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Editorial

Speaking of pandemics...

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We are in the midst of a persistent pandemic that threatens the health of the population we care for, particularly impacts on the socially disadvantaged, and whose control has been undermined by complacency and failure to generate consensus on adoption of measures to promote behavioral changes to contain its impact. Or perhaps two, because I am now referring to the tobacco pandemic, which in fact continues to be a greater threat than COVID-19. According to the World Health Organization, tobacco kills more than 8 million people a year around the world, 1.2 million of whom are non-smokers being exposed to second-hand smoke [1]. Nonetheless, tobacco use continues to be rampant, reported by 21% of adults in the U.S [2], and 26% in Europe (with significant variation among different countries); lower educational status and household income are important risk factors internationally [3].

The adverse effects of second hand tobacco smoke exposure (TSE) on the health of children has been known for a long time, and the association of TSE with poor lung function and growth in children with CF has been repeatedly documented over the last 30 years [4]. Surprisingly enough, the prevalence of tobacco use among parents of children with CF seems to be fairly similar to that of the general population [5–8].

Relatively recent analyses of the Early Pseudomonas Infection Control (EPIC) database [5] and of the Cystic Fibrosis Foundation Patient Registry (CFFPR) [6] quantified the impact of secondhand TSE on pediatric CF populations with an emphasis on its contribution to socioeconomic disparities. They found a similarly high (27%-29%) prevalence of secondhand TSE exposure in children with CF that varied more than two-fold by household income, parental education, and insurance status (private vs public). These studies found FEV1pp to be 4-8% lower in children with second hand TSE compared to those who had no TSE exposure; adjustment for several different measures of socioeconomic status reduced the apparent impact of smoke exposure to some degree, but overall both studies found that second hand TSE exposure was additive to and magnified SES-related disparities in lung function. The EPIC study also reported similar effects in relation to weight percentile [5].

Thus TSE is a highly prevalent risk factor for adverse pulmonary as well as nutritional outcomes in CF, and has a particular impact on low SES populations who are already at a disadvantage. In this context, it should be noted that despite an increased awareness of its existence, socioeconomic disparities in CF outcomes have been unchanged over the several decades since their initial recognition [9]. Counselling that focuses on TSE reduction presents a badly needed and relatively accessible tool in the our attempts to eliminate or at lease reduce these disparities [10]. A paper in this issue of JCF seems to show that attenuation of TSE can make a difference in lung function of people with CF. Oates et al [7] performed an analysis of data on reported TSE in the CFFPR and found a substantial reduction in pulmonary exacerbations in the first year of apparent cessation of TSE (as determined by exposure reported in the registry), with an additional smaller decrease for each additional year of cessation after that. TSE cessation was also associated with similar large improvements in ppFEV1 and BMI in the first year and then ongoing but smaller increases in follow-up years.

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A second paper in this issue of JCF by Baker et al [8] addresses how TSE might lead to an even larger relative deficit in lung function and differentially greater impact on socioeconomic disparities in the new era of highly effective CFTR modulators. Extrapolating from data in animals and humans that cigarette smoke inhibits CFTR function [11], they used CFFPR data to examine the benefit of newly prescribed tezacaftor/ivacaftor in pediatric patients 12 years of age and older over a two year period, comparing those who had TSE to those that did not. The study found that tezacaftor/ivacaftor use was associated with an improvement in FEV1pp in those patients who did not have TSE, but no improvement in those with TSE, leading to a small but statistically significant increase in the pre-existing difference in FEV1pp associated with smoke exposure. Of course, treatment of CF with tezacaftor/ivacaftor leads to relatively small increments in lung function, but if TSE similarly blunts the much greater impact of highly effective CFTR modulator treatment, the repercussions are obviously huge.

There are known limitations in self-report of tobacco use, particularly in populations facing possible disapproval [12], as well as known inconsistencies in pediatric providers' determination of parental smoking habits. The CFFPR data regarding smoke exposure is just starting to be used in analyses and has never been validated, but the consistency of the results of these two studies and the apparent dose-response relationship is reassuring in this regard. Furthermore, potential biases in reporting would most likely lead to under-ascertainment of exposures, which in turn would be more likely to underestimate rather than overestimate the apparent effect of TSE.

In summary, TSE leads to deficits in lung function and weight gain in people with CF and is an important contributor to socioeconomic disparities. There is a real potential concern that TSE will attenuate the effect of highly effective CFTR modulator drugs in people with CF who are exposed to smoke, and therefore lead to a magnification of SES-related disparities as we enter this new era of CF care. An optimistic note, however, is sounded by the findings of Oates et al indicating that cessation of TSE will lead to a reversal of its adverse effects, with a decrease in pulmonary exacerbations and improvements in FEV1 and weight. Feasible programs that can be initiated in the setting of pediatric practices have been shown to reduce second-hand TSE [13] and pediatric practice-based programs to counsel parents on smoking cessation have some (if limited) proven effectiveness [14]. Active intervention for smoking parents has been endorsed by the American Academy of Pediatrics [15]; surveys of parents show that advice on smoking cessation from their child's physician would be welcomed by most parents [16]. CF care providers, however, rarely offer any structured approach to smoking cessation counselling for parent caregivers, despite previous attempts to bring it to attention [17]; a recent survey of pediatric CF care providers found several reasons for this, including inadequate training, time constraints, reluctance to enter a therapeutic relationship with parents, and lack of attention from CF care guidelines and educational programs [18]. However, this was the state of mental health screening in CF just a few years ago, and sustained efforts on both sides of the Atlantic along with hard work by CF teams have resulted in dramatic improvements in mental health care [19]. Studies of general pediatricians show that education on smoke reduction counselling and a relatively small commitment of time can lead to success in screening, counselling, and behavior change [20]. Despite the barriers, given the large potential impact of eliminating secondhand smoke exposure in our most vulnerable patients, it behooves us to put TSE screening and counselling into CF care guidelines, educate care teams, and promote systematic screening and tobacco cessation counselling. We can do better [10].

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Declaration of Competing Interest

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