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IN THE NEWS

FROM TAT 2015

At the 13th International Congress on Targeted Anticancer Therapies (TAT), held in Paris this year, a wide range of preclinical and early phase I data were presented. In the session on new frontiers in immunotherapy, the importance of PD-1 expression on both immune cells and tumour cells was highlighted. Rationale combinations based on tumour biology should help to address why some patient's immune responses do not eradicate tumours. With the impressive results seen in patients with multiple solid tumours and, more recently, in haematological cancers, this session highlighted that immunotherapy combinations are likely to provide the next treatment breakthroughs.

The use of RECIST criteria is associated with many limitations, but alternative ways to assess tumour response, such as radiological approaches that are based on tumour metabolic behaviour, necrosis, and volumetric change, provide better predictive and prognostic information that may help improve patient selection. Progress in noninvasive radiogenomic analysis of wild-type and mutated tumours has provided the capacity to predict patient response to targeted therapies, which proved particularly useful for gliomas.

Another emerging area of increasing importance in clinical oncology is targeting tumour metabolism. A phase I study of AG-120, a first-in-class, selective inhibitor of mutated isocitrate dehydrogenase-1 (IDH1) in patients with a variety of advanced-stage haematological malignancies, which included relapsed or refractory acute myeloid leukaemia (AML), showed that this agent was well tolerated, with >25% of patients having complete remission. Extending discussion of early phase data to agents that target epigenetic mechanisms, the BET-bromodomain (BRD) inhibitor, OTX-015, was assessed in patients with AML and other haematological cancers, and had clinical activity and was well tolerated.

Regarding molecularly targeted therapies, rociletinib—an oral, irreversible inhibitor of mutant EGFR, including T790M EGFR—that delayed development of resistance in mouse models resistant to erlotinib treatment, provided durable clinical benefit in patients with advanced-stage *EGFR*-mutant non-small-cell lung cancer who had acquired resistance to first-generation and second-generation EGFR inhibitors. No cutaneous toxicity and very few grade 3–4 adverse effects were seen, illustrating the promise of these new-generation inhibitors, which are now being tested in trials of combination therapies.

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