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Molecular mimicry as a mechanism for the initiation of autoimmunity in Chagas disease

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Trypanosoma cruzi is the protozoan parasite that causes Chagas disease, a complex illness in which infected individuals may have no symptoms of infection or may develop cardiomyopathy or megadisease of the esophagus or colon. Several mechanisms may contribute to the development of cardiomyopathy, including autoimmunity. There are two major questions about autoimmune pathogenesis: What is its origin? and What is its role in pathogenesis? We have investigated both using a variety of combinations of parasite and mouse strains giving the varied cardiac outcomes observed in human infection. Infection of A/J mice with the Brazil strain of *T. cruzi* leads to massive cardiac inflammation with the presence of parasite nests. However, these mice also developed significant autoimmunity with cardiac myosin as the dominant autoantigen, of a magnitude similar to that observed in mice with purely autoimmune myocarditis induced by immunization with cardiac myosin (EAM). This implied that myosin autoimmunity contributes to inflammation. To test the possibility that molecular mimicry is responsible for autoimmunity, we immunized mice with a *T. cruzi* protein powder or heat-killed *T. cruzi* and, to our initial surprise, mice developed strong autoimmunity in addition to the expected parasite-specific immunity. Finally, injection of heat-killed *T. cruzi* led to cardiac damage and release of serum cardiac troponin I. The basis for the difference in whether the myosin autoimmunity is relatively benign (heat-killed *T. cruzi* immunization) or pathogenic (myosin immunization) is under investigation. Elimination of *T. cruzi* with benznidazole treatment caused a resolution of autoimmunity as well, which returned rapidly upon exposure to the parasite. Thus, although autoimmunity may be pathogenic, it is also dependent on the presence of the parasite and is not self-propagating.

Biography

David Engman received his MD-PhD from the University of Iowa in 1990 and joined the faculty of Northwestern University at that time. He is now Professor of Pathology and Microbiology and has also devoted the majority of his professional life to the training of physician-scientists as Director of the Northwestern University MD-PhD Program. Engman is also Attending Pathologist at Northwestern Memorial Hospital in the Diagnostic Molecular Biology Laboratory.

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