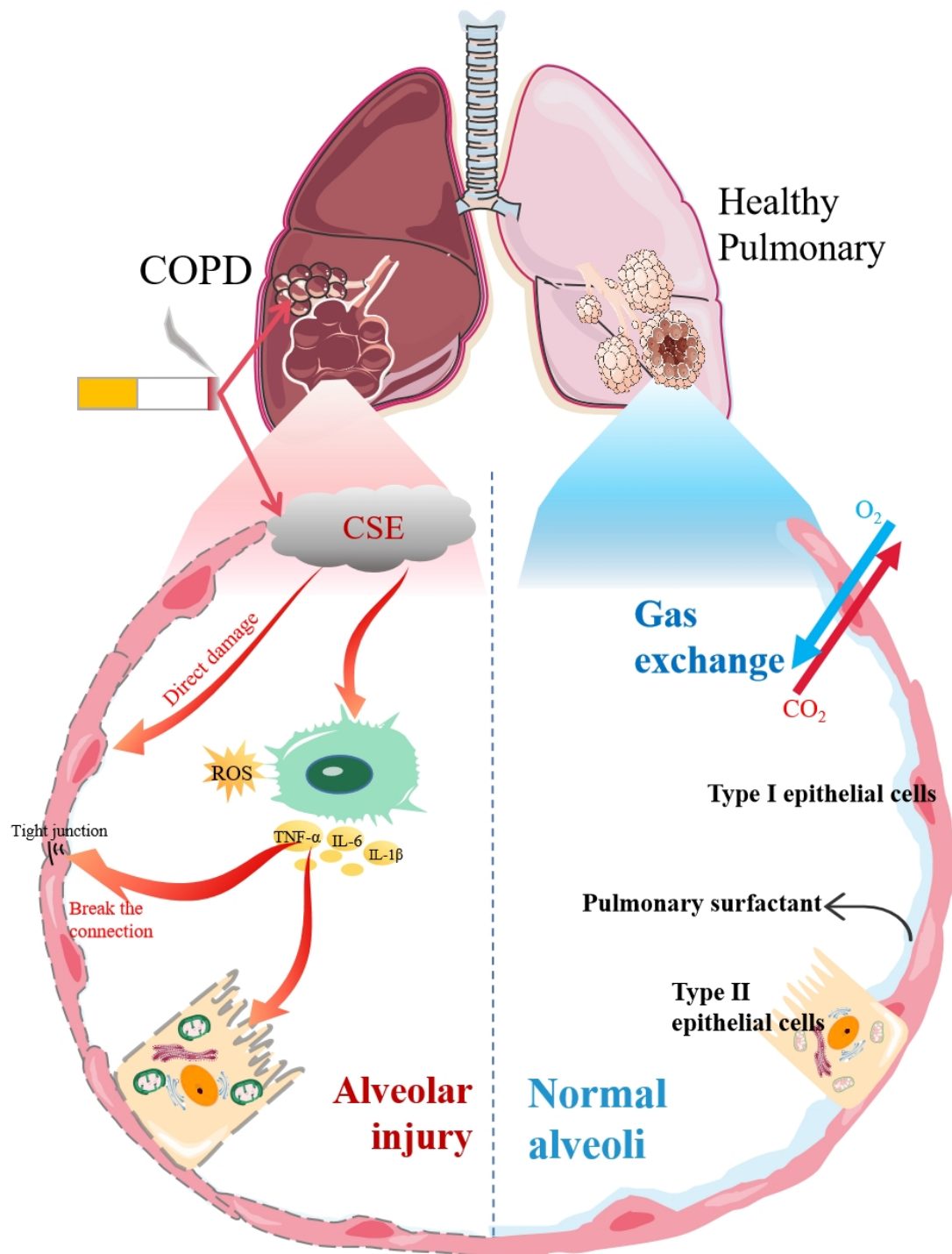
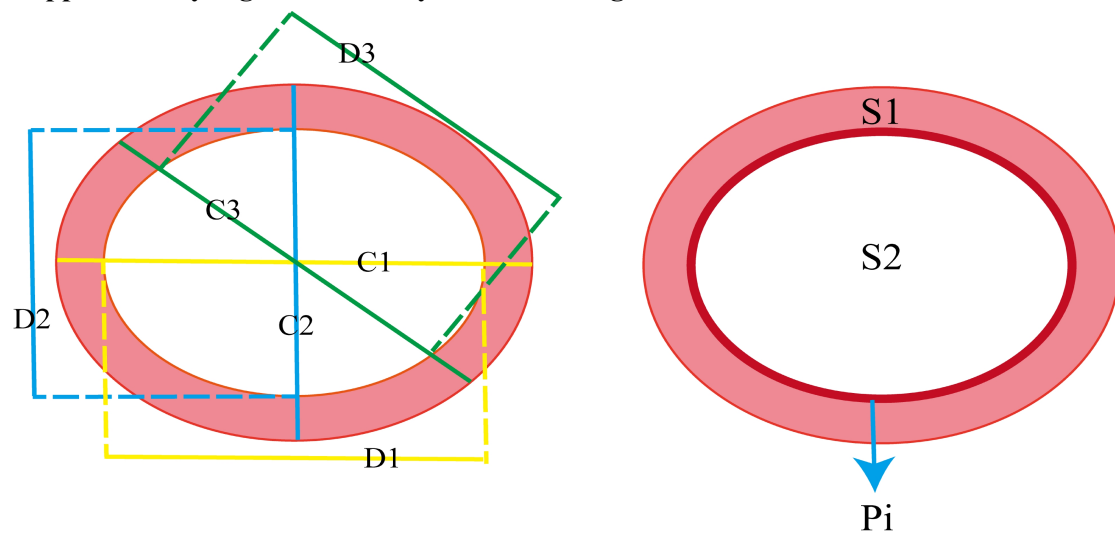


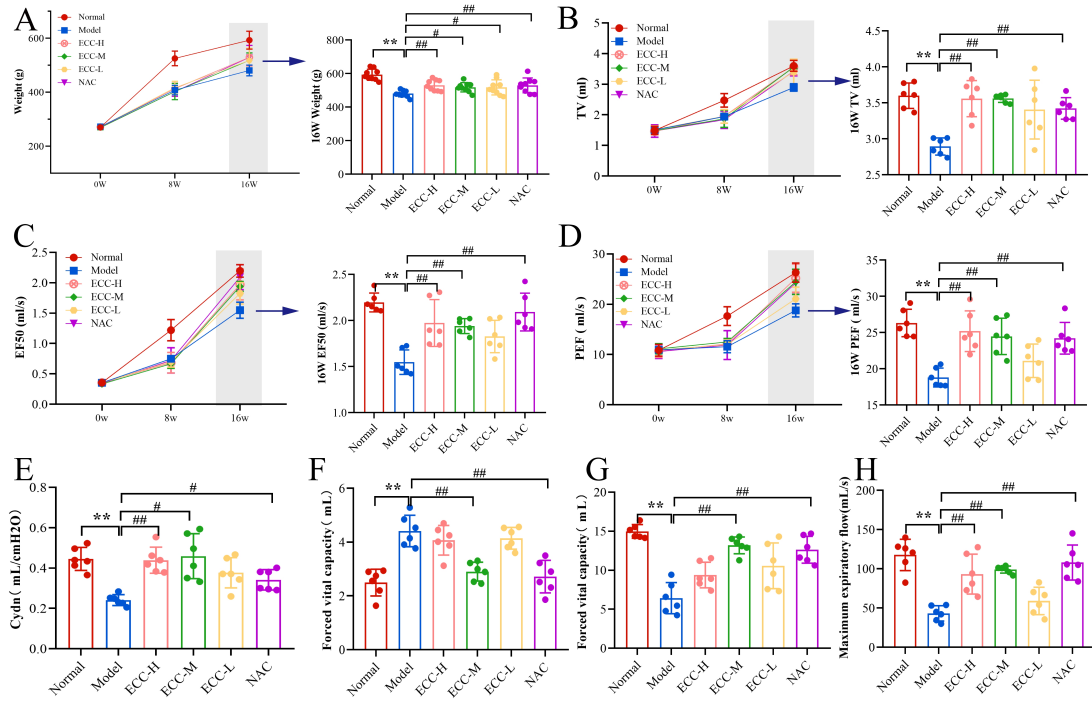
Supplementary Figure S1 Mechanism diagram of cigarette smoke induces inflammatory damage to alveolar epithelial barrier. Cigarette smoke can penetrate the alveoli, leading to inflammation and alveolar epithelial barrier damage. During alveolar epithelial barrier damage, inflammatory cells enter the alveolar space via impaired epithelial barrier integrity. Inflammatory mediators compromise the tight junctions between epithelial cells.



Supplementary Figure S2 Airway schematic diagram.



Supplementary Figure S3 The effect of the ECC-BYF on the COPD rats. (A) Weight. (B) tidal volume (TV). (C) expiratory flow at 50% tidal volume (EF50). (D) the peak expiratory flow (PEF). (E) dynamic compliance (C_{dyn}). (F) functional residual capacity (FRC) (G) forced vital capacity (FVC). (H) maximum expiratory flow (MMEF). *P* < 0.01, compared to the normal group. ##*P* < 0.01, #*P* < 0.05, compared to the model group.**



Supplementary Figure S4 ECC-BYF improved the damage and function of alveolar epithelial barrier induced by TNF- α . (A) The cell viability was detected using CCK8 reagent. (B) the effects of TNF- α on IL-6 mRNA in MLE-12 cells (C) the effects of TNF- α on TNF- α mRNA in MLE-12 cells. (D) The cell viability was detected using CCK8 reagent. (E) the effects of ECC-BYF on IL-6 mRNA in TNF- α -induced MLE-12 cells (F) the effects of ECC-BYF on TNF- α mRNA in TNF- α -induced MLE-12 cells (G) the content of SP-C in MLE-12 cells was measured using IF. DAPI blue fluorescent label; SP-C red fluorescent label; Merge Blue and red merge tags. The mean \pm SEM was used as the expression of values. ** $P < 0.01$, compared to the normal group. ## $P < 0.01$, compared to the model group.

