Cancer Drug Resistance

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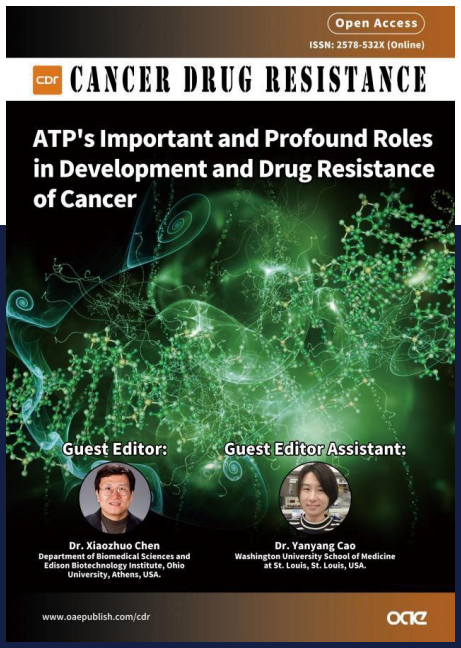
ATP's Important and Profound Roles in Development and Drug Resistance of Cancer



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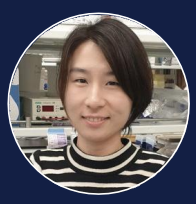

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Special Issue Introduction:

Adenosine 5'-triphosphate (ATP) is one of the most conserved small biochemical molecules in the bio-world, playing a wide variety of vital functions in both normal and cancer cells. It has long been well known that ATP plays roles in energy provision, transcription, and signal transduction. In the tumour microenvironment (TME), the level of ATP in the tumour interstitial reaches the hundred micromolar range, at least 103 times higher than those found in healthy tissues, but how tumour cells utilize the highly concentrated extracellular ATP (eATP) during cancer progression is still far from understood. More recent research results indicate that the high levels of eATP in the TME significantly and drastically affects the metabolism, growth, and cell differentiation of cancer cells in cancer cells and in tumours. Growing evidence indicates that tumour cells internalize eATP via macropinocytosis to drastically elevate intracellular ATP (iATP) levels. This increase in iATP levels contributes to tumour proliferation, survival, therapeutic response, drug resistance, induction of epithelial-to-mesenchymal transition (EMT), cancer stem cell (CSC) formation, and invasion. This list of newly found ATP functions has been keeping growing. In addition, eATP acts as an extracellular signalling molecule to activate various purinergic receptors (PR), leading to PR-mediated specific signalling for cancer progression.

This issue will summarize some of the most recent discoveries of ATP relative to development and drug resistance of cancer, and some of the most important and urgent scientific questions to be answered. By reading this issue, it may make readers wonder: what functions or processes does ATP not participate in?

We welcome research and review articles on a broad range of topics related to ATP and cancer, including but not limiting to:

- The functions and mechanisms of extracellular ATP in drug resistance;
- Molecular mechanisms of extracellular ATP-induced effects on cancer cells, including EMT, stemness, cell remodeling, and invasion;
- Mechanisms by which cancer cells exploit extracellular ATP in the tumor microenvironment to modulate drug resistance and overall cancer progression;
- How intracellular ATP regulates cancer cell metabolism in vitro and in vivo, with a focus on its role in drug resistance;
- Pre-clinical and clinical applications of targeting ATP and ATP-mediated signaling, encompassing P2 receptor signaling, and other innovative therapies aimed at combating drug resistance in cancer.

Submission Deadline: May 30, 2024

Journal Metrics

3.7 - 2-year Impact Factor
5.5 - CiteScore
6.1 - CiteScoreTracker 2023

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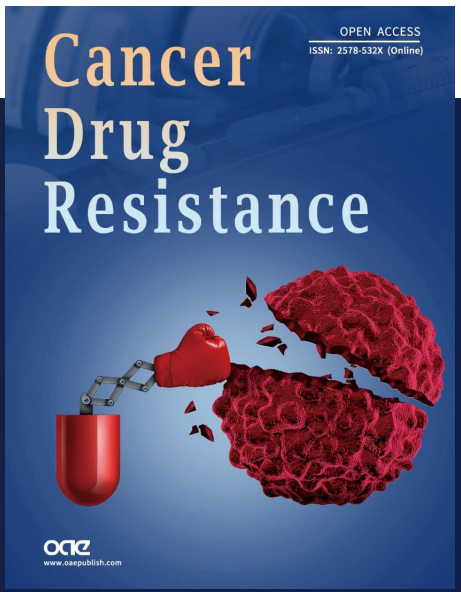
About the journal:

Cancer Drug Resistance (*CDR*) is an international peer-reviewed, open access, online journal.



Aims & Scope:

CDR is a continuously published journal committed to the rapid publication of high quality, peer-reviewed, original research. The journal publishes research articles, reviews, case reports, commentaries and letters on pharmacological aspects of drug resistance and its reversal, including drug design, drug delivery, drug distribution and cellular drug resistance. Molecular mechanisms of drug resistance also cover the cellular pharmacology of drug resistance such as influx and efflux pumps (including the ABC pumps), receptors and their ligands, cellular signaling pathways, drug activation and degradation (including Phase I and II metabolism), drug sequestration, target modification and DNA repair. Drug classes involved include DNA targeted drugs and antihormones as well as antibodies and protein kinase inhibitors. Both clinical and experimental aspects of drug resistance in cancer are included.

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