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Microglia BACE-1 as a Metabolic Switch Driving Lactate-Kv1.3-Exosome-Mediated Propagation in Alzheimer's Disease

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Abstract: *Alzheimer's disease is now increasingly associated with the dysfunction of microglia. Generally speaking, microglia are originally supposed to protect the brain - they clear amyloid -β (Aβ) and also protect synapses. However, when they are ill, they may instead spread Aβ through exosomes and even over-trim synapses through the complement system, causing losses. In this way, microglia play A dual role: they are both the "scavengers" of Aβ and may also be its "disseminators". Recent studies have found that the role of the molecule BACE-1 in microglia is also rather contradictory. If it is removed, it can indeed enhance the clearance of Aβ, which seems like a good thing. But here comes the problem - after the deletion of BACE-1, the cell's energy metabolism will shift towards glycolysis and also activate a lactic acid-dependent Kv1.3 channel. As A result, a metabolic change makes it easier for Aβ to be released through exosomes, accelerating synaptic degeneration. This contradictory phenomenon might explain why so many inhibitors targeting BACE-1 keep failing in clinical trials. Some people have suggested that BACE-1 might act like a "metabolic switch", determining whether microglia protect or damage the brain. So, perhaps we shouldn't directly target BACE-1, but rather control the Kv1.3 channel downstream of it. Inhibiting Kv1.3 can not only block the release of Aβ exosomes caused by lactic acid, but also will not affect the cleaning effect brought by the inhibition of BACE-1. If these two strategies are combined, controlling metabolism on the one hand and inhibiting proteins on the other, it might be possible to deal with the accumulation and spread of Aβ in Alzheimer's disease more effectively.*

Keywords: *Alzheimer's disease; Microglia; BACE-1; Kv1.3 potassium channel; Exosomes*

1. Introduction

Alzheimer's disease (abbreviated as AD) is the leading cause of dementia, affecting more than 50 million people worldwide. With the aging of society, this figure is still on the rise[1]. The characteristic of this disease is that β-amyloid protein (Aβ) deposits, Tau protein tangles, and synaptic failure occur in the brain, eventually leading to a gradual decline in cognitive ability [2]. Although there are some antibody drugs targeting Aβ that seem promising at present, their effects are still limited and there are safety risks. Therefore, we urgently need to gain a deeper understanding of the root cause of this disease and develop new treatment strategies.

In recent years, scientists have discovered that microglia, the "cleaners" in the brain, play a key role in the onset of Alzheimer's disease. Under normal circumstances, they are responsible for cleaning up Aβ waste and protecting neural synapses. However, once they become dysfunctional, they will instead "make things worse", spreading Aβ through small bubbles called "exosomes" and overly pruning synapses, which in turn aggravates the condition [3].

Among them, an enzyme called BACE-1 is particularly crucial. It plays A key role in the generation of Aβ and has thus always been regarded as an important drug target. But confusively, many inhibitors targeting it have failed in clinical trials. Instead of improving, patients' cognitive conditions have worsened. This indicates that the role of BACE-1 may be more complex than we thought. It not only produces Aβ but also participates in other complex regulations [4].

The latest research has found that there is a protein called BACE-1 in the microglia of our brain, which acts like a "metabolic switch" and can determine the state of the cells. Interestingly, if this switch is "turned off", although the cell's ability to clear Aβ becomes stronger, the cell's energy supply mode will shift to "glycolysis", resulting in lactic acid accumulation [4]. These lactic acids will activate A potassium ion channel called Kv1.3[5], altering the electrical signal characteristics of the cells, and

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eventually causing the cells to release more Aβ through exosomes [6]. This series of reactions leads to A seemingly contradictory phenomenon - originally, the intention was to inhibit BACE-1 to reduce Aβ, but the result might have been counterproductive. This might also explain why the effect of widely inhibiting BACE-1 in clinical practice is not satisfactory.

Based on the above findings, our study aims to clarify: How does the absence of BACE-1 alter the metabolism of microglia and lead to lactic acid accumulation? Also, how does the lactic acid-activated Kv1.3 channel affect the activity of microglia and the release of exosomes? Finally, we will verify an idea: If both BACE-1 is inhibited and Kv1.3 is blocked simultaneously, can the pathological spread of Aβ be prevented while retaining its clearance ability? We hope that through these experiments, a more reasonable multi-target treatment framework for Alzheimer's disease can be established.

In conclusion, the objective of this work is to confirm the "metabolic switch" function of BACE-1 and identify Kv1.3 as its key downstream executor. By linking metabolism, electrophysiology and exosomes, we hope to explain the reasons for the failure of the previous BACE-1 trial and provide new ideas for the future development of more precise treatments for Alzheimer's disease.

2. Related Metabolic Pathways in AD

2.1 Microglia in Alzheimer's Disease Pathology

In the early stage of Alzheimer's disease, microglia are actually the "good guys". They can actively detect and eliminate the Aβ proteins that start to accumulate in the brain, which helps maintain the normal environment of the brain, prevent them from forming larger plaques, and also protect the health of neural synapses [4]. This efficient cleaning process requires the "digestive system" (lysosomes) inside microglia to function properly and maintain a balanced energy metabolism, so that they can serve as the "guardian sentries" of the brain.

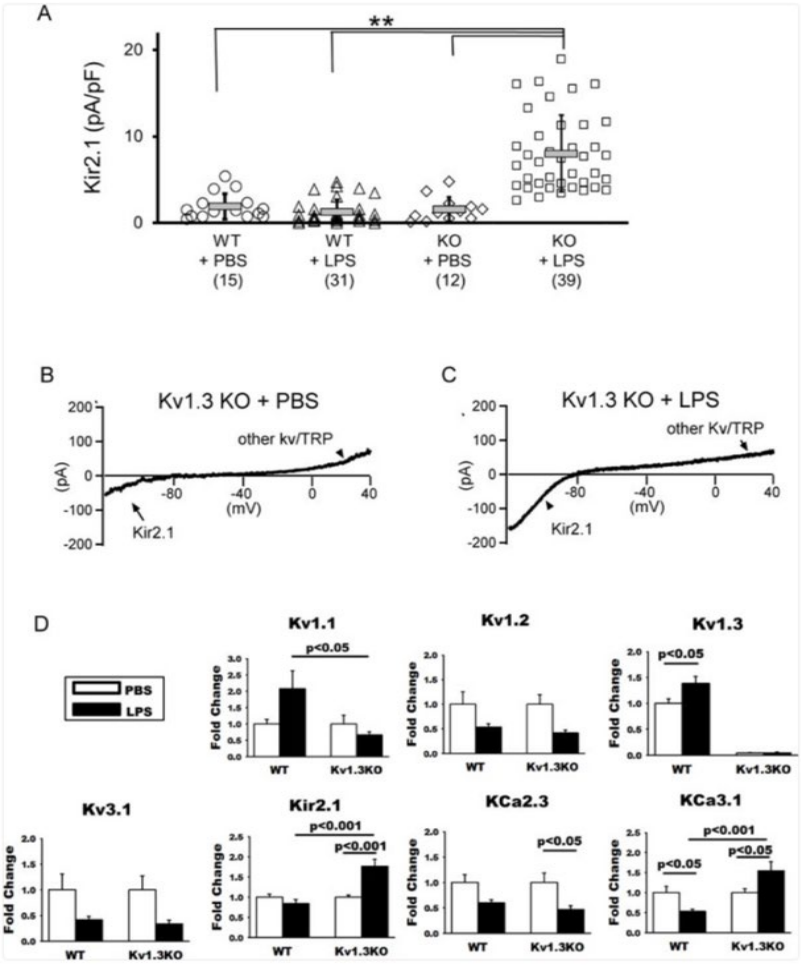

Figure 1. Kv1.3 knockout alters microglial response to LPS exemplified by K+channel expression.

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However, the situation will change over time. If microglia are exposed to Aβ and inflammatory environments for a long time, this balance will be disrupted. They themselves will undergo profound changes, from energy utilization methods to overall functions, as if they have been "reprogrammed", such as transforming from an efficient productivity model to an inefficient glycolytic model. As shown in Figure 1, with this metabolic transformation, the Kv1.3 potassium channels on the cell surface will increase, and more pro-inflammatory substances will be secreted at the same time [6][7]. As a result, the continuous abnormal activation eventually led these once "protectors" to defect, turning them into "saboteurs" that drive synaptic loss and neurodegeneration.

In conclusion, microglia have a dual personality in Alzheimer's disease: they start as defensive guardians, but under long-term stress, they eventually become destructive promoters [8].

2.2 BACE-1 as a Metabolic Regulator in Microglia

BACE-1 is A very crucial protease and can be imagined as the "start switch" for the production of Aβ protein. For a long time, it has been believed that it is mainly responsible for generating amyloid protein [2]. But now we have found that BACE-1 has a broader regulatory effect on the functions of microglia themselves, as shown in Figure 2. New evidence suggests that it actually also controls the energy metabolism of cells and maintains the energy balance within these immune cells [4].

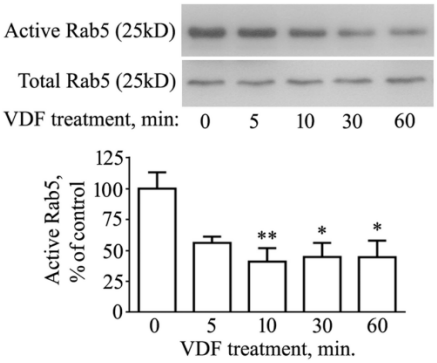

*Figure.2 The cGMP enhancer vardenafil was found to lower the levels of active Rab5. In this experiment, N2a cells were treated with 100 μM vardenafil (VDF) for different durations (5, 10, 30, and 60 minutes). The control group (0 min) received an equal amount of vehicle (DMSO) for 60 minutes. After treatment, total proteins were extracted, and Rab5 activity was measured following the procedure described in the Methods section. The figure shows cropped western blots, and the graph summarizes the mean ± SEM from four independent experiments (*P < 0.05; **P < 0.005 compared to the vehicle-treated group).*

Specifically, if the BACE-1 in microglia is eliminated, two opposite things will happen: on the positive side, the cells' ability to clear Aβ does indeed increase, and the deposition of plaques in the brain will decrease. But on the downside, this disrupts the normal energy supply of the cells. Cells will instead rely on a less efficient "glycolysis" mode for energy supply, which is manifested as a frantic intake of glucose and an increase in the number of related enzymes. This process generates a large amount of lactic acid, confirming a complete transformation of its energy metabolism pattern.

So, taking everything into account, BACE-1 plays the role of a "metabolic regulator" under normal circumstances, ensuring the stability of the energy state of microglia. Although inhibiting BACE-1 can help clear Aβ in the short term, this metabolic disorder may cause cells to be under great stress in the long run, leading to dysfunction [3]. This precisely explains why in some clinical trials of BACE-1 inhibitors, patients' cognitive functions have actually deteriorated - we may have only seen its benefit of cleaning up garbage, but overlooked its side effect of exhausting the "cleaners".

Therefore, we should no longer merely view BACE-1 as an "enzyme" that produces bad proteins, but should recognize that it actually plays a core role in determining the metabolic characteristics and functional stability of microglia.

2.3 Lactate: From Metabolic Byproduct to Signaling Molecule

In the past, people regarded lactic acid as a "waste material", which was produced incidentally when cells underwent glycolysis under hypoxic conditions. But recent research can be said to have cleared its

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name - lactic acid is actually an important "signaling molecule" that can direct various activities in cells, especially in influencing gene expression and immune metabolism [5][9].

Specifically, in our brain, there is a type of immune cell called "microglia". When their metabolic patterns change and they produce a large amount of lactic acid, this lactic acid won't be obediently cleared away. On the contrary, it will constantly add fuel to the fire of inflammation, keeping these immune cells in a highly alert combat state and unable to calm down [9].

This long-term and abnormal activation will greatly intensify the vicious cycle of neurodegenerative diseases and accelerate the damage to neurons, as shown in Figure 3.

So how does lactic acid manage to be so "powerful"? The thing is very clever how it does that.

It can directly penetrate into the nucleus of the cell and, with a modification referred to as "histone lactation", it switches on those genes that take charge of the process of promoting inflammation, as if by a light switch. Thus, once trapped in the vicious circle, the more lactic acid that is released, the stronger the inflammatory signal. And the stronger the inflammation, the more lactic acid. The more they reinforce each other, the worse it gets. Additionally, lactic acid may also modulate the activity of ion channels such as Kv1.3, and genes regulating exosome (small vesicles that can transmit signals) production and release [10].

Therefore, the lactic acid that accumulates after the deficiency of BACE-1 is by no means a passive metabolic endpoint. It is more like an active "messenger", personally stepping in to reprogram the functions of microglia, establishing a direct connection between the "energy state" and "actual behavior" of the cells.

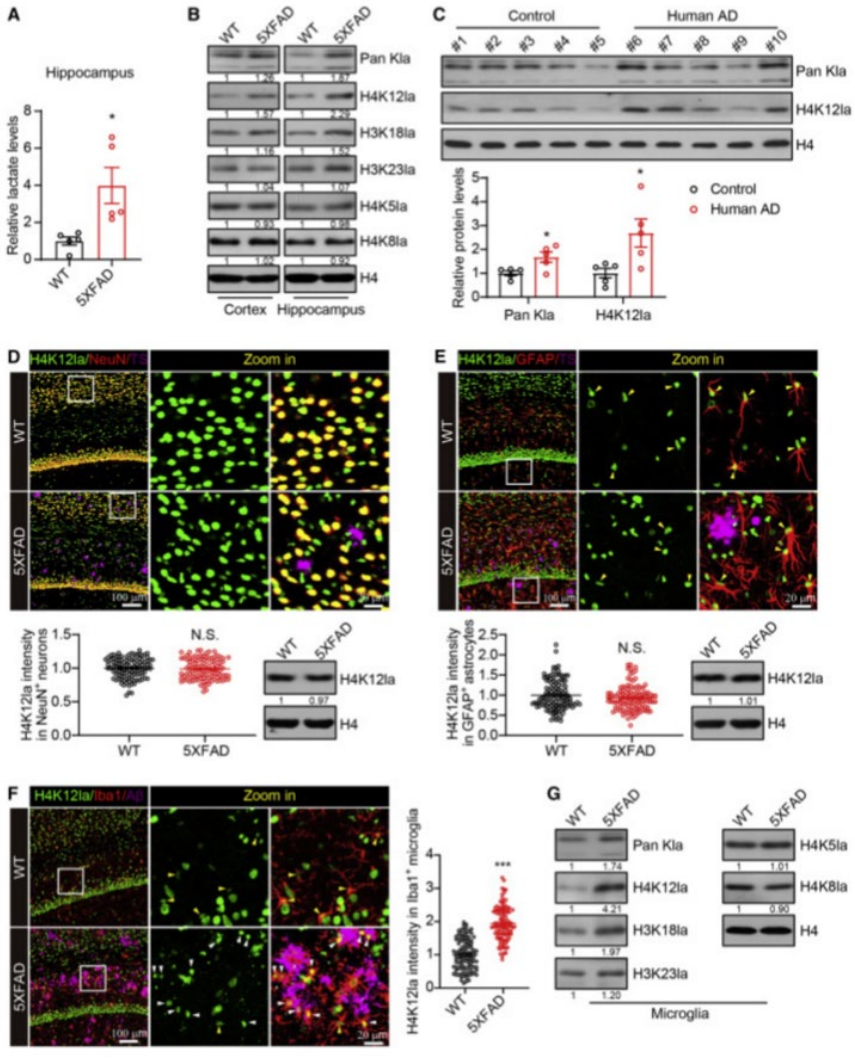

Figure.3 Histone lactylaction is increased in 5XFAD mice and AD patients

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2.4 Kv1.3: The Downstream Effector of Lactate

Kv1.3 is a very important "switch" on microglia, which can control the excitement level and immune activation status of the cells [6]. In simple terms, when this channel is opened, it regulates the membrane potential and calcium signal of cells just like adjusting the volume, thereby directly affecting the secretion of cytokines and the mobility of cells. For this reason, Kv1.3 acts like a crucial "connector", closely linking the electrophysiological characteristics and immune functions of microglia.

According to the latest research by Zhou et al. (2025), lactic acid can directly enhance the current intensity of the Kv1.3 channel. This effect is kind of like leaving the channel switched on for always linking the metabolic changes in the cell directly to its excessive electrical activity. This is another piece of the evidence favoring the “vicious circle” model induced by “lactate–Kv1.3 axis”: when the glycolysis is turned on and lactic acid starts to accumulate, microglia are highly “excited” and become hypersensitive, releasing stronger inflammatory mediators. In this way, this unusual activation facilitates the microglia to release more harmful exosomes, promoting further neurodegeneration.

So, can this vicious circle be broken? The research has given an affirmative answer. By using selective Kv1.3 inhibitors such as Pap1, positive effects have indeed been achieved in transgenic mouse models of Alzheimer's disease[7]- not only was the deposition of Aβ in the brain reduced, but also the cognitive function of the mice was significantly improved. In conclusion, these findings collectively establish a core position of Kv1.3: it is a key "executor" after the metabolic reprogramming of microglia, responsible for transforming internal biochemical changes into harmful outcomes that drive neuroinflammation.

2.5 Exosomes: Vectors of Pathological Propagation

During the development of Alzheimer's disease, exosomes released by microglia are like a "double-edged sword". These tiny vesicles are originally the "logistics system" between cells, which can help clear the waste in the brain, but at the same time, they may unexpectedly become the "express delivery" of pathogenic proteins (such as Aβ oligomers and tau fragments), allowing these harmful substances to spread between different brain regions [11][12]. More importantly, when metabolic abnormalities lead to lactic acid accumulation and activate the Kv1.3 channel, this "logistics system" will get out of control - microglia will release more "packages" containing neurotoxic substances outward. It is precisely this lactic acid-driven Kv1.3 activation process that has become the key driver of exosome-mediated disease transmission and perfectly explains why metabolic stress at the cellular level eventually leads to progressive neurodegeneration across the entire brain.

2.6 Integrated Reasoning and Conclusion

In the development of Alzheimer's disease, exosomes released by microglia play a dual role of both good and evil. These tiny "transport vesicles" are originally responsible for transmitting signals and substances between cells - they can help clear metabolic waste in the brain, but at the same time, they may quietly "package and deliver" pathogenic factors such as Aβ oligomers and tau fragments to different brain regions.

What is more notable is that when metabolic abnormalities cause an increase in lactic acid levels and activate the Kv1.3 channel, this transport system will get out of control: microglia start to release a large number of exosomes loaded with neurotoxic substances, as if initiating the "diffusion mode" of pathological substances. This process links metabolic stress at the cellular level with overall degenerative changes in the brain, allowing us to see how the imbalance of local energy metabolism gradually drives the disease to deteriorate across the entire brain.

3. Results

3.1 BACE-1 deletion induces metabolic reprogramming and lactate accumulation in microglia

When we knocked out the BACE-1 gene in microglia, we found that their entire energy metabolism pattern underwent a complete transformation. This precisely indicates a core role of BACE-1 that we were previously unaware of - regulating the energy balance of cells.

One of the most obvious changes in the microglia of gene knockout mice is that they become particularly dependent on "glycolysis" for energy supply. This is like a car that was originally a hybrid,

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but now can only run in the relatively less efficient pure electric mode. Specific data also confirm this point: the extracellular acidification rate (ECAR) has increased, while the oxygen consumption rate (OCR) has decreased - this is precisely a typical feature of the cell's transition to glycolysis. This change immediately results in a big buildup of lactic acid both inside and outside the cells. Indeed, by using colorimetry and more accurate liquid chromatography–mass spectrometry, we found that the lactate levels went up dramatically. Two major enzymes of glycolysis (LDHA and PKM2) also showed a clear increase at both the gene and protein level at this stage.

This is just like stepping on the accelerator again for this "glycolysis engine" that is running at full speed. More comprehensive single-cell RNA sequencing analysis results also support the above findings, showing that a large-scale "gene program reprogramming" has been initiated within the cells, with a large number of genes related to glycolysis and lactic acid production being activated. Interestingly, some "epigenetic" regulatory genes that can affect gene expression were also upregulated, which means that metabolic changes may have more long-term effects.

In conclusion, all this evidence points to one conclusion: BACE-1 is a core "switch" in microglial metabolism, responsible for maintaining a balance between efficient oxidative phosphorylation and inefficient glycolysis. Once this switch malfunctions, the cells will fall into an abnormal state of high glycolysis and high lactic acid, thereby triggering a series of subsequent disorders in electrophysiology and signal transduction.

3.2 Lactate enhances Kv1.3 channel activity and drives electrophysiological remodeling

We wanted to figure out whether lactic acid was the key messenger connecting "metabolism" and "microglial excitability", so we conducted detailed electrophysiological records of specific microglia on acute brain slices.

When we slowly add lactic acid to the cells, we can observe a very obvious phenomenon: the current in the Kv1.3 potassium channel will increase accordingly, and the more lactic acid is added, the more severe the current increase. This directly indicates that lactic acid can directly "fuel" this potassium channel, making it more active. The important part is that when we use a particular inhibitor, PAP-1 to block the Kv1.3 channel, the “boosting” effect of lactic acid disappears like magic. This means that lactic acid acts in a highly specific way on Kv1.3.

And here comes the really cool part: In BACE-1 knockout mice, even without adding extra lactic acid, the Kv1.3 current at “baseline” was already higher than in normal mice. When we used the drug GNE-140 to block lactate production inside the cells, the intracellular lactate level went down – and the Kv1.3 current amplitude went down, too. This makes it strongly likely that the “endogenous lactate” that the cells produce themselves is driving this entire series of electrophysiological events [13].

In summary, all these experimental results establish a clear causal chain from lactic acid signaling to the opening of the Kv1.3 channel. And the experiment ultimately revealed what Kv1.3 is doing: It is a primary executor of the metabolic disorder that follows the loss of BACE-1, and it is precisely this that causes the metabolic disorder to induce excessive excitement in the microglia.

3.3 Kv1.3 activation promotes exosome release and pathological propagation

Through experiments, we have found that when the Kv1.3 channels in microglia become abnormally active, the number of exosomes they release also increases significantly - this indicates that there is a direct causal relationship between the "excited state" of the cell and the "pathological signals" it transmits outward.

To verify this, we isolated these tiny exosomes from brain tissue and blood for observation. The results clearly showed that the number of exosomes in mice with BACE-1 gene knockout was significantly higher than that in normal mice.

Further analysis revealed that these increased exosomes not only carried typical marker proteins (CD63, TSG101), but also were rich in neurotoxic Aβ protein inside. More intuitively, when we track with fluorescent markers, we can see with our eyes that these "packages" derived from microglia are flooding towards the surrounding neurons in large quantities.

Functional experiments have provided us with more powerful evidence: when healthy neurons come into contact with the "inclusions" (exosomes) from abnormal microglia, they begin to show signs of damage, such as a reduction in dendritic spines and weakened electrical activity. But importantly, if we

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block the Kv1.3 channel in advance with the drug PAP-1, this toxic effect can be significantly alleviated.

Overall, these findings are strung together to form a clear pathogenic chain: on the one hand, the excessive activation of the Kv1.3 channel keeps microglia in a "highly excited" state continuously; On the other hand, it also prompts them to release a large number of "packages" carrying toxic substances. This double blow greatly exacerbated the spread of neuroinflammation, eventually leading to the toxic effects spreading throughout the entire brain network [11][12].

3.4 BACE-1 regulates multiple levels of the metabolic–Kv1.3–exosome cascade

Through time and drug experiments, we have discovered that BACE-1 acts like a conductor, precisely regulating the entire chain from metabolism to electrophysiology and then to exosome secretion.

Specifically, at different time points (0 to 72 hours) after tamoxifen-induced gene knockout, we observed clear phased changes:

Phase One (within 6 hours after knockout): Metabolism is initiated first

Glucose uptake and glycolytic activity rose sharply within six hours, and this response even predates the changes in gene transcription. This indicates that the regulation of glycolysis by BACE-1 occurs upstream of gene expression, acting like a master switch.

Phase Two (the later stage) : A vicious cycle of self-reinforcement is formed

Subsequently, two key glycolytic enzymes (LDHA and PKM2) became more active than before, and this change was accompanied by an enhancement of an epigenetic marker called "histone lactation" (H4K12la). This forms a "self-reinforcing" cycle: lactic acid promotes epigenetic changes, and these changes in turn keep the cells in a high lactic acid state continuously, resulting in the cells being firmly "locked" in the metabolic pattern of glycolysis [5][9].

Phase Three: The chain Reaction between electrophysiology and Secretion

Immediately after, we found that the expression level and current intensity of the Kv1.3 channel both began to increase, and at the same time, the number of exosomes released by the cells also significantly increased. A key finding is that even under the same lactic acid environment, gene knockout cells exhibit stronger Kv1.3 activation and exosome secretion than normal cells. This indicates that their entire reaction system has become extremely sensitive and is somewhat "overreacting".

We followed up with drug experiments to establish the precise order in this pathway. When we inhibited LDHA—in other words, lowered lactate production with drugs—we saw the downstream Kv1.3 activity suppressed as well. But what's really interesting is that, even when lactate is high, if you just block the Kv1.3channel directly, it'll pretty much knock out exosome release.

In conclusion, these results suggest that BACE-1 coordinates this pathway at multiple levels: it initiates glycolysis, amplifies the lactic acid-driven feedback loop, and ultimately shapes the electrophysiological behavior and secretory outcomes of the cells. This hierarchical regulatory mechanism highlights the profound impact of BACE-1 on microglial function and its core role in the pathogenesis of Alzheimer's disease.

3.5 Disruption of the BACE-1–lactate–Kv1.3–exosome axis drives synaptic dysfunction and cognitive decline

We systematically evaluated the overall impact of the "BACE-1-lactate-KV1.3-exosome" signaling axis in Alzheimer's disease through pathological, electrophysiological and behavioral experiments.

Tissue staining revealed that the plaques in the brains of BACE-1 knockout mice did indeed decrease, but the distribution range of soluble Aβ was actually wider - this is consistent with the speculation that pathogenic proteins are diffused in large quantities by exosomes.

Observations of microglia have shown that their morphology has become coarser and their branches more complex, which is a state of continuous activation rather than an abnormal short-term defense. Ultra-high-resolution imaging further revealed that the number of synapses in these mice was significantly reduced, corresponding to a decrease in LTP (an indicator of synaptic plasticity) in the hippocampus, indicating that the physiological basis of memory has been damaged.

At the behavioral level, these mice performed poorly in multiple memory tests, such as the water

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maze, Y-maze, and new object recognition, showing significant learning and memory impairments. The most crucial point is that when we simultaneously inhibit BACE-1 and Kv1.3, both synaptic function and learning and memory abilities are restored - this strongly demonstrates that the combined intervention of this signaling pathway has therapeutic potential[7][13].

The overall sequence of events can be described as follows: deletion of BACE-1 leads to lactate accumulation, which in turn causes misfolding of the Kv1.3 channel, ultimately leading cells to release large amounts of exosomes containing toxic material. This particular set of events seems to be a major cause of cellular dysfunction and behavioral defects in Alzheimer's disease. As a consequence, the key to the treatment of this disease may be to carefully balance and regulate all steps of the signaling pathway in fine detail. This may represent a novel strategy to slow disease progression.

4. Discussion

Our study provides a fairly complete framework to explain the seemingly paradoxical roles of BACE-1 in microglia. Long story short, here's what happens: on the one hand, if you inhibit BACE-1, that's great — it means that the microglia are better at clearing Aβ, and as long as it's only in the short-term, it will help protect neurons. On the other hand, however, that's where the good news ends, because that process sets into motion a series of unexpected events: the loss of BACE-1 leads to the accumulation of lactate, which then leads to the overactivation of Kv1.3 channel, which then leads to the exosomes that contain toxic substances being released by the microglia. And all that feels like a Pandora's box being opened, instead of preservation of synapses and cognition.

The combination of the positive and negative aspects perfectly explains why broad-spectrum BACE-1 inhibitors always fail in clinical trials: although they reduce amyloid plaques, they accidentally activate more dangerous pathogenic signaling pathways.

So, where is the solution? The key lies in finding the precise point of application of BACE-1 in this chain: If it mainly controls the initiation of glycolysis, then the combination of BACE-1 inhibitors and metabolic regulators (such as LDHA inhibitors) may yield better results. If its impact lies in amplifying a vicious cycle, then targeting the "histone lactation" stage might prevent the continuous abnormal activation of microglia. Or, if it controls the sensitivity of the Kv1.3 channel or the production of exosomes, then the direct combination use of Kv1.3 blockers will produce a synergistic protective effect.

In conclusion, our work has established that the "BACE-1 - lactate - Kv1.3 - exosome" signaling axis is the key mechanism driving Alzheimer's disease, and it has also opened up new ideas for future treatment: no longer pursuing a single target, but designing a reasonable multi-target combination. This more comprehensive perspective not only enables us to have a deeper understanding of the metabolic signals of microglia, but also points out a clear transformation direction for us: that is, how to precisely block the spread of their pathogenic behaviors while retaining their beneficial functions.

5. Conclusion

Through this research, we have finally revealed the clear "Pandora's box" of BACE-1's role in microglia: although enhancing the clearance of Aβ can be achieved by inhibiting BACE-1, this unintentionally opens another "Pandora's box". That is, BACE-1's absence leads to a significant accumulation of lactic acid, which then overstimulates the Kv1.3 potassium channel, and then microglia begin to spread toxic substances throughout the brain via exosomes, and then promote disease progression rather than delay it.

Moreover, this research also provides an explanation for the following question that has troubled us for a long time: Why have so many clinical trials targeting BACE-1 failed (or even worsen the disease)?

The answer is that BACE-1 has a dual effect: on one hand, it can remove amyloid proteins; on the other hand, it will also trigger the activation of hidden pathogenic pathways that play a contrasting role.

Therefore, in the future, we should abandon the current one-dimensional thinking of targeting Aβ and adopt multi-targeted combination therapy instead. For instance, if we combine the use of BACE-1 inhibitors with drugs that inhibit Kv1.3 channel or regulate lactate metabolism, in addition to clearing protein waste, we can also block the transmission chain of diseases, achieving more effective and longer-lasting neuroprotection.

From a broader perspective, through this research, we have also changed our understanding of

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microglia: they are not entirely “good” or “bad”, but like chameleons, their functions change according to their metabolic states. This new understanding provides a powerful theoretical basis to help us cross the current therapeutic stalemate, and meanwhile, we also found that “metabolism–ion channel–exosome” signaling pathway may be a new target for regulating microglia.

There are several interesting directions that are especially worth trying: Does this newly discovered pathway also participate in tau pathology or other neurodegenerative diseases?

Can we develop more precise and clinically applicable drugs to regulate it?

If we combine multi-omics approaches with high-resolution imaging technology, we may be able to map the dynamic changes of microglia during disease progression and thus provide us with more comprehensive understanding of the roles of these brain guardians in maintaining brain homeostasis and triggering neurogeneration.

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