



DDR1 and MT1-MMP expression are determinant for triggering BIK-mediated apoptosis by 3D type I collagen matrix in invasive basal-like breast carcinoma cells

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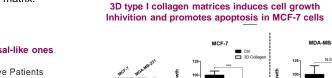
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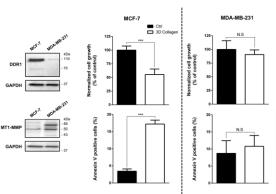


Background and aim

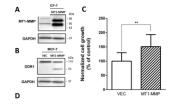
- During tumor progression, breast carcinoma cells are confronted to a type I collagen-rich environment.
- Discoidin Domain receptor 1 (DDR1) is a tyrosine kinase receptor of type I collagen, decreases cell growth and initiate apoptosis in non-invasive luminal breast carcinoma.
- Fibrillar organization of type I collagen is crucial for DDR1 activation,
- MT1-MMP is able to induce both type I collagen degradation and DDR1 cleavage.
- Here, we propose to investigate how apoptosis and cell growth are regulated in invasive basal-like breast carcinoma in type I collagen 3D matrix.

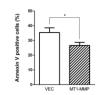


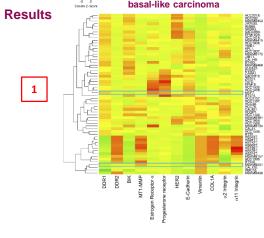




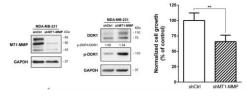
MT1-MMP overexpression in MCF-7 cells induces DDR1 cleavage and promotes cell growth

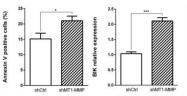


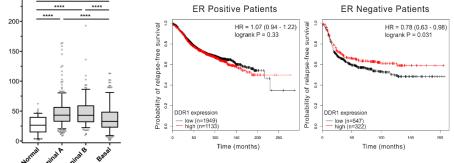




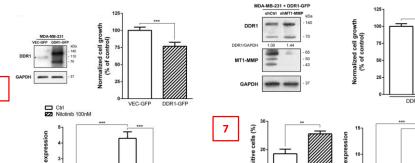
MT1-MMP depletion in MDA-MB-231 cells induces an increase in full length DDR1 expression and promotes cell apoptosis



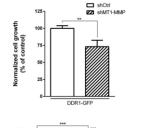


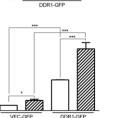


DDR1 overexpression in cells increases cell apoptosis MDA-MB-231 cells increases apoptosis level



DDR1 overexpression coupled with MT1-MMP depletion in MDA-MB-231





Conclusion

