INNATE IMMUNE STATUS IN ADVANCED DISEASES OF THE UPPER RESPIRATORY TRACT AGAINST THE BACKGROUND OF COVID-19 INFECTION

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Innate immunity is the first line of defense of the host and plays a key role in reducing the spread of pathogens. Unlike adaptive immunity, which works by creating special receptors against microbial antigens (for example, antibodies or TCR), innate immunity is based on the recognition of common molecular structures present in microbial structures.

These patterns of "pathogen-related molecular structures" (PAMP) are caused by groups of conserved receptors called detection receptors (PRR), which include cell surface receptors or endosome-like receptors (TLR), retinoic acid-induced gene I (RIG-I) and NOD-like receptors (NLR). RARs associated with SARS-CoV contain ssRNA of the viral genome and intermediates formed during virus replication, i.e. dsRNA, as well as viral proteins. Coronavirus otsrna and dtsrna molecules can be detected using multiple endosomal or cytosolic PRRS. It has been reported that both endosomal TLR3 and endosomal TLR3 are highly regulated after infection with coronavirus infection.

Similarly, cytoplasmic RNA sensors, RIG-I and the 5th gene differentially associated with melanoma (MD5), both respond to coronavirus RNA. Several studies on SARS-CoV and MERS-CoV have shown that IGE-mediated signaling plays an important role in the occurrence of interferon antiviral reactions to these viruses. "The Interferon Gene Stimulator (STING) is another PR commonly known as a cytosolic DNA sensor, and although coronaviruses do not contain DNA, SING-mediated signaling has been reported after coronavirus infections. Viral proteins are a different group of PAMPS. The SARS-CoV spike protein is known to interact with TLR2 and activate downstream signaling pathways.

Thus, the SARS-CoV M protein also plays the role of cytosolic PAMP and causes TLR-related interferon reactions.