The dopaminergic system in pulmonary hypertension

Dr. Pierdonato 17175 Bruno pierdo.bruno@gmail.com MD 1, Prof. Carmine Dario 17176 Vizza dario.vizza@uniroma1.it MD 2, Dr. Alberto 17177 Ricci alberto.ricci@uniroma1.it MD 1, Dr. Davide 17178 Scozzi davide_scozzi@libero.it MD 1, Dr. Maria Cristina 17179 Esposito mcristina.esposito@virgilio.it MD 1, Dr. Emanuela 17180 Cherubini emanuelacherubini@yahoo.it 1, Dr. Giorgia Amira 28403 Osman giorgiamira@hotmail.com MD 1, Dr. Silvia 17181 Papa silviapapa83@gmail.com MD 2, Dr. Martina 17187 Nocioni martinanocioni@yahoo.it MD 2, Dr. Mario 17188 Mezzapesa mario.mezzapesa@gmail.com MD 2 and Prof. Salvatore 17190 Mariotta salvatore.mariotta@uniroma1.it MD 1. 1 Clinic and Molecular Medicine, Sapienza University, Rome, Italy and 2 Cardiovascular, Respiratory Nephrology and Geriatric Sciences, Sapienza University, Rome, Italy.

Body: The purpose was to investigate the possible role of dopamine in the regulation of the pulmonary circulation in patients with pulmonary hypertension (PH). We enrolled 40 patients with PH (24 M/16 F; mean age 65) and 5 patients without PH (3 M/2 F; mean age 74); divided into groups according to the severity of PH as measured by pulmonary artery pressure (PAP) [severe=20 pts, moderate=10 pts, mild=10 pts]. 30 ml of blood from pulmonary artery and from peripheral arterial blood were taken out during each right heart catheterization for the determination of circulating catecholamines, receptors (D1DR-D5DR) and transporters (Mat-V-1, V-2-Mat, h-Dat) of dopamine. In patients (40 pts) with PH the increase of PAP was correlated (p<0.05) with a decrease in transmembrane transporters of dopamine (HDAT), with an increase of the concentration of adrenaline in the peripheral circulation (p<0.05), and with a decrease of the difference between lung and peripheral dopamine (p<0.05). In patients with mild PH (10 pts) the increase in right atrial pressure (PAD) and cardiac output (PO) is correlated (p<0.01) with an increase of dopamine receptors (D4R). From these observations, it might be theorized a decrease in the availability of dopamine in patients with pulmonary hypertension, which could cause an up regulation of its receptors and a reduction of its carriers; the decrease in the availability of dopamine may be a compensation to adrenergic hypertonia, given the correlation between PAP and circulating levels of adrenaline. The reduction of availability could be explained by an increased metabolism in the pulmonary circulation (inverse correlation between PAP and difference between lung and peripheral dopamine). Further studies are necessary.