Minimizing Transmission

Treat persons with active TB using anti-TB drugs.

This reduces the number and viability of tubercle bacilli in the sputum and reduces sputum production.

People producing sputum samples that are smear positive and/or culture positive for AFB should be isolated for a minimum of two weeks after start of treatment, AND until they have three consecutive smear-negative sputa samples.

The spread of tubercle bacilli can be controlled by mechanical means such as covering the nose and mouth, wearing a N95 mask while in transit, and remaining isolated in a facility with negative pressure ventilation.

All TB isolation rooms in a facility are to meet **Canadian Standards** for airborne respiratory isolation (http://shop.csa.ca/en/canada/health-care-facility-engineering/cancsa-z3172-10/invt/27013482010).

Exhaust is to be vented to the outside, and air exchange is to be greater than 12 air exchanges per hour (http://www.hss.gov.nt.ca/sites/default/files/nwtinfectioncontrolmanual.pdf).

Transmission Outcomes

After exposure to a source case, it can lead to one of **three different outcomes:**

1. The bacteria are destroyed by phagocytic cells of the immune system and cause no infection

   TST- / IGRA-

2. Infection will lead to primary disease (i.e. active disease) usually in less than 24 months

3. Cell-mediated immunity and delayed-type hypersensitivity reactions contain the infection at the site of implantation (i.e. via encapsulation) and the bacteria remains dormant

   (LTBI) TST+ / IGRA+

In the first situation, if bacteria are successfully cleared by the immune system, then test results will remain negative on the tuberculin skin test (TST) or interferon-gamma release assay (IGRA). In the latter situation, latent TB infection can stay dormant and never develop into active disease. This is the most probable occurrence. In less than 5% of cases and after a certain period of dormancy, the infection can progress to active disease during an individual's lifetime. It has been observed that the Aboriginal population tend to develop primary tuberculosis disease at higher rates within the first 24 month period of infection. This alludes to a possible genetic variation in the cytokine/vitamin D response as witnessed in the Dene/Cree population.

Figure 2.4: The Pathogenesis of Tuberculosis in the Infected Host (Adapted from the 7th Edition of the Canadian TB Standards)

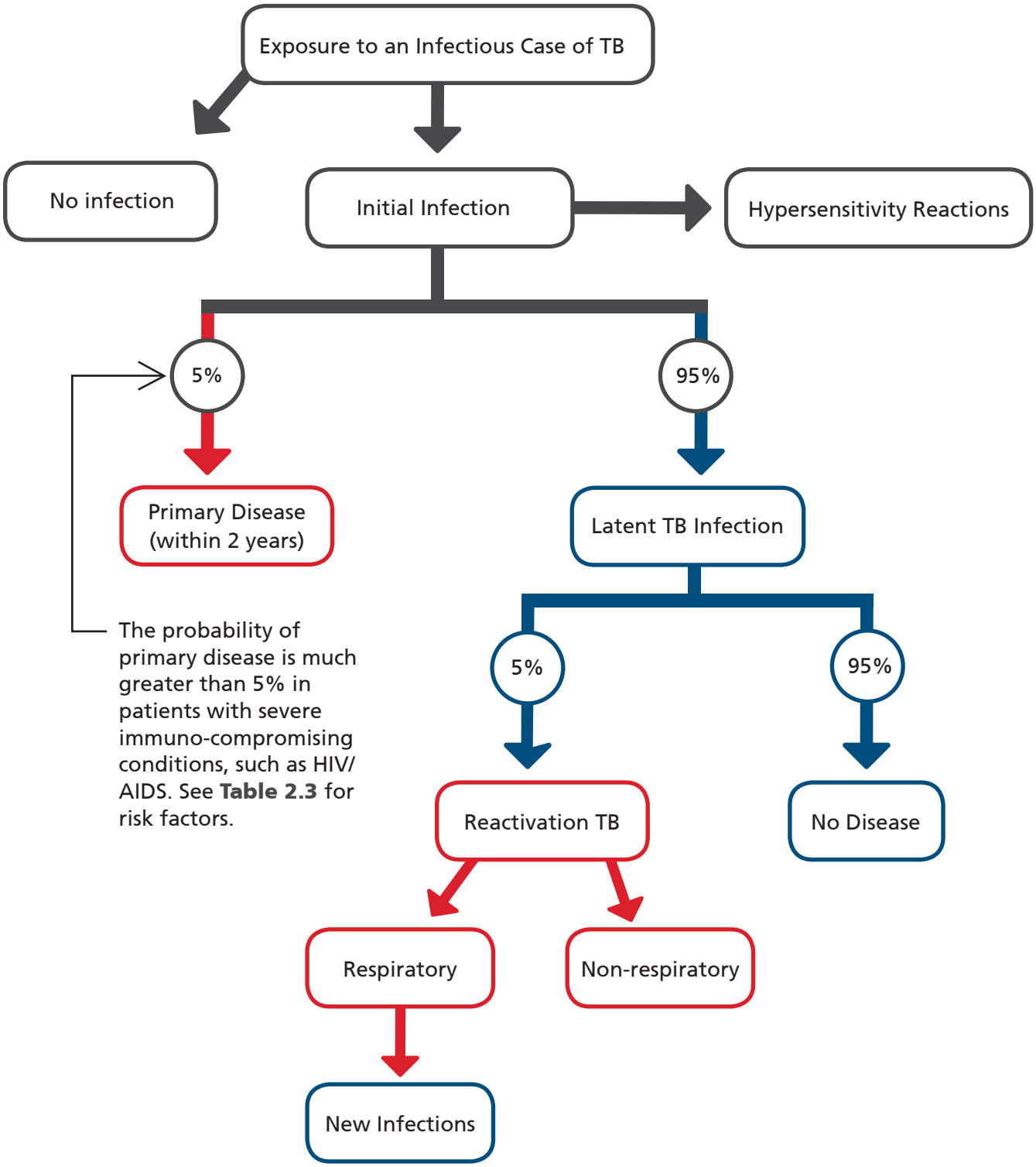
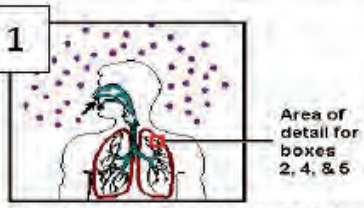


Figure 2.5: Defense Against Respiratory Infection

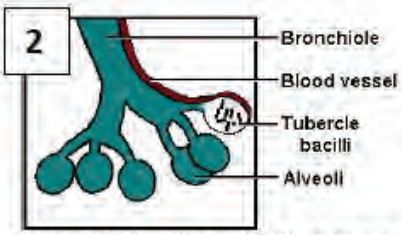
Adapted from www.niaid.nih.gov/factsheets/tb.htm

1



Droplet nuclei containing tubercle bacilli are inhaled, enter the lungs, and travel to the alveoli.

2



Tubercle bacilli multiply in the alveoli.

3

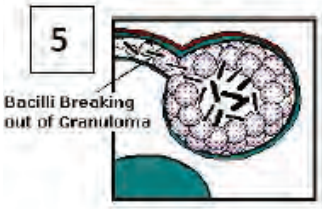
In the first 2-8 weeks:

- Alveolar macrophages scavenge alveolar surfaces ingesting organisms and other inhaled particles (non-specific defense).
- The stimulated T-cell releases chemical signals that produce inflammatory reactions (cell-mediated hypersensitivity) seen as induration when given a tuberculin skin test (seen above).
- The individual may have flu-like illness, erythema nodosum, and enlargement of hilar lymph nodes on chest x-ray (CXR) (also referred to as Primary TB).

4

- T cells also recruit and activate specialized cells to kill bacilli and wall off infected macrophages to form tiny, hard grayish nodules (granulomas).

5



- Bacilli may break out of the granulomas in the alveoli and travel to other sites (most often lungs or lymph nodes).
- The immune system kills many bacilli, but also immune cells and local tissue → producing vague symptoms (weight loss, fever, night sweats, and loss of appetite).
- As bacilli continues to replicate more lung tissue is destroyed, resulting in the development of a productive, infectious cough.
- Granulomas may then liquefy; bacilli multiply rapidly and spread, creating further lesions (chest pain and hemoptysis). This is also called Pulmonary TB.

Table 2.2: Respiratory vs. Non-respiratory TB Disease

Respiratory Tuberculosis Disease	Non-respiratory Tuberculosis Disease
• Lungs • Conducting airways • Pleura • Intrathoracic lymph nodes • Mediastinum • Nasopharynx • Nose • Sinus	• Peripheral lymph nodes • Genitourinary system • Bone and joint • Intestines/Peritoneum/Mesenteric glands • Brain/Meninges • Eye/Retina • Ear • Thyroid • Adrenals • Liver • Spleen • Skin • Myocardium/Epicardium/Pericardium • Oesophagus

Those with reactivated TB disease (in the past referred to as post primary disease) will manifest as respiratory, non-respiratory disease or both in rare cases. The differentiation of both diseases is described in **Table 2.2**.

Non-respiratory disease occurs when the immune system does not contain the bacteria and they gain access to the circulatory system and lymphatic system seeding other organ sites as a consequence. Non-respiratory tuberculosis involvement tends to increase in those with worsening immune compromise.

Risk Factors

Specific risk factors have been identified for the development of active TB among persons who are infected with MTB. The lifetime cumulative risk to develop TB disease is 10% and these risk factors influence at various levels of the immune response as it diminishes, leading to the possibility of reactivation.