drawn until recently. A 2015 systematic review on CRS and occupational and environmental exposures assessed 41 studies.[1020] There was substantial heterogeneity in the definition of CRS used and reporting of exposures was subject to bias in the form of self-report or industry/job title extrapolation. The authors concluded that limited conclusions can be drawn regarding the role of occupational or environmental exposures in CRS. Further and more recent work has, however, suggested a link between occupational and environmental exposures and CRS.

Additional studies since this review often continue to in adequately define their cohort with accepted diagnostic criteria, while also failing to specifically differentiate ARS from CRS. Further, self-reported outcomes are common, introducing a strong recall bias to these results. Consequently, the conclusions regarding the impact of these exposures and their effect on ARS or CRS should be tempered.

Nevertheless, several cross sectional studies have demonstrated a significant and independent association between environmental and occupational exposure and CRS.[1029–1031]

A cross-sectional study from Denmark showed that female blue-collar workers had higher rates of CRS compared to white-collar workers (adjusted risk ratio 1.64; 95% CI, 1.10-2.43), and that occupational exposures elevated the risks of CRS.[1032] Large cross-sectional studies of individuals in the U.S. and in South Korea identified associations between CRS and air quality, including pollution with particulate matter 10 (PM10).[1033,1034] Recent cross-sectional studies using a symptom-based diagnosis of CRS completed in China in 2016 and in Norway in 2018 determined that factors such as dust, poisonous gas, cleaning agents, animals, mildew and physically strenuous work were associated with CRS.[1029,1030] In general, statistically significant odds ratios for associations between these factors and CRS range from 1.2 to 2.7.[1029,1030,1034] A 2018 case-control study of textile and retail workers incorporating nasal endoscopy to diagnose nasal polyps identified significantly more nasal polyposis ($p = 0.001$), polypoid degeneration of the middle turbinate, ($p = 0.001$) and poorer Lund-Kennedy score (LK, $p < 0.001$) than those not exposed to dust.[1031] A 2015 case-control study demonstrated that higher serum levels of cadmium and nickel were associated with nasal polyposis, however these findings may have been confounded by smoking status.[1035] Research by the same group using atomic absorption spectrometry demonstrated a higher amount of heavy metals, including nickel, chromium, and arsenic, in nasal polyp tissue compared to non-polyp nasal mucosa from the same subjects, though again smoking status may have confounded these results.[1036]

Further study using novel techniques has corroborated that exposures contribute to CRS. Following the World Trade Center attack, dust exposure has been linked to increased prevalence of CRS.[1037] A 2018 investigation employed spatial monitoring techniques to estimate environmental exposures in individuals with confirmed diagnoses of CRSsNP and CRSwNP. The study correlated exposures of particulate matter 2.5 (PM2.5) and black carbon with measures of CRS severity and treatment, such as corticosteroids and ESS.[1038] When exposed to PM, this cohort of patients had a significantly greater likelihood to require ESS and revision ESS in a dose dependent relationship ($p = 0.015$). Additionally, black carbon (BC) was shown to be a significant predictor of SNOT-22 scores in a subgroup of patients that otherwise did not demonstrate sufficient mucosal inflammation to warrant surgery. These data showed that air pollutants correlated with symptom severity and that this may be influenced by exposure levels in patients with CRSsNP.[1038] A subsequent study in 2020 showed that occupational airborne exposures to vapors, gases, dusts, fumes, fibers, and mists correlated with increased rates of ESS and need for corticosteroids in individuals with CRS, while there was no correlation between pollutant levels and disease severity measures.[1039] These 2 studies employed guideline definitions to diagnose CRS in included subjects, strengthening the conclusions that can be drawn from these reports.[1038,1039] Interestingly, occupational exposure to several agents like hypochlorite, dust, cleaning agents and irritants have been associated with negative outcomes after ESS for CRS, as self-reported exposure to multiple irritants increased with the number of revision surgeries.[1040] The mechanisms of action of occupational agents leading to chronic sinonasal inflammation are most likely linked to epithelial barrier dysfunction with/without immune activation of the innate and adaptive immune system.[156] although the level of evidence linking pollution to CRS is limited, the existing literature does suggest that air pollution may play a role in the pathogenesis of CRS.[1041] Indeed, *in vivo* studies in mice have shown that air pollution results in eosinophilic RS in mice, highlighting an area for futher investigation and further lending credence to the theory that environmental pollutants may contribute to the development of CRS.[1042] Also, environmental irritants like hypochlorite in swimming pools have been associated with chronic inflammation and nasal hyperreactivity.

Overall, these data suggest that environmental and occupational exposures contribute to CRS (Tables IX-23 and IX-24). Further studies are needed to refine this association and establish causality. Ultimately, additional studies with larger patient population sizes and control groups, using current diagnostic criteria for ARS or CRS, and objective disease outcome measures (ie, SNOT-22, LM, LK, etc), are needed to establish the association between sinonasal disease and environmental/occupational allergens, while