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Exploring EGR-1 as a Master Regulator of Prostate Field Cancerization

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Support: Undergraduate Student Scholarly/Creative Grant from the Office of Undergraduate Research

METHODS

Cell Models

Human Tissues

RESULTS: Cell Models

• EGR-1 is a regulator of FAS, MIC-1, and PDGF-A expression
• EGR-1 down-regulated FAS and MIC-1 expression by 70% and 30% in PC-3 cells (p=0.005, p=0.015)
• EGR-1 up-regulated PDGF-A expression by 110-fold in PC-3 cells (p=0.004)

RESULTS: Human Tissues

• EGR-1, FAS, MIC-1, and PDGF-A have greater expression in adjacent tissue, which can be a molecular mechanism of field cancerization
• Ratio of either FAS, MIC-1, and PDGF-A to EGR-1 expression
• High ratios suggest a potential regulatory role of EGR-1
• EGR-1 is regulator of FAS, MIC-1, and PDGF-A expression
• Ratio of MIC-1 to EGR-1 expression increased significantly in adjacent and tumor tissues (p<0.05)

SUMMARY

Cell Models

Human Tissues

CONCLUSIONS

• EGR-1 is a regulator of FAS, MIC-1, and PDGF-A expression
• This novel analysis between cell models and human tissues is helpful identifying pathways in prostate field cancerization
• EGR-1, FAS, MIC-1, and PDGF-A are markers that can lead to improvements in prostate field cancerization

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