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Alterations in Endoplasmic Reticulum and Lysosomal–Mitochondrial Axis in Monocytes after treatment with Different Campylobacter jejuni Lysates

Barbara Canonico, Erica Cesarini, Francesca Luchetti, Gianna Di Sario, Loris Zamai, Raffaella Campana, Wally Baffone and Stefano Papa

Università Degli Studi Di Urbino, Carlo Bo, Disb, Urbino, Italia

Campylobacter jejuni, a Gram-negative spiral-shaped bacterium, often explicates its virulence through the cytolethal distending toxin (CDT). Infection by C. jejuni is commonly associated with human gastroenteritis, however it may also generate the development of the Guillain–Barré Syndrome, an acute peripheral neuropathy. An inflammatory response consequence of CDT-induced cell death is a possible cause of the disease. Monocytes are potent producers of both pro- and anti-inflammatory cytokines, playing a major role in innate immunity and in non-specific host responses. For this reason, we tested the effect of C. jejuni lysates obtained from different strains (expressing wild type or mutated-less functional CTD) on donor monocytes. The alteration induced on monocyte mitochondria and lysosomes were specifically evaluated by flow cytometry and confocal microscopy. Lysates from all strains induced endoplasmic reticulum (ER) stress in monocytes, suggesting that ER stress was not associated with CDT but to other C. jejuni virulence factors. The C. jejuni ISS 1 wild-type strain mostly induced lysosomal alterations, whereas the C. jejuni ATCC 33291 strain induced the most relevant mitochondrial alterations consistent with the induction of an intrinsic apoptotic pathway. Differently, the presence of CDT wild-type produced alteration in lysosomal acidic compartments and p53 down-regulation. In conclusion, the inhibition of p53 expression induced by CDT wild-type, would suggest that CDT, beside its direct cell death effects, is able to promote an apoptotic stimuli-resisting pathway.

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