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A Review on Possibility of Polio Vaccine to Combat Covid-19

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Abstract:- As COVID-19 is spreading fast and killing the people around the world, it made an emergency situation for looking a vaccine to control the lethal effect of COVID-19 on elderly and immunocompromised persons. The formulation and clinical trials of new specific vaccine for COVID-19 may take more than 6 months' time. Within this time, COVID-19 may cause the death of lakhs of people around the world. In this scenario, searching for an old vaccine which is having some protective effect against COVID-19 is easy solution for saving the life of many people. The present article reviews the features of Polio vaccine which can combat COVID-19.

Keywords:- *COVID-19; polio vaccine; picornavirus-like supercluster; OPV; enteroviruses.*

I. INTRODUCTION

COVID-19 is a highly infectious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). SARS-CoV-2 is large RNA virus with envelopand it belongs to genera Beta-coronavirus under the *Coronaviridae*family. It is having a single linear RNA segment with around 30000 nucleotides long and it is a positive-sense single-stranded RNA (+ssRNA). It is the seventh known coronavirus to infect people, after 229E, NL63, OC43, HKU1, MERS-CoV, and SARS-CoV [1]. The size ofvirion is approximately 50–200 nm in diameter. SARS-CoV-2 also having four structural proteins like other coronaviruses, known as the S (spike), E (envelope), M (membrane), and N (nucleocapsid) proteins. The N protein holds the RNA genome, and the S, E, and M proteins together create the viral envelope [2]. Protein modeling experiments on the spike protein of the virus suggested that SARS-CoV-2 has high affinity to the receptor angiotensin converting enzyme 2 (ACE2) on human cells to use them as a mechanism of cell entry [3]. Studies have shown that SARS-CoV-2 has a higher affinity to human ACE2 than the SARS virus strain [4].

As of May 11, 2020, 4,132,365 cases and 283,387 deaths due to COVID-19 had been reported worldwide [5]. At present there is no specific effective vaccination and medicine developed for COVID-19. Even if new effective vaccine discovered for COVID-19, at least minimum 6months are required for completing all the necessary steps before providing to public people. Within this time, COVID-19 may cause the death of lakhs of people around the world. In this scenario, searching for an old vaccine which is having some protective effect against COVID-19 is easy solution for saving the life of many people.

Poliovirus (PV) is an enterovirus and also known as Enterovirus C whichbelongs to the *Picornaviridae*family. It is the etiological agent of poliomyelitis. Poliomyelitis is caused by three serotypes of poliovirus. Polio vaccination is a more than 50 yrs old vaccination using in all over the world. Mainly, there are two types of polio vaccines which are Inactivated Polio Vaccine (IPV) and Oral Polio Vaccine(OPV). The worldwide use of oral polio vaccines (OPVs) since the 1960s has resulted in three things.First thing is the eradication of wild poliovirus type 2 (WPV2), second thing is the lack of WPV3 detection since November 2012 and third thingis the confinement of WPV1 to areas of Afghanistan, Nigeria, and Pakistan by 2016[6,7]. The OPV vaccine was easier to administrate and provide a herd effect by inducing long-lasting protective systemic, humoral and cellular immunity as well as local mucosal protectionfrom PV infection. One drawback of OPV is that Sabin strains are genetically unstable. In rare cases, the attenuated strains can revert to neuro virulence while replicating in the vaccinee which cause vaccine-associated paralytic poliomyelitis (VAPP). In addition, in populations with low immunity against PV, the attenuated vaccine viruses can also give rise to neurovirulent and transmissible vaccine-derived poliovirus (VDPV) strains which have caused outbreaks in 17 countries [8,9].

The OPV and IPV have successfully eliminated poliomyelitis in most parts of the world and both vaccines differ in the induction of mucosal immunity, which is important for protection against poliovirus infection (or reinfection). Mucosal immunity is essential for reducing virus circulation [10,11]. Trivalent OPV (tOPV) and IPV protect against all three types of polio viruses (types 1, 2 and 3). Bivalent OPV (bOPV) targets type 1 and type 3, but not type 2. At the mucosal surfaces, IgA antibodies are the first line of defense against invasion of ingested pathogens, such as poliovirus. OPV is more efficient than IPV in inducing IgA [12,13]. The mucosal immunity provided by poliovirus-specific IgA has been demonstrated only in persons who have been in contact with live poliovirus, either wild type or OPV [14,15]. The present review is to establish relationship between the type of immunity behind children and young people against COVID-19 with polio vaccination and also to suggest polio vaccine for combating COVID-19.

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II. RELATIONSHIP BETWEEN AGE AND COVID-19 FATALITY RATE

The worldwide fatality rate of COVID-19 shows that fatality rate decreases with decreasing age. Age is a factor of defense against COVID-19.The estimated overall death rate due to COVID-19 was 0.66% which wasincreasing sharply to 7.8% in people aged over 80 and decreasing to 0.0016% in children aged 9 and below [16]. There are many studies show that children and young people are having less complication with COVID-19 than old age people. COVID-19 is not able to establish disease in majority of children.Gudbjartssonand colleagues conducted a population-based study in Iceland and they found that children under 10yrs of age and females had a lower incidence of SARS-CoV-2 infection than adolescents or adults and males [17]. As per worldwide fatality data of COVID-19, it is seen that the fatality rate is very low in young people of age below 18 yrs. Normally viral respiratory infections badly affect children below 5yrs and elderly persons above 65yrs of age. The immunity in children and young people indicates the presence of an adaptive immunity from childhood vaccination or infections.

III. RELATIONSHIP BETWEEN SEX AND COVID-19 FATALITY RATE

As per the available data of COVID-19 confirmed cases, men are dying at a higher rate than women across the countries. The death rate of men in Italy and China are more than double those of women. Around 61% of patients who die in New York city are men. The relationship with sex and COVID-19 fatality rate indicate a role of adaptive immunity against COVID-19.Stephanie and colleagues reported that the role of sex to modulating vaccine induced immunity has gained attention over the last years. Specifically, females typically show higher antibody responses and experience more adverse events following vaccination than males. This enhanced immune reactogenicity among females givewoman more resistant to infectious diseases. It alsocontributes to higher incidence of autoimmunity among females [18]. Sex differences in immune responses change for the duration of life and are influenced by both the age and reproductive status of an individual. The differential regulation of immune responses between the sexes are contributing by Sex chromosome genes and sex hormones, including oestrogens, progesterone and androgens. The Sex differences in immune responses which make a differential susceptibility in males and females towards autoimmune diseases, malignancies and infectious diseases, as well as affecting the outcome of vaccination [19].

IV. ASYMPTOMATIC CARRIERS OF COVID-19

Robert Redfield, the director of the Centres for Disease Control and Prevention(CDC), told that 25% of people infected with COVID-19 don't show any symptoms or fall ill but they can still transmit the illness to others. The CDC had tested for COVID-19 patients on the Diamond Princess cruise ship, which was quarantined in Japan in February 2020. CDC found that 712 tested positive out of the 3,711 people onboard and also noticed that almost 50% of them had no symptoms at the time of testing. Haiyan Q and colleagues reported that large proportion of asymptomatic children indicates the challenge in identifying paediatric patients who do not have clear epidemiological information, leading to a risky situation in community-acquired infections[20].A small study among Japanese ex-pats evacuated from Wuhan in February 2020 revealed that 30.8 % of people who tested positive for COVID-19 showed no symptoms. There are many reports state that most of the children and young people positive for COVID-19 are asymptomatic.Asymptomatic children and young people in the communities are a sign that SARS-CoV-2 virus cannot establish a disease in most of the children and young people. In the human body, prior infection or vaccination, allow rapid, specific mobilization of defenses to prevent disease symptoms, promote rapid clearance of the virus, as a result, most secondary viral challenges are asymptomatic [21]. SARS-CoV-2 is new virus, so the immunity may be due to cross reacting antibodies of previous infection or vaccinations.

V. RELATIONSHIP WITH CO-MORBIDITY AND COVID-19 FATALITY RATE

Worldwide COVID-19 fatality reports show that 8 out of 10 deaths due to COVID-19 are occurring in persons with at least one underlying co-morbidity, in specific those with cardiovascular diseases, hypertension, diabetes and also with a range of different chronic underlying conditions. Memory B cell activation is one of the important step in immunity produced by vaccination. Andrea M and colleagues reported that activation of B memory cell can cause heart failure in heart disease patients [22]. Xiao Z and colleagues reported that in obese diabetic subjects, B cell adaptive response is impaired and potentially contribute to overall higher inflammation [23]. The COVID-19 fatality data of world shows that the low immunity persons in young age group like pregnant woman, HIV patients and organ transplanted persons were shown more recoveries than people above 60yrs of age. Nan Yu and colleagues reported the clinical features and obstetric and neonatal outcomes of pregnancy with COVID-19 in Wuhan, China. Seven pregnant women with COVID-19 were examined and the onset symptoms were similar to those reported in non-pregnant adults with COVID-19[24]. In pregnant woman, evidence indicates that the maternal immune system may tolerate fetal antigens by suppressing cell-mediated immunity while retaining normal humoral immunity. The above facts show that immunocompromised persons in younger age groups

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may have some humoral immunity against COVID-19 and this humoral immunity may be due to previous vaccinations or past infections.

VI. WHAT WILL MAKE POLIO VACCINATION AS A GOOD OPTION FOR COMBATTING COVID-19?

The factors described above regarding fatality rate of COVID-19 indicate the presence of an acquired immunity in children and young people and this immunity may be acquired from previous infections and past vaccinations. Human coronaviruses (HCoV) are the second most prevalent cause of the common cold and account for 15–30% of proven viral infections. Coronaviruses like 229E, OC43, NL63 HKU1 and SARS-CoV can infect human and produce respiratory infections in children and adults. Because of these infections, they may have antibodies against these coronaviruses and these antibodies may be can cross react with SARS-CoV2 antigens. childhood vaccinations may be also playing an important role in giving protection against COVID-19 in children and young people and many of these vaccinations are compulsory vaccinations for all the infants born in different countries. There are many childhood vaccines may be contributing immunity especially vaccines contain single stranded RNA viruses like polio vaccine, MMR (Measles, Mumps and Rubella) vaccine etc.

VII. PICORNAVIRUS-LIKE SUPERCLUSTER VIRUSES

The positive-sense RNA of viruses is directly translated into one or more polyproteins which are further cleaved by virus proteases into mature or intermediate viral proteins. Some positive-sense RNA viruses can be classified further into the picornavirus-like supercluster based on genetic analysis of RNA-dependent RNA polymerase. The above supercluster includes viruses belonging to the *Picornaviridae*, *Caliciviridae*, and *Coronaviridae*families[25]. The main human viruses included in *Picornaviridae* family are human rhinovirus (HRV), enterovirus 71 (EV71), poliovirus (PV), foot-and-mouth disease virus (FMDV), hepatitis A virus (HAV), and porcine teschovirus (PTV) [26]. The viruses included in the picornavirus-like supercluster is having a common feature that they possess a viral 3C or 3C-like protease (3Cpro or 3CLpro) respectively. These proteases are responsible for the majority of cleavages of the corresponding viral polyprotein into mature or intermediate virus proteins [27,28]. The 3Cpro and 3CLpro share several common characteristics, including a typical chymotrypsin- like fold which contains a cysteine residue as an active site nucleophile in the catalytic triad (or dyad), composed of cysteine, histidine, and glutamic acid (or aspartic acid) residues and a preference for a glutamic acid or glutamine residue at the P1 position on the substrate (in the nomenclature of Schechter and Berger) [29].

The *Picornaviridae* family includes a large group of non-enveloped viruses that have a major impact on human health. The viral RNA genome contains one open reading frame encoding a single polyprotein that can be further processed by viral proteinases. The crucial 3C proteinases (3Cpros) of picornaviruses share similar spatial structures and it plays a significant role in the viral life cycle and virus host interaction. The proteinase and RNA-binding activity of 3Cpro are important for the viral polyprotein processing and the initiation of viral RNA synthesis. It also can induce the cleavage of certain cellular factors required for transcription, translation and nucleocytoplasmic trafficking to modulate cell physiology for viral replication. Due to interactions between 3Cpro and these essential factors, 3Cpro is also involved in viral pathogenesis to support efficient infection[30]. SARS-CoV-2 contains a 3C-like protease (3CLpro) analogous to the 3Cpro of picornaviruses, responsible for processing two overlapping polyproteins, pp1a and pp1ab. Other members of human Coronaviruses including SARS-CoV ,CoV- 229E, CoV-OC43, CoV-HKU1, and CoV-NL63 also require a 3CLpro in the maturation of viral proteins. The main picornavirus-like supercluster viruses use in vaccinations are Poliovirus and Hepatitis A virus. Because of this similarity in proteases of picornaviruses and coronaviruses, scientists are trying to develop a common drug which can target these proteases for preventing infections caused by these viruses.

VIII. NEGATIVE INTERACTION BETWEEN CORONAVIRUSES AND ENTEROVIRUSES IN VIRAL RESPIRATORY DISEASES

The main causes of worldwide morbidity and mortality are due to Acute respiratory infections (ARIs), mainly in children during the first years of life [31]. The viruses most commonly associated with ARIs are parainfluenza viruses 1-4 (PIV 1- 4), influenza viruses A and B (IFV A and IFV B), respiratory syncytial virus (RSV), human adenovirus (ADV), human coronavirus (HCoV), human rhinovirus (HRV), enterovirus (EV), human bocavirus (HBoV) and human metapneumovirus (HMPV). Many researchers tried to isolate causative viral agents of respiratory illness and table I shows the different viruses isolated from individuals belongs to different parts of the world. One of the most common and important diseases in children is respiratory tract viral infection. Respiratory infective viruses can be detected with help of Polymerase chain reaction (PCR) tests. The interpretation of clinical significance of PCR positivity is difficult due to the reflection of a past, imminent or active asymptomatic infection because of their high sensitivity. Many times, single respiratory viruses have been detected in samples from children with symptoms. However, other respiratory viruses can also be co-detected simultaneously which make difficult to interpret the clinical significance of these findings [32]. It is seen that many viruses are co-detected with other viruses and table II shows co-detection of HCoV with other viruses.HCoV was detected more times with viruses like RSV, RHV, IFV, PIV, HMPV, ADV and HBoV and in rare situations,HCoV co-detected with enteroviruses(EV). Ren L and colleagues co-detected one

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case of HCoV along with EV after screening 3434 numbers of ARI patients aged between 26 to 65yrs old [33]. HCoV infections are less in children compared to adult persons. Johanna N K and colleagues conducted a study in which they taken nasopharyngeal aspirates from 107 children without concurrent respiratory symptoms and analyzed sample with help of RT-PCR. They found that Thirty-one (29%) of the nasopharyngeal aspirates were positive for viral RNA,18% for HRV, and 11% for EV RNA. HCoV RNA was not found in any of the children [35].

The respiratory viral diseases follow a seasonal cycle which has been widely recognized for thousands of years. The annual epidemics of the common cold and influenza disease catch the human population every year in the winter season in temperate regions. Most of the respiratory viruses have seasonal oscillation of their outbreaks as per the epidemiological studies in temperate regions [48]. IFV, HCoV, and RSV clearly show peak incidences in the winter months (leading to them sometimes being called winter viruses) [49-54]. Conversely, ADV, HBov, HMPV, and HRV can be detected throughout the year (all-year viruses) [53-55]. For some enteroviruses, detection frequency and case numbers increase in summer (summer viruses) [56,57]. Although infection rates peak in spring and fall, disease severity caused by rhinovirus infection increases in winter [58,59]. Furthermore, PIV shows a type-specific pattern of seasonal circulation[60] and the details of seasonal variation of respiratory viruses shown in fig.1[48]. EV (Non rhino enteroviruses) infections are usually seen between June to October and HCoV infections are seen between November to April [48]. Both viral infections are rarely seen together. This may be due to many factors. Replication conflicts among those respiratory viruses can contribute to the non-overlapping peak incidence with respect to one another. The above negative interaction between EV and HCoV are not studied deeply. Similar studies have done with other respiratory viruses. The epidemiological observation of interference between respiratory viruses has been shown that influenza viruses and RSV do not share peaks during the same period even though both are prevalent in winter [61]. A recent study by using statistical approaches shows that a strong negative interaction between seasonal IVF A virus and RHV at both the population and individual levels [62]. The mainly proposed possible mechanisms of the interference between respiratory viruses includes the disruption of cell surface viral receptor, cell death, or the host interferon (IFN) responses [63,64]. Protective antibody-driven interferences have also been proposed for the conflict of genetically close viruses such as PIV, HMPV, and RSV [65].

IX. POLIO VACCINE AND COVID-19

IPV is a “killed” vaccine. Hence, it is not associated with the risks of VAPP or VDPV outbreaks. Consequently, there is a growing consensus that OPV vaccination must cease alongside a transition to worldwide IPV vaccination as the Global Polio Eradication Initiative (GPEI) approaches the endgame. As of April 2013,47 countries were using IPV only for polio vaccination,24 countries and 6 territories were using OPV/IPV and 126 countries were using only OPV. The above countries details are shown in fig. 2[66]. USA has transferred from OPV to IPV in the year 2000 and they are not using OPV at present. Most of the European countries like UK, Italy, Spain are using only IPV. The countries like Mongolia, Fiji, Vietnam etc. had not introduced IPV even in 2015.A comparison is made between two groups of countries and named them as Group-A & Group-B against confirmed COVID-19 cases and death due to COVID-19. Under Group-A, the countries selected which had not introduced IPV as on 2015. Group-B consists of the countries which have completely transferred from OPV to IPV as on 2013.The details of above comparison are given in table III and the data show that number of COVID-19 cases and deaths were more in Group-B countries compared to Group-A. The countries like Russia and Brazil were using both IPV and OPV for polio vaccination in 2013 and number of confirmed COVID-19 cases are high in these counties compared to group-A countries. The reason behind this phenomenon may be OPV give good cellular immunity than IPV. OPV became the favored vaccine in many countries due to the ease of oral administration, the lower costs of production, and its ability to replicate in the gastrointestinal tract, stimulating both local secretory IgA(SIgA) in the pharynx and gastrointestinal tract and circulating IgG [67,68]. Mostly SIgA are specific to antigen. But there are studies show that secretion of non-specific SIgA on mucosal surfaces for the protection against different pathogens like bacteria, virus and parasites [69,70]. The lack of mucosal immunity in oral and intestine in case of IPV, may have triggered asymptomatic transmission of COVID-19.

PV in OPV can pass to other persons from vaccinee through stool, saliva etc. Hence, children getting OPV can immunize a society where they live. The people living in OPV using countries will continuously in touch with vaccine PV and thus a strong immunity will develop against PV. Because of this, countries which provide only OPV can produce more polio immunized persons compared to IPV only using countries. The Inactivation step in IPV production by formaldehyde cause the partial destruction of specific antigenic epitopes which result in some loss of immunogenicity [71]. IPV produce immunity only to person get vaccination. In countries like USA,UK, children only getting IPV vaccination and they cannot transfer their immunity to others. The life styles of people living in different countries also play an important role in circulation of vaccination’s PV. In developed countries which use still OPV, old age people may not be staying with grandchildren, mostly in old age homes which also make them less contact with circulation PV. The country like New Zealand belongs to Group-B is having less COVID-19 confirmed cases and deaths which may be due to less population size and the speedy execution of remedial actions such as complete lockdown and social distancing for preventing thespreading of COVID-19 in that country.

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X. HOW LONG POLIO VACCINATION IMMUNITY CAN LAST?

Normally people say that polio vaccine immunity is lifelong. Many studies prove that 99% of polio vaccine immunized individuals will protect for minimum 18 years from polioviruses. The last booster dose of polio vaccine may be giving at age of 4-6yrs and therefore a high immunity against poliomyelitis will be present in individuals upto the age of 22-24 yrs. Generally, the people with immunity against poliovirus show poliovirus antibody cutoff of ≥1:8 which has been known to be protective against wild or Sabin attenuated poliovirus strains. Subhash C and Nirmala A reported in a study that out of 150 individuals aged 20–50 yrs, 10(7%) had titers below the minimum protective level of ≥1:8 against vaccine-derived poliovirus strain [72]. Vincenzo Baldo and colleagues conducted a serological survey among 318 young adult University Students in Northern Italy for assessing the immunological status against polioviruses based on their country of origin. The neutralizing antibody titration in tissues cultured on microplates was used for assessing the immunity to poliomyelitis. They found that poliovirus antibody titer was <1:8(seronegative) in 26.7% against poliovirus type 1, 7·2% against type 2, and 22·6% against type 3 in the study population. Out of 318 individuals in the above study, 219 (68·9%) were Italian and 99 (31·1%) were from outside the European Union (EU). The individual with poliovirus antibody titer ≥1:8(seropositive) to polioviruses 1 and 3 decreased significantly with older age. This age-related decrease in antibody titer against poliovirus was more in Italian group than among the non-EU individuals [73]. The above studies show that the old age persons above 60 years of age may not have high protective immunity against poliomyelitis. If polio vaccination is having some control effect on COVID-19, old age people (above 60yrs) may not get benefit from polio vaccine. It is observed that the COVID-19 death rate among adults aged between 25-45 yrs old is more in underdeveloped and developing countries compared to developed countries which may due to late introduction of polio vaccination in underdeveloped and developing countries.

XI. HOW POLIO VACCINE WILL PROTECT FROM SARS-COV-2?

Polio vaccine will boost both innate immunity and adaptive immunity. Innate immunity is the first line of defense mounted against the virus before the adaptive immune system is generated. One of the essential functions of the innate immune system is recognition of evolutionarily conserved microbial structures, known as pathogen-associated molecular patterns (PAMPs). The main molecular structures of different microorganisms recognized by innate immune system as PAMPs are glycoproteins, lipopolysaccharides, proteoglycans, and nucleic acid motifs. These are essential for the survival or infectivity of the microbe.The human Immune cells have proteins for sensing the presence of microbial invasion known as germ line-encoded pattern recognition receptors (PRRs) [74]. The PRRs sense the PAMPs on microorganism inversed in to the human body which upregulates the transcription of genes involved in inflammatory responses like the genes encode pro-inflammatory cytokines, chemokines, type I interferons (IFNs), and antimicrobial proteins. The expression patterns of the inducible genes differ among activated PRRs [75]. The central role in induction of antiviral responses is by the Production of type I IFNs (primarily alpha IFN [IFN- α] and IFN-β) as they trigger the transcription of many IFN-inducible genes. These genes influence the protein synthesis, growth regulation, and apoptosis. Type I IFNs also play an important role as link between innate and adaptive immune responses by enhancing the maturation of dendritic cells (DCs), cytotoxicity of natural killer (NK) cells, and the differentiation of virus-specific cytotoxic T lymphocytes [76].

One important component of the innate immunity boost by polio vaccine for combatting COVID-19 is the activation of PPRs which can sense foreign single stranded RNA. Because both PV and SARS-CoV-2 are +ssRNA viruses. One of the most extensively studied family of PRRs is Toll-like receptors (TLRs). At the time of infection, TLRs play an important role in the initiation of an antiviral response. The human's TLR multigene family consist of 10 members. Among these, TLR2, -3, -4, -7, and -8 are important for the recognition of structural components of RNA viruses, including viral double-stranded RNA (dsRNA), single-stranded RNA (ssRNA), and surface glycoproteins. The majority of the TLR family members are being expressed at the cell surface. The TLR3, -7, and -8 are present in the intracellular compartments, such as the endoplasmic reticulum(ER), endosomes, lysosomes, and endolysosomes and they recognize nucleic acid motifs of viruses[74].Toll-like receptor 7 (TLR7) present in the endosomes of plasmacytoid dendritic cells (pDCs) and B cells senses ssRNA oligonucleotides containing guanosine- and uridine-rich sequences on RNA of the viruses [74,77]. Toll-like receptor 8 (TLR8) is also recognizes ssRNA in the viruses like HIV and other RNA viruses. TLR8 is phylogenetically and functionally closely related to TLR7. TLR8 is preferentially expressed in myeloid DCs and monocytes. Both poliovirus and SARS-CoV-2 virus produce dsRNA as replication intermediate of ssRNA which can be recognized by Toll-like receptor 3 (TLR3).TLR3 is present both intracellularly and on the surfaces of NK cells, epithelial cells, and fibroblasts. It also presents in the intracellular compartment of macrophages, B lymphocytes, and cDCs. Toll-like receptor 4 (TLR4) also play a role in antiviral defense to RNA viruses by sensing the fusion protein and the envelope proteins of RNA viruses.

RIG-I-like receptors (RLRs) are the another PPRs which sense the viral RNA species and they are cytosolic proteins. The retinoic acid-inducible gene I product (RIG-I) and melanoma differentiation-associated antigen 5 (MDA5) are the two members of RLRs which help in the recognition of viral RNA species [78,79].The ssRNA/dsRNA (ss/dsRNA)-binding C-terminal domain (CTD) of Retinoic

acid-inducible gene I (RIG-I) act as a repressor domain (RD) when it is in unbound condition [80]. Two repeats of a cysteine-aspartic protease (caspase)-recruiting domain (CARD)-like region at the N terminus of RIG-I are exposed when binding to viral RNA structures produced during viral replication and this event help RIG-1 to interact with other CARD containing proteins to trigger downstream signaling mechanisms. The virus-associated RNA species, including dsRNA and 5′-triphosphate ssRNA can be specifically recognized by RD [81]. RD can differentiate self ssRNA, such as mRNA and tRNA from viral RNA by having an exposed 5′-triphosphate. Melanoma differentiation-associated antigen 5 (MDA5) is very similar to RIG-I, and they are having the same overall domains. MDA5 exposes a CARD when binding to long dsRNA fragments which initiates cytokine and type I IFN production similarly to RIG-I. The RLR signaling pathways for inducing gene expression for the synthesis and release of type I Interferons (IFNs) and pro-inflammatory cytokines in order to launch an antiviral inflammatory responses similar to the pathways also utilized by the TLRs.

Type I IFNs like IFN-α and IFN-β produced by action of PPRs will help in blocking viral replication and also activate the immune response by enhancing T-cell recognition of infected cell. The best inducer of IFN-α and IFN-β production is dsRNA produced as the replicative intermediates of RNA viruses. One dsRNA molecule per cell is sufficient to induce the production of interferon. The proteins like nsp1 and nsp3 present in HCoV can interfere with function and production of interferons as mechanism to escape from host defense. Hence, boosting of innate immunity for fighting against ss+RNA viruses is very important for providing immunity to COVID-19. In old age people, innate immune system will be weak and take more time to recognize the SARS-CoV-2 virus. Within this time, virus will multiply and spread to neighboring cells. The delay in the action of innate immunity will cause spreading of virus to numerous cells and later vigorous inflammatory response by immune system will cause cytokine storm which leads to death of person. Here, the polio vaccine plays a role in activation of innate immunity and indirectly adaptive immunity against COVID-19. The live attenuated poliovirus in OPV will multiply in epithelial cells in intestine and nasopharynx will increase innate immunity to ss+RNA viruses by giving a training to immune system for combating COVID-19. The non-specific innate immunity generated by OPV may be lasting for maximum 2-3months only. If old age people infect with SARS-CoV-2 within this 2-3 months' time, their immune system can easily combat COVID-19 without serious symptoms.

OPV also provide mucosal immunity by producing more amount of SIgA on mucosal surfaces. Mostly SIgA are specific to antigen. But there are studies show that secretion of non-specific SIgA on mucosal surfaces for the protection against different pathogens like bacteria, virus and parasites [70,71]. The ACE2 and PVR, cell receptors of PV and SARS-CoV-2 are present in respiratory cells and intestinal cells. This feature may give a competition between two viruses. Poliovirus receptor (PVR) also known as cluster of differentiation 155(CD155) is a Type I transmembrane glycoprotein in the immunoglobulin superfamily. It is recently emerging as a promising target in immunotherapy. There are studies show that PVR enhances the existing anti-tumor responses and it also involved in important cellular processes, such as adhesion, contact inhibition, migration, proliferation, and the immune response [82].

XII. CONCLUSION

The relation between COVID-19 fatality rate with age, sex and Co-morbidity show a role of an adaptive immunity by past vaccination. There may be many childhood vaccinations play role in adaptive immunity against COVID-19. The priority should be given to Polio vaccine as poliovirus has few interesting features with respect to SARS-C0V-2 which includes both viruses belong to Picornavirus-like supercluster virus family andthe negative interaction between EV and HCoV in establishing viral respiratory illness. These features show the possibility of polio vaccine to combat COVID-19 as it contains an enterovirus. Additionally, polio vaccine boosts innate immunity and adaptive immunity by giving training to immune system for combatting ss+RNA viruses. The non-specific innate immunity generated by OPV against COVID-19 may be lasting for maximum 2-3months only.

OPV show better immune responses compared to IPV due to the presence of attenuated poliovirus. As the science behind polio vaccine is well known, it can be administered to people without more clinical trials, and negligible side effects make this vaccine harmless. Till a specific vaccine develop for COVID-19, polio vaccine may be considered as an option for combatting COVID-19. The above fact may be considered by the medical field for assessing its suitability and acceptability as a research trial.

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Place of study & year	Study group	EV	HCoV	RSV	PIV	HMPV	HBoV	RHV	IFV	ADV	Reference
Taiwan 1997-1999	6986 children (<12yrs)	889	0	120	137	-*	-	-	562	277	Huey et.al [34]
Finland 1999-2000	107 children	12	0	-	-	-	-	19	-	-	Johanna et.al [35]
France 2003-2004	449 children	6	11	158	9	17	-	91	19	5	Franc et.al [36]
UK 2001-2006	815 samples from 198 children	-	47	89	44	17	-	394	35	13	Merci et.al [37]
Spain 2004-2006	338 children (<3yrs)	-	22	67	38	39	48	46	25	2	Gustavo et.al [38]
China 2005-2007	5808 RTI patients (>14yrs)	188	65	30	252	19	-	376	1119	51	Ren et.al [39]
Malaysia 1982-2008	10269 children (<5yrs)	-	-	1913	357	-	-	-	297	141	Chee-Sieng et.al [40]

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Netherlands 2006–2009	142 children (2yrs)	10	8	104	6	9	6	43	7	13	Kim et.al[41]
Brazil 2007-2009	162 individuals	2	5	12	10	5	5	14	23	1	Ronaldo et.al [42]
China 2009-2010	720 children (<14 yrs)	29	18	35	128	26	29	132	95	10	Yanqin et.al [43]
Greece 2010-2011	611 children (<14 yrs)	12	0	225	118	15	25	73	56	31	Kouni et.al [44]
India 2011-2013	300 children (<5 yrs)	1	3	61	22	14	11	54	42	39	Pravakar et.al [45]
Saudi Arabia. 2014-2015	2266 Children (<14yrs)	92	79	336	120	99	171	644	146	344	Saleh et.al [46]
EV, enterovirus; HCoV, coronavirus; RSV, respiratory syncytial virus; PIV, parainfluenza virus; HMPV, human metapneumovirus; HBoV, human bocavirus; RHV, human rhinovirus;IFV, influenza virus; ADV, human adenovirus. *No data available											

Table 1:- Details of respiratory viral agents isolated from nasopharynx swab samples.

Place of study & year	Study group	EV + HCoV	RSV + HCoV	PIV + HCoV	HMPV + HCoV	HBoV + HCoV	RHV + HCoV	IFV + HCoV	ADV + HCoV	Reference
Italy 2003-2004	2,060 children (<15yrs)	-*	7	1	4	-	2	3	5	Susanna et.al [47]
Spain 2004-2006	338 children (<3yrs)	-	2	1	1	1	1	1	-	Gustavo et.al [38]
China 2005-2007	5808 RTI patients (>14yrs)	1	-	2	-	-	4	1	-	Ren et.al [39]
Brazil 2007-2009	162 individuals	0	1	0	1	0	0	0	0	Ronaldo et.al [42]
China 2009-2010	720 children (<14yrs)	0	1	4	1	1	3	1	2	Yanqin et.al [43]
India 2011-2013	300 children (<5yrs)	0	2	0	0	0	0	2	0	Pravakar et.al [45]
*No data available										

Table 2:- The reports of co-detection of coronaviruses with other respiratory viruses.

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Group A			Group B		
Country	No. of Confirmed cases	No. of deaths	Country	No. of Confirmed cases	No. of deaths
Cambodia	122	0	USA	1367568	80652
Cook Islands	0	0	UK	223060	32065
Fiji	18	0	Italy	219070	30560
Kiribati	0	0	Spain	227436	26744
Lao PDR	0	0	Germany	172010	7580
Mongolia	42	0	Denmark	10429	529
Nauru	0	0	Norway	8105	219
Papua NG	8	0	Sweden	26322	3225
Philippines	11086	726	Finland	5962	267
Samoa	0	0	Ireland	22996	1458
Solomon Is.	0	0	Portugal	27581	1135
Tonga	0	0	France	139063	26380
Tokelau	0	0	Malaysia	6726	109
Tuvalu	0	0	South Korea	10909	256
Vanuatu	0	0	Japan	15847	633
Vietnam	288	0	New Zealand	1147	21
Libera	0	0	Australia	6927	97
Ghana	4263	22			
Burkina faso	751	49			
Angola	43	2			
Zambia	267	7			
Zimbabwe	36	4			
Tanzania	509	21			
Rwanda	284	0			
Burundi	15	1			
Egypt	9400	525			
Eritrea	39	0			
Uzbekistan	2453	10			
Sierra leone	338	19			
Azerbaijan	2519	32			

Group A countries; countries selected which had not introduced IPV as on 2015.
Group B countries; countries selected which had completely transferred from OPV to IPV as on 2013.
Source: Ref. 5.

Table 3:- Confirmed COVID-19 cases in different countries as on 10.05.2020.

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Month	June	July	Aug.	Sep.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May
Winter virus						Influenza virus						
							HCoV					
						RSV						
All-year virus	Adenovirus/HBoV											
Type-specific	PIV3		PIV1									
Spring	HMPV											
Spring/Fall	Rhinovirus											
Summer virus	Enteroviruses											

HCoV, coronavirus; RSV, respiratory syncytial virus; HBoV, human bocavirus; PIV1, parainfluenza virus 1; PIV3, parainfluenza virus 3; HMPV, human metapneumovirus. Source: Ref. 48.

Fig 1:- Schematic of seasonality of respiratory virus infection in temperate regions.

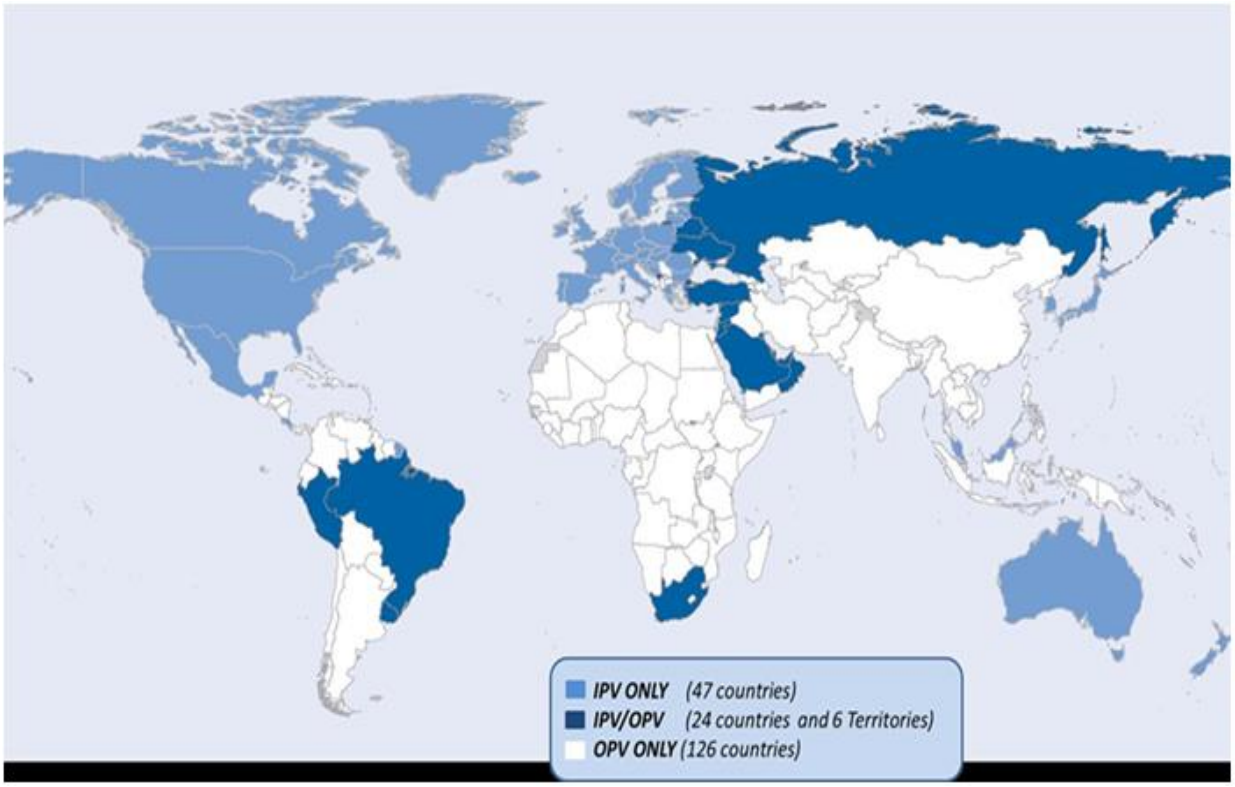

Fig 2:- Countries which were using IPV and OPV as on 2013. Source: Ref. 66.