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# Pediatric Infectious Diseases

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Review Article

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ABSTRACT

Pediatric infectious diseases are becoming a major concern now a day as there is an increase in the number of emerging microbial strains which are affecting children. It is time to wake up and take care of our future generation as we have already lost many infected children by the grace of these infectious diseases. This article is containing basic information about various types of infectious diseases, their transmission and control.

## INTRODUCTION

Disease can be defined as an abnormal condition of particular function or structure within the body of particular individual and makes health condition worse. These diseases are mainly categorized as non-infectious and infectious diseases. Many of the non-infectious diseases are long-lasting and they will not transmit from one to another. Infectious diseases generally causes through pathogenic microorganisms of various kinds such as bacteria, parasites, viruses or fungi. Infectious diseases are contagious in nature and spread from one person to another by direct or indirect contact.

There has been the presence of many infectious diseases from ancient times for which people got suffered, affected and died too [1-3]. There are certain types of infectious diseases which have more dominance in both infantile and childhood stages of human life and these are generally referred as pediatric infectious diseases. Amoebiasis, AIDS, Candidiasis, Chagas disease, Cholera, Typhoid, Dental

caries, Diphtheria, Infectious mononucleosis, Influenza, Measles, Meningitis, Mumps, Pneumonia, Polio, Whooping cough, etc.

## AMOEBIASIS

Amoebiasis is a diarrheal infection, caused by parasitic protozoan named Entamoeba histolytica (anaerobic pathogen) and it has been integrated with malnutrition of children [4]. Acute diarrhea is the most often gastroenterological infection and the main cause in childhood is dehydration. It can be manifested by a sudden occurrence of three or more watery stools a day lasting for 7-10 days, fourteen days at most. It mainly occurs until 5 years in children and until 2 years in neonates. Primary causes of Amoebiasis include gastrointestinal infections, bacterial, viral, alimentary intoxications and other factors [5]. Cutaneous Acanthamoebiasis is an infrequent infection in organ recipients and immunocompromised patients. Advanced diagnosis is important as cutaneous Acanthamoebiasis can disseminate to the CNS and cause granulomatous amoebic encephalitis, which is generally fatal [6].

## AIDS

Acquired immune deficiency syndrome (AIDS) is a disorder of immune system caused by virus named human immunodeficiency virus (HIV) [7]. Although there is evidence for the prevalence of Acquired Immunodeficiency Syndrome (AIDS) and Human Immuno-Deficiency Virus (HIV) is declining or stabilising in some countries, their impact remains a significant health care challenge that affects communities, families and health care systems [8]. Primary target of the HIV are CD4 cells and loss of these cells results in immune response weakening and renders the host susceptible to various infections, ultimately leading to AIDS. Success or failure of immunological treatment can be measured through an increase or decrease count of CD4 cells [9]. Oral cavity is not considered as the primary a route of transmission for HIV virus, with some exceptions like oral sex or breastfeeding [10].

## CANDIDIASIS

Candidiasis is the most common fungal disease affecting skin, mucosa, nails and internal organs of children. There are more than one hundred species of Candida and almost twenty species are pathogenic for humans [11]. Infections due to Candida species remains as regular infections in neutropenic patients, but Chronic Disseminated Candidiasis is a different form of Candida infection, with the involvement of the spleen, liver and also kidneys in rare cases [12]. Candida albicans is fungal species which commonly colonizes human mucosal surfaces. C. albicans can proliferate and cause an array of infections that are often life-threatening when host defences are weak [13]. Autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy is an infrequent, autosomal recessive disease

which can be characterized by the onset of mucocutaneous candidiasis in young adulthood, followed by adrenal insufficiency and hypoparathyroidism in a subset of patients [14]. Prevalence of Candida infection is increasing day by day, leading to higher frequency of diseases like candidaemia and candidiasis [15]. Protection against disseminated candidiasis can be achieved by vaccination with Met6 peptide induced antibody production [16]. Treatment of oral or esophageal candidiasis includes a first-line antifungal drug called Fluconazole [17].

## CHAGAS DISEASE

Chagas disease, can also be called as American trypanosomiasis, generally caused by hemoflagellated protozoan pathogen, Trypanosoma cruzi which generally transmits to humans by triatomine bugs [18-21]. Chagas disease was discovered by Brazilian scientist Carlos Chagas in 1909 [21,22], and it is named after that scientist chagas. Diagnosis of chronic infection by Trypanosoma cruzi relies on serological methods. Chagas disease can affect esophagus, bowel systems and heart. These organs lose normal function after swelling. Heart is the targeted system with congestive heart failure, symptoms include feet swelling, shortness of breath and heart palpitations [23]. There is no vaccine availability to prevent chagas disease and treatment is restricted to limited drugs, benznidazole and nifurtimox. There is a requirement of new effective drugs with lower side-effects [22].

## CHOLERA

Cholera remains a significant cause of mortality in childhood all over the world [24]. It is an acute watery diarrheal disease caused by gram-negative pathogen Vibrio cholera [25,26]. Vibrio cholerae can be found mostly in water, soil and other host organisms. It is an opportunistic pathogen and causative agent of multidrug-resistant nosocomial infections. It is also the main cause of chronic lung infections in patients who are diagnosed with cystic fibrosis [27-33].

Vibrio cholerae includes more than 180 serogroups and it is responsible for approximately 3-5 million cases and an estimated 1,20,000/annum worldwide. Cholera remains a major public health problem in few countries with poor access to proper sanitation and safe water [34,35]. Symptomatic infections include acute watery diarrhoea and vomiting, which can rapidly lead to dehydration and finally death if not treated immediately [36]. If rehydration management is not proper, it will lead to fatal stage within hours. Poor hygiene behaviours, inadequate water supplies and poor sanitation are largely responsible for cholera disease [37].

Treatment with prescribed antibiotics for patients who lost a large volume of stool during rehydration treatment is also recommended. There is an availability of many antibiotics like doxycycline (suggested for adults) and azithromycin (suggested for children and pregnant women). Antibiotics should be given

along with aggressive hydration to avoid secondary transmission of cholera. Patients resistance to tetracycline can be treated with doxycycline [38].

## TYPHOID FEVER

Typhoid fever is a food and water borne microbial infection caused by non-spore forming gram negative facultative anaerobic rod shaped bacterium called Salmonella Typhi [39,40]. It is one of the most serious forms of enteric fever with estimated number of more than 21 000 000 typhoid cases and 2,00,000 deaths [41]. It has more prevalence in developing countries because of poor sanitation and antibiotic resistance [42]. Typhoid fever is an important cause of morbidity with an estimated 12-33 million cases per year [43]. Culture techniques, serology and molecular techniques plays vital role in the diagnosis of typhoid fever [44]. General preventive methods like improved general public sanitation and clean water supplies may lead to typhoid free society [45].

## DENTAL CARIES

Dental caries is one of the most common preventable oral infectious disease affecting children all over the world [46-51]. There are many factors responsible for dental caries like poor oral hygiene, hereditary factors, concentration of bacteria in oral cavity, cariogenic diet and reduced level of fluoride in drinking water, hyperglycemia, dyslipidemia, fermentable substrates, microflora and saliva [52-56]. It is a complex, biofilm-induced disease involving oral bacteria and demineralization/remineralization process of an affected tooth [57].

Dental caries is a process of progressive demineralization in hard tissue of tooth surface by organic acids derived from sugar-containing or fermentable foods [58]. This disease develops in crowns and roots of children teeth, and arise as an aggressive tooth decay in early childhood that affects the primary teeth of infants and children [59]. Dental caries can be diagnosed by using a visual method with mouth mirrors, dental probes and daylight illumination [60]. If dental caries develops after the eruption of permanent dentition, it may lead to total damage and spread of infection throughout the body may also occur. Utmost care must be taken to reduce the formation of dental caries. Early diagnosis with proper treatment is also necessary [61,62].

## DIPHTHERIA

Diphtheria is an infectious disease caused by pathogenic rod shaped bacterium called Corynebacterium diphtheriae [63]. Trivalent combination vaccines against pertussis, tetanus and diphtheria infections have been widely used in pediatric immunization since 1940s, resulting in a significant decline in incidence and prevalance of pertussis [64,65].

## INFECTIOUS MONONUCLEOSIS

Acute infectious mononucleosis is generally caused by viruses such as γ-herpes virus, Epstein-Barr virus or human β-herpes virus and cytomegalovirus. Primary infection of mononucleosis appears predominantly in adolescents, children and young adults [66,67]. Symptoms start with arthralgia, subfebrility, malaise and myalgia, similar to other common upper respiratory tract infections [66,68,69]. After the prodromal symptoms, tonsillopharyngitis, fever, cervical lymphadenopathy, hepatosplenomegaly and leukocytosis can develop. The positive heterophil tests and clinical features are usually sufficient to diagnose Infectious Mononucleosis [60,70].

## INFLUENZA

Influenza is a respiratory tract infection caused by influenza virus. It is an RNA virus composed of three general types: influenza A, B and C according to nucleoprotein antigenicity [71]. Influenza virus belongs to the viral family called Orthomyxoviridae with seven or eight single-stranded, negative-sense RNA segments [72]. Influenza A viruses are the major pathogens compared with influenza B and C. Influenza viruses are causing approximately five million cases of severe illness, 250,000-500,000 deaths annually and remains as a major infectious disease and public health challenge with a global annual attack rate estimated at 5%-10% in adults and 20%-30% in children. Birds and Mammals are the major reservoirs of newly emerging pandemic influenza viruses and immunization process remains the most effective method to prevent influenza [73-75].

## MEASLES

Measles is a common infection in children which is vaccine-preventable, caused by Measles virus (single stranded, non-segmented, enveloped negative-sense RNA virus belonging to the family Paramyxoviridae [76-82]. Measles eradication is going to be a much difficult challenge [83] and there are no treatments available to kill measles virus. Treatment maily aims to clear the infection from immune system and to ease symptoms [84].

## MENINGITIS

Meningitis may be defined as an inflammatory response to infection (bacterial, viral) of cerebrospinal fluid. It can be characterized by sudden onset of febrile illness with signs and symptoms of meningeal involvement [85]. Most pathogenic microbes in humans are able to cause meningitis. However, Neisseria meningitides, Haemophilus influenzae and Streptococcus pneumoniae are the most common pathogens

that cause bacterial meningitis [86]. Although there is an availability of newer antibiotics and preventive strategies, meningitis remains an important source of mortality as it is one of the most significant infections in children it in developing countries. Meningitis is generally a complication of a primary bacteremia in children below 2 years of age [87].

## MUMPS

Mumps is an acute viral infection, generally characterized by enlargement of salivary and parotid glands. It is a most common disease in childhood [88,89]. Mumps virus belongs to the family Paramyxoviridae. It has an incubation period of 2-4 weeks and typically causes a prodrome of low-grade fever, parotitis and malaise. Neurologic complications are the most common manifestations and generally start developing within one week. Some of the symptoms include deafness, encephalitis, facial neuritis, hydrocephalus, cerebellar ataxia, transverse myelitis and polyradiculitis [90].

## PNEUMONIA

Streptococcus pneumoniae is a major pathogen that can cause infection in childhood. These bacteria colonize the nose and pharynx and move to the middle ear, blood and lungs, which causes bacterial meningitis, otitis media and septicemia. We are losing almost 1 million people every year because of the infection of Streptococcus pneumonia and drug resistant Streptococcus pneumoniae is widely distributed around the world and increasing its prevalance [91,92].

## POLIO

Polio is a viral infection which can be otherwise called infantile paralysis. It is a subgroup of family picornavirdae with three major serotypes PV1, PV2 and PV3. Immunity against all three serotypes is essential for prevention of disease. This virus generally transmitted through oral discharge from oral routes. Virus multiplies in the oral larynx and small intestine after entering human body. It usually attacks local lymphoid tissues and then enters into the main blood system, at the time of nervous system attack [93]. An estimated two lakhs fifty thousand children are now alive and another five million children are walking by the grace of polio eradication initiative [94].

## WHOOPING COUGH

Whooping cough (pertussis), a respiratory infection caused by a non-invasive pathogen Bordetella pertussis, accounts for more than three lakh deaths per year. B. pertussis localises mainly in the upper respiratory tract and produces a large array of potential virulence factors, many of which play vital role in

the pathogenesis [95-99]. Overcrowding, poor hygiene, waning immunity are the main reasons for high incidence of whooping cough. The whole cell pertussis vaccine is the generally used vaccine all over the world, consisting of a killed B pertussis strain, origin of which may differ in different parts of the world, generally given in combination with genetically inactivated diphtheria and tetanus toxoids, with or without Haemophilus influenzae type b, hepatitis B or inactivated polio virus vaccine, depending upon the manufacturer [100].

## CONCLUSION

There has been the existence of many infectious diseases from ancient times, which are emerging day by day to attack our innocent pediatric population. The emergence of these pediatric infectious diseases reflects various factors like microbiological, social and political factors. Finally we need to concentrate more on prevention and elimination of all pediatric infectious diseases as these are becoming a substantial burden on future generation and demands global action and attention.

## REFERENCES

1. Earla P. Impact of Microbes on Ancestors. RRJMB. 2015;4:1-5.
2. Earla P. Long Lasting Disease: Leprosy. J Infect Dis Ther. 2015;3:R1-001.
3. Earla P. Ancient Diseases-Microbial Impact. J Anc Dis Prev Rem. 2014;2:R1-001.
4. Nair G, et al. Detection of Entamoeba histolytica by Recombinase Polymerase Amplification. Am J Trop Med Hyg. 2015;93:591-595.
5. Radlovic N, et al. Acute Diarrhea in Children. Srp Arh Celok Lek. 2015;143:755-762.
6. D'Auria A, et al. Cutaneous Acanthamoebiasis with CNS Involvement Post-Transplantation: Implication for Differential Diagnosis of Skin Lesions in Immunocompromised Patients. J Neuroparasitol. 2012;3:1-7.
7. Jeevani T and Aliya S. HIV Infections-Acquired Immuno Deficiency Syndrome Malignancies. J AIDS Clinic Res. 2011;2:131.
8. Maluleke TX, et al. Perceptions of Professional Nurses in Rural Hospitals of the Limpopo Province Regarding Nursing Care of Patients with Human Immuno-deficiency Virus and Acquired Immunodeficiency Syndrome. J AIDS Clinic Res. 2012;3:176.
9. Kotwal J, et al. Evaluation of Surrogate Markers for Prediction of CD4 Counts in People Living with Human Immunodeficiency Virus/ Acquired Immunodeficiency Syndrome. J AIDS Clin Res. 2016;7:573.
10. Almeida EC, et al. Langerhans Cells in Oral Mucosa from Patients with Acquired Immunodeficiency Syndrome. J Infect Dis Ther. 2014;2:160.

11. Rani M, et al. In-Vitro Cytokine Induction and Neutrophil Respiratory Burst Activity by Candida Isolates from HIV Seropositive Patients with Oropharyngeal Candidiasis. RRJMB. 2014.
12. Catano JC and Taffur CC. Chronic Disseminated Candidiasis: The New Face of an Old Disease. J Clin Case Rep. 2012;2:189.
13. Jabra-Rizk MA. Oral Candidiasis: An Opportunistic Infection of AIDS. J AIDS Clin Res. 2014;5:i101.
14. Seton M. Hydroxychloroquine Reverses Alopecia in Autoimmune Polyendocrinopathy- Candidiasis-Ectodermal Dystrophy (APECED): A Case Report. J Clin Cell Immunol. 2013;4:177.
15. Mascellino MT. Epidemiology of Candidaemia: A Prospective Comparison between Invasive Candidiasis in Italy and All Over the World. J Clin Exp Pathol. 2016;6:e120.
16. Xin H. Double Chimeric Peptide Vaccine and Monoclonal Antibodies that Protect Against Disseminated Candidiasis. J Vaccines Vaccin. 2014;5:241.
17. Gaona-Flores V, et al. In vitro Sensitivity to Fluconazole through Vitek II Systems, of Strains of Candida Spp. In Patients with Oropharyngeal Candidiasis and HIV/AIDS. J AIDS Clin Res. 2013;4:230.
18. Zanluqui NG, et al. Macrophage Polarization in Chagas Disease. J Clin Cell Immunol. 2015;6:317.
19. Apt W, et al. Chagas Disease: A Global Neglected Disease that Require Continuous Medical Education. J Community Med Health Educ. 2013;4:260.
20. Guzman TA, et al. Stroke in Chagas Disease Patients in Ecudaor. J Neuroparasitol. 2011;2:1-4.
21. Reinaldo B and Bestetti. The Non-Award of the Nobel Prize of 1921 to Carlos Chagas: A Tragic Mistake. J Infect Dis Ther. 2015;3:220.
22. Mandal S. Epidemiological Aspects of Chagas Disease - a Review. J Anc Dis Prev Rem. 2014;2:117.
23. Castro AAM, et al. Clinical Course Development of the Chagas Heart Disease in a Brazilian Patient: A Case Report. J Clin Case Rep. 2014;4:449.
24. Sandle T. Exhuming Skeletal Remains: How Cholera Deaths of the Past Could Shine a Blue-Light of Hope. J Anc Dis Prev Rem. 2015;3:e121.
25. Dutta S, et al. Human Enteric Vaccines. J Vaccines Vaccin. 2014;5:252.
26. Abd H, et al. Survival of Vibrio cholerae Inside Acanthamoeba and Detection of Both Microorganisms From Natural Water Samples May Point out the Amoeba as a Protozoal Host for V. cholerae. J Bacteriol Parasitol. 2011;S1:003.
27. Bagchi A. Characterization of the Leucine-Responsive Transcription Factor from Pathogenic Vibrio cholerae Using Molecular Modelling and Molecular Dynamics Simulations. Curr Synthetic Sys Biol. 2014;2:110.
28. Feng L, et al. A Recalibrated Molecular Clock and Independent Origins for the Cholera Pandemic Clones. PLoS ONE. 2008;3:e4053.
29. Cheng K, et al. Spread of beta-lactam-resistant Pseudomonas aeruginosa in a cystic fibrosis clinic. Lancet. 1996;348:639-642.

30. Workentine M and Surette MG. Complex Pseudomonas population structure in cystic fibrosis airway infections. Am J Respir Crit Care Med. 2011;183:1581-1583.
31. Smith EE, et al. Genetic adaptation by Pseudomonas aeruginosa to the airways of cystic fibrosis patients. Proc Natl Acad Sci. 2006;103:8487-8492.
32. Winstanley C, et al. Genome Res. 2009;19:12-23.
33. Fouts DE, et al. Whole genome sequencing and comparative genomic analyses of two Vibrio cholerae O139 Bengal-specific Podovirusesto other N4-like phages reveal extensive genetic diversity. Virol J. 2013;10:165.
34. 34.Atif AB, et al. Isolation of Vibrio cholerae in Homogenized Tissues of Liver, Gall Bladder and Bile in Rabbit Model. Microinflammation. 2014;1:103.
35. Haque F, et al. Cholera Outbreaks in Urban Bangladesh In 2011. Epidemiol. 2013;3:126.
36. Thompson KM, et al. Managing Cholera as a Preventable Global Threat. J Vaccines Vaccin. 2013;4:183.
37. Pun SB, et al. An Outbreak of Vibrio cholerae in 2012, Kathmandu, Nepal. Trop Med Surg. 2013;1:115.
38. Nazar-ul-Islam, et al. Review of Trends in Cholera. Air Water Borne Diseases. 2015;4:118.
39. Haque SS. Antioxidant Status of Formulated Drugs Against Typhoid. Biochem & Anal Biochem. 2011;1:102.
40. Vagholkar K, et al. Abdominal Complications of Typhoid Fever. Journal of Surgery [Jurnalul de chirurgie]. 2015;11:359-361.
41. Ishaleku D, et al. The Re-emergence of Chloramphenicol Sensitive Salmonella species among Typhoid Fever Patients in the Southern Geographical Zone of Nasarawa State, Nigeria. J Infect Dis Ther. 2015;3:219.
42. Agwu E. Distribution of Community Acquired Typhoid Fever among Febrile Patients Attending Clinics in Bushenyi, Uganda: Case Study of the Year 2005. J Medical Microbiol Diagnosis. 2012;1:101.
43. Iheukwumere I, et al. Manifestations, Mismanagement and Diagnostic Challenges of Malaria and Typhoid Fever. Malar Chemoth Cont Elimination. 2013;2:109.
44. 44.Typhoid Diagnostics for the Developing World - Are We Looking in the Wrong Haystack?. J Med Microb Diagn. 3:e124.
45. Johnson OK. Pilot Case Series Demonstrating Unsuspected Ulceration in Perforated Ileum from Typhoid Fever. J Gastrointest Dig Syst. 2016;6:445.
46. Perillo L, et al. Influence of Occlusal Disorders, Food Intake and Oral Hygiene Habits on Dental Caries in Adolescents: A Cross-Sectional Study. Dentistry. 2016;6:358.
47. Agarwal R, et al. Prevention of Dental Caries-Measures beyond Fluoride. Oral Hyg Health. 2014;2:122.

48. Ambarkova V and Ivanova V. Dental Caries Experience among Primary School Children in the Eastern Region of the Republic of Macedonia. OHDM. 2014;13:1-7.
49. Pollick HF. What is the evidence that fluoride works to prevent dental caries?. OHDM. 2004;2:1-11.
50. Rashkova M, et al. Study of the risk factors for the development of dental caries and creation of a system for assessment the risk of caries in children in Bulgaria. OHDM. 2008;2:1-11.
51. Hysi D, et al. Dental Caries Experience and Oral Health Behaviour Among 12-YearOlds in the City of Tirana, Albania. OHDM. 2010;4:229-234.
52. Hegde MN, et al. Salivary IgA and Dental Caries in Type II Diabetics in Indian Adult Population. J Interdiscipl Med Dent Sci. 2014;2:133.
53. Kodama Y, et al. Are Dyslipidemia Following Obesity and Hyperglycemia as an Indicator of Diabetes Involved in Dental Caries Development in Zucker Rats?. J Diabetes Metab. 2014;5:340.
54. Damle SG, et al. Quantitative Determination of Inorganic Constituents in Saliva and their Relationship with Dental Caries Experience in Children. Dentistry. 2012;2:131.
55. Moin M and Malik A. Frequency of Dental Caries and Level of Risk among Type II Diabetics. Dentistry. 2015;5:334.
56. Helaly M and Abuaffan AH. Association between Sickle Cell Disease and Dental Caries among Sudanese Children. J Mol Imag Dynamic. 2015;5:120.
57. Wiener RC. Children with Special Health Care Need's Association of Passive Tobacco Smoke Exposure and Dental Caries: 2007 National Survey ofChildren's Health. J Psychol Abnorm Child. 2013;1:104.
58. Pratiwi NL. The Trend Analysis of the Availability of Dental Caries and Dental Health Personnel in Indonesia. Dentistry. 2016;6:360.
59. Pashayev AC, et al. An Investigation into the Prevalence of Dental Caries and its Treatment Among the Adult Population With Low Socio-Economic Status in Baku, Azerbaijan. OHDM. 2011;10:7-12.
60. Gambhir RS, et al. Vaccine against Dental Caries- An Urgent Need. J Vaccines Vaccin. 2012;3:136.
61. Datta P and Datta PP. Prevalence of Dental Caries among School Children in Sundarban, India. Epidemiol. 2013;3:135.
62. Lopes Coutinho TC. Dental Caries: A Problem of low Socioeconomic Background and Our Responsibility for Helping change the Situation. J Socialomics. 2012;1:e107.
63. Bensahi I, et al. Nontoxigenic Corynebacterium diphtheriae: A Rare Cause of Infective Endocarditis in Native Valve. J Infect Dis Ther. 2015;3:216.
64. Sánchez GF, et al. Safety Profile of the Tetanus-Diphtheria-Acellular Pertussis Combination Vaccine as a Single Booster (5th dose) among Spanish Children Aged 4-6 years Old with Different Vaccination Schedules. J Vaccines Vaccin. 2015;6:284.

65. Deshpande RP and Ghongane B. Current status of vaccines against Diphtheria, Pertussis, Tetanus, Hepatitis B and Hib: A Review. RRJMHS. 2014.
66. Ónodi-Nagy K, et al. Antibiotic Induced Cutaneous Rash in Infectious Mononucleosis: Overview of the Literature. J Allergy Ther. 2015;6:222.
67. Chovel-Sella A, et al. Incidence of rash after amoxicillin treatment in children with infectious mononucleosis. Pediatrics. 2013;131:e1424-e1427.
68. Leung AK and Rafaat M. Eruption associated with amoxicillin in a patient with infectious mononucleosis. Int J Dermatol. 2003;42:553-555.
69. Treffel P and Gabard B. Measurement of sodium lauryl sulfate-induced skin irritation. Acta Derm Venereol. 1996;76:341-343.
70. Balfour HH Jr, et al. Infectious mononucleosis. Clin Transl Immunology. 2015;4:e33.
71. Zhao J, et al. Synthesis and In Vitro Anti-Influenza Evaluation of Rupestonic Acid Analogues: Effect of Configuration and Substitution at C (3). Med chem. 2016;6:322-326.
72. Gohil D, et al. Oseltamivir Resistant Influenza A (H1N1) Virus Infection in Mumbai, India. J Antivir Antiretrovir. 2015;7:108-114.
73. Ramezanpour B, et al. Cross-Sectoral Perspectives of Market Implementation of the MVA Platform for Influenza Vaccines: Regulatory, Industry and Academia. J Vaccines Vaccin. 2016;7:318.
74. Ma J, et al. Leukocyte Interferon-α-n3 Inhibits Influenza A Viral Replication in Human Alveolar Epithelial A549 Cells. J Antivir Antiretrovir. 2015;7:104-107.
75. Kiseleva I and Rudenko L. Potentially Pandemic Live Influenza Vaccines Based on Russian Master Donor Virus are Genetically Stable after Replication in Humans. J Vaccines Vaccin. 2016;7:317.
76. Ariad S, et al. Measles Virus: Association with Cancer. J Clin Cell Immunol. 2011;S5:002.
77. Jiatong Z. The Strategy to Further Control and Elimination Measles in China Based on the Analysis of 10 Years Measles Suspects Accumulation in Guangxi. J Antivir Antiretrovir. 2011;S3.
78. Akalu HB. Review on Measles Situation in Ethiopia; Past and Present. J Trop Dis. 2015;4:193.
79. Shrivastava SR, et al. 2015 CalifornianMeasles Outbreak: An Eye Opener for Local Public Health Authorities. Biol Med. 2015;S3:004.
80. Batirel A and Doganay M. Clinical Approach to Skin Eruption and Measles: A Mini Review. J Gen Pract. 2013;1:118.
81. Jiatong Z and Ge Z. Measles Control in Guangxi, China: High Risk Counties Selection and its Mass Campaign from 1999-2008. J Antivir Antiretrovir. 2013;5:021-027.
82. Durrheim DN and Dahl-Regis M. The Ethical Imperative to Eradicate Measles. J Clinic Res Bioeth. 2014;5:183.
83. Chaturvedi S. Reluctance to Advance the Age of Measles Immunisation: Ethics of Best Bargain, Policies of Denial, and Programs of Verticality. J Clinic Res Bioeth. 2015;6:216.

84. Van Buynder PG. Large Measles Outbreak in a Religious Community in British Columbia. J Vaccines Vaccin. 2014;5:246.
85. Alzein KJ. Revision of Meningitis Surveillance System in Jordan during 2001 and 2014 Years. Epidemiology. 2016;6:220.
86. Pinheiro DML, et al. Polymorphisms in DNA Repair Gene XRCC1 (Arg194Trp) and (Arg399Gln) and their Role in the susceptibility of Bacterial Meningitis. J Meningitis. 2016;1:105.
87. Ghuneim N, et al. Epidemiology of Different Types of Meningitis Cases in Gaza Governorates, Occupied Palestinian Territory, December 2013- January 2014. J Antivir Antiretrovir. 2016;8:026-034.
88. Kolekar PS, et al. Genotyping of Mumps viruses based on SH gene: Development of a server using alignment-free and alignment-based methods. Immunome Res. 2011;7:1-7.
89. Choudhury SA and Matin Fl. Seroprevalence of Antibodies to Measles, Mumps and Rubella (MMR) Vaccines in Previously Vaccinated Human Immunodeficiency Virus-Infected Children and their Control Counterparts. J Vaccines Vaccin. 2014;5:255.
90. Suvorit SB, et al. Postencephalitic Parkinsonism in a Patient with Mumps Infection: A Case Report. J Neuroinfect Dis. 2014;5:162.
91. Li J, et al. Effects of Microgravity on the Phenotype, Genome and Transcriptome of Streptococcus pneumonia. RRJMB. 2016.
92. Hammond TG and Hammond JM. Optimized suspension culture: the rotating-wall vessel. Am J Physiol Renal Physiol. 2001;281:F12-F25.
93. Ali L, et al. An Assessment of Religious Impediments to Polio Vaccination in Tehsil Khwazakhela, Swat. J Pain Relief. 2016;5:241.
94. Fetene NW and Sherani A. Determinant Factors for Implementing Polio Eradication Activities under Security Compromised Settings of Pakistan. J Trop Dis. 2013;2:127.
95. Mukkur T and Richmond P. Alternative Whooping Cough Vaccines: A Minireview. J Vaccines Vaccin. 2013;4:175.
96. Marzouqi I, et al. Development of improved vaccines against whooping cough: current status. Hum Vaccin. 2010;6:543-553.
97. Elahi S, et al. The benefits of using diverse animal models for studying pertussis. Trends Microbiol. 2007;15:462-468.
98. He Q and Mertsola J. Factors contributing to pertussis resurgence. Future Microbiol. 2008;3:329-339.
99. Mattoo S and Cherry JD. Molecular pathogenesis, epidemiology, and clinical manifestations of respiratory infections due to Bordetella pertussis and other Bordetella subspecies. Clin Microbiol Rev. 2005;18:326-382.
100. Mukkur T and Richmond P. Development of Affordable Effective Vaccines against Whooping Cough for the Developing World. J Vaccines Vaccin. 2012;3:e107.