Invasion mechanism in human melanoma cells

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To the editor,

We are glad to congratulate Ciolczyk-Wierzbicka for their article [1] in which they observed the possible role of N-cadherin and selected downstream protein kinases: PI3K, ERK1/2, and mTOR in cell invasion in four human malignant melanomas (WM793, Lu1205, WM115, and WM266-4). After separate use or combination treatment of siRNA for N-cadherin and downstream protein kinases inhibitors, all tested melanoma cell lines showed reduction in invasion via conventional Boyden transwell methods and activity decline of MMP-2 and MMP-9 by western blot and gelatin zymography compared to control cells. Finally, the mechanism of invasion through N-cadherin/FGFR interaction, PI3K signaling, and MEK-ERK pathway was discussed.

In Ciolczyk-Wierzbicka et al.'s study, they cited suggestion of Hazan et al. [2] which was the 21st reference that N-cadherin functionally interacted with FGF receptor, causing sustained downstream signaling by PI3K, and through MEK-ERK promotes cell survival, migration, and invasion. However, in Hazan's article, PI3K was not mentioned at all. Moreover, U0126 was mistakenly written into U126 in all of figures and someplace of text part. U0126 is a ERK 1/2 kinase inhibitor via blockade MEK1/2 kinase activity [3]. It

is likely to cause confusion when read as U126. These issues should be amended in the current study.

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Compliance with Ethical Standards

Conflict of interest The authors declare no conflict interest.

Ethical approval There are no human participants and/or animals contained in this paper.

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