Editorial

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Viral Haemorrhagic Fever Outbreaks and Children: A Forgotten Toll

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Editorial

Viral haemorrhagic fever outbreaks of a previously unexpected magnitude developing at Africa slummy urban areas, propagating among immunologically naive, pictures an epidemiologist nightmare, and a worrisome perspective to the world because of the permanent risk of global spread. Long lasting consequences of emerging virus diseases like Ebola or potentially yellow fever in developing countries might endure after closing the emergency [1].

Ebola for instance, last outbreak initiated probably by a child, contributes to worsening an humanitarian and orphan crisis, as 2014-2016 West Africa Ebola outbreak left at least 1000 children without one or both parents, and children survivors might not resume school in the near future although study cycle is on-going. Not to mention an unacceptable death toll will be paid by the very young in the near future as resilience to preventable endemic diseases is compromised once Ebola virus decimated health personnel and wreck in weak health system, hampering lifesaving health promotion activities like vaccination.

Interestingly, children and adolescents cluster apart on regard to immune responses, clinical profile, and death rate due to Ebola in an age related manner, even when exposure is accounted [2]. If this alleged reduced susceptibility to Ebola finds biological support, it might guide future vaccine and treatment developments. Thereby, it is already known that an outstandingly fast and effective antibody response is needed in order to clear virus before Ebola subverts specific antibody response redirecting it, to focus on epitopes shared with sGP blanks. Kind of antibodies like monoclonal Mab100 and mab 114 effectively interfere functional binding of glycoprotein GP to its receptor, mediates rvsv-Z-BoV-GP protection against ebola virus. Whereas low real time PCR CT values and pro inflammatory cytokine exhaustion through I-IFN response hallmark bad prognosis in Ebola virus disease and Yellow fever.

A new cue to the puzzle, coming from the study Influenza 3I14mAB, and a Sos antibody accelerated response generated through intra clonal diversification [3] may shed light to a number of Ebola insensitive children being in close in contact to the virus. Whether mechanism based on structure would cope to RNA virus diversity or not and its relevance for Ebola virus disease vaccine development is a question only time will answer.

In pursuit of therapeutic targets, tertiary structures from most of Ebola encoded proteins have been described. Such an approach has rendered the discovery of molecules which binds Ebola GP inhibiting viral fusion. Same approach, using *in silico* modelling of the EBOV RNA-dependent RNA polymerase active site revealed key structural elements that might help in the development of molecules but also Ebola virus phylogenetic tree [4].

Although international emergency called by WHO to confront 2014 west Africa outbreak has ended, most ecological and human conditions prevailing before the outbreak are replicating along a wide belt across Africa. Bats immune system evolved to harbour a number of RNA viruses. In the last one, (2014) only one amino acid substitution was necessary for human adaptation. Do we are ready to take real action? Certainly, there will no better response than developing effective vaccines.

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