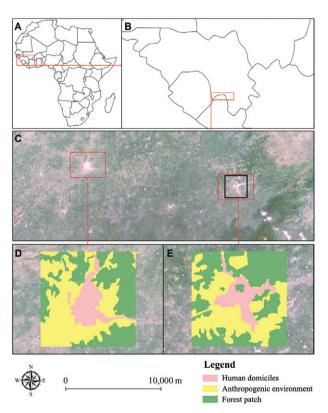
LETTER TO THE EDITOR

## Landscape fragmentation and Ebola outbreaks

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The 2014 Ebola outbreak started in early-March, Guinea (Baize et al. 2014). Following, the outbreak spread into Liberia in March, Sierra Leone in May and Nigeria in July. Today every city in the affected regions embedded in the West Africa is at risk (Pigott et al. 2014). Hypotheses have been raised about possible mechanisms that made dynamics of Ebola transmission exacerbated (Bausch & Schwarz 2014). The coupled pathogen-reservoir system has evolved from a zoonotic-only transmission, involving bats and non-human primates, into a



Epicenter of the 2014 Ebola outbreak. A: affected region in the West Africa; B: area of emergence of a new Ebola virus strain in South Guinea; C: satellite image showing initial locations of the outbreak of Ebola virus disease; D: Guéckédou landscape with forest patches, anthropogenic environment and human domiciles; E: Macenta landscape with forest patches, anthropogenic environment and human domiciles.

human-human transmission mode dynamics (Gire et al. 2014, Vogel 2014). This could be possible under a structural property of the landscape in where epicenter of the Ebola outbreak originated.

The cities of Guéckédou and Macenta (Guinea) are fragmented landscapes containing anthropogenic environment, i.e., urban plus rural areas and forest patches (Figure). Anthropogenic environment represents 50-60% of the total landscape area (100 km<sup>2</sup>). Edge density between anthropogenic environment and forest patches is 10-12 times higher than landscapes without fragmentation, which makes possible human density increase between anthropogenic and forest habitats. Landscape fragmentation can have two roles in the Ebola transmission dynamics: (i) serve as epidemiological corridors in where pathogen-carrier reservoirs can maintain and spread zoonotic cycles and (ii) make a frontier of contact between forest fringes and anthropogenic environment. The former mechanism can be responsible to increase variability of genetic pools of the pathogen within the zoonotic Ebola transmission cycle, whereas the latter is the cause of contact between humans and the wild pathogen. These mechanisms may ultimately be linked to the mutant Ebola virus that infect humans and is responsible for the 2014 Ebola outbreak. Could therefore these mechanisms be inferred as determinants of Ebola outbreaks in the future?

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