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The Aetiology of Pneumonia and Lung Lipid Metabolism

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DESCRIPTION

During the COVID-19 pandemic, pneumonia, an acute respiratory disease with varying etiologies, received a lot of attention. Among the many aspects of pneumonia that have received extensive research, lipid metabolism has received insufficient attention. Both covid-19 associated and non-covid-19 associated pneumonias in human lungs are examined here for abnormal lipid metabolism. Morphometric analysis revealed lipid depositions both extracellular and intracellular, particularly in vessels adjacent to inflamed regions, where they appear to impede blood flow. On Sudan III and Oil Red Ostained cryosections, as well as on OsO_4-contrasted semi and ultra-thin sections, lipids were visible. Inflammation sites had a higher content of unsaturated fatty acids than intact lung sites, according to chromatographic mass spectrometry. qPCR and *in silico* RNA sequence analysis revealed that pneumonia caused a down regulation of the lipid metabolism genes.

Pneumonia includes a variety of life-threatening conditions that can have a variety of aetiologies, such as bacterial, viral, fungal, mycoplasma, or lipoid, and affect the alveoli, distal bronchioli, or interstitial lung tissue. It, along with other infections of the lower respiratory tract, is responsible for over 2.49 million deaths in 2019, with the highest mortality rate among patients over 70 years old (1.23 million deaths), making it the most fatal infectious disease. The seriousness and gamble with factors rely upon pneumonia type and host science, including invulnerable obstruction and tissue versatility, and are overall completely contemplated. The specific dysregulations in lipid metabolism that are associated with lung inflammation of various etiologies have been addressed much less, despite the fact that a large number of morphological and physiological abnormalities in inflamed lungs have been reported to date. Since lung surfactant lipid synthesis plays the most important protective role against lung disease, this requires significant attention. Since the lung is SARS-CoV-2's primary organ of attack, a number of aspects of pneumonia in the context of covid-19 have recently received special attention. The increase in the activity of secreted phospholipase A2 (sPLA2) in blood plasma is the most significant change in lipid-modulating enzymes for covid-19 that has been reported thus far. However, it is still unknown whether these systemic abnormalities in lipid metabolism have an effect on the structure of inflamed lung tissue. In addition, it is unclear whether these lipid metabolism characteristics are unique to covid-19 associated pneumonia or if they are a general phenomenon. In this study, we investigated lipids in pneumonias of various causes to answer these queries. Lung samples stained with Sudan III, Oil Red O, or contrasted with OsO_4 lipids were examined with light and electron microscopy to examine the patterns of lipid distribution in various pneumonias and the inflamed lung versus the intact lung. To compare pneumonias of various etiologies and non-inflamed lungs, we also utilized chromato-mass spectrometry of fatty acids and evaluated the expression of key enzymes of lipid metabolism *in silico* (through RNA-seq database analysis) and directly on autopsy specimens (through qPCR). We discovered that the lipids in inflamed lung tissue were deposited as droplets in the cytoplasm of cells, in the extracellular space, and within the small blood vessels regardless of covid-19 status. The lipid beads were for the most part situated inside cells around the vessels or in their area. Inflammed areas of the same lung were more likely to accumulate lipids than non-inflamed ones, and control intact lung tissue was not affected. Increased levels of unsaturated fatty acids and decreased expression of enzymes involved in lung lipid metabolism were linked to lipid deposition.

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CONFLICT OF INTEREST

The author declares there is no conflict of interest in publishing this article.

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