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## Theobald Smith – The discoverer of ticks as vectors of disease

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**Summary.** The cause of Texas fever in cattle, which is characterised by lysis of erythrocytes leading to anaemia, icterus, haemoglobinuria, and death, remained unsolved for many decades and assorted theories were proposed as an explanation for a disease being transmitted by apparently healthy animals. From 1889 to 1893, Theobald Smith and Frederick L. Kilbourne could demonstrate in elegantly conducted experiments how the disease was spread from cattle to cattle by ticks serving as the vector of transmission. Furthermore, they were able to identify the pathogen of Texas fever, an intra-erythrocytic protozoan which Smith named *Pyrosoma bigeminum*. Today it is recognised that either of two species of the now renamed genus *Babesia*, *Babesia bigemina* and *Babesia bovis*, may be involved in Texas fever and that babesiosis is generally transmitted by ticks. In animals, genera like *Boophilus spp.*, *Derma-centor spp.* and *Rhipicephalus spp.* are possible vectors. The first case of tick-transmitted babesiosis in a human was reported by Skrabalo and Deanovic in 1957 and occurred near Ljubliana in the small town of Strmec, Croatia. In humans, the vectors of most reported cases are ticks of the genus *Ixodes*, which are among the most predominant ticks in Austria. However, cases of human babesiosis in Austria remain to be studied. Smith and Kilbourne's work was the first demonstration that ticks transmit disease of any kind. Furthermore, by proving that ticks carry *Babesia microti* – which causes babesiosis in animals and humans – this is the first account of a zoonotic disease and the foundation of all later work on the animal host and the arthropod vector.

**Key words:** Texas fever, Theobald Smith, Frederick L. Kilbourne, Skrabalo and Deanovic, *Babesia bigemina*, *Babesia bovis*, *Babesia microti*.

### Background

In 1868 more than 70,000 Texas Longhorns, trailed from Texas to Kansas, were shipped by rail to the midwest and east. Within a month of their arrival, native northern cattle began dying from a mysterious disease. Many cattle herds were totally lost or had major fatalities. The Texas cattle appeared healthy, and only northern cattle became ill and died. Farmers started calling the disease *Texas fever*. To protect their cattle, states along the cattle trails

passed quarantine laws directing cattle away from settled areas or restricting the passage of herds to the winter months when there was less danger from Texas fever [1, 2].

Though Texas fever was clearly associated with Texas cattle, its cause remained a mystery for many years. Various theories were proposed to account for a fatal disease being transmitted by apparently healthy animals. There was a lack of knowledge about where the disease came from, how it was transmitted and how to treat the disease.

It was obvious that the disease occurred wherever southern cattle were trailed. The spread of the disease stopped with the first frost every fall. Some cattlemen suggested that the disease might be related to ticks found on many of the southern Texas cattle even though the ticks disappeared during the northern winter [3].

In 1869, after a study commissioned by the Department of Agriculture, scientists concluded that the disease might be associated with the grass grazed by cattle, but "there is not the slightest foundation for the view that ticks disseminate the disease".

### Experiments

From 1889 to 1893 Theobald Smith and his co-worker Frederick L. Kilbourne demonstrated in elegantly designed experiments [4] how the disease was transmitted from healthy southern cattle to cattle from the north.

On experimental fields with southern cattle and ticks, northern cattle died. On fields with southern cattle but where the ticks had been previously removed, northern cattle did not acquire Texas fever. On tick-infested fields with northern cattle but no southern cattle, northern cattle died. Smith concluded: no ticks, no Texas fever.

Moreover, he was able to identify the pathogen of Texas fever. He demonstrated that the disease is caused by a protozoan that inhabits erythrocytes. Smith named this organism *Pyrosoma bigeminum*. Now it is recognized that either of two species of the renamed genus *Babesia*, *Babesia bigemina* and *Babesia bovis*, may be involved in Texas fever.

### Pathogenesis

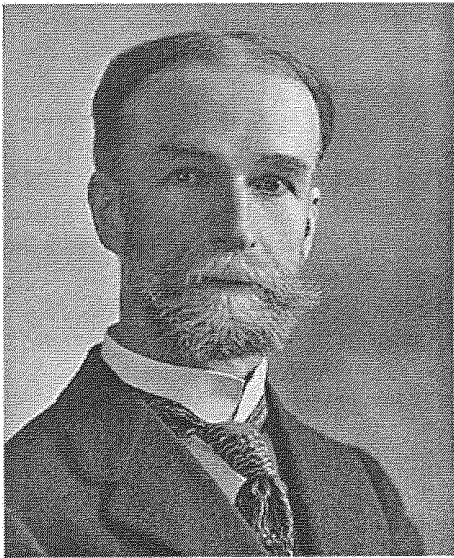
The only ticks capable of transmitting Texas fever in North America are the cattle fever ticks *Boophilus annulatus* and *Boophilus microplus*. These one-host ticks ordi-

narily spend all stages of their life cycle on the same cow. After engorging with blood the adult female tick drops from the cow, lays eggs on the ground, and then dies. *Babesia bigemina* is transmitted through the eggs to the next generation of the ticks, which after hatching attach to passing cattle. This life cycle explains why grass appeared to be poisoned by the Longhorns. The grass did not cause the disease; ticks lurking in the grass were responsible. Because the first frost killed ticks on the ground, cattle crossing the area in the winter were safe from the disease.

Babesiosis is generally transmitted by ticks. In animals, genera like *Boophilus spp.*, *Dermacentor spp.* and *Rhipicephalus spp.* are possible vectors, whereas in humans, the vectors of most reported cases are ticks of the genus *Ixodes*. The principal vector of *Babesia microti* in the U.S. is *Ixodes dammini* [5]. The first reported case of tick-transmitted babesiosis was observed in Yugoslavia in 1957 by Skrabalo and Deanovic [6].

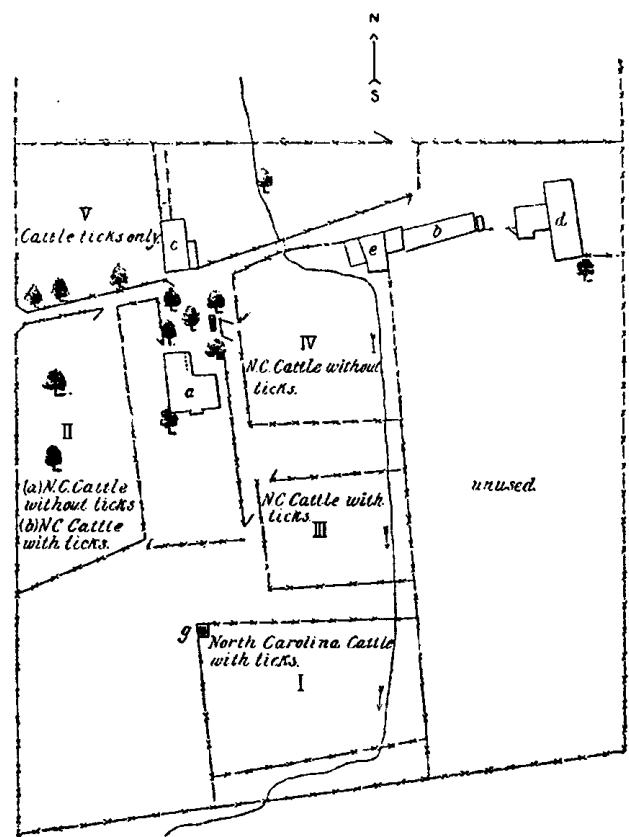
### Immunity

Identification of the pathogen and vector of babesiosis still did not explain the apparent good health of the Texas cattle that carried the disease. It is now understood that calves are born with a natural partial resistance to infection that lasts a month or two after birth and goes away

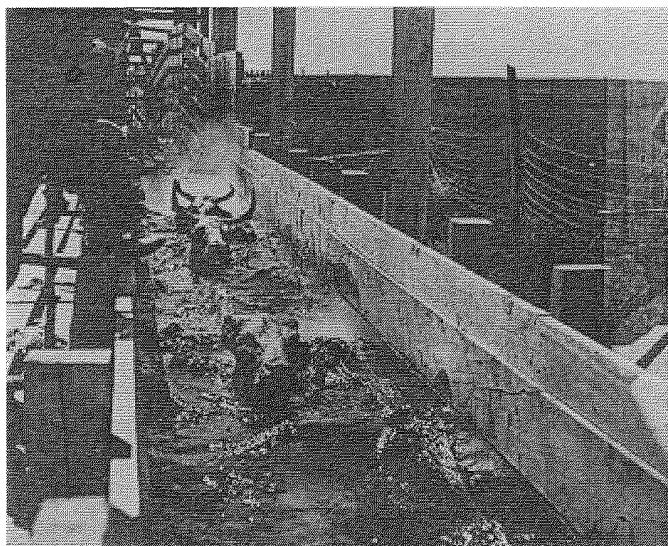


**Fig. 1.** Theobald Smith, son of German immigrants coming from Rhineland to the U.S. in 1854, was born in Albany, New York on 31 July 1859. Beside demonstrating the etiology of Texas cattle fever, he differentiated between human and bovine tubercle bacilli, and was responsible for identifying the cause of several other animal diseases. Later, he and D.E. Salmon injected heat-killed whole-cell vaccine of *Vibrio cholerae* into pigeons and demonstrated immunity to subsequent administration of a live microbial culture. Smith declined the position of director of the Rockefeller Institute (now Rockefeller University) at its founding in 1901. However, after working for several years in Washington and at the Harvard Medical School, he joined the Princeton laboratories of the Rockefeller Institute in 1915, where he remained as director of the Department of Animal Pathology until his retirement in 1929. He died in New York on 10 December 1934

gradually. In areas where the disease was widespread, the calf suffered a mild attack at an early age, then developed enough immunity to keep from being overwhelmed but not enough to rid itself of the pathogen. By the time the animal reached adulthood, it had an unsteady balance with its protozoan parasites that allowed it to live in reasonably good health while remaining a carrier as long as the babesia remained in the blood. Cattle staying in the Texas fever area were continually reinfected, so they remained immune. When they left the area, their immunity persisted for several years, but they were carriers of the disease. In contrast, animals first infected as adults often died. Cattle anywhere north of southern Texas had no chance of developing resistance to the disease as calves, and the Longhorns on the drives carried not only Texas fever but also the ticks necessary to transmit the disease.



**Fig. 2.** Experimental field from 1889. Scale:  $\frac{1}{4}$  inch = 33 feet. a dwelling house; b station laboratory; c house stable; d cow stable; e breeding pens; f tool house; g shed in field; field V: field of healthy Texas Longhorns heavily infested with ticks; field IV: field of healthy North Carolina Cattle without ticks; experimental fields I–III: On experimental field I, Texas Longhorns grazed together with North Carolina Cattle. North Carolina Cattle became infested with ticks, and subsequently they developed Texas Cattle fever and died. On experimental field II, Texas Longhorns without ticks (ticks were removed manually by Smith and Kilbourne) grazed together with healthy North Carolina Cattle. North Carolina Cattle remained healthy. Next, healthy North Carolina Cattle were transferred to the heavily tick-infested experimental field I where the cattle became infested with ticks, developed Texas Cattle fever, and died. As a last step, Smith transferred grass and ticks from Experimental field I to Experimental field III. Subsequently, healthy North Carolina Cattle became tick-infested and died of Texas Cattle fever



**Fig. 3.** Cows running through dipping vats containing arsenic water

### Eradication

Once transmission of the disease was understood, it became apparent that if all cattle fever ticks in South Texas were eliminated, Texas fever could be eliminated as well. Because cattle fever ticks are one-host ticks restricted to cattle, their eradication was possible.

Smith: „Eliminate the ticks on cattle and you eradicate the ticks because they cannot live elsewhere”. Consequently, all cattle in the Texas fever area were run through dipping vats containing arsenic to kill the ticks, and dip-

ping was repeated periodically until no more ticks could be found. Since 1942 cattle fever ticks have been considered eradicated from the United States.

### Smith's contribution

The importance of Smith and Kilbourne's work can hardly be overestimated. It was the first demonstration that ticks transmit disease of any kind. Furthermore, by proving that ticks carry *Babesia microti* – the cause of babesiosis in animals and humans – this was the first account of a zoonotic disease and the foundation of all later work on the animal host and the arthropod vector.

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