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Aspergillosis includes a spectrum of diseases caused by different Aspergillus spp. New insights into the cellular and molecular mechanisms of resistance and immune tolerance to the fungus in infection and allergy have been obtained in experimental settings. The fact that virulence factors, traditionally viewed as fungal attributes, are contingent upon microbial adaptation to various environmental stresses encountered in the human host implies that the host and fungus are jointly responsible for pathogenicity. Ultimately, despite the occurrence of severe aspergillosis in immunocompromised patients, clinical evidence indicates that aspergillosis also occurs in the setting of a heightened inflammatory response, in which immunity occurs at the expense of host damage and pathogen eradication. Thus, targeting pathogenicity rather than microbial growth, tolerance rather than resistance mechanisms of defense may pave the way to targeted anti-inflammatory strategies in difficult-to-treat patients. The challenge now is to translate promising results from experimental models to the clinic.