



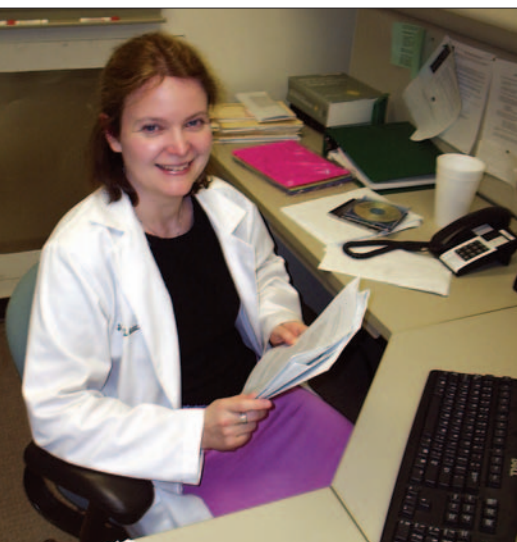
DMS geneticist Victor Ambros, Ph.D., presented data at a meeting in Europe on the use of a new assay for profiling microRNA in samples of the human brain cancer glioblastoma multiforme.

## A look at metabolism in breast tumors

If being a resident is like learning to ride a bike—you're pushing the pedals but are supported by training wheels or a parent running alongside—then resident Jennifer Quinn, M.D., M.P.H., will soon be trading one training bike for another.

**Skills:** Quinn, who is about to complete her residency in internal medicine, will stay on at DHMC as a Tiffany Blake Fellow. The fellowship, underwritten by the Hitchcock Foundation, funds a year of supervised study and research to help a physician develop the skills to become an effective investigator and to compete for independent grant support. Quinn will be working at Dartmouth's Norris Cotton Cancer Center in the lab of William Kinlaw, M.D., studying the role of glucose metabolism and progesterone in the development of breast tumor cells.

Quinn began her medical career in Boston, in the office of an ophthalmologist, where she worked with people who had degenerative eye diseases. In 2002, she graduated from the Tufts University combined M.D.-M.P.H. program.



LEE MCDAVID

Quinn looks forward to investigating how tumors grow, as well as to mentoring undergraduates.

She will suspend her clinical duties next year while she investigates sugar-to-fat metabolism and how it might promote breast cancer growth. Specifically, she'll look at a protein called CHREBP—a sugar sensor found in cancerous breast tissue but not in normal breast cells—and the role it plays in the synthesis of lipids, or fats, and in the growth and survival of breast cancer cells. She will also investigate the role CHREBP plays in how progesterone modulates sugar signaling. "It's one part of a very big story," she says.

Quinn hopes her studies will shed light on why breast tumors have a high rate of fat synthesis, which encourages breast cancer. "These tumors are hypermetabolic and gobbling up sugar," she notes. "Is that somewhere we can intervene?"

**Grassroots:** Most of Quinn's training has been clinical, and most of her research has been epidemiological—related, that is, to the incidence and control of disease in broad populations—so she now wants to beef up her laboratory experience. "I convey information much better if I understand it at a grassroots level myself," she says. "I don't like to just quote evidence to patients. I like to know where that evidence came from."

As a Tiffany Blake Fellow, Quinn will be able to develop the skills and contacts required to do independent clinical research, but she is also looking forward to mentoring Dartmouth College undergraduates—including at least one student participating in the Women in Science Program. She enjoys the role of mentor and teacher, as well as researcher. "If I had my druthers, my ideal career would be one where I could be both a physician and a scientist," she says. "Ultimately, the goal is to do something that will help people. . . . I think I see where [my research] could do that. Not tomorrow, not in a year, but maybe down the road." LEE MCDAVID

### Protein of pain

DMS researchers recently demonstrated how a receptor protein in the central nervous system, dubbed TLR4, contributes to neuropathic pain—a debilitating condition resulting from damaged or dysfunctional nerves. The finding, published in the *Proceedings of the National Academy of Sciences*, reveals new therapeutic possibilities for treating chronic pain. "The results of this study are significant because they demonstrate the potential for very novel drug targets," says the lead researcher, Joyce DeLeo, Ph.D., director of the Neuroscience Center at Dartmouth.



### Modeling disease

Human autoimmune pancreatitis (AIP)—a rare and poorly understood disease—may become less mysterious and more treatable thanks to a DMS study published in the *American Journal of Pathology*. The research team, led by pathologists Daniel Longnecker, M.D., and William Hickey, M.D., figured out how to induce a disease analogous to AIP in rats, thereby creating "an immunologically intact animal model to dissect basic immunological mechanisms" of the disease. "Furthermore," they wrote in the paper, "this model represents a novel means for the study of organ-specific autoimmunity in general."

