〔Brain Nerve, **40**, 673 (1988)〕

Brain Tissue Leukotrienes in Cerebral Ischemia and Effect of Inhibitor of SRS-A Release on Postischemic Cerebral Edema.

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In order to test the development of lipoxygenase metabolites of arachidonic acid in cerebral ischemia, we measured free arachidonic acid, SRS-A and LTC_4 in the brain tissue. Moreover, we studied the influence of inhibitor of SRS-A release on postischemic cerebral edema. The arachidonic acid concentration increased strikingly during the ischemia and decreased rapidly after recirculation. The concentrations of SRS-A and LTC_4 increased significantly after recirculation and remained higher levels at 180 min after recirculation than the preischemic levels. Tranilast significantly reduced the increasing of postischemic brain water contents. It is suggested that leukotrienes may play some role in the pathogenesis of postischemic cerebral edema.

〔Arch. Biochem. Biophys., **264**, 492 (1988)〕

Subcellular Distribution and Properties of Carbonyl Reductase in Guniea Pig Lung.

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Carbonyl reductase activity in guinea pig lung was found in the mitochondrial, microsomal, and cytosolic fractions; the specific activity in the mitochondrial fraction was more than five times higher than those in the other fractions. The enzymes were solubilized with 1% Triton X-100 and 1 M KCl from the particulate fractions, and purified to homogeneity. The enzymes from the three fractions were almost identical in catalytic, structural, and immunological properties. Carbonyl reductase, synthesized in a rabbit reticulocyte lysate cell-free system, was apparently the same in molecular size as the subunit of the mature enzyme purified from cytosol.

〔Biochem. J., **252**, 17 (1988)〕

Kinetic Mechanism of Pulmonary Carbonyl Reductase

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The kinetic mechanism of guinea pig lung carbonyl reductase was studied at pH 7 in the forward reaction with five carbonyl substrates and NAD(P)H and in the reverse reaction with propen-2-ol and NAD(P). In each case the enzyme mechanism was sequential, and product-inhibition studies were consistent with a di-iso ordered bi bi mechanism, in which NAD(P)H binds to the enzyme first and NAD(P) leaves last and the binding of cofactor induces isomerization. The kinetic and binding studies of the cofactors and several inhibitors such as pyrazole, benzoic acid, Cibacron blue and benzamide indicate that the cofactor and Cibacron blue bind the free enzyme whereas the other inhibitors bind to the binary and/or ternary complexes.