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Implications of Non-targeted effects for advanced radiotherapy

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Our understanding of the mechanisms of radiation response in biological systems has been changing from studying the role of radiation damage at the DNA and cellular level to integrated models of tissue and whole organism responses. Key to this has been a growing appreciation of bystander responses where cells respond to their neighbours being irradiated and intercellular signalling mechanisms play a role. Bystander responses have been observed locally between cells but also over longer ranges including between tissues in the intact organism. Microbeam approaches, where localised radiation beams can be delivered, have been key experimental tools which are allowing the delineation of mechanisms underpinning bystander signalling in a range of models.

For advanced radiotherapies, delivery of dose into tumours is highly modulated in both space and time. A major consequence is the production of dose-gradients across tumours and normal tissues as dose is "painted" into the treatment volume. Little is known about the underlying biological rationale optimising these approaches and the role, if any bystander signalling will play. More recently studies with modulated photon beams have begun to address these issues to develop biologically driven models for radiotherapies which can take into account any impact of bystander signalling.

This is leading to a redefinition of bystander signalling, away from the hit versus non-hit signalling hypothesis, to one where the response of cells and tissues to radiation exposures is via an integrated response to oxidative damage.

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