

## Introduction to Feline Acromegaly

Acromegaly in cats results from excess growth hormone (GH) secreted from an adenoma within the pituitary pars distalis. The anabolic effects of GH are mediated by insulin-like growth factor (IGF)-I (*somatomedin C*) and include an increase in body size, enlargement of the abdomen and head, weight gain and organomegaly of the heart, kidney, liver and tongue. Insulin antagonism occurs due to a GH-induced post-receptor defect in glucose transport leading to hyperinsulinism and a down regulation of insulin receptors. Acromegaly typically is seen in male DSHs, over 10 years old, with poorly controlled diabetes mellitus, or less commonly, neurological signs. Conformational alterations are insidious in onset and often not noted by owners. Occasionally weight loss is seen. Later clinical signs include degenerative arthropathies, congestive heart failure and renal failure. Pituitary tumours may cause local neurological damage resulting in stupor, somnolence, poor appetite, adipsia, temperature deregulation, circling, seizures, and changes in behaviour. Most of the laboratory test abnormalities in acromegaly are due to the diabetes: hyperglycaemia, glycosuria, hypercholesterolemia, and mild increases in ALT and ALP. Ketonuria is an infrequent finding. Persistent hyperphosphataemia without concurrent azotemia, mild erythrocytosis and a persistently raised total protein (*up to 95 g/L*) may also be found. Associated renal failure may result in azotemia, isosthenuria, and proteinuria. A tentative diagnosis of acromegaly may be made in a cat with insulin-resistant diabetes (*requiring > 2 iu/kg insulin*) and consistent conformational changes. Ideally other causes of insulin resistance are ruled out, a pituitary CT or MR scan is performed and an increased serum IGF-1 concentration is found (*GH measurement is not readily available commercially*). Some cases of acromegaly have IGF-I values in the high normal range.

Successful therapy for acromegaly has not been established in the cat. Potential treatments include radiation therapy, medical therapy (*Octreotide*), and hypophysectomy. The prognosis is guarded to poor with survival times of 4-42 months (*median, 21 months*). The tumour usually grows slowly-neurologic signs are uncommon until late in the course of the disorder, diabetes mellitus can usually be somewhat controlled with large doses of insulin given twice daily (*because insulin resistance often fluctuates, to avoid hypoglycaemia, insulin doses should not exceed 12 to 15 units per injection*) but most cats die from heart or renal failure.