NOTED

- **The number of US cancer survivors rose to 13.7 million as of January 2012,** noted the *Cancer Progress Report 2013* (www.cancerprogressreport.org), released in September by the American Association for Cancer Research (AACR). Continued progress "will only be possible if we make funding for cancer research and biomedical science a national priority," emphasized Charles Sawyers, MD, AACR president and chair of the human oncology and pathogenesis program at Memorial Sloan-Kettering Cancer Center in New York, NY.
- **The U.S. Food and Drug Administration granted accelerated approval to Genentech's Perjeta (pertuzumab) for neoadjuvant treatment of early-stage breast cancer,** the first drug to receive such approval. "We are seeing a significant shift in the treatment paradigm for early-stage breast cancer," said Richard Pazdur, MD, director of the agency's Office of Oncology Drug Products.
- **Merck is cutting about one fifth of its global workforce,** with a new round of about 8,500 layoffs in addition to an earlier reduction of about 7,500 employees.
- **In a phase III trial of 602 women with advanced HER2-positive breast cancer whose cancer recurred or progressed despite previous treatments, those given Genentech's Kadcyla (T-DM1, ado-trastuzumab emtansine) achieved median progression-free survival (PFS) of 6.2 months,** compared with PFS of 3.3 months for those who received their physician's choice of treatment. Patients given Kadcyla also experienced fewer serious adverse side effects than those in the control group. The study was reported at the European Cancer Congress (ECC) 2013, held in Amsterdam, the Netherlands, from September 27 through October 1.
- **During the next 50 years, the dominant contributions to the global cancer burden will be from India, China, and Nigeria,** predicted the *State of Oncology 2013* report (www.i-pri.org/oncology 2013), presented at the ECC.
- **Nike cofounder Phil Knight pledged to give Oregon Health and Science University (OHSU) a $500 million award for cancer research**, but with an important footnote: For the award to be delivered, OHSU must raise an equivalent sum within 2 years.

higher in malignant colorectal tumors than in surrounding tissues. What wasn't clear, however, was whether the microbe was a cause or a consequence of cancer.

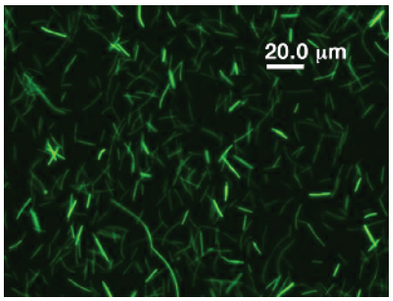
Now, two studies published in *Cell Host & Microbe* suggest that *F. nucleatum* creates a proinflammatory microenvironment and promotes colorectal carcinogenesis through E-cadherin and β-catenin signaling.

"*Fusobacteria* may provide not only a new way to group or describe colon cancers, but also, more importantly, a new perspective on how to target pathways to halt tumor growth and spread," says Wendy Garrett, MD, PhD. Garrett is an assistant professor of immunology and infectious disease and medicine at Harvard School of Public Health, Harvard Medical School, and Dana-Farber Cancer Institute, all in Boston, MA, and senior author of one of the studies (Cell Host Microbe 2013;14:207–15).

Building on the earlier research, Garrett's team examined colonic adenomas and normal tissue from 29 patients. They found an increased abundance of *Fusobacteria* sp. in adenomas compared with normal tissue, suggesting that the microorganism is involved in the initiation or progression of neoplasms. They also examined stool samples from healthy controls as well as patients with colorectal adenomas and carcinomas and found that levels of the bacteria increased as disease progressed.

Additionally, in a mouse model of intestinal tumorigenesis, the researchers found that *F. nucleatum* potentiated tumor development and recruited differentiated myeloid cells—including dendritic cells, myeloid-derived suppressor cells, and macrophages—that promote angiogenesis and tumor progression.

In the other study, a team led by Yiping Han, PhD, a microbiologist who has studied *F. nucleatum* for more than a decade and a professor of periodontics at Case Western Reserve University School of Dental Medicine in Cleveland, OH, showed that a small molecule called FadA on the surface of *F. nucleatum* binds to and modulates E-cadherin, a tumor suppressor, on colorectal epithelial cells (Cell Host Microbe 2013;14:195–206). β-catenin signaling is then activated, leading to the increased expression of transcription factors, oncogenes, *Wnt* genes, and inflammatory genes, and stimulating the growth of cancer cells.



Yiping Han

Abundant in the mouth, *Fusobacterium nucleatum* may cause colorectal cancer. Researchers don't know for certain whether the microbe plays a role in other cancers of the gastrointestinal tract.

Han's team also found specific *fadA* gene copy levels in normal colorectal tissue, adenomas, and carcinomas, with a 10-fold jump between each tissue type. "The differences were very clear," she says. "There was very little overlap between the three groups, and no overlap between normal and carcinoma."

Because *fadA* is unique to *F. nucleatum,* its expression could be used to diagnose colorectal cancer and identify people at risk for the disease, Han suggests. It could also be used to guide and assess possible treatments. "If FadA levels are reduced after treatment, perhaps it is an effective treatment," she says. "If it doesn't reduce the levels, perhaps the danger is still there."

As part of the research, Han's lab derived a synthetic peptide from a region of E-cadherin. Tested in mice, the peptide prevented *F. nucleatum* from binding to and invading cells, inhibiting tumor growth and inflammatory responses. Although the researchers didn't test the compound against other disorders related to *F. nucleatum,* she says there could be value in doing so.

"If we could block systemic proliferation of *Fusobacterium nucleatum,*" Han suggests, "we could kill many birds with one stone." ■

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