Collectin Interaction With Human Rhinovirus

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RationaleThe most prevalent cause of the common cold is the non-enveloped virus known as human rhinovirus (HRV), potentially inducing hospitalization in neonates, aggravate long-term respiratory disorders, and enhance the risk of pneumonia in people with immunocompromised conditions. Patients with chronic airway disease may potentially experience exacerbations due to HRV infections which currently have no approved treatment.The lung lining fluid surfactant contains the collectins such as surfactant protein-A (SP-A) and SP-D are crucial to innate pulmonary defences. They are composed of four regions: a N-terminal domain, a neck region, a collagenous region, and a carbohydrate recognition domain (CRD). The neck, CRD, and a short collagenous segment of SP-D make up a recombinant of human fragment of SP-D (rfhSP-D), which has been developed and harbouring many of the functions found for the native human full-length SP-D protein. The antiviral properties of SP-A and SP-D against respiratory syncytial virus and influenza A virus have been shown both in vitro and in vivo. We have previously shown that both SP-D and rfhSP-D can also bind to HRV, which is a non-enveloped and non-glycosylated virus, in a calcium dependent manner whereas no binding was observed for SP-A. The current investigation focused on the location of SP-D binding to the three viral surface capsid proteins on HRV: VP1, VP2, and VP3. MethodsThe HRV's virus capsid proteins were expressed in Escherichia coli. The binding of SP-D and rfhSP-D was assessed to recombinant VP1, VP2, and VP3's using surface plasmon resonance (SPR) and enzyme linked immunosorbent assay (ELISA). Results Binding between VP3 and both SP-D and rfhSP-D was observed using SPR. The binding was calcium- and concentration-dependent and no binding was seen in the presence of EDTA. No binding was seen for SP-A. Also, no binding was observed for SP-D/rfhSP-D and VP1 or VP-2, respectively. The binding between SP-D/rfhSP-D and VP3 was confirmed by ELISA. This indicates that SP-D binds to VP3 on the surface of HRV and is involving the CRD region of SP-D. ConclusionsThis finding potentially broadens the function of SP-D in the immune system to a more general anti-airway viral protein. More research is needed to evaluate if this interaction can be exploited for therapeutic purposes in the future against acute and chronic respiratory diseases in both neonates, children, and adults.

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Methods

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Results

Binding between VP3 and both SP-D and rfhSP-D was observed using SPR. The binding was calcium- and concentration-dependent and no binding was seen in the presence of EDTA. No binding was seen for SP-A. Also, no binding was observed for SP-D/rfhSP-D and VP1 or VP-2, respectively. The binding between SP-D/rfhSP-D and VP3 was confirmed by ELISA. This indicates that SP-D binds to VP3 on the surface of HRV and is involving the CRD region of SP-D.

Conclusions

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