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Structural changes of pulmonary surfactant induced by bacterial lipopolysaccharide and by Polymyxin B

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Pulmonary surfactant (PS) is a mixture of lipids (~90 %) and 8-10 % specific surfactant associated proteins. PS lines the interior of the lung alveoli and acts to lower interfacial tension. The absence of PS due to prematurity, or its damage, is treated by exogenous PS in neonatal medicine. Curosurf (Cur) is one such clinically used replacement surfactants. It is an extract of porcine lung tissue consisting of at least 50 different phospholipids and contains a small amount of the essential protein SP-B (~2 wt%). Structurally, Cur is a mixture of uni-, oligo- and multilamellar vesicles. After inhalation, bacterial endotoxin, lipopolysaccharide (LPS) interferes with PS. We evaluated functional and structural changes of Cur in the presence of LPS using pulsating bubble surfactometer, optical microscopy, small angle neutron (SANS) and X-ray scattering (SAXS/WAXS). LPS bound to the lipid bilayer of Cur and disturbed its lamellar structure by swelling. The structural changes were attributed to the surface charge unbalance of the lipid bilayers due to LPS insertion. Polymyxin B (PxB) is an antimicrobial peptide primarily used in clinical practice to treat infections by resistant Gram-negative bacteria. In addition, PxB improves the surface properties of exogenous pulmonary surfactant [1]. Our SAXS experiments revealed that PxB acts as an inhibitor of structural disarrangement induced by LPS and restores original lamellar packing [2]. The lipid bilayer thickness was determined from SANS curves using the model of vesicles.

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References

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