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Differential regulation and function of SP-A1 and SP-A2: Proteins of lung innate immunity

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Surfactant protein A(SP-A) plays an important role in innate immunity and surfactant-related functions. Dysregulation of immunity and/or surfactant function occur in most pulmonary diseases. Because levels and genetic variants of the surfactant protein A have been associated with susceptibility in several pulmonary diseases it is imperative that we understand their regulatory mechanisms with the hope of identifying points of therapeutic intervention. Two functional genes (hSP-A1; hSP-A2) encode SP-A in humans, and several genetic and splice variants have been identified for each gene. Functional and regulatory differences have been observed between hSP-A1 and hSP-A2. Both 5' and 3' untranslated regions (UTR) play important roles in the translational regulation and mRNA stability of SP-A1 and SP-A2. The 5'UTRs exhibit alternative splicing of untranslated exons resulting in different splice variants for hSP-A1 and hSP-A2, and the 3'UTRs exhibit sequence variability. The SP-A2 ABD/ABD' 5'UTR contains exon B (eB) which is absent from the SP-A1 AD 5'UTR. eB is an enhancer of transcription and translation and contains cis regulatory elements. Specific trans-acting factors including several members of the 14-3-3 family of proteins bind eB and inhibition of specific members of the 14-3-3 family results in a decrease of the SP-A2 protein, but not SP-A1, indicating the role and specificity of 14-3-3 in the differential regulation of SP-A1 and SP-A2. Polymorphisms at the 3'UTR are involved in the regulation of SP-A1 and SP-A2 variants as these provide differential binding sites for miRNAs, noncoding RNAs, shown previously to regulate gene expression by affecting mRNA stability and/or translation. The available data indicate that both 5' and 3'UTR are important regulators of SP-A1 and SP-A2 as these contain cis-elements for trans-acting protein and/or miRNA binding. A better understanding of the SP-A1 and SP-A2 may provide points for therapeutic intervention in disease where there is an SP-A-dependent derangement of innate immunity and/or surfactant. Because SP-A variants differentially affect the function and protein expression profile of the alveolar macrophage, the sentinel cell of lung innate immunity, the knowledge gained may further help to better understand macrophage functions such as phagocytosis and clearance of bacteria and inflammatory processes.

Biography

Joanna Floros is curently serving as professor of Pediatrics at Penn State University in College of Medicine. Her interest is in Differential regulation and function of SP-A1 and SP-A2: Proteins of lung innate immunity.

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