Each quarter, the editorial board selects an area to highlight from the broad range of topics that fall under the umbrella of chemistry in cancer research. This quarter, we are focusing on new opportunities for kinase inhibitors. CICR Editorial Board members, Drs. Kevin Kuntz and Martin Swarbrick have taken the lead in assembling an overview of the topic.

**Kevin W. Kuntz, PhD**
Vice President, Molecular Discovery
Ribon Therapeutics
Lexington, Massachusetts

**Martin E. Swarbrick, PhD**
Senior Group Leader, Discovery Chemistry
Cancer Research UK Therapeutic Discovery Laboratories
Cambridge, United Kingdom
Kinase inhibitors for the treatment of cancer

It has been almost two decades since the first kinase inhibitor, imatinib, was approved for the treatment of cancer. This was a remarkable achievement – until this first proof of concept, kinases were often thought of as a challenging target for drug discovery, particularly due to the similarity of the ATP pocket shared by all kinases leading to potential problems with discovering selective inhibitors. However, since that first approval of imatinib in 2001, the veritable floodgates have opened; now more than 40 kinase inhibitors have been approved as cancer treatments. These compounds target about 20 different kinases, with receptor tyrosine kinases being the most frequently targeted class.

Against the backdrop of such a large volume of prior work, many excellent reviews of the field are extant. The interested reader is turned to a few recent reviews of kinase inhibitors, including "Kinase-targeted cancer therapies: progress, challenges and future directions" by Khushwant and Bhullar and "New Perspectives, Opportunities, and Challenges in Exploring the Human Protein Kinome" by Wilson and colleagues. In addition, see our research highlights in this issue for an article that contains an excellent summary of the targets and properties of the FDA-approved kinase inhibitors.

While these drugs already represent significant steps forward in cancer treatment, more than 100 new kinase inhibitors are currently in clinical trials, with many targeting kinases for which there is not yet an approved drug (see a curated list of kinase inhibitors in clinical trials).

As shown by the work of Dr. Chandra Miduturu, the Early-career Researcher profiled in this issue, FGFR4 is one of these newer kinase targets, with fisogatinib being explored in FGFR19-overexpressing hepatocellular carcinoma. Blueprint Medicines is not the only company targeting FGFR4; Novartis has FGF401, H3 Biosciences has H3B-6527, and Incyte has INCB062079, all of which are reported to be selective FGFR4 inhibitors in clinical testing. Other FGFR kinases also are being targeted by novel inhibitors. Earlier this year, erdafitinib, a pan FGFR inhibitor from Janssen, was approved for metastatic urothelial carcinoma harboring FGFR3 or FGFR2 genetic alterations. Several other companies are also developing pan-FGFR inhibitors, including futibatinib from Taiho, infgratinib from QED Therapeutics, pemigatinib from Incyte, and rogaratinib from Bayer. Clearly, the FGFR kinase family represents a target class that remains of great interest in cancer research.

Newer generation kinase inhibitors are also being developed in cancers for which resistance has evolved to an earlier kinase inhibitor. For example, avapritinib is being tested in metastatic gastrointestinal stromal tumors (GIST) which have become resistant to imatinib and other approved kinase inhibitors. Imatinib is thought to work in GIST by inhibiting the KIT or PDGFR kinases and not through inhibition its original target of BCR-ABL. Avapritinib is selective for KIT and PDGFRA mutant kinases and does not have activity against BCR-ABL. Indeed, new inhibitors overcoming resistance to earlier inhibitors has been a fruitful area in the field of kinase inhibitors and is likely to continue to be an active area of future research.

Cyclin-dependent kinase (CDK) inhibitors are a class that has also shown promise in treating cancers. Several CDK4/6 inhibitors have recently been shown to be useful in treating breast cancer. Other CDKs are also attracting interest for treating other types of cancer. For example, Syros has recently opened a trial for SY-1365, a selective CDK7 inhibitor. CDK7 is thought to be important in cancer due to its role in cell-cycle progression. Also, Tolero Pharmaceuticals is testing a selective CDK9 inhibitor, alvocidib, for the treatment of myelodysplastic syndromes. Dinaciclib is reported to be
a pan-CDK inhibitor and is being studied by Merck in several cancers. It has an interesting pyridine N-oxide as part of the structure – one only a few N-oxides in a clinical stage kinase inhibitor (and possibly the only one!).

Harkening back to some of the earliest concerns around the structural similarity across kinase family members, the challenge of identifying and optimizing ever more selective small molecule inhibitors of specific kinases continues to be a focus of drug discovery in this field. As pointed out above with imatinib working in GIST via inhibition of KIT and PDGFR, co-targeting kinases can present an opportunity for additional patient benefit. However, selectivity can also be a toxicity risk, of course, and can even cloud the interpretation of results. Although not exclusively focused on kinase inhibitors, an important recent article by Lin et al made headlines, sounding a cautionary note around the contribution of off-target effects to the mechanism of action of some cancer drugs undergoing clinical trials. Using CRISPR-Cas9 mutagenesis, the authors found that effects on cell survival were unaffected by the loss of the supposed target for this set of compounds. One of the compounds, OTS964, developed as an inhibitor of PBK, was found to be a potent inhibitor of another cyclin-dependent kinase CDK11, which may explain the observed effects.

It is clear from the past successful development of kinase inhibitors for the treatment of cancer and the current active development of new inhibitors, that the kinase field will be an important area for cancer treatment for years to come.

Join the new AACR-CICR LinkedIn Network Group

We look forward to your participation in our conversations that are designed to enhance your CICR initiative. Join the group.

View the CICR Newsletter Archives / the CICR Early-career Researcher Profiles Archives

For past issues of the CICR Newsletter, visit its Archives. We are also pleased to provide past issues’ Profiles of Early-career Researchers in its own Archives.
BOS172722 is a highly potent, selective and orally bioavailable MPS1 inhibitor currently in the clinic. BOS172722 treatment in combination with paclitaxel induces gross chromosomal segregation defects caused by MPS1 inhibitor-mediated abrogation of the mitotic delay induced by paclitaxel treatment. MPS1 inhibition led to a significant sensitisation of TNBC cells to death and regression of patient-derived xenografts. The most important discovery in this paper from Anderhub et al. is that MPS1 inhibition induces significant cell killing, particularly in highly proliferative cancers. They anticipate that use of proliferation markers for patient selection may be beneficial for the success of MPS1 inhibitors in clinic.
News from the CICR Steering Committee

Contributed by CICR Chair, Andrew J. Phillips, PhD
President and Chief Executive Officer
C4 Therapeutics
Watertown, Massachusetts

Congratulations to Dr. Joachim Rudolph, CICR Chairperson-elect 2020-2021!

Best wishes to Dr. Joachim Rudolph who was recently elected as CICR Chairperson-elect 2020-2021! We appreciate his and his fellow candidate, Dr. Zoran Rankovic's, availability to serve the CICR Working Group in this way. We look forward to Dr. Rudolph's leadership on the Steering Committee and to the future success of all CICR Working Group initiatives. Plan to meet Dr. Rudolph during the CICR Town Hall Meeting on Sunday, April 26, 2020, 5:30-7 p.m. at the AACR Annual Meeting 2020 in San Diego, California. For information on Dr. Rudolph's background and goals, visit the CICR Election Result webpage. Thanks to all CICR members who participated in the election!

CICR Town Hall Meeting and Reception held at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics on Oct. 27, 2019 in Boston, Massachusetts

I was pleased to greet many of you, along with CICR Past Chair, Dr. Julian Blagg, at the CICR Town Hall Meeting and Reception at this informative conference, where an update on the CICR Working Group initiatives was presented. Almost 70 individuals attended. Afterwards, it was impressive to participate in the discussions and cordial networking held over appetizing refreshments. On behalf of the CICR Steering Committee, we hope to see many of you at similar functions in the future!
Plan to attend the CICR Town Hall at the AACR Annual Meeting 2020 in San Diego!

We look forward to an interesting Town Hall on Sunday, April 26, 2020, 5:30-7 p.m., during the [AACR Annual Meeting](https://www.aacr.org). This event will take place after that day's final session of the popular "New Drugs on the Horizon" drug development special session. Come join us for some excellent discussion on CICR activities and productive networking after the formal program.

CICR Scholar-in-Training Awards (SITAs) to be presented at the AACR Annual Meeting 2020

Early-career investigators, be sure to check the [AACR Annual Meeting 2020 website](https://www.aacr.org) to learn how you can submit an abstract to be considered for one of these inaugural travel awards to the AACR Annual Meeting 2020 in San Diego, California. Abstract submission deadline is Thursday, Dec. 5, 2019. Generous support of these awards is provided by WuXi AppTec.

Join the Conversation on the New CICR LinkedIn Network

We cordially invite those of you not yet "CICR-connected" to join the [CICR LinkedIn Network](https://www.linkedin.com/groups/8813575/). This network will enhance communication among CICR members and the greater cancer research community interested in cancer chemistry research! Be part of this exciting forum to interact and connect, while sharing recent research updates of interest. Access the network, join, and contribute. We look forward to your active participation and be sure to share with your colleagues! Reference URL: [https://www.linkedin.com/groups/8813575/](https://www.linkedin.com/groups/8813575/)

*Thank you for your CICR membership!*

Contact us at [cicr@aacr.org](mailto:cicr@aacr.org).
**Selected Research and Review Highlights**

**High Proliferation Rate And A Compromised Spindle Assembly Checkpoint Confers Sensitivity To The MPS1 Inhibitor BOS172722 In Triple Negative Breast Cancers**


BOS172722 is a highly potent, selective and orally bioavailable MPS1 inhibitor currently in the clinic. BOS172722 treatment in combination with paclitaxel induces gross chromosomal segregation defects caused by MPS1 inhibitor-mediated abrogation of the mitotic delay induced by paclitaxel treatment. MPS1 inhibition led to a significant sensitisation of TNBC cells to death and regression of patient-derived xenografts. The most important discovery in this paper from Anderhub et al. is that MPS1 inhibition induces significant cell killing, particularly in highly proliferative cancers. They anticipate that use of proliferation markers for patient selection may be beneficial for the success of MPS1 inhibitors in clinic.

**Structure-based design of potent selective inhibitors of protein kinase D1 (PKD1)**


The authors expand on a series of kinase inhibitors, based on a tricyclic propargyl alcohol chemotype, that were previously described as potent inhibitors of methionine gatekeeper kinases NIK, AKT, and PAK4/5/6. In all these cases the stereochemistry of the propargyl alcohol was critically important for their potency, with the R-enantiomer preferred. These observations were rationalized by observed hydrogen bonding interactions between the alcohol and the DFG motif, as observed in X-ray crystallographic structures. The authors used this chemotype as a starting for the development of potent and selective PKD1 kinase inhibitors. Unlike what was observed with other kinases, hydrogen bonding to the propargyl alcohol was not required for potent PKD
inhibition. This observation, in particular, led to highly selective PKD inhibitors such as compound (S)-12, with significant selectivity over NIK.

Properties of FDA-approved small molecule protein kinase inhibitors


This review summarizes available information for the 48 FDA-approved kinase inhibitors — many of them for oncology indications — as of the time of the report. The inhibitors are classified by the kinase targeted for inhibition, as well as the overall target class (25 receptor tyrosine kinases, 10 non-receptor protein kinase, and 13 protein-serine/threonine protein kinases). Contained herein is a summary of the uses of each molecule in both oncology and non-malignant diseases. Further, the author describes the X-ray co-crystal structures of the compounds bound to their respective targets, if that information is known. While all but one of the reported inhibitors is orally efficacious, the author finds that 20 of the 48 molecules exceed at least one of the Lipinski “rule of five” metrics.

RNA errors could be a new source of cancer vaccines

https://www.nature.com/articles/s41598-019-50738-4

Immunotherapy approaches to cancer treatment are thought to work by activating the patient’s own immune system to fight off the disease. Triggering this response requires identification of neoantigens present in the tumor tissue, and non-responders appear to have lower levels of neoantigens. The authors of this recent report propose a new option that might expand the scope of personal cancer vaccines that could increase tumor response rates. They have discovered that certain transcriptional errors give rise to “frameshift neoantigens” which appear to be highly immunogenic. Due to relatively predictable patterns in these errors, a peptide array that can represent the bulk of them can be created and used to detect an antibody response in patient blood samples. Further, these peptide “vaccines” were shown to delay or prevent tumor growth in mouse models. This suggests that cancer vaccines based on RNA mistakes, rather than DNA mutations, may one day be created from these frameshift neoantigens.

Drug Discovery Targeting Anaplastic Lymphoma Kinase (ALK)

https://pubs.acs.org/doi/10.1021/acs.jmedchem.9b00446

This article describes recent drug discovery efforts targeting anaplastic lymphoma kinase (ALK), a receptor tyrosine kinase that is validated to play an important role in cancers such as anaplastic large cell lymphoma (ALCL), non-small cell lung cancer (NSCLC), and neuroblastomas. There are currently five FDA approved small molecule inhibitors of ALK: crizotinib (Pfizer, 2011), ceritinib (Novartis, 2014), alectinib (Chugai/Roche, 2015), brigatinib (ARIAD Pharma, 2017), and lorlatinib (Pfizer, 2018). The article describes these inhibitors and others, focusing on their chemotypes, activity, selectivity, and resistance as well as potential therapeutic strategies to overcome drug resistance. Finally, the authors describe the recent application of the PROTAC technique in developing ALK degraders, which opened a new avenue for targeted ALK therapies.
Identification of Novel, Potent, and Orally Available GCN2 Inhibitors with Type I Half Binding Mode

https://pubs.acs.org/doi/10.1021/acsmedchemlett.9b00400

General control nondepressible 2 (GCN2), a serine/threonine-protein kinase, is a master regulator of amino acid homeostasis and is linked to cancer survival in the tumor microenvironment. The authors present their efforts at optimizing an aryl sulfonamide with an unusual core alkyne spacer separating the hinge binding region from the allosteric pocket binder. The chemotype was recognized for its selectivity over other kinases, which the authors propose is due to the aryl sulfonamide inducing a conformational change around the alphaC-helix with DFG-in folding. Development of this series of compounds focused on improvements in solubility and PK parameters. An optimal inhibitor from this series exhibited in vitro and in vivo proof of concept efficacy by suppressing GCN2 pathway activation with asparaginase treatment in CCRF-CEM cells and a mouse xenograft model.
Chandra Miduturu, PhD, is currently a principal scientist/associate director in the Medicinal Chemistry group at Blueprint Medicines. He joined Blueprint Medicines as a founding chemist in 2011 after completing his postdoctoral research work with Prof. Nathanael Gray at the Dana-Farber Cancer Institute (DFCI) and Harvard Medical School (HMS), Boston. As one of Nathanael’s first postdoctoral researchers, he contributed to a new strategy of discovering novel kinase inhibitors and scaffolds by utilizing a high-throughput kinase screening method across the entire human kinome. He also worked on developing novel small molecule tools to interrogate kinases and phosphatases utilizing both cell and chemical biology techniques. He joined the DFCI/HMS postdoctoral program after obtaining his PhD in chemistry with Prof. Scott Silverman from the University of Illinois at Urbana-Champaign Chemical Biology program developing DNA constraints to study macromolecular structure and folding. Prior to his graduate studies, he obtained his bachelor’s and master’s degrees in chemistry from the Loyola College, Chennai, India, and the Indian Institute of Technology, Madras, India.

Starting operations in 2011, Blueprint Medicines has conducted kinase-focused drug discovery and development with four investigational medicines in clinical development targeting genomically validated kinases. Its approach empowers the rapid design and development of potent and selective treatments and increases the likelihood of clinical success. As a founding chemist at Blueprint Medicines, Chandra contributed to the design and synthesis of kinase-focused small molecule libraries. Annotating this library for potency and selectivity across more than 450 kinases has enabled Blueprint Medicines to identify potentially ideal starting points for medicinal chemistry programs. This early foundational work supports the rapid and reproducible discovery of precision therapies, and has helped Blueprint Medicines be among a few select companies that may bring two wholly discovered medicines, if approved, to patients within its first ten years of operation.

Chandra has spent most of his time at Blueprint Medicines devoted to several kinase inhibitor programs for oncology. Notably, Chandra is a key contributor to discovering two drug candidates now in clinical development: the mutant-selective KIT and PDGFRα inhibitor avapritinib and covalent FGFR4 selective inhibitor fisogatinib (also known as BLU-554). Avapritinib has received Food & Drug Administration (FDA) Breakthrough Therapy Designations for two distinct disease indications, and its first New Drug Application was accepted by the FDA in August 2019.

Most recently, Chandra has served as the lead chemist in Blueprint Medicines’ collaboration with Roche, targeting up to five immuno-kinases. Currently approved immunotherapies are primarily antibodies that address immune checkpoint drug targets outside the cell; in collaboration with Roche, Blueprint Medicines is looking at...
cancer immunotherapy differently by identifying intracellular immunokinases that play a role in antitumor immune responses, and precisely targeting them with small molecule therapies.

Chandra has co-authored 10+ peer-reviewed publications and has nine published patent applications. He has been an invited speaker to give first disclosures of some of this work, including the 2017 ACS first time disclosures, the 2017 Medicinal Chemistry Gordon Research Conference and 2017 Royal Society of Chemistry medical chemistry symposium.
Spotlight on World News

Note: Discussion of FDA approvals for this issue are limited to new chemical entities approved to treat cancer since the previous CICR Newsletter issue (August 2019).

FDA Approval of Darolutamide (Nubeqa)

On July 30, 2019, Bayer & Orion received approval for the non-steroidal antiandrogen (NSAA) darolutamide for the treatment of adult patients with non-metastatic castration-resistant prostate cancer (nmCRPC). The approval of this second generation NSAA follows enzalutamide (Astellas, 2012) and apalutamide (Janssen, 2018), a pair of structurally similar androgen receptor antagonists. See related blogpost.

Source: FDA

FDA Approval of Pexidartinib (Turalio)

The CSF1R antagonist pexidartinib (Turalio, Plexxikon) was approved on August 2, 2019 to treat adult patients with symptomatic tenosynovial giant cell tumor (TGCT). TGCT is a rare cancer that commonly affects the tendons of the fingers, hands and wrists. Although the tumor is rarely malignant, it causes tendon sheaths to overgrow causing damage to surrounding tissue. Colony stimulating factor 1 receptor (CSF1R) is a cell surface protein that is known to be over-expressed in many cancers and on tumor-associated macrophages.

Sources: FDA; FDA News
FDA Approval of Entrectinib (Rozlytrek)

Roche received approval for entrectinib (Rozlytrek) on August 15, 2019 to treat adult patients with metastatic non-small cell lung cancer (NSCLC) whose tumors are ROS1-positive. The inhibitor is selective for pan-Trk tropomyosin receptor kinases (targeting TrkA, TrkB, and TrkC), ROS1 kinase, and ALK kinase. The approval marks the third tissue agnostic FDA approval, in which the therapeutic can be used to treat patient tumors that has tested positive for a specific biomarker. See related blogpost.
Source: FDA

FDA Approval of Fedratinib (Inrebic)

On August 16, 2019, Celgene received approval for the Janus kinase 2 (JAK-2) inhibitor fedratinib (Inrebic) to treat adult patients with intermediate-2 or high-risk primary or secondary myelofibrosis. The compound is a low nM inhibitor of JAK-2, but also inhibits FLT3, RET, and JAK-3 kinases. The approval marks the second approval for myelofibrosis after the JAK inhibitor ruxolitinib (Jakafi, 2011).
Source: FDA; Leukemia

Oncologist Stephen Hahn appears to be frontrunner for FDA head

The position of commissioner of the FDA has not had a permanent occupant since Scott Gottlieb, MD left the post earlier this year. While Ned Sharpless, MD, the former director of the National Cancer Institute (NCI), has held the position as a stop-gap measure, U.S. President Trump appears poised to nominate Stephen Hahn, MD, for a permanent role. Hahn is a radiation oncologist by training, and has held positions at the NCI, the University of California – San Francisco, and the University of Pennsylvania School of Medicine. He is currently the Chief Medical Officer at MD Anderson Cancer Center. This lengthy experience at academic medical centers is slightly different than that of many of his precursors. If Hahn does, indeed, receive the nod from the President, his nomination would be subject to the normal US Congressional vetting process.
Source: BioCentury; Stat

AMG510 shows limited clinical efficacy in colon cancer

The last two issues of our newsletter have profiled the first covalent KRASG12C inhibitor to hit the clinic. In lung cancer, this compound has shown some impressive results. In contrast, colon cancer patients have not yet realized similar benefit. As presented at a September meeting of the European Society of Medical Oncology (ESMO), of the 12 patients with KRASG12C mutation who received the highest dose, only 1 partial response was noted. However, most patients did present with stable disease, and no prominent safety concerns were noted. This is compared with 13 patients with NSCLC, of which more than half achieved a partial response.
Source: FierceBioTech; Amgen
Upcoming Conferences

Third FBDD Down Under
Nov. 12-15, 2019, Melbourne, Australia

San Antonio Breast Cancer Symposium
Dec. 10-14, 2019, San Antonio, Texas

Advancing Precision Medicine Drug Development: Incorporation of Real-World Data and Other Novel Strategies
Jan. 9-12, 2020, San Diego, California

Sixth AACR-IASLC International Joint Conference: Lung Cancer Translational Science from the Bench to the Clinic
Jan. 11-14, 2020, San Diego, California

Cancer Evolution and Combinatorial Cancer Therapies: Concepts and Challenges
Jan. 19-23, 2020, Banff, Alberta, Canada

American Chemical Society National Meeting and Expo
March 22-26, 2020, Philadelphia, Pennsylvania

15th CHI Fragment-Based Drug Discovery
April 13-17, 2020, San Diego, California

AACR Annual Meeting 2020
April 24-29, 2020, San Diego, California

Kinase 2020: 9th RSC/SCI symposium on kinase inhibitor design
June 1-2, 2020, London, UK

Fragment-Based Lead Discovery 2020
Sept. 20-23, 2020, Cambridge, UK.
### Funding Opportunities

See this page: [https://www.aacr.org/FUNDING/PAGES/FUNDING-LISTING.ASPX](https://www.aacr.org/FUNDING/PAGES/FUNDING-LISTING.ASPX)
For a current listing of available AACR grant opportunities

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CLOSED  AACR-Bristol-Myers Squibb Midcareer Female Investigator Grant

Grant Amount: $225,000 USD  Application Deadline: 11/19/2019  Decision Date: 3/2020  Start of Grant Term: 7/1/2020
Grant Duration: 3 years