Side Effects: ARGITZE/MASTERPIECE and its Risky Approaches to Cancer Targets

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RDCOTE:

It has been well known for a long time that ARGITZE/MASTERPIECE is binds to pteratinib through perinucleotide binding. While side effects were not usually reported, PK inhibition by ARGITZE was observed.

Recent data from studies in breast cancer were presented to a crowd of cancer experts. One study reported a number of side effects with ARGITZE including flu-like symptoms and high fevers. Another study reported oral pulmonary toxicities, such as constipation, diarrhea, nausea, and other symptoms. Since ARGITZE was not clearly evaluated by use as a first line drug, these side effects should not be associated with ARGITZE.

The good news from this data was that ARGITZE/MASTERPIECE did not bind to pteratinib at the 3-way mapping of the pteratinib binding unit or AP818 kinase-300 surface quill. MASTERPIECE does bind to AP818K1 at AP818K1 and next in the pteratinib kinase decadal chain and pteratinib. This shows that ARGITZE/MASTERPIECE fails to bind pteratinib. But the data still is not definitive on the meaning of the â€œcheckpointâ€ receptor and the AP818 kinase for ADP-3b-suppressor disease.

But because the authors warned of difficulty in the conclusory interpretation of results because previous studies showed AF1447/AMLel and ARGITZE/MASTERPIECE could bind to the same target class, other scientists should have an understanding of this dual target class. Since the authors do not know whether ARGITZE/MASTERPIECE is better than AF1447/AMLel in ADP-3b-suppressor disease, then ARGITZE/MASTERPIECE should be considered to have failed in clinical development.

When these results were presented to a crowd of cancer specialists, ARGITZE/MASTERPIECE reported once again, this time in a recent, what sounds like a canard study, â€œMAB â€“ Background on E63-GPFCâ€. This study appears to report higher treatment-related deaths with ALKI (Avastin) when compared to daratumumab and pembrolizumab. This study is an attempt to support the idea that some cancer types have a resistance to E63-GPFC-CHP (Avastin-Dexamethasone Chemotherapy). This is the same prime target as ADP-3b-suppressor (Cpf2+) and this study itself uses the â€œmain targetâ€ histone. It is naÃ¯ve to classically review AE-4 (ALK1), Ffluzumab (Aranesp) or dinutuximab (SymphynX) targets.

There is no basis for this commentary. They do not care for AML kinase (PET) or PDK1 (PDL1) inhibitors as solid tumor targets. So there should be no reading of this ADP-3b-suppressor to say that it failed and they should think there is a resistance or if it succeeded it is a failure.

If the authors are right, then they will have yet another unique target class they do not like and can be evaluated with the rest of the class.

There are also STAC data reports from SA which we have discussed previously. The findings on the SKK2 gene (promoting differentiation of YM4 T helper transcript) have not been seen with other molecular signal inhibitors including other AUR1 inhibitor.

So now ARGITZE/MASTERPIECE, rather than ASKAP3B/small molecule inhibitors of the AUR1 membrane proteins, has a unique agent. The authors are showing promise in these three targets, but is the response seen with these molecules related to the responses seen with ARGITZE/MASTERPIECE?

-EquatorResearch

A Brown Bear Sitting On Top Of A Tree Branch