NEWS IN BRIEF

FDA approvals for the first 6 months of 2016

The FDA approved 13 new drugs in the first 6 months of this year (TABLE 1). These Center for Drug Evaluation and Research (CDER) approvals consisted of 8 small molecules and 5 biologics.

FDA approvals tend to be back-loaded to the second half of the year. Last year, the FDA approved 14 new drugs in the first half of the year and 31 in the second half, for a total of 45 approvals (*Nat. Rev. Drug Discov.* **15**, 73–76; 2016).

The agency also approved one biosimilar. Celltrion's infliximab-dyyb became the first FDA-approved biosimilar of a monoclonal antibody.

Asher Mullard

Table 1 | **FDA approvals in the first 6 months of 2016**

Drug (trade name)	Lead company	Indication
Brivaracetam (Briviact)	UCB	Partial-onset seizures
Obiltoxaximab (Anthim)*	Elusys Therapeutics	Anthrax
Ixekizumab (Taltz)*	Eli Lilly	Plaque psoriasis
Reslizumab (Cinqair)*	Teva	Severe asthma
Defibrotide sodium (Defitelio)	Gentium	Hepatic veno-occlusive disease
Venetoclax (Venclexta)	AbbVie	Chronic lymphocytic leukaemia
Pimavanserin tartrate (Nuplazid)	Acadia Pharmaceuticals	Hallucinations and delusions associated with Parkinson's disease psychosis
Atezolizumab (Tecentriq)*	Genentech	Urothelial carcinoma
Obeticholic acid (Ocaliva)	Intercept	Primary biliary cholangitis
[^{18}F]fluciclovine (Axumin)	Blue Earth Diagnostics	Suspected prostate cancer recurrence
Daclizumab (Zinbryta)*#	Biogen	Multiple sclerosis
Gallium Ga 68 dotatate (Netspot)	Advanced Accelerator Applications	Neuroendocrine tumours
Sofosbuvir and velpatasvir (Epclusa)	Gilead Sciences	Hepatitis C virus genotypes 1–6

*Biologics. #The FDA first approved daclizumab in 1997 for kidney transplant rejection, but its then-sponsor Roche withdrew the drug in 2009 citing declining demand.

PARPs pick-up

Tesaro's poly(ADP-ribose) polymerase (PARP) inhibitor niraparib hit its primary end point in its first Phase III trial, lifting hopes for a class of anticancer drugs with a turbulent history.

Companies first started working on PARP inhibitors in the 1990s. Because PARP proteins facilitate the repair of DNA damage, researchers theorized that PARP inhibitors would lead to accumulation of DNA damage and therefore kill highly mutated cancer cells. The field stalled in 2011 with the Phase III failure of Sanofi's iniparib in triple-negative breast cancer, but made a comeback following the discovery that iniparib was not a bona fide PARP inhibitor (*Nat. Rev. Drug Discov.* **12**, 725–727; 2013).

AstraZeneca secured a first approval for the drug class in 2014 with olaparib, but the newly released data have renewed interest in follow-on candidates.

In the Phase III trial of niraparib in patients with platinum-sensitive ovarian cancer, median progression-free survival for patients with germline BRCA mutations who received niraparib was 21 months compared with 5.5 months in the control arm. Although it is difficult to compare the results of two different trials — which can include patients with different baseline characteristics — investors interpreted these data as a win for Tesaro. In AstraZeneca's 2011 Phase II study of olaparib, median progression-free survival of patients with germline BRCA mutations was 11.2 months, compared with 4.1 months in the control arm.

Tesaro will present full data at the European Society for Medical Oncology (ESMO) congress in October. It also plans to file for regulatory approval in both the United States and the European Union in the fourth quarter of 2016.

Other PARP inhibitors in development include Clovis's rucaparib, which the company filed for FDA approval in February, Medivation's talazoparib, which is in Phase III trials for breast cancer and AbbVie's veliparib, which is in Phase III trials for breast cancer and non-small cell lung cancer.

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Cancer model consortium debuts

Although cancer researchers have made important discoveries with currently available cancer cell lines, several shortfalls limit the utility of these models. Cell lines don't exist for all cancer types, and don't capture the full genetic diversity of any cancer type. Two-dimensional models don't necessarily reflect the biology of three-dimensional tumours. And a lack of clinical data about how cancer-cell donors responded to treatment complicates interpretation of preclinical results.

To address these deficits, four US and European organizations are launching the Human Cancer Model Initiative (HCMI) to generate 1,000 new cancer models.

"This new project is timed perfectly to take advantage of the latest cell culture and genomic sequencing techniques to create models that are representative of patient tumours and are annotated with genomic and clinical information," said Louis Staudt, director of the US National Cancer Institute (NCI)'s Center for Cancer Genomics, in a press statement. Contributing scientists will also use reprogrammed stem cells and organoids — 3D organ-like cell structures — to generate better models of disease. "This effort is a first step toward learning how to use these tools to design individualized treatments," he said.

The NCI, the Sanger Institute in Cambridge, UK, Cancer Research UK and the Hubrecht Institute in Utrecht, the Netherlands, are funding and contributing to the HCMI.

The initiative will generate models for common cancers as well as for rare and childhood cancers. Cancer models, and associated data sets and annotations, will be shared with the research community.

If all goes to plan, the HCMI could eventually generate thousands more models as well. Researchers may ultimately need about 10,000 models to fully capture the diversity of relatively common genetic subtypes of cancer, Staudt told *Nature News*.

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