Advances in sirtuin research in lung diseases (Review)

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Abstract. Sirtuins, which comprise a family of NAD+-dependent deacetylases, have emerged as critical regulators in the pathogenesis of pulmonary diseases because of their pleiotropic roles in oxidative stress, metabolic homeostasis and inflammation. Growing evidence indicates that sirtuins modulate key cellular processes, including mitochondrial function, cell survival and death pathways, via protein deacetylation, thereby influencing disease progression in chronic obstructive pulmonary disease, pulmonary fibrosis and lung cancer. Notably, dysregulated sirtuin expression and activity are implicated in various lung pathologies, highlighting their dual potential as diagnostic biomarkers and therapeutic targets. The present review synthesized recent advances in sirtuin research, focusing on molecular mechanisms and translational applications, to provide a foundation for developing novel strategies for lung disease prevention and treatment. A comprehensive literature review of the relationship between sirtuins and lung-related diseases was conducted. Literature published in PubMed and Web of Science up to May 2025 was searched using the keywords 'sirtuin,' 'SIRTs,' 'lung disease,' and 'pulmonary disease.'

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1. Introduction

Respiratory diseases, including pulmonary fibrosis (PF), chronic obstructive pulmonary disease (COPD), lung cancer and acute lung injury (ALI), are characterized by high morbidity and mortality globally, representing a major health burden (1). The intricate pathophysiology underlying these conditions, coupled with the current limitations in therapeutic interventions, imposes substantial clinical and socioeconomic challenges that markedly impair patients' quality of life (2). In China, chronic respiratory diseases are prevalent diseases, with COPD, lung cancer, PF and infectious diseases (particularly tuberculosis and pneumonia) being the most prevalent forms (3). Epidemiological data indicate that ~13.7% of the Chinese population aged ≥40 years experience COPD, corresponding to nearly 100 million affected individuals (4). The primary etiological factors include tobacco smoking and ambient air pollution, which are collectively responsible for a substantial proportion of the disease burden (4). Lung cancer represents a major public health challenge in China and it is characterized by high incidence and mortality rates, with low early diagnosis rates and substantial treatment costs posing significant barriers to effective disease management (3). Similarly, PF and infectious pulmonary diseases (especially drug-resistant tuberculosis) present ongoing clinical challenges. The incidence of interstitial lung diseases, including PF, has continued to increase. Although anti-fibrotic agents such as pirfenidone and nintedanib have been incorporated into clinical practice, their therapeutic benefits remain limited and these drugs are associated with considerable financial burdens. Given the complex etiological factors contributing to pulmonary pathologies and their substantial mortality rates, the development of novel therapeutic strategies is of paramount importance. In this context, sirtuins, comprising a conserved family of NAD+-dependent deacetylases that regulate crucial cellular processes including metabolism, aging, stress responses and epigenetic modulation, have emerged as promising targets (5,6). Notably, specific sirtuin isoforms (SIRT1, SIRT3 and SIRT6) have been increasingly implicated in pulmonary pathophysiology, with accumulating evidence supporting their critical roles in various lung disease processes (7) (Fig. 1).

2. Classification and functions of sirtuins

Sirtuins were originally identified in yeast as silent information regulator 2 (Sir2) proteins (8). These proteins, encoded by the SIR gene family, were initially characterized for their roles in chromatin silencing through modulation of chromatin structure accessibility in *Saccharomyces cerevisiae* (9,10). Subsequent

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research has revealed that Sir2 homologs are ubiquitous across all biological domains, including archaea, bacteria and eukaryotes (11). Sirtuins have emerged as crucial regulators of diverse cellular processes that enhance cellular survival and longevity, including DNA repair, transcriptional regulation, metabolic homeostasis and stress response pathways (12). At the molecular level, sirtuins catalyze NAD+-dependent deacetylation, removing modified acyl groups from lysine residues from both histone and non-histone protein substrates. This enzymatic activity yields three products: deacetylated proteins, nicotinamide and 2'-O-acyl-ADP-ribose (13,14). Notably, the NAD+ dependence of sirtuins establishes them as cellular energy sensors, enabling the integration of metabolic status with targeted lysine deacetylation in various subcellular compartments (15,16).

The sirtuin family of class III histone deacetylases, which are evolutionarily conserved from prokaryotes to eukaryotes, are uniquely inhibited by nicotinamide (17). Mammalian systems express seven distinct sirtuin isoforms (SIRT1-7) that can be phylogenetically divided into four classes based on their amino acid sequence homology: Class I (SIRT1-3), class II (SIRT4), class III (SIRT5) and class IV (SIRT6-7) (12,18,19). Structurally, sirtuins exhibit conserved catalytic domains of ~270 amino acids that mediate their functional diversity. The canonical sirtuin architecture comprises a large Rossmann fold domain responsible for NAD+ binding and a smaller zinc-binding domain containing helical structures. These domains form a cleft that accommodates acetylated substrates, facilitating their binding and subsequent deacetylation (20,21).

The seven mammalian sirtuin isoforms display distinct subcellular localization patterns, enzymatic activities and substrate specificities, reflecting their diverse functional roles (22,23). SIRT1-7 are encoded by genes located at unique chromosomal loci (10q22.1, 19q13, 11p15.5, 12q, 5p23, 19p13.3 and 17q25, respectively) and their gene sizes varying from 6-37 kb (24,25). Subcellular compartmentalization critically determines sirtuin function. SIRT1 primarily localizes to the nucleus but it exhibits nucleocytoplasmic shuttling, as evidenced by heterokaryon assays (26,27). This ubiquitously expressed deacetylase participates in numerous physiological and pathological processes, including cell cycle regulation and apoptosis, DNA damage repair, metabolic homeostasis, oxidative stress response and cellular senescence and longevity modulation (28). SIRT2 displays dynamic localization, transitioning between the cytoplasm and nucleus during G_2/M phase progression (29). Beyond its established roles in microtubule dynamics and cell cycle control, emerging evidence has implicated SIRT2 in neurodegenerative disease pathogenesis (30). The mitochondrial sirtuins (SIRT3-5) serve as crucial regulators of oxidative metabolism and energy homeostasis (22). Among these, SIRT3 plays particularly diverse roles through the coordination of mitochondrial signaling pathways, including the substrate-specific deacetylation modulation of oxidative phosphorylation, reactive oxygen species detoxification, fatty acid β-oxidation, tricarboxylic acid cycle flux and amino acid metabolism (31). Beyond their canonical deacetylase function, SIRT4-6 exhibit additional enzymatic activities that are essential for metabolic regulation and stress adaptation. Notably, SIRT4 demonstrates robust ADP-ribosyltransferase activity, through which it post-translationally modifies target proteins to modulate critical processes including insulin secretion and amino acid metabolism (32). SIRT5 is a key metabolic regulator that removes malonyl and succinyl groups from lysine residues, thereby influencing mitochondrial function and urea cycle activity (33). SIRT6 possesses dual enzymatic capabilities, functioning as both a deacetylase and a long-chain deacetylase, facilitating its central role in maintaining genomic stability through DNA repair mechanisms and telomere maintenance (34). Although both SIRT6 and SIRT7 are nuclear proteins, they exhibit distinct subnuclear distributions. Specifically, SIRT6 primarily associates with chromatin, whereas SIRT7 predominantly localizes to the nucleolus. This compartmentalization facilitates their coordinated regulation of transcriptional and metabolic networks (35,36). Collectively, these multifunctional enzymes both orchestrate metabolic and bioenergetic pathways and serve as crucial guardians of cellular homeostasis under stress conditions. These diverse activities position sirtuins as promising therapeutic targets for age-related and metabolic disorders.

3. Sirtuins and lung disease

Sirtuins and PF. PF represents a terminal pathological manifestation of numerous chronic lung disorders and constitutes the most prevalent form of interstitial lung disease. This progressive condition ultimately leads to respiratory failure and severe complications, with current therapeutic approaches offering only palliative management rather than curative solutions (37,38). Idiopathic pulmonary fibrosis (IPF), the most common subtype of PF, has an exceptionally complex pathogenesis that remains incompletely elucidated. Current standard-of-care pharmacotherapies, primarily comprising glucocorticoids and immunosuppressants, aim to slow disease progression, but their clinical efficacy is limited (39,40). Although several approved treatments are available, their utility is constrained by significant adverse effects and marginal improvements in disease outcomes, underscoring the urgent need for novel therapeutic agents (41,42).

Emerging research has identified sirtuins as critical modulators of PF progression, positioning them as promising molecular targets for anti-fibrotic drug development (43). Among these proteins, SIRT1 is particularly well characterized, both serving as a key endogenous regulator of fibrotic processes and demonstrating diagnostic potential for IPF. Mechanistically, SIRT1 exerts its anti-fibrotic effects by modulating the Smad2/3 signaling pathway, thereby suppressing the expression of pro-fibrotic genes and proteins in activated fibroblasts (44,45). Emerging evidence demonstrates that pharmacological inhibition of SIRT2 suppresses fibroblast activation and attenuates PF progression via modulation of the Smad2/3 signaling pathway (46). Furthermore, accumulating studies suggest that SIRT3, SIRT6 and SIRT7 have potential protective roles in the pathogenesis of PF (43). SIRT3 is particularly noteworthy, as it regulates p53-mediated senescence pathways in lung epithelial cells, thereby influencing both the initiation and progression of fibrotic processes (47). According to experimental studies, SIRT3 deficiency exacerbates PF by increasing mitochondrial DNA damage and apoptosis in alveolar epithelial cells (48), whereas SIRT3 overexpression protects against asbestos-induced fibrosis by preserving mitochondrial

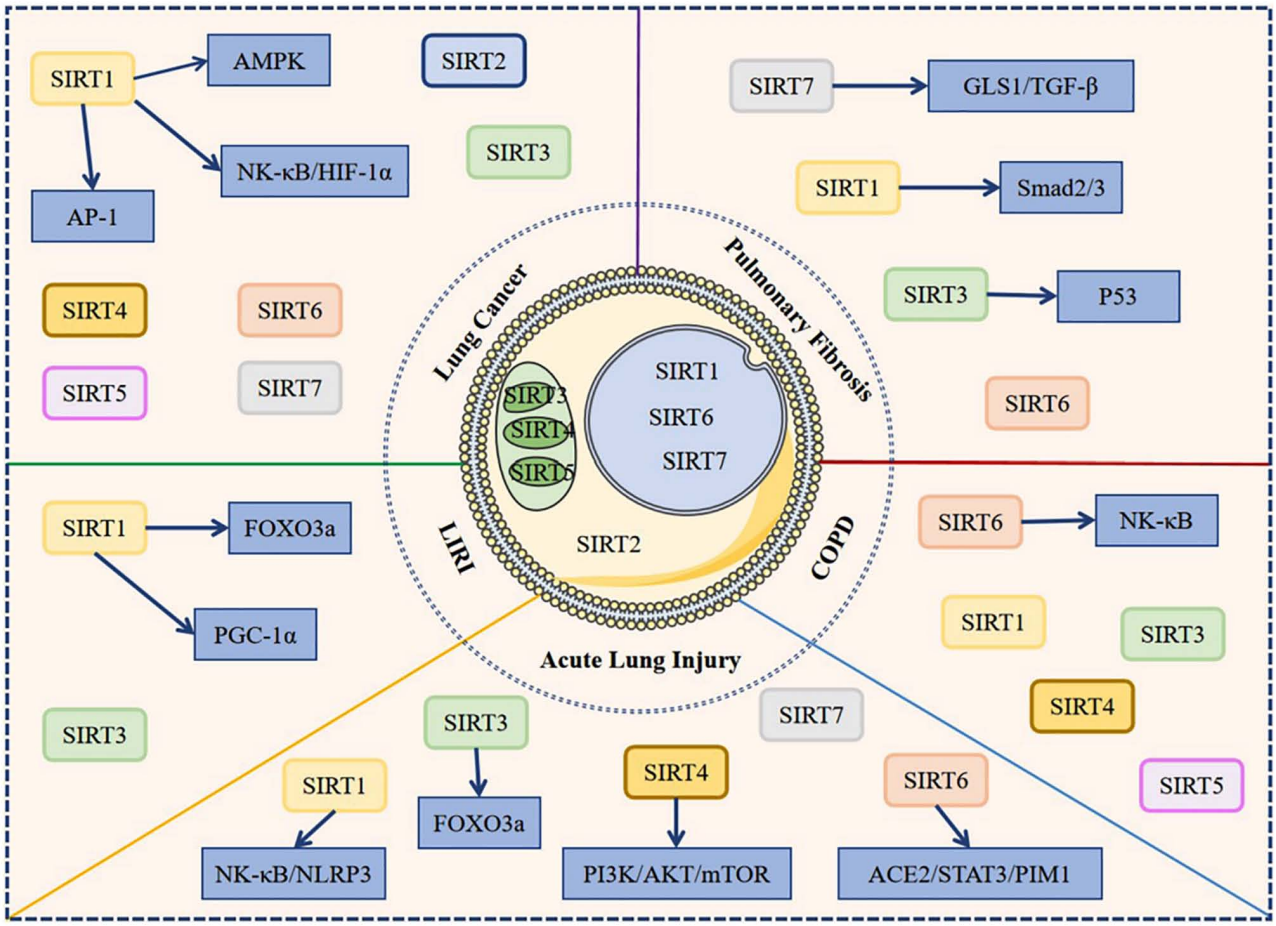

Figure 1. Comprehensive regulatory network of sirtuins in pulmonary pathophysiology. SIRT1-SIRT7, Sirtuin 1-Sirtuin 7; AMPK, AMP-activated protein kinase; NF-κB, Nuclear factor-kappa B; HIF-1α, Hypoxia-inducible factor-1α; AP-1, Activator protein-1; FOXO3a, Forkhead box O3a; PGC-1α, Peroxisome proliferator-activated receptor γ coactivator-1α; NLRP3, NOD-like receptor family pyrin domain-containing 3; GLS1, Glutaminase 1; TGF-β, Transforming growth factor-β; Smad2/3, Mothers against decapentaplegic homolog 2/3; p53, Tumor protein p53; PI3K, Phosphatidylinositol 3-kinase; AKT, Protein kinase B; mTOR, Mammalian target of rapamycin; ACE2, Angiotensin-converting enzyme 2; STAT3, Signal transducer and activator of transcription 3; PIM1, Proviral integration site for Moloney murine leukemia virus 1; LIRI, Lung ischemia-reperfusion injury; COPD, Chronic obstructive pulmonary disease.

integrity and reducing fibroblast recruitment (49). Similarly, SIRT6 can mitigate bleomycin-induced PF by activating lipolytic metabolic pathways (50). Complementing these findings, Choudhury *et al* identified SIRT7 as a novel endogenous regulator of glutaminase 1 expression, demonstrating its capacity to inhibit transforming growth factor-β-mediated fibrotic responses (51).

Sirtuins and lung cancer. Lung cancer is a leading cause of cancer-related death globally (52) and its morbidity and mortality are strongly associated with smoking patterns (53). Multiple risk factors for lung cancer have been identified, including smoking, domestic biomass fuel exposure, occupational exposure, diet and nutrition, genetic factors and environmental air pollution (54). Lung cancer is usually categorized into small-cell and non-small-cell types and the most common clinical symptoms include cough, hemoptysis, dyspnea, weight loss and anorexia (54,55).

The role of SIRT1 in cancer pathogenesis remains controversial, with accumulating evidence demonstrating both oncogenic and tumor-suppressive functions in a context-dependent manner. Elevated SIRT1 expression has been documented in several malignancies, including acute myeloid leukemia, prostate cancer and non-melanoma skin cancer, suggesting its potential tumor-promoting effects (56,57). Conversely, downregulation of SIRT1 has been observed in other cancer types such as breast carcinoma, bladder cancer, glioblastoma, ovarian cancer and prostate cancer (58). In lung cancer, emerging evidence indicates that SIRT1 activation suppresses cancer cell migration by inhibiting epithelial-mesenchymal transition (EMT) (59). Furthermore, under hypoxic conditions, SIRT1 modulates key metastatic regulators including NF-κB and hypoxia-inducible factor-1α, thereby influencing lung cancer progression (60). This regulatory pattern appears conserved across cancer types, as demonstrated by studies in ovarian cancer, in which hypoxia-mediated SIRT1 downregulation promotes EMT (61). Notably, the anti-inflammatory properties of SIRT1 might contribute to its tumor-suppressive effects. During lung cancer metastasis, pro-inflammatory factor accumulation is counteracted by the SIRT1-mediated suppression of NF-κB and activator protein 1 signaling pathways (62). Conversely, SIRT1 deficiency leads to constitutive activation of these pathways, resulting in sustained inflammatory responses that can exacerbate malignant progression (63,64). In addition, quercetin has been demonstrated to induce apoptosis in A549 and H1299 lung cancer cell lines through

SIRT1/AMPK pathway-mediated autophagy (65). Similarly SIRT1, the precise role of SIRT2 in tumorigenesis remains controversial, with studies reporting both tumor-suppressive and oncogenic functions (66,67). Notably, SIRT2-mediated deacetylation of extracellular proteins has been reported to promote lung cancer metastasis (68). In contrast to the dual roles of SIRT1 and SIRT2, SIRT3 exhibits consistent tumor-suppressive activity in lung cancer (69). SIRT3 inhibits the growth and metastasis of small-cell lung cancer, enhances DNA damage repair and confers radioresistance in non-small cell lung cancer (NSCLC) cells (70,71). NSCLC, one of the most prevalent malignant tumors, has significant associations with multiple sirtuin family members. Current evidence indicates that all seven sirtuins participate in the pathogenesis of NSCLC through distinct mechanisms. Specifically, SIRT4 regulates mitochondrial dynamics to suppress NSCLC progression (72), SIRT5 induces apoptosis by inhibiting DNA damage (73), SIRT6 silencing triggers cell cycle arrest and apoptosis (74) and SIRT7 promotes tumor progression through destabilization of the ARF tumor suppressor (75).

Sirtuins and COPD. COPD is the third leading cause of mortality globally and it is characterized by progressive and irreversible airflow limitation (76,77). This debilitating respiratory disorder manifests through two primary pathological phenotypes: small airway disease resulting from peribronchial fibrosis and emphysema caused by alveolar wall destruction (78). Clinically, COPD presents with persistent respiratory symptoms including chronic cough and sputum production, markedly impairing patients' quality of life while posing substantial socioeconomic burdens (79).

Emerging evidence has highlighted the crucial involvement of sirtuins in the pathogenesis of COPD. SIRT1 provides protective effects by mitigating endoplasmic reticulum stress, apoptosis, oxidative stress and inflammation in COPD models, positioning it as a potential therapeutic target (80-82). Similarly, SIRT3 exerts protective effects against cigarette smoke-induced COPD by suppressing airway epithelial mitochondrial oxidative stress through MnSOD upregulation, suggesting its potential as a preventive target (83). SIRT6, which exhibits reduced expression in lung tissues from patients with COPD, plays a protective role by inhibiting cigarette smoke extract-induced cellular senescence. Mechanistically, SIRT6 deficiency leads to NF-κB activation; telomere shortening; and β-catenin, VEGF and NRF2 downregulation, thereby exacerbating oxidative stress and accelerating emphysema progression (84,85). Although the mitochondrial sirtuins SIRT4 and SIRT5 can theoretically influence COPD pathogenesis through mitochondrial regulation, their specific roles remain poorly characterized and further investigation is warranted.

Sirtuins and pulmonary ischemia-reperfusion. Lung ischemia-reperfusion injury (LIRI) represents a severe complication following lung transplantation that markedly compromises graft function and recipient survival while increasing postoperative morbidity and mortality rates (86). This pathophysiological process, resulting from unavoidable organ ischemia and subsequent reperfusion during transplantation, typically manifests as acute aseptic inflammation (87). The underlying mechanisms of LIRI involve multiple interconnected pathological processes, including oxidative stress, calcium overload, endoplasmic reticulum stress, inflammatory responses, dysregulated autophagy and apoptotic pathways (88,89). SIRT1 has emerged as a key regulator in the pathogenesis of LIRI. Experimental evidence demonstrates that nicotinamide pretreatment activates SIRT1 in isolated perfused rat lungs, attenuating oxidative stress and preserving pulmonary barrier function (90), β-hydroxybutyrate pretreatment suppresses alveolar macrophage pyroptosis via the SIRT1/Forkhead Box O3 (FOXO3) axis, thereby mitigating LIRI (91); Kaempferol administration ameliorates LIRI through SIRT1/PGC-1α-mediated mitochondrial protection, thereby reducing oxidative stress and apoptosis (92).

In addition to SIRT1, SIRT3 has been identified as another crucial sirtuin involved in the pathogenesis of LIRI. Experimental studies have demonstrated that pharmacological SIRT3 activation by melatonin, hydrogen sulfide and procyanidin B2 protects against various forms of LIRI (93-95). Conversely, SIRT3 downregulation exacerbates LIRI through enhanced mitochondrial fission and oxidative stress (96). These collective findings position both SIRT1 and SIRT3 as promising therapeutic targets for LIRI treatment. Notably, although SIRT1 has been well documented to participate in ischemia-reperfusion injuries across multiple organ systems including the brain, intestine and kidneys (97-99), the potential pleiotropic effects of SIRT3 in extrapulmonary ischemia-reperfusion contexts remain to be elucidated.

Sirtuins and ALI. ALI is characterized by acute hypoxemic respiratory failure resulting from diffuse damage to the alveolar-capillary membrane caused by various direct or indirect insults, leading to interstitial and alveolar edema. Emerging evidence has implicated multiple sirtuins (SIRT1, SIRT3, SIRT4, SIRT6 and SIRT7) in the pathogenesis of ALI. Among these, SIRT1 has been the most extensively investigated because of its multifaceted roles in different ALI models, followed by SIRT3. The contributions of other sirtuins to ALI remain relatively unexplored.

Current research demonstrates that pharmacological agents including metformin, quercetin and artesunate protect against lipopolysaccharide- or sepsis-induced ALI through distinct SIRT1-dependent mechanisms, including modulation of the SIRT1/NF-κB/NLRP3 inflammasome pathway (100), regulation of the SIRT1/P53/SLC7A11 axis (101); activation of the Sirt1/Nrf2/Gpx4 antioxidant system (102) and enhancement of SIRT1/AMPK signaling (103). These findings underscore the central role of SIRT1 in ALI therapeutics.

Similarly, SIRT3 plays a crucial protective role in sepsis-induced ALI through FOXO3a regulation. Under septic conditions, decreased SIRT3 levels lead to FOXO3a hyperacetylation, exacerbating oxidative stress and cellular apoptosis, thereby aggravating lung injury (104). Conversely, SIRT3 upregulation promotes FOXO3a deacetylation and activation, attenuating apoptotic pathways and ameliorating ALI severity. Emerging evidence demonstrates that pharmacological activation of SIRT3 protects against ALI through distinct molecular mechanisms. Menaquinone-4 mitigates ALI by activating the SIRT3/p53/SLC7A11 pathway (105), whereas stellate ganglion block ameliorates LPS-induced ALI through

the SIRT3-mediated regulation of oxidative stress (106). These findings collectively highlight SIRT3 modulation as a promising therapeutic strategy for ALI prevention and treatment.

Although less extensively studied, other sirtuins also participate in the pathogenesis of ALI. SIRT4 overexpression attenuates sepsis-induced ALI by modulating the JAK2/STAT3 and PI3K/AKT/mTOR signaling axes (107). SIRT6 protects against LPS-induced lung injury through the ACE2/STAT3/PIM1-mediated suppression of epithelial cell inflammation and apoptosis (108), with SIRT6 deficiency exacerbating p53-dependent ferroptosis in murine ALI models (109). In addition, the miRNA-762/SIRT7 axis has been implicated in regulating LPS-induced ALI pathogenesis (110).

4. Summary and outlook

Emerging evidence has highlighted the crucial involvement of sirtuins in the pathogenesis of various pulmonary disorders. Extensive preclinical investigations have established that sirtuins are master regulators of multiple pathological processes in lung diseases, including PF, COPD, lung carcinoma and ALI, primarily through their modulation of oxidative stress, inflammatory cascades, mitochondrial dynamics, programmed cell death and fibrogenesis.

Among the seven mammalian sirtuin isoforms, SIRT1 and SIRT3 are the most extensively characterized members involved in pulmonary pathophysiology. These proteins exert their protective effects through two principal mechanisms: epigenetic regulation of pro-inflammatory and pro-fibrotic gene expression via the deacetylation of key transcription factors (FOXO3a, p53 and NF-κB) and preservation of cellular homeostasis through the maintenance of mitochondrial integrity and enhancement of endogenous antioxidant defenses. Although preliminary studies have suggested the potential involvement of other sirtuins (SIRT4, SIRT6, SIRT7) in pulmonary disease modulation, their precise molecular mechanisms and pathophysiological significance remain incompletely understood and warrant systematic investigation.

Despite significant advances in understanding sirtuin biology in pulmonary diseases, several critical knowledge gaps warrant further investigation. First, although the functional roles of SIRT1 and SIRT3 have been extensively characterized, those of other sirtuin isoforms (particularly SIRT2 and SIRT5) in lung pathophysiology remain poorly understood. Systematic studies are needed to elucidate their cell type-specific functions across distinct pulmonary cell populations, including alveolar epithelial cells, fibroblasts and immune cells. Second, the environmental regulation of sirtuin activity represents an important but understudied area. Key questions include the ability of common environmental compounds (such as cigarette smoke, particulate matter and occupational toxins) to modulate sirtuin expression and enzymatic activity and the potential roles of epigenetic mechanisms (such as DNA methylation, histone modifications and non-coding RNAs) in mediating these effects. Addressing these questions is essential for understanding disease heterogeneity and developing personalized therapeutic approaches.

From a translational perspective, several challenges must be overcome. First, current sirtuin modulators (such as resveratrol and nicotinamide) lack sufficient target specificity and optimal pharmacokinetic profiles. Second, tissue-specific delivery systems are needed to achieve therapeutic sirtuin modulation in the lungs while minimizing off-target effects. Third, the pleiotropic roles of sirtuins in aging and metabolic regulation necessitate comprehensive safety assessments in preclinical models to evaluate potential adverse effects.

Future research should focus on integrated multiomics analysis (combining transcriptome, proteome and metabolomics to systematically analyze the regulatory network of sirtuins in lung diseases), gene editing and organoid modeling (constructing lung organoids genetically modified by sirtuins using CRISPR-Cas9 technology to simulate the disease process and screen for potential drugs) and clinical translational research (promoting clinical trials of sirtuin modulators in clinical trials, especially efficacy assessments in patients with IPF and COPD).

In conclusion, the mechanisms of action of sirtuins in lung diseases and translational medicine research are being extensively explored. In-depth exploration of their molecular regulatory networks and the development of targeted drugs combined with precision medicine strategies are expected to provide breakthroughs in the treatment of lung diseases. Future research should combine basic and clinical aspects to promote the translation of sirtuin-targeting strategies from laboratory research to practical medical applications.

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Availability of data and materials

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Authors' contributions

SX performed research and wrote the main manuscript text. WC, XL and YW conducted an investigation on research methods and processes. YK provided funds, checked the manuscript, participated in the present study and made substantial contributions to the conception, design and interpretation of data. Data authentication is not applicable. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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