Role of Saliva in Tick/Host Interactions

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ABSTRACT


Although several hosts mount efficient anti-tick immunity, natural tick/host associations are characterized by inefficient or non-existent anti-tick immunity. The absence of efficient anti-tick immunity in natural hosts could result from either host immune incompetence or the ectoparasite’s successful evasion of the host’s immune response. In this review I discuss data supporting the immune-evasion hypothesis and discuss its consequences to tick/host interactions.

INTRODUCTION

The classical paper authored by William Trager in 1939 indicated that guinea pigs were able to mount a very efficient immune response to the tick, Dermacentor variabilis. This immune response was characterized by a local reaction at the feeding site that prevented the tick from obtaining the blood meal. In addition, attachment of the tick to an immune host enhanced grooming, further reducing the ectoparasite’s survival. Thus, a previously exposed guinea pig would allow only 3–5% of added larvae to feed, as opposed to more than 50% when naive animals served as hosts. A different result was obtained when the white-footed mouse Peromyscus leucopus was the host for D. variabilis. A single exposure would never confer anti-tick immunity and, after multiple exposures, only a partial immunity developed. Trager concluded that unnatural D. variabilis hosts, such as guinea pigs, were able to develop efficient anti-Dermacentor immunity, but natural hosts, such as the white-footed mouse, were not.

The immunological basis of tick resistance, studied mostly in guinea pigs, has been the subject of much research. Trager (1939) indicated that partial

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immunity could be transferred with serum, pointing to the mediation of antibodies in the rejection response; additional serum factors, such as the alternative-complement pathway, were implicated (Wikl and Allen, 1977). Cellular immunity also plays a role in tick rejection reactions; in particular, the role of basophils was well characterized and, indeed, tick/immune-host interactions became and experimental model for the study of the basophil-delayed hypersensitivity (Allen, 1973; Askenase, 1977; Brown et al., 1982). However, the lack, or weakness, of anti-tick immunity in natural tick/host associations remained poorly understood.

In theory, the lack of anti-tick immunity could be due to the host’s inability to mount an effective immune response (host immune incompetence), or to the ectoparasite’s ability to evade its host’s immunological reaction (parasite immune evasion). The host immune incompetence theory has been assumed, without proof, by many investigators in their pursuit of effective anti-tick vaccines that could be useful in scenarios of natural tick/host associations (Wikl and Allen, 1982). The purpose of the present review is to give support to the hypothesis of parasite immune evasion in the explanation of stable tick/host associations, to outline a basic protocol for its test in different tick/host associations, and to speculate on its consequence in predicting the outcome of any tick/host interaction.

HOW UNNATURAL HOSTS REJECT TICKS

Edema is the most conspicuous histological observation at the tick feeding sites on resistant hosts (Tatchell and Moorhouse, 1968; Wikl and Allen, 1982). In some hosts, a basophil infiltrate exists which, through its released mediators, may increase vascular permeability at the feeding site and contribute to enhanced edema formation. Edema may be the significant component in tick rejection reactions. The availability of a nutritious meal is reduced because only a protein-poor serum transudate is available to the tick at edematous sites. Many ticks which survive this meal either do not molt or molt to abnormally small nymphs or adults (Wikl and Allen, 1982).

Many pharmacological mediators are efficient edema promoters. Among them are the vasoactive amines histamine and serotonin, the leukotrienes, particularly C4 and D4, and peptides such as bradykinin and the anaphylatoxins. Adenosine triphosphate, released by injured cells, also promotes edema. Prostaglandins, particularly of the E series, induce vasodilation and may potentiate the edema induced by the above agonists by increasing the hydrostatic perfusion pressure at the capillary bed (Williams and Peck, 1977; Bach, 1982; Ribeiro, 1987a).

Edema-promoting mediators may either be released from cells or result from acellular reactions. Basophils, mast cells and platelets have stored in their