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Sommario	Molecular basis of myosin VI alternative splicing in cellular transformation Rossella Scotto Di Perrotolo1, Carlos Niño1 and Simona Polo1,2 1IFOM, Fondazione Istituto FIRC di Oncologia Molecolare, Via Adamello 16, 20139, Milan, Italy. 2DIPO, Dipartimento di Oncologia ed Emato-Oncologia, Universita’ degli Studi di Milano, Via Festa del Perdono 7, 20122 Milan, Italy. Alternative splicing is a finely regulated process that plays a role in cancer development through largely unknown molecular mechanisms. Our lab characterized an alternatively-spliced exon cassette, called large insert, in the actin motor protein myosin VI. The inclusion of the large insert generates a long isoform involved in endocytosis, while the skipping produces a short isoform involved in cell migration. This finding is relevant to ovarian cancer where exon skipping dictates addiction to myosin VI short for tumor cell migration. Aim of this thesis was to understand how the isoform choice is regulated. We have set up convenient 2D and 3D epithelial cell culture models to modulate isoforms expression and to characterize the molecular mechanism behind. Our study identified a signaling pathway involved in the AS-reprogramming during the conversion from an epithelial/static to a mesenchymal/motile phenotype and three splicing regulators contributing to the myosin

	VI AS decision that appears to be promising therapeutic targets for ovarian cancer treatment.
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