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Review Article

# Critical review on analytical detection of first line and second line anti tubercular agent by various modern analytical method

**TS Basuri[1]*, Ranjit Mohapatra[2]**

[1] *Mayurbhanj Medical Academy, Baripada, Odisha, India*
[2] *Utkal University, Bhubaneswar, Odisha, India*

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ABSTRACT

Tuberculosis is a chronic inflammatory, granulomatous bacterial infectious disease caused by Mycobacterium tuberculosis. About 10 million people worldwide are ill with MTB in 2018, 5.7 million are males, 3.2 million are females and 1.1 million are infants, 1.6 million died from the disease. This article includes epidemiology, classification, pathogenesis, diagnosis and treatment of Tuberculosis. It includes the drug profile of antitubercular agents such as isoniazid, pyrazinamide, ethambutol, rifampicin, paraminosalicylic acid, thiacetazone, ethionamide, kanamycin, amikacin. cycloserine, viomycin, morphazinamide and some newer drugs such as ofloxacin, ciprofloxacin, clarithromycin and rifabutin. It contains analytical detection methods of antitubercular agents by HPLC.

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## 1. Introduction

*Tuberculosis (TB):* Tuberculosis is a chronic granulomatous inflammatory infectious bacterial disease caused by Mycobacterium tuberculosis, which is most commonly affect the lungs. [1]

### *1.1. Epidemiology of TB*

MTB has very ancient origins: it has lasted more than 70,000 years and actually infects almost 2 billion people worldwide in 2016, with about 10.4 million new cases of TB annually, about 33.33% of the world's population carries TB bacillus and is at risk of developing active disease. [2]

Approximately 10 million people worldwide are infected with MTB in 2018, 5.7 million men, 3.2 million women and 1.1 million girls, 1.6 million people expired from the disease currently available for treating TB, it remains incompetent, taking 6 to 9 months to cure the drug-susceptible variant and up to 2.5 years to cure MDR-TB. [3]

### *1.2. Classification of Tuberculosis (TB 4:)*

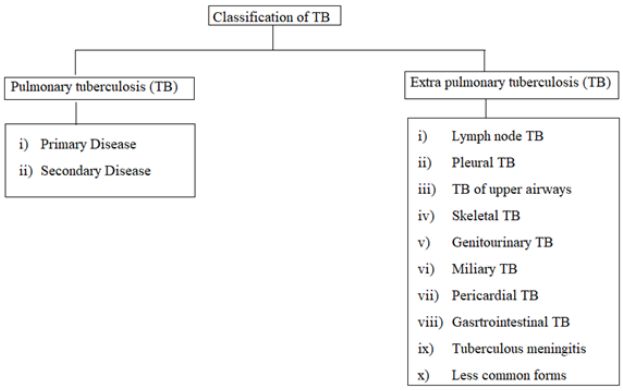


**Figure 1:** Classification of tuberculosis

* *Corresponding author*.
*E-mail address*: tbasuri@gmail.com (T. S. Basuri).

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### 1.3. TB Pathogenesis

1. TB is an infectious disease caused by M. Tuberculosis usually affects the lungs.[4]
2. M. Tuberculosis is transmitted to atmosphere as airborne droplets, owing to runny noses of individuals with pulmonary tuberculosis (TB). Transmission done due to breathing of nuclei of these droplets which goes from the nasal cavities to upper respiratory tract, bronchi and then lastly goes to the alveoli of lungs.[5]
3. First on M. Tuberculosis enters the alveoli and is swallowed by alveolar macrophages that cause a large ratio of inhaled tuberculosis bacilli to be destroyed.[6]
4. The minor unchanged ratio reproduces inside the macrophages and is liberated upon death of the macrophages.[7]
5. Approximately within 2 to 8 weeks,[8] an immune defensive mechanism is activated which permits leukocytes to extinguish major proportion of the tubercle bacilli. The encapsulation by the leukocytes leads to the barrier formation around the tubercle bacilli developing a granuloma.[7]
6. Once if it goes inside the hindrance shell, the tubercular bacilli are to be under control and it establish a stage of latent tuberculosis infection (LTBI). Persons will not give any indications of TB and infection are unable to spread.[9]
7. On other hand, if the defensive mechanism is unable to keep the tubercular bacilli under control, then bacilli are quickly reproduced which leads to a development from latent tuberculosis infection to tuberculosis TB.[10]

### 1.4. Diagnosis of TB[11,12]

1. The medical history of patients
2. Physical test
3. M. Tuberculosis test
4. Chest X-ray
5. Bacteriologic examination

### 1.5. Treatment of tuberculosis:[13,14]

*Anti-tubercular agent:* These are the medicines which are used for the tuberculosis treatment.

Drug profile of antitubercular agentsTable 1
*Analytical Detection of Antitubercular Agents*
*First line antitubercular drugs*Table 1
*Second line antitubercular drugs:*Table 2
New antitubercular drugsTable 4

## 2. Conclusion

Tuberculosis is a prolonged bacterial infection caused by Mycobacterium tuberculosis, characterised by the development of granulomas in infected tissues and by hypersensitivity mediated by the cells. About 10 million people worldwide became contaminated with MTB in 2018, 5.7 million men, 3.2 million women and 1.1 million kids, 1.6 million die from illness. This article includes epidemiology, classification, pathogenesis, diagnosis and treatment of Tuberculosis. It includes the drug profile of antitubercular agents such as isoniazid, pyrazinamide, ethambutol, rifampicin, paraminosalicylic acid, thiacetazone, ethiomnamide, kanamycin, amikacin. cycloserine, viomycin, morphazinamide and fewer new mwdicines include ofloxacin, ciprofloxacin, clarithromycin and rifabutin. It contains analytical detection method of

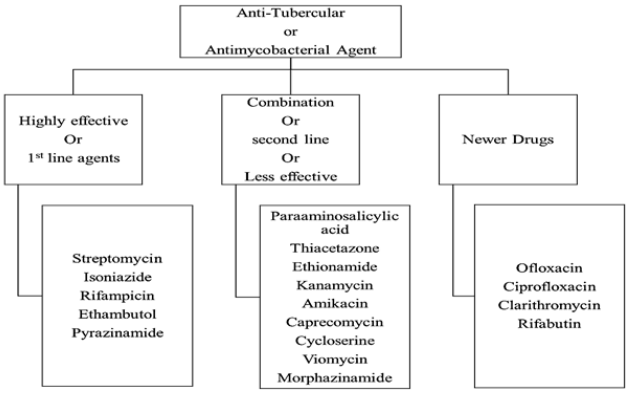


**Figure 2:** Class of TB drugs

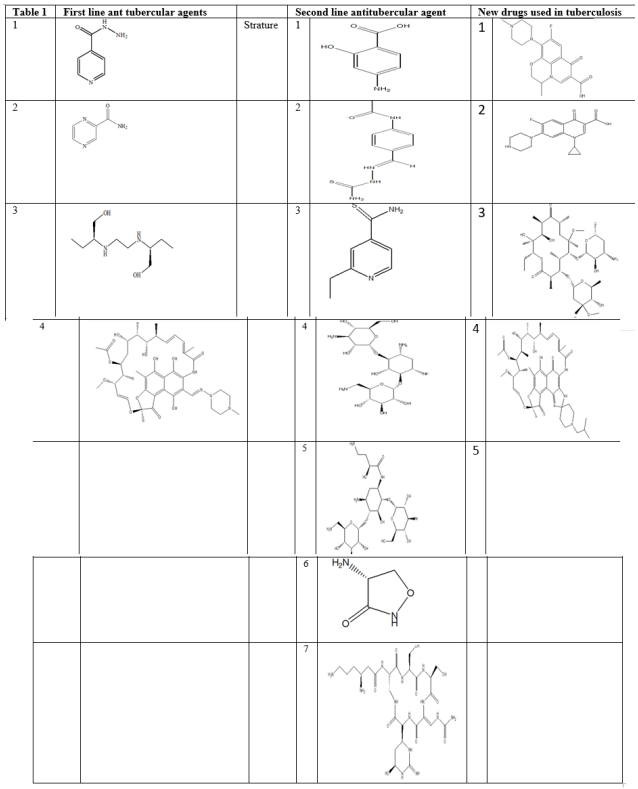


Diagram 1: Table 1+ Structure

**Table 1:** Drug description of antitubercular agent

| Sr. no. | Drug | Mechanism of action |
|---|---|---|
| 1. | | |
| i) | Isoniazide [15,16] | Isoniazid interferes with the cell wall creation by inhibiting the synthesis of mycolic acid. |
| ii) | Pyrazinamide [17,18] | Pyrazinamide is a synthetic pyrazinoic acid amide derivative having bactericidal property. Pyrazinamide is mainly dynamic against gradually multiplying intracellular bacilli by an unfamiliar mechanism of action. Its bactericidal action is based on the bacterial pyrazinamidase, which eliminates the amide group to generate active pyrazinoic acid. |
| iii) | Ethambutol [19–21] | Ethambutol prevent the transfer of mycolic acid in M. tuberculosis cell wall. Which leads to the weakening of cell wall resulting into cell death. |
| iv) | Rifampicin [22,23] | Rifampicin works by blocking DNA dependent RNA polymerase, which results in depletion of synthesis of RNA and death of cell. |
| 2. | | |
| i) | Paraminosalicylic acid [24] | Aminosalicylic acid works by two route. $1^{st}$- It inhibit folic acid production. Aminosalicylic acid bind to pteridine synthetase enzyme which is use for synthesis of folic acid. As bacteria is not able to use exterior source of folic acid leads to slow down the multiplication and growth of cell. $2^{nd}$ - It may hinder the production of the cell wall constituent, mycobactin, which leads to dropping iron uptake by M. tuberculosis. |
| ii) | Thiacetazone [25] | It is a bacteriostatic agent. Exact mechanism of thiacetazone is unknown. It can inhibit the synthesis of mycolic acid in mycobacterium tuberculosis leading to cell wall weakening which results in cell death. |
| iii) | Ethionamide [26] | Ethionamide is a derivative of nicotinamide. Actual mechanism of ethionamide is mysterious. It might prevent the creation of mycolic acid which leads to bacterial cell wall interruption and cell lysis. |
| iv) | Kanamycin [27] | Kanamycin is an antibiotic class of the aminoglycoside. Aminoglycosides function by binding to the bacterial ribosomal subunit 30S, producing misinterpretation of t-RNA, Leads to bacteria is not able to synthesize protein which are essential for growth of cell. |
| v) | Amikacin [28] | Amikacin fixes to bacterial 30S ribosomal subunits and affects with mRNA binding lead to inhibit protein synthesis which are necessary for its growth. |
| vi) | Cycloserine [29] | Cycloserine blocks peptidoglycan formation, allows the cell wall to collapse and contributes to cell death. |
| vii) | Viomycin [30,31] | Viomycin restricts translocation which obstructs the synthesis of proteins, leading to death of bacterial cells. |
| 3. | | |
| i) | Ofloxacin [32] | Ofloxacin is a fluoroquinolone antibiotic. Ofloxacin inhibits bacterial topoisomerase II and topoisomerase IV mechanism, which are participating in duplication and repair of DNA, leads to cell death. |
| ii) | Ciprofloxacin [33] | Ciprofloxacin is a synthetic broadspectrum fluoroquinolone antibiotic. Ciprofloxacin inhibits bacterial DNA gyrase mechanism, leads to obstruction of DNA replication, resulting in cell death. |
| iii) | Clarithromycin [34] | Clarithromycin attach to 50S subunit of ribosome and hunder protein production in bacteria which is important for its growth. |
| iv) | Rifabutin [35] | Rifabutin hinders bacterial DNA-dependent RNA polymerase, which inhibit transcription or RNA synthesis. Which leads to inhibition of protein synthesis which are essential for cell growth. |

**Table 2:** Analytical detection of first line antitubercular agents by HPLC

| Sr. no | Method | Mobile phase | Stationary phase | Flow rate (ml/min) | Wavelength (nm) | Retention time (min) |
|---|---|---|---|---|---|---|
| i) | Isoniazide[36] | Ethanol: water: 1%acetic acid (5.3:93.7:1, v/v/v) | C-18 column | 1.5 | 265 | 2.28 |
| | Isoniazide[37] | Water: methanol (85:15, v/v) | C-8 column (250 x 4.6mm,5m) | 1.2 | 274 | 4.1 |
| ii) | Pyrazinamide[38] | 0.02M Potassium dihydrogen phosphate (pH 2.6): acetonitrile (98:2, v/v) | C-18 column (25 cm × 4.6 mm, 5 $\mu$m) | 1 | 268 | 8.4 |
| iii) | Ethambutol[39] | Methanol: water: glacial acetic acid (70:30:0.2, v/v/v) | C-18 column | 1 | 210 | 29.26 |
| iv) | Rifampicin[40] | Methanol: acetonitrile: (0.075 M) monopotassium phosphate: (1.0 M) citric acid (28:30:38:4, v/v) | C-18 column (150 × 4.6 mm, 5m) | 2 | 254 | 2.91 |

**Table 3:** Analytical detection of second line antitubercular agents by HPLC

| | | | | | | |
|---|---|---|---|---|---|---|
| i) | Paraminosalicylic acid[41] | 20 mM phosphate buffer, 20mM tetrabutylammoninum hydrogen sulphate and 16% (v/v) methanol adjusted to pH 6.8 | C-18 column (50 mm × 4.6 mm, 5 $\mu$m) | 1 | 233 | 2.783 |
| ii) | Thiacetazone[42] | Water: acetonitrile (92.5:7.5, v/v) | Internal surface reversed-phase (ISRP) mixed-functional phenyl column (Capcell Pak, 50x4.6 mm, 5 $\mu$m) | 1 | 322 | 10.9 |
| iii) | Ethionamide[43] | Acetonitrile: phosphate buffer (75:25, v/v) | C-18 column (250×4.6mm, 5$\mu$m) | 1.5 | 291 | 3.8 |
| | Ethionamide[44] | Methanol: water (40:60, v/v) | C-18 column (250 × 4.6 mm, 5 $\mu$m) | 1 | 275 | 5.467 |
| iv) | Kanamycin[45] | 22 mM disodium 1,2-ethanedisulfonate and 5 mM sodium octanesulfonate in a water-acetonitrile mixture (80:20, v/v) | C-18 column (40 × 8 mm,10 $\mu$m) | 1.5 | Ex: 351 Em: 440 | 9 |
| | Kanamycin[46] | Acetonitrile:0.1 M sodium acetate buffer (pH 5.0; 25:75, v/v) | C-18 column (250 x 4.6 mm, 5 $\mu$m) | 2 | 330 | 19.5 |
| v) | Amikacin[47] | Acetonitrile:0.1 M sodium acetate buffer (pH 5.0; 25:75, v/v) | C-18 column (250x4.6 mm, 5 $\mu$m) | 2 | 330 | 8.7 |
| | Amikacin[48] | Methanol:acetonitrile: acetate buffer (75:20:05 v/v) | C-18 column (250 mm x 4.6 mm, 5 $\mu$m) | 1 | 212 | 4.61 |
| vi) | Cycloserine[49] | 20mM Sodium hydrogen phosphate: acetonitrile (95:05, v/v) | Agilent Zorbax SB phenyl column (250 × 4.6 mm, 5 $\mu$m) | 1 | 335 | L- 10.4 D- 11.8 |
| | Cycloserine[50] | 10 mM Phosphate buffer: acetonitrile (90:10, v/v) | Atlantis T3, (150 × 4.6 mm, 3 $\mu$m) | 0.4 | 240 | 5.1 |

**Table 4:** Analytical detection of new antitubercular agents by HPLC

| | | | | | | |
|---|---|---|---|---|---|---|
| i) | Ofloxacin[51] | Acetonitrile: Buffer (35:65, v/v) | C-8 column (250 cm × 4.6 mm, 5 $\mu$m) | 1.5 | 315 | 10 |
| | Ofloxacin[52] | Triethylamine: acetonitrile :0.3% o-phosphoric acid ((0.02:20:80, v/v/v) | C-18 column (250 × 4.6 mm,5 $\mu$m) | 1 | 290 | 6.15 |
| | Ofloxacin[53] | 0.03M Potassium dihydrogen phosphate: methanol (30:70, v/v) | C-18 column (150 × 4 mm,4 $\mu$m) | 1 | 294 | 5.17 |
| ii) | Ciprofloxacin[54] | Phospahte buffer (2.7 pH): Acetonitrile (77:23, v/v) | C-18 column (250 x 4.6 mm, 5 $\mu$m) | 1.5 | 277 | 3.26 |
| | Ciprofloxacin[55] | 2% Acetic acid: acetonitrile (84:16, v/v) | C-18 column (150 × 4.6 mm, 5 $\mu$m) | 1 | 280 | 6.5 |
| | Ciprofloxacin[56] | 5% Acetic acid: acetonitrile: methanol (90:5:5, v/v/v) | C-18 column (150 × 6 mm, 5 $\mu$m) | 1 | 280 | 12 |
| | Ciprofloxacin[57] | Water: acetonitrile: triethylamine (80:20:0.3, v/v/v) | C-18 column (125 x 4 mm,5 $\mu$m) | 1 | 279 | 2.4 |
| C | Ciprofloxacin[58] | 0.025M Orthophosphoric acid (3 pH): methanol (60:40, v/v) | C-18 column (125x4mm, 5$\mu$m) | 2 | 278 | 1.75 |
| | Ciprofloxacin[59] | 0.1M Potassium dihydrogen phosphate: acetonitrile (80:20, v/v) | C-18 column (250 mm × 4.6 mm, 10) | 1.5 | 276 | 5.15 |
| iii) | Clarithromycin[60] | Acetonitrile: methanol:0.04 M phosphate buffer (pH 6.9) (52:9:39, v/v) | Perkin-Elmer Spheri-5 cyano column (100 × 4.6 mm, 5 $\mu$m) | 1 | ECD | 9.2 |
| | Clarithromycin[61] | 0.05M Phosphate buffer (3.2 pH): acetonitrile (50:50, v/v) | C-18 column (250 × 4.6 mm,5 mm) | 1 | 205 | 2.21 |
| | Clarithromycin[62] | Acetonitrile: methanol: potassium dihydrogen phosphate buffer (7.5 pH) (40:6:54, v/v) | C-8 column (125 × 4.0 mm) | 1.5 | Amperometric detector | 4.8 |
| | Clarithromycin[63] | Acetonitrile: formic acid: water: trifluoroacetic acid (70:15:14.9:0.1, v/v) | C-18 column (250x4.6 mm, 5 $\mu$m) | 1 | ELSD | 4.7 |
| | Clarithromycin[64] | Acetonitrile: phosphate buffer (11 pH) (60:40, v/v) | Asahipak Shodex ODP-50 4E column (250 mm × 4.6 mm, 5 $\mu$m) | 1 | 210 | 6.46 |
| | Clarithromycin[65] | Acetonitrile: 0.035 M potassium dihydrogen phosphate (pH 4.4) (55: 45, v/v) | C-18 column (150× 4.6 mm,5 $\mu$m | 0.6 | 210 | 4.1 |
| iv) | Rifabutin[66] | 50mM Phospahte buffer (4.2 pH): acetonitrile (53:47, v/v) | C-18 column (250 x 4.6mm,5m) | 1.2 | 265 | 8.5 |
| | Rifabutin[67] | Methanol: Water (75:25, v/v) | C-8 column (250 × 4.6 mm,5 $\mu$m) | 1 | 240 | 5.5 |
| | Rifabutin[68] | Acetonitrile + Methanol (1:1): Water (75:25, v/v) | C-18 column (250 x 4.6mm,5m) | 1 | 242 | 5.3 |

antitubercular agents by HPLC. From the study we can conclude that there are various analytical detection methods are available. The current work contains compilation of the analytical detection method antitubercular agents by HPLC.

## 3. Source of Funding

None.

## 4. Conflict of Interest

None.

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## Author biography

**TS Basuri,** Associate Professor

**Ranjit Mohapatra,** Assistant Professor

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