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Information about the [Vucic Lab](#)



Tuesday 22.03.2022 at 5 pm

“Drug discovery adventures of a cell death cowboy”



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Abstract:

Cell death is involved in all aspects of life, from the development of multicellular organisms to fighting off infections and maintaining tissue homeostasis. Thus, it is not surprising that multiple diseases that are caused by deregulated or dysfunctional cell death signaling. Consequentially, there is a growing interest in modulating cell death to treat diseases. Early therapeutic efforts focused on inducing apoptotic cell death in tumor tissues using Bcl-2 and IAP antagonists, as well as activators of TNF family death receptors. The role apoptosis and of other forms of cell death, such as necroptosis and pyroptosis, and their functional connectivity, is also evident in various inflammatory diseases. TNF is critical for the development and proper functioning of the immune system by mediating cell survival and cell death inducing signaling. Disruption of these signaling events can disturb the balance and the composition of signaling complexes, potentially resulting in severe inflammatory diseases. RIP1 kinase is a particularly important regulator of inflammation and cell death signaling emanating from TNF and other tissue-damaging stimuli. Inactivation and/or inhibition of RIP1 in preclinical animal disease models has validated its kinase activity as a key factor and established RIP1 as an attractive therapeutic target for the treatment of inflammatory diseases. In my presentation I will emphasize the instrumental and protective role for RIP1 kinase inhibition in inflammatory disease, and discuss our current drug discovery efforts based on cell death pathways.

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Information on the Cell Death Club seminar series and registration are advertised via the local mailing lists and is provided on the [website of the SFB 1403](#) and on [twitter](#).