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Corrigendum

Corrigendum to "GDC-0449—A potent inhibitor of the hedgehog pathway" [Bioorg. Med. Chem. Lett. 19 (2009) 5576]

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Page 1— *lines 59–61 should read*: There is also strong evidence linking mutations in the Hh pathway to sporadic BCC,[5,6] and medulloblastoma[7,8]. In addition, a larger series of cancers has been associated with ligand activation of the Hh pathway including tumors of the gastro-intestinal tract[21], among others.[9,10]

Page 1— *lines 62–67 should read*: The Hh pathway has been studied extensively using the natural product cyclopamine, a steroidal alkaloid that blocks Hh signaling with an EC_{50} of approximately 300 nM and has been shown to bind SMO,[11] to modify Hh pathway activation in explants of embryonic neural tubes,[12] to inhibit the growth of human medulloblastoma allografts,[8] and basal cell carcinoma skin explants.[13]

Page 5— *lines 220–225 should read*: Compound **31** was explored further in a Hh pathway dependent medulloblastoma allograft model generated from Ptch+/−mice[22,25] and produced complete regression at doses as low as 12.5 mg/kg BID (Fig. 3). These data are consistent with the potency previously reported for another HPI in a similar model of medulloblastoma.[26] In addition to the mutation-driven autocrine signaling medulloblastoma model, Hh antagonists have been shown to delay growth in primary human tumor xenograft models.[21]

Additional references

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