globulin. Fasting serum glucose concentrations did not change significantly, but fasting insulin concentrations decreased and insulin sensitivity improved. Rosiglitazone treatment also improved endothelium-dependent vascular responses.

The authors conclude that rosiglitazone improves insulin sensitivity in women with PCOS. It helps to restore ovulation and decrease androgen production without significant adverse side effects. They suggest that reducing insulin resistance could modify cardiovascular risk in women with PCOS by improving endothelial dysfunction and low-grade chronic inflammation; but confirmation requires larger placebo-controlled studies.

Rebecca Ireland

Original article Tarkun *et al.* (2005) Effect of rosiglitazone on insulin resistance, C-reactive protein and endothelial function in non-obese young women with polycystic ovary syndrome. *Eur J Endocrinol* **153:** 115–121

GHRH antagonists: a novel therapy for non-Hodgkin's lymphoma

Growth-hormone-releasing hormone (GHRH) gene expression is deregulated in several hematologic malignancies. Furthermore, GHRH antagonists have antitumor effects in many cancer models. These are thought to be mediated indirectly by suppressing hepatic production of insulin-like growth factor I (IGF-I) or directly by actions on the tumor cells. Keller *et al.* evaluated whether GHRH antagonists can inhibit non-Hodgkin's lymphoma (NHL).

Athymic (nude) mice bearing xenografts of the human NHL cell lines, RL or HT, were treated with the GHRH antagonists, MZ-5-156 or MZ-J-7-138. As measured by tumor volume, this treatment inhibited tumor growth *in vivo* by up to 74%. A similar effect on proliferation was observed *in vitro* when NHL cell lines were exposed to the drugs, although the effect of MZ-5-156 was inferior to that of MZ-J-7-138 at lower concentrations.

Both cell lines expressed GHRH and the IGF-I-receptor. The GHRH-receptor splice variant 1 was also expressed, and provided a high-affinity binding site for the radiolabeled GHRH antagonist, JV-1-42. Treatment of nude mice with GHRH antagonists resulted in reduced levels of serum and liver IGF-I in the presence of MZ-5-156 but not MZ-J-7-138. In RL-derived and HT-derived tumors, both antagonists suppressed basic fibroblast growth factor, but had no effect on vascular endothelial growth factor.

The authors suggest that GHRH antagonists exert their antitumor affects directly, by GHRH receptor occupancy, and speculate that these drugs could offer a potential therapy for NHL.

Vicky Heath

Original article Keller G *et al.* (2005) Effective treatment of experimental human non-Hodgkin's lymphomas with antagonists of growth hormone-releasing hormone. *Proc Natl Acad Sci USA* **102:** 10628–10633

Hypocortisolism in survivors of SARS

After the outbreak of severe acute respiratory syndrome (SARS) in Singapore in March 2003, many survivors suffered from psychosomatic symptoms reminiscent of endocrinopathies. Leow *et al.*, therefore, aimed to find out whether any chronic endocrine conditions have occurred in this population as a result of SARS.

In this prospective study in Singapore, 61 survivors of SARS were recruited, approximately 3 months after their recovery. Patients who had an endocrine disorder before the occurrence of SARS were excluded. Blood samples were collected from each of the participants and analyzed for factors such as cortisol, adrenocorticotropic hormone, dehydroepiandrosterone sulphate, and free T_4 and T_3. Hormone measurement techniques such as immunochemiluminometric assay and radioimmunoassay were also carried out. In total, 24 patients were diagnosed with hypocortisolism, of whom 2 had thyrotoxicosis, 3 had central hypothyroidism and 1 had primary hypothyroidism. In 15 of the patients diagnosed with hypocortisolism, the hypothalamic–pituitary–adrenal axis dysfunction resolved completely within 1 year. Low levels of dehydroepiandrosterone sulphate were also detected in 8 patients.

The authors conclude that the hypothalamic–pituitary–thyroid and hypothalamic–pituitary–adrenal axes are targets of SARS-associated coronavirus, but *in vitro* studies, animal and clinical studies are needed to provide further explanation.

Marie Lofthouse

Original article Leow MKS *et al.* (2005) Hypocortisolism in survivors of severe acute respiratory syndrome (SARS). *Clin Endocrinol* **63:** 197–202