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Review

Importance of diagnosis: As a premise in the management and control of viruses

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ABSTRACT

The nutritional status of the host has long been associated with both severity and susceptibility to infectious disease. The accepted model system proposes that inadequate nutrition impairs the functioning of the immune system, thus resulting in increased susceptibility to infection. However, current work suggests that not only can the nutritional status of the host affect the immune response, but it can also affect the viral pathogen. In a mouse model, a benign strain of coxsackievirus B3 became virulent and caused myocarditis in selenium- and vitamin E-deficient mice. This change in pathogenicity was due to mutations in the viral genome, which changed an avirulent virus into a virulent one.

Keywords: Virus, types, diversity, outlook, conclusion, virulence, diagnostic.

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INTRODUCTION

Pathogenic viruses are viruses that can infect and replicate within human cells and cause diseases. The continuous emergence and re-emergence of pathogenic viruses have become a major threat to public health. Whenever pathogenic viruses emerge, their rapid detection is critical to enable the implementation of specific control measures and the limitation of virus spread. Further molecular characterization to better understand these viruses is required for the development of diagnostic tests and countermeasures. Advances in molecular biology techniques have revolutionized the procedures for the detection and characterization of pathogenic viruses. The development of PCR-based techniques together with DNA sequencing technology has provided highly sensitive and specific methods to determine virus circulation. Pathogenic viruses potentially having global catastrophic consequences may emerge in regions where the capacity for their detection and characterization is limited. The development of a local capacity to rapidly identify new viruses is therefore critical. This article reviews the molecular biology of pathogenic viruses and the basic principles of molecular techniques commonly used for their detection and characterization. The principles of good laboratory practices for handling pathogenic viruses are also discussed. This review aims at providing researchers and laboratory personnel with an overview of the molecular biology of pathogenic viruses and the principles of molecular techniques and good laboratory practices commonly implemented for their detection and characterization (Artika et al., 2020).

VIRAL DISEASES

The emergence of infectious diseases has been a threat to public health and global stability. Historically, emerging infectious diseases have caused the deadliest catastrophic pandemics such as the 1918 influenza pandemic (claiming about 50 million lives), the HIV/AIDS pandemic (claiming about 35 million lives so far), etc). Emerging infectious diseases are defined as infections whose incidence in humans has increased within the past two decades or threaten to increase in the years to come. The disease emergence can be caused by the spread of a new pathogen, or by the reappearance (or re-emergence) of a known pathogen after a decline in infection (biological, social, and environmental factors have been linked to the emergence of infectious diseases. These include changes of the pathogens through evolution, changes in the way human populations interact with each other, and with their environment. In addition, increased susceptibility to infection, increased ease of international travel, climate and weather changes, have also been associated with new diseases emergence. One of the major agents responsible for causing emerging infectious diseases is the virus. Pathogenic viruses that cause emerging diseases are called emerging viruses (Chua and Gubler, 2013). Nipah virus first emerged in 1998 during a large outbreak of encephalitis and respiratory disease in Malaysia and Singapore, causing 276 cases of encephalitis with 106 fatalities. Since 2001, outbreaks of the Nipah virus have occurred almost every year in Bangladesh with a strikingly high case-fatality rate of up to 90%, with 24 cases of Nipah virus occurring to date in 2013. The recurrent outbreaks of the Nipah virus in Bangladesh have been epidemiologically associated with the consumption of date palm sap, which has led to the hypothesis that Nipah virus zoonosis is a result of drinking date palm sap contaminated by infected fruit bats (de Wit et al., 2014; Chua et al., 2000. Syed, 2018a).

Diversity of viral diseases

Chikungunya virus (CHIKV) is an arthropod-borne virus that is transmitted by *Aedes* (*Ae.*) mosquitoes. It was first isolated in 1952 in the Makonde Plateau of the southern province of Tanzania (former Tanganyika). The virus transmission cycle requires infection of female mosquitoes via a viraemic bloodmeal taken from a susceptible vertebrate host and, following a suitable extrinsic incubation period, transmission to another vertebrate host during subsequent feeding (Thiberville et al.,2013; Stein, 2008). Since the Indian Ocean outbreak in 2005–2006, the information available for the scientific community relating specifically to the clinical characteristics of patients infected by CHIKV has significantly increased (Thiberville et al.,2013; Krishnamoorthy et al., 2009). Arboviruses (*ar*thropod-*bo*rne viruses) are a group of viruses that exist in a transmission cycle between blood-feeding arthropod vectors and amplifying, vertebrate hosts. With most arboviruses, human involvement in this transmission cycle is incidental. In terms of public health significance, the mosquito is the most important vector of arbovirus transmission. It is estimated that approximately 3.9 billion people, living in more than 120 different countries, are at risk of becoming infected with any of the three major arboviruses: Chikungunya virus (CHIKV), Dengue virus (DENV) and Zika virus (ZIKV) (Harapan et al., 2019; Syed, 2018b; Weaver and Reisen, 2010).

Definition of the term and history

Towards the end of the first decade of the 21st century, during December 2019, numerous pneumonia incidences of unidentified cause appeared in Wuhan, Hubei, China, with clinical presentations greatly resembling Flu and viral pneumonia. After virus isolation and analysis of viral genome sequence from infected patient's samples, a novel coronavirus named severe acute respiratory syndrome-related coronavirus 2 or SARS-CoV-2 (initially designated as novel coronavirus or nCoV-2019) was identified from an unknown source. SARS-CoV-2 is the causative agent of respiratory disease which is recently named Coronavirus disease 2019 (COVID-19) by the World Health Organisation (WHO). Human-to-human transmission of SARS-CoV-2 is a major concern for the health care workers and a preliminary R0 (Reproductive number: as the *number* of new infections one infected person generates on average throughout its infectious period) measure of 1.4 to 2.5 was reported by the WHO (https://www.who.int/health-topics/coronavirus) (Biswas et al., 2020). COVID-19 is one of the most alarming diseases in the globe at this moment. The number of patients infected with SARS-CoV-2 is increasing at an almost steady rate, although in some days fewer number cases were reported. The total number of cases reached 20,000 in the first 12 days, 40,000 in the next 7 days, and more than 80,000 in just 33 days. Infections are causing varied clinical manifestations from mild symptoms to severe respiratory attacks, although there is a possibility of asymptomatic infection. It has spread in many other countries beyond China, therefore proper handling and management of the disease are critically important to prevent a pandemic (Syed, 2020).

Pathogenesis differences of the term

Varicella-zoster virus (VZV), a member of the human alphaherpesvirus family, causes chickenpox (varicella) mostly in children and establishes a latent infection in cranial, dorsal root, and autonomic ganglia. Latent VZV can reactivate decades later to produce shingles (zoster). VZV-specific cell-mediated immunity (CMI) declines with age, resulting in zoster and associated neurological complications that are also seen in immunocompromised organ transplant recipients and patients with cancer or AIDS. The actual mechanism of reduction in VZV-specific CMI and associated virus reactivation remains unclear. VZV produces chickenpox and shingles only in humans, underscoring the need for an animal model to study VZV neuropathogenesis. Attempts by multiple groups to establish VZV infection in guinea pigs and mice by experimental inoculation have resulted in seroconversion without clinical symptoms (Mahalingam et al., 2019; Myers et al., 1980; Syed, 2019a).

Characteristics

Ebola virus disease (EVD) is a severe and frequently lethal disease caused by the Ebola virus (EBOV). EVD outbreaks typically start from a single case of probable zoonotic transmission, followed by human-to-human transmission via direct contact or contact with infected bodily fluids or contaminated fomites. EVD has a high case–fatality rate; it is characterized by fever, gastrointestinal signs, and multiple organ dysfunction syndrome. Diagnosis requires a combination of case definition and laboratory tests, typically real-time reverse transcription PCR to detect viral RNA or rapid diagnostic tests based on immunoassays to detect EBOV antigens. Recent advances in medical countermeasure research resulted in the recent approval of an EBOV-targeted vaccine by European and US regulatory agencies. The results of a randomized clinical trial of investigational therapeutics for EVD demonstrated survival benefits from two monoclonal antibody products targeting the EBOV membrane glycoprotein. New observations emerging from the unprecedented 2013 to 2016 Western African EVD outbreak (the largest in history) and the ongoing EVD outbreak in the Democratic Republic of the Congo have substantially improved the understanding of EVD and viral persistence in survivors of EVD, resulting in new strategies toward prevention of infection and optimization of clinical management, acute illness outcomes and attendance to the clinical care needs of patients (Jacob et al., 2020). The 2013-2016 Western African EVD outbreak was the first to be largely characterized by molecular epidemiological evidence. Deep-sequencing efforts often performed on-site and in parallel by several groups, resulted in the determination of >1,600 coding-complete (all open reading frames) or near-complete (typically coding-complete plus parts of leaders and/or trailers) EBOV genomes directly from human patient samples (Gire et al., 2014; Carroll et al., 2015; Syed, 2019b). The honey bee queen is the central hub of a colony to produce eggs and release pheromones to maintain social cohesion. Among many environmental stresses, viruses are a major concern to compromise the queen’s health and reproductive vigor. Viruses have evolved numerous strategies to infect queens either via vertical transmission from the queens’ parents or horizontally through the worker and drones with which she is in contact during development, while mating, and in the reproductive period in the colony. Over 30 viruses have been discovered from honey bees but only a few studies exist on the pathogenicity and direct impact of viruses on the queen’s phenotype. An apparent lack of virus symptoms and practical problems are partly to blame for the lack of studies, and we hope to stimulate new research and methodological approaches. To illustrate the problems, we describe a study on sublethal effects of the Israeli Acute Paralysis Virus (IAPV) that led to inconclusive results. We conclude by discussing the most crucial methodological considerations and novel approaches for studying the interactions between honey bee viruses and their interactions with queen health (Amiri et al., 2020). Chronic hepatitis B is a global health problem. The clinical outcomes of chronic hepatitis B infection include asymptomatic carrier state, chronic hepatitis (CH), liver cirrhosis (LC), and hepatocellular carcinoma (HCC). Because of the spontaneous error rate inherent to viral reverse transcriptase, the hepatitis B virus (HBV) genome evolves during infection under the antiviral pressure of host immunity. The clinical significance of pre-S/S variants has become increasingly recognized in patients with chronic HBV infection. Pre-S/S variants are often identified in hepatitis B carriers with CH, LC, and HCC, which suggests that these naturally occurring pre-S/S variants may contribute to the development of progressive liver damage and hepatocarcinogenesis. This paper reviews the function of the pre-S/S region along with recent findings related to the role of pre-S/S variants in liver diseases. According to the mutation type, five pre-S/S variants have been identified: pre-S deletion, pre-S point mutation, pre-S1 splice variant, C-terminus S point mutation, and pre-S/S nonsense mutation. Their associations with HBV genotype and the possible pathogenesis of pre-S/S variants are discussed. Different pre-S/S variants cause liver diseases through different mechanisms. Most cause the intracellular retention of HBV envelope proteins and induction of endoplasmic reticulum stress, which results in liver diseases. Pre-S/S variants should be routinely determined in HBV carriers to help identify individuals who may be at a high risk of less favorable liver disease progression. Additional investigations are required to explore the molecular mechanisms of the pre-S/S variants involved in the pathogenesis of each stage of liver disease (Chen, 2018; Syed, 2018c). Understanding transmission routes and directionality of viral spread is a crucial first step in determining the effect and epidemiology of a given pathogen because transmission routes have direct effects on the prevalence and virulence of viruses (Martin et al., 2012; Shapiro-Ilan et al., 2005).

DISCOVERY OF NOVEL VIRUSES

The interactions between viruses and their honey bee queen hosts have practical importance for maintaining pollinator health because the queens play an important role in the vertical transmission of many health-relevant viruses. More research is needed to document the distribution of viruses and their dynamics across space and time. The continued discovery of novel viruses or virus strains can be anticipated, necessitating continued monitoring efforts. This is particularly true to honey bee queen breeding operations that widely distribute their bees to their customers. Alternative to virus monitoring of queen breeders, local, small-scale queen breeding efforts could mitigate the risk of human-assisted virus spread over long distances (Moritz et al., 2010). Beyond the practical importance of more virus research in honey bee queens, honey bees present unique opportunities of academic interest to study the relations between host physiology, virus transmission and replication, and virus pathogenicity (Rangel and Fisher, 2019). Specifically, the interplay between caste, potentially differential virulence evolution, and social immune mechanisms should be of great general interest. However, we believe that many experimental difficulties that complicate investigations into the interactions between viruses and honey bee queens need to be addressed before conclusive studies can improve our current understanding of queen–virus relationships and honey bee virology in general (Vanengelsdorp and Meixner, 2010).

CONCLUSION

Pathogen virulence is a complex interplay of both host and pathogen properties. Host nutritional status has long been considered a risk for infection susceptibility and severity and is now implicated in shaping viral evolution. Continued studies on the molecular consequences of obesity and malnutrition at the macro- and micronutrient levels will reveal which host defenses are impaired through malnutrition and how they control quasispecies development and viral pathogenesis. Similarly, as we gain insight into how hosts influence quasispecies formation and pathogen virulence, we too can exploit these features for host benefit (Honce and Schultz-Cherry, 2020). Viral infections are either acute or persistent. An acute infection generally has a rapid course with an incubation period of days to weeks and the virus clears the body within 2 to 3 weeks of disease onset. Persistent infections may last months to years and can be characterized as late complications of acute infection or latent, chronic, or slow infections. Persistent infections may be reactivated and cause acute episodes or cause late sequela to infections. They may be associated with immunopathologic disease, may lead to neoplasia, and are important epidemiologically as a result of recurrence of or continual shedding (Kennedy and Greenacre, 2005).

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