In this study, we investigated the inhibitory mechanisms of resorcinol in B16F10 mouse melanoma cells. We found that resorcinol reduced both the melanin content and tyrosinase activity in these cells. In addition, resorcinol suppressed the expression of melanogenic gene microphthalmia-associated transcriptional factor (*MITF*) and its downstream target genes tyrosinase, tyrosinase-related protein (*TRP*)-*1*, and *TRP-2*. In addition, we found that resorcinol reduced intracellular cAMP levels and protein kinase A (PKA) activity, and increased phosphorylation of the p38 mitogen-activated protein kinase (MAPK). Resorcinol was also found to directly inhibit tyrosinase activity. However, resorcinol-induced decrease in melanin content, tyrosinase activity, and tyrosinase protein levels were attenuated by SB203580, a p38 MAPK inhibitor. Taken together, these data indicate that anti-melanogenic activity of resorcinol is be mediated through the inhibition of cAMP signaling and activation of p38 MAPK, indicating that resorcinol may be a possible ameliorating agent in the treatment of hyperpigmentation skin disorders.

Schematic diagram of resorcinol function in melanogenesis.