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Correction

Correction: de Almeida et al. Acute Promyelocytic Leukemia (APL): A Review of the Classic and Emerging Target Therapies towards Molecular Heterogeneity. *Future Pharmacol.* 2023, *3*, 162–179

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Figure Legend

In the original publication [1], there was a mistake in the legend for Figure 7.

The description of the figure is "Gilteritinib and midostaurin mechanism through *FLT3* kinase inhibition". The correct legend appears below. The authors state that the scientific conclusions are unaffected. This correction was approved by the Academic Editor. The original publication has also been updated.

Figure 7. Venetoclax mechanism through BCL-2 inhibition. BCL-2 associated protein X (BAX) is a pro-apoptotic protein that is recruited by Venetoclax. In the presence of an apoptotic signal, BAX is translocated from the cytoplasm to the vicinity of the mitochondria, where it undergoes activation and conformational modification before adhering to the outer mitochondrial membrane. Small units of activated BAX proteins form oligomers that eventually penetrate the outer mitochondrial membrane and release cytochrome c, which activates the cell cascade to apoptosis via caspases (adapted from Kucukyurt and Eskazan, [37]).

Reference

1. de Almeida, T.D.; Evangelista, F.C.G.; Sabino, A.d.P. Acute Promyelocytic Leukemia (APL): A Review of the Classic and Emerging Target Therapies towards Molecular Heterogeneity. *Future Pharmacol.* **2023**, *3*, 162–179. [CrossRef]

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