16-kDa Prolactin Inhibits Endothelial Cell Migration by Downregulating the Ras-Tiam1-Rac1-Pak1 Signaling Pathway

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SUPPLEMENTARY FIGURE LEGEND

Supplementary Fig. S1. 16k PRL attenuates primary human endothelial cell migration.

Primary human umbilical vein endothelial cells (HUVEC) were incubated in media containing 10% FBS and grown to 80–90% confluence in a 12-well plate. HUVEC monolayers were scratched (dotted line) with a 1 ml pipet tip and cell migration at 2, 6 and 24 hours after wound scratch was recorded by phase contrast microscopy. Control HUVEC (panel a) first began migration at about 6 hours after wound scratch and reached 100% sealing 24 hours after wound scratch. 16k PRL treated HUVEC (panel b) also migrated into the wound area, but at a lowered rate, reaching about 80% sealing 24 hours after wound scratch. The scratched area into which endothelial cells have migrated after 24 hours is enlarged for a better view of the level of wound sealing. Similar results were obtained in a second independent experiment. Together, these results suggest that 16k PRL attenuated wound-induced cell migration in primary endothelial cells.