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PCR array profiling of antiviral genes in human embryonic kidney cells expressing *Human coronavirus OC43* structural and accessory proteins

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Background & Aim: *Human coronavirus OC43* (HCoV-OC43) causes common cold, and is associated with severe respiratory symptoms in infants, elderly and immunocompromised patients. HCoV-OC43 is a member of Betacoronavirus genus that includes also the severe acute respiratory syndrome (SARS) and the Middle East Respiratory Syndrome (MERS) coronaviruses. Both SARS-CoV and MERS-CoV were shown to express proteins with the potential to evade early innate immune responses. However, the ability of HCoV-OC43 to antagonise the intracellular antiviral defenses has not yet been investigated. The objective of this study was to investigate the role of HCoV-OC43 structural (membrane and nucleocapsid) and accessory (ns5a and ns2a) proteins in the modulation of antiviral gene expression profile in human embryonic kidney 293 (HEK-293) cells using PCR array analysis.

Methods: HCoV-OC43 membrane (M), nucleocapsid (N), ns5a and ns2a mRNA were amplified and cloned into the pAcGFP1-N expression vector (Clontech), followed by transfection in HEK-293 cells. Expressions of M, N, ns5a and ns2a proteins were confirmed by indirect immunofluorescence test. Three days post-transfection, the cells were challenged by Sendai virus. The human antiviral response PCR array system (Qiagen) was used to profile the antiviral gene expression in HEK-293 cells, using the fold regulation comparison and the manual normalization methods.

Results: Around 50-60 genes were down-regulated by HCoV-OC43 proteins, the most prominent genes being those critical for the activation of transcription factors involved in the antiviral response like interferon regulatory factors (IRFs) and activator protein 1 (AP-1). Among the most important down-regulated genes were those coding for interferons (IFNs) mitogen-activated protein kinases (MAPKs), pro-apoptotic and pyroptotic proteins (caspases, cathepsins, and tumor necrosis factor), pro-inflammatory cytokines (interleukins), pattern recognition receptors (PRRs; toll-like receptors and NOD-like receptors) and their signaling transduction proteins (TICAM1, MAVS).

Conclusion: This study shows for the first time that similarly to SARS-CoV and MERS-CoV, HCoV-OC43 has the ability to down-regulate the transcription of genes critical for the activation of different antiviral signaling pathways.

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