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Comphrensive study on Crimean Congo haemorrhagic fever

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DESCRIPTION

The Crimean-Congo hemorrhagic fever virus is a deadly illness with a mortality rate of up to 40%. The CCHFV is regarded as a priority agent for public health because there are no approved medical countermeasures. When used therapeutically or prophylactically, the non-neutralizing mouse monoclonal antibody 13G8 targets the CCHFV glycoprotein GP38 and shields mice from a lethal CCHFV challenge. Here, we present the structures of GP38 bound to a newly isolated CC5-17 mAb from a human survivor and a human chimeric 13G8 mAb.

These mAbs have shifted the angles at which they bind overlapping epitopes. We looked at the broad-spectrum potential of c13G8 and CC5-17 as well as the viability of applying them to the closely related nairoid *Aigai* virus. According to binding studies, CCHFV mAbs are unable to bind to *Aigai* virus GP38 because the corresponding region contains non-conserved amino acids. Future mAb therapeutics that are effective against a variety of CCHFV strains are made possible by this information and their *in vivo* efficacy.

Hemorrhagic fever and death are brought on by the Crimean-Congo Hemorrhagic Fever Virus, a member of the Nairoviridae family of tick-borne viruses. Because of its propensity to spread and prevalence throughout the Eastern Hemisphere, the WHO has identified CCHFV as a serious public health risk for which no approved vaccines or treatments are available. The CCHFV strains are divided into five clades, I through III. Because former CCHFV genogroup IV strains, which infrequently cause serious illness, were reclassified as the species *Aigai* virus, a sixth clade was no longer present. A negative-sense tri-segmented RNA genome with three segments a large, medium, and small segment—accompanies CCHFV. The majority of efforts to develop new medications and vaccines have focused on proteins derived from the M-segment encoded glycoprotein precursor. A clinically relevant strain of GP38's structure was determined in this study, both on its own and in complex with the human chimeric 13G8 mAb. The next step was to isolate seven human anti-GP38 mAbs from a CCHFV survivor, which revealed two new antigenic sites on GP38. It was also possible to solve the GP38 complex with CC5-17, one of the human-derived mAbs. At the same antigenic site, CC5-17 and c13G8 compete with one another, but CC5-17 binds free GP38 with a lot more affinity. Specific amino acid sites were found to be responsible for the mAbs' strong affinity for the five phylogenetic clades of CCHFV but not for the closely related nairovirus species Aigai virus. *In vivo* studies were carried out to determine whether the higher affinity of CC5-17 in comparison to c13G8 equated to greater protection, and they showed that c13G8 is more effective than the higher affinity CC5-17.

CONCLUSION

By measuring the binding affinities of c13G8 against GP38 from the CCHFV strains IbAr10200, Afg09, Turkey-2004, and Kosovo/Hoti, Bio Layer Interferometry was used to examine the effects of up to 20% differences in amino acid sequence between CCHFV strains. There was evidence of a similar sub-nanomolar affinity among Clades III–V members. Testing of c13G8 against the Aigai virus strain Pentalofos, the most closely related virus to CCHFV, was done to see if it had the potential to be broad-spectrum against other nairoviruses. It's interesting to note that when c13G8 interacted with GP38 from the Aigai virus, binding was completely eliminated and BLI could not detect it.