**Immune Response in SARS-CoV-2 Infection, Inflammation, and Cancer Immune Evasion and Tumour Progression**

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The immune response to SARS-CoV-2 infection plays a pivotal role in determining disease severity and long-term outcomes, including potential links to carcinogenesis. Initial immune activation leads to both **acute and chronic responses**, characterized by the release of pro-inflammatory cytokines and the recruitment of immune cells to the site of infection, which can contribute to severe respiratory complications and afterwards to long COVID, characterised by prolonged inflammation. Chronic inflammation resulting from persistent SARS-CoV-2 infection may invoke **immune dysregulation,** promoting an environment favouring tumour development through mechanisms such as **reactive oxygen species (ROS) production, immune exhaustion**, and **viral reactivation** of oncogenic pathogens​ like human papillomavirus (HPV) or Epstein-Barr virus (EBV), known to drive cancer development. Long COVID further complicates the relationship between SARS-CoV-2 and cancer, as ongoing inflammation and tissue damage resemble processes that promote carcinogenesis. This inflammatory state can lead to the upregulation of oncogenes and impair the immune system’s ability to monitor and eliminate emerging tumour cells. Additionally, long COVID may induce metabolic alterations and hypoxic conditions that favour tumour growth, alongside psychological stressors that exacerbate the risk of cancer​. Understanding the connections between SARS-CoV-2, chronic inflammation, and cancer progression is essential for developing comprehensive surveillance strategies for cancer in COVID-19 survivors. Future research must elucidate the molecular pathways involved and assess the long-term oncogenic risks associated with SARS-CoV-2 infections, guiding strategies to mitigate these risks.