Vein grafting, revascularization with drug-eluting stents is quickly becoming the preferred treatment for these patients. In the future, drug-eluting stents may be used for the prevention of acute coronary syndrome. It is believed that acute coronary syndrome is caused by unstable plaques of mild to moderate stenosis (< 50%) that do not result in angina symptoms. Given the high event-free survival rate achieved with drug-eluting stents, one could argue for treating these lesions with stent therapy and potentially changing their natural history. In addition, the effect of drug-eluting stents on other difficult lesion subsets, such as left main coronary artery disease, bifurcation disease and chronic total occlusions, is currently under investigation.

However, it is important to keep in mind that the efficacy of the drug-eluting stent is based on avoiding recurrent angina and additional revascularization procedures. These stents do not reduce the incidence of myocardial infarction or death. From an economic perspective, a recent analysis of the SIRIUS trial showed that, even though the use of drug-eluting stents significantly increased the immediate procedural cost, there was no significant difference in cost at 1-year follow-up because of the reduced need for repeat revascularization.

Over the past 27 years percutaneous coronary intervention has undergone changes at a breathtaking pace. With the recent development of the drug-eluting stent, an even more spectacular evolution is taking place that will forever change the landscape of interventional cardiology.

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Do stem cells cause gastric cancer?

Although the link between Helicobacter pylori infection and gastric cancer is well established, new research suggests that stem cells play an important role in the development of this malignant disease. JeanMarie Houghton and colleagues recently showed that H. pylori-induced inflammation in mice caused the migration of stem cells originating from bone marrow to the stomach, where they subsequently developed into gastric tumours. Previous evidence suggests that bone-marrow-derived cells have a reparative function on being recruited to areas of injury or inflammation. The idea that these cells might also play a role in the development of cancer revisits a concept that arose partly from the observation in the 1970s that only 1% of leukemia cells grow into colonies in vitro, an ability that later earned these cells the label “cancer stem cells.” Houghton and colleagues’ research suggests that similar stem cells may give rise to gastric cancer, a finding that presents a new way of thinking about the pathogenesis of a disease that is the second leading cause of cancer-related deaths worldwide, killing nearly 600 000 people each year.

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Bone marrow-derived stem cell that has differentiated into a gastric epithelial cell. Reprinted, with permission, from Houghton et al.© 2004 American Association for the Advancement of Science.

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**H. pylori and cancer**

*H. pylori* is one of the most common chronic bacterial infections worldwide. The bacterium is the only one known to consistently tolerate the acidic environment of the stomach. Without treatment, *H. pylori* infection can persist for many years, causing chronic inflammation.

Up to 80% of patients with gastric cancer have a current or past *H. pylori* infection. This has led the World Health Organization to classify *H. pylori* as a group 1 carcinogen (www.cie.iarc.fr/monoeval/crthgr01.html). How the bacterium contributes to the development of cancer is still not entirely clear, although bacterial proteins, the immune response and hormonal responses have all been implicated. In addition, current research is beginning to link inflammation to the formation of tumours, with the inflammation-induced protein NF-kB emerging as a key factor.

### Are stem cells to blame?

Houghton and colleagues’ work suggests an unexpected alternative to the inflammation theory. The research group focused on the idea that bone-marrow-derived cells move into areas of chronic injury or inflammation to effect repairs. What long-term consequence this recruitment has on chronic inflammation is largely unknown. Houghton and colleagues wondered if these stem cells could be involved in the development of gastric cancer.

To study this question, Houghton and colleagues used a strain of mice (C57BL/6) and an in vivo model of gastric cancer in humans, since no markers are available. Nevertheless, these findings are a warning against the premature development of stem cell therapies and are bound to spark debate on the pathogenesis of gastric cancer.

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