Perfect pathogen

Literature, Russian Literature

My idea of a perfect pathogen would be one that can survive and persist long enough to cause infection and to be transmitted to the next host. And this multiplicity of behaviours depends at least on the generation of diverse cell surface structures and phenotypic variants to evade the host immune response. One such strategy to achieve this in bacteria is antigenic variation, a strategy also employed by viruses, fungi, and protozoa, which all face similar challenges when infecting a host, that is to overcome the host immune response. Antigenic variation generally encompassed in two forms: phase variation (on-off expression of a particular antigen) and true antigenic variation (expression of alternative forms of a particular antigen). An infecting organism having the ability to alter their cell surface structures such as capsule, pili etc., which are displayed to the host immune system during the course of an infection, is of essence to prevent host recognition and destruction. Consequently, it also effectively confronts the host with a continually changing population that makes it difficult or impossible to eliminate by the host. This will not only help increase the persistence and survival of the pathogen, extending the length of the infection, but it will allow the pathogen to multiply undetected (at least temporarily) and increases the likelihood of transmission and contributes to the success of the pathogen and possibly reinfection. Such alterations in surface structures can also facilitate adaptation to new environments by conferring a fitness advantage in certain environments in the face of selective pressure such as naturally acquired and vaccine-induced immunity. Capsular switching in Neisseria meningiditis whereby the capsular phenotype of the organism changes is one such mechanism. Capsular switching, which occurs through

horizontal gene transfer allows it to acquire large sequences of DNA, presumably during cocolonization of the human nasopharynx with at least two strains. Increasing evidence suggests that outbreaks of meningococcal infection can be initiated or sustained through capsular switching, presumably allowing escape from natural immunity against the original serogroup. Therefore, any naturally acquired and vaccine induced anti-capsular antibody will become ineffective in controlling the pathogen. Another example is phase variation - having the ability to switch on and off a virulence factor during different courses of the infection metabolically conserved energy by regulating the gene expression of products that are not needed in response to the environment. A classic example is Neisseria meningitidis is capsulated during airbone transmission between hosts but once in the respiratory tract, it switches off capsular expression because non-capsulated bacteria is more invasive but when it disseminates in the blood and the CSF, it requires its capsule to protect it from phagocytosis and thus capsular expression is switched on. Because of a common need to evade the host immune system for any pathogen, I believe that genomic plasticity is a feature no perfect pathogen can omit.