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Commentary

# Development of Human Immunity from Prenatal to Geriatrics

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## DESCRIPTION

Innate and adaptive immune responses involve more than 1600 genes. These genes play a critical role in surviving in hazardous conditions. However, the immune system is weak at birth and must evolve during the course of a lifetime of exposure to many outside stressors, from prenatal to geriatric (including pregnancy), to adult decline. Humans have acquired mechanisms of innate immunity and immunological memory to survive recurrent infections as a long-lived species. These immunological mechanisms, on the other hand, alter with time, initially to adapt to the transition from foetal to newborn, then to mature and expand during growth, subtly changing during pregnancy, and finally diminishing in senescence. As the older person has encountered and created a memory bank too many diseases over their lifetime, the output of naive lymphoid cells and the ability to form new immunological memory become less essential. It's possible that the myeloid bias and greater release of pro-inflammatory cytokines that occur as people age are required for better phagocytosis of an increasing number of senescent cells, raising the question of whether the ageing immune system has a purpose.

Evolution has principally shaped the immune system to respond effectively to acute infections in young adults, to adapt to pregnancy and transfer protection to children, and to cope with various chronic illnesses that can continue for decades. Aside from combating viruses, bacteria, fungi, and parasites, the immune system is also responsible for tissue regeneration, wound healing, the removal of dead and malignant cells, and the establishment of a healthy gut flora. We may have to pay for genetic features selected to ensure early-life fitness by the later development of immunological phenotypes like chronic inflammation, assuming there is no strong selecting pressure on people beyond reproductive age. Massive ageing and enhanced longevity are relatively new phenomena that have emerged in a highly tuned environment. Aging may be a result of evolutionary unpredicted antigenic exposure across an individual's lifespan, and so alterations in the ageing immune system may simply be a result of this.

The immune system of the elderly resembles that of a newborn in certain ways, with diminished antimicrobial activity by neutrophils and macrophages, decreased antigen presentation by myeloid dendritic cells and decreased NK killing, and partially weakened adaptive lymphocyte responses. In dealing with a typical viral infection such as influenza, both the very young and old immune systems are similarly impaired, whereas the young (non-pregnant) adult organism appears to be perfectly adapted for this task. The key function of the young adult in the survival of the species for its procreative capacity may be reflected in the evolution of the immune system inside an individual.

The immune system undergoes significant remodelling and deterioration as people get older, with serious consequences for their health and survival. Older persons are more susceptible to acute viral and bacterial infections as a result of immunological senescence. Furthermore, when compared to younger adult patients, the death risk for these illnesses is three times higher in the elderly. In the developed world, infectious diseases remain the fourth leading cause of death among the elderly. Furthermore, abnormal immunological responses in the elderly can aggravate inflammation, perhaps contributing to cancer, cardiovascular disease, stroke, Alzheimer's disease, and dementia, among other maladies of old age. During a typical influenza season, those over 65 account for almost 90% of the additional deaths. In addition, vaccine efficacy is affected by inadequate immune responses. Immune aging also causes dormant viruses to emerge, such as the varicella-zoster virus, which produces shingles and persistent neuralgia. At the same time, as the immune system ages, it loses its ability to retain full tolerance to self-antigens, leading to an increase in autoimmune ailments. This is most likely related to lymphopaenia, which causes excessive homeostatic lymphocyte proliferation, as well as diminished regulatory T-cell function and macrophage clearance of apoptotic cells as people get older.

## CONCLUSION

Most current findings has shown that during prenatal and geriatric, important developmental changes occur that alter every layer of the innate immune system, from exterior barriers to single cell function. The age at which many functions settle to adult levels varies greatly between research, which can be related to different methodology as well as the unique characteristics of

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each population. Despite this, there is still a lot to learn about the age-dependent development of inherent components in various populations.