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MICROGRAVITY INDUCES DIFFERENTIAL RESPONSE TO RHINOVIRUS INFECTION IN LUNG CELLS

Abstract

Purpose: Space travel presents many risks to astronauts including microgravity, and radiation. Another risk to consider in human spaceflight is viral infection susceptibility and response. Respiratory infections have been reported on past space missions, the first during Apollo 7, where one astronaut contracted rhinovirus which due the confined nature of space travel, was quickly transmitted to the remaining crew. Although a quarantine procedure is now in place, infections remain a risk of space travel. It is well known that microgravity induces immune system impairment, however the direct consequences of this impairment on response to infection in a systemic and respiratory context is less well understood. From studies using blood samples of astronauts, and simulated microgravity studies, the altered function of immune cells is documented. However, the immune function of non-immune cells like epithelial cells has not been investigated and these cells are the primary site of infection and defence.

Methodology: Bronchial epithelial cell line BEAS-2B and alveolar epithelial cell line A549 were cultured in T175 flasks. At 80% confluency, the media was replaced with quiescent media. After 24 hours, the cells were trypsinised, counted and seeded into 1.75ml tubes in quiescent media. The tubes were completely filled with media to avoid bubble formation and shear stress. Half of the BEAS-2B and A549 cells were exposed to rhinovirus 16 (RV16), then control and virus exposed cells were attached to a random positioning machine (RPM) to simulate microgravity. The remaining tubes were placed on the base of the RPM to account for any vibrational effects. After 24- and 48-hours supernatant was collected for ELISA.

Results: BEAS-2B and A549 cells were cultured in a scaffold free 3D non-adherent model that resulted in the formation of spheroid-like structures. At a basal state, BEAS-2B cells had a trend towards decreased IL-8 release under microgravity conditions compared to normal gravity, while baseline IL-6 release was unchanged between conditions. RV16 exposed BEAS-2B cells in microgravity had decreased release of IL-8 compared to RV16 gravity controls at 48 hours ($p<0.05$, $n=4$), however there was an increased released of IL-6 in the RV16 microgravity group compared to RV16 gravity control ($p<0.05$, $n=4$).

Conclusions: The results reveal that cells in a microgravity environment have a differential response to viral infection compared to normal gravity as well as differential effects on pro-inflammatory cytokines IL-8 and IL-6. The driving mechanisms of these changes should be investigated further.