors are characterized by their interaction with G protein and are containing seven hydrophobic transmembrane regions. This gene encodes the receptor for the

tachykinin substance P. The encoded protein is also involved in phosphatidylinositol metabolism of substance P. The encoded NK1R protein structure incorporates the characteristics of G protein-coupled receptors, and NK1R subtypes are expressed in the central nervous system and surrounding tissues (Hayase et al. 2015).

NALCN is a conservative cation channel that can conduct permanent sodium leakage current and regulate resting membrane potential and neuronal excitability. It is part of the NALCN channel body, a large ion channel complex, which is composed of a variety of proteins including UNC80 and UNC79. The main neuronal expression patterns and their functions indicate an important role in neuronal function and disease. Extracellular Ca^{2+} affects neuronal excitability in a G protein-dependent manner through the UNC79-UNC80-NALCN complex (Lu et al. 2010).

In this experiment, a rat model of dysphagia after stroke was constructed, and ARID1B overexpression plasmid was injected into the rat body through tail vein injection. The number of swallows and the swallowing response time induced by distilled water in each group of rats on the 7th and 14th day after modeling were detected. Two weeks after model establishment, the rat brain tissues were collected, part of the brainstem nucleus tractus solitarius and nucleus suspicious tissues were analyzed with a Ca^{2+} fluorescent indicator to analyze the changes in the concentration of Ca^{2+} inside and outside the cells within 2 min. For a part of the brainstem nucleus tractus solitarius and suspected nucleus tissues, immunohistochemistry was used to analyze the expression characteristics of genes ARID1B and TACR1 related proteins. The cerebrospinal fluid of brain tissue was collected, and the expression of gene TAC1 related protein in cerebrospinal fluid was analyzed by ELISA. For a part of the brainstem nucleus tractus solitarius and suspicious nucleus tissues, western blot was used to analyze the expression of proteins SMARCA1, UNC80, and NALCN. Through these tests, explore the molecular mechanism of ARID1B affecting dysphagia after stroke in rats.

2. Investigations and results

2.1. Swallowing feature detection

The results of swallowing feature detection are shown in Fig. 1. The NC group and the sham-operated group averaged 10 swallows in 5 min, and the average swallowing response time was 2-3 s. The ARID1B overexpression group averaged 6 swallows in 5 min, and the average swallowing response time was 6-8 s. The average number of swallows in 5 min in the model group was 3 times, and the average swallowing reaction time was 25-28s.

2.2. Fluo-3 AM Ca^{2+} fluorescent probe test

The results of Fluo-3 AM Ca^{2+} fluorescent probe test are shown in Fig. 2. Through the combination of the green fluorescence of Ca^{2+} and the red fluorescence of the cell membrane, the yellow fluorescence in the overlay image can be obtained as intracellular Ca^{2+}, while the green fluorescence is extracellular Ca^{2+}. The intracellular Ca^{2+} concentration is positively correlated with swallowing intensity, which is consistent with the results of swallowing feature detection.

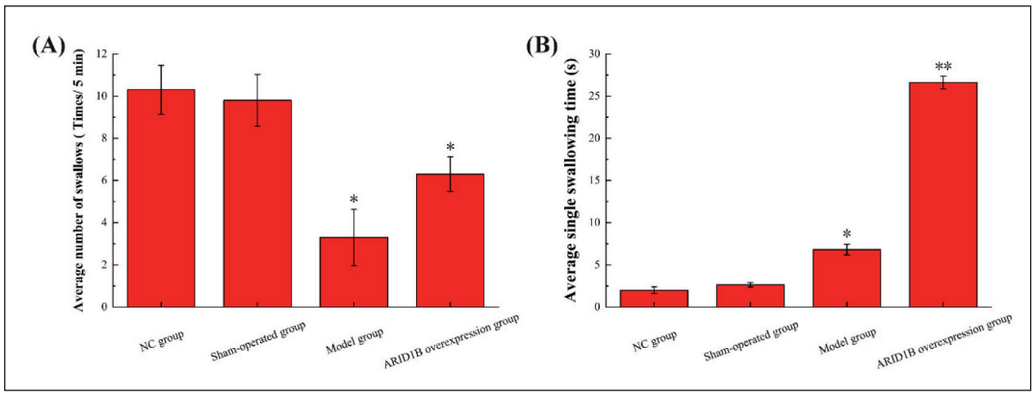

Fig. 1: The results of swallowing feature detection. The data of NC group is consistent with the normal value. (A) Average number of swallows in 5 minutes. (B) Average time of single swallow. The symbol * means P<0.05, ** means P<0.01 (compared to the NC group).

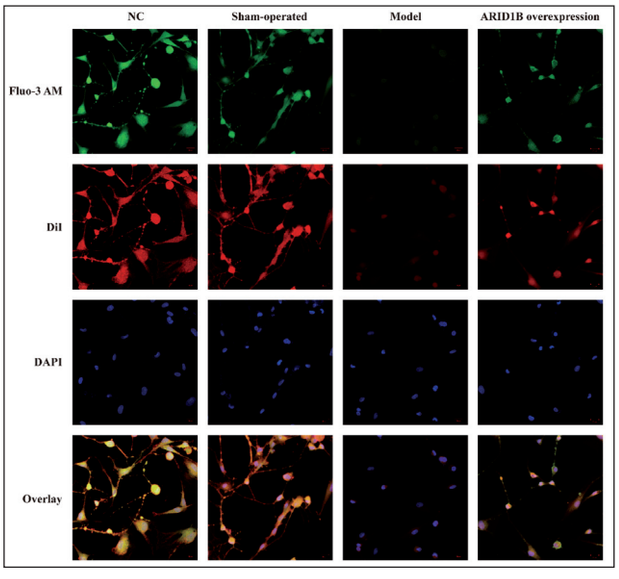

Fig. 2: The results of Fluo-3 AM Ca^{2+} fluorescent probe test. The data of NC group is consistent with the normal value. The yellow fluorescence in the overlay image is intracellular Ca^{2+}.

2.3. Immunohistochemistry analysis

The results of the immunohistochemical analysis are shown in Fig. 3. The yellowish-brown spots in the first horizontal row represent the protein expressed by the gene ARID1B, and the yellow-brown parts on the cell membrane in the second horizontal row represent the protein expressed by the gene TACR1. The protein content expressed by genes ARID1B and TACR1 shared the same trend. The expression level in the NC group was slightly higher than that in the sham-operated group, and the expression level in the model group was the least, while the expression level in the ARID1B overexpression group increased compared with the model group.

2.4. ELISA analysis

The results of the ELISA analysis are shown in Fig. 4. The contend of protein expressed by gene TAC1 is consistent with the results in the immunohistochemical analysis. According to the results of the two analysis experiments, the neural pathways related to the genes ARID1B, TACR1 and TAC1 are closely related to the swallowing function. At the same time, it can be known that the overexpression of ARID1B can improve the swallowing ability of stroke patients to a certain extent.

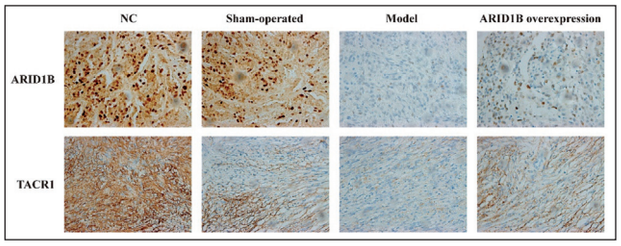

Fig. 3: The results of immunohistochemical analysis. The data of NC group are all consist with the normal value. The yellowish-brown spots in the first horizontal row represent the protein expressed by the gene ARID1B, and the yellow-brown parts on the cell membrane in the second horizontal row represent the protein expressed by the gene TACR1.

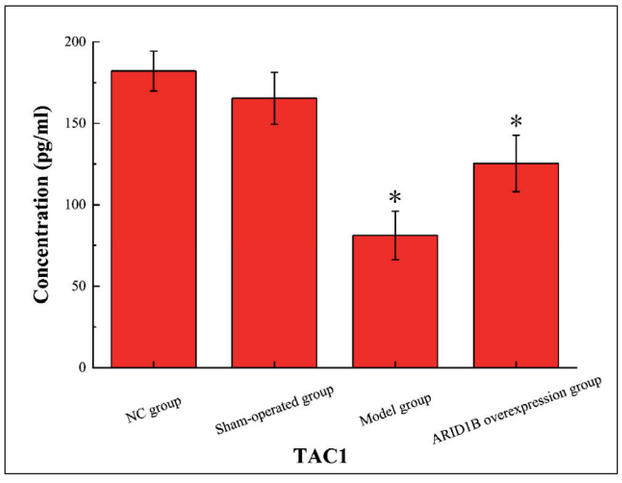

Fig. 4: The results of ELISA analysis. The data of NC group is consistent with the normal value. The symbol * means P<0.05 (compared to the NC group).

2.5. *Western blot analysis*

The results of Western blot analysis are shown in Fig. 5. The content trends of the protein expressed by gene SMARCA1, protein UNC80 and NALCN are consistent with the results of immunohistochemical analysis and ELISA analysis. This indicates that the neural pathways related to SMARCA1-UNC80-NALCN are also related to the swallowing function, and once again confirms the foregoing conclusion.

obstruction, suffocation, dehydration, and malnutrition. Aspiration after stroke is associated with a high risk of developing pneumonia (Nakamori et al. 2020).

Ball palsy and pseudo-ball palsy of stroke can cause swallowing disorders. Ball palsy refers to the injury of the medullary motor nucleus or cranial nerve, and the injury of the lower motor neuron. The symptoms of dysphagia are more severe than dysarthria, the pharyngeal reflex is absent or very weak, the tongue muscles are atrophy or there is fasciculation. Ball palsy has poor compensatory ability and poor rehabilitation effect. Pseudobulbar palsy refers to bilateral cortical medulla oblongata injury, upper motor neuron injury, and the lower motor neuron innervating the swallowing muscle is not damaged. Dysargia is more serious than dysphagia, the pharyngeal reflex is present (but delayed, uncoordinated), the compensatory ability is strong, and the rehabilitation effect is better (Benjapornlert et al. 2020; Cabib et al. 2020).

In molecular biology, SWI/SNF (SWItch/Sucrose Non-Fermentable), is a subfamiliy of ATP-dependent chromatin remodeling complexes, which is found in eukaryotes. In other words, it is a group of proteins that associate to remodel the way DNA is packaged. This complex is composed of several proteins – products of the SWI and SNF genes (SWI1, SWI2/SNF2, SWI3, SWI5, SWI6), as well as other polypeptides. It possesses a DNA-stimulated ATPase activity that can destabilize histone-DNA interactions in reconstituted nucleosomes in an ATP-dependent manner, though the exact nature of this structural change is unknown. The SWI/SNF subfamily provides crucial nucleosome rearrangement, which is seen as ejection and/or sliding. The movement of nucleosomes provides easier access to the chromatin, allowing genes to be activated or repressed (Wang et al. 2017).

The transcriptional activator SWI can increase the expression of gene ARID1B, and the protein expressed by gene ARID1B increases the expression of genes TAC1, TACR1 and SMARCA1 through chromosome remodeling (Shibutani et al. 2017; Niedermaier et al. 2019; Kruizinga et al. 2020). The protein expressed by gene TAC1 affects the excitability of neurons and the biological behavior of the organism, and the protein expressed by gene TACR1 is the receptor for the protein expressed by gene TAC1 (Hu et al. 2017; Jakimiuk et al. 2017). In addition, the protein expressed by the gene SMARCA1 forms an ion channel with the proteins UNC80 and NALCN (Parsa et al. 2019). The protein UNC80 affects the ion transport of neurons in the brain and regulates the function of the protein NALCN (Shamseldin et al. 2016; Lu et al. 2007; Bramswig et al. 2018). The three neural pathways together affect the swallowing function of stroke patients, forming a molecular mechanism that affects swallowing disorders after stroke in rats (Lalli et al. 2020).

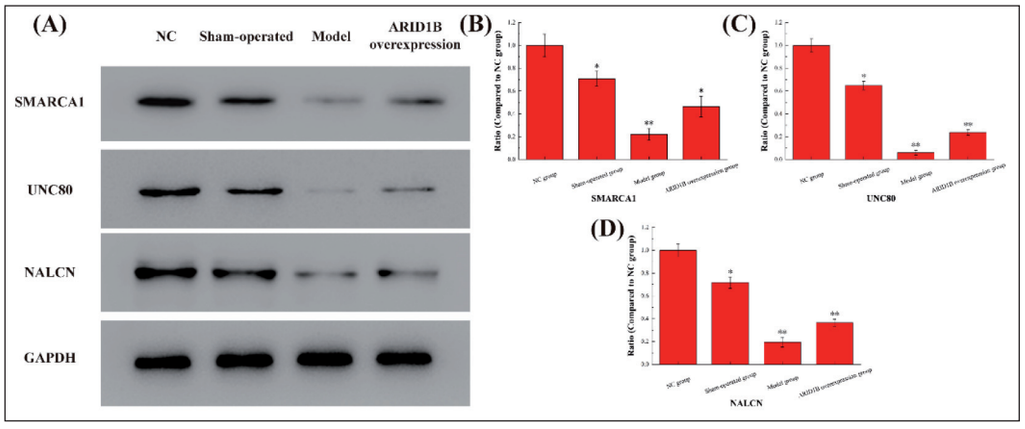

Fig. 5: The results of Western blot analysis. The data of NC group are all consistent with the normal value. (A) Original gel electrophoresis image. (B-D) The ratio of protein expressed by gene SMARCA1, protein UNC80 and NALCN expression compared to NC group. The symbol * means P<0.05, ** means P<0.01 (compared to the NC group).

3. Discussion

Dysphagia is a common symptom of stroke patients, with an incidence rate of 50% to 65%, which often has a serious impact on the physical and mental health of patients. Physiologically, decreased swallowing function can cause aspiration, bronchospasm, airway

In this experiment, we established a rat model of dysphagia after stroke, and then injected the ARID1B overexpression plasmid into rats through tail vein injection to explore the molecular mechanism of dysphagia after stroke. Through swallowing feature detection, Ca^{2+} concentration detection, and detection of protein expression in

the above three neural pathways, the molecular mechanism related to dysphagia was finally explored to a certain extent. At the same time, we found that the overexpression of the gene ARID1B can improve the swallowing function. The clarification of the molecular mechanism requires further in-depth experimental research.

4. Experimental

4.1. Study design

Our study is a typical causal study on the purpose of exploring molecular mechanisms. In the experiment, we used 48 male SD rats aged 8-10 weeks and randomly separated them into normal group, sham-operated group, model group and ARID1B overexpression group. The latter two groups were used to temporarily block the middle cerebral artery for 90 min to establish dysphagia model. The ARID1B overexpression group received a tail vein injection of plasmid for 14 days. During model building, the tension converter was connected to the anterior abdomen of the digastric muscle of the rat's neck. The number of swallows and the swallowing reaction time triggered by distilled water in each group of rats on the 7th and 14th days after the modelling were detected. Two weeks after successful model establishment, rats were anesthetized with ether and their necks were dissected to death. The brain tissues of the rats were collected. Part of the brainstem nucleus tractus solitarius and nucleus suspicious tissues were analyzed with Fluo-3 AM Ca^{2+} fluorescent probe to analyze the intracellular concentration of Ca^{2+}. For a part of the brainstem nucleus tractus solitarius and suspected nucleus tissue, immunohistochemical analysis were applied to detect the expression characteristics of the proteins expressed by gene ARID1B and TACR1. The cerebrospinal fluid of brain tissue was collected, and the expression of protein expressed by gene TAC1 in the cerebrospinal fluid was analyzed by ELISA. For a part of the brainstem nucleus tractus solitarius and suspicious nucleus tissue, Western blot analysis was used to analyze the expression of protein expressed by gene SMARCA1 and protein UNC80 and NALCN.

4.2. Swallowing feature detection

During the establishment of the model, the tension converter was connected to the anterior abdomen of the digastric muscle of the rat neck. Based on the feedback data, the number of swallows within 5 min and the swallowing response time triggered by distilled water in each rat on the 7th and 14th day were detected.

4.3. Fluo-3 AM Ca^{2+} fluorescent probe test

Add 16.5mg Pluronic F127 to Fluo-3 AM/DMSO solution, which can prevent Fluo-3 AM from polymerizing in HBSS and help it enter cells. Dilute the Fluo-3 AM solution with HBSS to prepare working solution with a concentration of 4 μM. Add Fluo-3 AM working solution to the cells and incubate at 37 °C for 20 min. Add 5 times the volume of HBSS solution containing 1% FBS (fetal bovine serum), and continue to incubate for 40 min. Wash the cells 3 times with HEPES buffer saline, and then resuspend the cells with HEPES buffer saline to make a solution of 1×10^5 cells/ml. Incubate at 37 °C for 10 min, and then detect with a fluorescent confocal microscope.

4.4. Immunohistochemistry analysis

Cells of each group were seeded on a six-well plate and fixed in 4% paraformaldehyde at room temperature for 24 h. All these samples were washed three times with PBS solution. The cells were then sealed with 5% BSA for 30 minutes. The primary antibody FBP17 and PPRC1 (1: 500, anti-human, Abcam, USA) were diluted to the used concentration. After incubating on a 4 °C shaker overnight and washed in PBS three times, the samples were incubated with secondary antibody (1: 1000, Abcam, USA) for 30 min in the dark at room temperature. Finally, the samples were observed under an inverted microscope with pictures taken at the same time. Image J software was used for positive area analysis.

4.5. ELISA analysis

Five standard wells were set on the ELISA-coated plate. The standard samples were added to the wells according to the concentration requirements and serially diluted. The sample volume in each well was 50 μl. Then a blank control well and the sample well to be tested were set. The blank control well would not add sample and enzyme-labeled reagent; the other steps were the same with the sample well. Add 40 μl of sample diluent to the sample well, and then add 10 μl of the sample. The final dilution of the sample is 5 times. Seal the plate with a sealing film and incubate at 37 °C for 30 min. After incubation, wash the plate with washing solution and dry it. After drying, add 50 μl of enzyme-labeled reagent to each well except the blank well. Repeat the incubation and washing steps. Then add 50 μl of developer A and 50 μl of developer B in each well sequentially, and develop color at 37 °C for 15 min in the dark, and lastly add 50 μl of stop solution to stop the reaction (the blue turns to yellow immediately). Set the blank control well as zero, and measure the absorbance (OD value) of each well in sequence at 450 nm wavelength.

4.6. Western blot analysis

Collect cells from each group, and add 200 μl of cell lysate to each six-well plate. After sonication, the cells were lysed on ice for 1 h. The lysed cell sample was centrifuged at 12,500 rpm for 15 min at 4 °C. Then, transfer the supernatant in the centrifuge tube to a clean centrifuge tube. GAPDH protein quantification kit was used to quantify protein concentration. The measured protein samples were stored at -80 °C. In Western blot electrophoresis, the protein loading concentration was 50 μg per well. After SDS-PAGE electrophoresis, the membrane was transferred and blocked. Protein expressed by gene SMARCA1 and protein UNC80 and NALCN primary antibody (1: 500, anti-human, Abcam, USA) were diluted to use concentration. The samples were incubated overnight on a shaker at 4 °C. After washing with PBS, the samples were incubated with the secondary antibody (1: 1000, anti-human, Abcam, USA) for 30 min at room temperature in the dark. Finally, the developer was used for development and photography.

4.6. Statistical analysis

The experimental results are expressed as mean ± standard deviation. Statistical analysis was performed using SPSS 22.0 software. The figures were produced with Origin 2020 and Adobe Illustrator 2020 software.

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Conflicts of interest: None declared.

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