Silencing of Human ADAM9 Increases Tumor Cell Proliferation

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The ADAM protein family comprises multi-domain and multi-functional membrane proteins, which contain disintegrin and metalloprotease domains (**A** **D**isintegrin **A**nd **M**etalloprotease). These proteins are expressed in different animal species such as mammals and insects and their adhesive domains play a key role in many physiological processes, such as the fertilization, myoblast fusion, cell survival, migration and proliferation. ADAMs can also be involved in some diseases, including cancer spreading and metastases. The expression of the ADAM9 is increased in tumor cells such as prostate cancer cells (DU-145) and breast tumor cells (MDA-MB-231) as compared to fibroblasts. The aim of this work was to generate knockout clones lacking ADAM9 expression using silencing RNA techniques. MDA-MB-231 cells were transfected with Lipofectamine (Invitrogen) and silencing primers for the disintegrin domain (sense sequence: 5' - rCrCrArGrArGrUrArCrUrGrCrArArUrGrGrUrUrCrUrUrCTC - 3' and antisense sequence: 5'- rGrArGrArArGrArArCrCrArUrUrGrCrArGrUrArCrU rCrUrGrGrArA - 3') of ADAM9. Knocking out of ADAM9 and GAPDH was confirmed by real time PCR. Silenced cells showed significant enhanced proliferation rate 8 days after transfection but not after 15 days. These results suggest a role for ADAM9 in the control of tumor cell proliferation.

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