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Doctoral College
Metabolic & Cardiovascular Disease

Medical University of Graz

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WNT PATHWAY TUMOUR SUPPRESSOR MUTATIONS IN CANCER: BEYOND LOSS-OF-FUNCTION?

GUEST LECTURE by

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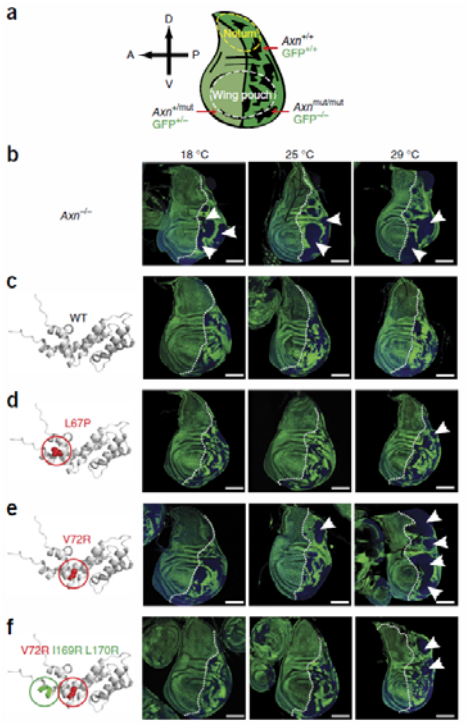
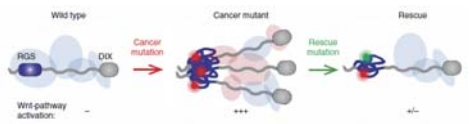
Tuesday, 25.04.2017
17:00

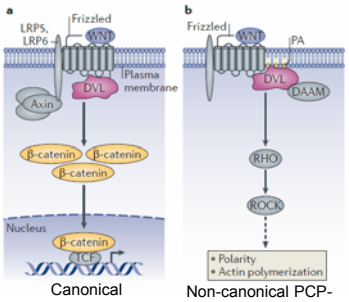
SR 07.11, Preclinics, MUG
(Harrachgasse 21, 1st floor)



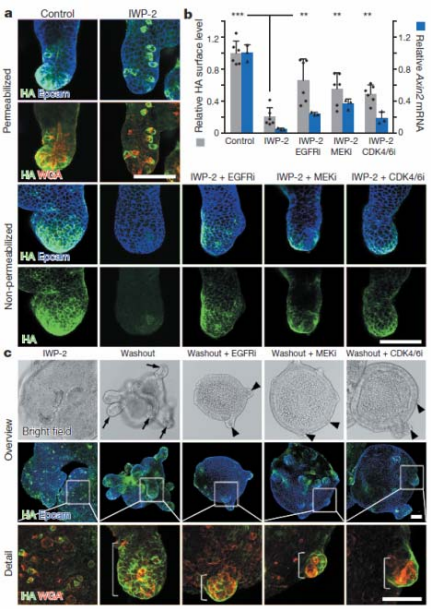
In vivo temperature-dependent hyperplastic growth induced by *Drosophila* Axin cancer mutants is rescued by aggregon mutations. Anvarian et al. (2016) Nat Struct Mol Biol 23(4):324-32



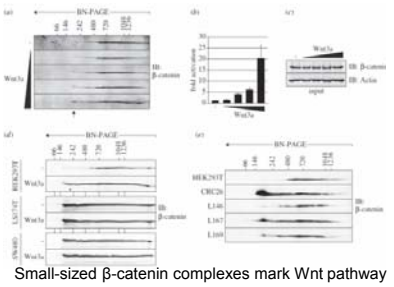
Model for the mechanism of action of Axin RGS cancer variants. Anvarian et al. (2016) Nat Struct Mol Biol 23(4):324-32



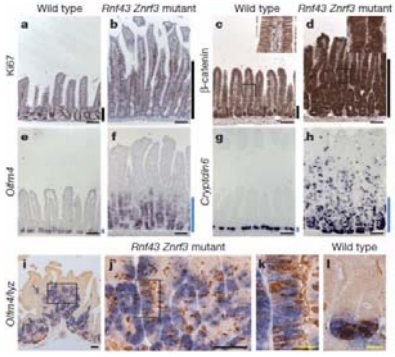
Canonical Non-canonical PCP-WNT signaling
Consonni et al. (2014) Nat Rev Mol Cell Biol 15:357-62



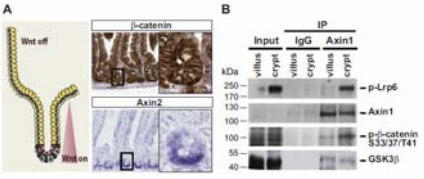
Cell proliferation influences Wnt3 surface level and signaling range. Farin et al. (2016) Nature 530:340-3



Small-sized β-catenin complexes mark Wnt pathway activation in primary human tumor cells. Gerlach et al. (2014) Open Biol 4:140120



Strong proliferation of the *Rnf43 Znrf3* compound mutant intestine is accompanied by Wnt/β-catenin activation as well as stem cell and Paneth cell metaplasia. Koo et al. (2012) Nature 488(7413):665-9



Wnt-induced β-catenin phosphorylation within the destruction complex is confirmed in primary tissues. Li et al. (2012) Cell 149:1245-56