"Bronchial thermoplasty decreases airway remodelling by blocking epithelium-derived heat shock protein-60 secretion and protein arginine methyltransferase-1 in fibroblasts." Qingzhu Sun, Lei Fang, Michael Roth, *et al*. *Eur Respir J* 2019; 54: 1900300.

Figure 8 from the above mentioned article was originally published in an incorrect form. The first three immunofluorescence image panels in figure 8b were inadvertently duplicated from figure 7b. The corrected figure 8 with the relevant images in panel 8b replaced is shown below, and the original research article has been corrected and republished online.

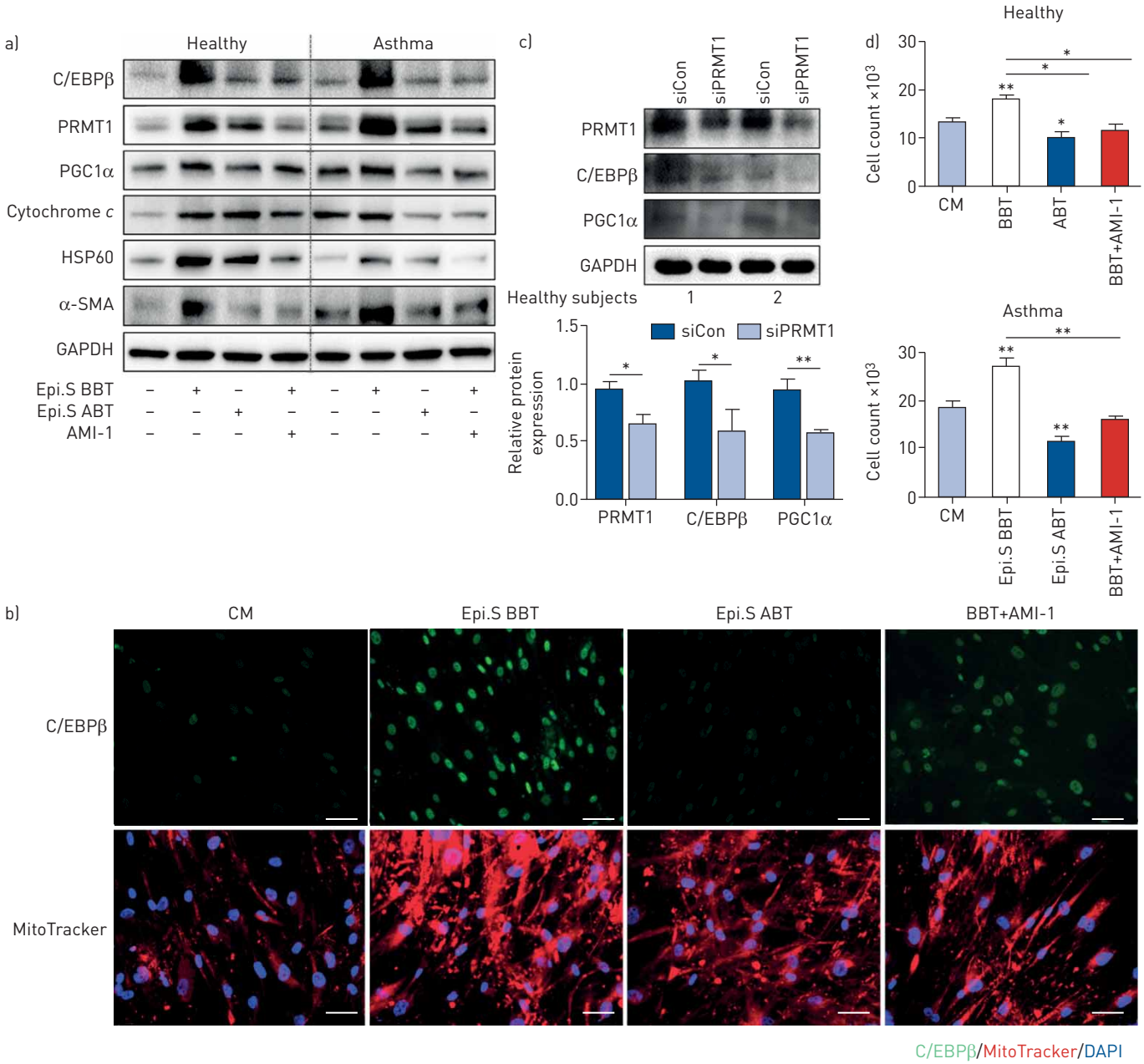

FIGURE 8 Protein arginine methyltransferase-1 (PRMT1) inhibition reduced mitochondrial mass and regulators. C/EBPβ: CCAAT enhancer-binding protein-β; PGC1α: peroxisome proliferator-activated receptor-γ coactivator-1α; HSP60: heat shock protein-60; α-SMA: smooth muscle actin; GAPDH: glyceraldehyde 3-phosphate dehydrogenase; Epi.S: epithelial cell culture supernatant; BT: bronchial thermoplasty; BBT: before BT; ABT: after BT; si: small interfering; Con: control; CM: control medium. a) BBT Epi.S-induced expression of mitochondria activators was reduced by AMI-1. The quantitative analysis of all Western blots (n=4 in each group) is provided in supplementary figure S5b. b) Representative immunofluorescence of BBT Epi.S-induced expression of C/EBPβ and MitoTracker in the presence and absence of AMI-1. Scale bars: 10 µm. c) Representative Western blot of siRNA for PRMT1 (siPRMT1) on the expression of PGC1α and C/EBPβ. The quantitative analysis of all Western blots (n=3) is shown in the bar chart below the blot. d) The effect of AMI-1 on BBT Epi.S-induced fibroblast proliferation (n=3). Data are presented as mean±SEM. *: p<0.05; **: p<0.01, comparing control *versus* other groups.