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Errata

"Effect of oxygen on breath markers of oxidative stress". M. Phillips, R.N. Cataneo, J. Greenberg, R. Grodman, R. Gunawardena, A. Naidu. *Eur Respir J* 2003; 21: 48–51.

Unfortunately, the p-value was omitted from the legend for figure 2. The corrected legend is reproduced below with the figure.

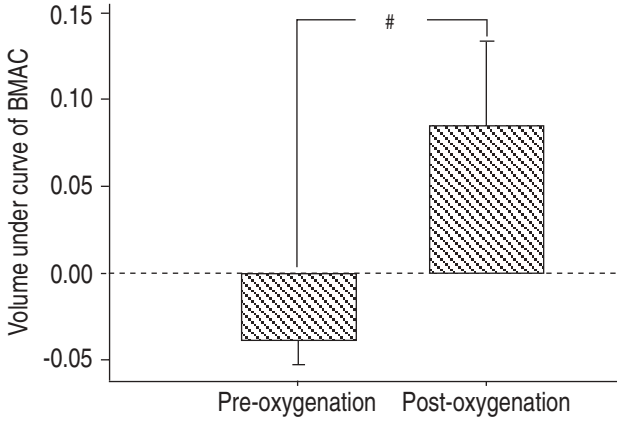

Fig. 2. – Mean volume under the curve (VUC) of the breath methylated alkane contour (BMAC) before and after oxygenation. This diagram displays the mean VUC of the BMACs in figure 1 (error bar=SEM). The change demonstrates a significant global increase in the volatile markers of oxidative stress that comprise the BMAC. #: $p<0.02$.

"Genomic DNA extraction from small amounts of serum to be used for α_1-antitrypsin genotype analysis". S. Andolfatto, F. Namour, A-L. Garnier, F. Chabot, J-L. Gueant, I. Aimone-Gastin. *Eur Respir J* 2003; 21: 215–219.

Unfortunately, there was an error in the first paragraph of the introduction on page 215. The PI ZZ prevalence for Western and Northern Europe should read 1:1,000–1:4,500 and not 1:1,000–1:145,000. The PI ZZ prevalence for Central Europe should read 1:4,500–1:10,000 and not 1:45,000–1:10,000. The corrected paragraph is produced in full below.

The α_1-antiprotease inhibitor (Pi), or α_1-antitrypsin (α_1-AT), is the principal serum inhibitor of lysosomal proteases, such as neutrophil elastase [1]. The α_1-AT is a polymorphic single chain glycoprotein of 52 kDa and 394 amino acids, synthesised in the liver and normally present in serum at 150–350 $mg \cdot dL^{-1}$ [2]. It displays >90 different genetically determined phenotypes [3]: phenotype M is the normal variant (90% of the population) and phenotypes S and Z are the two most frequent abnormal variants [3]. Calculated values of PI ZZ prevalence are approximately: 1:1,000–1:4,500 in Western and Northern Europe; 1:4,500–1:10,000 in Central Europe; and 1:10,000–1:90,000 in Eastern Europe and in the southernmost and northernmost areas of the continent. In the white population of USA, Canada, Australia and New Zealand, PI ZZ phenotype prevalence ranges from 1:2,000–1:7,000 individuals. In nonwhite populations α_1-AT deficiency is thought to be a rare or nonexistent disease [4, 5]. Homozygosity for the Z phenotype is the principal cause of α_1-AT deficiency. It typically leads to the development of diverse liver diseases in children and adults and to early adult onset emphysema, with plasma level of α_1-AT in homozygous PiZ individuals reaching only 10–15% of α_1-AT concentration observed in PiM individuals [6, 7]. Although individuals' MS or SS are unaffected, SZ subjects may be symptomatic. More recently, α_1-AT deficiency has been associated with asthma, bronchiectasis, vasculitis and panniculitis [8, 9].