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Interaction of Host Genome with Infectious Virus.

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Introduction

Mucosal surfaces provide portals of entry into the body—for substances essential to survival, like food, water, and air—and for several pathogens, including viruses. Over evolutionary time, hosts have developed. A battery of anti-viral immune mechanisms to combat these attacks. When an epidemic encounters a mucosal surface, the interplay between viral invasion and host antiviral defense strategies ultimately determines the severity and outcome of the infection. Like all traits, the host anti-viral response is dictated partially by genetics and may vary with genetic polymorphisms across the population. Similarly, other host proteins important for virus infection, like cell surface receptors utilized by viruses, can vary across the host population. The importance of those variations might not be clear when studying the pathogenesis of a highly virulent virus in susceptible hosts. However, recent advances in viral detection are starting to paint a replacement picture of mucosal viral infections in humans and other hosts—a picture during which viruses are frequently present, often within the absence of a clear-cut disease association. Within the context of frequent, less virulent infections, variations within the host anti-viral response and host viral targets may take center stage in determining the physiological impact of a virus infection.

PCR and other genetic methods have revolutionized our ability to detect viruses in samples from infected hosts. Since the 1970s, viruses are routinely detected in clinical and research laboratories using viral culture together with various antibody-based techniques1. While these methods are often powerful, they're also labor-intensive, can require multiple days for a result, and may fail to detect many virus types. Since the invention of PCR2, macromolecule based techniques are increasingly applied to the detection of viral genetic material within host fluids and tissues. As compared to viral culture, a PCR-based test is fast, requiring only several hours to finish, and is extremely sensitive. With the event of the many reagents and platforms over the past decade, macromolecule amplification techniques became widely available and accessible; they're now the first method for detecting viruses in research and are increasingly being adopted in clinical settings.

For many years, investigators have sought infectious causes for autoimmune diseases like Type 1 diabetes. Autoimmune diseases, like infections, involve activation of the immune reaction and inflammation. However, no straightforward causal relationship between these diseases and particular pathogens has been uncovered. Recent evidence puts this search during a new context. Within the last several years, autoimmune diseases are investigated extensively in genome-wide association studies (GWAS). With a spread of approaches, investigators have sought to find out how the “hits” in GWAS relate to disease pathogenesis. For both Type I Diabetes and inflammatory bowel disease, new evidence links disease development to regions of the host genome involved in host-virus interactions.

Here, we'll discuss how genetic and genomic technologies have widened our view of mucosal virus infections. In contrast to a 1 pathogen-one disease model, we describe a model of the human virome during which we are nearly continually exposed to viruses, which can or might not cause symptoms. During this context, the virome is a crucial component of the environment which will interact with host genetic traits to contribute to the pathogenesis of complex diseases. We highlight several recent examples during which interactions between host genetics and viral infections are implicated in autoimmune and inflammatory diseases.

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