Apoptosis (2023) 28:1503–1504
https://doi.org/10.1007/s10495-023-01895-5

Rising impact of cell death research

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Accepted: 11 September 2023 / Published online: 26 September 2023

Since we took over editing *Apoptosis* in 2016, a steady and continuous rise in the ISI/Clarivate impact factor from 3.8 to 5.6 in 2021 could be noted. Last June we received the impact factor of 2022 and it appeared that the impact rose with 1.6 points to a firm 7.2. This is a great achievement due to the increasing interest in programmed cell death mechanisms and the resulting increased submission of high-impact and high-quality manuscripts. As editors of Apoptosis, we keep being determined to improve the journal's quality. We continuously report on the progress of the journal [1–3] and celebrated recently the quarter-century anniversary of *Apoptosis* [4]. The journal is heading forward while we will continue to maintain the rigorous, fair and fast peer-review process of submitted manuscripts, recruit invited reviews and aim for special issues on trending topics in the field of programmed cell death. Of course, *Apoptosis* would be nowhere without the trust of authors and the continued support of our excellent editorial board members and outside reviewers.

The above-mentioned rise in interest in, and impact of, cell death mechanisms is reflected by the increasing number of reports on the different mechanisms of programmed cell death [5–7]. *Apoptosis* published a number of reports on ferroptosis, the iron-dependent cell death pathway that is biochemically distinct from other cell death mechanisms. A paper reported a ferroptotic gene signature identifying targets that could be used to improve immunotherapy of renal cell carcinoma [8]. A review on damage-associated molecular patterns described the relationship with ferroptosis [9]. Two reviews discussed the topic, one on the role of ferroptosis in myocardial infarction, generating novel insights on the prevention and treatment of this condition by targeting the process of ferroptosis [10], and one on targeting lipid metabolism to induce ferroptosis and affect tumor cell survival [11]. Another programmed mode of cell death is necroptosis, the programmed version of necrosis. Regulation of necroptosis got major attention recently, in one paper aiming for increasing cardiac tolerance to ischemia–reperfusion injury, in order to reduce mortality rates in patients with acute myocardial infarction [12]. Another report aims to delineate the role of ubiquitylation in the process of necroptosis to facilitate the development of new therapeutic strategies for necroptosis-related diseases [13]. Pyroptosis is an inflammatory form of lytic programmed cell death often happening as an anti-microbial cellular response, but also taking part as a regulatory pathway in different pathologies. Regulation of pyroptosis was part of several recent publications [14, 15] and disease-specific mechanisms were described [16–19]. Autophagy is a degradation and recycling pathway in cells for adaptation to stress signals and is therefore often closely associated with cell death mechanisms. This currently appears to be a major topic of research and fundamental studies were published in *Apoptosis* [20–22]. Regulation or targeting of autophagy was also presented in studies to harness it as a strategy for cancer treatment [23–26], heart ischemia and reperfusion [27] and other diseases [28, 29].

Since the beginning of the COVID-19 pandemic, reports on cell death mechanisms related to virus infection frequented *Apoptosis*. Since COVID-19 is a vascular pathology [30] the virus strikes in many tissues. It was recently published that SARS-CoV-2 also disrupts spermatogenesis [31] and neurogenesis [32]. In addition, it was hypothesized that apoptosis induced by SARS-CoV-2 can be targeted for the treatment of COVID-19 [33].

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Declarations

Competing interests The authors declare no competing interests.

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