Pneumoconiosis: current status and future prospects

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Abstract

Pneumoconiosis refers to a spectrum of pulmonary diseases caused by inhalation of mineral dust, usually as the result of certain occupations. The main pathological features include chronic pulmonary inflammation and progressive pulmonary fibrosis, which can eventually lead to death caused by respiratory and/or heart failure. Pneumoconiosis is widespread globally, seriously threatening global public health. Its high incidence and mortality lie in improper occupational protection, and in the lack of early diagnostic methods and effective treatments. This article reviews the epidemiology, safeguard procedures, diagnosis, and treatment of pneumoconiosis, and summarizes recent research advances and future research prospects.

Keywords: Pneumoconiosis; Epidemiology; Diagnosis; Treatment; Emerging technologies

Introduction

Pneumoconiosis is a group of heterogeneous occupational interstitial lung diseases caused by the inhalation of mineral dust in the lungs, which leads to lung dysfunction.[1] This dust is primarily inorganic particles, such as free silica dust, asbestos fibers, dust from coal mines, and mixed silicate dust. The pathological characteristics of the disease are chronic pulmonary inflammation and fibrosis.[2] Inflammation can promote pulmonary fibrosis, and then lead to pneumoconiosis.[3]

Pneumoconiosis is prevalent worldwide, and has maintained a relatively high incidence in recent years.[4,5] It remains a severe global public health issue due to the lack of prevention of dust in the workplace, the failure of diagnosis of the disease at the early stages, and limited effective treatments for the disease. New diagnostic methods and therapeutic targets provide hope for solving the clinical problems of pneumoconiosis, and various new research techniques, such as high-throughput omics technology and the rapid development of bioinformatics technology provide opportunities for in-depth study.

Therefore, we have summarized the latest epidemiological research and current methods of the diagnosis and treatments for pneumoconiosis. We also analyzed various emerging technologies and summarized current advances in pneumoconiosis research to identify potential treatment targets and areas for future research.

Epidemiology of Pneumoconiosis

Over the past few decades, many measures have been put in place to protect workers against dust inhalation. However, pneumoconiosis is still a threat to public health.[4,6-9] According to the Global Burden of Disease studies,[10-13] although the worldwide prevalence of pneumoconiosis has shown a downward trend since 2015, there are still a large number of patients. The prevalence of pneumoconiosis is around 527,500 cases, with over 60,000 new patients reported globally in 2017.[10] The mortality of pneumoconiosis patients remained at a high level in recent years,[14-17] with over 21,000 deaths each year since 2015.

Alarmingly, pneumoconiosis has re-emerged even in the United States and Australia, countries with highly developed healthcare systems, high standards of workplace safety procedures, and highly mechanized mining practices that reduce workers' exposure to particles.[9] It is possible that other less developed countries, especially those with inadequate reporting systems, have many patients that have not yet been diagnosed and reported.

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This makes it difficult to determine the exact numbers of existing pneumoconiosis cases. In addition, a diagnosis of pneumoconiosis can cause workers to lose their jobs, meaning many of them are unwilling to submit to physical examinations, even if they are experiencing symptoms. This can lead to pneumoconiosis patients not being detected in time.[18] Therefore, pneumoconiosis likely has higher rates of morbidity and mortality than previously thought.

Safeguard Procedures of Pneumoconiosis

The Occupational Safety and Health Administration (OSHA) modified standards of occupational exposure to respirable crystalline silica (RCS) in 2016. The permissible exposure was limited to 50 (mg/m^3) and stricter exposure limits were imposed on specific industries. OSHA estimates this rule will substantially lower the risk of material impairment to health. Additionally, OSHA also made clear provisions for safeguard procedures, which include a series of provisions to protect employees, such as methods of controlling dust concentration, assessment of exposure, using tools to prevent exposure, regular physical examination, and pathological records preservation.

Silicosis still occurs in new industries such as the manufacturing of denim jeans and the processing of artificial stones (AS). Sandblasting is involved in the process of manufacturing denim jeans, where the high pressure results in high concentrations of RCS.[19] AS have become popular in recent decades because of their increasing affordability. However, the silica content of AS is much higher than that of natural marble or granite stone. Cutting and polishing AS also results in high levels of exposure to RCS dust. Long-term inhalation of high concentrations of RCS during sandblasting and AS processing has resulted in the rapid progression of accelerating silicosis.[20-24] It is commonly found in young workers, meaning that patients may suffer severely from this disease and die at a relatively young age, which is a cause of widespread concern.[6] Additionally, the development of nanotechnology has increased the use of nanomaterial. It is currently an emerging industry and there are few patients presently reported, but the risk for exposure is still worth noting since studies have shown that nano-silica is likely to cause lung inflammation and fibrosis.[25]

It is clear that dust exposure in these industries is far more dangerous than previously thought, and this reflects failure to recognize and control risks associated with silica exposure in modern work practices.[6] More vigilance is needed to identify and control occupational exposure to these dangerous dust particles.

Diagnosis of Pneumoconiosis

Current situation

The screening of pneumoconiosis primarily relies on observing a history of exposure to harmful dusts and performing chest radiography. We have summarized the general process of pneumoconiosis diagnosis in Figure 1. The current criteria were compiled by the International
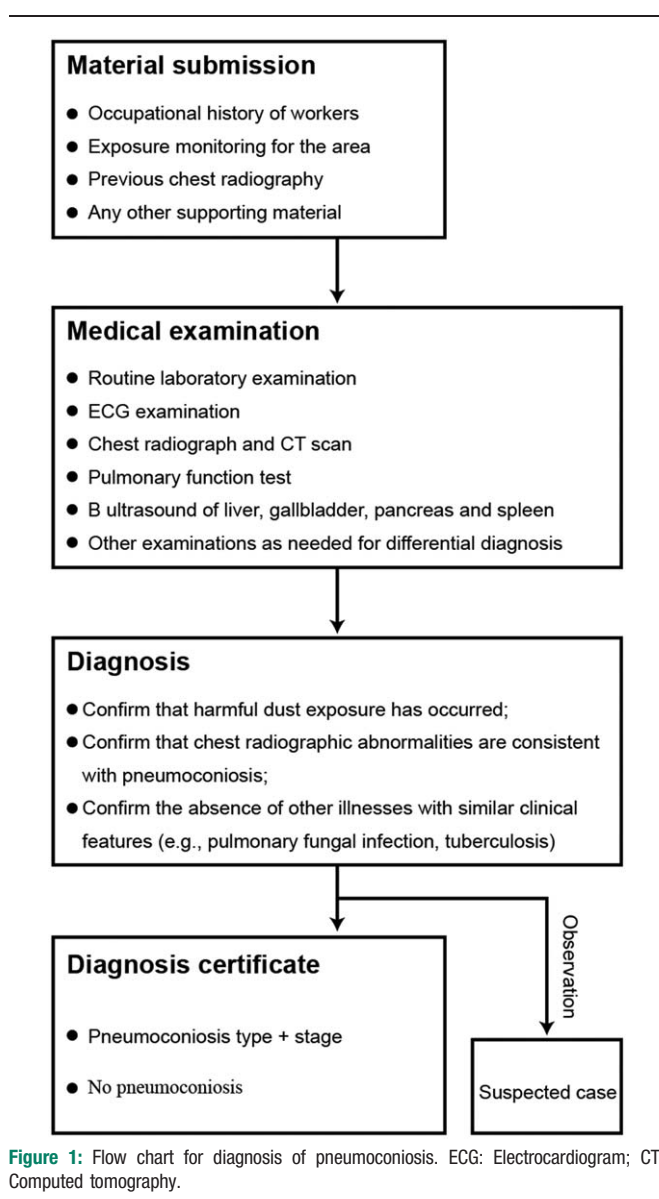

Figure 1: Flow chart for diagnosis of pneumoconiosis. ECG: Electrocardiogram; CT: Computed tomography.

Labour Organization (ILO). The last revision of the ILO International Classification of Radiograph of Pneumoconiosis (ILO/ICRP) was made in 2011. This version introduced digital standard images and specified the quality of diagnostic monitors for the screening of pneumoconiosis. The National Institute of Occupational Safety and Health (NIOSH) of the United States currently certifies the proficiency of technicians responsible for reading digital radiographs of pneumoconiosis.[26] The use of digital standard images makes telemedicine and remote counseling possible, and provides ample opportunity for the accurate and timely diagnosis of pneumoconiosis. While high-resolution computed tomography (HRCT) is more sensitive to the detection of early signs than chest x-rays are,[27] the International Classification of HRCT for Occupational and Environmental Respiratory Diseases (ICOERD) has developed and recommended a list of diagnoses for occupational and environmental related lung diseases.[28] ILO classification remains valid for practical reasons, because there is currently no global standard.[8,27]

Because imaging techniques cannot assess the functional status of the patient, pulmonary function tests (PFT) are used as a supplementary method of the evaluation of the disease. PFT can be used to assess dyspnea, distinguish between obstructive and restrictive diseases, and assess disease severity.[27] It is difficult to differentiate pneumoconiosis from similar diseases because of its long incubation period and vague clinical symptoms. Diagnosis relies on laboratory examinations, such as bronchoalveolar lavage fluid (BALF), to observe the sediment in the alveoli and exclude similar pulmonary diseases.

In recent years, coal worker's pneumoconiosis (CWP) has reappeared in Australia. However, it has not been diagnosed as pneumoconiosis in a timely fashion due to the lack of experienced medical personnel.[8] Diagnosing pneumoconiosis is difficult due to complicated assessment procedures and the need for highly-trained medical personnel, since pneumoconiosis requires diagnostic methods that are far more complicated than a clinical diagnosis. Once a patient has been diagnosed with pneumoconiosis, their conditions are usually already extremely serious and difficult to treat. In order to control this disease, there is an urgent need to diagnose it at the preclinical stage, which would reduce its incidence and minimize its severity among affected workers. Significant advances in basic and clinical research are needed to identify feasible diagnostic biomarkers or methods.

Potential new diagnostic methods for pneumoconiosis

Electrical impedance tomography (EIT) may be a potential method for the early diagnosis of pneumoconiosis. The clinical symptoms of pneumoconiosis are difficult to observe during the early stages of the disease. However, the electrical conductivity of the cells significantly changes before clinical symptoms appear. EIT can accurately detect changes in electrical conductivity to diagnose pneumoconiosis prior to the appearance of clinical symptoms.[29] EIT is relatively harmless and can be used for dynamic monitoring. This method is presently applied in clinical settings to evaluate lung function.[30] Once relevant diagnostic criteria are established, this technology can immediately be used to diagnose pneumoconiosis in clinical practice. However, because this method has no diagnostic specificity, it cannot replace the chest x-rays as the diagnostic standard of pneumoconiosis.

Another diagnostic method is the three-dimensional magnetopneumography magnetic dipole model (3D-MPG-MDM). Recent research has found that 3D-MPG-MDM can non-invasively and inversely track the amount and distribution of particles within the lungs. These researchers have found that 3D-MPG-MDM can diagnose pneumoconiosis caused by inhalation of metal particles.[31] This is a powerful and effective technique that can assess the risk of pneumoconiosis by identifying how many particles are deposited in the lungs, which promotes earlier diagnosis and subsequent intervention. Although the incidence of metal particle-related pneumoconiosis could be effectively controlled using this novel technique, its safety, sensitivity, and specificity have not yet been systematically evaluated. Much work is required before this method can be applied in clinical settings.

Additionally, epigenetic pathways have received significant attention, while microRNA (miRNA) shows potential as a clinical diagnostic biomarker. miRNA is widely involved in post-transcriptional gene regulation, and is related to pneumoconiosis progression. Recently, researchers found that miRNA-155 expression levels were positively correlated with lung fibrosis in mice.[32] Because miRNA also possesses characteristics that are detectable by serum, has a stable morphology, and can be repeatedly freeze-thawed, it is suitable for clinical applications as a biomarker.[33] Further advances in miRNA-related research could provide biomarkers for assisting early diagnosis of pneumoconiosis.

Treatment of Pneumoconiosis

Current situation

Despite the high incidence of pneumoconiosis over the past few decades, established clinical treatments for pneumoconiosis are still very limited. The pneumoconiosis treatment regimen is detailed in Figure 2. The only life-saving therapeutic option in end-stage pneumoconiosis is lung transplantation.[34] Additionally, reports have demonstrated that some clinical treatments may relieve symptoms and possibly improve life quality.[35] The treatment of pneumoconiosis includes integrated treatments and whole lung lavage.[36] Integrated treatments are primarily based on the patient's common clinical symptoms, including cough, chest pains, and shortness of breath. Furthermore, treating pneumoconiosis-related complications (eg, respiratory infections, tuberculosis, chronic obstructive pulmonary disease, and pneumothorax) and encouraging patients to perform rehabilitative exercises can improve lung function and help relieve some symptoms.[37,38]

Whole lung lavage is used to remove phlegm, secretion, and dust or fibrotic cytokines from the patient's airway to delay the progression of pneumoconiosis. This procedure typically works best when used in the early stages of this disease,[39] when most of the inhaled dust is still in the pulmonary alveoli. However, so far, there is no evidence to support that whole lung lavage exhibits beneficial effects on pulmonary function or lung fibrosis. As an invasive procedure, it is not known whether whole lung lavage has long-term negative impacts on lung homeostasis. Performing lung transplantation is a feasible method for end-stage lung diseases, including silicosis, and is most promising in young patients.[34] The three-year survival rate of silicosis patients post-lung transplantation can reach 76%.[34] However, lung transplant recipients have a short median survival of only 6 to 7 years.[6] The limit in available lungs, significant contraindications, high cost, difficulty of the procedure, and the high risk of the operation have severely restricted the application of lung transplantation. The vast majority of pneumoconiosis patients suffer from this disease with no effective treatment to slow disease progression.

Despite all of this, pneumoconiosis still receives scant attention from researchers. There have been relatively few articles on the pathogenesis of pneumoconiosis or studies on drugs published in respected journals over the last
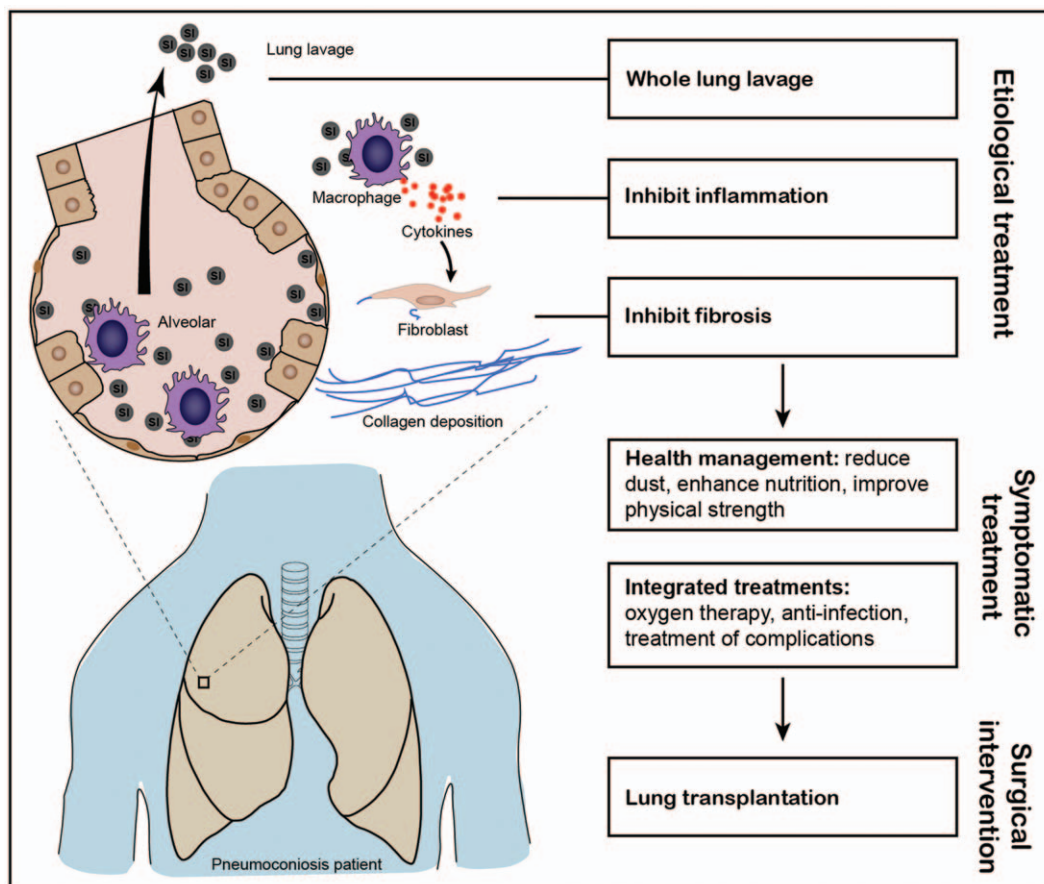
Figure 2: Clinical treatment of pneumoconiosis. SI: Silica.

5 years. Prevention alone is not effective, as evidenced by the CWP outbreak in Australia and the United States. Additionally, workers often choose not to wear protective equipment at work, while oftentimes new hazardous materials are not detected in time. All of this can cause harmful dust exposure. In order to address the pneumoconiosis problem, we must focus on its prevention, diagnosis, and treatment. There is an urgent need for effective therapeutic drugs to improve the quality of life and prolong survival time, which will require researchers to identify therapeutic targets and treatments.

Potential therapies for pneumoconiosis

New drugs

Currently, some drugs have been identified as having therapeutic effects on pneumoconiosis, and the related agents are summarized in Table 1. Drugs used in the treatment of other diseases may have the ability to treat pneumoconiosis. For example, the anti-fibrosis drug pirfenidone used for idiopathic pulmonary fibrosis (IPF),[40] the anti-inflammatory immune response drugs hydroxychloroquin,[41] corticosteroids, and infliximab[42], the antioxidant drug N-acetylcysteine,[43] and the vasodilators nicorandil and carvedilol[44,45] may inhibit pulmonary inflammation or fibrosis in pneumoconiosis experimental models. Corticosteroids with anti-inflammation properties have been shown to

relieve the clinical symptoms of patients with chronic beryllium disease.[46] Traditional Chinese medicine extracts such as dioscin,[47] astragaloside IV,[48] kaempferol,[49] tanshinone IIA[50] and dihydrotanshinone,[51] have been shown to relieve inflammation and fibrosis and alleviate silicosis in animal models.

Among these medications, pirfenidone appears most promising for pneumoconiosis treatment due to its clear anti-fibrotic properties. Pirfenidone is a pyridine compound exhibiting broad-spectrum anti-fibrotic effects.[52] It has the potential ability to decrease mortality from fibrosis by inhibiting pulmonary fibrosis and scar-formation by regulating or suppressing the expression of fibroblast growth factor (FGF), connective tissue growth factor (CTGF), transforming growth factor (TGF)-β, oxidative factors, and some pro-inflammatory factors in the lungs and kidneys.[53] An experimental study on silica-induced lung fibrosis in rats demonstrated that pirfenidone can slow the transformation from epithelial to mesenchymal cells when administered in 14- and 28-day treatments. These treatments were associated with a significant down-regulation of vimentine and up-regulation of E-cadherin,[40] suggesting that pirfenidone can exhibit an inhibiting effect on silica-induced epithelial-mesenchymal transition (EMT) in rats. Guo et al[40] further demonstrated that pirfenidone inhibited the expression of TGF-β and Smad2/3, suggesting that the effect of pirfenidone on EMT

Table 1: Recently discovered potential therapeutic agents for pneumoconiosis.

Therapeutic agents	Targets/mechanisms	Treatment effects	Models/patients	References
Clinical medications				
Pirfenidone	Inhibits the epithelial-mesenchymal transition	Ameliorates fibrosis	Rat silicosis model	[40]
Hydroxychloroquine	Blocks toxicity and lysosomal membrane permeability	Prevents silica-induced lung damage	Silica-exposed mouse	[41]
Infliximab	Anti-tumor necrosis factor-α	Improves inflammation	Rat silicosis model	[42]
N-acetylcysteine	Inhibits oxidative stress; downregulate proinflammatory cytokines	Ameliorates fibrosis and inflammation	Mouse silicosis model	[43]
Carvedilol	Modulates P-AKT/mTOR/TGF-β1 signaling	Ameliorates fibrosis and inflammation	Rat silicosis model	[44]
Nicorandil	Downregulates inflammatory and fibrotic cytokines; restores oxidant/antioxidant balance	Ameliorates fibrosis and inflammation	Rat silicosis model	[45]
Corticosteroids	Not reported	Improves symptoms	Patients with chronic beryllium disease	[46]
Traditional Chinese medicine extracts				
Dioscin	Promotes autophagy and reduces apoptosis	Ameliorates fibrosis and inflammation	Mouse silicosis model	[47]
Astragaloside IV	Continuously phosphorylates Smad3 in the TGF-β1/Smad signaling pathway	Ameliorates fibrosis	Rat silicosis model	[48]
Kaempferol	Modulates autophagy	Ameliorates fibrosis and inflammation	Mouse silicosis model	[49]
Tanshinone IIA	Suppresses TGF-β1/Smad signaling; inhibits NOX4 expression and activates the Nrf2/ARE pathway	Ameliorates fibrosis and inflammation, antioxidant	Rat silicosis model	[50]
Dihydrotanshinone I	Inhibits STAT1 and STAT3	Modulates T helper responses and improves inflammation	Mouse silicosis model	[51]

ARE: Antioxidant response element; mTOR: Mammalian target of rapamycin; NOX4: Nicotinamide adenine dinucleotide phosphate oxidase type 4; Nrf2: NF-E2-related factor 2; P-AKT: Phosphorylated-protein kinase B; STAT: Signal transducer and activator of transcription; TGF-β1: Transforming growth factor-beta1.

was likely mediated by the TGF-β/Smad2/3 signaling pathway. However, the clinical efficacy of pirfenidone in pneumoconiosis has not yet been reported.

Stem cell therapy

Stem cell therapy holds tremendous potential for treating pneumoconiosis, and research into this treatment is currently ongoing. Preclinical studies have demonstrated that intratracheal or tail vein administration of mesenchymal stem cells (MSCs) can inhibit inflammation and fibrosis, and have demonstrated therapeutic effects in the silicosis murine model.[54,55] Early phase clinical trials of stem cell therapy for pneumoconiosis are currently underway. One non-randomized uncontrolled trial on four silicosis patients showed that combining the treatments of MSCs and hepatocyte growth factor (HGF) could exhibit therapeutic effects. The average age of the four patients was 41.5 ± 6.6 years; mean exposure time to silica

was 9.0 ± 6.2 years. All patients displayed coughing and chest distress, and were confirmed to have lung lesions by high kilo-voltage chest X-rays and computed tomography (CT) scans. In that experiment, MSCs/HGF was injected intravenously once a week for three consecutive weeks at a dose of 2×10^6 cells/kg. Six months after the treatment, pulmonary function tests, chest X-ray, and CT were performed; blood lymphocyte and serum immunoglobulin G concentrations were evaluated. The treatment was found to be generally safe. Symptoms such as coughing and chest pains gradually improved, while lung function significantly improved. Images of the lungs showed that the lesion was partially absorbed (clinical trial registry number NCT01977131).[56]

While preclinical and clinical studies of MSC in pneumoconiosis have confirmed its effectiveness as a treatment, the mechanism is not yet fully understood. Some studies have suggested that MSC could have anti-inflammatory and

anti-fibrotic properties. Research performed by Huang et al[57] used imaging-guided MSC-based therapy in a silicosis murine model, and showed that MSCs have various anti-inflammatory properties. This included down-regulation of pro-inflammatory cytokines, suppression of macrophage activation, and a delay in fibrosis progression. An additional study found that MSCs regulate macro-phage function by a variety of mechanisms, which contributes to their anti-inflammatory activity and is associated with MSC function and survival.[58]

The increased use of induced pluripotent stem (iPS) cells enables researchers to extract mature cells from the patient's skin or blood and reprogram these cells to an immature, embryonic state. These iPS cells can then be used to culture mature cell types, even lungs, in the laboratory, which provides a method for developing personalized treatments.[59] Stem cell therapy has been used to treat hematological diseases via bone marrow and epidermal transplants. Recently, it was successfully used in the clinical treatment of macular degeneration and junctional epidermolysis bullosa.[52-55] These reports have demonstrated that stem cell therapy is promising for use against fibrotic lung disorders.

While stem cell therapy has been proven to be an effective treatment for pneumoconiosis, many obstacles remain. First, the safety and efficacy of this treatment must be evaluated. Second, stem cell therapy is in its early stages and there are still technical barriers to overcome. Moreover, the rigorous clinical trials and the myriad ethical issues must be addressed before its clinical application. These experimental therapies are also ex-tremely expensive. However, despite these many obstacles, stem cell therapy shows much promise and will drive research in the coming years.

Future Research Prospects

Emerging techniques bring new hope

The emergence of high-throughput omics technology, the rapid development of bioinformatics, and breakthroughs in gene-editing technology all show promise for the in-depth study of pneumoconiosis. Recently, high-through-put omics have been used in pneumoconiosis research. Genomics studies on silicosis suggest that single nucleotide polymorphisms are associated with susceptibility to pneumoconiosis and several disease-related single nucleo-tide polymorphisms have been found (eg, rs73329476, rs12812500).[60] Epigenomics studies have shown that some miRNAs are closely associated with pneumoconiosis risk and disease progression, and have the potential to serve as non-invasive biomarkers and prognostic markers for early pneumoconiosis.[61,62] Transcriptomics and proteomics identify a range of differentially expressed protein-coding genes and proteins. Numerous key targets (eg, activating transcription factor 3, chemokine [C-C motif] ligand 2 [CCL2], C-C motif chemokine receptor 2 [CCR2], protein tyrosine phosphatase non-receptor type 2 (PTPN2), p-signal transducer and activator of transcrip-tion [STAT]3 and signaling pathways have been identified (eg, Janus kinase-STAT [JAK-STAT] pathway, nucleotide

binding oligomeric domain [NOD]-receptor interacting protein 2 [RIP2]-nuclear factor-kappa B [NF-κB] path-way).[63-66]

In the future, application of new omics technologies to the study of pneumoconiosis, such as integrating omics and spaceomics, may provide more comprehensive and unbi-ased approaches, which will greatly enhance our ability to study the pathological and molecular mechanism of pneumoconiosis. These new technologies provide hope for identification of effective diagnostic and therapeutic treatments for pneumoconiosis, including silicosis.[59]

However, the targets selected by omics must be verified before their clinical application. The emergence of gene editing technologies has greatly accelerated this process. The development of gene-editing technologies, such as clustered regularly interspaced short palindromic repeats (CRISPR)/CRISPR-associated systems (Cas9) technology, has greatly improved our ability to modify genes with unprecedented flexibility. We can now directly and effectively edit cell- or animal-specific target genes and alter their function, providing significant advantages over other methods.[67] The combination of gene editing technologies and omics technologies will contribute to the further discovery of new genes and proteins with important functions and applications, and lay the foundation for the diagnosis and treatment of pneumoco-niosis. Figure 3 visually demonstrates the application of these technologies. While gene-editing technologies are still primarily used in cell experiments and animal model building for research, they have shown promise for controlling disease and xenotransplantation.

The use of gene-editing technologies and omics methods to treat pneumoconiosis is still in the early stages of research. While there are still some technical difficulties to overcome before its clinical application, these technologies are at the core of future research for treatments against pneumoconiosis.

New targets bring new possibilities

The primary pathophysiological mechanisms of pneumo-coniosis relate to inflammation and fibrosis. Current studies suggest that inflammatory processes play a key role in pneumoconiosis, which in turn promotes fibrosis progression.[68,69] The targets identified by omics are primarily focused on inflammatory signaling pathways relating to the immune system. When dust settles in the lungs, it activates an immune response. Inflammatory cells are then recruited and activated, secreting related inflammatory factors such as cytokines, chemokines, and adhesion molecules. This activates fibroblasts and pro-motes fibrosis progression. Chronic inflammation is a key symptom of pneumoconiosis in lung tissues and BALF.[70] As such, they could be effective therapeutic targets in the immunoinflammatory pathway. Macrophages were the first observed, and the Heppleston theory was proposed in 1969, which states that the death and disintegration of macrophages engulfing dust results in fibrosis.[71] In addition to macrophages, other related immune inflam-matory cells such as T[72,73] and B[74,75] lymphocytes have
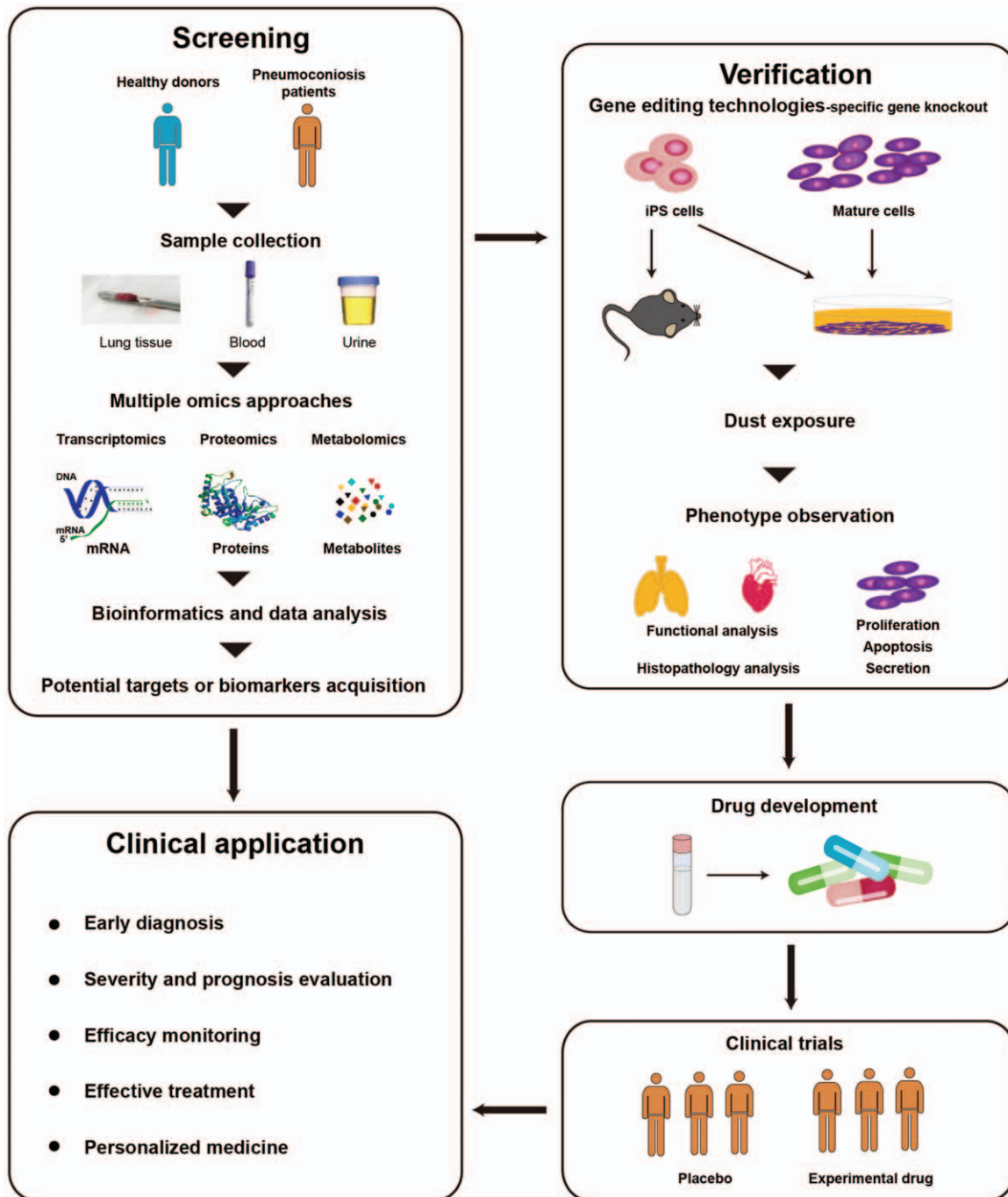
Figure 3: Application of new technologies in the research of pneumoconiosis. iPS cells: Induced pluripotent stem cells; mRNA: Messenger RNA.

been observed in the progression of pneumoconiosis. Multiple inflammation-related targets and signaling pathways have also been found to be therapeutic targets for pneumoconiosis. One study showed antagonizing macrophage receptor with collagenous structure (MARCO) with PolyG can ameliorate silica-induced fibrosis in rats.[76] Inhibiting NOD-like receptor protein 3 (NLRP3) inflammasome with MCC950 also showed anti-fibrosis in silica-treated bronchial epithelial cells.[77] Blocking some signaling pathways, including CD44-ras homolog family member A (RhoA)-Yes-associated protein (YAP), RhoA/ Rho kinase, and Fas cell surface death receptor (FAS)-

caspase-8 can also relieve pneumoconiosis in animal models.[78-80] miRNAs are key regulators of gene transcription, and regulate the production of intracellular messenger RNA (mRNA) and subsequent proteins. A study showed that miRNA-29 (which regulates the Wnt/ β-catenin pathway) and miRNA-326 (which targets tumor necrosis factor superfamily-14 and polypyrimidine tract-binding protein) have the effect of inhibiting silicosis fibrosis.[81,82] These studies can help with finding drugs or drug therapeutic targets. However, these targets have remained in the preclinical stage and have not been used clinically due to a lack of research.

Research on the mechanisms behind immune inflammation pathways is not sufficient. Most studies focus on a single aspect, while the pathological mechanisms behind pneumoconiosis are complex. The cells associated with lung inflammation form a complex cell network, while the biological effects of the cytokines they produce interact with each other. Blocking a single target might be insufficient to change the degree of inflammation and to effectively treat the disease, which could be why it is difficult to clinically administer the newly found targets. Therefore, research into multiple targets of the pathological mechanisms of pneumoconiosis is needed. The identification of these new targets will provide new possibilities for treating pneumoconiosis.

Summary

In this review, we summarized the current clinical status and research prospects of pneumoconiosis. At present, the lack of effective dust prevention, early diagnosis methods, and disease-specific drug treatments are major problems in the management of pneumoconiosis. Pneumoconiosis is still a worldwide threat, and therefore more extensive research into its pathological mechanisms is of great importance. Such research will assist in solving the problems related to the dust prevention and clinical treatment of pneumoconiosis, and the development of new technologies will provide powerful tools for these studies. Although there are many obstacles, we expect that further study will reveal the mechanisms behind pneumoconiosis, help with the development of dust-prevention methods, identify new biomarkers, evaluate therapeutic responses, and develop new drugs effective against pneumoconiosis, thus improving prognosis for patients worldwide.

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Conflicts of interest

None.

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