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INTERNATIONAL JOURNAL OF CURRENT RESEARCH

International Journal of Current Research
Vol. 13, Issue, 03, pp.16620-16622, March, 2021

ISSN: 0975-833X

DOI: https://doi.org/10.24941/ijcr.40988.03.2021

REVIEW ARTICLE **OPEN ACCESS**

INFLUENZA VIRUSES

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ARTICLE INFO

Article History:
Received 18th December, 2020
Received in revised form
07th January, 2021
Accepted 15th February, 2021
Published online 26th March, 2021

Key Words:

Influenza, Flu virus, Swineorigin, Avian OIV, COVID-19.

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ABSTRACT

The history of influenza viruses teaches that influenza originates from volatile, generically aquatic animals, and then passes to man through the "jump" in pigs. The promiscuity of the farms, as it is used in Asia, determines this passage and then the spread. This gave rise to the Spanish flu (1918, H1N1), the Asian flu (1957, H2N2), that of Hong Kong (1968, H3N2) and so on. The strains spread in some years may also present relationships with those of other years. The swine origin influenza virus (S-OIV) detected in April 2009 in Mexico, Canada and USA exibthed an unique genome composition not shown before. According to the flu vaccinations compaign influenza vaccine is produced before the start of the flu season, which means that the virus almost never coincided with does tested early to produce the vaccine. The main componet of the viral family for human pathology was the agent of the small pox, the first disease to be eraticated by a vaccine.

Citation: Giulio Tarro. "Influenza viruses*", 2021. International Journal of Current Research, 13, (03), 16620-16622.*

INTRODUCTION

Since the emergence of a novel aquatic bird flu agent in humans maybe detected in near future, approaches to early diagnosis and prompt therapy are welcome. Younger people have antibodies to the most important antigens of the strains they have come into contact with. As we age, we observe a broader spectrum immunity which is reflected in the polyvalent antibodies that are acquired through contact with numerous primary and secondary antigens present in the viral strains encountered over the following years (Tarro, 2016a). But each subsequent contact with a type A influenza virus involves not only strictly specific antibodies, but also an increase in those directed towards the strain responsible for the subject's first influenza infection (Davenport phenomenon or doctrine of "original antigenic sin") (Tarro, 2017a). In this way, immunization against a specific strain, widespread a given period, progressively involves an increasing difficulty in its further distribution and creates the selective advantage, for some variant of the virus, to multiply and spread. The new strains will be able to multiply in hosts, regardless of whether or not they have had an immunological experience with the previous strains. As a result of this, shortly after the appearance of a new type, the old forms will disappear and the new family will become dominant for a period that generally covers 10-20 years, in which we witness, for the appearance of minor antigenic variations, to the subdivision of this into various subtypes (Tarro, 2019). Thus, epidemic manifestations can occur with those strains that possess dominant antigens that adapt to the deficiency, or rather, to the antibody absences of the population. In conclusion, it seems that the influenza A virus shows a capacity and an aptitude for survival which is based on the possibility of the emergence of new antigenic models that allow the virus to easily blend into populations still partially immune to previous antigenic forms. According to this view, the variations of influenza A viruses can be conceived a unitary sense, in the context of an evolutionary principle and development, known by Burnet as "immunological drift" or immunological steering (Tarro, 2017b).

Epidemiology: It is very important to remember that the presence of antibodies to the most recent Asian strains of 1957 (A2) has been demonstrated precisely in the oldest segment of the population at the time: in Asian flu, strains with dominant antigenic characters, different from those that had characterized the more or less previous years, but similar to those of the strains spread much earlier (Pandemic 1889-90) (Zimmer, Burke, 2009). For the emergency created by the "chicken flu" epidemic in Asia, it is right not to have created alarmism by being victims of bad information. The possibility of the avian virus reaching other parts of the world was there as was the case with all types of influenza viruses.

It is clear that the dead animal is harmless, so there are other veterinary and agricultural interests at the bottom. There is a potential risk of genetic recombination with human influenza viruses that could result a viral variant capable of human-to-human transmission (Enserink, Coen, 2009). During the avian flu epidemic that affected Asian countries in 2006 (China, Pakistan, Thailand, Cambodia, Indonesia, South Korea, Taiwan, Laos and Vietnam) with 80 million dead or sacrificed chickens and 42 human fatal cases, the H5N1 virus has been identified as an etiological agent, the same that in 1997 caused an epidemic outbreak in Hong Kong with 18 infected humans and 6 deaths and the sacrifice of 1.5 million chickens (Tarro, Esposito, 2011). Avian influenza epidemics followed one another with the implication of different viral strains such as H9N2 in 1999, two infected children and also other individuals, and in 2003, an infected boy in Hong Kong, while H5N1 affected three subjects of the same family also killing two in 2003. At the same time in the Netherlands an outbreak of H7N7 avian influenza affected 83 people and killed a veterinarian. In 20 06 in the USA outbreaks of avian influenza were identified in Texas and Delaware (H7N2 virus), in the latter state together with territories of Maryland and Virginia worked 14,000 people and 1,900 families that produced 8% of the American poultry meat, with a budget of one and a half billion dollars (Chowel et al, 2009). In 2003, American exports to Europe had reached the quota of eight million and eight hundred thousand eggs and 452 thousand chicks, respectively for 20 million and 3 million euros.

Viral transmission: If for SARS (2002-2003) direct contact was necessary, to put it in practical terms the so-called Pflugge droplets, for this avian flu it was different, in fact, it spreads through the air even at a distance. It was completely useless to set up a panic syndrome that often occurs through bad information or a lack of knowledge of the phenomenon. No alarmism because the number of victims was decidedly lower than other forms of flu. In the spring of 2009, a new influenza virus type A (H1N1) infecting humans was found in Mexico, Canada and the USA: the swine virus then caused a mild to severe febrile respiratory infection worldwide (Dawood et al, 2009) (Lister et al, 2009). In Campania the flu peak preceded the incidence of cases at national level by two weeks, moreover the percentage of patients positive for the virus was much higher in the city of Naples, compared to the other provinces of Campania. The most affected age, between 7 months from birth to 17 years, (43.45%) was similar to that reported nationally suggesting the conclusion that the high incidence and mortality of influenza in Campania was probably due to the fact that this region is the most densely populated in Italy and revealing important indications for future prevention campaigns (Morens et al, 2009) (Esposito et al, 2012). It is reasonable to hypothesize that for many deaths recorded elsewhere at the time, and we obviously refer to other regions, there was not the same ability and possibility of correlating the virological diagnosis to the cause of death, especially when the influenza virus acts as a cofactor. The problem of overdiagnosis has been solved in the fact that the definitive H1N1 influenza A label has been given to many laboratory diagnosed patients who otherwise would not have actually proved so affected. This phenomenon known as Will Rogers, is widely known since it was studied for the IQ (intelligence quotient) of the Oklahoma inhabitants who migrated to California, and then referred to many other aspects that lead to false interpretations of statistical observations. In conclusion, once again a problem as important as that of health, scientific authority has been lacking at national, regional and local level, because, as is constant in our country, professionalism does not pay. The consideration that last Summer in Australia, New Zealand, South Africa, Argentina, Chile there was the epidemy of coronavirus COVID-19, but there was no viral influenza, suggests that it is useless to get vaccinated (WHO, 2020). Furthermore it is now known that flu vaccinated subjects are fragile towards the coronavirus because this virus is activated in these subjects at 36% of cases as already reported (Krause, 2020).

CONCLUSION

Being able to identify sources of infection quickly and accurately has important implications for protecting the environment and monitoring potential pathogens. Appropriate control of viral infections will depend on the right choice of health regulations by the appropriate authorities and on the regulation of actual viral parameters: this will allow the development of surveillance systems with which to monitor and reduce known viral diseases more effectively and perhaps even prevent those emerging (Tarro, 2016b). To be able to talk about a real prevention technique, based on the use of the pathogen itself to immunize the organism, it is necessary to take a time jump up to 1796, the year in which the English doctor and naturalist Edward Jenner developed the first vaccine. Jenner had noticed that the milkmaids who lived in the county and who had contracted the minor disease called cowpox did not get sick even when smallpox was spread epidemically in the community. Consequently, on May 14, 1796, Jenner used material infected with cowpox virus, obtained from a milking machine, to inoculate an eight-year-old boy. In July of the same year, the doctor deliberately inoculated the same child with biological material taken from a subject with human smallpox. The child did not develop the disease and this demonstrated the effectiveness of the first form of smallpox vaccination (Tarro, 2015). Thanks to the use of this vaccination, universally adopted in the world since the nineteenth century, the World Health Organization has declared that smallpox has been completely eradicated from the planet, starting in 1979.

Conflict of Interests: The author has no conflict of interests to declare.

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