Peer Community In
Evolutionary Biology

Ebola evolution during the 2013-2016 outbreak

Sylvain Gandon and *Sébastien Lion* *based on peer reviews by*

Submitted: 31 March 2017, Recommended: 31 March 2017

Cite this recommendation as:
Gandon, S. and Lion, S. (2017) Ebola evolution during the 2013-2016 outbreak. *Peer Community in Evolutionary Biology*, 100019. 10.24072/pci.evolbiol.100019

Published: 31 March 2017

The Ebola virus (EBOV) epidemic that started in December 2013 resulted in around 28,000 cases and more than 11,000 deaths. Since the emergence of the disease in Zaire in 1976 the virus had produced a number of outbreaks in Africa but until 2013 the reported numbers of human cases had never risen above 500. Could this exceptional epidemic size be due to the spread of a human-adapted form of the virus? The large mutation rate of the virus [1-2] may indeed introduce massive amounts of genetic variation upon which selection may act. Several earlier studies based on the accumulation of genome sequences sampled during the epidemic led to contrasting conclusions. A few studies discussed evidence of positive selection on the glycoprotein that may be linked to phenotypic variations on infectivity and/or immune evasion [3-4]. But the heterogeneity in the transmission of some lineages could also be due to environmental heterogeneity and/or stochasticity. Most studies could not rule out the null hypothesis of the absence of positive selection and human adaptation [1-2 and 5]. In a recent experimental study, Urbanowicz et al. [6] chose a different method to tackle this question. A phylogenetic analysis of genome sequences from viruses sampled in West Africa revealed the existence of two main lineages (one with a narrow geographic distribution in Guinea, and the other with a wider geographic distribution) distinguished by a single amino acid substitution in the glycoprotein of the virus (A82V), and of several sub-lineages characterised by additional substitutions. The authors used this phylogenetic data to generate a panel of mutant pseudoviruses and to test their ability to infect human and fruit bat cells. These experiments revealed that specific amino acid substitutions led to higher infectivity of human cells, including A82V. This increased infectivity on human cells was associated with a decreased infectivity in fruit bat cell cultures. Since fruit bats are likely to be the reservoir of the virus, this paper indicates that human adaptation may have led to a specialization of the virus to a new host. An accompanying paper in the same issue of *Cell*

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by Diehl et al. [7] reports results that confirm the trend identified by Urbanowicz et al. [6] and further indicate that the increased infectivity of A82V is specific for primate cells. Diehl et al. [7] also report some evidence for higher virulence of A82V in humans. In other words, the evolution of the virus may have led to higher abilities to infect and to kill its novel host. This work thus confirms the adaptive potential of RNA virus and the ability of Ebola to specialize to a novel host. In this context, the availability of an effective vaccine against the disease is particularly welcome [8]. The study of Urbanowicz et al. [6] is also remarkable because it illustrates the need of experimental approaches for the study of phenotypic variation when inference methods based on phylodynamics fail to extract a clear biological message. The analysis of genomic evolution is still in its infancy and there is a need for new theoretical developments to help detect more rapidly candidate mutations involved in adaptations to new environmental conditions.

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