Valproic acid inhibits the invasion of PC3 prostate cancer cells by upregulating the metastasis suppressor protein NDRG1.

Lee JE, Kim JH.

Abstract

Valproic acid (VPA) is a clinically available histone deacetylase inhibitor with promising anticancer attributes. Recent studies have demonstrated the anticancer effects of VPA on prostate cancer cells. However, little is known about the differential effects of VPA between metastatic and non-metastatic prostate cancer cells and the relationship between the expression of metastasis suppressor proteins and VPA. In the present study, we demonstrate that inhibition of cell viability and invasion by VPA was more effective in the metastatic prostate cancer cell line PC3 than in the tumorigenic but non-metastatic prostate cell line, RWPE2. Further, we identified that the metastasis suppressor NDRG1 is upregulated in PC3 by VPA treatment. In contrast, NDRG1 was not increased in RWPE2 cells. Also, the suppressed invasion of PC3 cells by VPA treatment was relieved by NDRG1 knockdown. Taken together, we suggest that the anticancer effect of VPA on prostate cancer cells is, in part, mediated through upregulation of NDRG1. We also conclude that VPA has differential effects on the metastasis suppressor gene and invasion ability between non-metastatic and metastatic prostate cancer cells.

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