Comparative in vitro study of interactions between particles and respiratory surface macrophages, erythrocytes, and epithelial cells of the chicken and the rat

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Abstract

In mammals, surface macrophages (SMs) play a foremost role in protecting the respiratory system by engulfing and destroying inhaled pathogens and harmful particulates. However, in birds, the direct defense role(s) that SMs perform remains ambiguous. Paucity and even lack of SMs have been reported in the avian respiratory system. It has been speculated that the pulmonary defenses in birds are inadequate and that birds are exceptionally susceptible to pulmonary diseases. In an endeavour to resolve the existing controversy, the phagocytic capacities of the respiratory SMs of the domestic fowl and the rat were compared under similar experimental conditions by exposure to polystyrene particles. In cells of equivalent diameters (8.5 μ m in the chicken and 9.0 μ m in the rat) and hence volumes, with the volume density of the engulfed polystyrene particles, i.e. the volume of the particles per unit volume of the cell (SM) of 23% in the chicken and 5% in the rat cells, the avian cells engulfed substantially more particles. Furthermore, the avian SMs phagocytized the particles more efficiently, i.e. at a faster rate. The chicken erythrocytes and the epithelial cells of the airways showed noteworthy phagocytic activity. In contrast to the rat cells that did not, 22% of the chicken erythrocytes phagocytized one to six particles. In birds, the phagocytic efficiencies of the SMs, erythrocytes, and epithelial cells may consolidate pulmonary defense. The assorted cellular defenses may explain how and why scarcity of SMs may not directly lead to a weak pulmonary defense. The perceived susceptibility of birds to respiratory diseases may stem from the human interventions that have included extreme genetic manipulation and intensive management for maximum productivity. The stress involved and the structural–functional disequilibria that have occurred from a ‘directed evolutionary process’, rather than weak immunological and cellular immunity, may explain the alleged vulnerability of the avian gas exchanger to diseases