Summary and conclusion

Our study provides experimental evidence that suggests MAGEA3 is an important survival molecule for pancreatic cancer cells under metabolic and genotoxic stress conditions. The mechanistic study revealed that CCL2 and/or survivin are two possible functional mediators of MAGEA3. Thus, we propose that targeting MAGEA3 may have a better impact on PCA therapy. We also report the involvement of MAGEA3 in different signalling pathways. Our proteomics approach to identify interacting partners of MAGEA3 provided a preliminary data with a list of proteins involved in different molecular regulatory networks. Our in vivo experiments proved the involvement of MAGEA3 in pancreatic cancer progression. We tried to transform mouse primary pancreatic epithelial cell with huMAGEA3 and/or muKRAS\textsuperscript{G12D} in vitro but failed, however we observed upregulation of survivin upon MAGEA3 overexpression, which suggests that MAGEA3 alone may not be a driver oncogene. The contribution of MAGEA3 to oncogeneic process need to be further evaluated along with other genetic alterations.

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