

## Apprehensive Perception of Disease Vaccines in Work

Ding Xue Zhi\*

Northwest Institute of Plateau Biology, Chinese Academy of Sciences Xining Lanzhou University, China

### Abstract

Different types of white blood cells fight infection in different ways. Macrophages are white blood cells that swallow up and digest germs and dead or dying cells. The macrophages leave behind parts of the invading germs, called antigens.

**Keywords:** T-lymphocytes; COVID-19; Vaccination; Vector vaccines; Treatment; Paracetamol

### Introduction

The body identifies antigens as dangerous and stimulates antibodies to attack them. B-lymphocytes are defensive white blood cells. They produce antibodies that attack the pieces of the virus left behind by the macrophages. T-lymphocytes are another type of defensive white blood cell. They attack cells in the body that have already been infected. The first time a person is infected with the virus that causes COVID-19, it can take several days or weeks for their body to make and use all the germ-fighting tools needed to get over the infection [1]. After the infection, the persons immune system remembers what it learned about how to protect the body against that disease. The body keeps a few T-lymphocytes, called memory cells that go into action quickly if the body encounters the same virus again. When the familiar antigens are detected, B-lymphocytes produce antibodies to attack them. Experts are still learning how long these memory cells protect a person against the virus that causes COVID-19. COVID-19 vaccines help our bodies develop immunity to the virus that causes COVID-19 without us having to get the illness. Different types of vaccines work in different ways to offer protection. But with all types of vaccines, the body is left with a supply of memory T-lymphocytes as well as B-lymphocytes that will remember how to fight that virus in the future [2]. It typically takes a few weeks after vaccination for the body to produce T-lymphocytes and B-lymphocytes. Therefore, it is possible that a person could be infected with the virus that causes COVID-19 just before or just after vaccination and then get sick because the vaccine did not have enough time to provide protection [3]. Sometimes after vaccination, the process of building immunity can cause symptoms, such as fever. These symptoms are normal signs the body is building immunity. Currently, there are three main types of COVID-19 vaccines that are approved or authorized for use in the United States or that are undergoing large-scale clinical trials in the United States [4]. Below is a description of how each type of vaccine prompts our bodies to recognize and help protect us from the virus that causes COVID-19. None of these vaccines can give you COVID-19. mRNA vaccines contain material from the virus that causes COVID-19 that gives our cells instructions for how to make a harmless protein that is unique to the virus. After our cells make copies of the protein, they destroy the genetic material from the vaccine. Our bodies recognize that the protein should not be there and build lymphocytes that will remember how to fight the virus that causes COVID-19 if we are infected in the future [5]. Protein subunit vaccines include harmless pieces of the virus that causes COVID-19 instead of the entire germ. Once vaccinated, our bodies recognize that the protein should not be there and build T-lymphocytes and antibodies that will remember how to fight the virus that causes COVID-19 if we are infected in the future. Vector vaccines contain a modified version

of a different virus than the one that causes COVID-19. This is called a viral vector. Inside the shell of the modified virus, there is genetic material from the virus that causes COVID-19. Once the viral vector is inside our cells, the genetic material gives the cells instructions to make a protein that is unique to the virus that causes COVID-19 [6].

### Discussion

Using these instructions, our cells make copies of the protein. This prompts our bodies to build T-lymphocytes and B-lymphocytes that will remember how to fight that virus if we are infected in the future. While COVID-19 vaccines were developed rapidly, all steps have been taken to ensure their safety and effectiveness. Booster shots enhance or restore protection against COVID-19 which may have decreased over time [7]. An additional primary dose is for people who are moderately to severely immune-compromised and did not build enough or any protection from their primary vaccine series. 10-15% of the patient those who present with severe Chikungunya progress to Sub-acute or chronic phase. Acute phase is characterized by the triad of Fever, Arthralgia/arthritis and Rash. Fever is sudden onset, high grade with chills and lasts for 3 to 5 days. Fever can be biphasic. Joint pain occurs initially or begins 2-5 days after onset of fever. Arthralgia is bilateral, symmetrical, distal joints more than proximal joints. Most affected joints are hands, wrists, and ankles. Involvement of the axial skeleton was noted in 34 to 52% of cases. Periarticular edema or swelling has been observed in 32 to 95% of cases [8]. Large joint effusions were noted in 15% of cases. Skin rash has been reported in 40 to 75% of patients which may be macular or maculopapular, usually occurs within first 4 days of fever, starts on the limbs and trunk, pruritic in 25 to 50% of patients. Atypical dermatologic manifestations include bullous skin lesions (most often in children) and hyper pigmentation. Cervically lymphadenopathy may occur in 10-40% of patients, mostly in children or young adults. Few patients may present or develop complications like severe sepsis or septic shock, meningo-encephalitis, Guillian-Barres syndrome, myocarditis, decomposition of cardiovascular diseases, fulminant hepatitis in patients with chronic liver disease,

**\*Corresponding author:** Ding Xue Zhi, Northwest Institute of Plateau Biology, Chinese Academy of Sciences Xining Lanzhou University, China, E-mail: Ding\_xue\_zhi@edu.cn.

**Received:** 23-May-2022, Manuscript No. AWBD-22-68953; **Editor assigned:** 25-May-2022, PreQC No. AWBD-22-68953 (PQ); **Reviewed:** 08-Jun-2022, QC No. AWBD-22-68953; **Revised:** 14-Jun-2022, Manuscript No. AWBD-22-68953 (R); **Published:** 21-Jun-2022, DOI: 10.4172/2167-7719.1000158

**Citation:** Zhi DX (2022) Apprehensive Perception of Disease Vaccines in Work. Air Water Borne Dis 11: 158.

**Copyright:** © 2022 Zhi DX. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

pancreatitis, extensive epidermolysis, kidney failure, respiratory failure. Complications occur in patients of extreme ages - > 65, children, pregnancy, co-morbidity –diabetes mellitus, hypertension, ischemic heart disease and other co-infection. In the sub-acute phase the predominant features is arthritis/arthralgia and fatigue. In 10-15% patients the joint symptoms progress beyond 3 months among whom 50 % patients evolve into rheumatoid arthritis or sero negative spondylo-arthritis or non-specific viral arthritis. Investigations include testing blood or serum for direct or indirect evidences of CHIKV infection. Complete blood count will reveal leukopenia with marked lymphopenia, or sometimes pancytopenia and mild thrombocytopenia [9]. In all patients dengue should be excluded by doing NS-1. For confirmation of the case at least one of the followings in the acute phase of illness should be present: virus isolation by cell culture, detection of viral RNA by real-time reverse-transcription polymerase chain reaction, detection of antibody by enzyme linked immune-sorbent assay -Viral specific IgM antibody in single serum sample collected within 5 to 28 days of onset fever or four-fold rise of IgG antibody in samples collected atleast three weeks apart. In CNS affected patients cerebro-spinal fluid maybe studied. Treatment of CHIKV in acute phase is mostly supportive with rest, adequate electrolyte containing fluid and paracetamol upto maximum 4 gm/day for fever and joint pain [10]. Additional tramadol ortapentadol may be used if joint pain is not relieved by paracetamol. There is no specific anti-viral therapy. There is no indication of steroid in acute phase. Non-steroidal anti-inflammatory drugs should best be avoided in the acute phase, but if required may be used once dengue is excluded, but avoid aspirin. Cold compression of joints is helpful. Hospitalization is required for only those with hypo-tension, altered consciousness, bleeding manifestations, co-morbidity, extreme age or decreased intake. In the sub-acute phase the mainstay of treatment is adequate dose of NSAIDs and physiotherapy. In some patients short course systemic steroid may be necessary. In the chronic phase patient must be evaluated by a rheumatologist for the initiation of disease modifying anti-rheumatic drugs. Prognosis is good compared to dengue. Younger patients recover within 5 to 15 days. Recovery is delayed in the elderly. In few patients joint pain can persist up to 2years [11]. The risk of death is around 1 in 1,000 which is relatively low compared to dengue haemorrhagic fever. After a single infection it is believed most people become immune. Preventing mosquito bite and vector control by control of mosquito breeding places are the best way to prevent CHIKV infection. Cases of SARS were first noted in Guangdong Province, China, in November 2002. Initial cases were recognized as atypical pneumonia characterized by high fever, shortness of breath, cough, and pneumonia. Most early cases were associated with people in the wild animal trade and their contacts. The index case for the illness in Hong Kong was a physician from Guangdong Province who travelled to Hong Kong five days after the onset of symptoms, was identified as the source of 16 cases which led to the world-wide outbreak. By the time the epidemic was contained in August 2003, more than 8400 cases and more than 900 fatalities were identified. Cases of SARS occurred in 29 countries in Asia, Europe, and North America. China, including Hong Kong, had 83 percent of all cases. Transmission was believed primarily by droplet spread, and less frequently by direct contact or fomites [12]. Viral shedding in faeces' also has been reported. The etiologic agent was identified as a past unrecognized Corona virus, an enveloped single stranded positive-sense RNA virus. Clinically, cases present following 2 day incubation with fever. Frequently there is malaise, non-productive cough, dyspnea, chills, rigors, and headache. Rhinorrhoea and a sore throat are rare [13]. Radiological signs after the onset of fever show consolidation that increases progressively in size, predominantly in the

lower lung fields but pleural effusions are absent. Biopsy shows interstitial inflammation and oxygen saturation is decreased in about half of patients. Laboratory tests show leucopenia, lymphocytopenia and thrombocytopenia. Diagnosis can be done by viral isolation and characterization, RT-PCR or serology. However, the duration of detectable viremia or viral shedding is unknown. Currently, paired sera and aero conversion or a rising titer is considered confirmation of suspected cases but is not used clinically. Diagnosis is based on clinical findings and exclusion of other causes of pneumonia. Treatment No specific treatment is currently recommended except for meticulous supportive care. As with other viral infections, antibacterial agents are ineffective. In addition, no antiviral agents have been found to provide benefit for treating SARS. Prognosis Mortality was strongly age-dependent, with children and young adults rarely developing fatal disease, while more than half of the clinical cases over the age of 65 years died. Overall mortality was close to 10%. Emerging viral diseases are a major cause of morbidity and mortality in different parts of the world. Humans are responsible for this to a large extent by causing disruption of environmental homeostasis and intruding and altering wild life habitat as well as domestic animals. In order to survive these deadly viruses, much attention is needed to be paid to prevention of transmission of these zoonotic diseases and preserving a healthy environment for both human being and animals. Among all of these the recent outbreak of Chikungunya in our country has threatened the health sector just like Dengue fever did in year 2000. Hopefully with growing knowledge and clinical experience we will be able to overcome this hurdle and contain the disease. The 20th century has seen significant reductions in ecosystems and biodiversity and equally dramatic increases in the numbers of people and domestic animals in habiting the Earth. In fact, continued in advertent human activities like land use changes, population growth, increased contacts with wild animal reservoirs and the degradation of health care resources has increased the opportunity for various pathogenic agents including some deadly viruses to pass from the wild and domestic animals to human beings causing emergence of new diseases. Emerging viral diseases are nothing new. Smallpox probably reached Europe from Asia in the 5th century, and yellow fever emerged in the Americas during the 16th century as a consequence of the African slave trade. Dengue fever arose simultaneously in South-East Asia, Africa, and North America during the 18th century. In 1918-1919 theseo-called Spanish flu spread like wildfire through all five continents, killing between 25 and 40 million people. The second half of the 20th century saw the emergence of HIV/AIDS, among other viral diseases. Even more worrying is the fact that emerging and re-emerging viral diseases have had a tendency to spread more quickly and more widely during the last decade, invading whole countries and continents. It is estimated that the majority, some estimates place it as high as 75%, of these emerging diseases are derived from animals [14, 15].

## Conclusion

Those satisfying tests of homogeneity have been constituted into cholera districts. It was found that the size of the 19 cholera districts thus created varied considerably from single thanas to combinations of up to 20 such local administrative units. As stated in the third article of the series of publications presently under review. Found that in 9 of these 19 cholera districts a state of endemic existed, whereas 10 experienced only epidemic cholera.

## Acknowledgement

None

## Conflict of interest

None

## References

1. Mantur BG, Amarnath SK (2008) Brucellosis in India - a review. *J Biosci IND* 33: 539-547.
2. Mohammad AS, Esmail MM (2012) A review of epidemiology, diagnosis and management of brucellosis for general physicians working in the Iranian health network. *SID ME* 5: 384-387.
3. Kassiri H, Amani H, Lotfi M (2013) Epidemiological, laboratory, diagnostic and public health aspects of human brucellosis in western Iran. *Asian Pac J Trop Biomed CN* 3: 589-594.
4. Cosford KL (2018) *Brucella canis*: An update on research and clinical management. *Can Vet J US* 59: 74-81.
5. Golshani M, Buozari S (2017) A review of brucellosis in Iran: epidemiology, risk factors, diagnosis, control, and prevention. *Iran Biomed J ME* 21: 346-359.
6. M Barbaiya, KH Costenbader (2016) Environmental exposures and the development of systemic lupus erythematosus. *Curr Opin Rheumatol US* 28: 497-505.
7. Gergianaki I, Bortoluzzi A, Bertias G (2018) Update on the epidemiology, risk factors, and disease outcomes of systemic lupus erythematosus. *Best Pract Res Clin Rheumatol EU* 32: 188-205.
8. Estel GJP, Gil MFU, Alarcon GS (2017) Epidemiology of systemic lupus erythematosus.
9. Cooper GS, Parks CG (2004) Occupational and environmental exposures as risk factors for systemic lupus erythematosus. *Curr Rheumatol Rep EU* 6: 367-374.
10. Parks CG, Santos ASE, Barbaiya M, Costenbader KH (2017) Understanding the role of environmental factors in the development of systemic lupus erythematosus. *Best Pract Res Clin Rheumatol EU* 31: 306-320.
11. Wu D, Meydani SN (2014) Age-associated changes in immune function: impact of vitamin E intervention and the underlying mechanisms. *Endocr Metab Immune Disord - Drug Targets UAE* 14: 283-289.
12. Koenig A, Buskiewicz I, Huber SA (2017) Age-associated changes in estrogen receptor ratios correlate with increased female susceptibility to coxsackievirus B3-induced myocarditis. *Front Immunol EU* 16: 1584-1585.
13. Naradikian MS, Hao YI, Cancro MP (2016) Age-associated B cells: key mediators of both protective and auto-reactive humoral responses. *Immunol Rev EU* 269: 118-129.
14. Kovaoui RD, Loebenstein BG (2006) Age-associated changes within CD4+ T cells. *Immunol Lett EU* 107: 8-14.
15. Wu D, Meydani SN (2008) Age-associated changes in immune and inflammatory responses: impact of vitamin E intervention. *J Leukoc Biol US* 84: 900-914.