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**中国大陆学者发表的结核病英文文章摘要**

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**1. Microbiol Res. 2025 Jul 28;300:128292. doi: 10.1016/j.micres.2025.128292. Online ahead of print.**

Advancing roles of nitric oxide in tuberculosis: Promising targets for novel

anti-TB therapeutics.

Kong X(1), Yang J(1), Wang J(1), Li J(1), Jin X(1), Cai J(2), Ruan Y(2), Chen

R(3), Shen L(4), Pi J(5).

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Tuberculosis (TB), a chronic zoonotic infectious disease caused by Mycobacterium

tuberculosis (Mtb) infection, remains a major public health burden worldwide.

The increasing threatens of multidrug-resistant/extensively drug-resistant TB,

human immunodeficiency virus (HIV) co-infection, lack of effective vaccines and

diagnosis methods, as well as the low treatment efficacy of anti-TB therapeutics

lead to multiple difficulties and challenges in TB control. Host immune defense

is critical for the processes and outcome of Mtb infection control due to the

complex immune evasion mechanisms of Mtb, thus, it's of vital importance to

characterize the host immune responses and mechanisms during Mtb infection.

Nitric oxide (NO) has complex physiological functions in different conditions

and has been shown to play important roles in the immune defenses against Mtb

infection and even direct killings of Mtb, which still requires further systemic

evaluations. In this review, we summarized the current understanding for the

roles and mechanisms of NO in host defenses during Mtb infection, as well as the

role of NO in the occurrence, development and treatment of TB, which may provide

theoretical basis for the development of novel strategies in the prevention and

control of TB and drug-resistant TB.

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**2. Microb Pathog. 2025 Jul 31:107944. doi: 10.1016/j.micpath.2025.107944. Online**

**ahead of print.**

IL-32 positively regulates the AEBP1-IκBα-NF-κB-TNF-α axis to inhibit

Mycobacterium tuberculosis infection in human macrophages.

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S(1), Bao F(4), Xia X(5), Liu A(6).

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Mycobacterium tuberculosis (MTB), the causative agent of tuberculosis, survives

in host macrophages, the primary effector and antigen-presenting cells involved

in the host immune response. We previously showed that Interleukin-32 (IL-32) is

significantly increased in the peripheral blood plasma of tuberculosis patients,

can act as an anti-MTB agent. However, the underlying molecular mechanism for

its effect remains unknown. Here, we showed that inhibiting IL-32 with

monoclonal antibody increases MTB loads that was positively correlated with

higher antibody concentrations and longer exposure times. RNA-Sequencing result

indicated that 3797 genes were shown to be up-regulated in response to IL-32

inhibition, while 1365 genes were down-regulated. GO and KEGG analysis indicated

that classical signaling pathways, including TNF, cell cycle, and Wnt were

significantly enriched. Consistent expression trends were observed in NF-κB

pathway-related antibacterial factors that are functionally capable of

inhibiting MTB. Using differentially expressed gene and protein-protein

interaction analysis, AEBP1 was the gene with the most significant difference in

expression and regulated by IL-32. These findings suggest a dynamic molecular

and cellular mechanism by which IL-32 positively regulates the

AEBP1-IκBα-NF-κB-TNF-α axis to inhibit MTB infection in human macrophages.

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**3. Clin Chim Acta. 2025 Jul 30:120523. doi: 10.1016/j.cca.2025.120523. Online ahead of print.**

Exosomal cargoes as potential biomarkers for latent tuberculosis infection: a

promising frontier in diagnosis?

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This article examines the potential of exosome analysis in diagnosing latent

tuberculosis infection (LTBI). Tuberculosis (TB), caused by Mycobacterium

tuberculosis, remains a significant public health challenge due to its high

incidence worldwide. LTBI is a critical link in TB prevention and control but is

also a major obstacle to achieving TB eradication. Current diagnostic methods,

such as the tuberculin skin test and interferon-γ release assays, have

limitations. Exosomes, small vesicles released by cells, contain DNA, RNA,

proteins, and lipids that reflect the host cell's pathological state and may

serve as novel biomarkers for LTBI diagnosis. The article introduces exosome

formation and extraction methods, explores the pathological mechanisms of

mycobacterial vesicles and exosomes from infected hosts, and reviews research

progress on exosomal DNA, non-coding RNA, proteins, and lipids as diagnostic

markers for LTBI. Despite their potential, exosome research and application face

challenges, including complex separation and purification processes, the dynamic

nature of LTBI, and biomarker specificity. Future research will require

multidisciplinary collaboration to develop efficient exosome-separation

techniques and to further investigate the clinical value of these biomarkers,

ultimately promoting the application of exosomes in LTBI diagnosis and

contributing to global TB prevention and control efforts.

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**4. Clin Respir J. 2025 Aug;19(8):e70114. doi: 10.1111/crj.70114.**

Association of T-Cell Profiles With Disease Severity, Drug-Induced Liver Injury,

and Treatment Completion in Tuberculosis.

He Y(1), Zheng X(1), Dang Z(2), Hao X(1), Liu Y(1), Wang P(1), Chen Y(3), Wang

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**BACKGROUND:** Tuberculosis (TB) treatment is challenged by a long duration, poor

adherence, and the high risk of drug-induced liver injury (DILI). T-cell

immunity is essential for anti-mycobacterial defense, but current

immune-monitoring methods poorly reflect disease severity and treatment

response. Correlations of immune subpopulations with TB severity, DILI, and

treatment prognosis remain poorly understood.

**METHODS:** Peripheral blood mononuclear cells were collected from confirmed TB

patients (n = 40). Multiparameter flow cytometry analysis was used to assess

previously defined TB-associated T-cell phenotypes based on the co-expression of

cytokines and immune checkpoint molecules following stimulation with two

Mycobacterium tuberculosis peptides: culture filtrate protein 10 and early

secreted antigenic target 6. Patients were subgrouped by disease severity, DILI,

and treatment regimen (16-week short course vs. 24-week standard).

**RESULTS:** Specific subsets (14/124) were found to be associated with disease

severity. Notably, six of 14 subsets were positive for programmed death-ligand 1

(PD-L1), indicating its potential role in disease progression. DILI was

associated with three interleukin (IL)-21+ subsets (naïve CD4+, memory CD8+, and

interferon [IFN]-γ- CD4+ T cells) and IL-17+ memory CD8+ T cells, along with

PD-L1+TIM-3+CD4+ T cells (all p < 0.05). The 16-week and 24-week treatment

groups showed a significant difference in IFN-γ+ naïve CD8+ T cells at week 16

(p = 0.013), but not at treatment completion (p = 0.393), despite the different

durations.

**CONCLUSIONS:** This study identifies specific T-cell phenotypes associated with TB

severity, DILI, and treatment dynamics, highlighting potential immune markers

for disease monitoring and DILI prediction.

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**5. J Sep Sci. 2025 Aug;48(8):e70235. doi: 10.1002/jssc.70235.**

A Universal Strategy for Evaluation and Quantification of Potential Genotoxic

Impurities of Hydrazine Derivatives in Isoniazid Injection.

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Isoniazid is an irreplaceable first-line anti-tuberculosis drug. Its synthesis

requires hydrazine as a starting material, which is classified as a Class 2

genotoxic impurity according to the ICH M7 guideline. Consequently, there is a

risk of introducing potential genotoxic impurities (PGIs) containing

hydrazine-related alerting structural during production. This study employed a

systematic approach to assess the risks of PGIs in isoniazid and developed a

novel LC-MS/MS method for accurate control and quantification of these

impurities. First, candidate impurities were preliminarily predicted using

quantitative structure-activity relationship (QSAR) systems based on expert rule

and statistics. Subsequently, acceptable impurity limits were established based

on the prediction results. Finally, an LC-MS/MS method was developed to quantify

the selected PGIs. The results indicated that the impurities were classified as

Class 3 (3,5-bis(4-pyridyl)-4-amino-1,2,4-triazole), Class 4 (benzohydrazide,

picolinohydrazide, and nicotinohydrazide), and Class 5

(3,6-di(4-pyridyl)-1,4-dihydro-1,2,4,5-tetrazine). According to the ICH Q3B

guidelines and the threshold of toxicological concern (TTC) based on the

duration of administration, the limits of Class 4 and 5 impurities were set at

0.1%, and the limit of Class 3 impurities was set at 0.0066%. The established

method demonstrated excellent linearity (r > 0.999) within the range of

0.2-25 ng/mL. And good recoveries were observed in the range of 88.1%-113.0%.

The method was successfully applied to quantify impurities in 77 batches of

isoniazid injections, all of which complied with the established acceptance

limits. This universal strategy enhances quality control of isoniazid

formulations, ensuring clinical safety through robust impurity assessment and

validated analytical methodology.

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**6. J Clin Tuberc Other Mycobact Dis. 2025 Jul 17;40:100553. doi:**

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Retrospective analysis of systemic lupus erythematosus patients with latent

tuberculosis infection: A 5-year follow-up study.

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Patients with systemic lupus erythematosus (SLE) have a higher incidence of

tuberculosis (TB)infection compared to the general healthy population. The use

of glucocorticoids and immunosuppressive agents for SLE management further

elevates TB risk. This study aimed to evaluate the prevalence of latent

tuberculosis infection (LTBI) in SLE patients and explore risk factors for

progression to active TB (ATB) in those with concurrent SLE and LTBI. We

conducted a retrospective analysis of SLE patients treated at the Department of

Rheumatology and Immunology, Peking University Shenzhen Hospital, between 2014

and 2023. During a five - year follow - up period, LTBI was detected in 122

patients (24.11%). Of these, 11 individuals (all from the subgroup of 108

patients who did not receive tuberculosis preventive treatment [TPT]) progressed

to ATB. A comparative analysis between the 11 ATB cases and 111 non-progressing

LTBI patients revealed significant differences: ATB cases showed higher

cyclophosphamide (CTX) usage, elevated high-sensitivity C-reactive protein

(hs-CRP) levels, and less frequent hydroxychloroquine (HCQ)administration. These

findings underscore the need for regular monitoring during prolonged CTX

therapy, especially in moderate-to-high TB burden regions, and highlight the

potential protective role of HCQ.

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**7. J Biol Chem. 2025 Jul 28:110534. doi: 10.1016/j.jbc.2025.110534. Online ahead of print.**

Modeling Mycobacterium tuberculosis Pathogenesis in Lung Epithelial Organoids

Reveals Strain-Specific Host Responses and Intercellular Crosstalk.

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Emerging studies have identified alveolar epithelial cells (AECs) as a conducive

niche for Mycobacterium tuberculosis (Mtb) replication and spread during early

infection. However, the host-pathogen interactions and intercellular crosstalk

within the lung epithelial microenvironment remain inadequately understood.

Here, we developed a lung epithelial organoids co-culture model and exposed it

to the virulent H37Rv strain or the avirulent BCG strain to investigate TB

pathogenesis. Transcriptomic analyses revealed that Mtb infection markedly

alters cell death patterns in organoids and modulates signal transduction

pathways in Peripheral blood mononuclear cells (PBMCs). Western blot indicates

the H37Rv strain induced ferroptosis, autophagy, and apoptosis while suppressing

necroptosis in organoids. In contrast, BCG predominantly enhanced autophagy.

PBMCs also exhibited strain-specific responses, with BCG strongly activating the

Hippo and Notch signaling pathways, whereas H37Rv primarily engaged the TNF

signaling pathway. Furthermore, Mtb significantly reshaped the paracrine and

autocrine signaling dynamics between PBMCs and organoids. NicheNet network

analysis identified TNFSF15 and BDNF, induced by H37Rv, as key mediators.

Experimentally, overexpression of TNFSF15 and BDNF suggested that TNFSF15 from

organoids promoted BDNF expression in PBMCs via paracrine signaling. In turn,

BDNF from PBMCs then inhibited ferroptosis in organoids, contributing to

restrict Mtb growth. Overall, our study provides a conceptual framework for

understanding the mechanisms of TB pathogenesis within AECs and offers valuable

insights to prevent and control TB transmission in humans.

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**8. Mol Immunol. 2025 Jul 28;185:105-115. doi: 10.1016/j.molimm.2025.07.012. Online ahead of print.**

miR-146a-5p targets IRAK1/TRAF6 to promote bacillus Calmette-Guérin survival by

exosome-mediated autocrine actions.

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**BACKGROUND:** Exosomes carry signaling molecules between cells and play important

roles in the interaction between macrophages and Mycobacterium tuberculosis

(Mtb). This study aimed to examine the function and content of exosomes secreted

by macrophages infected with Bacillus Calmette-Guérin (BCG).

**METHODS:** THP-1 monocytes and HEK293T cells were used. Macrophages were infected

with BCG. A Transwell system was used to evaluate the effect of the exosomes

secreted by macrophages. Cells were transfected with the miR-146a-5p plasmid or

inhibitor to examine the effects of miR-146a-5p overexpression or inhibition.

qRT-PCR was employed to investigate the expression levels of miR-320a-5p,

miR-27a-5p, miR-26a-5p, miR-146a-5p, and miR-223-5p and the mRNA expression of

IL-6, TNF-α, and IL-1β. Western blot was used to investigate the protein

expression of IRAK1, TRAF6, CD63, CD81, GRP94, Alix, TSG101, P65, and p-P65. A

dual luciferase assay was performed to investigate whether miR-146a-5p targets

IRAK1 and TRAF6.

**RESULTS:** The infected cells contained high miR-146a-5p levels that could be

secreted into exosomes. Exosomal miR-146a-5p promoted Mtb survival and

proliferation after uptake by host cells. Bioinformatics showed that high

miR-146a-5p levels were found in exosomes from BCG-infected macrophages and

blood samples from patients with tuberculosis. The phagocytosis of exosomes

containing miR-146a-5p by BCG-infected macrophages suppressed the expression of

inflammatory factors by regulating the IRAK1-TRAF6-NF-κB signaling pathway,

ultimately leading to the inhibition of inflammatory factor expression in

macrophages and a decrease in the macrophage BCG killing capacity.

**CONCLUSION:** The findings indicate a new immune evasion mechanism of Mtb.

miR-146a-5p secreted in exosomes by BCG-infected macrophages can decrease the

bactericidal potential of macrophages. The results offer a novel theoretical

basis and potential biomarkers for diagnosing, treating, and managing

tuberculosis.

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**9. BMC Med. 2025 Jul 28;23(1):445. doi: 10.1186/s12916-025-04269-7.**

Global, regional, and national burden and trends of multidrug-resistant

tuberculosis and extensively drug-resistant tuberculosis in adolescents and

adults aged 15-49 years from 2010 to 2021: insights from the global burden of

disease study 2021.

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**BACKGROUND:** Multidrug-resistant tuberculosis (MDR-TB) and extensively

drug-resistant tuberculosis (XDR-TB) among those aged 15-49 years pose a severe

public health challenge, yet our understanding of the burden of these diseases

in this age group remains limited. This study aimed to evaluate the trends in

MDR-TB and XDR-TB burden among this population from 2010 to 2021 across global,

regional, and national levels.

**METHODS:** This study extracted four key indicators-incidence, prevalence, deaths,

and disability-adjusted life years (DALYs) per 100,000 population-for MDR-TB and

XDR-TB among 15-49 years from the 2021 Global Burden of Disease (GBD) study. It

assessed the burden trends using percentage change (PC) and estimated annual

percentage change (EAPC), with further analysis by age, sex, and

sociodemographic index (SDI).

**RESULTS:** In 2021, the global incidence, prevalence, deaths, and DALYs of MDR-TB

among adolescents and young adults were 241,399, 336,746, 33,285, and 1,896,002,

respectively. Global MDR-TB incidence and DALYs rates showed slight decreases

since 2010, with EAPCs of -0.76 and -2.61, respectively. In 2021, the global

incidence, prevalence, deaths, and DALYs of XDR-TB among adolescents and young

adults were 12,861, 14,039, 2442, and 133,610, respectively. Since 2010, global

XDR-TB incidence rates have increased, with an EAPC of 0.57, while prevalence

and death rates have decreased, with EAPCs of - 2.67 and - 2.87, respectively.

The incidence and prevalence rates of MDR-TB were significantly decreased since

2010 in high SDI, high-middle SDI, and low SDI regions. The prevalence rate of

XDR-TB was significantly decreased since 2010 in the high SDI and high-middle

SDI regions, while a significant increase was observed in the middle SDI,

 low-middle SDI, and low SDI regions. Furthermore, a gradual decline was

observed in the burden of MDR-TB and XDR-TB as the SDI level increases. The

burden of MDR-TB and XDR-TB showed an upward trend during the COVID-19 epidemic.

**CONCLUSIONS:** The burden of MDR-TB and XDR-TB among adolescents and young adults

remained very severe, particularly in the middle SDI and low-middle SDI regions.

The COVID-19 pandemic may impact the global burden of these drug-resistant

tuberculosis. Targeted interventions are crucial to address this issue.

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**10. Eur J Med Chem. 2025 Jul 26;298:118010. doi: 10.1016/j.ejmech.2025.118010.**

**Online ahead of print.**

Discovery of novel Cytochrome bd oxidase inhibitors against Mycobacterium

tuberculosis.

Wu X(1), Zhang X(2), Xia W(3), Zhang Y(2), Huang K(1), Zhao F(3), Ji C(1), Wang

J(4), Zhou B(5), Zhang JZH(6).

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Drug-resistant Mycobacterium tuberculosis remains a major challenge to effective

tuberculosis (TB) control. Cytochrome bd oxidase (Cyt-bd), a critical terminal

oxidase within the electron transport chain of M. tuberculosis, maintains

bacterial survival under respiratory stress but remains an underexplored

therapeutic target. Here, we developed an integrated computational and

experimental workflow to identify novel Cyt-bd inhibitors. Among the candidates,

TB25, TB25-2, and TB25-14 exhibited substantial antimycobacterial efficacy, each

reducing intracellular M. tuberculosis survival in macrophages by over 94 %.

Importantly, co-treatment with Q203, a cytochrome bcc oxidase (Cyt-bcc)

inhibitor, resulted in pronounced synergistic bactericidal effects. These

findings highlight the potential of dual Cyt-bd/Cyt-bcc inhibition as a new

strategy for treating drug-resistant and latent TB, and validate the

effectiveness of our virtual screening pipeline for discovering new anti-TB

agents.

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**11. Microorganisms. 2025 Jun 29;13(7):1524. doi: 10.3390/microorganisms13071524.**

Tuberculosis Patients' Serum Extracellular Vesicles Induce Relevant Immune

Responses for Initial Defense Against BCG in Mice.

Xu W(1), Hou Y(1), Zhang J(1), Cao T(1), Dai G(1), Wang W(1), Tian N(1), Liu

D(1), Chu H(1), Sun H(1), Sun Z(1).

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Extracellular vesicles (EVs) can be distributed in various bodily fluids, such

as serum and urine, and play an essential role in immune regulation, substance

transport, and other aspects. Tuberculosis (TB) is an infectious disease caused

by Mycobacterium tuberculosis (Mtb), which places a tremendous burden on public

health prevention and control within society. Researchers are committed to

developing various diagnoses and treatment plans to eliminate TB effectively.

The results of some studies conducted to date demonstrate that the serum EVs of

TB patients, which carry components related to Mtb, can be used as relevant

markers for TB detection and improve diagnostic efficiency. However, no relevant

reports exist on the particular physiological functions such EVs perform, thus

warranting further exploration. In this study, we collected serum EVs from both

healthy individuals and TB patients. After identifying the morphology,

concentration, and expression of classic markers (CD63, CD81, and CD9) of EVs,

we explored their physiological functions at the cellular level and their

physiological functions and effects on BCG colonization in the lungs at the

mouse level. It was found that EVs were abundant in TB patients and healthy

individuals, and the number of CD63 and CD9 markers co-expressed on the surface

of serum EVs in healthy individuals was greater than that in TB patients. Serum

EVs in patients with TB can stimulate cells to secrete more immune cytokines,

such as TNF-α and IL-6, compared with those in healthy individuals; induce an

increase in the M1/M2 ratio of macrophages in the peripheral blood mononuclear

cells of mice; and inhibit the colonization of Mycobacterium bovis bacillus

Calmette Guérin (BCG) in the lungs of mice. In addition, they can inhibit the

occurrence of inflammatory responses in the lung tissue of mice. The above

results suggest that serum EVs in TB patients may exert their physiological

function by regulating immune responses. This finding also indicates that

exploring serum EVs in TB patients with regard to their physiological functions

shows excellent potential.

DOI: 10.3390/microorganisms13071524

PMCID: PMC12298580

PMID: 40732033

**12. J Microbiol Methods. 2025 Jul 26;236:107202. doi: 10.1016/j.mimet.2025.107202.**

**Online ahead of print.**

Diagnostic potential of recombinant Mycobacterium tuberculosis PcaA antigen and

its enhancement of protective efficacy as a subunit vaccine booster following

BCG priming.

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Bacillus Calmette-Guérin (BCG) is the only vaccine currently used in clinical

practice to prevent tuberculosis (TB), however, it remains limited in preventing

latent infection, TB reactivation, and providing comprehensive protection. In

this study, the pcaA gene, an antigen associated with persistent infection, was

selected, and the recombinant plasmid pET28a-PcaA was successfully constructed

for protein expression and purification. The specificity of the antigen was

further verified using chemiluminescence, enzyme-linked immunosorbent assay

(ELISA), flow cytometry, and mycobacterial growth inhibition assay (MGIA).

Significant differences were observed in the expression levels of IFN-γ, IL-2,

IL-8, and IgG in the peripheral blood of patients with Mycobacterium

tuberculosis (M. tb) following stimulation with the PcaA antigen in the Active

Tuberculosis (ATB) and Latent Tuberculosis Infection (LTBI) groups. An IL-8

combined diagnostic model could effectively distinguish between ATB and LTBI,

while anti-PcaA IgG demonstrated strong performance in ruling out M. tb

infection. Recombinant PcaA protein (rPcaA) was formulated with liposome

dimethyl dioctadecylammonium bromide (DDA) / colloidal manganese salt (MnJ)/DM

to immunize mice. Serum-specific antibody levels, cytokines secreted by

splenocytes, and the number of multifunctional T cells in splenocytes were

assessed. The results indicated that the BCG + rPcaA-DM vaccine group exhibited

significantly elevated levels of Th1-type cytokines, antibody titers, and the

frequencies of IFN-γ+/TNF-α+ single and double-positive CD4+ and CD8+ T cells

compared to the BCG group. Furthermore, splenocytes and lung cells from

immunized mice significantly inhibited mycobacterial growth. These findings

suggest that the rPcaA-DM vaccine, as a BCG booster, significantly enhances Th1

polarization and provides robust protective efficacy, with potential to prevent

LTBI progression to ATB.

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PMID: 40720996

**13. Immunotargets Ther. 2025 Jul 22;14:773-786. doi: 10.2147/ITT.S518628.**

**eCollection 2025.**

Mitophagy: A Potential Therapeutic Target for Tuberculosis Immunotherapy.

Gao S(#)(1), Yang Z(#)(1), Yu J(2), Zhang F(1)(3), Tang S(4), Pang Y(1).

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Mitophagy serves as a cytoprotective mechanism that is essential for eliminating

dysfunctional or superfluous mitochondria, thereby fine-tuning mitochondrial

quantity and maintaining cellular homeostasis. Recent studies underscore the

critical role of mitophagy in determining the fate and function of host cells

infected by Mycobacterium tuberculosis. The successful pathogen strategically

integrates into the host's mitochondrial network, manipulating processes such as

apoptosis, metabolic reprogramming, mitochondrial fusion and fission, and

reactive oxygen species production. Therefore, understanding those mechanisms is

critical for the advancements of host-directed therapies against tuberculosis.

This study offers a comprehensive overview of the interplay between

Mycobacterium tuberculosis and mitophagy, emphasizing the associated signaling

pathways and potential therapeutic targets involved in mitophagy in

Mycobacterium tuberculosis infection. Activating mitophagy in infected host

cells represents a promising avenue for improving therapeutic outcomes against

tuberculosis. This review aims to summarize potential research direction for

agents targeting induction of mitophagy. Notably, evidence suggests that

BNIP3/NIX-mediated mitophagy may serve as a potential therapeutic target.

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PMCID: PMC12301142

PMID: 40727489

**14. Genes (Basel). 2025 Jul 17;16(7):832. doi: 10.3390/genes16070832.**

Analysis of Key miRNA/mRNA Functional Axes During Host Dendritic Cell Immune

Response to Mycobacterium tuberculosis Based on GEO Datasets.

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**BACKGROUND:** Dendritic cells (DCs) play an important role as a bridge between

innate and adaptive immunity, and changes in gene expression of DCs during the

immune response to Mycobacterium tuberculosis (M.tb) may affect the development

of tuberculosis.

**METHODS:** Using systems biology methods, mRNA and miRNA expression profile data

of DCs infected with M.tb were obtained. A total of 1398 differentially

expressed mRNAs and 79 differentially expressed miRNAs were identified, and a

corresponding miRNA-mRNA regulatory network was constructed using Cytoscape

3.9.1 software. The functional annotations and pathway classifications of the

miRNA-mRNA network were identified using the DAVID tool. Then, the key pathway

modules in the miRNA-mRNA network were screened and subjected to PPI network

analysis to identify hub nodes. Subsequently the miRNA/mRNA axis was determined,

validated by qRT-PCR, and evaluated through ROC curve analysis.

**RESULTS:** The TNF signaling pathway and the Tuberculosis pathway were key pathway

modules, with miR-34a-3p/TNF and miR-190a-3p/IL1B being the greatest

correlations with the two pathway modules. qRT-PCR results showed that IL1B and

miR-190a-3p exhibited significant differences in both the H37Ra and BCG

infection groups. The AUC of two factors (IL1B and miR-190a-3p) was 0.9561 and

0.9625, respectively, showing high sensitivity and specificity.

**CONCLUSIONS:** Consequently, miR-190a-3p/IL1B might be a good candidate marker to

characterize the immune response of DCs to M.tb and a transition signal from

innate to adaptive immunity.

DOI: 10.3390/genes16070832

PMCID: PMC12294207

PMID: 40725488 [Indexed for MEDLINE]

**15. Phytother Res. 2025 Jul 27. doi: 10.1002/ptr.70042. Online ahead of print.**

Salvianolic Acid B Inhibits ZBP1-Mediated PANoptosis in Mycobacterium

tuberculosis-Infected Macrophages by Targeting TNFR1.

Shen J(1)(2), Fu Y(1), Liu F(1), Wu J(1), Zhang H(1), Sun J(1), Miao Z(1), Jiang

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The increasing rates of drug resistance in Mycobacterium tuberculosis (Mtb) have

made controlling tuberculosis more challenging. Excessive programmed cell death

helps mediate Mtb transmission. Salvianolic acid B (Sal B), a water-soluble

extract of Salvia miltiorrhiza, has been reported to inhibit programmed cell

death and excessive inflammation. This study aimed to investigate the potential

inhibitory mechanism of Sal B on PANoptosis. The inhibitory effect of Sal B on

PANoptosis was evaluated by western blotting, ELISA, and other techniques in an

in vitro model of Mtb H37Ra-infected macrophages. The roles of ZBP1 and TNFR1 in

PANoptosis were explored by small interfering RNA transfection. In addition, the

inhibitory effect of Sal B on PANoptosis and the hyperinflammatory response was

verified by western blotting, hematoxylin and eosin staining, and

immunohistochemistry in an in vivo model of inflammatory injury in the lungs of

LPS-infected mice. Sal B inhibited the protein levels of key molecules of

Mtb-mediated PANoptosis and hindered the assembly of the PANoptosome consisting

of ASC, ZBP1, RIPK1, RIPK3, and Caspase 8. Sal B may further inhibit PANoptosis

by binding to TNFR1 and suppressing ZBP1 levels. In addition, the results of

in vivo studies verified that Sal B could ameliorate LPS-induced pathological

injury in mouse lung tissues. Sal B can target TNFR1 to achieve a regulatory

effect on macrophage PANoptosis. This provides new ideas for Sal B as a

host-directed therapy drug to attenuate the excessive inflammatory response

induced by Mtb infection.

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PMID: 40717039

**16. Infect Drug Resist. 2025 Jul 23;18:3661-3670. doi: 10.2147/IDR.S524986.**

**eCollection 2025.**

Diagnostic Utility of Nanopore Sequencing for Tuberculous Serous Effusions.

Chen Y(#)(1), Ling Y(#)(2), Xu X(3), Shen Y(4), Xu K(3), Yu G(3).

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**OBJECTIVE:** Early and precise diagnosis of tuberculous serous effusions is a huge

challenge. Nanopore sequencing is a potentially efficient assay. The objective

of the current study was to evaluate the diagnostic accuracy of nanopore

sequencing for tuberculous serous effusions using clinical specimens directly,

and to provide a new pathway for the early and precise diagnosis of tuberculous

serous effusions.

**METHODS:** This was a retrospective analysis of the effectiveness of nanopore

sequencing as a diagnostic method for tuberculous serous effusions using

clinical specimens (pleural fluid, pericardial effusion, and ascitic fluid).

Using clinical diagnosis as reference standard, the diagnostic accuracy

indicators such as sensitivity, specificity, positive predictive value (PPV),

negative predictive value (NPV), and area under the curve (AUC) for the tests in

question were evaluated.

**RESULTS:** In total, 132 patients were eligible for inclusion. Nanopore sequencing

showed sensitivity of 93.3%, specificity of 85.2%, PPV of 96.1%, NPV of 76.7%,

and AUC of 0.89 for tuberculous serous effusions. The diagnostic accuracy of

nanopore sequencing was significantly superior than that of Xpert MTB/RIF and

culture. Similar results were observed in different types of tuberculous serous

effusions (pleural tuberculosis, pericardial tuberculosis, and peritoneal

tuberculosis).

**CONCLUSION:** Nanopore sequencing was efficient for the rapid diagnosis of

tuberculous serous effusions and had a very positive effect. For paucibacillary

tuberculous serous effusions, nanopore sequencing might become an effective

method for detecting pathogenic bacteria.

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PMCID: PMC12296698

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**17. Front Med (Lausanne). 2025 Jul 22;12:1632764. doi: 10.3389/fmed.2025.1632764.**

**eCollection 2025.**

Renal epidermoid cyst mimicking renal tuberculous abscess: a case report.

Gao J(1), Luo H(1), Zhu H(1), Liu Z(1), Li M(1), Chen Y(1), Wang S(1), Zhou

C(1), Li Z(1), Liang G(1), Chen S(1).

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Renal epidermoid cysts (RECs) are exceedingly rare benign cystic lesions, with

only 15 histologically confirmed cases reported worldwide to date. Due to their

non-specific clinical and radiological features, they are often misdiagnosed

preoperatively as infectious or neoplastic conditions. Here, we report a

25-year-old man in whom a complex renal cyst was incidentally identified during

a routine health examination. Retrospectively, the patient reported mild urinary

frequency and low-grade fever. Imaging suggested a non-enhancing heterogeneous

cyst in the lower pole of the right kidney. Laparoscopic partial nephrectomy was

performed, revealing abundant yellow-white caseating material intraoperatively,

prompting empirical anti-tuberculosis therapy in the context of regional

endemicity. However, histopathological analysis confirmed a diagnosis of RECs,

and anti-tuberculous treatment was subsequently withdrawn. On postoperative day

5, the patient developed gross hematuria due to a renal artery pseudoaneurysm,

which was successfully managed with selective arterial embolization. This case

highlights the diagnostic challenges posed by atypical cystic renal lesions and

underscores the importance of integrating imaging, intraoperative findings, and

histopathology. Including RECs in the differential diagnosis may prevent

unnecessary antituberculous therapy and overtreatment.

Copyright © 2025 Gao, Luo, Zhu, Liu, Li, Chen, Wang, Zhou, Li, Liang and Chen.

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PMCID: PMC12321514

PMID: 40766064

**18. Front Public Health. 2025 Jul 22;13:1588196. doi: 10.3389/fpubh.2025.1588196.**

**eCollection 2025.**

Prediction of risk factors associated with the development of

multidrug-resistant tuberculosis in patients with tuberculosis.

Chen H(1)(2), Tong Z(1), Zhong J(1), Tong Y(1), Zeng Q(1), Shen B(1), Song Q(1),

Qian F(2)(3), Xiao X(3).

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**OBJECTIVE:** This study aimed to develop and validate a reliable nomogram based on

clinical factors to predict development of multidrug-resistant tuberculosis

(MDR-TB) associated with individuals with tuberculosis (TB), so as to reduce the

harm and economic burden caused by disease progression.

**METHODS:** The study included 4,251 individuals with TB who received treatment at

Huzhou Central Hospital between January 2016 and December 2023, excluding 87

individuals because of infection with non-TB mycobacterium or incomplete

information (including missing laboratory or clinical data). A total of 4,164

individuals (2,261 sputum smear-positive and 1,903 sputum smear-negative

patients) were ultimately included in the analysis. This analysis incorporated

clinical baseline presentations, demographic information, imaging findings,

laboratory results, and clinical diagnoses to develop a predictive model for

MDR-TB.

**RESULTS**: This study demonstrated that sex, age, a history of anti-TB therapy,

body mass index (BMI) ≤ 18.5, smoking history, occupation, previously diagnosed

TB, pulmonary cavitation, comorbidities, poverty, and C-reactive protein

(CRP) ≥ 37.3 mg/L were major risk factors for MDR-TB in patients with TB. The

area under the receiver operating characteristic (ROC) curve was 0.902 for the

training group and 0.909 for the validation group. Calibration curve analysis

revealed that the predicted and actual incidence rates of MDR-TB in the two

groups were in good agreement, whereas decision curve analysis (DCA) indicated

that the predictive model resulted in better clinical benefit.

**CONCLUSION:** The nomogram model established in this study included 11 clinical

characteristics and demographic features of patients with TB, which may be

valuable for predicting MDR-TB.

Copyright © 2025 Chen, Tong, Zhong, Tong, Zeng, Shen, Song, Qian and Xiao.

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PMCID: PMC12321860

PMID: 40766036 [Indexed for MEDLINE]

**19. Int J Gen Med. 2025 Jul 30;18:4119-4129. doi: 10.2147/IJGM.S527840. eCollection 2025.**

Construction and Validation of a Nomogram-Based Predictive Model for Acute

Kidney Injury Caused by Drug Resistance to Tuberculosis.

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**OBJECTIVE:** Acute kidney injury (AKI) is a common and serious adverse effect

during tuberculosis (TB) treatment in clinical settings, particularly in

patients with drug-resistant TB. AKI may lead to treatment interruption and poor

prognosis. Early identification of patients at high risk for AKI is crucial to

improve clinical outcomes.

**METHODS:** We retrospectively enrolled 571 TB patients, divided into training and

validation cohorts. LASSO and multivariate logistic regression were used to

identify risk factors, and the nomogram was evaluated using AUC, calibration,

and decision curve analysis (DCA).

**RESULTS:** This study included 571 patients with TB. In this study, five variables

(age, hypertension, diabetes, Scr, and ALB) were included to construct a

nomogram for predicting AKI caused by drug resistance to TB. The AUC of the

training set and validation set were 0.809 (95% CI: 0.7480-0.871, P < 0.001) and

0.841 (95% CI: 0.765-0.918, P < 0.001), respectively, indicating that the

prediction model had good discriminative performance. The calibration curve

shows that the predicted values of the model are basically consistent with the

actual values, indicating good performance. DCA suggests that almost all ranges

of TB patients can benefit from this new predictive model, indicating good

clinical utility.

**CONCLUSION:** The nomogram model of AKI caused by drug resistance to TB

established in this study has good predictive value and helps identify high-risk

populations.

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PMCID: PMC12320139

PMID: 40761921

**20. Front Microbiol. 2025 Jul 21;16:1622017. doi: 10.3389/fmicb.2025.1622017.**

**eCollection 2025.**

Lycium barbarum and Lactobacillus acidophilus synergistically protect against

anti-tuberculosis drug-induced male reproductive injury via gut

microbiota-independent pathways in mice.

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**BACKGROUND:** As first-line anti-tuberculosis drugs, rifampicin (RIF) and

isoniazid (INH) are associated with reproductive impairment during their use,

accompanied by sustained dysbiosis of the gut microbiota (GM). Lycium barbarum

(Wolfberry), a substance that can be used both as medicine and food, is often

used in traditional Chinese medicine to treat male reproductive-related

diseases. However, the potential of wolfberry to mitigate reproductive injury

induced by anti-tuberculosis (anti-TB) drugs via modulation of the GM has not

been reported. This study aimed to explore the protective effect and mechanism

of wolfberry on the reproductive injury of male mice induced by anti-TB drugs.

**METHODS:** Forty male Kunming mice were randomly assigned to normal, model,

wolfberry, and levocarnitine groups (n = 10/group). The normal group received a

daily gavage of ultrapure water, while the other three groups were administered

ultrapure water, wolfberry decoction, and levocarnitine, respectively, via

gavage 3 h prior to the daily administration of RIF and INH for 21 days. Another

40 mice were rendered pseudo-germ-free via oral administration of antibiotic

(ATB) water for 1 week, then divided into ATB, ATB + Wolfberry,

ATB + Lactobacillus acidophilus (L. acidophilus), and ATB + Wolfberry+L.

acidophilus groups. Prior to the administration of RIF and INH by gavage, the

mice were administered ultrapure water, wolfberry decoction, L. acidophilus, or

a combination of wolfberry and L. acidophilus via gavage for 21 consecutive

days. Afterwards, sperm motility, count, and serum follicle-stimulating hormone

(FSH), luteinizing hormone (LH), and testosterone (T) levels were evaluated. Gut

contents were collected for 16S rRNA sequencing and real-time PCR, and

testicular tissues were subjected to pathological and transcriptomic analyses.

**RESULTS:** Wolfberry improved sperm quality in mice with reproductive injury

induced by anti-TB drugs. Specifically, wolfberry increased sperm count and

motility, alleviated testicular pathological damage, and regulated the levels of

sex hormones, including FSH, LH, and T. Besides, wolfberry restored intestinal

barrier function, enhanced the abundance of L. acidophilus in the gut, and

modulated key processes involved in spermatid differentiation, sperm

development, and the meiotic cell cycle. Notably, the combination of wolfberry

and L. acidophilus yielded the most significant protective effects against

reproductive injury induced by anti-TB drugs.

**CONCLUSION:** Our findings suggest that wolfberry protects against reproductive

injury induced by anti-TB drugs, partially mediated through modulation of the

GM, though this effect is not entirely dependent on the microbiota. Importantly,

wolfberry and L. acidophilus play a synergistic role in protecting against the

reproductive injury induced by anti-TB drugs.

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**eCollection 2025.**

Development and validation of a prediction model based on a nomogram for

tuberculous pleural effusion.

Liu S(#)(1)(2), Yang Y(#)(1)(2), Wang D(#)(3), Gao L(1)(2), Qin J(1)(2), Wu

Y(1)(2), Li D(1)(2), Li X(4), Chen M(5), Wang H(1)(2), Shen Y(1)(2), Wen

F(1)(2), Chen F(6).

**Suli Liu, Yao Yang, Dongmei Wang, Lijuan Gao, Jiangyue Qin, Yanqiu Wu, Diandian Li, Xiaohua Li, Mei Chen, Hao Wang\*, Yongchun Shen\*, Fuqiang Wen, Fangying Chen**

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**BACKGROUND:** Diagnosing tuberculous pleural effusion (TPE) is challenging. There

is a lack of cross-sectional lateral comparisons among TPE prediction models.

**OBJECTIVES:** We aimed to develop and validate a novel TPE prediction model and

compare its diagnostic performance with that of existing models.

**METHODS:** Patients with pleural effusion were included in the training, testing,

and external validation sets. Variable selection strategies included LASSO and

logistic regression. The discriminability, calibration, and clinical efficacy of

the prediction model were estimated in the three sets. The performance of the

model was compared with that of two existing prediction models.

**RESULTS:** Fever, tuberculosis interferon-gamma release assays, pleural adenosine

deaminase, the pleural mononuclear cell ratio, the ratio of pleural lactate

dehydrogenase to pleural adenosine deaminase, pleural carcinoembryonic antigen,

and pleural cytokeratin 19 fragment were selected to establish the prediction

model. The AUCs were 0.931 (0.903-0.958), 0.856 (0.753-0.959), and 0.925

(0.867-0.984) in the training, testing, and external validation sets,

respectively. The AUCs of the two existing prediction models were 0.793

(0.737-0.850) and 0.854 (0.816-0.892). The calibration curves revealed that this

model had good consistency. Decision curve analysis revealed the acceptable

clinical benefit of this model.

**CONCLUSION:** Compared with the existing models, the TPE prediction model

developed in this study demonstrated good diagnostic performance.

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The exacerbating effects of stable pulmonary tuberculosis on the deterioration

of inflammatory response, coagulation function, and pulmonary function in COPD:

A propensity score-matched retrospective study.

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Chronic obstructive pulmonary disease (COPD) and tuberculosis pose significant

public health challenges, particularly in tuberculosis-endemic developing

regions where their co-prevalence may exacerbate the disease burden of chronic

airflow obstruction. This study aimed to investigate the impact of stable

pulmonary tuberculosis on inflammatory status, coagulation function, and

pulmonary function in COPD patients during acute exacerbations.We conducted a

retrospective analysis of 68 COPD patients with acute exacerbation and stable

pulmonary tuberculosis (observation group) admitted between December 2019 and

December 2023. Using propensity score matching based on age and gender, we

selected 68 COPD patients without stable pulmonary tuberculosis as the control

group. Comparative analysis of laboratory tests and pulmonary function

parameters revealed that the observation group had significantly elevated levels

of erythrocyte sedimentation rate (ESR), interleukin-6 (IL-6), lymphocyte count,

platelet count (PLT), and D-dimer, along with significantly reduced pulmonary

function parameters including forced expiratory volume in 1 s/forced vital

capacity (FEV1/FVC) and forced expiratory flow between 25 %-75 % of vital

capacity (FEF25-FEF75) (all P < 0.05). Correlation analysis demonstrated

positive associations between ESR/IL-6 levels and D-dimer, PLT, and fibrin(ogen)

degradation product (FDP) levels (P < 0.05).Binary logistic regression analysis

of 14 significantly different variables identified IL-6 (OR = 1.056), ESR

(OR = 1.022), PLT (OR = 1.005), D-Dimer (OR = 1.002), FEV1/FVC (OR = 0.962), FEF50 (OR = 0.534), and FEF75 (OR = 0.089) as independent factors associated with acute exacerbation in COPD patients with stable pulmonary tuberculosis (all P < 0.05). Our findings indicate that elevated IL-6, ESR, PLT, and D-Dimer levels coupled with decreased FEV1/FVC, FEF50, and FEF75 levels represent distinctive clinical characteristics of these patients.This study demonstrates that COPD patients with stable pulmonary tuberculosis exhibit enhanced inflammatory responses, prothrombotic tendencies, and more severe pulmonary function impairment, providing a scientific basis for developing individualized treatment strategies for this patient population.

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Clinical characteristics of pulmonary sarcoidosis in China: a retrospective,

multicenter study.

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**BACKGROUND: P**atients with pulmonary sarcoidosis or intrathoracic lymph node

tuberculosis (TB) may present with comparable clinical manifestations that pose

challenges in differentiation. This study aims to improve the diagnostic

accuracy of pulmonary sarcoidosis.

**METHODS:** A retrospective analysis of patients diagnosed with pulmonary

sarcoidosis or intrathoracic lymph node TB within the past decade at four

tertiary hospitals in China was conducted. According to the inclusion and

exclusion criteria, a total of 968 patients were ultimately enrolled in the

study, comprising 477 individuals diagnosed with pulmonary sarcoidosis and 491

individuals diagnosed with intrathoracic lymph node TB. The analysis focused on

general information, clinical manifestations, and auxiliary examination results,

with a comparative analysis between the two groups.

**RESULTS:** The median age of onset for pulmonary sarcoidosis was 50 years, with

females accounting for 68.94% of the patients. Common symptoms of pulmonary

sarcoidosis included cough, sputum production, dyspnea, and chest pain, while

approximately 34.12% of patients were asymptomatic. Fever, fatigue, and night

sweats occurred less frequently in pulmonary sarcoidosis patients than in those

with intrathoracic lymph node TB. Uveitis and myocardial sarcoidosis were

observed exclusively in pulmonary sarcoidosis patients. The median time from

symptom onset to the diagnosis of pulmonary sarcoidosis was up to three months.

Approximately 47.29% of pulmonary sarcoidosis patients had reduced peripheral

blood lymphocyte counts, and 94.12% exhibited symmetric enlargement of hilar

lymph nodes on chest CT. Both pulmonary sarcoidosis and intrathoracic lymph node

TB showed granulomatous inflammation, with 64.36% of intrathoracic lymph node TB

cases presenting necrotic foci. Bronchoscopy was the primary method for biopsy,

and only 11.06% of pulmonary sarcoidosis patients had multiple nodules in the

tracheal or bronchial mucosa, with a low positivity rate for pathogen tests.

**CONCLUSION:** Pulmonary sarcoidosis predominantly affects middle-aged and young

women and can be differentiated from intrathoracic lymph node TB by the presence

of uveitis and myocardial sarcoidosis, although these manifestations are rare. A

significant proportion of pulmonary sarcoidosis patients experience a reduction

in their peripheral blood lymphocyte count. Chest CT scans often reveal

symmetric bilateral enlargement of hilar lymph nodes, and in some cases,

multiple nodules in the tracheal or bronchial mucosa. Both pulmonary sarcoidosis

and intrathoracic lymph node TB show granulomatous inflammation, but

tuberculosis lesions are more likely to necrose.

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Rapid Diagnosis of Mycobacterium tuberculosis Infection and Drug Resistance

Based on Real-Time Fluorescence PCR.

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Tuberculosis (TB) is a major health concern that disproportionately affects

vulnerable populations. The emergence and spread of drug-resistant TB pose a

serious threat to global public health. Traditional diagnostic methods for

Mycobacterium tuberculosis (MTB), such as smear microscopy and culture, have

significant limitations and often result in delayed clinical treatment. To

address this challenge, an efficient diagnostic scheme based on real-time

fluorescent PCR was developed. High-quality MTB nucleic acids can be extracted

from test samples, and resistance status can be identified at key resistance

sites. Compared to traditional methods, this approach significantly reduces

detection time to just a few hours, enabling the determination of infection and

resistance status shortly after a patient's initial visit. Detection sensitivity

exceeds 85%, and specificity reaches 95%. This non-invasive technology not only

minimizes patient discomfort but also supports accurate diagnosis in grassroots

medical institutions due to its low cost and high efficiency. Overall, the

approach represents a significant improvement in TB diagnostics.

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