Author’s response to reviews

Title: Inhibition of STAT3-Interacting Protein 1 (STATIP1) promotes STAT3 transcriptional up-regulation and imatinib mesylate resistance in the chronic myeloid leukemia

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Author’s response to reviews: see over
To Dra. Dafne Solera, Executive Editor of “BMC Cancer Journal”,

Dear Dra Solera.

Please find enclosed our manuscript, titled “Inhibition of STAT3-Interacting Protein 1 (STATIP1) promotes STAT3 transcriptional up-regulation and imatinib mesylate resistance in the chronic myeloid leukemia”, for consideration for publication in BMC Cancer Journal.

This manuscript describes a novel form of STAT3 transcriptional regulation in chronic myeloid leukemia (CML). Herein, we characterized the STATIP1 protein in K562 cells and its ability to regulate the transcriptional activity of STAT3, which has been widely implicated in the development of cancer, particularly leukemias. Furthermore, we also showed that STATIP1 is down-expressed in Lucena, an imatinib mesylate (IM)-resistant cell line and also STATIP1 and STAT3 are inversely expressed in CML patients (IM-responsive and IM-resistant). STATIP1 inhibition could confer resistance IM in the K562 cell line. Our data suggest that STATIP1 is a negative regulator of STAT3 and demonstrate a novel mechanism in which a STAT3-interacting protein is involved in IM resistance. Thus, we reveal a new cell signaling pathway mechanism related to leukemia biology that could improve our understanding of outgrowth in other cancers.

The authors declare that they have no competing interests. The manuscript was drafted according to the “instructions for authors” guide available at the journal’s homepage. The text of this manuscript was professionally edited by an American Journal Experts editor (www.journalexperts.com), the certificate for which is attached as a supplemental data file.

Thank you for considering this manuscript for publication.

Best regards,

Dr. André Mencalha.