

naso-pharyngeal mucosa, thence by the lymphatics of the neck to the peribronchial lymph-nodes, and so to the lungs. In fact, not a few hold now that penetration of airborne bacilli into the lungs is difficult, if not impossible. Vausteenberghe and Grysez state that anthracosis, to quote an almost parallel case, is not produced in animals by making them breathe an atmosphere saturated with soot so long as they are prevented from swallowing the accumulations of the nose and pharynx. The same thing applies to attempts to infect the lungs with tubercle bacilli by respiration. Calmette and Guérin of the Pasteur Institute at Lille confirm these observations and have enquired more particularly into the part played by intestinal infection. It will be noted that their work in certain points corroborates that of von Dungern and Smidt.

Calmette and Guérin have studied the question in kids and goats. The young were suckled by goats whose udders had been inoculated with tuberculous virus, human, bovine, and avian. In the older animals the infective material was introduced into the stomach by an oesophageal sound in order to obviate the possibility of infection through the upper air-passages. The result of these experiments goes to show that kids and goats, are very susceptible to tubercular infection, especially that of bovine origin, and moreover, that they readily contract tuberculosis by way of the intestinal tract. In the young, virulent bacilli, absorbed in small quantity, were retained for varying periods in the mesenteric lymph-nodes and might later infect the lungs. The bacilli in adults, however, produced almost immediately tuberculosis of the lungs. Similar results were obtained in bovines and rodents. In their opinion it would seem logical to infer that a similar state of things may obtain in man, and that von Behring is justified when he states that pulmonary tuberculosis in the adult results from the tardy evolution of an intestinal infection contracted in early life. They find that tubercular bacilli may pass the intestinal wall without producing any lesion. They are then taken up by phagocytes in the chyloferous vessels. Hence, they get into the venous circulation, and the dead or dying leucocytes, carrying the bacilli, and having lost their amoeboid properties have a tendency to become arrested in the capillaries, particularly those of the lungs, which they block as a foreign body. Should these infection-bearing leucocytes pass through the pulmonary circulation they may be arrested in the capillaries of the meninges, joints, kidneys, and other structures. It is noteworthy, that their experiments showed that animals that had recovered from tuberculosis were rendered partially immune against subsequent tubercular