Author's response to reviews

Title: Pyrrolo[1,2-B][1,2,5]Benzothiadiazepines (PBTDS) induces apoptosis in K562 cells

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The BCR-ABL fusion gene, deriving from the reciprocal translocation between chromosomes 9 and 22, is responsible for the myeloid cell expansion and apoptosis inhibition in CML. The resulting cytoplasmic p210 BCR-ABL protein is an activated form of the abl tyrosine kinase protein. Abnormal protein tyrosine kinases are responsible for several other human leukemias. The ABL inhibitor Imatinib (Gleevec) showed remarkable efficacy in the clinical management of CML. However, resistance to kinase inhibitors (PTK-Is) is a major emerging problem that may limit long-term therapeutic efficacy. Thus novel effective drugs are urgently needed. We found that pyrrolo[1,2-b][1,2,5]benzothiadiazepines (PBTDS) are able to induce apoptosis in a concentration and time dependent manner in K62 cell. These findings suggest that PBTDS are promising candidates as novel therapeutic agents for BCR-ABL-positive leukaemias.