Persistent rhinitis and epithelial remodeling induced by cyclic ozone exposure in the nasal airways of infant monkeys.

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Abstract Children chronically exposed to high levels of ozone (O(3)), the principal oxidant pollutant in photochemical smog, are more vulnerable to respiratory illness and infections. The specific factors underlying this differential susceptibility are unknown but may be related to air pollutant-induced nasal alterations during postnatal development that impair the normal physiological functions (e.g., filtration and mucociliary clearance) serving to protect the more distal airways from inhaled xenobiotics. In adult animal models, chronic ozone exposure is associated with adaptations leading to a decrease in airway injury. The purpose of our study was to determine whether cyclic ozone exposure induces persistent morphological and biochemical effects on the developing nasal airways of infant monkeys early in life. Infant (180-day-old) rhesus macaques were exposed to 5 consecutive days of O(3) [0.5 parts per million (ppm), 8 h/day; "1-cycle"] or filtered air (FA) or 11 biweekly cycles of O(3) (FA days 1-9; 0.5 ppm, 8 h/day on days 10-14; "11-cycle"). The left nasal passage was processed for light microscopy and morphometric analysis. Mucosal samples from the right nasal passage were processed for GSH, GSSG, ascorbate (AH(2)), and uric acid (UA) concentration. Eleven-cycle O(3) induced persistent rhinitis, squamous metaplasia, and epithelial hyperplasia in the anterior nasal airways of infant monkeys, resulting in a 39% increase in the numeric density of epithelial cells. Eleven-cycle O(3) also induced a 65% increase in GSH concentrations at this site. The persistence of epithelial hyperplasia
was positively correlated with changes in GSH. These results indicate that early life ozone exposure causes persistent nasal epithelial alterations in infant monkeys and provide a potential mechanism for the increased susceptibility to respiratory illness exhibited by children in polluted environments.