Compensatory growth occurs in the developing liver, but not the pancreas

In many organisms, cellular loss during organ development initiates compensatory growth, mostly regulated by extrinsic factors, which ensures normal organ size. Grafting experiments have shown that some vertebrate tissue grows to a predetermined size regardless of the host environment, which suggests that intrinsic factors can also determine final organ size. Stanger and colleagues used mouse models to investigate organ size regulation in the pancreas and liver—two organs that develop from adjacent regions of the endoderm.

In one of the transgenic mouse models, ablation of pancreatic progenitor cells could be switched on and off at various developmental time points. These authors showed that if the number of progenitor cells is reduced during the early-bud stage, the remaining progenitor cells are unable to compensate. Pancreata from these mice grew at a normal rate after birth, but remained smaller than those of control mice. In chimeric mice created by addition of pancreatic progenitor cells to pancreatic-progenitor-deficient blastocytes, pancreas size correlated with the extent of chimerism. By contrast, compensatory growth occurred rapidly and precisely in the developing liver after hepatic progenitor cell ablation in transgenic mouse embryos.

The authors speculate that there may be two kinds of tissue: those like the liver, which undergo compensatory growth regulated by extrinsic factors, both during development and during regeneration in adulthood; and those that, like the pancreas, are subject to intrinsic growth control and are dependent on the number of progenitor cells available during an early window of development.

Original article Stanger BZ *et al.* (2007) Organ size is limited by the number of embryonic progenitor cells in the pancreas but not the liver. *Nature* **445:** 886–891

A safe and effective palliative treatment for malignant hilar biliary obstruction

Malignant hilar biliary obstruction is often unresectable at the time of diagnosis. Biliary drainage is a priority because it palliates the obstructive symptoms, but biliary drainage procedures have a high complication rate. Endoscopic placement of a metal stent is the preferred procedure, and unilateral stent placement can provide sufficient drainage for symptom palliation in these patients. Intraluminal brachytherapy (ILBT)—which delivers high-dose radiation to a bile-duct tumor whilst minimizing exposure of nearby organs—prolongs stent patency by slowing tumor ingrowth, so combining the use of both unilateral metal stenting and ILBT could provide an effective option for palliation of malignant hilar biliary obstruction.

In this pilot study Singh *et al.* evaluated stent patency and survival in eight patients with type II malignant hilar biliary obstruction who underwent endoscopic, contrast-free placement of a metal stent, followed 4 weeks later by endoscopic ILBT. These patients were compared with a historical control group of 10 patients who underwent contrast-free stent placement alone. Stent patency duration was longer in the ILBT-treated group than the control group: (mean ± SD) 305 ± 183.96 days versus 143.9 ± 115.11 days, respectively, $P = 0.03$. Survival (mean ± SD) was also prolonged in the ILBT-treated group compared with the control group: 310 ± 192.68 days versus 154.9 ± 122.51 days, respectively, $P = 0.05$. Estimated median survival by Kaplan–Meier analysis was 225 days for ILBT-treated patients and 100 days for controls ($P = 0.025$). None of the patients developed major complications related to stent or ILBT treatment.

The authors call for a large, randomized, controlled trial to confirm the favorable findings of their pilot study.

Original article Singh V *et al.* (2007) Endoscopic intraluminal brachytherapy and metal stent in malignant hilar biliary obstruction: a pilot study. *Liver Int* **27:** 347–352

Dietary nitrates might have a gastroprotective effect

A high dietary nitrate intake has traditionally been linked with gastrointestinal cancer, but more published studies contradict than support this association. In humans, dietary nitrate (mostly from green leafy vegetables) is converted to nitrite in saliva, and to nitric oxide (NO) in the acidic stomach. Petersson and