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Activation of TNF-α from Human Endothelial Cells by Exposure of the Mitochondrial Stress Protein (Hsp60) Secreted from THP-1 Monocytes to High Glucose

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Abstract : Inflammation of the endothelium is an important process leading to diabetic atherosclerosis. However, the molecular mechanisms by which diabetes contributes to endothelial inflammation remain to be established. Using In-vitro cultured Human cells and Hsp60 specific ELISA assays, we show that Hsp60 is not only induced in Human monocyte cells under hyperglycaemic conditions but that the Hsp60 is also secreted from these cells. Furthermore, we also demonstrate that the Hsp60 secreted from these monocyte cells is also able to activate Toll-like receptor-4 (TLR4) from Human endothelial cells. This suggests that a potential link may exist between the hyperglycaemia-induced expression of Hsp60 in monocyte cells and vascular inflammation. Circulating levels of Hsp60 due to mitochondrial stress in diabetes patients could, therefore, be an important modulator of inflammation in endothelial cells and thus contribute to the increased incidences of atherosclerosis in diabetes mellitus.

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