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Title: Severity of ventilator induced lung injury does not contribute to ventilator induced diaphragmatic dysfunction

Dr. Christian 2037 Bruells cbruells@ukaachen.de MD [1], Mrs. Kathy 2038 Reiss kreiss@ukaachen.de [3], Mrs. Ashley 2039 Smuder asmuder@hhp.ufl.edu [2], Dr. Matthew 2040 Hudson mhudson@hhp.ufl.edu [2], Mr. Bradley 2041 Nelson bnelson@ufl.edu [2], Prof. Dr Stefan 2042 Uhlig suhlig@ukaachen.de [3] and Prof. Dr Scott 2043 Powers spowers@hhp.ufl.edu [2]. [1] Department of Anesthesiology, Aachen University Clinic of the RWTH Aachen, Aachen, Germany, 52074 ; [2] Inst of Applied Physiology and Kinesiology, College of Human Health and Performance, University of Florida, Gainesville, FL, United States, 32611 and [3] Inst. of Pharmacology and Toxicology, RWTH Aachen, Aachen, Germany, 52074 .

Body: Mechanical ventilation (MV) is a life-saving intervention for patients with respiratory failure. Even 12h of MV can promote diaphragmatic contractile dysfunction and atrophy (referred to as ventilator-induced diaphragmatic dysfunction, VIDD).The pathophysiology remains unclear but could be linked to inactivity, the physiological impact of positive pressure ventilation (PPV) on the diaphragm and/or ventilator-induced lung injury (VILI). We tested the hypothesis, if negative pressure ventilation (NPV) compared to PPV will diminish VIDD. The concomitant influence of VILI on VIDD was also examined. Rats were ventilated with either PPV or NPV or breathed spontaneously (control) for 12h. We measured diaphragmatic contractile properties, fiber size and markers of oxidative damage. Lungs were histologically examined and cytokine levels were assayed in bronchoalveolar lavage for evidence of VILI. Compared to control, both PPV and NPV resulted in significant oxidative damage to the diaphragm along with fiber atrophy and contractile dysfunction. No significant differences existed in these measures between PPV and NPV groups. Both the PPV and NPV groups experienced VILI, graded by histologic scores or cytokines. Note, that the severity of VILI varied between animals within both the PPV and NPV groups. Nonetheless, the severity of VILI was not significantly correlated with the degree of VIDD. Both PPV and NPV promote VIDD and VILI. The magnitude of VILI is not correlated with the degree of VIDD. Although these findings do not eliminate the possibility that VILI may play a role in VIDD, our results are consistent with the concept that diaphragmatic inactivation as major contributor to VIDD.