Ebolavirus has been posing a threat to human health since 1976, when it first appeared, causing severe symptoms and often leading to death [1]. While at first the outbreaks were limited to a small number of cases, ranging from 32 to 318, in 2013 an outbreak occurred, that lasted until 2016, with over 28,000 recorded cases [2]. This was the first outbreak that occurred in West Africa and was also reported in places outside Africa and due to its duration and magnitude it was characterised as the largest Ebola epidemic [1]. During this outbreak there was a concern that ebolavirus is mutating faster, which could increase its virulency, transmissibility and antigenicity [1]. This assumption was based on phylogenetic analyses of genome data that were carried out early during the epidemic and estimated atypically high molecular evolutionary rates for the virus, that were almost two times higher than what had been observed until that time [3]. However, later studies that included a larger number of viral sequences sampled over the whole epidemic, found lower evolutionary rates [4]. These studies revealed that Ebola Makona, which is the Ebola strain responsible for this outbreak, has an evolutionary rate between 0.87e-3 and 1.42e-3 [8], that is within the range of substitution rates for RNA viruses, 10e-2 – 10e-5 [9]. It has now been shown that initial high rate estimates for this outbreak were due to the small number of samples and the limitations of the computational methods used, and the fact that evolutionary rates sampled over short periods of time can lead to higher mutation rate estimations as many deleterious mutations have not been weeded out yet by natural selection [4].

In May of 2018 a new Ebola outbreak was reported in DRC, with 3318 cases reported by December of 2019 [5]. Although there are signs that the number of cases is declining, the epidemic continues to spread [5], and because of this it is harder to assess its magnitude and make assumptions about the evolutionary rate. Despite the fact that we do not yet have information about the entirety of this outbreak, it has been clear since its beginning that it is one of the largest Ebola virus outbreaks to have occurred [5]. Further studies have shown that the epidemiological features as well as the case fatality ratio are similar to other outbreaks that have previously occurred [6]. Sequencing analysis of early samples revealed that the Ebola virus Tumba, which evolves at a slower rate than other ebolavirus variants, was the cause of this outbreak. Although there were some differences found in the rate of evolution between ebolavirus Tuba and ebolavirus Makona, their intra-outbreak rates were found to be similar [7].

In recent years there have been developments in the field of immunology that allow in depth genomic surveillance of viruses through next-generation sequencing, so more sequences are available for observation and new ones can be easily obtained [1]. This, in combination with the use of better computational models, that allow for a more relaxed set-up that doesn’t heavily rely on assumptions, have led to more clear rate estimations [4]. The two previously mentioned Ebola outbreaks were the first outbreaks on which real-time genomic surveillance was applied [8]. During the 2013-2016 Ebola outbreak, Quick et al. created a portable genome sequencing device, that could generate readings within an hour. This was of great importance as it allowed researchers to observe the evolution and transmissibility of the virus in real time and they were able to quickly infer conclusions from that information and take measures to prevent the epidemic spread faster[8].

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