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Developmental Arrest of Mechanosensory Bristles in the *Drosophila* Temperature-Sensitive Mutant, *shibire*

Seokjoo Kim* and Yun-Taik Kim

Department of Life Science, Sogang University, Seoul 121-742

The X-linked temperature-sensitive recessive mutant of *Drosophila, shibire^{ts1} (shi^{ts1})* paralyzes at 30°C but behaves normally at 22°C. Heat pulses of 30°C during pupal development in *shi^{ts1}* flies induce abnormal epidermis without macrochaete bristles. We examined a temperature-sensitive defect in development of mechanosensory bristles in head and thorax when exposed to brief heat pulses at various times in development. White prepupae were collected and raised at 17°C to allow a more accurate developmental staging. Vials of pupae were exposed to 30°C for 4 hrs at successive 4-hr intervals thoughout puparium stage. All the macrochaetes on the head and thoraces were examined after eclosion. We found that all the bristles have a specific temperature sensitive period (TSP) during development. Heat pulses during TSP caused a failure of normal bristle development. Specifically, bristles in head and notum were affected by high temperature during 30-50 hr after puparium formation (apf). The effects were greatly reduced on most macrochaetes over 50 hr apf and no effect was found over 68 hr apf. Control tests of wild type flies showed no defect in the development of bristles. Since the macrochaete and the underlying sensory cells are derived from a common progenitor cell, our results suggest a developmental time windows for sensitivity of each bristle and their sensory cells to *shi* mutation.

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Involvement of Glucocorticoid Receptor in the Induction of Differentiation by Ginsenosides in F9 Teratocarcinoma Cells

Youl-Nam Lee[1*], Hae-Young Chung[2], Shin-Il Kim[3], and Kyu-Won Kim[1]

[1]Department of Molecular Biology, [2]Department of Pharmacy, Pusan National University, Pusan, 609-735, [3]Korea Ginseng and Tobacco Research Institute, Daejeon, 305-345, Korea.

Ginsenosides Rh1 and Rh2 induced differentiation in cultured F9 teratocarcinoma stem cells. To elucidate the mechanism of differentiation-inducing effects of Rh1 and Rh2, we examined the possible involvement of glucocorticoid receptor (GR) in the differentiation effects of these agents because of their structural similarity with glucocorticoids. Treatment of F9 cells with Rh1 or Rh2 induced nuclear translocation of GR as did dexamethasone, a synthetic glucocorticoid. Furthermore, using gel shift assay, we determined a binding protein in F9 cells induced by Rh1 or Rh2 using glucocorticoid response element (GRE). Protein complex was detected in the nuclear extracts of F9 cells and increased in Rh1 or Rh2 treated F9 cells. To confirm whether this protein complex is a GR, we performed the competition assay with unlabled GRE as a specific competitor. In addition, supershift assay using a GR antibody showed supershift bands in Rh1 or Rh2-treated F9 cells. Moreover, overexpression of GR by cotransfection of GR expression vector and GRE-luciferase vector enhanced the transactivation activity of GRE promoter in the presence of Rh1 or Rh2 and further confirmed by examining the inhibitory effect of RU486, a glucocorticoid antagonist with a high affinity for the GR. These results suggest that the differentiation-inducing action of ginsenosides Rh1 and Rh2 in F9 cells is due to the interaction with a GR.