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# Antigen Stasis can Predispose Primary Ciliary Dyskinesia (PCD) Patients to Asthma

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**Abstract :** Introduction: We have observed that many patients with Primary Ciliary Dyskinesia (PCD) benefit from asthma medications. In healthy airways, the ciliary function is normal. Antigens and irritants are rapidly cleared, and NO enters the gas phase normally to be exhaled. In the PCD airways, however, antigens, such as Dermatophagoides, are not as well cleared. This defect leads to oxidative stress, marked by increased DUOX1 expression and decreased superoxide dismutase [SOD] activity (manuscript under revision). $H_2O_2$, in high concentrations in the PCD airway, injures the airway. NO is oxidized rather than being exhaled, forming cytotoxic peroxynitrous acid. Thus, antigen stasis on PCD airway epithelium leads to airway injury and may predispose PCD patients to asthma. Indeed, recent population genetics suggest that PCD genes may be associated with asthma. We therefore hypothesized that PCD patients would be predisposed to having asthma. Methods. We analyzed our database of 18 million individual electronic medical records (EMRs) in the Indiana Network for Patient Care research database (INPCR). There is not an ICD10 code for PCD itself; code Q34.8 is most commonly used clinically. To validate analysis of this code, we queried patients who had an ICD10 code for both bronchiectasis and situs inversus totalis in INPCR. We also studied a validation cohort using the IBM Explorys® database (over 80 million individuals). Analyses were adjusted for age, sex and race using a 1 PCD: 3 controls matching method in INPCR and multivariable logistic regression in the IBM Explorys® database. Results. The prevalence of asthma ICD10 codes in subjects with a code Q34.8 was 67% vs 19% in controls ($P < 0.0001$) (Regenstrief Institute). Similarly, in IBM*Explorys, the OR [95% CI] for having asthma if a patient also had ICD10 code 34.8, relative to controls, was =4.04 [3.99; 4.09]. For situs inversus alone the OR [95% CI] was 4.42 [4.14; 4.71]; and bronchiectasis alone the OR [95% CI] =10.68 (10.56; 10.79). For both bronchiectasis and situs inversus together, the OR [95% CI] =28.80 (23.17; 35.81). Conclusions: PCD causes antigen stasis in the human airway (under review), likely predisposing to asthma in addition to oxidative and nitrosative stress and to airway injury. Here, we show that, by several different population-based metrics, and using two large databases, patients with PCD appear to have between a three- and 28-fold increased risk of having asthma. These data suggest that additional studies should be undertaken to understand the role of ciliary dysfunction in the pathogenesis and genetics of asthma. Decreased antigen clearance caused by ciliary dysfunction may be a risk factor for asthma development.

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