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ROLE OF WNT/BETA-CATENIN SIGNALING IN OVARIAN TUMOUR GROWTH AND ANGIOGENESIS. A CROSSTALK WITH NOTCH SYSTEM

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Wnt/β-catenin and Notch are highly conserved pathways that regulate a diversity of cell processes, including proliferation, apoptosis and differentiation. We analyzed the role of both systems in ovarian cancer using specific inhibitors. For this purpose we performed three *in vivo* experiments. A human ovarian adenocarcinoma cell line (IGROV-1) was subcutaneously injected in 6-8 weeks-old female nude mice. Once the tumours were palpable, we injected the inhibitors: the first and second experiments were carried out using Wnt/β-catenin inhibitors (XAV939: 2.5, 5 mg/kg; ICG-001: 5 and10 mg/kg). The third experiment was a combination of ICG-001 (5mg/kg) and DAPT (5 mg/kg), a Notch inhibitor. Mice were injected every two days three times and they were euthanized 3 days after the last injection. Our results showed a significant decrease in tumour size when mice were treated either with XAV939 or with ICG-001. When compared with tumours from non-treated animals, both experiments showed a significant decrease in cell proliferation (KI67) and a decrease in the endothelial and periendothelial cell area stained with CD31 and α-Smooth-muscle-actin, respectively. When mice were treated with XAV939, a significant decrease in VEGF levels and Angiopoietin 1/2 was observed. Regarding the experiment with the combination of inhibitors, there was a significant decrease in tumour size and a decline in tumour cell proliferation (KI67). Both inhibitors administered simultaneously produced a decrease in cell proliferation at the same extent as individually administrated. In conclusion, we demonstrate a clear involvement of Wnt/β-catenin in ovarian tumour growth and angiognesis. We suggest an interaction of this pathway with Notch system.