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Gaps and bridges in evolving rationale of COPD pharmacotherapy

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COPD pharmacotherapy aims at treatable traits of a disease previously considered irreversible. The evolution of COPD pharmacotherapy follows two different paths, related to the British and Dutch hypotheses, respectively. The first path seeks "COPD specific" traits, e.g., ameliorating air-trapping and combating non-eosinophilic inflammation, currently represented by the long acting bronchodilators and PDE4 inhibitors. The second path is a "copy-paste" approach utilizing asthma therapeutics in COPD, best represented by the current overuse of inhaled corticosteroids (ICS) to treat "COPD at-large". In the past, short-acting bronchodilators were used to treat COPD with modest effectiveness until the advent of long-acting beta agonists (LABA). Early clinical research of ICS in COPD showed some symptomatic improvement with a questionable effect on exacerbations without affecting FEV1 decline or mortality. The availability of ICS-LABA combination inhales and the development of long-acting anti-muscarinics (LAMA) set the stage for debating which pharmacotherapy is better for moderate-severe COPD. The COPD mega-trials, ToRCH and UPLIFT failed to achieved statistical significance in their primary outcomes. However, these studies demonstrate the salutary effects of LABA and LAMA on COPD outcome measures including exacerbations. Factorial analysis of ToRCH contributed to understanding the limited role of ICS, weighed against the increased risk of pneumonia. In the SUMMIT study, ICS-LABA failed to decrease cardiovascular mortality in moderate COPD. While "systemic-COPD" affects comorbidity, ICS-LABA treatment failed to show effectiveness in this regard. The recent definition of Asthma COPD overlap syndrome along with the, yet untested, recommendation for the use of ICS-LABA in these patients deserves further consideration. GOLD 2017 prefers LAMA to LABA as the first line long-acting bronchodilator, and recommends using LAMA-LABA combination before ICS in exacerbation prone COPD. An argument can be made for better definition at diagnosis (FEV1/FVC < LLN threshold) as well patient phenotyping aiming to improve the therapeutic success.

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