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LETTER TO THE EDITOR

CERK inhibition might be a good potential therapeutic target for diseases

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We read with great interest the article by Pastukhov *et al.* (2014a) which reported that NVP-231, the inhibitor of ceramide kinase (CERK), could reduce cell viability, DNA synthesis, colony formation and induced apoptosis of the breast and lung cancer cell lines MCF-7 and NCI-H358. The results suggested that CERK inhibition might be a good potential therapeutic target for breast and lung cancer.

Moreover, Pastukhov *et al.* (2014b) had demonstrated that proliferation was reduced via CERK inhibition in renal mesangial cells and fibroblasts. It suggested that CERK inhibition was a potential target for treating mesangioproliferative kidney diseases. Furthermore, Payne *et al.* (2014) had indicated that CERK was required for tumour recurrence and survival and CERK was up-regulated in tumour cells and during tumour recurrence in mouse breast cancer. Furthermore, gene expression profiles showed that up-regulated CERK was related to an increased risk of recurrence in women for breast cancer. These results suggested that CERK played a vital role in breast cancer recurrence and CERK inhibition might be a potential target for tumour recurrence. In addition, Bini *et al.* (2012) had showed that CERK inhibition reduced cell proliferation in human neuroblastoma cells.

Together, these findings indicated that CERK played a functional role in diseases. We read with considerable interest and thought that CERK inhibition might be a good potential therapeutic target for diseases, including cancer, mesangioproliferative kidney diseases and neuroblastoma cells.

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Conflict of interest

The authors declare no conflicts of interest to disclose.

References

Bini F, Frati A, Garcia-Gil M, Battistini C, Granado M, Martinesi M *et al.* (2012). New signalling pathway involved in the anti-proliferative action of vitamin D_3 and its analogues in human neuroblastoma cells. A role for ceramide kinase. Neuropharmacology 63: 524–537.

Pastukhov O, Schwalm S, Zangemeister-Wittke U, Fabbro D, Bornancin F, Japtok L *et al.* (2014a). The ceramide kinase inhibitor NVP-231 inhibits breast and lung cancer cell proliferation by inducing M phase arrest and subsequent cell death. Br J Pharmacol 171: 5829–5844.

Pastukhov O, Schwalm S, Römer I, Zangemeister-Wittke U, Pfeilschifter J, Huwiler A (2014b). Ceramide kinase contributes to proliferation but not to prostaglandin E2 formation in renal mesangial cells and fibroblasts. Cell Physiol Biochem 34: 119–133.

Payne AW, Pant DK, Pan TC, Chodosh LA (2014). Ceramide kinase promotes tumor cell survival and mammary tumor recurrence. Cancer Res 74: 1–12.