tissue as self. That is, it is a foreign material in the lungs and therefore the body reacts to it by producing fibrosis. Again, these two theories, the autoimmune theories, are not widely accepted. However, the fourth theory, the stagnation of phagocytes, which is somewhat similar to the last one I mentioned, says that the phagocytes disintegrate in situ or in place in the lungs and in doing so release sclerosing agents which are probably lipids or lipoproteins. This is the theory which is currently favored as explaining the development of asbestosis.

(Next Slide) Moving on from asbestosis, we come to bronchogenic cancer. This is the type of cancer of the lung which one normally refers to when speaking of lung cancer; that is, the same type of cancer that occurs in cigarette smokers. It is cancer of the bronchial tubes. In the 1930's, an association was discovered between asbestosis and bronchial cancer. This was especially true in asbestos textile workers in whom an excess risk was firmly established in the 1950's. I mention this because this group of workers apparently has a high exposure to asbestos dust. In other groups of workers this relationship has not been found to be as strong. The association appears to be with asbestosis rather than simply exposure to the asbestos dust. Cigarette smoking is an important additional and possible synergistic factor. In fact, Dr. Selikoff estimates an increased risk factor of 90. That is, the risk of developing cancer in an asbestos worker who smokes would be 90 times greater than an asbestos worker who doesn't smoke. This is quite something and I think it emphasizes the importance that if one is an asbestos worker, he should not smoke. The latent period here is 20 to 30 years; if you remember, this contrast with 10 to 20 years for the onset of asbestosis.