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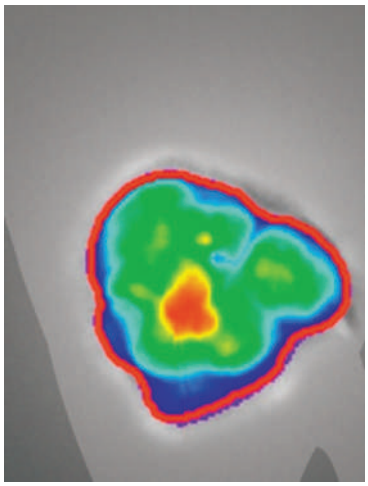

The RON receptor tyrosine kinase is overexpressed in premalignant pancreatic intraepithelial neoplasia and in the majority of pancreatic cancers; however, the pathobiological significance of RON overexpression in pancreatic cancers has yet to be fully established. In this study, Logan-Collins and colleagues show that in both murine and human pancreatic cancer cells, RON signaling regulates the expression of genes implicated in cancer cell survival. shRNA-mediated silencing of RON in pancreatic cancer xenografts inhibited their growth, primarily by increasing susceptibility to apoptosis and by sensitizing them to gemcitabine treatment. In addition, the authors show that escape from RON silencing is associated with re-expression of RON and/or expression of phosphorylated forms of the related c-Met or epidermal growth factor receptors. Given these findings, the authors propose that RON signaling mediates cell survival and *in vivo* resistance to gemcitabine in pancreatic cancer, and they reveal mechanisms through which pancreatic cancer cells may circumvent RON-directed therapies. For details, see the article by Logan-Collins and colleagues on page 1130 of this issue.

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