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REVIEW ON LIFE CYCLE OF CORONA VIRUS AND INBUILT IMMUNITY WEAPONS AGAINST COVID-19

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Abstract:

The most Major, dangerous, Chronic and leading problem of many countries are facing is the COVID-19 spread. The main purpose of this survey review is to know the molecular structure and the life cycle of corona virus. The origin and life cycle of corona virus is briefly discussed. The worldwide viral spread and its epidemiology/Pandemiology make it important to know about overall pathogenesis and their main targets and preventive measures or strategies against this COVID infection. The paper deals with the biology and replication with pathophysiology of Corona virus and awareness of preventive and defense mechanism against any viral or bacterial infection which is present inside our body already on every level.

Keywords: *COVID-19, upper respiratory, Corona, adaptive, Innate, Humoral, Cell mediated*

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1. INTRODUCTION:

Corona viruses are a group of enveloped viruses. They are non-segmented and have single stranded positive sense RNA. They affect vertebrates such as pigs and chickens. Apart from that six corona viruses have been known to infect human hosts. This leads to respiratory diseases among them, severe acute respiratory syndrome corona virus-2 (SARS-CoV2) and Middle East respiratory syndrome coronavirus (MERS-CoV) highly pathogenic corona viruses species that have resulted in regional and global outbreaks.

The international committee on taxonomy of viruses has classified corona virus as follows:

Table.1. Classes of Corona as per ICTV

Order	Nidovirales
Family	Coronqaviridae
Subfamily	Coronavirinae
The corona virus divided into 4 genera namely	• *Alphacoronaviru* • *Betacoronavirus* • *Gammacoronavirus* • *Deltacoronavirus*.

The beta-coronavirus is assigned with four specific lineages namely: A, B, C & D among the six known human corona viruses (HCoVs), HCoV-229E and HCoV-NL63 belong to *Alphacoronavirus*, whereas HCoV-OC43 and HCoV-HKU1 belong to lineage A, SARS-CoV to lineage B, and MERS-CoV to lineage C *Betacoronavirus*[1]

2. MOLECULAR BIOLOGY OF HUMAN CORONA VIRUS

Human corona virus is enveloped and non-segmented. It is approximately 120nm in diameter and is likely to be spherical. It consists of petal shaped projections called as spikes. These spikes are made up of spike protein which is in turn made up of heavily glycosylated type I glycoprotein. A subset of corona virus has an additional layer made up of hemaglutinin-esterase protein (HE), which is termed as short spikes. These short spikes are not important for viral infectivity. The envelope is made up of lipid bilayer and the large and sharp spikes are anchored into the bilayer. The lipid bilayer is formed by the virus budding from intracellular membranes. Other number of elements adhere to the envelope are integral membrane protein (M), small membrane protein (sM) or envelope (E). Inside the envelope is a ribonucleoprotein (RNP) core, which comprises the RNA genome and a single species of nucleocapsid protein N. The corona virus genome is non-segmented, single stranded and positive sense [2]. Its size ranges from 27 to 32kb. The genomic RNA is topped and polyadenylated and contains different open understanding edges (ORFs). The invariant quality request is Replicase-S-E-M-N-3? With various little ORFs (encoding embellishment proteins) dispersed among the basic qualities. The coronavirus replicase is encoded by two huge covering ORFs (ORF1a and ORF1b) involving around 66% of the hereditary succession or genome and is legitimately interpreted from the genomic RNA [3].

3. STRUCTURAL PROTEINS

3.1 SPIKE GLYCOPROTEN(S)

The outermost component of corona virus is the S glycoprotein. Its main function is the attachment of the viral antigen to the host cells. It is also responsible for the fusion of virus envelope with the cell membranes. The host's immune response first attacks the S glycoprotein. The S protein is large in size, ranging from 1160 to 1452 amino acids. Inside a coronavirus animal varieties, arrangement variety is generally shown more by S proteins; the variety of the Spike proteins (S1&S2) grouping most likely gives a specific bit of leeway in insusceptible creatures. There are numerous potential N-connected glycosylation locales. The spike protein has N-terminal sign succession and a film mooring plan close to the C end. The S protein might be cut into S1 and S2 subunits. The cell type determines the extent of S cleavage. This cleavage leads to generation of two major glycopolypeptides as follows: N-terminal S1 and C-terminal S2. S1 and S2 are linked to each other by non-covalent linkage. The breakage can be caused without disrupting the spikes. This is done by the treatment of trypsin of MHV virions. The S1 can be released from the virion by urea or mild alkali treatment. As the whole genome of the corona virus is considered, the S2 is more conserved than S1.The S2 polypeptide has two areas with seven build up periodicity which structures wound structure. The two major role of the Spike (S1 &S2) protein are as follows:

i) It is responsible for the fusion of the virus bilayer with the cellular membranes.

ii) This is necessary for viral entry into the cell. S protein is sufficient for the membrane fusion.

iii) S protein is responsible for viral binding to the receptors of the target cells [3].

3.2 INTEGRAL MEMBRANE PROTEIN (M):

The M protein is the structural protein essential for the production of corona virus like particles. The M polypeptide is made up of 225-230 amino acids. The amino terminal 20 or so residue of mature M protein of all corona viruses is hydrophilic. They are exposed to the virion surface. They contain small number of glycosylation sites. The remainder of the N-terminal half of the molecules form three helical membrane. The structure of the C-terminal half is not known for sure, yet it is accepted to [3].

3.3 HEMAGLUTININ-ESTERASE GLYCOPROTEIN:

This is considered to be the mysterious gene. The corona virus belonging to the MHV group possess the HE gene. The product of this gene is not necessary for the viral replication. Not all virus strains express the HE protein

3.4 SMALL MEMBRANE PROTEIN (E):

It was earlier considered that the corona virus consist of three structural proteins namely S, M, N or four (HE). It is now proved that the virion contains an additional protein called as the small membrane protein which is very essential for the virion assembly. It is clearly known that the E and M protein are very essential for the virion assembly.

3.5 NEUCLEOCAPSID PROTEIN (N):

The N protein is a 50-60 kilo-Dalton phosphoprotein. This along with the genomic RNA forms a helical nucleocapsid. This is about 9-11 or 14-16nm in diameter. The nucleocapsid protein provides only limited protection to the RNA genome against the various enzymes like ribonucleases. The N protein contains 377 to 455 amino acids. This is highly basic in nature and has high serine content (7-11%)[3]

4 REPLICATION CYCLE OF CORONA VIRUS:

Virus are naturally obligate intracellular parasite which carry their genome (RNA/DNA) or you can call it as functional proteins which are necessarily required in replication process or in early steps of replication cycle.

Actually, the virus replication is completely depending on host cell machinery

Virus must command to host machinery for successful replication. The replication cycle produces 100-1000's new virus particle per cycle in which they generate 2 components: 1) functional RNA's and proteins and 2) genomic RNA or DNA and structural proteins and this replication is might be cytolytic or non-cytolytic.

The various steps involved in the replication cycle of the corona virus are as follows:

1) Attachment: the virus attaches to specific receptor on cell surface of host
2) Penetration: Enveloped virus penetrate host cell through fusion of viral envelope with host cell membrane
3) Uncoating: this step makes viral nucleic acid available for transcription to permit multiplication to proceed
4) Transcription: In this step by using host machinery the virus makes RNA copy of gene sequence and this copy called mRNA which enters into the cytoplasm where it directs synthesis of proteins, which it encodes.
5) Translation: it is a process in which ribosome in cytoplasm and endoplasmic reticulum synthesize protein after transcription of DNA and RNA in cell nucleus
6) Assembly and release: virus get assembled into infected host cell nuclei and mature at the inner lamella of nuclear membrane
7) Replication of Corona virus

4.1 ATTACHMENT AND ENTRY:

The first step of coronavirus replication is the binding of S protein to the cell surface receptors. The S protein has two subunits: S1 called as the bulb which is responsible for the receptor binding and S2 called as the stalk responsible for the fusion of the cell membranes. When the S1 binds to the receptor, it triggers a triggers a conformational change in the S2 subunit. This inturn results into fusion between the virus envelope and the cellular membrane and release of nucelocapsid in the cytoplasm. Some of the cell surface enzymes like aminopeptidase N (APN) for HCoV-229E, angiotensin converting enzyme 2 (ACE2) for HCoV-NL63 and SARS-CoV, and dipeptidyl peptidase 4 (DPP4) for MERS-CoV are used as receptors by some human corona virus.One or more host proteases governs the cleavage of S protein into S1 And S2. Host factors could also restrict the attachment and entry of HCoV. For example, interferon-inducible transmembrane proteins (IFITMs) exhibited broad-spectrum antiviral functions against various RNA viruses. The entry of SARS-CoV, MERS-CoV, HCoV-229E, and HCoV-NL63 was restricted by IFITMs [4]

4.2 TRANSLATION OF VIRAL REPLICASE:

After the entry and Uncoating, the RNA serves as a transcript [5]. This leads to cap dependent translation of the open reading frame-1a to produce polyprotein-1a(pp-1a). The slippery sequence and an RNA pseudoknot near the end of open reading frames-1a enable 25-30% of the ribosome to undergo frame shifting. This produces longer polyprotein which is termed as pp-1ab. The autoproteolytic cleavage of pp1a and pp1ab generates 15–16 nonstructural proteins (nsps) with various functions [6]. The RNA-dependent RNA polymerase activity is encoded in nsp12. The papain like protease and main protease activity is encoded at nsp3 and nsp5. The other non-structural protein starts the rearrangement of cellular membrane to form double membrane vesicles. This

leads to the coronavirus replication transcription complex [7]

4.3 GENOME REPLICATION AND TRANSCRIPTION:

The genomic RNA is used as a template to synthesize negative-sense antigenome. This is then used as a template to synthesize the new genomic RNA. There are specific sites called the transcription regulated sequences where the polymerase can switch template during discontinuous transcription [7].

4.4 VIRION ASSEMBLY AND RELEASE:

The particle assembly is governed by M protein. This occurs in the ER-Golgi intermediate compartment. The interactions of M protein lead to the virion morphogenesis [8]. The Interaction between the M protein and the S protein as well as M protein and N protein lead to transfer of structural components to the assembly site. The E protein also contributes to the virion assembly. Finally, the coronavirus particles are transported to the smooth wall vesicles and released by exocytosis. You can see in given Figure (fig.2) Whole replication cycle in brief flowchart clipart for reference [7].

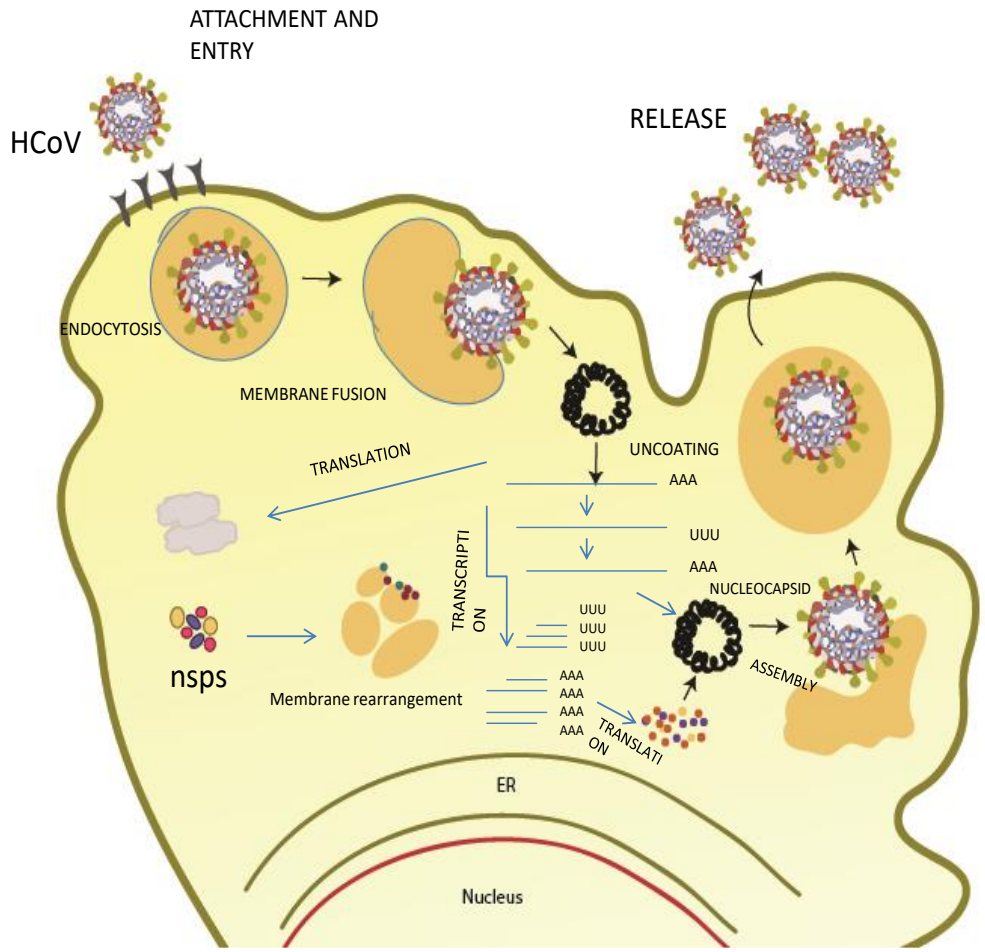

Figure 1.replication cycle of Covid-19

5 COVID-19 EPIDEMIOLOGY:

The new public health crisis threatening the world is the emergence and spread of the novel corona virus. The virus originated in bats and was transmitted to humans through other unknown intermediary animals. This started in Wuhan, China in December 2019. This disease has affected many people worldwide. Till 5th march 2020, there were 96000 reported cases of coronavirus disease and 3300 reported deaths[11]. The transmission of this disease takes place by inhalation or close contact with the infected person. This disease is mild in most cases. If the severity increases it may lead to pneumonia, acute respiratory distress syndrome and multi organ dysfunction. Many people are asymptomatic. The fatality rate is estimated to be 2-3%. This disease has had a huge global impact. It is continuing to affect people all over the country [8].

Table 2: The number of cases and death of COVID – 19 outbreaks according to WHO situation reports 20th May, 2020.

Country	Cases	Death	Region
China	4,999,235	325,125	Asia
Singapore	29,364	22	Asia
Hong Kong	1,056	4	Asia
Thailand	3,034	56	Asia
South Korea	11,110	263	Asia
Japan	16,367	768	Asia
Malaysia	6,978	114	Asia
Germany	177,827	8,193	Europe
Australia	7,079	100	Australia
Vietnam	324	00	Australia
United State	1,570,583	93,533	North America
France	180,809	28,022	Europe
Macao	124,603	7,119	Asia
United Kingdom	248,818	35,341	Europe
United Arab emirates	25,063	227	Asia
India	106,886	3,303	Asia

Updated data of COVID-19 Pandemic in India (20-22 may 2020)

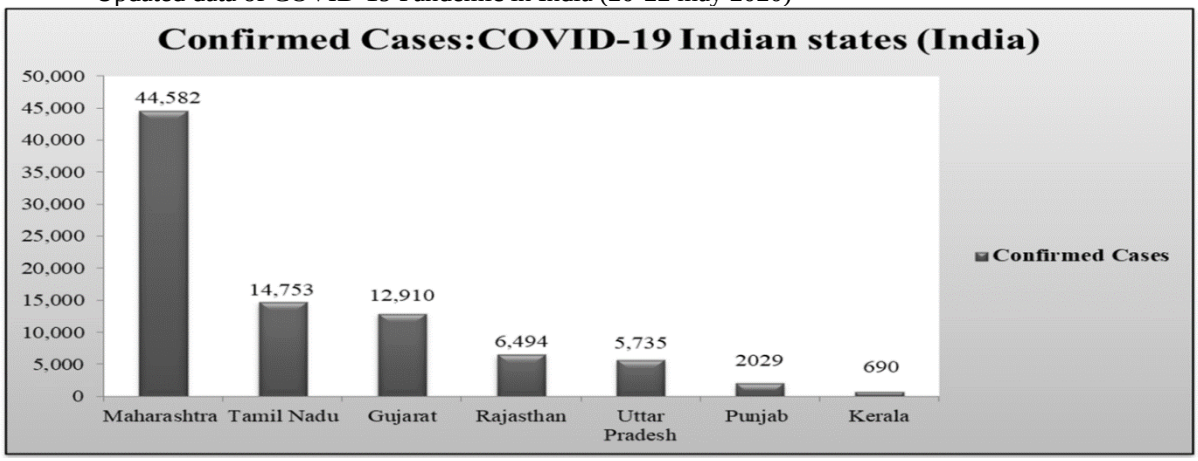

Figure.2. Confirmed Case statistic of COVID-19 in India

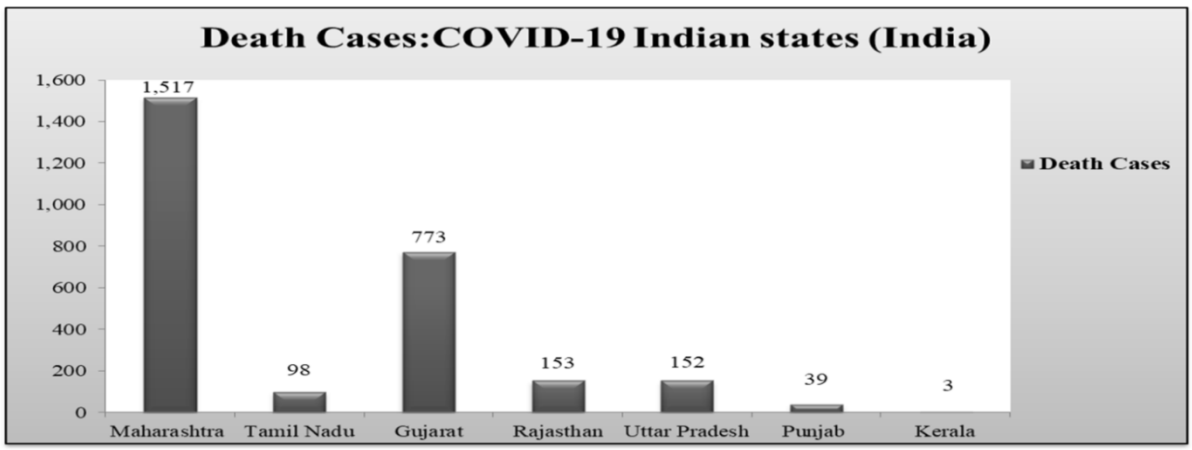

Figure .3.Statistic of Death cases (COVID-19) in India

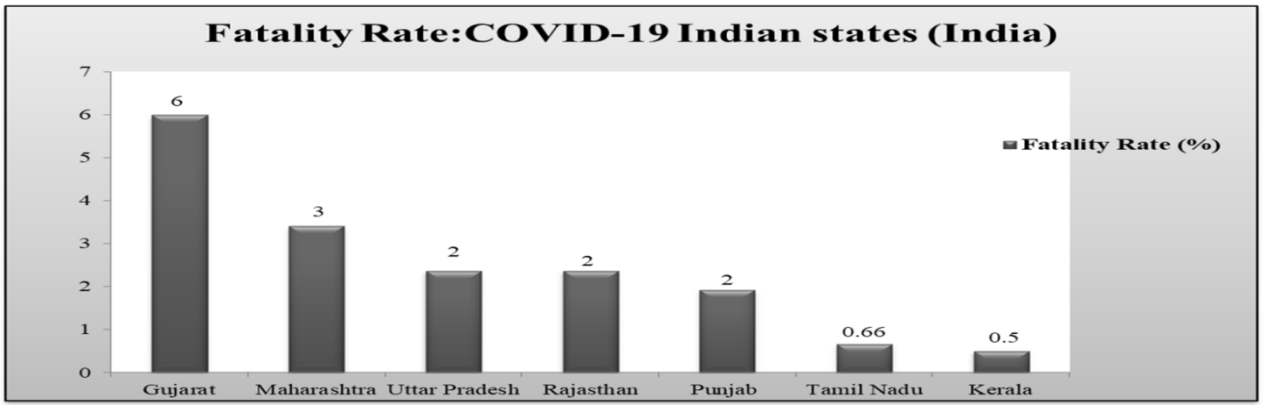

Figure4. % fatality rate in India

1. ROLE OF INNATE AND ADAPTIVE IMMUNE SYSTEM IN VIRAL INFECTION:

The immune framework has evolved to shield the host from a vast expanse of pathogenic microorganisms that are themselves continually developing. The immune system additionally enables the host to eliminate poisonous or allergenic substances that enter through mucosal surfaces. Integral to the resistant framework's capacity to prepare a reaction to an attacking pathogen, toxins or allergen is its capacity to separate self from non-self. The host utilizes both innate and adaptive systems to recognize and wipe out pathogenic microorganisms. Both of these components incorporate self-oneself segregation. This review recognizes key instruments utilized by the immune system to react to attacking organisms and different exogenous dangers and identifies settings in which upset immune capacity worsens tissue injury

The immune reaction to viral contamination involves inborn and versatile adaptive barriers. The natural reaction or innate response, which we have talked about beforehand, works persistently in an ordinary host without presentation to any infection. Most viral diseases are constrained by the innate safe immune system. Nonetheless, if viral replication outpaces inborn safeguards system, the adaptive reaction must be prepared. The versatile barrier or adaptive defence comprises of antibodies and lymphocytes, regularly called the humoral reaction and the cell mediated immune reaction. The term 'adaptive' alludes to the separation of self from non-self, and the fitting of the reaction to the specific outside trespasser. The capacity to shape the response in an infection explicit way relies on correspondence between the natural and adaptive immune frameworks. This correspondence is done by cytokines that bind to cells, and by cell-cell interaction between dendritic cells and lymphocytes in lymph nodes. This communication is urgent to the point that the versatile reaction can't happen without an inborn insusceptible framework or natural innate immune system.

The cells of the adaptive immune framework are lymphocytes – B cells and T cells. B cells, which are gotten from the bone marrow, become the cells that produce antibodies. Immune system microorganisms, which develop in the thymus, separate into cells that either take part in lymphocyte development, or slaughter infection tainted cells. Both humoral and cell mediated reactions are essential for antiviral protection. The contribution of each varies, depends upon the causative agent and the host. Antibodies by and large tie to infection particles in the blood and at mucosal surfaces, along these lines hindering the spread of contamination. Conversely cells perceive and execute infected cells.

A key element of the adaptive immune framework is memory. Rehash infection by a similar virus are met promptly with a solid and explicit reaction that generally successfully stops the disease with less dependence on the intrinsic or innate immune framework. At the point when we state we are invulnerable to contamination with an infection, we are discussing resistant memory. Antibodies or antibodies ensure us against disease on account of safe memory. The principal adaptive e reaction against an infection is called the primary response or reaction frequently takes days to develop and to become mature. Interestingly, a memory reaction creates inside after long periods of disease. Memory is kept up by a subset of B and T lymphocytes called memory cells which get by for a considerable length of time in the body. Memory cells stay prepared to react quickly and productively to a resulting experience with a pathogen. This supposed secondary reaction is frequently stronger than the primary response to disease. Subsequently, youth contaminations secure grown-ups, and invulnerability presented by inoculation can keep going for a considerable

length of time. The idea of the adaptive immune reaction can obviously decide if an infection contamination is cleared or makes harm the host. Be that as it may, an uncontrolled or wrong adaptive response can likewise be harming. A total comprehension of how infections cause illness requires energy about the versatile invulnerable reaction [10].

2. VIRAL ACTIVATION OF IMMUNITY:

Immunity against any viral infection is produced by a variety of mechanism it might be either specific or nonspecific. The time lapse, activation and power or magnitude of immune response is depend on how the virus interacts with host cells (on whether it is steady state, cytolytic, latent, or integrated infection) and on how the virus spread (by local, primary hematogenous and nervous system spreads.) therefore, viral antigen might be present in various parts of infection. Actually the host has number of immune defence mechanisms that can eliminate causative agent or virus and viral disease

Table no. 3. Defense mechanism in the upper airways

Defense	Humoral	Cellular
Mechanical	Mucus	Cilliary epithelium
A specific immunological defense	Complement lysozyme lactoferrine	Granulocyte Macrophage
Specific immunological defense	Immunoglobins	Lymphocytes

2.1. Humoral Immunity: virus as well as infection tainted cells can initiate or potentiate B lymphocytes
to create counter acting agent called antibodies (explicit for viral antigens) Antibody balance is best when infection is available in enormous liquid spaces (e.g., serum) or on wet surfaces (e.g., the gastrointestinal and respiratory tracts). IgG, IgM, and IgA have all been appeared to apply antiviral activity. Immune response can kill infection
by:
1) Blocking virus cell interaction or
2) Perceiving viral antigens on infection contaminated cells which can prompt neutralizer subordinate cytotoxic cells (ADCC) or supplement interceded lysis. IgG
antibodies are liable for most antiviral activity in serum, while IgA is the most significant counter acting agent when infections contaminate mucosal surfaces.

a. **Cell-Mediated Immunity:** The term cell-mediated immunity refers to the recognition and additionally murdering of virus and infection tainted cells by white blood cells and the formation of various dissolvable elements (cytokines) by these phones when invigorated by infection or infection contaminated cells. Cytotoxic T lymphocytes, regular executioner (NK) cells and antiviral macrophages can recognize and slaughter infection contaminated cells. Partner T cells can perceive infection contaminated cells and produce various significant cytokines. Cytokines delivered by monocytes (monokines), T cells, and NK cells (lymphocytes) play significant work in managing resistant capacities and creating antiviral safe capacities. The early, nonspecific reactions limit viral replication and proliferation during the intense and acute period of infection diseases. The later explicit immune (humoral and cell intervened) reactions capacity to help dispense with infection toward the finish of the intense stage, and in this way to keep up explicit protection from reinfection it resembles immunization or vaccination [10].

3. RESPIRATORY INFECTION DEFENCE MECHANISM:

3.1. Defense mechanism in the upper airways:
The nose is the climate control and air conditioner system of the airways. Since typical relaxing is through the nose, most airborne particles are sifted there; henceforth the nasal mucosa is the primary line of protection against particles noticeable all around. Pathogenic and non-pathogenic antigens constantly shell the epithelium of the nasal airways. These antigens are basically expelled non-immunologically by the main guard layer of the mucosa, comprising of bodily fluid, ciliated epithelial cells, and glycoproteins/lysozymes. On the off chance that the antigen passes this safeguard layer, explicit and vague immunological guard systems exist. The vague protection comprises of phagocyting cells like neutrophils and macrophages and the supplement actuation. The particular guard component (bringing about a particular immunological response according to a specific antigen) is framed by the antibodies, for the most part secretory IgA and to a lesser degree IgG and immunocompetent cells in the nasal mucosa. Initiation of the particular barrier instruments may prompt irritation which can be unfavorably susceptible. The serious co-activity of mechanical, explicit immunological resistance brings about a firmly controlled harmony between an appropriate safeguard against pathogens and extreme touchiness. Disappointments in these safeguard instruments, or their co-activity, brings about upper respiratory contamination as well as sensitivity [11].

3.2. Explicit cell guard of respiratory system:
The particular guard system (bringing about a particular immunological response comparable to a

specific antigen) is framed by the antibodies, basically secretory IgA and to a lesser degree IgG and immune able cells in the nasal mucosa. Activation of the particular defense systems may prompt irritation and inflammation which can be hypersensitive allergic. Powerful mucosal invulnerability relies upon antigen priming of both T and B cells. Leukocyte receptors can just react to antigen that has been handled and by antigen presenting cells. The antigen presentation procedure can happen locally in the lamina propria and in the regional draining de lymphoid structures of the nasal mucosa known as Waldeyer's ring. This comprises of the palatine and lingual tonsils, the adenoid and the cervical lymph node. The general happening induction of tolerance, regardless of the bombardment of antigens, proposes a carefully managed T-cell framework. Dynamic immunosuppression appears to play a significant role in the separation among minor and potential pathogenic antigens and results in the ordinary circumstance in a resistance for insignificant antigens [11].

Coronaviruses are enormous, enveloped RNA infections of both clinical and veterinary significance. Enthusiasm for this viral family has increased in the previous barely any years because of the identification of a recently emerged coronavirus as the causative operator of severe acute respiratory syndrome (SARS). At the sub-atomic level, coronaviruses utilize an assortment of bizarre systems to achieve a perplexing project of quality expression [11]. Coronavirus replication involves ribosome outline moving during genome interpretation, the amalgamation of both genomic and numerous subgenomic RNA species, and the gathering of descendants virions by a pathway that is exceptional among encompassed RNA infections. Progress in the examination of these procedures has been upgraded by the improvement of opposite hereditary frameworks, a development that was up to this time impeded by the huge size of the coronavirus genome [12]. The sign of coronavirus translation is the creation of numerous subgenomic mRNAs that contain groupings comparing to the two parts of the bargains. (Translation is characterized as the procedure whereby subgenome-sized mRNAs are created, and coronavirus replication is the procedure whereby genome-sized RNA, which additionally works as mRNA, is delivered.) Thus, the age of subgenomic mRNAs includes a procedure of irregular transcription [14]. The coronavirus genomic RNA of roughly 30,000 nucleotides encodes basic proteins of the infection, non-structural proteins that have a basic job in viral RNA amalgamation (which we will allude to as replicasetranscriptase proteins), and non-structural proteins that are insignificant for infection replication in cell culture yet seem to present a particular bit of leeway in vivo (which we will allude to as specialty explicit proteins). At any rate one specialty explicit protein, non-structural protein 2 (nsp2), and one basic protein, the nucleocapsid (/look into/infection/hcov-nucleocapsidoverview) protein (N), are engaged with viral RNA union. The declaration of the coronavirus replicase-transcriptase protein qualities is interceded by the interpretation of the genomic RNA. The replicasetranscriptase proteins are encoded in open-perusing outline 1a (ORF1a) and ORF1b and are integrated at first as two enormous polyproteins, pp1a and pp1ab. The blend of pp1ab includes modified ribosomal outline moving during interpretation of ORF1a. During or after blend, these polyproteins are separated by infection encoded proteinases with papain-like (PLpro) and chymotrypsin-like folds into 16 proteins; nsp1 to nsp11 are encoded in ORF1a, and nsp12 to nsp16 are encoded in ORF1b. The replicase-transcriptase proteins, together with other viral proteins and, potentially, cell proteins, amass into membranebound replication-interpretation edifices (RTC)[2]. (We will utilize the term RTC to portray edifices replicating or delivering genome-or subgenome-length RNA.) These buildings amass at perinuclear locales and are related with twofold film vesicles. Hydrophobic transmembrane spaces are available in nsp3, nsp4, and nsp6 and likely serve to stay the incipient pp1a/pp1ab polyproteins to films during the initial step of RTC formation. The system of coronavirus replication thusly will take the coronavirus replication of MHV (mouse hepatitis infection) for instance. to the host-cell receptor CEACAM-1 through cooperation of the spike (S) glycoprotein. Infection section into the host cell can happen through combination with the outside of the host cell, with the ensuing arrival of the genomic RNA into the cytoplasm. On the other hand, MHV can enter the host cell through the arrangement of endocytic vesicles, and genomic RNA is discharged into the cytoplasm following combination with the vesicle film [13]. Interpretation of the positive-strand genomic RNA offers ascend to an enormous polyprotein that experiences proteolytic preparing to produce a RNA-subordinate RNA polymerase. Through the activity of the RNA polymerase, a full-length, antisense negative-strand layout is created. Subgenomic mRNAs are blended, apparently from subgenomic negative-strand formats. Interpretation of subgenomic mRNAs offers ascend to auxiliary viral proteins. S glycoprotein is communicated on the outside of the host cell and this may add to combination with neighboring uninfected cells by official to CEACAM-1. Infection get together happens inside vesicles, trailed by infection discharge by combination of virion-containing vesicles with the plasma film. Discharged infection

can taint different cells and can imitate inside the parent cell through official to CEACAM-1. E, envelope protein; ER, endoplasmic reticulum; M, layer protein; N, nucleocapsid protein; ORF, open understanding casing [13].

CONCLUSION:

Corona viruses are a group of enveloped viruses. Over the past 50 years the emergence of many different coronaviruses that cause a wide variety of human and veterinary diseases has occurred. It is likely that these viruses will continue to emerge and to evolve and cause both human and veterinary outbreaks owing to their ability to recombine, mutate, and infect multiple species and cell types. The4 viral replication of the Corona virus is almost same as common replication cycle of general.

Future research on coronaviruses will continue to investigate many aspects of viral replication and pathogenesis. First, understanding the propensity of these viruses to jump between species, to establish infection in a new host, and to identify significant reservoirs of coronaviruses will dramatically aid in our ability to predict when and where potential epidemics may occur. As bats seem to be a significant reservoir for these viruses, it will be interesting to determine how they seem to avoid clinically evident disease and become persistently infected. Second, many of the non-structural and accessory proteins encoded by these viruses remain uncharacterized with no known function, and it will be important to identify mechanisms of action for these proteins as well as defining their role in viral replication and pathogenesis. These studies should lead to a large increase in the number of suitable therapeutic targets to combat infections. And in case of prevention we have our upper and lower respiratory defenders mechanism to combat so, focused is to strengthen the respiratory system in all sense and after this we have cell mediated and humoral immunity to combat so this mechanism building and strengthening may help as Prevention strategy and in other ways The particular guard system (bringing about a particular immunological response comparable to a specific antigen) is framed by the antibodies, basically secretory IgA and to a lesser degree IgG and immune-able cells in the nasal mucosa is to protect.

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