Aphidicolin-induced stress pathway in pre-implantation embryos

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The cell cycle is a ubiquitous and complex process that is essential for the proper growth and development of the pre-implantation embryo. There has been increasing evidence that correlates the cell cycle with the activity of ion channels, in particular potassium channels. In a previous study we have shown that aphidicolin-induced G1 cell cycle arrest of pre-implantation embryos results in the constitutive activation of a cell cycle-related potassium channel (Day *et al.*, 1998). The present study was aimed at identifying the various signalling pathways activated upon the administration of aphidicolin using flow cytometry and microarrays and from there, decipher the link between these pathways and potassium channel activity.

Results suggest that aphidicolin-induced cell cycle arrest was due to stimulation of the stress-activated kinase pathway (SAPK) that proceeds via p38MAP kinase. This provides a potential link between the mitogen-activated kinase (MAPK) pathways and the activity of the cell cycle-related potassium channels present in embryos.

Day, M.L., Johnson, M.H. & Cook, D.I. (1998) *EMBO Journal*, **17**: 1952-1960.