Body: Background. Alveolar macrophages (AM) in experimental models can express a proinflammatory M1 or an antiinflammatory M2 phenotype. The presence and function of these phenotypes in human disease have not been established. Aim. To study if the M1 and M2 phenotypes of AM are expressed in humans, in particular in lung tissue of nonsmokers and smokers with and without COPD. Methods. Alveolar macrophages were immunostained in sequential slides for M1 (iNOS) and M2 (CD206) in lungs of 11 severe COPD, 13 smokers without COPD and 11 nonsmokers. M1 and M2 AM were counted and expressed as percentage of total AM. Results. In COPD the proportion of M1 [53(16-81)\%] and M2 [76(40-98)\%] was higher than in nonsmokers: M1 [24(11-44)\%] and M2 [44(0-88)\%], p<0.05 for both. The proportion of M1 was also higher in smokers without COPD [52(31-81)\%], than in nonsmokers [24(11-44)\%] (p=0.009). In COPD there were more M2 than M1 AM (p=0.03). The sum of the percentage of AM expressing M1 and M2 markers exceeded 100% in most of COPD subjects [132(85-159)\%] and smokers without COPD [124(87-144)\%] suggesting that a high proportion of AM expressed both M1 and M2 markers. From these data we calculated that the alveolar macrophages M0 (no M1, no M2) decreased from a minimum of 32% in nonsmokers to a minimum of 15% in smokers with COPD (p=0.004). Conclusions. Alveolar macrophages can be present as M0, M1 and M2 in normal and diseased lungs. Nonsmokers have a high proportion of M0 macrophages that decreases markedly in COPD. With the development of the chronic inflammation characteristic of COPD alveolar macrophages express both M1 and M2 markers in high proportion, often simultaneously.