Thromboxane mediates the increase in alveolar surfactant pool induced by free fatty acid infusion in the rabbit

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Intravenous infusion of free fatty acid (FFA) produces pulmonary edema and an increase in the alveolar surfactant content of the rabbit. In order to identify a likely mediator of this lung response to FFA, we used inhibitors of cyclo-oxygenase (indomethacin, 15 mg.kg⁻¹ i.v., or meclofenamate, 5 mg.kg⁻¹ i.v.) and thromboxane syn-thetase inhibitors (imidazole, 50 mg.kg⁻¹ i.v. or dazoxiben, 2 mg.kg⁻¹ i.v.) which were administered before FFA, 20 mg. kg⁻¹ min⁻¹ i.v., in four different experimental series (n = 54). Lung surfactant was measured in bronchial-alveolar lavage fluid by determining disaturated phosphatidylcholine (DSPC). Both kinds of inhibitors blocked the increase in FFA-induced DSPC. They increased the survival rate but they only slightly changed the post-FFA morphofunctional pulmonary alterations. We conclude that the increase in alveolar surfactant induced by FFA is likely mediated by thromboxane. This mediator