# Tumor Metastasis with a Twist

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*Protein is key to early embryonic development, but later promotes spread of cancer*

In the early stages of human embryogenesis, a transcription factor called *Twist1* plays a key regulatory role in how the embryo assumes form and function. Much later in life, however, researchers at the University of California, San Diego School of Medicine, say *Twist1* can re-emerge, taking a darker and more deadly turn.

In a paper published in the March 15, 2011 issue of *Cancer Cell*, UCSD scientists led by Jing Yang, PhD, assistant professor of pharmacology and pediatrics, identify a unique function of *Twist1* in later life: it promotes the formation of invadopodia in tumor cells, a vital step in the spread of such cells (metastasis) to surrounding tissues and other parts of the body.

Invadopodia (meaning "invasive feet") are tiny protrusions of tumor cells that extend into the extracellular matrix – the surrounding connective tissue and fibers that provide support. Invadopodia concentrate enzymes that degrade the matrix so that tumor cells can break away and metastasize.

Previous studies have linked the expression of *Twist1* to many aggressive, solid-tumor cancers, including melanomas, neuroblastomas, as well as breast and prostate cancer. The new research by Yang and colleagues describes in detail how *Twist1* initiates the multi-step pathway resulting in invadopodia formation and matrix degradation. The research also reveals places in the process that may present potential targets for future anti-metastasis therapies.

After embryogenesis, *Twist1* is normally suppressed. Cancer cells, however, reactivate the transcription factor, enabling *Twist1* to initiate its complex pathway leading to metastasis. Drug designers, however, have yet to successfully devise a way to directly inhibit transcription factors like *Twist1*. The UCSD study points to other possibilities.

"We hope to inhibit downstream targets of *Twist1* (such as platelet-derived growth factor receptors) to inhibit invadopodia formation and function," Yang said. "Our study suggests that inhibition of invadopodia-mediated matrix degradation could be an effective way to suppress metastasis."

If that happens, a cancer tumor becomes a stable, unmoving and easier target for other types of therapeutic treatments.

Co-authors of the paper include Mark A. Eckert and Andrew T. Chang, UCSD Molecular Pathology Graduate Program and Biomedical Science Graduate Program; Thinzar M. Lwin and Etienne Danis, UCSD Department of Pharmacology; and Jihoon Kim and Lucila Ohno-Machado, UCSD Division of Biomedical Informatics.

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