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Research Progress on Cellular Immune Mechanisms and New Vaccine Platforms in the Development of COVID-19 Vaccines

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Abstract. In recent years, the novel coronavirus (SARS-CoV-2) caused novel coronavirus pneumonia (COVID-19) in the global serious impact, and the number of symptomatic and asymptomatic infections continued to rise, the World Health Organization (WHO) to publicly declare COVID-19 as a pandemic. Vaccines, along with human specific and non-specific immunity, are powerful means of combating SARS-CoV-2 and can to some extent control the speed and route of COVID-19 transmission. Therefore, it is crucial to have a deep understanding of the characteristics of SARS-CoV-2, the transmission routes and methods of COVID-19, the immune mechanisms of the body in the face of SARS-CoV-2 and the immune escape methods of the virus, the types and advantages and disadvantages of vaccines, and to enhance the performance, safety, and effectiveness of vaccine development. The article mainly elaborates on the characteristics and immune escape of SARS-CoV-2, the immune response of the human body in the face of virus invasion, and the characteristics of different COVID-19 vaccines, as well as the prospects for future vaccine development and improvement.

Keywords: COVID-19, SARS-CoV-2, vaccine, immune mechanism.

1. Introduction

At the end of 2019, a global epidemic broke out in Wuhan, Hubei Province, China. It was a respiratory disease caused by the novel coronavirus (full name: Severe Acute Respiratory Syndrome Coronavirus 2, hereinafter referred to as SARS-CoV-2). This virus spreads globally in a short period of time, according to data released by the World Health Organization.

By January 16, 2021, the cumulative number of confirmed cases of novel coronavirus pneumonia (COVID-19) worldwide has exceeded 92 million, and the cumulative number of deaths has exceeded 2 million [1]. When the disease occurs, it is often accompanied by symptoms such as difficulty breathing, persistent high fever, limb weakness, muscle soreness, etc. However, compared to other respiratory diseases, although the mortality rate of SARS-CoV-2 is much lower, its transmissibility is higher than other respiratory diseases. It can be seen that SARS-CoV-2 can remain latent in the human body for a long time and then spread to the public through asymptomatic or symptomatic patients. In summary, combining history and current social conditions, the outbreak of

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epidemics is undoubtedly a major challenge to the global public health system, and the development and improvement of COVID-19 vaccines are important tasks at present.

In history, vaccines have helped humanity overcome multiple epidemics such as smallpox, measles, and poliomyelitis. SARS-CoV-2 virus belongs to a enveloped positive stranded RNA virus with a genome of approximately 30000 nucleotides. And in this epidemic, mRNA vaccines have provided great help to humans, especially mRNA vaccines have advantages and guarantees in safety due to their short half-life in the human body. Moreover, the development cost of mRNA vaccines is relatively low, and the high transcription efficiency and yield in vitro meet the advantages of rapid production and low cost of mRNA vaccines in the short term. These advantages have led to the widespread production and development of mRNA vaccines in the market, making significant contributions to the fight against COVID-19.

MRNA vaccines are still full of innovative value in today's market. In 1982, China first artificially synthesized active yeast propargyl tRNA and entered the forefront of international RNA research. In recent years, the level of RNA research in China has developed rapidly and vigorously, which has also brought objective profits and opportunities to the market. In addition, the investment and support of domestic project funds in improving the immune mechanism of COVID-19 vaccines and reducing side effects have amplified the research prospects of this sector. It is undoubtedly important to explore the cellular immune mechanism and improve the research value of COVID-19 vaccines in depth. Therefore, the text will further explore and discuss the cellular immune mechanisms in the development of COVID-19 vaccines and the research progress of new vaccine platforms.

2. Structure and transmission pathways of SARS-CoV-2

2.1. Structure of novel coronavirus

SARS-CoV-2 was first discovered in Wuhan, China in 2019. It belongs to the beta coronavirus 2B lineage, and in subsequent tests, SARS-CoV-2 was highly similar to SARS of the same lineage, with sequence similarity exceeding 79%. Later, scholars compared SARS-CoV-2 with the SARS like coronavirus strain BatCov RaTG13 in bats and found that the relationship between the two was the closest, with an identity of up to 96% [2,3]. In the future, most scholars speculated and believed that SARS-CoV-2 evolved from BatCov RaTG13 in bats, and some groups also enjoyed eating wild bats, believing it to be a way of praying for blessings. In short, further research is needed to determine the origin and evolution of SARS-CoV-2.

In further research, scholars noticed that the new coronavirus is a typical envelope virus, which is round or oval in shape and 60-120 mm in diameter [3]. The envelope consists of lipids and glycoproteins, the spike like spike protein embedded in the envelope, which is 9-12 mm in diameter [3], the nucleocapsid composed of nucleic acid and protein capsid in the body of the virus, and the matrix protein between the envelope and the nucleocapsid. As a single stranded positive RNA virus, the genome of novel coronavirus usually starts with 5 '- UTR and ends with 3' - UTR [4]. The gene length of SARS-CoV-2 is 30kb, with a G+C content of 38%, and has been annotated to have 10 ORFs (Open reading frames) encoding at least 26 proteins [3]. These proteins play a crucial role in the survival rate and toxicity of SARS-CoV-2. For example, membrane protein (M protein) can alleviate the host's immune response and enhance pathogenicity by interfering with cellular pathways during SARS-CoV-2 immune escape.

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2.2. Transmission route of novel coronavirus

In recent years, COVID-19 caused by SARS-CoV-2 has undoubtedly been a global public health crisis, with tens of thousands of people infected and causing a series of health problems. This is due to the faster transmission speed of SARS-CoV-2, surpassing SARS CoV and MERS CoV. A large part of this is because SARS-CoV-2 has a wider transmission route compared to the two viruses, a stronger affinity for receptors on its spike protein surface that interact with the host, and faster virus updates and mutations.

SARS CoV-2 is usually transmitted through body fluid, aerogel and excreta through close contact without isolation protection between infected persons and non infected persons [5]. In 2019, there were also cases of COVID-19 infection in Wuhan, China through the consumption of wild bats, and the SARS like coronavirus strain BatCov RaTG13 carried by bats is highly similar to SARS-CoV-2. Therefore, the SARS virus carried by animals can also be transmitted to humans through mutation. In 2020-2021, there were large-scale COVID-19 infections in multiple mink breeding bases in Denmark. SARS-CoV-2, after mutating in minks, re infected humans, such as mink-CoV-2.

2.3. Pathogenic mechanism of novel coronavirus

SARS-CoV-2 is the seventh member of the coronavirus family that causes disease in humans, and the fourth human coronavirus (HCoV-229E, HCoV-NL63, HCoVOC43, and HCoV-HKU1). ACE2 is the main receptor for the spike protein S of SARS-CoV-2 virus. The S1 subunit of the S protein on the surface of SARS-CoV-2 virus binds to ACE2 on the surface of host cells, and then the S protein is cleaved into S1 and S2 subunits under the action of host TMPRSS2, promoting the fusion of the virus envelope and host cell membrane, and finally releasing viral RNA into the cytoplasm.

3. Immune response mechanisms

3.1. Innate immune response

Innate immunity is an innate ability of the human body, and this immune response does not target a specific pathogen. It is the first line of defense for the human immune system. The innate immune response is composed of multiple parts of the body. Firstly, there are physical barriers such as the skin and mucous membranes that can prevent viruses from entering the body directly from the outside world. Secondly, there are chemical barriers such as gastric acid (mainly composed of HCL hydrochloride), sweat, tears, and other substances that can further kill viruses entering the body from the outside world. In summary, innate immune response can respond to multiple pathogens, but does not generate immune memory because the pattern of each immune response is the same and can quickly take effect and play a role before adaptive immunity.

Cellular immunity is an important part of the human immune system, which is distributed throughout the body. Macrophages are distributed in tissues and can engulf pathogens and release cytokines during immune responses, ultimately initiating inflammatory responses. White blood cells are also members of macrophages, circulating throughout the body with the blood. People's attention to natural killer NK cells is constantly increasing, and a series of experiments have been conducted using mice. NK cells have been detected in most lymphoid organs of mice, with extremely high levels and activity in the spleen and lymph nodes. In the collaboration of Dr. W.H. West and others, the distribution of NK cells in various lymphoid organs was examined. Unlike mice, humans detect

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a large number of NK cells in spleen cells, but have almost no cytotoxic activity in thymus, lymph nodes, and tonsils [6].

Interferon (IFN) belongs to an antiviral drug or a cellular product. In early research, Isaacs and Linden mann reported in 1957 that chicken cells infected with influenza virus produced a secreted factor to combat the virus. Later in 1958, Nagano and Kojima also published and described similar findings, laying the foundation for future research on the IFN system. The IFN system includes the response of synthetic cells to external stimuli, followed by the establishment of an antiviral state through the response of IFN and infected cells [7]. And the action of IFN represents the active defense of early hosts, occurring before the immune response begins. The duality of cytokines and drugs makes IFN an important bridge between innate immunity and clinical treatment.

3.2. Adaptive immune response

Adaptive immunity is the process in which the body's immune system recognizes external antigens and produces specific antibodies through activation and differentiation of T and B cells to fight against external viruses, thereby generating an immune response and gaining memory to quickly respond the next time an antigen is detected. Adaptive immune response can be summarized into four characteristics. 1) Specificity refers to the ability to generate an adaptive immune response specifically targeting an antigen. 2) Memory, which generates memory T and B cells after the first contact with an antigen and produces an immune response, can quickly respond to the second invasion of the same antigen into the body. 3) Tolerance can prevent immune diseases in the body by not responding to antigens produced by oneself. 4) Diversity, the different arrangements of multiple genes can generate a large number of different types of antigen receptors, which can cover the vast majority of foreign antigens in the world [8].

T cells differentiate from bone marrow hematopoietic stem cells and mature in the thymus. If T cells are differentiated in detail, three types of T cells can be seen in the human body: helper T cells (CD4+Th cells), cytotoxic T lymphocytes (also known as Tc1 cells), and regulatory T cells (Treg). Assistive T cells are mainly responsible for secreting cytokines, regulatory T cells are responsible for the body's homeostasis and balance, and suppress excessive immune responses. The most clear effect of cytotoxic T lymphocytes is the CD8+T cell subset, which plays a role in killing pathogens in adaptive immune responses. Cytotoxic T lymphocytes can selectively kill cells carrying target antigens by releasing cytotoxic molecules through immune synapses, and secrete cytokines such as IFN - γ, which will further accelerate the natural and adaptive immunity of cells in the body [9].

3.3. COVID-19 immune control

Under normal circumstances, IFN - α/β produces an intracellular antibacterial state to provide the first line of defense against the virus and affect its replication process. What sets COVID-19 apart is that it actively inhibits a series of reactions mediated by IFN - α/β, to the extent that it may not be able to control virus replication. However, compared with IFN - β, IFN - α can effectively inhibit the replication of SARS CoV, but the inhibitory effect is 50-90 times lower than IFN - β [10]. Various research data indicate that IFN - β may be a more effective antiviral component in combating COVID-19 at present. In the later DisCoVeRy experiment, the subcutaneous injection of INF - β combined with the treatment of lopinavir/ritonavir, hydroxychloroquine and remdesivir [10].

Part of the innate immune physical barrier is located in the lung epithelium of the human body, which inevitably introduces pathogenic particles into the body when inhaling large amounts of air every day. Therefore, covering the entire mucus layer and body fluids is crucial and forms a defense

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line. This line of defense is composed of dendritic cells, respiratory epithelial cells, and alveolar macrophages. The severity of the disease is closely related to the depletion of retained alveolar macrophages and the aggregation of inflammatory macrophages in bronchoalveolar lavage fluid. However, further evidence is needed to demonstrate the infectivity of the macrophage lineage to SARS-CoV-2 [10]. Mucosal associated invariant T (MAIT) cells are a type of natural T cell primarily used to recognize metabolites presented by the MR1 protein and secrete TNF - α (tumor necrosis factor - α), IFN - γ, IL-17A (interleukin-17A), IL-22, as well as granzyme B and perforin for antibacterial purposes. The latest data shows that CD69 activator on blood MAIT cells can predict the severity of diseases [10].

3.4. Virus immune escape strategy

The essence of SARS-CoV-2's immune escape from the host is to invest a large amount of resources to combat and block the antiviral response. SARS-CoV-2 sometimes attempts to mask inflammatory RNA during immune evasion. In research, it has been shown that the viral genome ribonucleoprotein complex enters the cytoplasm, causing the genome to dissociate from the virus's N protein, which can then be directly translated by the host's ribosome [11]. The initial translation of viral RNA is consistent with the host translation rules, on the one hand to integrate into the host and function, and on the other hand to evade the host's immune checkpoint. Afterwards, due to the virus's mRNA being disguised as the host's mRNA, the initial translation of the replication enzyme did not trigger an immune response in the cells. As viral RNA is continuously synthesized in the host's body, the emergence of dsRNA intermediates can make host cells detect and produce immune responses, because both viral RNA and sgRNA need to go through negative chain intermediates [11]. These opposite and complementary RNA transcription substances undoubtedly increase the frequency of dsRNA occurrence, which can be further detected by the host immune system.

So, the detection of dsRNA determines the success of virus immune escape, and SARS-CoV-2 is no exception. Minimizing the virus or keeping it away from the host sensor is undoubtedly the best choice, and SARS-CoV-2 perfectly utilizes these two points. The process of establishing RO is also achieved by interfering with or utilizing autophagy biology to reduce host recognition [11]. After the virus successfully establishes RO, SARS-CoV-2 will leave the DMV and enter the cytoplasm, and then translate and assemble particles in a safer environment.

4. The core mechanism of 3-cell immunity in COVID-19 vaccine development

4.1. T cell-mediated immune response

The immune response mediated by T cells, as the name suggests, refers to the differentiation, proliferation, and transformation of T cells in the body into sensitized T cells when they recognize and are stimulated by antigens. When the body detects a secondary entry of antigens, sensitized T cells will directly kill the antigens. In addition, the cytokines released by sensitized T cells will have a synergistic effect. CD4+T cells are mainly expressed by helper T cells (Th cells) and are receptors for Th cell T cell receptors (TCR) to recognize antigens. CD4+T cell-mediated cellular immunity is mediated by CD4+T cell-specific immune responses, which may cause exudative inflammation in macrophages and T lymphocytes. In addition, CD4+cells have the function of assisting cellular and humoral immune responses. CD8 molecules are differentiated from white blood cells, and CD8+T cells are important effector cells, CD4+T cells have stronger self reactivity [12].

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Current research has shown that cellular immunity mediated by CD4+, CD8+, and T lymphocytes runs through the onset and recovery of COVID-19 from start to finish. Transient immunodeficiency and other issues may occur in acute COVID-19 patients, but CD4+, CD8+, and T cell fluctuations are not significant. Significant reductions in CD4+, CD8+, and T cell numbers can be clearly observed in patients with severe or critical COVID-19 [12]. CD4+T cells play an auxiliary role in CD8+T cells, with the core being signaling or cytokines, further promoting the differentiation and memory of CD8+T cells. CD8+T cells play a regulatory role on CD4+T cells. Firstly, CD8+Tregs suppress autoimmunity by clearing CD4+Tfh/Th17 cells to achieve inhibitory effects. Secondly, Tc17 cells induce the pathogenicity of CD4+Th17 cells through IL-17, exacerbating autoimmune levels and achieving a promoting effect. There is cross regulation and immune balance between CD4+T cells and CD8+T cells, which is manifested in the fact that the differentiation of CD8+T cells can affect a series of reactions mediated by CD4+T cells, achieving "bidirectional regulation" [9].

4.2. The role of immune checkpoint

Immune checkpoints are regulatory molecules on the surface of cells involved in immune responses, mainly used to adjust the timing and intensity of immune responses, achieve balance in immune activity in the human body, and avoid damage caused by excessive responses. The main expression sites of PD-1 (programmed death receptor 1)/PD-L1 (programmed death ligand 1) are different. The former is mainly expressed in activated T cells, B cells, and NK cells, while the latter is mainly expressed in tumor cells, APCs (antigen-presenting cells), and some normal tissue cells. After combining the two, the TCR signaling pathway is inhibited, causing T cell apoptosis and limiting immune response. In some tumors, upregulation of PD-L1 expression can be achieved to evade detection and attack by the immune system. Nivolumab is a good example in blocking pathways and restoring T cell killing power against tumors [13]. Multiple pieces of evidence have shown that PD-1 plays a role in regulating COVID-19. By comparing blood samples from COVID-19 infected and healthy individuals, PD-L1 RNA expression levels were observed, demonstrating the prognostic role of PD-L1 in COVID-19 patients, and PD-L1 dysregulation is also related to the pathogenesis of COVID-19. Furthermore, SARS-CoV-2 induces upregulation of PD-L1 while infecting epithelial cells, leading to dysregulation of PD-L1 expression in the patient's immune cells [13].

5. New platform and research and development progress for COVID-19 vaccine development

5.1. mRNA vaccine platform

With the comprehensive outbreak of SARS-CoV-2, WHO is also regularly releasing a list of vaccines under development to the public [14]. And according to the data, there are 183 vaccines currently undergoing clinical trials and 199 vaccines currently undergoing preclinical research. And most of the late selected targets are involved in receptor binding, and the membrane is fused to the host's surface facial mask S protein [15]. As early as the early 1930s, two virologists in the United States, Jonas Salk and Thomas Francis, began developing vaccines for influenza. Afterwards, they chose fertilized eggs as the culture medium and created an inactivated influenza A vaccine produced from chicken embryos.

Compared to other SARS-CoV-2 vaccine platforms, mRNA vaccines have advantages such as low production process cost and speed, making them more marketable and attractive. As early as August 2020, two mRNA vaccines, mRNA-1273 and BNT162b1, had entered phase III clinical

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trials. The former can stabilize the S protein by fusing the conformation before encoding, and can induce humoral and cellular immunity in animal models, demonstrating overall good safety. Based on the good safety of mRNA immunity, there is no need to handle viruses during the production process, which can effectively control the risk of infection. MRNA also has no risk of insertion mutations, so it does not need to penetrate deep into the nucleus to express antigens [15]. Although mRNA vaccines are by no means a perfect product, there is still room for improvement in stability and RNA translation efficiency. Contemporary scholars are still committed to improving mRNA vaccines to stabilize their efficiency.

5.2. Virus vector vaccine

Virus vector vaccines have also demonstrated many unique advantages in combating SARS-CoV-2. Firstly, it should be clarified that the core purpose of the vaccine is to further train the human immune system and cells to recognize antigens faster when the corresponding virus invades the host, thereby activating the immune response and better protecting the host. Understanding a vaccine should first ensure its safety. Virus vector vaccines are modified by removing toxic genes, retaining their ability to enter cells but not causing disease. The target simulates the viral infection pathway, even partially removing genes that allow replication [16]. Stability is crucial in the production, transportation, and storage of vaccines, and some mRNA vaccines are required to be stored at ultra-low temperatures to avoid degradation. As mentioned earlier, mRNA-1273 and BNT162b1 also require ultra-low temperature storage. The latter can be stored between -80 ℃ and -20 ℃ for 6 months, while the former can be stored at -20 ℃ for 6 months. However, adenovirus vector vaccines can be stored at lower temperatures for the same time as mRNA vaccines, and Ad26.COV2S can survive for 6 months at 2 ℃ to 8 ℃ [16]. In terms of immunity, viral vector vaccines can present antigens and are more likely to be delivered in one step without the need for booster shots, which can save costs and avoid the impact of large personnel movements on epidemic prevention and control during the epidemic period.

6. Conclusion

Through the research on the cellular immune mechanism and new vaccine platform in the development of COVID-19 vaccine, it is found that there are two types of mainstream vaccines against novel coronavirus in the market, mRNA vaccine and viral vector vaccine. Therefore, further exploration of the advantages and disadvantages of the vaccines themselves leads to the conclusion that the two vaccines have different advantages and disadvantages, as well as different manufacturing processes, efficiency, and costs. Moreover, there are also differences in the types of vaccines that are practical in different environments under the current epidemic environment. Therefore, both types of vaccines play a crucial role. This study focuses on introducing the appearance and transmission routes of COVID-19, the immune response of the body in the face of COVID-19, and the escape strategy of SARS-CoV-2 in the face of host cell immunity. In addition, the focus is on describing mRNA vaccines and viral vector vaccines and introducing their advantages and disadvantages. This is helpful for scholars who wish to compare mRNA vaccines with viral vector vaccines, understand immune mechanisms and immune escape. In future research, it is hoped that the storage temperature and time of mRNA vaccines can be increased and extended to improve RNA translation efficiency and reduce the risk of infection when viral vector vaccines are virus-free. Make more varieties of novel coronavirus vaccines complete the phase III clinical

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trials, more and better put into market use, serve public health and reduce the emergence of COVID-19 patients.

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