TALES OF MAN'S TRAVAILS AND TRIUMPHS AS THE BATTLE WITH VIRUSES RAGES ON. SEPT. 24, 2024

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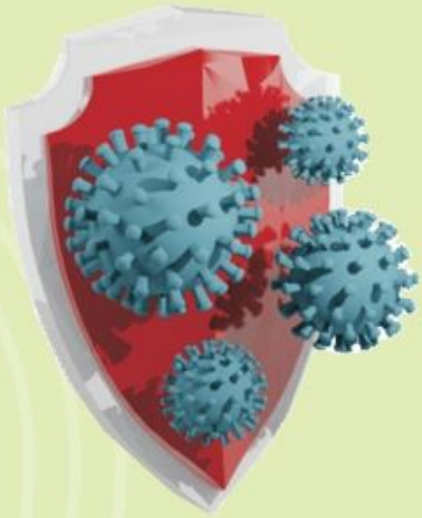

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Professor of Medical Microbiology (Virology)
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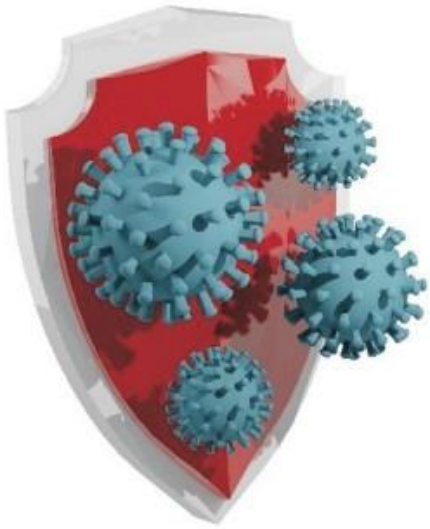

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NATIONAL LIBRARY OF NIGERIA CATALOGUING-IN-PUBLICATION DATA

Tales of Man's Travails and Triumphs as the Battle with Viruses Rages on / by Junaid, A. Surajudeen
1. Viruses
2. Microorganism
I. Fulafia Faculty of Health Science (College of Medicine)
Inaugural Lecture Series No. 20

1. Title

QR 360.J95	2024	579.2
ISBN 978.978.769-052-9		(Pbk)
ISSN (Print) 1115-991X		AACR2
ISSN (Online) 1115-9901		

Publisher:
FULAFIA PRESS,
Federal University of Lafia,
P.M.B 146, Lafia,
Nasarawa State,
Nigeria.

Print @
Castle Mart Hubs,
No. 2, Abonnama Close,
Off Emeka Anyaoku Street,
Area 11, Garki Abuja.

DEDICATION

To Almighty Allah, the beneficent, the merciful And My beloved mother, Hajia Rabi, for good upbringing and your love.

THE PRESENTER

PROF. SURAJUDEEN ALIM JUNAID

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Department of Medical Laboratory Science

Faculty of Health Science

College of Medicine

Federal University of Lafia

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Salutations

The Vice Chancellor, Federal University of Lafia
Members of the Governing Council of the federal University of Lafia
Principal Officers of our great University
Deans and Directors
Members of Senate
All Professors present
Members of the Congregation
Medical Laboratory Science Council of Nigeria
Association of Medical Laboratory Scientists of Nigeria
West African Postgraduate College of Medical Laboratory Science
Invited Friends and Colleagues
Invited Guests
Members of my nuclear and extended family
My Dear Students
Students of Federal University of Lafia
Distinguished Ladies and gentlemen

Preamble

Mr Vice Chancellor, Distinguish Guests, I am here to share a storyline which captures my thoughts around viruses and the immune system. They are thoughts that have dominated my mind at every junction of my academic journey. I hope you find these thoughts relevant to our present travails and successes against emerging and re-emerging viral infections.

Sir, you are aware that man lives in a complex and competitive environment with other biotic and abiotic elements. His survival means victory on many fronts as he is part of an ecosystem where he is either depending on other elements to survive or they are depending on him to survive.

One of these relationships is with microorganisms. These microbes find man as a host which they invade without permission and colonise without tenancy agreement. To be fair to the microorganisms, they mean no harm. They are just on an innocent expedition to survive and perpetuate themselves. Unfortunately, the presence and activities of these organisms in humans, animals and plants cause injury, disease and in most cases, eventual death.

Viruses are a group of microorganisms that can only grow and multiply in living cells. Their presence brings the immune system to live. This is where the battle begins as the immune players begin to demand answers from the intruding viruses with attendant collateral damage. This is the remote cause of disease and can manifest with several symptoms depending on cells, organs, tissues and systems that are involved.

Interestingly, man and other living things do not go down without a fight when invaded by viruses. Nature has endowed man with capabilities to actively recognise incursion and invasion by viruses and other pathogens. Through a systematic and orderly event, the body mounts a formidable front that goes after the intruders to decapitate and destroy them once recognised as foreign bodies.

Consequently, man is in an unending battle with viruses as he encounters them daily through the air, he breaths, the food and water he takes, contact with man and fomite or through practices that breach the skin or lifestyles that allow exchange of body fluids from infected individuals or materials to susceptible hosts. In many cases, the formidable immune system through their active surveillance and patrol of the body, round up these pathogens and destroy them before they establish themselves to cause harm. In other cases, the viruses are so crafty that they overwhelm or outsmart the immune system and cause significant havocs, leading to severe disability and eventual death.

It is a battle that begins from the womb against the foetus and spans throughout lifetime with man developing strategic fronts to fight for survival.

This lecture gives a clear story of this battle and why it still rages on now than ever before.

CHAPTER ONE

1.0 INTRODUCTION

Mr Vice Chancellor sir, it will be right to begin this story by telling us what a virus is. A succinct definition of a virus has it that it is an ultramicroscopic, obligate intracellular parasite that is not capable of self-replication. It thus depends on its host replication machinery to produce its kind and has either DNA or RNA as its genome.

Other authors have defined it as a chain of nucleic acids (DNA or RNA) which lives in a host cell, uses parts of the cellular machinery to reproduce, and releases the replicated nucleic acid chains to infect more cells. Fig 1.0 below represents a typical viral particle.

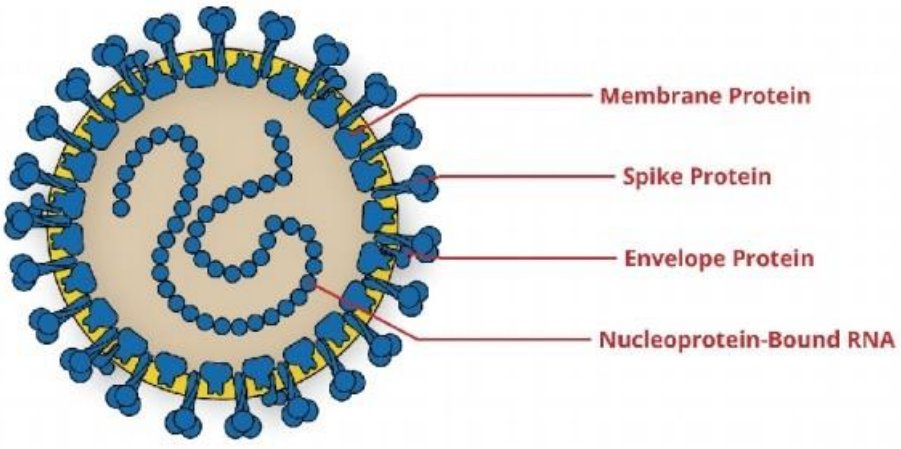


Fig 1.0: A typical virus structure

It is instructive to highlight that the earliest indications of the biological nature of viruses came from studies in 1892 by the Russian scientist Dmitry I. Ivanovsky and in 1898 by the Dutch scientist Martinus W. Beijerinck, Beijerinck first surmised that the virus under study was a new kind of infectious agent, which

he designated *contagium vivumfluidum*, meaning that it was a live, reproducing organism that differed from other organisms. Both investigators found that a disease of tobacco plants could be transmitted by an agent, later called tobacco mosaic virus, passing through a minute filter that would not allow the passage of bacteria. This virus and those subsequently isolated would not grow on an artificial medium and were not visible under the light microscope. In independent studies in 1915 by the British investigator Federick W. Twort and in 1917 by the French-Canadian scientist Felix H. d'Herelle, lesions in cultures of bacteria were discovered and attributed to an agent called bacteriophage ("eater of bacteria"), now known to be viruses that specifically infect bacteria. (Wagner and Krug, 2024).

The unique nature of these agents meant that new methods and alternative models had to be developed to study and classify them. The study of viruses confined exclusively or largely to humans, however, posed the formidable problem of finding a susceptible animal host. In 1933 the British investigators Wilson Smith, Christopher H. Andrewes, and Patrick P. Laidlaw were able to transmit influenza to ferrets, and the influenza virus was subsequently adapted to mice. In 1941 the American scientist George K. Hirst found that influenza virus grown in tissues of the chicken embryo could be detected by its capacity to agglutinate (draw together) red blood cells.

A significant advancement was made by the American scientists John Enders, Thomas Weller, and Frederick Robbins, who in 1949 developed the technique of culturing cells on glass surfaces; cells could then be infected with the viruses that cause polio (poliovirus) and other diseases. (Until this time, the poliovirus could be grown only in the brains of chimpanzees or the spinal cords of monkeys). Culturing cells on glass surfaces opened the way for diseases caused by viruses to be identified by their effects on cells (cytopathogenic effect) and by the presence of antibodies to them in the blood. Cell culture then led to the

development and production of vaccines (preparations used to elicit immunity against a disease) such as the poliovirus vaccine (Wagner and Krug, 2024).

1.1 Viral Strategy for the Battle

Mr Chairman, someone may want to imagine how a virus particle as simple as it appears, has remained topical on discussion agenda when infectious diseases are being discussed or when pandemics hit with the most recent, the SARS-CoV2.

a. Ultramicroscopic:

By being ultramicroscopic, it means that man is up against an enemy that he cannot readily see with the unaided eyes. What you can see is what you can evidently avoid or attack. But viruses represent a group of microorganisms that cannot be seen with the ordinary light microscope. They can only be seen by electron microscopes whose resolution power is far stronger. The smallest virus has a diameter of approximately 20nm while the biggest is approximately 300nm. The strategy here is to come in obscurity and deal a deadly blow.

b. Obligate intracellular parasite:

As an obligate intracellular parasite, the virus lives and multiplies only within living cells. It lives at the expense of its host, its presence causing significant disruption to biological and metabolic activities within the cells and by extension organs and tissues.

c. Not Capable of Self Replication:

The inability of the viral particle to have a replication machinery of its own but having the prowess to ambush and take over the replication mechanism of the host cell and commandeer it to make viral proteins instead of host cell proteins represents an enemy that is armed to the teeth, a case of a tenant taking over from the landlord.

1.2 The Battle Line

a. The Virus Armoury: Viral Pathogenesis

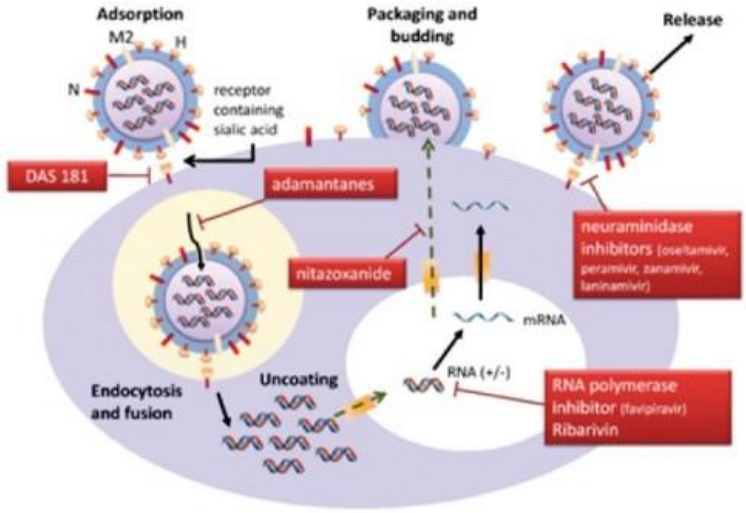


Fig 2.0: Virus armoury for infectivity

Mr Chairman, Fig 2.0 above shows the endowments that a virus has. They help it to locate a susceptible cell, invade it, defy its resistance and overwhelm it by comfortably surviving and multiplying inside it with the cell eventually getting destroyed in the process.

Following my curiosity about viruses and their ability to access and infect human cells and cause diseases, I embarked on explorative research to demonstrate and study this phenomenon to generate verifiable evidence as a scientist. In one study, I studied the invasion of rotaviruses in children and how the immune system fights back to eliminate the viruses in the body (Junaid *et al.*, 2011a). In another study, I delved into exploring the factors that aid abet the transmission and eventual assault of these viruses on susceptible cells (Junaid *et al.*, 2014).

Viral pathogenesis is the study of the process and mechanisms by which viruses cause diseases in their target hosts, often at the cellular or molecular level. It is a specialized field of study in virology (Nathanson, 2016).

Pathogenesis is a qualitative description of the process by which an initial infection causes disease (Albrecht *et al.*, 1996). Viral disease is the sum of the effects of viral replication on the host and the host's subsequent immune response against the virus (Racaniello, 2014). Viruses can initiate infection, disperse throughout the body, and replicate due to specific virulence factors (Albrecht *et al.*, 1996).

There are several factors that affect pathogenesis. Some of these factors include virulence characteristics of the virus that is infecting. To cause disease, the virus must also overcome several inhibitory effects present in the host. Some of the inhibitory effects include distance, physical barriers and host defences. These inhibitory effects may differ among individuals due to the inhibitory effects being genetically controlled.

Viral pathogenesis is affected by various factors: (1) transmission, entry and spread within the host, (2) tropism, (3) virus virulence and disease mechanisms, (4) host factors and host defence (Ryan and Ray, 2014).

b. Mechanism of Viral Infection

Viruses need to establish infections in host cells to multiply. For infections to occur, the virus must hijack host factors and evade the host immune response for efficient replication. Viral replication frequently requires complex interactions between the virus and host factors that may result in deleterious effects in the host, which confers the virus its pathogenicity (Morse *et al.*, 2019).

c. Important Steps of Virus Life Cycle that Shape Pathogenesis

Important Steps of Virus Life Cycle that Shape Pathogenesis

- Transmission from a host with an infection to a second host
- Entry of the virus into the body
- Local replication in susceptible cells
- Dissemination and spread to secondary tissues and target organs
- Secondary replication in susceptible cells
- Shedding of the virus into the environment
- Onward transmission to third host

1.3 Primary transmission

Three requirements must be satisfied to ensure successful infection of a host. Firstly, there must be enough virus available to initiate infection. Cells at the site of infection must be accessible, in that their cell membranes display host-encoded receptors that the virus can exploit for entry into the cell, and the host anti-viral defence systems must be ineffective or absent (Racaniello, 2014; Morse *et al*., 2019).

i. Entry to the Host

Viruses causing disease in humans often enter through the mouth, nose, genital tract, or through damaged areas of skin, so cells of the respiratory, gastrointestinal, skin and genital tissues are often the primary site of infection (Ryan and Ray, 2014; Mitchell, 2010). Some viruses are capable of transmission to a mammalian fetus through infected germ cells at the time of fertilization, later in pregnancy via the placenta, and by infection at birth (Albrecht, 1996).

ii. Local Spread

Following initial entry to the host, the virus hijacks the host cell machinery to undergo viral amplification. Here, the virus must

modulate the host innate immune response to prevent its elimination by the body while facilitating its replication. Replicated virus from the initially infected cell then disperse to infect neighbouring susceptible cells, possibly with spread to different cell types like leukocytes. This results in a localised infection, in which the virus mainly spreads and infects adjacent cells to the site of entry (Morse *et al*., 2019). Otherwise, the virus can be released into extracellular fluids. Examples of localised infections include common cold (rhinovirus), flu (parainfluenza), gastrointestinal infections (rotavirus) or skin infections (papillomavirus) (Albrecht, 1996).

a. Dissemination and Secondary Replication

In other cases, the virus can cause systemic disease through a disseminated infection spread throughout the body. The predominant mode of viral dissemination occurs through the blood or lymphatic system, some of which include viruses responsible for chickenpox (varicella zoster virus), smallpox (variola), HIV (human immunodeficiency virus). A minority of viruses can disseminate via the nervous system (Ryan and Ray, 2014). Notably, the poliovirus can be transmitted via the fecal-oral route, where it initially replicates in its site of entry, the small intestine and spread to regional lymph nodes. Then, the virus disseminates via the bloodstream into different organs in the body (e.g. liver, spleen), followed by a secondary round of replication and dissemination into the central nervous system to damage motor neurons.

1.4 Disease Mechanisms: How Viruses Cause Diseases

A viral infection does not always cause disease. A viral infection simply involves viral replication in the host, but disease is the damage caused by viral multiplication. An individual who has a viral infection but does not display disease symptoms is known as a carrier (Furuya-Kanamori, 2016).

a. Damage Caused by the Virus

Once inside host cells, viruses can destroy cells through a variety of mechanisms. Viruses often induce direct cytopathic effects to disrupt cellular functions (Fuentes-González *et al.,* 2013; Cann 2015). This could be through releasing enzymes to degrade host metabolic precursors, or releasing proteins that inhibit the synthesis of important host factors, proteins, DNA and/or RNA. Namely, viral proteins of herpes simplex virus can degrade host DNA and inhibit host cell DNA replication and mRNA transcription (Kumar *et al.*, 2014). Poliovirus can inactivate proteins involved in host mRNA translation without affecting poliovirus mRNA translation. In some cases, expression of viral fusion proteins on the surface of the host cells can cause host cell fusion to form multinucleated cells. Notable examples include measles virus, HIV, respiratory syncytial virus (MacLachlan and Dubovi, 2011).

Importantly, viral infections can differ by the "lifestyle strategy". Persistent infections happen when cells continue to survive despite a viral infection and can be further classified into latent (only the viral genome is present, there is no replication occurring) and chronic (basal levels of viral replication without stimulating an immune response). In acute infections, lytic viruses are shed at high titres for rapid infection to a secondary tissue/host, whereas persistent viruses undergo shedding at lower titres for a longer duration of transmission (months to years) (Flint *et al*., 2019).

Lytic viruses are capable of destroying host cells by incurring and/or interfering with the specialised functions of host cells. An example would be the triggering of necrosis in host cells infected with the virus. Otherwise, signatures of viral infection, like the binding of HIV to co-receptors CCR5 or CXCR4, can also trigger cell death via apoptosis through host signalling cascades by immune cells. However, many viruses encode proteins that can modulate apoptosis depending on whether the infection is

acute or persistent. Induction of apoptosis, such as through interaction with caspases, will promote viral shedding for lytic viruses to facilitate transmission, while viral inhibition of apoptosis could prolong the production of virus in cells, or allow the virus to remain hidden from the immune system in chronic, persistent infections. Nevertheless, induction of apoptosis in major immune cells or antigen-presenting cells may also act as a mechanism of immunosuppression in persistent infections like HIV. The primary cause of immunosuppression in HIV patients is due to the depletion of CD4+ T helper cells.

Interestingly, adenovirus has an E1A protein to induce apoptosis by initiating the cell cycle, and an E1B protein to block the apoptotic pathway through inhibition of caspase interaction (Dimmock *et al*., 2016).

Persistent viruses can sometimes transform host cells into cancer cells. Viruses such as the human papillomavirus (HPV), human T-lymphotropic virus (HTLV) can stimulate growth of tumours in infected hosts, either by disrupting tumour suppressor gene expression (HPV) or upregulating proto-oncogene expression (HTLV).

b. Damage Caused by Host Immune System

Sometimes, instead of cell death or cellular dysfunction caused by the virus, the host immune response can mediate disease and excessive inflammation. The stimulation of the innate and adaptive immune system in response to viral infections destroys infected cells, which may lead to severe pathological consequences to the host. This damage caused by the immune system is known as virus-induced immunology (Sehrawat *et al*., 2010).

Specifically, immunopathology is caused by the excessive release of antibodies, interferons and pro-inflammatory

cytokines, activation of the complement system, or hyperactivity of cytotoxic T cells. Secretion of interferons and other cytokines can trigger cell damage, fever and flu-like symptoms. In severe cases of certain viral infections, as in avian H5N1 influenza in 2005, aberrant induction of the host immune response can elicit a flaring release of cytokines known as a cytokine storm (Tisoncik, 2012).

In some instances, viral infection can initiate an autoimmune response, which occurs via different proposed mechanisms: molecular mimicry and bystander mechanism. Molecular mimicry refers to an overlap in structural similarity between a viral antigen and a self-antigen. The bystander mechanism hypothesizes the initiation of a non-specific and overreactive antiviral response that tackles self-antigens in the process. Damage caused by the host itself due to autoimmunity was observed in the West Nile virus (Hawkes, 2018).

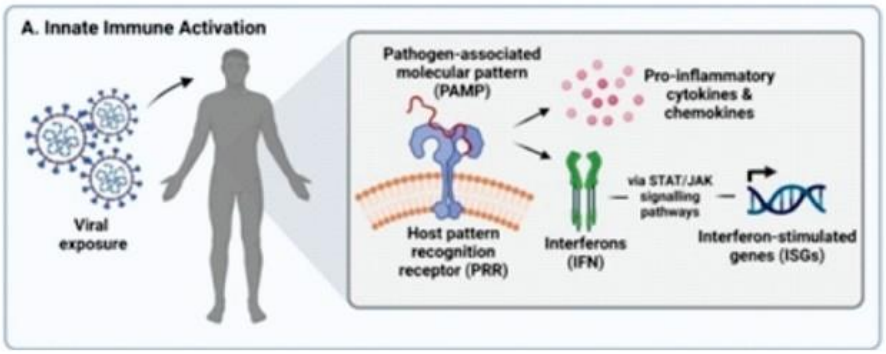
CHAPTER TWO

2.0 THE IMMUNE SYSTEM

Mr Chairman, ladies and gentlemen, we have seen the virus and its potentials to cause harm. It is important to see how the immune system is endowed to give the viruses a fight in equal or greater measure.

The immune system fights germs on the skin, in the tissues of the body, and in bodily fluids such as blood. It is made up of the innate (general) immune system and the adaptive (specialized) immune system. These two systems work closely together and take on different tasks. I designed and executed research initiatives that revealed the adaptive immune response in the face of Hepatis E virus invasion in both humans and animals and the dynamics of the immune response to the challenge showed the intense battle between viruses and humans (Junaid *et al.,* 2014a)

Figure 3.0 below shows the scope of the immune system.



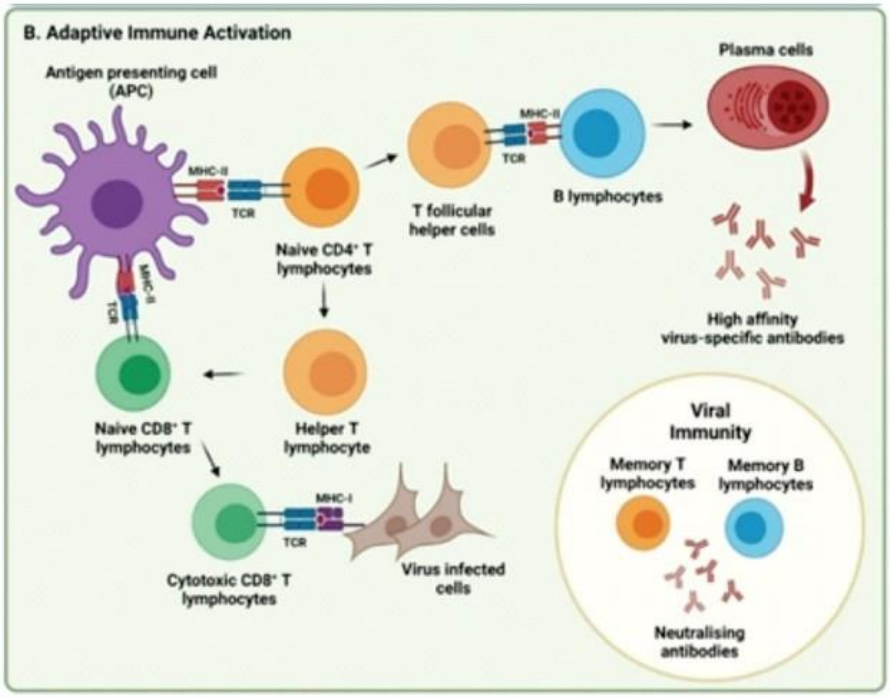


Fig. 3.0: Sketch of the major divisions of the immune system

2.1 Innate Immune System

The innate immune system is the body's first line of defence against intruders. It responds in the same way to all germs and foreign substances, which is why it is sometimes referred to as the "non-specific" immune system. It acts very quickly – for instance, it makes sure that bacteria that have entered the skin through a small wound are detected and destroyed on the spot within a few hours. But the innate immune system can't always stop germs from spreading.

The innate immune system provides

- protection offered by the skin and mucous membranes
- protection offered by immune system cells and proteins

a. Protection by Skin and Mucous Membrane

All outer and inner surfaces of the human body are a key part of the innate immune system. The closed surface of the skin and mucous membrane already forms a physical barrier that stops germs from entering. On top of that, substances like acid, enzymes and mucus prevent bacteria and viruses from growing. Certain movements in the body also stop germs from settling – for example, movements of hair-like structures (cilia) in the lungs or movements of the bowel muscles. Some fluids in the body have a similar effect – including tear fluid, sweat and urine (which flushes the organs of the urinary system.

b. Protection by Immune Cells and Proteins

If germs get past the skin or mucous membranes and enter the body, the innate immune system fights them using special immune system cells and proteins.

If, for example, an area of skin becomes infected, immune system cells spring into action – either by moving to the area or by being activated locally. Certain cells of the immune system release substances to make the blood vessels wider and more "leaky." This causes the area around the infection to swell, become warm and turn red – visible signs of the inflammation that has developed. A fever may develop too. Then the blood vessels get wider and even more immune system cells arrive to fight the infection.

Bacteria or viruses that enter the body can be stopped right away by phagocytes, also known as scavenger cells. These special white blood cells (leukocytes) enclose germs and "digest" them, making them harmless. The remains of the germs move to the surface of the phagocytes, where they can be detected by the adaptive immune system.

There are also other types of immune system cells that release substances to kill bacteria and other germs. But it's not only the germs that die – tissue cells and immune system cells die and break down too. Together, their remains form a yellowish fluid called pus.

c. The role of proteins

Several proteins (enzymes) help the cells of the innate immune system. A total of nine different enzymes activate each other in a kind of chain reaction: One enzyme in the first stage alerts several enzymes in a second stage, each of which activates several enzymes in a third stage, and so on. This allows the immune response to grow stronger very quickly.
The tasks of these enzymes include:

- marking germs as targets for phagocytes,
- attracting other immune system cells from the bloodstream,
- destroying bacteria cell walls to kill the bacteria, and
- fighting viruses by destroying the viral envelope (the outermost layer of a virus) or cells that have been infected with viruses.

d. Natural killer cells: Searching for body cells that have changed

The natural killer cells are the third major part of the innate immune system. Their main job is to identify cells that have been infected by a virus, as well as abnormal cells that may turn into (or have turned into) tumor cells. To do this, they search for cells with an abnormal surface, and then destroy the cell surface using substances called cytotoxins.

2.2 The Acquired Immune System

If the innate (general) immune system fails to destroy the germs, the adaptive (specialized) immune system takes over. The adaptive immune system specifically targets the type of germ

that is causing the infection. But to do that, it first needs to recognize the germ as such. This means that it's slower to respond than the innate immune system, but it's more accurate when it does respond. It also has the advantage of being able to "remember" germs. So the next time the adaptive immune system faces a germ it has already met, it can start fighting the germ faster.

This memory is also the reason why there are some illnesses you can only get once in your life, because afterwards your body becomes “immune” to them. It may take a few days for the adaptive immune system to respond the first time it meets the germ, but the next time the body can react immediately. The second infection then usually goes unnoticed or is at least milder.

The adaptive immune system is made up of:

- T cells in the tissue between the body's cells
- B cells (also in the tissue between the body's cells)
- Antibodies in the blood and other bodily fluid

2.2.1 T Cells

T cells (also called T lymphocytes) are made in bone marrow. They travel in the bloodstream to the thymus, where they mature. The "T" in their name comes from “thymus."
T cells have three main jobs:

- They use chemical messengers to activate other cells of the immune system, starting the adaptive immune system response (T helper cells).
- They detect tumor cells or cells that have been infected by viruses and destroy them (cytotoxic T cells).
- Some T helper cells become memory T cells after the infection has cleared up. They "remember" the germ that was fought off and are then ready to activate the adaptive immune system quickly if the body is infected by the same germ again.

T cells have specific features (receptors) on their surfaces that germs can attach to – like a lock that one particular key will fit. The immune system can make a matching T cell type for each germ within a few days of infection.

Then if a germ attaches to a matching T cell, the T cell starts to multiply – making more T cells that can specifically fight that germ. Because only the cells that match the germ multiply, the immune response is "tailor-made."

2.2.2 B Cells

B cells (B lymphocytes) are made in the bone marrow, where they mature into specialized immune system cells. They take their name from the "B" in "bone marrow." Like the T cells, there are many different types of B cells that match particular germs.

B cells are activated by T helper cells: T helper cells send signals to B cells that match the same germs as they do. This stimulates the B cells to make copies of themselves and turn into plasma cells. The plasma cells quickly make very large amounts of antibodies and release them into the blood. Because the T helper cells only activate the B cells that match the attacking germs, the body only makes the exact antibodies that are needed.

Some of the activated B cells turn into memory cells and become part of the "memory" of the adaptive immune system.

The different cells of the adaptive immune system communicate either directly or through soluble chemical messengers such as cytokines (usually proteins). These chemical messengers are made by various cells in the body.

2.2.3 Antibodies

Antibodies (proteins with sugar groups attached to them) travel around the body in the bloodstream. They are made by the

immune system to fight germs and foreign substances. Antibodies can quickly recognize germs and other potentially harmful substances, and then attach to them. This makes the "intruders" harmless and attracts other immune system cells to help. Antibodies are made by B cells. Germs and substances that can trigger the production of antibodies are called "antigens."

An antibody only attaches to an antigen if it matches exactly, like a key in the lock of the antibody. In this way, antibodies recognize matching germs and trigger the fast response of the adaptive immune system.

Antibodies have three main functions:

- They make germs harmless – for example, by directly attaching to the cell surface of viruses or bacteria, or by binding harmful substances made by these germs. This prevents the germs from latching onto normal body cells and infecting them.
- They activate other immune system cells by attaching to their surface. Also, it's much easier for phagocytes to fight off germs that have a lot of antibodies attached to them.
- They activate proteins that help in the immune system response.

So, the antibodies of the adaptive immune system also help the innate immune system to do its job.

CHAPTER THREE

3.0 THE SPECTRUM OF VIRUS SHAPES

Mr Chairman, it will interest you to know that viruses come in many different shapes and sizes, but all are made of two essential components: a core of genetic material, either DNA or RNA, which is surrounded by a protective protein coat called a capsid. Packaged together, a single virion comes in four different shapes: helical, polyhedral, spherical, and complex (Fig 4.0)

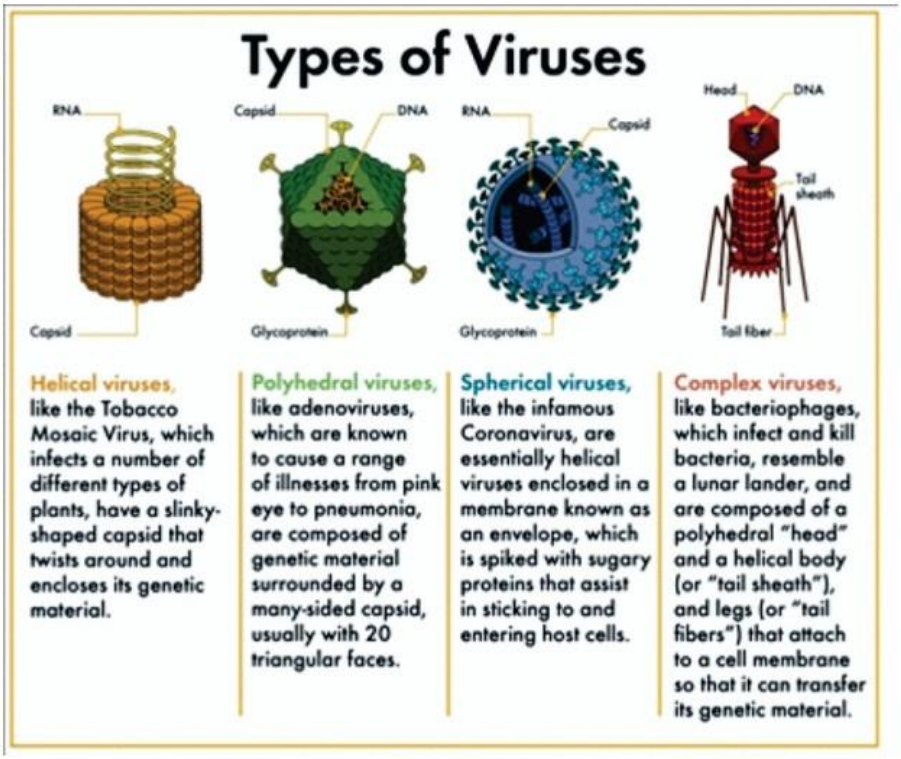


Fig 4.0 Spectrum of virus shapes

The exact structure of a virus is dependent upon which species serves as its host. A virus which replicates in mammalian cells will have a protein coat which enables it to attach to and infiltrate

mammalian cells. The shape, structure, and function of these proteinschanges depending on the species of virus.

The above virus shows the typical structure a virus takes, a viral genome surrounded by a shield of proteins. The various envelope proteins will enable the virus to interact with the host cell it finds. Part of the protein coat will then open, puncture through the cell membrane, and deposit the viral genome within the cell. The protein coat can then be discarded, as the viral genome will now replicate within the host cell. The replicated virus molecules will be packaged within their own protein coats and be released into the environment to find another host. While many virus particles take a simple shape like the one above, some are much more complicated.

The above image shows a *phage*, a type of virus which specializes on bacterial cells. The protein coat of a phage is much more complex and has a variety of specialized parts. The head portion contains the viral genome. The collar, sheath, base plate, and tail fibres are part of an intricate system to attach to and inject the genome into a bacterial cell. The tail fibres grasp the bacterial cell, pulling the base plate up to the cell wall or membrane. The sheath and collar compress, puncture the cell, and deposit the DNA into the bacterial cell.

Some virus molecules have no protein coat whatsoever or have never been identified making on. In some plant virus species, the virus is passed from cell to cell within the plant. When seeds are created within the plant, the virus spreads to the seeds. In this way the virus can live within cells its entire existence, and never need a protein coat to protect it in the environment. Other virus molecules have even larger and more complex protein coats and specialize on various hosts.

3.1 Are Viruses Living Things?

Mr Chairman, there has been a debate as to whether viruses are living or non-living things. This is a complicated question. A cell is living because it contains all the necessary components to replicate its DNA, grow, and divide into new cells. This is the process all life takes, where it is a single-celled organism or a multi-cellular organism. Some people do not consider a virus living because a virus does not contain all the mechanisms necessary to replicate itself. They would say that a virus, without a host cell, cannot replicate on its own and is therefore not alive.

Yet, by the definition of life laid out before, it seems that when a virus is inside of a host cell it does have all the machinery it needs to survive. The protein coat it exists in outside of a cell is the equivalent of a *bacterial spore*, a small capsule bacteria form around themselves to survive harsh conditions. Scientists who support a virus being a living organisms note the similarity between a virus in a protein coat and a bacterial spore. Neither organism is active within their protective coat, they only become active when they reach favourable conditions.

In fact, the only reason a virus affects us at all is because it becomes active within our cells. Further, a virus tends to evolve with its host. Most dangerous viruses have just recently jumped to a new species. The biochemistry they evolved to live within the other species is not compatible with the new species, and cell damage and death occur. This causes a number of reactions, depending on which cells were infected. The HIV virus, for instance, attacks immune cells exclusively. This leads to a total loss of immune function in patients. With the virus causing the common cold, the virus attacks respiratory cells and damages them as it does its work.

Yet, not all virus infections will be detrimental to the host. A virus that kills the host will be less successful over time,

compared to a virus which doesn't harm the host. A healthy host increases the number of virus molecules released into the environment, which is the goal of the virus. In fact, some virus particles may benefit the host. A good example is a form of herpes virus, found in mice. This virus, while it is infecting a mouse, provides the mouse with a good defence against the bacteria which carry the plague. While the mechanism is not clear, the virus somehow prevents the bacteria from taking hold in the mouse's system.

When viewed in this light, it is easy to see how a virus is very similar to bacteria. The bacteria create and maintains the tools needed to reproduce DNA, where the virus steals them. This is the only real difference between a virus and a bacterium. Because of this, many scientists consider a virus a living organism. Scientists who study viruses, *virologists*, note that virus particles (alive or not) have been evolving with life probably as long as the first cells were present. Because of this, there is a virus which specializes on almost every single species on the planet.

From my research stables, we adsorbed viruses on maize offal as vaccine seed. They served as vehicle for administration of viral vaccines to poultry. Though dry and with elevated temperatures, the viruses were only inactive by not inactivated as they elicited immunologic responses once administered to chicken (Echeonwu *et al*., 2011). The findings added a dimension to debate as to whether viruses are living or non-ling things.

3.2 Virus Host Range

Mr Chairman, what makes viruses a global concern is their host range. The host range, a key property of viruses, reflects the diversity of species that viruses can naturally infect. To be a member of the exclusive "*host range*" club of a virus, the host should support the replication or life cycle of the virus; i.e., the

virus should be able to successfully enter a host cell and complete a series of tasks, including unencapsidation and replication in the initial cell and movement to adjacent cells and throughout the host. Nonetheless, it is often difficult to define the host range of a virus as a number of other factors need to be incorporated, such as the host susceptibility to infection, as well as the ability of the virus to undergo sustained transmission from host to host. Moreover, the actual breadth of the host range can be reduced by barriers that prevent contact between vectors and hosts, and the unsynchronized seasonal timing between (1) available infected hosts in a viremic stage and feeding activity of vectors and (2) available uninfected species and infectious vectors in each environment. And it can be expanded. Depending on the virus, the the range may expand to secondary hosts and/or vectors, as a result of selection pressure on the virus generated either in vectors or in hosts as well as of the degree of promiscuity of host seeking behaviour of the vectors involved. Moreover, other viruses, such as rhabdoviruses, replicate in their insect vectors. Thus, the majority of known rhabdovirus species have two natural hosts, either insects and plants, or insects and vertebrates. The virus host range is also expanded when there are "*spillover*" infections into alternative hosts. In some instances, viruses gain the ability to spread efficiently to a new host that was not previously exposed or susceptible. These transfers involve either increased exposure or the acquisition of genetic variations that allow the virus to overcome barriers to infection of the new host. Phylogenetic studies suggest that these host shifts are frequent in the evolution of most pathogens, but why viruses successfully jump between some host species but not others is only just becoming clear.

For these reasons, the host range is highly variable among viruses. Some, such as dengue and mumps viruses, who's only known mammalian host are humans, are referred to as specialist viruses. These viruses have evolved to become specialized in

infecting one or very few host species. In contrast, generalist viruses successfully infect hosts from different species and even hosts from a higher taxonomical rank. Examples of generalist viruses include *Cucumber mosaic virus* (CMV; *Bromoviridae*), which infects more than 1000 plant species, and *Influenza A virus* (*Orthomyxoviridae*), which infects birds and several different species of mammals. Opinions have been divided regarding the evolutionary significance of host range variation. Some argue that host–virus relationships with a narrow, specific host range are more advanced, while others posit the opposite. The advantages of generalism are more obvious: a generalist virus would be able to exploit multiple hosts and thus enhance its fitness. Since generalist viruses are not the norm, it is generally assumed that generalism comes with a cost. It has also been suggested that evolution should favour specialists because evolution proceeds faster with narrower niches. The answers may lie with genome sequencing. Through genome sequencing, it is now apparent that a variety of organisms carry genes that reflect past infection events by viruses. By using data from multiple potential host species, it may be possible to determine whether extant viruses characterized as presently having a broad host range have been resident in the genomes of their hosts for longer times than current viruses with a narrow host range. Such evidence may suggest that families of viruses with broad host ranges are more evolutionarily ancient and may have benefited from a greater ability to avoid extinction. Also, it is increasing clear that understanding the evolution and biology of a species cannot be achieved without examining the interactions between the members of the holobiont; i.e., the prokaryotic symbionts, the eukaryotic symbionts, the viruses, and the host. Metagenomics, coupled with biological studies, promise further characterization of these host–virus interactions.

3.3 Virus Life Cycle

Mr Chairman, this is the battle ground. All viruses depend on

cells for reproduction and metabolic processes. By themselves, viruses do not encode for all the enzymes necessary for viral replication. But within a host cell, a virus can commandeer cellular machinery to produce more viral particles. Bacteriophages replicate only in the cytoplasm, since prokaryotic cells do not have a nucleus or organelles. In eukaryotic cells, most DNA viruses can replicate inside the nucleus, with an exception observed in the large DNA viruses, such as the poxviruses, that can replicate in the cytoplasm. With a few exceptions, RNA viruses that infect animal cells replicate in the cytoplasm. An important exception that will be highlighted later is Influenza virus.

a. Life cycle of Viruses with prokaryotic Host

The life cycle of bacteriophages has been a good model for understanding how viruses affect the cells they infect, since similar processes have been observed for eukaryotic viruses, which can cause immediate death of the cell or establish a latent or chronic infection. Virulent phages typically lead to the death of the cell through cell lysis. Temperate phages, on the other hand, can become part of a host chromosome and are replicated with the cell genome until such time as they are induced to make newly assembled viruses, or progeny viruses.

b. The Lytic Cycle

During the lytic cycle of virulent phage, the bacteriophage takes over the cell, reproduces new phages, and destroys the cell. T-even phage is a good example of a well-characterized class of virulent phages. There are five stages in the bacteriophage lytic cycle.

Attachment is the first stage in the infection process in which the phage interacts with specific bacterial surface receptors (e.g., lipopolysaccharides and OmpC protein on host surfaces). Most phages have a narrow host range and may infect one species of

bacteria or one strain within a species. This unique recognition can be exploited for targeted treatment of bacterial infection by phage therapy or for phage typing to identify unique bacterial subspecies or strains. The second stage of infection is entry or penetration. This occurs through contraction of the tail sheath, which acts like a hypodermic needle to inject the viral genome through the cell wall and membrane. The phage head and remaining components remain outside the bacteria.

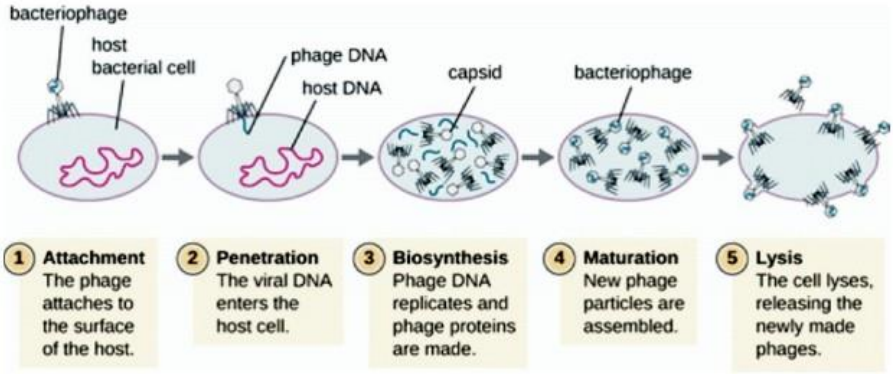


Fig 5.0: Virus Lytic Cycle

The third stage of infection is biosynthesis of new viral components. After entering the host cell, the virus synthesizes virus-encoded endonucleases to degrade the bacterial chromosome. It then hijacks the host cell to replicate, transcribe, and translate the necessary viral components (capsomeres, sheath, base plates, tail fibers, and viral enzymes) for the assembly of new viruses. Polymerase genes are usually expressed early in the cycle, while capsid and tail proteins are expressed later. During the maturation phase, new virions are created. To liberate free phages, the bacterial cell wall is disrupted by phage proteins such as holin or lysozyme. The final stage is release. Mature viruses burst out of the host cell in a process called lysis and the progeny viruses are liberated into the environment to infect new cells.

a. The Lysogenic Cycle

In a lysogenic cycle, the phage genome also enters the cell through attachment and penetration. A prime example of a phage with this type of life cycle is the lambda phage. During the lysogenic cycle, instead of killing the host, the phage genome integrates into the bacterial chromosome and becomes part of the host. The integrated phage genome is called a prophage. A bacterial host with a prophage is called a lysogen. The process in which a bacterium is infected by a temperate phage is called lysogeny. It is typical of temperate phages to be latent or inactive within the cell. As the bacterium replicates its chromosome, it also replicates the phage's DNA and passes it on to new daughter cells during reproduction. The presence of the phage may alter the phenotype of the bacterium, since it can bring in extra genes (e.g., toxin genes that can increase bacterial virulence). This change in the host phenotype is called lysogenic conversion or phage conversion. Some bacteria, such as *Vibrio cholerae* and *Clostridium botulinum*, are less virulent in the absence of the prophage. The phages infecting these bacteria carry the toxin genes in their genome and enhance the virulence of the host when the toxin genes are expressed. In the case of *V. cholera*, phage encoded toxin can cause severe diarrhoea; in *C. botulinum*, the toxin can cause paralysis. During lysogeny, the prophage will persist in the host chromosome until induction, which results in the excision of the viral genome from the host chromosome. After induction has occurred the temperate phage can proceed through a lytic cycle and then undergo lysogeny in a newly infected cell.

d. Transduction

Transduction occurs when a bacteriophage transfers bacterial DNA from one bacterium to another during sequential infections. There are two types of transduction: generalized and specialized transduction. During the lytic cycle of viral

replication, the virus hijacks the host cell, degrades the host chromosome, and makes more viral genomes. As it assembles and packages DNA into the phage head, packaging occasionally makes a mistake. Instead of packaging viral DNA, it takes a random piece of host DNA and inserts it into the capsid. Once released, this virion will then inject the former host's DNA into a newly infected host. The asexual transfer of genetic information can allow for DNA recombination to occur, thus providing the new host with new genes (e.g., an antibiotic-resistance gene, or a sugar-metabolizing gene). Generalized transduction occurs when a random piece of bacterial chromosomal DNA is transferred by the phage during the lytic cycle. Specialized transduction occurs at the end of the lysogenic cycle, when the prophage is excised, and the bacteriophage enters the lytic cycle. Since the phage is integrated into the host genome, the prophage can replicate as part of the host. However, some conditions (e.g., ultraviolet light exposure or chemical exposure) stimulate the prophage to undergo induction, causing the phage to excise from the genome, enter the lytic cycle, and produce new phages to leave host cells. During the process of excision from the host chromosome, a phage may occasionally remove some bacterial DNA near the site of viral integration. The phage and host DNA from one end or both ends of the integration site are packaged within the capsid and are transferred to the new, infected host. Since the DNA transferred by the phage is not randomly packaged but is instead a specific piece of DNA near the site of integration, this mechanism of gene transfer is referred to as specialized transduction. The DNA can then recombine with host chromosome, giving the latter new characteristics. Transduction seems to play an important role in the evolutionary process of bacteria, giving them a mechanism for asexual exchange of genetic information.

CHAPTER FOUR

4.0 LIFE CYCLE OF VIRUSES WITH ANIMAL HOST

Lytic animal viruses follow similar infection stages to bacteriophages: attachment, penetration, biosynthesis, maturation, and release. However, the mechanisms of penetration, nucleic-acid biosynthesis, and release differ between bacterial and animal viruses. After binding to host receptors, animal viruses enter through endocytosis (engulfment by the host cell) or through membrane fusion (viral envelope with the host cell membrane). Many viruses are host specific, meaning they only infect a certain type of host; and most viruses only infect certain types of cells within tissues. This specificity is called a tissue tropism. Examples of this are demonstrated by the poliovirus, which exhibits tropism for the tissues of the brain and spinal cord, or the influenza virus, which has a primary tropism for the respiratory tract.

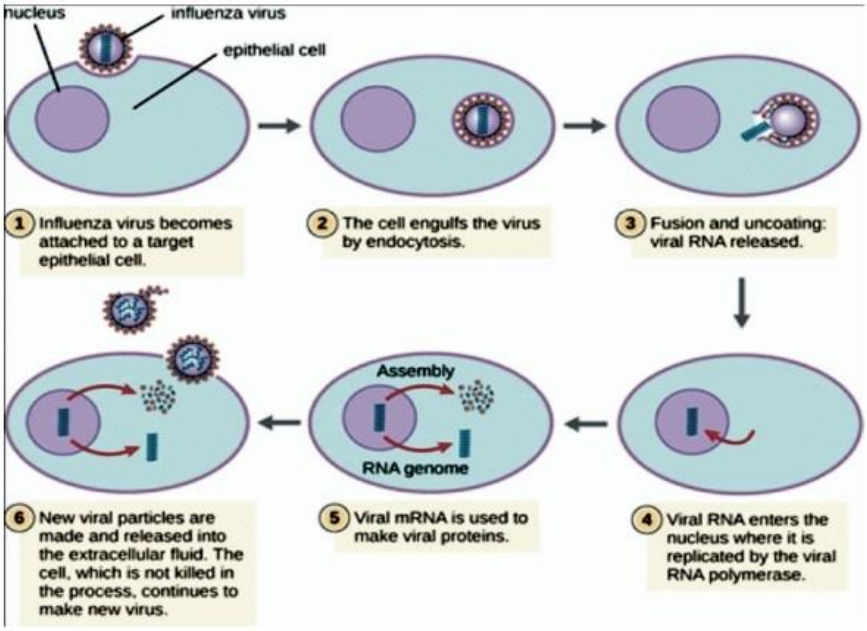
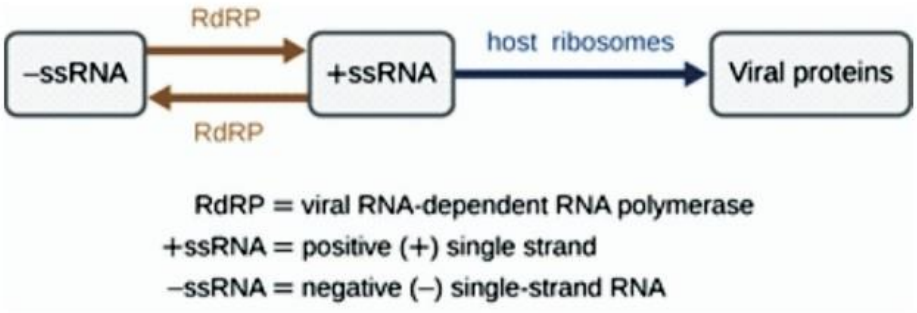


Fig 6.0 Virus replication in humans

Animal viruses do not always express their genes using the normal flow of genetic information—from DNA to RNA to protein. Some viruses have a dsDNA genome like cellular organisms and can follow the normal flow. However, others may have ssDNA, dsRNA, or ssRNA genomes. The nature of the genome determines how the genome is replicated and expressed as viral proteins. If a genome is ssDNA, host enzymes will be used to synthesize a second strand that is complementary to the genome strand, thus producing dsDNA. The dsDNA can now be replicated, transcribed, and translated similar to host DNA.

If the viral genome is RNA, a different mechanism must be used. There are three types of RNA genome: dsRNA, **positive (+) single-strand (+ssRNA)** or **negative (-) single-strand RNA (-ssRNA)**. If a virus has a +ssRNA genome, it can be translated directly to make viral proteins. Viral genomic +ssRNA acts like cellular mRNA. However, if a virus contains a -ssRNA genome, the host ribosomes cannot translate it until the -ssRNA is replicated into +ssRNA by viral RNA-dependent RNA polymerase (RdRP). The RdRP is brought in by the virus and can be used to make +ssRNA from the original -ssRNA genome. The RdRP is also an important enzyme for the replication of dsRNA viruses, because it uses the negative strand of the double-stranded genome as a template to create +ssRNA. The newly synthesized +ssRNA copies can then be translated by cellular ribosomes.



An alternative mechanism for viral nucleic acid synthesis is observed in the retroviruses, which are +ssRNA viruses. Single-stranded RNA viruses such as HIV carry a special enzyme called reverse transcriptase within the capsid that synthesizes a complementary ssDNA (cDNA) copy using the +ssRNA genome as a template. The ssDNA is then made into dsDNA, which can integrate into the host chromosome and become a permanent part of the host. The integrated viral genome is called a provirus. The virus now can remain in the host for a long time to establish a chronic infection. The provirus stage is similar to the prophage stage in a bacterial infection during the lysogenic cycle. However, unlike prophage, the provirus does not undergo excision after splicing into the genome.

4.1 Persistent Infections

Persistent infection occurs when a virus is not completely cleared from the system of the host but stays in certain tissues or organs of the infected person. The virus may remain silent or undergo productive infection without seriously harming or killing the host. Mechanisms of persistent infection may involve the regulation of the viral or host gene expressions or the alteration of the host immune response. The two primary categories of persistent infections are latent infection and chronic infection. Examples of viruses that cause latent infections include herpes simplex virus (oral and genital herpes), varicella-zoster virus (chickenpox and shingles), and Epstein-Barr virus (mononucleosis). HepatitisCvirus (HCV) and HIV are two examples of viruses that cause long-term chronic infections.

4.2 Latent Infection

Not all animal viruses undergo replication by the lytic cycle. There are viruses that are capable of remaining hidden or dormant inside the cell in a process called latency. These types of viruses are known as latent viruses and may cause latent infections. Viruses capable of latency may initially cause an acute infection before becoming dormant.

For example, the varicella-zoster virus infects many cells throughout the body and causes chickenpox, characterized by a rash of blisters covering the skin. About 10 to 12 days postinfection, the disease resolves and the virus go dormant, living within nerve-cell ganglia for years. During this time, the virus does not kill the nerve cells or continue replicating. It is not clear why the virus stops replicating within the nerve cells and expresses few viral proteins but, in some cases, typically after many years of dormancy, the virus is reactivated and causes a new disease called shingles. Whereas chickenpox affects many areas throughout the body, shingles is a nerve cell-specific disease emerging from the ganglia in which the virus was dormant.

4.3 Chronic Infection

A chronic infection is a disease with symptoms that are recurrent or persistent over a long time. Some viral infections can be chronic if the body is unable to eliminate the virus. HIV is an example of a virus that produces a chronic infection, often after a long period of latency. Once a person becomes infected with HIV, the virus can be detected in tissues continuously thereafter, but untreated patients often experience no symptoms for years. However, the virus maintains chronic persistence through several mechanisms that interfere with immune function, including preventing expression of viral antigens on the surface of infected cells, altering immune cells themselves, restricting expression of viral genes, and rapidly changing viral antigens through mutation. Eventually, the damage to the immune system results in progression of the disease leading to acquired immunodeficiency syndrome (AIDS). The various mechanisms that HIV uses to avoid being cleared by the immune system are also used by other chronically infecting viruses, including the hepatitis C virus.

CHAPTER FIVE

5.0 MY BIRTH AND THE BATTLE AT THE TIME

Mr Chairman, permit me to share how I was birthed into a battle with microbes which would have taken my live like it did for thousands of lives, especially of infants at the time.

I was born at a time, in the heat of the H3N2 strain of Asian flu which spread to Africa in 1969. The air was badly polluted with influenza viruses that claimed the lives of hundreds of thousands of children.

Fig 7.0: Global Map showing the H3N2 Pandemic

The 1968 flu pandemic was a global outbreak of influenza that originated in China in July 1968 and lasted until 1970. The outbreak was the third influenza pandemic to occur in the 20th century; it followed the 1957 flu pandemic and the influenza pandemic of 1918- 1919. The 1968 flu pandemic resulted in an estimated one million to four million deaths, far fewer than the 1918–1919 pandemic, which caused between 25 million and 50 million deaths.

The 1968 pandemic was initiated by the emergence of a virus known as influenza A subtype H3N2. It is suspected that this virus evolved from the strain of influenza that caused the 1957 pandemic. The 1957 pandemic flu virus, or influenza A subtype H2N2, is thought to have given rise to H3N2 through a process called antigenic shift, in which the haemagglutinin (H) antigen (a substance that stimulates an immune response) on the outer surface of the virus underwent genetic mutation to produce the new H3 antigen. Because the new virus retained the neuraminidase (N) antigen N2, persons who had been exposed to the 1957 virus apparently retained immune protection against the 1968 virus. This would explain the mildness of the 1968 outbreak relative to the pandemic of 1918–1919.

Although the 1968 flu outbreak was associated with comparatively few deaths worldwide, the virus was highly contagious, a factor that facilitates its rapid global dissemination. Indeed, within two weeks of its emergence in July in Hong Kong, some 500,000 cases of illness had been reported, and the virus proceeded to spread swiftly throughout Southeast Asia. Within several months it had reached the Panama Canal Zone and the United States, where it had been taken overseas by soldiers returning to California from Vietnam. By the end of December, the virus had spread throughout the United States and had reached the United Kingdom and countries in western Europe. Australia, Japan, and multiple countries in Africa, eastern Europe, and Central and South America were also affected. The pandemic occurred in two waves, and in most places the second wave caused a greater number of deaths than the first wave.

The 1968 flu pandemic caused illness of varying degrees of severity in different populations. For example, whereas illness was diffuse and affected only small numbers of people in Japan, it was widespread and deadly in the United States. Infection

caused upper respiratory symptoms typical of influenza and produced symptoms of chills, fever, and muscle pain and weakness. These symptoms usually persisted for between four and six days. The highest levels of mortality were associated with the most susceptible groups, namely infants and the elderly. Although a vaccine was developed against the virus, it became available only after the pandemic had peaked in many countries.

The H3N2 virus that caused the 1968 pandemic is still in circulation today and is a strain of seasonal influenza. In the 1990s a closely related H3N2 virus was isolated from pigs. Scientists suspect that the human H3N2 virus jumped to pigs; infected animals may show symptoms of swine flu.

The battle line was drawn between me and polluted air with viruses finding susceptible hosts in infants and dealing deadly blows on them.

5.1 The Battle in the Womb

Mr Chairman sir, this battle against viruses begins at conception, right in the womb. This is because pregnant mothers could be carrying latent, chronic or active viral infections, placing foetuses at risk of getting infected.

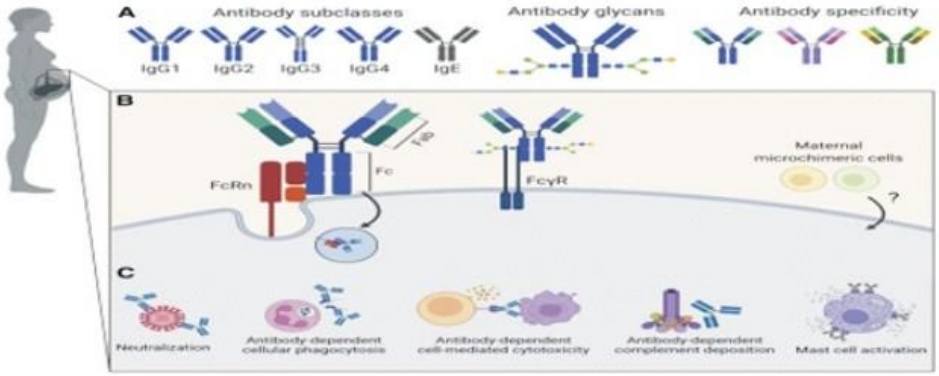
At this stage, the foetus is very vulnerable and completely unequipped to immunologically to face the virus. However, by divine providence, God has made it possible that the placenta which houses the foetus is impervious to viruses and other pathogens. Fig 8.0 below shows how shielded the foetus in because of maternal defence through the placenta

Fig 8.0: Picture of foetus in en utero

The leading viruses in maternal-newborn dyads include adenovirus C, Cytomegalovirus (CMV), Epstein-Barr virus (EBV), Herpes simplex virus 1 (HSV-1), rubella and rhinovirus.

The foetus has no capacity to engage the virus in the battle. The fetus survives the assault by through the defence mechanisms of mother.
In the innate immunity arm of the response, the placenta helps prevent the mother's immune system from rejecting her own foetus by not recognising it as a foreign body. It also prevents microbes from accessing the foetus by surrounding it with an impervious placenta.

Not done, the maternal immune system passively immunises the foetus by vertically transferring some of her antibodies for the mother to use. Throughtranscystosis, the kindness of mothers come to life as she donates majorly IgG and recently IgE throughneonatal Fc receptor (FcRn)maternal immune cells (Maternal microchimeric cells). IgG1 dominates as the most efficiently transferredsubclass, followed by IgG and then IgG2 and IgG4.



(A) Antibody types in transplacental transfer.
(B) Mechanisms of antibody and maternal-microchimeric-cell transplacental transfer.
(C) Effector functions of maternal IgG antibodies

Fig 9: Picture of immune induction pathway at infancy

Battle line: maternal hypergammaglobulinaemiablocks FcRN and interfere with process of mounting response by neonates.

5.2 Post Natal Life

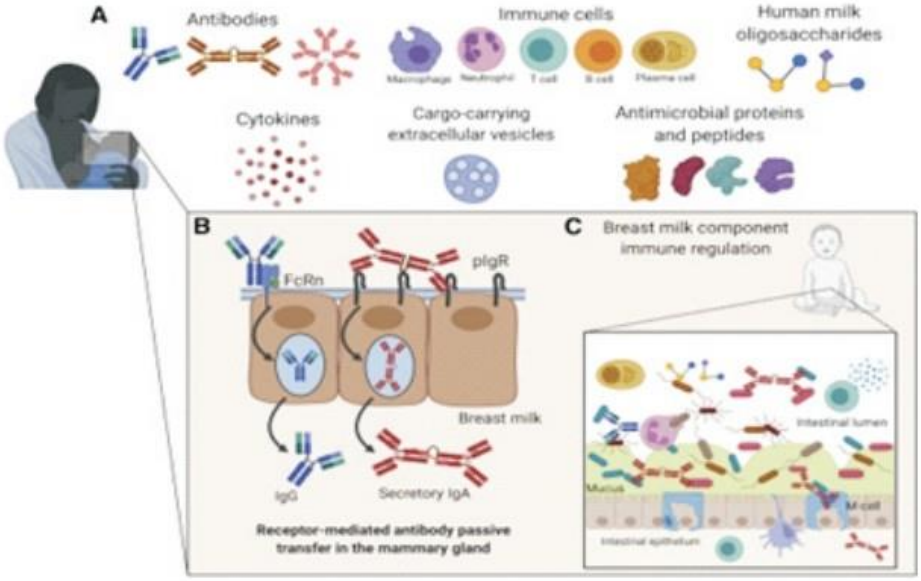


Fig 10: Picture of immune system specialisation pathway

After birth, the milk of kindness from mothers is not just food for nourishment, it is also armoury to fortify infants against viral infections. Colostrum is the early milk from the mother. It is rich in antibodies, majorly IgA, then IgG and IgM. Since the immune organs in infants are yet to take up the responsibility of producing armoury for the fight, the infant fights with the weapons supplied by the mother through breast milk.

Fraction of leucocytes transferred in breast milk to infant, memory B cells and a population of T cells are also transferred.

It has also been observed that exclusive breast feeding reduces diarrheal diseases due to high IgA concentration.

5.3 Induction into the Battle

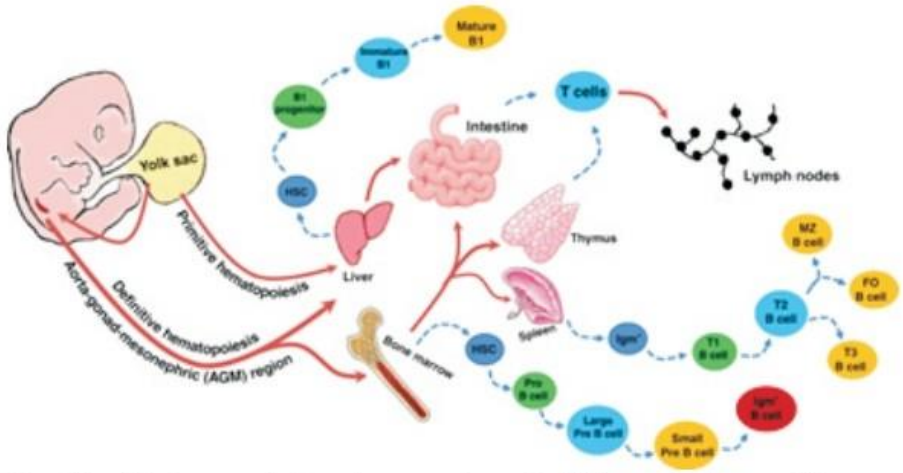


Fig 11: Picture of development and differentiation of immune players in early life

Mr Chairman, by and by, from infancy, we get inducted into this battle. Immune pathway and players in infants begin to develop as they and readied for the battle. The foetal immune system develops and generates both tolerogenic and protective immune responses to tolerate both self- and maternal antigens.

The fetal T cells with pro-inflammatory potential are born in a tolerogenic environment and are tightly controlled by both cell-intrinsic and -extrinsic mechanisms. Fetal B-1 and B-2 B cells involved in innate and adaptive immune responses, respectively. Innate immunity protects against infection while adaptive immunity creates memory after an initial response to a specific pathogen.

I made attempts at generating data from personal research around the battle children have with viruses and how their immune systems rise to the occasion by measuring levels of some secretary antibodies against rubella and rotaviruses, given their significance in early life (Junaid *et al.*, 2011a; Junaid *et al.*, 2011b).

5.4 Victoryfor Vaccines shortly after my Birth

Mr Chairman, I was very young in late 70s when smallpox was eradicated in the world. The feat was achieved through the invention and administration of vaccines.

Smallpox was a highly infectious disease caused by Variola virus (often called smallpox virus), which belongs to the genus Orthopoxvirus. The last naturally occurring case was diagnosed in October 1977, and the World health Organisation (WHO) certified the global eradication of the disease in 1980, making smallpox the only human disease to have been eradicated to date.

The initial symptoms of the disease included fever and vomiting. This was followed by formation of ulcers in the mouth and a skin rash. Over several days, the skin rash turned into the characteristic fluid-filled blisters with a dent in the centre. The bumps then scabbed over and fell off, leaving scars. The disease was transmitted from one person to another primarily through prolonged face-to-face contact with an infected person or (rarely) via contaminated objects. Prevention was achieved

mainly through the smallpox vaccine. Once the disease had developed, certain antiviral medications could potentially have helped, but such medications did not become available until after the disease was eradicated. The risk of death was about 30%, with higher rates among babies. Often, those who survived had extensive scarring of their skin, and some were left blind.

A vaccine is a substance used to stimulate the production of antibodies and provide immunity against one or several diseases, prepared from the causative agent of a disease, its products, or a synthetic substitute, treated to act as an antigen without inducing the disease.

Since the invention of vaccines, they have played significant roles in preventive medicine. They are handy from conception and throughout life with the frequency of administration more at infancy to help all of us win this life battles against pathogens.

My research team has been neck-deep in viral vaccines research, either to develop viable vaccine seeds, improve on vaccine delivery in poultry (Echeonwu *et al*., 2011) or challenges around vaccinations in humans globally (Enitan *et al.*, 2020). The findings have been revolutionary as they highlight why vaccines have become the mainstay in our public health initiatives.

CHAPTER SIX

6.0 SOME GLOBAL PANDEMICS IN MY LIFETIME

6.1 HIV Pandemic (1980-2003)

Mr Chairman, I was a young man full of life within the bracket of the HIV pandemic. Having been graciously assisted by maternal immune system to survive virus assaults at infancy, I was responsible enough to take my destiny in my hands while the HIV pandemic lasted. Many young men at my age were not so lucky as they fell by the rampaging sword of the virus.

The said pandemic started in the period October 1980–May 1981, 5 young men, all active homosexuals, were treated for biopsy-confirmed *Pneumocystis carinii* pneumonia at 3 different hospitals in Los Angeles, California. Two of the patients died. All 5 patients had laboratory-confirmed previous or current cytomegalovirus (CMV) infection and candidal mucosal infection. Case reports of these patients follow." The five patients described in this historic US Centers for Disease Control and Prevention (CDC) report on June 5, 1981, were previously healthy gay men aged 29–36 years. Their illnesses and deaths marked the dark dawn of the recognition of AIDS. The HIV/AIDS pandemic has now been with us for four decades and at least 32 million lives have been lost.

HIV infection is caused by a retrovirus that infects and replicates in human lymphocytes and macrophages, eroding the integrity of the human immune system over a number of years, culminating in immune deficiency and a susceptibility to a series of opportunistic and other infections as well as the development of certain malignancies.

At the initial consultation with the medical practitioner, an infected patient may be at any stage of the natural history from acute to chronic infection, ranging from asymptomatic through

to severely unwell. The initial assessment is key for prognosis and formulation of short- to long-term management plans.

AIDS (a syndrome of a constellation of infections, conditions, or malignancies) occurs because of HIV infection, and usually develops over 10-15 years.?

This battle was partly against unbridled sexual urges and largely against the immune players, particularly the CD4 cells.

6.2 The CD4 Cells and HIV

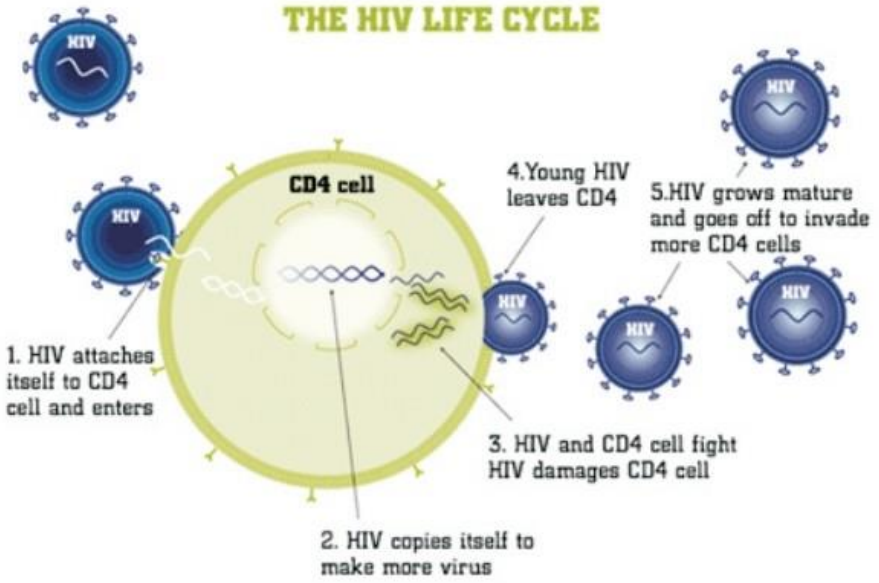


Fig 12: The CD4 cell HIV infection

CD4+ T-cells are the central mediators of immune response in humans, crucially coordinating cellular and humoral immune responses against infections. Very early studies on subjects with

AIDS documented lymphopenia, low lymphocyte proliferative responses after stimulation with antigens, and an inversion in the ratio of helper T-cells to cytotoxic T-cells (Ei *et al,* 1995). Further studies in this line confirmed that HIV selectively infects CD4+T- cells and destroys them for its own benefits (Nashimura *et al*., 2005). Later, it was shown that suppressing HIV replication with antiretroviral therapy (ART) rapidly increased peripheral blood CD4+ T-cell counts and reversed immunodeficiency (Nowak *et al*., 1997). Now, most researchers agree that HIV majorly infects CD4+ T-cells and leads to progressive loss of the cells from circulation and from the total body stores.

Upon *in vitro* infection with HIV, productive infection of CD4+ T-cells takes place and leads to either cell lysis or giant cell/syncytia formation, in which both infected and uninfected cells fuse, leading to spread of infection. Animal models of SIV infection also documented severe depletion of CD4+ T-cells in the gut-associated lymphoid tissue (GALT), which is the major producer of CD4+ T-cells in the body. Subsequent studies provided evidence that the same phenomenon of depletion of GALT CD4 reservoirs occurs in human HIV infection as well Quantitative estimates of absolute CD4+ T-cell count, and percentage have been shown to correlate strongly with the progression of disease. A normal adult harbour about 22?×?10^{11} CD4+ T-cells, whereas in the HIV-infected individual, this number is halved by the time the peripheral blood CD4+ T-cell count falls to 200?cells/microliter of blood. In more advanced disease, destruction of parenchymal lymphoid spaces is so extensive that enumeration of the total body CD4+ T-cell count cannot even be attempted. Since HIV induces both quantitative and qualitative defects in the CD4+ T-cell compartment, numbers of circulating CD4+ T-cells in HIV+ subjects have been the most widely used tool for predicting the onset of overt immunodeficiency and the best surrogate marker for monitoring severity of the disease (Okoye and Picker, 2013).

6.2 The SARS Pandemic of 2002-2003

Mr Chairman sir, another pandemic I have witnessed in my lifetime is the Severe Acute Respiratory Syndrome(SARS). SARS is a viral respiratory disease caused by a SARS-associated coronavirus. It was first identified at the end of February 2003 during an outbreak that emerged in China and spread to 4 other countries.

SARS is an airborne virus and can spread through small droplets of saliva in a similar way to the cold and influenza. It was the first severe and readily transmissible new disease to emerge in the 21st century and showed a clear capacity to spread along the routes of international air travel.

SARS can also be spread indirectly via surfaces that have been touched by someone who is infected with the virus.

Most patients identified with SARS were previously healthy adults aged 25–70 years. A few suspected cases of SARS have been reported among children under 15 years. The case fatality among persons with illness meeting the current WHO case definition for probable and suspected cases of SARS is around 3%.

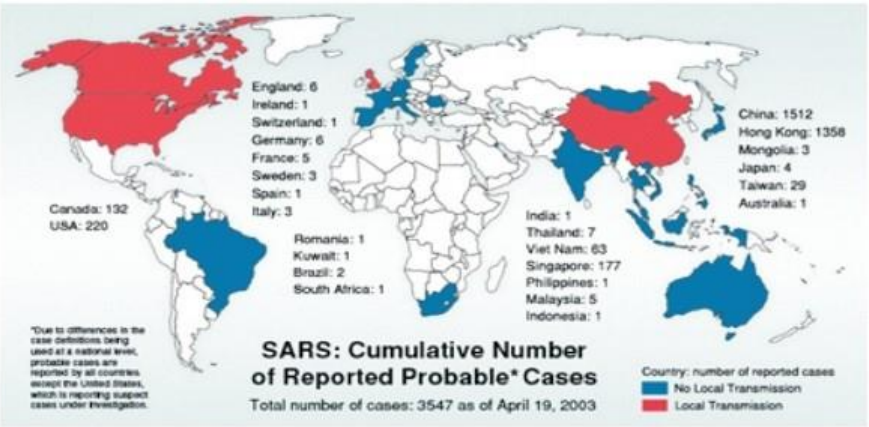


Fig 13: Map showing SARS Pandemic in 2003

In November 2002, doctors in the Guangdong province of southeastern China began to see the first cases of what would become known as SARS, or severe acute respiratory syndrome. Over the next several months, 8,096 people in 26 countries contracted the new viral illness, leading to 774 deaths. Although the slow reporting of initial SARS cases helped the illness spread, globally enforced medical practices eventually helped end the outbreak.

The reasons for the slow reporting of SARS are complicated. Doctors had never seen the viral illness before, and at first, those in Guangdong province thought the SARS cases they were seeing might be atypical pneumonia.

This pandemic saw viruses draw a battle line between man and animal reservoirs which are either a tourist attraction, a bush meat delicacy or an integral element in the ecosystem.

6.2.1 Role of Animals in Disease Epidemics

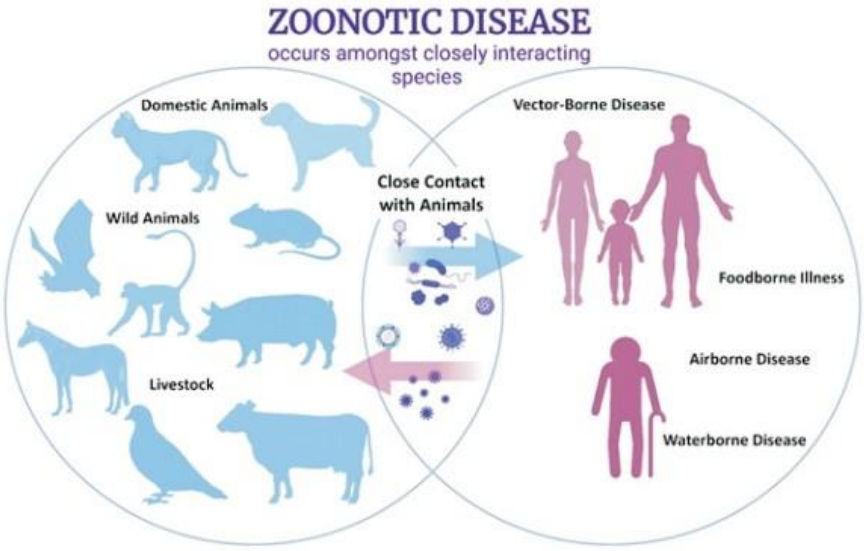


Fig 14: Picture of virus escape from animals to humans and vice versa

Mr chairman, the question on many minds, not least the epidemiologists and public health experts struggling to contain SARS, was: From where did SARS come? Over the course of the outbreak, epidemiological guesses about SARS' origin were made. Although, at the time of this writing, none of the guesses had been definitively proved, the leading hypothesis is that the SARS virus was transmitted from an animal species to humans somewhere in Guangdong Province, China. Species-jumping pathogens are nothing new in the world of public health. Many of the great disease scourges of human history began when viruses or bacteria were transmitted from animal hosts to human populations. Tuberculosis is, for example, believed to have jumped from animals to humans during the process of human domestication of livestock. Many experts also think that HIV/AIDS originated in simian or primate species before jumping into humans sometime in the twentieth century. The eventual identification of Guangdong Province as the origin of the species-jumping SARS virus also came as no great surprise for epidemiologists. The southern region of China has long been considered a particularly potent microbial incubator. Guangdong Province is, for example, 'famous for its "wet markets," where a bewildering variety of live fauna are offered for sale (sometimes illegally) for the medicinal properties or culinary potential. The opportunity for contact, not only with farmed animals but also with a variety of otherwise rare or uncommon wild animals, is enormous'

This region of China is also important to global surveillance efforts on influenza because of the role experts believe the region's animal–human milieu plays in nurturing strains of the influenza virus. The southeast Asian region had also been the location of two previous scary but ultimately limited viral outbreaks – the H5N1 avian influenza outbreak in Hong Kong in 1997 and the Nipah virus outbreak in 1998–99 in Malaysia.

The H5N1 virus spread from birds to humans, and the Nipah virus spread from pigs to people. Because neither the H5N1 virus nor the Nipah virus developed efficient human-to-human transmission, the outbreaks remained limited in scope and impact. The H5N1 and Nipah viruses constituted, however, warnings that species-jumping viruses were jumping and potentially dangerous. Public health experts have kept an eye on southern China and southeast Asia as a possible, if not the probable, source of the long-anticipated, killer pandemic influenza virus. In late May 2003, WHO reported that researchers in Hong Kong and Shenzhen, China announced they had detected several viruses closely related genetically to the SARS virus in two wild animal species, the masked palm civet and the raccoon-dog. The researchers also found antibodies to the SARS virus in another wild animal species, the Chinese ferret badger

According to WHO (2003j-2), 'these and other wild animals are traditionally considered delicacies and are sold for human consumption in markets throughout southern China.' These studies prompted researchers to posit in June 2003 'that the earliest cases of SARS, in Guangdong Province, China, may have had contact, during slaughter or due to proximity to so-called "wet" markets, with certain wild animals species consumed as delicacies in southern China' (WHO, 2003). At the time of this writing, scientists still had not proved or disproved these hypotheses about the origin of SARS. As WHO (2003u-2) argued in June 2003, additional studies are urgently needed before any firm conclusions can be reached. Answers to these questions will also greatly assist predictions of the future evolution of SARS.

6.3 The Ebola Outbreak of 2014

Fig 15: Map of Ebola virus pandemic

Mr Chairman, not long ago, we witnessed the Ebola virus pandemic which stood out as the fastest spreading infectious agent in history.

Ebola first appeared in 1976 in two simultaneous outbreaks, one in what is now Nzara, South Sudan, and the other in Yambuku, Democratic Republic of the Congo. The latter occurred in a village near the Ebola River, from which the disease takes its name.

The virus family Filoviridae includes 3 genera: Cuevavirus, Marburgvirus, and Ebolavirus. Within the genus Ebolavirus, 6 species have been identified: Zaire, Bundibugyo, Sudan, Taï Forest, Reston and Bombali.

It is thought that fruit bats of the *Pteropodidae* family are natural Ebola virus hosts. Ebola is introduced into the human population through close contact with the blood, secretions, organs or other bodily fluids of infected animals such as fruit bats, chimpanzees, gorillas, monkeys, forest antelope or porcupines found ill or dead or in the rainforest.

Ebola then spreads through human-to-human transmission via direct contact (through broken skin or mucous membranes) with:

- blood or body fluids of a person who is sick with or has died from Ebola; and
- objects that have been contaminated with body fluids (like blood, feces, vomit) from a person sick with Ebola or the body of a person who died from Ebola.

Health-care workers have frequently been infected while treating patients with suspected or confirmed Ebola. This occurs through close contact with patients when infection control precautions are not strictly practiced.

Burial ceremonies that involve direct contact with the body of the deceased can also contribute to the transmission of Ebola.
The symptoms of Ebola infection can be sudden and include fever, fatigue, muscle pain, headache and sore throat. These are followed by vomiting, diarrhoea, rash, and internal and external bleeding.

The time from when someone gets infected to having symptoms is usually from 2 to 21 days. A person with Ebola can only spread the disease once they have symptoms. People can spread Ebola for as long as their body contains the virus, even after they have died.

The battle line was in its transmission rate. The 2013–2016 epidemic of Ebola virus disease, centred in West Africa, was the most widespread outbreak of the disease in history. It caused major loss of life and socioeconomic disruption in the region, mainly in Guinea, Liberia and Sierra Leone. The first cases were recorded in Guinea in December 2013; the disease spread to neighbouring Liberia and Sierra Leone, with minor outbreaks occurring in Nigeria and Mali. Secondary infections of medical

workers occurred in the United States and Spain. Isolated cases were recorded in Senegal, the United Kingdom and Italy. The number of cases peaked in October 2014 and then began to decline gradually, following the commitment of substantial international resources.

Fig 16: picture of response strategy

Given the rapid spread of the virus at the time, it was a battle between the virus and human movement especially at the global front. It asked questions on global health security at entry and exit ports across nations.

6.4 The SARS COV-2 Pandemic of 2019

Mr Chairman, we will all not forget the 2020 in a hurry as tiny virus particles locked us down in our homes. We were scared to go out to the streets and became scared and suspicious of each other. We disconnected physically from ourselves in a weird battle to contain the rampaging onslaught of viruses.

In late December 2019, several health facilities in Wuhan, in Hubei province in China, reported clusters of patients with

pneumonia of unknown cause. Similarly to patients with SARS and MERS, these patients showed symptoms of viral pneumonia, including fever, cough and chest discomfort, and in severe cases dyspnea and bilateral lung infiltration. Among the first 27 documented hospitalized patients, most cases were epidemiologically linked to Huanan Seafood Wholesale Market, a wet market located in downtown Wuhan, which sells not only seafood but also live animals, including poultry and wildlife. According to a retrospective study, the onset of the first known case dates to 8 December 2019. On 31 December, Wuhan Municipal Health Commission notified the public of a pneumonia outbreak of unidentified cause and informed the World Health Organization (WHO).

Fig 17: Timeline of SARS-CoV2 Pandemic

The first recorded cases were reported in December 2019 in Wuhan, China. Over the course of the following 10 months, more than 30 million cases have been confirmed worldwide. COVID-19, coronavirus disease 2019; ICTV, International Committee on Taxonomy of Viruses; PHEIC, public health emergency of international concern; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; WHO, World Health Organization.

Rattled by the pandemic, my team and I sought to understand the role of international travels in the SARS-Cov2 pandemic (Enitan *et al.*, 2020b)

6.5 Impact of 2019 Pandemic on Health Systems

The Coronavirus disease 2019 (COVID-19), the global pandemic, causing Severe Acute Respiratory Syndrome Corona virus -2 (SARS CoV-2), has devastated the world resulting in several million infections and close to five million deaths till late October 2021.

Though COVID-19 pandemic has affected all sectors directly or indirectly, the crisis is worse on the already overburdened health systems in many countries. A significant level of health service delivery was impacted, especially, during the early times owing to the steady spread of the virus across all settings. It posed challenge on health human resource management, facility utilization and medical supply management.

Evidence indicates that the COVID-19 pandemic has made significant disruption in health service delivery particularly in resource-limited countries. The disruption is not only due to the direct effects of the COVID-19 pandemic but also it pressurized the health systems and stretched others beyond their capability indirectly. The COVID-19 pandemic has exposed the existing gaps in the health system. The COVID-19 pandemic has disrupted both preventive and curative services for communicable and noncommunicable diseases. Many of essential services have been delayed by the healthcare facilities, patients were also unable to attend follow-ups and acute care visits due to the fear and anxiety they experienced during the pandemic waves.

The COVID-19 pandemic, in addition to the direct disease burden, it posed a significant risk of indirect morbidity and

mortality from other preventable and treatable diseases as a result of essential health services disruption. The most common reasons mentioned for critical gaps or reducing services during COVID-19 were shifting of health care workers to support COVID-19 services, cancellations of planned treatments, decrease in public transport, loss of income to pay for services and limit utilization and high rates of morbidity and mortality among health care workers, were another reason leading to staff shortages. Many countries have reported shortage of medicines, diagnostics and other technologies as the main reasons for disruption of services.

Similarly, in Ethiopia, following the first COVID-19 reported case in March 2020, the health system was challenged heavily. The ever-increasing COVID-19 cases demanded reshuffling health care workers and repurposing health care facilities. On the other hand, patients with various disease conditions shy away from visiting health facilities. Comparison of the pre-COVID-19 era service utilization with the COVID-19 period showed that there was a substantial disparity in service delivery practice of the health facilities. A huge decline in the patient flow for routine services was noted.

The COVID-19 challenge passed strong message to the world on the need of building resilient and sustainable health system. In doing so, strong investment to strengthen the health systems including the health workforce development, creating a decent working condition, providing training and equipment, especially in relation to personal protective equipment and occupational safety is required. Social dialogue is essential to building resilient health systems, and therefore has a critical role both in crisis response and in building a future that is prepared for health.

Countries that invest in research and innovation of products ranging from vaccines to drugs and to test kits stand a big chance of surviving pandemics of these proportion. Along with my

research team, we also shifted our attention to viral drug development initiatives for possible trials and packaging against viral agents (Chollom *et al*., 2022).

CHAPTER SEVEN

7.0 IMMUNE RESPONSE IN THE ELDERLY

Mr Chairman, I have given you a rundown of how we survived viral assaults from infancy through several junctions in life. Age is catching up with us and again, these viral agents do not respect age. So how is the immune system in the elderly built? It has been described as immunosenescence, a situation where the vibrancy of the immune system begins to lose sensitivity and response strategy to match the virus firepower.

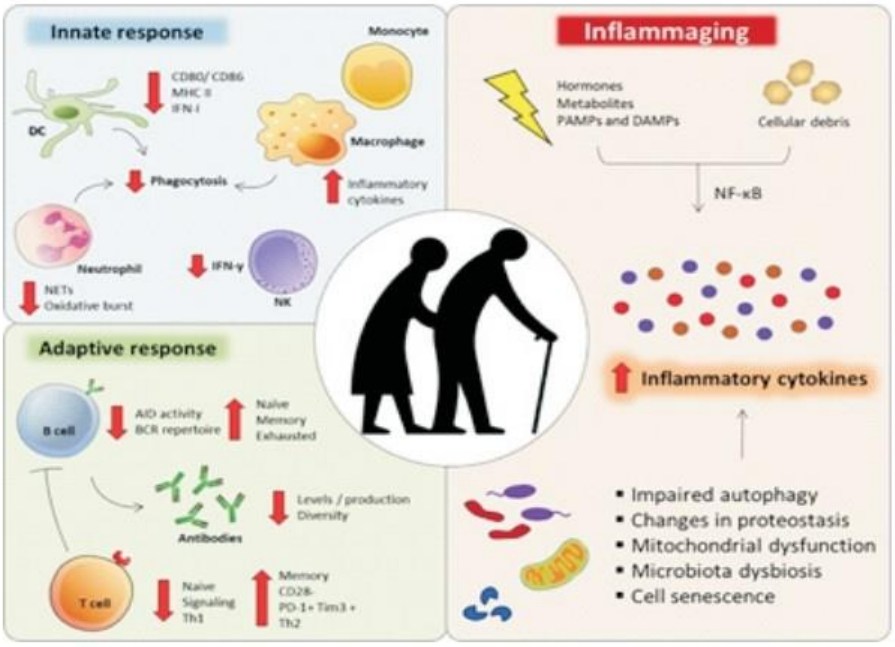


Fig 18: Picture of Immune response in the elderly

The aging process can be understood as a progressive and natural decrease in the biological functions of an organism. Despite its enormous plasticity and capacity for renewal, the immune system is also affected during the aging process. Since a functional immune response is essential for maintaining homeostasis and health, the immune aging process, called

immunosenescence, contributes to the increased susceptibility to infections, cancers and autoimmune diseases.

A very striking feature of the immunosenescence process is a low-grade proinflammatory state, with an increase in serum inflammatory mediators, such as IL-6, IL-1RA, TNF-α, IL-1, and C-reactive protein (CRP). This low-grade inflammatory state named "inflammaging" is associated with the diminished ability to mount efficient immune responses during the aging process.

Inflammaging is caused by a set of hormonal, metabolic and immune factors that constantly provide stimuli that are recognized by innate receptors, favouring an inflammatory environment. In addition, senescent cells commonly experience changes in their intracellular homeostasis, including telomeric perturbations and oxidative stress, leading to the activation of signalling pathways such as nuclear factor κB (NF-κB) and increased secretion of cytokines, chemokines, growth factors and lipids. This condition in which senescent cells change their secretory phenotype is called the senescence-associated secretory phenotype (SASP) and is a potential contributor to inflammaging. The exacerbated inflammatory process associated with age may also be due to a failure to resolve inflammation since many regulatory factors are also deficient in older individuals.

The inflammatory stimuli that support the phenomenon of inflammaging can be triggered by several factors, including chronic infections and microbiota changes, which are going to be more detailed further in this text. However, sterile components naturally produced during cell cycle can also contribute to this phenomenon. Cellular debris resulting from the cell death process that occurs daily due to chemical and physical stresses as well as the accumulation of metabolic

products and cellular catabolic products, such as lipofuscins and beta-amyloid proteins play a crucial role in inflammaging. Under the physiological conditions of cell proliferation, such components are usually diluted between dividing cells. However, as the cell proliferation rate reaches its lowest levels due to aging, these molecules accumulate and can be recognized by pattern recognition receptors (PRRs).

In addition, infectious processes during aging can further accentuate the inflammatory condition by releasing pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs). During cytomegalovirus (CMV) infection, which infects 40–100% of the population worldwide, inflammatory mediators such as prostaglandin E2, IL-6 and TNF-α are released, highlighting the important contribution of this pathogen to inflammaging. However, a 10-year longitudinal study compared the impact of CMV infection on the serum levels of inflammatory cytokines in 249 individuals and showed that cytokine production in CMV-seropositive and CMV-seronegative individuals is similar

7.1 Why this Battle will Continue to Rage on

Mr Chairman, we have come a long way in this battle, but there is no end in sight yet. Three fundamental, interrelated factors fuel the microbial comeback, experts say. Across the globe, people are abandoning the countryside for life in the city, leading to rapid, unplanned urban expansions. In crowded conditions with limited access to health care and poor sanitation, pathogens like Ebola, Zika and influenza enjoy lush opportunities to spread. With more infections mingling, there are also more opportunities for pathogens to share their virulence genes.

At the same time, global demand for meat has quadrupled over the last five decades by some estimates, driving the spread of industrial livestock farming techniques that can allow benign

microbes to become more virulent. The use of colistin in livestock agriculture in China, for example, has been associated with the emergence of *mcr-1*, which was first discovered during routine surveillance of food animals there. Genetic analyses suggest that siting factory farms full of chickens and pigs in proximity to wild waterfowl has played a role in the emergence of highly virulent strains of avian influenza. Crosses of Asian and North American strains of avian influenza caused the biggest outbreak of animal disease in U.S. history in 2014–2015. Containing that virus required the slaughter of nearly 50 million domesticated birds and cost over $950 million. Worryingly, some strains of avian influenza, such as H5N1, can infect humans.

Since we can rarely develop drugs and vaccines fast enough to stanch the most dangerous waves of disease, early detection will be key moving forward. Researchers have developed a welter of models and pilot programs showing how environmental cues such as temperature and precipitation fluctuations and the insights of wildlife and livestock experts can help pinpoint pathogens with pandemic potential before they cause outbreaks in people. Chlorophyll signatures, a proxy for the plankton concentrations that are associated with cholera bacteria, can be detected from satellite data, potentially providing advance notice of cholera outbreaks.

Even social media chatter can be helpful. Innovative financing methods, such as the World Bank's recently launched Pandemic Emergency Financing Facility?—?a kind of global pandemic insurance policy funded by donor countries, the reinsurance market and the World Bank?—?could help ensure that resources to isolate and contain new pathogens are readily available, wherever they take hold. Right now, emerging disease expert Peter Daszak points out, “we wait for epidemics to emerge and then spend billions on developing vaccines and drugs.” The non-

profit organization that Daszak directs, EcoHealth Alliance, is one of a handful that instead aim to detect new pathogens at their source and proactively minimize the risk of their spread.

Burnet died in 1985, two years after the discovery of HIV, one of the first of the latest wave of new pathogens. His vision of a contagion-free society was that of a climber atop a foothill surrounded by peaks, mistakenly thinking he'd reached the summit. The challenge of surviving in a world of pathogens is far from over. In many ways, it's only just begun.

CONCLUSION

Mr Chairman, I hope I have not bored you with this storyline of our survival against viruses. That we are here today, means that we are victorious against them. I dare say that we all have been invaded by these destroyers in many ways than few, but our immune system has always risen to the occasion to overwhelm and flush them out of our systems.

Unfortunately, not everyone has been lucky to win this battle. We recall with pains the millions of children that died due to viral infections before the advent of vaccines aged 0-5years. We remember many that still die or get incapacitated today by vaccine preventable diseases due to either lack of awareness, lack of accessibility to quality vaccines or due to other factors.

It is clear from the storyline that the immune system is adequately built to overwhelm the assault of viruses but at certain times, the viruses reinvent themselves in form of genetic mutation where they acquire more weaponry to subdue the immune system, cause disease and death, through ingenuity, God has given scientists the ability to augment and support the immune system through the development of vaccines for prevention and drugs for therapy. I have shown from the

foregoing how my research team and I have laboured in search for vaccines and drug candidates for use in man and animals to win this battle against viral diseases (Echeonwu *et al*., 2011; Chollom *et al*., 2022)

While we accept that some of them are hiding in secluded places in our bodies in latency, waiting for an opportunity to strike when our immune system wanes, we must remember to indulge in lifestyles that do not weaken our immune systems. From what we consume, to the physical exercises that we undertake to adhering to routine vaccine schedules and observing clear preventive strategy protocols, we must be determined to keep our armoury intact to confront viruses and other pathogenic organisms that are on the rampage

Indeed, we have seen that the battle is between man and viruses, but we have also seen that this battle is aided and abetted by several factors and actors in the ecosystem as depicted from some of my research output (Ndako *et al* 2011; Junaid *et al* 2014b). The quality of water we take is important as this could expose us to more battles against polio virus, hepatitis E virus, rotavirus and many others. Similarly, the quality of air that we breath in could expose us to infections like influenza and parainfluenza viruses. Also, contact with blood and blood or other body fluids could initiate a battle with HIV, hepatitis B and hepatitis C viruses amongst others. Quite aware of how closely knitted man is with animals in the ecosystem, I dedicated some of my research endeavors to researching on animal and human viruses as well as made significant efforts in vaccine and drug development in that light (Echeonwu *et al*., 2011; Junaid *et al*., 2014; Chollom *et al*., 2022).

The passion to know more about the relationship between man and viruses made me to take the challenge of doctoral research on hepatitis E virus. The rigors of this research took me to South Africa where I could access current technologies to study the

virus more closely. Through that journey, I appreciated more the interrelationship between animal, human and environmental health as captured in the one health philosophy. Hepatitis E affects both humans and animals, with the transmission circle aided by poor sanitary measures and lapses in personal or community hygiene. My PhD research titled seroprevalence and molecular characterisation of hepatitis E virus in Plateau state clearly paints the picture. It is therefore important to emphasise that there will never be healthy humans where there are sick animals as there is no human health with animal and environmental health.

While we do our best to keep away from these viruses, we cannot guarantee that they will stay away from us. As such, we must keep a consistent regime of routine medical checks and religiously follow treatment protocols whenever prescribed as our immune system cannot do it all alone.

RECOMMENDATIONS

The highlight on this battle has revealed what we need to do to improve our chances in winning this battle.

Since this battle keeps evolving and re-emerging generation after generation, we certainly cannot de-emphasise the need for continuous effort in unravelling how the viruses reinvent themselves and how they have sustained themselves to stay relevant in spite of changing environmental factors like climate change. We must also know the remote causes and triggers of pandemics and their regimes if we must win this war. Our curiosity must also take us further into unpacking the viral proteome and study it alongside human proteome to see why the virus has often succeeded in some instances to defeat the array of defensive and offensive arsenals that man is made of.

The continuous search for extraneous solutions to upregulate the

immune system and its mechanisms is also a task that must be on the front burner. We need to reconsider our approach to vaccine and drug development to make them more potent and effective. The phenomena where viruses change and mutant swiftly and made nonsense of vaccine endeavours need to be unravelled. This applies to drug and vaccine development as we now live in an era where the race against antimicrobial resistance is gaining pace and needs to be won in time to avert a bigger pandemic in years to come. The robust use of Artificial Intelligence (AI) in the battle against viruses is hereby advocated.

The cycle of viral infections could be complex but not unbreakable. As simple as handwashing is, it is very effective in winning this battle before the immune system is called upon. This is within the content of personal and community hygiene. For this to be effective however, public awareness needs to be right. With the right sensitisation, the public will be fully enlightened on preventive measures, including vaccinations. Unfortunately, we woke up to an era of serious misinformation around viruses, their evolution and vaccine safety and intent following the SARS Cov2 pandemic of 2019. The misinformation was a pandemic of its own and it almost made nonsense of our response strategies globally and locally. It has become extremely important to professionalise health information dissemination through strategic efforts and health education.

Funding has continued to retard efforts around viral diagnosis and research. Not many health centres carryout comprehensive diagnosis on viral diseases to support clinical management of cases. The few that do are limiting it to either the use of rapid diagnostic kits or serological platforms without verifiable data on standards and quality. Unfortunately, most virus diagnosis require molecular studies or cell culture platforms for effective diagnosis. These facilities are lacking due to funding. As a result,

researchers on viruses who are determined to go the full length must look beyond the shores of the country to either access funding or placements to make progress. The call for investment in this area is hereby amplified.

ACKNOWLEDGEMENTS

Distinguished ladies and gentlemen, Life is such a mystery. In the journey of life, the only constant thing through triumphs and travails is that our paths will always cross with others. This was what defined what I became today. I had craved for opportunities like this to acknowledge the immense contribution of many individuals and groups to the making of the story of my life. To all of you, I remain eternally grateful. I would love to pay tribute to all of you individually unfortunately, due to constraint of time and space, this will not be possible, however I beg your pardon to limit it to my recall of the significant contribution and impact in my life.

Mr Chairman sir, first and foremost, kindly permit me to acknowledge the Almighty Allah the beneficent, the merciful for everything. All thanks go to Allah without whom I wouldn't have been here to deliver this lecture. *Alhamdulillah*.

Permit me also to acknowledge the exemplary and inspiring leadership of the vice chancellor of the federal University of Lafia who since coming on board has given both the institution and staff a lift. I am particularly grateful to you, sir. I specially appreciate our hard working and result oriented Vice Chancellor Professor Shehu Abdul-Rahman, a role model and mentor per excellence, for the opportunity to stand before you all today to deliver my inaugural lecture.

It is a great privilege to still have one's parents alive at this stage of life. My immeasurable gratitude goes to my parents Alhaji Alim Junaid and Hajia Rabiat for all your sacrifices, proper training, disciplined upbringing, and consistent prayers. I recall with nostalgia, how my beloved mother will pass life lessons to us through night-time stories and core values. May Allah reward you both with Aljanna Firdaus.

My profound gratitude goes to my siblings, Abdul-Hakeem, Mansur, Sururat, Muhideen, Teslim, Harirat, Abdul-Azeez, for the brotherhood and comradery that typified our relationship all along. Keep the flame burning.

I appreciate the short yet impactful contribution of my late wife Amina(Mina), leaving behind 4year old Zainab that has grown to a woman. The loss paused my academic sojourn and dampened morale, that was the "dark phase", but your footprints are still visible. May the Almighty forgive her shortcomings and grant her Aljanna Firdaus.

To my darling wife Hajia Amina (Aminoo), I am struggling to find the most appropriate words to express my appreciation for your unparallel support, but let me say thank you so much, for helping me through the "dark phase" to the resurgence. I am here today because of your strong unwavering support and prayers. Thank you for your love and friendship I pray that all your dreams will come true and that you will enjoy the fruits of your labour as well. To my children Zainab, Ammar, Muhammad - Alamin, Mustapha and Mukthar, I thank you all for your patience, understanding obedience and friendship. May Almighty Allah bless you all abundantly and perfect his plan for you. I want to also register my appreciation to my wonderful and ever supportive in-laws, especially Hajia Muhibba (Mamoo) and Alhaji Abdulrazak (Papo). Thank you for everything.

To my uncles, Alhaji Muhammad Junaid and Hajia, my elder brother; Alhaji Hamza Abdulsalam, cousins, nephews, nieces too numerous to mention but a few. I want to appreciate the parts you all played over the years that culminated to what I became. Thank you all.

I recall and appreciate the foundational contribution of my primary and Arabic school teachers, whose early molding and

guidance was key to what I became today. Even though I can only vaguely remember your names, you are all well acknowledged.

I remember with nostalgia my secondary school teachers notably; Mr. Thomas, Mr. Mathews, Mrs. Mathews, Mr. Husseini, Mr. Noshi, and a host of others for their immense role in shaping me for the next critical stage of my journey.

To my secondary school mates especially the class of 1987; Government College Pankshin; POBA '87, whom I am proud to be associated with and elated to have your presence today to honour the clarion call; I remain indebted to our friendship and togetherness through the years. Let the true brotherhood we share continue to rage on. Great POBA! Thank you so very much you are all greatly acknowledged. Space and time will not allow me to mention you individually.

I thank all the friends and colleague I met in the National Veterinary Research Institute, Vom, the University of Jos and the University of Cape Town, South Africa in the quest of seeking knowledge whose impact I cannot easily forget; my very good friend; Sunday Adebayo, a loyal friend, and a confidant, Arzard Samuel and Muhammed Bala Abdu, both of blessed memory. We shared very fond memories. Rest on, while life battles continue to rage on.

Sadiq Yahaya, AbdulSalam Babakayode, Obisesan Oluwatuyi, Solomon Umukoro, Muhammed Salisu, Haruna Ogbole, Onovoh, Okeke O, Dr. Shola Dayo Ajayi, Shola Oshadeyi, Dr. Nick Nwakpa, Dr. Jide Owolodun, Dr. Jim Banda, my childhood friends; Suleiman Shitu and AbdulAzeez, among others are all appreciated.

To my community in Vom, you all are special. You will always be family and home any time and day.

My appreciation goes to the entire FCVMLT and NVRI family. I spent a substantial part of my life with most of you. I thank your contribution through these years, for the love, care, support, ups and downs, especially; Dr. Okwori, Dr. Oluwatayo, Dr. TKC Udeani, Adanma, Hauwa, late Jalmet, Joshua, Mrs Oluwatayo, Okeke, Dr. Chukwu; Julie, Oluchi, Ashi, Mathias Dalyop, Umar Baba, Abubakar Alhaji, Late Dr.Elayo, Prof. Ndako, Victor Abiodun, Coker; the list is inexhaustive.

To my mentors academically, religiously, and professionally, I am here because you stood by me. I rode on your shoulders to attain this height today.

My special gratitude goes to my teachers, lecturers, and mentors including Late Dr. Olabode who sharpened my interest in Virology and Mentored me through my Fellowship in Medical Virology and my first Masters degree in Medical immunology, Dr. G. O. N. Echeonwu, Late Dr Shidali, late Dr. Jemitola, Late Dr, Nwobu. Late Dr. H. O. Okpala, Mr. Onwe, Late Mr. Ogbonna, Dr. Adeyanju, Prof. F. A.C. Onwuliri. and others. How can I forget Prof. S.E. Agina under whose supervision I bagged my second Masters and my PhD degrees respectively.

My Special appreciation and commendation go to Dr. Solomon C. Chollom for his unflinching support, hard work, resilience, and selflessness. Time and time again, you proved to be a worthy ally, always come through at all times of need. You are a true friend indeed. I also wish to commend and appreciate Dr. Joseph Dlama Zira a worthy ally, confidant, and true friend. I must not fail to mention my good friend Dr. A.I. Akyala, for his contribution and selflessness, Sophia Osawe of Institute of Human Virology (IHVN) for her significant assistance at a critical time of need.

My Vice Chancellor Sir, permit me to express my gratitude by

acknowledging the following colleagues in FULafia though in no particular order or preference. I acknowledge and appreciate the immediate past Vice Chancellor, Prof. Muhammad Sanusi Liman who facilitated my coming to the Federal University of Lafia. Prof. Is-haq Ali Shugaba, Deputy Vice Chancellor, Administration, and Provost College of Medicine, former Provost Prof. Yakubu A. Ashuku, Prof. Mohammed Isa Kida, Prof. Jibril Danasabe, Prof. Samaila Usman Dakyes, Deputy Vice Chancellor, Research & Partnerships, Professor Aleruchi Chuku, Deputy Vice Chancellor, Academic Affairs, Professor Josephine Odey, Professor Victor Samson Dugga, Professor Amos Ambo, Prof. Adam Sirajudeen, Prof. Victoria Pam, Bashir Ozomata, Hajia Amina Makunguji, Mr. Wilson A. (Bursar), Nuradeen Abdu (Registrar), Edibo Sunday Abel, David, Alh. Saidu, Prof. Bako Ara, Dr, Rene Mathew, Dr. Ahmed Rabiu and Dr. Lawal Abdul Mumuni. All Deans and Heads of Departments in FULafia, all staff of the Faculty of Health Sciences, all Senate members, and staff of the Academic Unit.

To the entire university community, you have been extremely supportive. Every team has been unique and every individual that I have met, has added a layer of knowledge to me.

Sir, permit me to specially appreciate the following secondary school classmate and very good friends who has made and continue to make my stay in Lafia memorable. They include but not limited to Timothy Ihuman, Suleiman Dahiru (Malamawan Lafia), late Mustapha Ibn Agwai, Engr. Williams and Kefas.

I sincerely thank the staff of Department of Medical Laboratory Science my immediate constituency, teaching, and non-teaching. I recognize the relentless services rendered by Dr.Akpulu Peter, Dr. S. C. Chollom, Dr. John Ndubuisi, Abdullahi Mairiga, Usman A. Itakure, Emmanuel Onovoh, all Visiting lecturers, Mrs Ogwuche, Mrs Rosemary Ogenyi,

Yankat, Justina Rabi, Faith, Mustapha, Jesse Samson, Isa Dankawagi, Muazu, Balkisu Karim, Maimuna, Awazi, Umar Farouk. You are all acknowledged for your efforts.

My profound gratitude goes to the Association of Medical Laboratory Scientists of Nigeria (AMLSN) family for the privilege to serve at various times at the chapter, Branch and as the Assistant National Secretary and the National Secretary. Thank you all. Particularly, Dr. T. Y. Rahim, Mr. Manason Rubainu and Dr, G. C. Okara former presidents under whom I served. I also acknowledge the Registrar/CEO Medical Laboratory Science Council of Nigeria (MLSCN); Tosan Erhabor and indeed the entire MLSCN for the privilege and opportunity to serve the council. My appreciation also goes to the G50 family for the comradery and the West African Postgraduate College of Medical Laboratory Science WAPGCMLS for yet another golden opportunity to serve.

Mr. Chairman sir, permit me to appreciate chairman and my fellow members of the former Governing Council of the University of Calabar Teaching Hospital (UCTH), for the memorable time we spent on the board, I wish to appreciate our chairman Late Col. Muhammadu Abdu (Rtd.), Hon. Rotimi Raman, Hon. Segun Omoworare, Barr. Emeka Akwaka and others.

To my students I admire your quest for success, discipline, and resilience, I appreciate your unwavering support and cooperation. Mr. chairman sir, the pioneer set MLS students of FULAFIA will be graduating in a few days. I am particularly proud and elated to have the privilege of midwifing the process as the pioneer Head of Department.

I acknowledge the role of the inaugural lectures committee lead by chairman, committee of Provosts, Deans and Directors, Prof.

Josephine Odey, thank you for the guidance that enabled me to prepare and deliver this lecture.

Ones again I wish to thank our amiable Vice Chancellor for this opportunity to deliver the 20th Inaugural lecture in the series, also the first to be delivered from Department of Medical Laboratory Science, Faculty of Health Science and indeed the first from the College of Medicine, of this great University. May Almighty Allah continue to guard and guide you aright as you pilot the affairs of this great University.

Finally, my profound gratitude and appreciation go to the Vice Chancellor, Professor Shehu Abdul Rahman for the enabling platform to build my academic castle and for appointing me Dean, faculty of Health Science, Deputy Provost College of Medicine, and Head Department of medical Laboratory Science. I cannot thank you enough for your magnanimity, and confidence repose in me to deliver. I appreciate all the opportunities accorded me to serve our dear University, thank you for believing in me.

Mr. Vice Chancellor sir, this is my story, thank you all for coming and for being part of my audience.

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CITATION OF PROFESSOR SURAJUDEEN A. JUNAID

FWAPCMLS, FMLSCN, FAMLSN, MAMLSN

Mr Chairman, Ladies and Gentlemen!

It is my profound honour to present to you a man whose journey through the academic and professional landscape has been nothing short of extraordinary. Standing before you is a man who rode on the wings of hard work and determination and has epitomised the values of academic excellence, earning his placeamong the intellectual elites that has elevated him to the peak of the Ivory Towers, as a pathfinder and pacesetter in Medical Virology. He is an embodiment of humility, a polished administrator, and an intelligentsia to reckon with. Prof. Surajudeen Alim Junaid was born in 1970 in the beautiful city of Jos, Plateau state. Observant of the signs of responsibility and productivity demonstrated in early life, his parents did not waste time in nurturing him into the academic masterpiece he is today in character and learning as they enrolled him into the renown Nurudeen Primary School between 1976 and 1982 where he obtained the certificate of primary education. Eager to continue in his educational pursuit, young Junaid took the long journey from Jos city to Government College Bokkos and then to the serene, elevated altitudes in Pankshin Local Government where he joined the famous Government College Pankshin, Plateau state between 1982-1987 for his secondary education and obtained his GCE O' levels.

As one who has developed passion for inquiry into nature and determined to walk into the unseen world of viruses, the smart Surajudeen headed for medical laboratory science profession by enrolling in the University of Jos and the Federal College of Veterinary and Medical Laboratory Technology, Vom between 1987 and 1997 where he specialised and graduated as Medical Laboratory Scientist, specialising in Medical Microbiology.

He proceeded to Taraba State for his one-year mandatory national youth service (NYSC) and was posted to Wukari town for his primary assignment, where he received special commendation of excellent service. Upon completion of his NYSC, he was awarded with automatic employment and posted first to General Hospital Jalingo and later to Gembu in Sardauna Local Government Area as the medical laboratory scientist-in-charge of the General Hospital and the Local Government Area, from 1998 to 1999, a responsibility he discharged with passion and commitment. He received another offer of appointment with the National Veterinary Research Institute, Vom, Plateau State, in 1999, an offer he accepted.

The young graduate wasted no time as he enrolled for the Fellowship of Medical Laboratory Science Council of Nigeria between 1999 and 2000, specialising in Medical Virology. In a remarkable fashion, he pursued two Master of Science degrees between 2003 and 2007 and bagged Master of Medical Laboratory Science (MMLS) in Medical Immunology (2005) from Ambrose Alli University, Ekpoma and MSc Applied Microbiology from the prestigious University of Jos in 2007. Not done with his academic pursuit, this egghead, a colossus of wisdom and a fastidious reader enrolled for Doctor of Philosophy at the prestigious University of Jos in 2007 and bagged a PhD in 2014 in Applied Microbiology with thesis in Virology where he thoroughly explored the serologic and molecular peculiarities of Hepatitis E virus to consolidate his giant stature in the comity of virologists in Nigeria.

This Professor of Medical Microbiology who has an unbridled bias for virology has been active in service to his fatherland. Following a successful national service in Taraba State between 1997 and 1998, he picked up an appointment as a lecturer with the Federal College of Veterinary and Medical Laboratory Science Vom in 1998 where he taught bacteriology to students of

medical laboratory science. He rose through the ranks to become a Chief Lecturer and subsequently, the Head of Bacteriology Department from 2010 -2019. Prof. Through dedication to duty and excellence in service, the then Dr Junaid was elevated to the position of Dean of Postgraduate studies Federal College of Veterinary and Medical Laboratory Technology, Vom; where he oversaw the fellowship programmes, a position he held between 2019 to 2021 before relinquishing it to join this prestigious citadel of knowledge in Lafia, the Federal University of Lafia.

Academic, Professional and Administrative Career

Following sterling performances administratively and otherwise, the Vice Chancellor, Prof. Shehu Abdul Rahman appointed Prof Junaid as the Pioneer Dean of Faculty of Health Science in 2022, re-appointed as Dean in 2023 and Pioneer Deputy Provost, College of Medicine in 2023. Prof Junaid was Ag, Head of Department (HOD) in 2020 and was re-appointed as Pioneer HOD in 2024. As the first Dean of the Faculty of Health Science, he was able to transform the faculty to meet up with excellent academic and clinical standard, with a well formidable administrative structure and prowess. He has shown sagacity in combining these responsibilities and delivering excellent outcomes in all of them.On assuming work at this renown University and thrilled with his qualities and experience in teaching, research and administration, Prof. Junaid was tasked with the enormous responsibility of heading the young department of Medical Laboratory Science. His immediate tasks were to give the department an administrative and functional face as well as achieve NUC and Medical Laboratory Council of Nigeria(MLSCN) accreditations. Through the support of the Vice Chancellor, the workaholic HOD worked round the clock to secure lecture halls, offices, laboratories, library, and all essentials, including personnel to give the department shape. Today, the department is in full swing, full accreditation has been achieved from NUC and MLSCN. The first set of Bachelor

of Medical Laboratory Science (BMLS) graduates from the department are about to be celebrated in a month or two. Also, postgraduate programmes are about to commence as administrative works in that regard are at advanced stage.

Ladies and Gentlemen, at the professional front, Prof. Junaid is an Associate Member of the Association of Medical Laboratory Science, A Fellow of the Medical Laboratory Science, a Fellow of the West African College of Medical Laboratory Science, and a Member of the Society for Scientists in Infectious Diseases. This academic is a consummate teacher and an astute researcher, he has a colourful academic cap decorated with authored book chapters, research publications, a long list of outstanding mentees he has groomed with doctoral degrees, outstanding record of community service and a dominant trait of leadership excellence with a touch of unionism

Scientific Impact, Publications and Scholastic work

A prolific researcher, Prof. Junaid has published over 50 research papers in high-impact international journals indexed with reputable publishers and database such as PubMed, Scopus, Scimago, Thompson Reuters, Tailor and Francis, Elsevier, Willey and Oxfords Publishing. He has also co-authored several influential book chapters in Medical and Health sciences. His works have made substantial contributions to the fields of virology and medical microbiology, earning him recognition both nationally and internationally. He has also served as a resource person and examiner for the Medical Laboratory Science Council of Nigeria and has been a visiting professor at several universities, including Benue State University, the University of Abuja, and Nasarawa State University. He has presented quality research works in National and international conferences and his contributions from research work has addressed and solved numerous health care problems in Nigeria. Professor Junaid has attended several

capacity building activities including conferences and workshops home and abroad. He has presented lectures, chaired technical sessions and lead panel discussions in several scientific workshops and conferences, distinguishing himself as an A-list intelligentsia.

Contribution to Medical Laboratory Science Training

This academic guru has served as resource person and examiner for the Medical Laboratory Science Council of Nigeria for several professional examinations He has also undertaken several technical tasks for MLSCN such as resource verification of universities, accreditation of programmes and laboratory inspections just to mention but a few. Among them are

- MLSCN Rep First Professional Examinations Baze University, Abuja
- MLSCN Rep Final Professional Examinations University of Jos
- External Examiner First Professional Examinations University of Jos
- MLSCN Rep First Professional Examinations University of Jos
- External Examiner Final Professional Examinations University of Jos
- **External Assessor** (MLSCN), First Professional Examinations, ABUTH, Zaria, 2004.
- **Coordinator** (MLSCN) Medical Laboratory Assistant Examinations, October 2003; March 2004; 2006, September 2008.
- **Coordinator** (MLSCN) Medical Laboratory Technician Examinations, March, 2005; 2007.
- **Coordinator** First Professional Examination, June 2007.
- **Coordinator** FinalProfessional Examination, (Medical. Microbiology). October 2006.
- **External Examiner** Final Professional Examination,

Medical Microbiology, October 2000; October 2006.

- **Examiner** (MLSCN) Medical Laboratory Technician Examination,2000, 2001, 2003, 2004, 2005, 2006.

Visiting appointments

On visiting appointments, Prof Junaid is a Professor, Department of Medical Laboratory Science, College of Health Sciences, Benue State University, Makurdi, Benue State. Nigeria since 2021; Professor, University of Abuja, Department of Medical Laboratory Science 2024 and Professor, Nasarawa State University, Keffi.

Ladies and gentlemen, this professor is not just an academic success, he is also an indomitable unionist with modern day board room skills. He has been a force to reckon with in his profession and other places that he has worked. He was the National Secretary of Association of Medical Laboratory Scientists of Nigeria (AMLSN). August 2014 – 2017; The Chairman, Academic Staff Union of Colleges of Agriculture (ASUCA) FCVMLT, Vom, 2012 – 2019; The National Assistant Secretary, Association of Medical Laboratory Scientists of Nigeria (AMLSN). August 2008 – 2011, The Branch Secretary, Association of Medical Laboratory Scientists of Nigeria (AMLSN), Plateau State Branch, 2001 – 2006; The Chapter Secretary, Association of Medical Laboratory Scientists of Nigeria (AMLSN), Vom Chapter 2001 – 2006; The General Secretary Senior Staff Association (SSA), N.V.R.I., Vom, 2002 – 2015.

Leadership Qualities

Upon joining the university community in Lafia, this golden fish could not hide as his sterling qualities of leadership and academic excellent brought him out. Prof Junaid is the current Dean, Faculty of Health Science, Federal University Lafia, he doubles as the current Deputy Provost, College of Medicine,

federal University Lafia and triples as the head of Department, Medical Laboratory Science. The three-in-one cap have been worn by the ebullient professor with grace as he keeps churning out success after success. Leading up to these positions, Prof. Junaid has served as Departmental Examination Officer, and is currently a Departmental, Faculty, College Board Member as well as Senate member of the university.

He has also been called upon to serve in the following committees: Member, College of Postgraduate College Board, Federal University, Lafia since 2021; Member, Students Disciplinary Committee, Federal University, Lafia since 2021; Member, Faculty Board, Faculty of Basic Medical Sciences, FU, Lafia since 2021; Member, Awards / Rewards System Committee, FU, Lafia since 2022 and Member, Committee of Provosts, Deans and Directors, Fed. University, Lafia since 2022.

Professor Junaid has not gone without notice before the curious eyes of the West African Postgraduate College of Medical Laboratory Science. He was the Faculty Secretary (Faculty of Virology) West African Post Graduate College of Medical Laboratory Science (WAPCMLS) 2021 – 2023. He is the current Faculty Chairman, (Faculty of Virology) West African Post Graduate College of Medical Laboratory Science (WAPCMLS). He is also the current secretary of the Nigerian Chapter of the College.

Community Service

Prof. Junaid has demonstrated a deep commitment to community service. His leadership roles in these organizations have further solidified his reputation as a consummate leader and mentor. Ladies and Gentlemen, this Professor is not without community impact. He has been a Road Safety Special Marshall with the Federal Road Safety Corps since 2014, a Police

Community Relation Committee (PCRC) since 2014, a Rotarian, Rotary club of Jos South since 2008, a National Youth Service Corps (NYSC) between 1998-1999, a National Youth Service Corps (NYSC), Road Safety Cadet, 1998-1999 and a Rotaract International Club, District 912, Rotaract Club of Vom, 1997.

While at the research hub and vaccines home at the National Veterinary Research Institute, Prof Junaid was a Member, NVRI, Vom Ad-Hoc Committee to Establish a Viable Fuel Dump in the Institute, March 2003; a Member, NVRI, Vom, Petroleum Products Committee, May 2003; a Member, FCVMLT, Vom, ad-hoc committee on Students Conduct/Discipline, June 2003; a Member, FCVMLT ad-hoc Committee on Employment Interview Panel, August, 2005, a member, Servicom Committee, FCVMLT Vom, 2006 and member, Bird Flu Committee NVRI, Vom, 2006.

Prof. S.A. Junaid has not gone without Presidential recognition as he was appointed member of the Governing Board of the University of Calabar Teaching Hospital by the President of the Federal Republic of Nigeria (Gen. Muhammadu Buhari)from 2018 to 2023. Within the Board, Prof Junaid was the Chairman, Appointment, Promotion and Disciplinary Committee between 2018 and 2023 where he boosted staff Morales through merited and timely promotions and deserved appointments, Member Finance and General-Purpose committee, Staff training and Development Committee, Litigation matters committee, Hospital Services Committee among other powerful committees of the Board.Prof. Junaid is also Member Standing Committee, Employment Interview Panel, National Institute for Policy and Strategic Studies (NIPSS), Kuru since 2006.

Awards and Honours

Prof SA Junaid has been recognized severally for his

outstanding service to humanity as he has continued to scoop awards at all fronts. Some of the awards include: the Nigeria Medical Laboratory Science Students Association (NIMELSA) FULafia, Distinguished Award of Excellence – 2024; College of Medicine Staff Award for Excellent leadership -2023; Academic Staff Union of Colleges of Agriculture (ASUCA) Humanitarian Merit Award – 2020; Association of Medical Laboratory Science Council of Nigeria (AMLSN) Meritorious Award -2011; SSAUTHRIAI Merit Award -2011; SSAUTHRIAI Merit Award -2007; AMLSN, (Vom Chapter) Merit Award -2007; NYSC Award -1998; NYSC Road Safety Cadet Merit Award -1998; Students' Union Government (SUG) Merit Award -1997; Odua Student merit award -1997.

Membership in Professional Bodies

Prof. Junaid has an intractable network of professionals that have impacted on his net worth. He is a member of many professional organizations. They include: The Association of Medical Laboratory Scientist of Nigeria (AMLSN); The Association of University Academic Medical Laboratory Scientists; The Microbiology Society of Nigeria (MSN); The American Society for Microbiology (ASM); The Biotechnology Society of Nigeria (BSN); The Society for Scientists in Infectious Disease (SSID); The Anti-Microbial Resistance Association of Nigeria (AMRAN); The G50 Initiative for Health and the Anti-Microbial Resistance Association Initiative **(AMRAI).**

National and International Travels

Professor Junaid is widely travelled. He has visited all the states in Nigeria and has friends and associates in every major city in the country. He has also visited countries like United Kingdom, Saudi Arabia, USA, South Africa, Angola, Niger Republic, Ghana, Burkina Faso, Togo, Benin Republic, and Cameroun.

Hobbies

With Prof. Junaid, all is not about work. His hobbies include reading, travelling and sports, He does this to recreate, refresh and reinvent himself for greater productivity

The Family

This unassuming Professor is a proud family man. He would not have achieved much without the support of his lovely, dedicated, obedient and hardworking wife, Hajia Amina Abdul Razak, who is humility personified. The family is blessed with five children namely, Zainab, Ammar, Muhammad Al-Amin, Muhmmad Mustapha and Muhammad Mukthar.

Ladies and gentlemen today is an epoch-making event in the life of this colossus as he takes the bold stage to share with us the landmarks experiences that dotted his academic journey from the city slums to the citadel of knowledge where he now bids to seat professorially as he continues to do exploits in Medical virology and Medical Microbiology as a whole.

INAUGURAL LECTURES SERIES IN FEDERAL UNIVERSITY OF LAFIA

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20.	Prof. Surajudeen A. Junaid	Tales of Man's Travails and Triumphs as the Battle with Viruses Rages on	24th Sept. 2024

SIGNIFICANCE OF INAUGURAL LECTURES IN FULAFIA

The rite of passage to become a professor in a university has for hundreds of years included the test of having to profess one's knowledge to a lay audience and fellow academics. Indeed, the origin of the title 'professor' comes from the need to profess, or declare publicly, one's knowledge. The occasion of inaugural lecture presentation is therefore an essential component of the University's public events through which the institution engages with audiences with a broader interest in its research, including funders and decision makers from government, academia and industry. Professionals and academics gain a unique opportunity to engage across knowledge boundaries for the benefit of mankind.

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TALES OF MAN'S TRAVAILS AND TRIUMPHS AS THE BATTLE WITH VIRUSES RAGES ON.

SEPT. 24, 2024