Table 1. Summary of the main differences/relationships between glucocorticoids, androgens and estrogens

	Glucocorticoids	**Androgens**	**estrogens**
Active/less active forms	cortisol/cortisone *corticosterone/dehydrocorticosterone*	testosterone/androstenedione	estradiol/estrone
Main transporting protein in plasma	CBG	SHBG *(not in rodents)*	
Effects on body protein	wasting	increasing deposition and stores	protecting their integrity
Effects on carbohydrate metabolism	increase liver glucose output; increase glycemia; glycogen wasting		
Effects on lipid metabolism	increased overall lipogenesis from glucose and amino acids; enhanced lipid storage	limited lipid storage	decrease lipid storage; protection of lipids from oxidation
Effects on energy metabolism	favour lipids at the expense of carbohydrates and protein; alter the ponderostat setting	increase thermogenesis	increase thermogenesis; precursors of postulated ponderostat signal
Effects on mineral deposition in bone	mobilization (up to osteoporosis)	maintenance / retention	
Actions on inflammation	decrease (cytokine inhibition); synergistic effect with estrogens	decrease?	decrease (antioxidant); synergistic effect with glucocorticoids
Effects on the immune system	depress	enhance (protein/energy availability)	
Effects on insulin	induce insulin resistance	synergistic effects with insulin favouring protein deposition and growth	counteract glucocorticoid effects on insulin
Effects on steroid hormone synthesis and function	strongly inhibit androgen synthesis and actions; also (less strongly) estrogen action	block some glucocorticoid effects; DHEA is an antiglucocorticoid	decrease androgens (substrate for their synthesis); block some glucocorticoid effects

CBG: corticosteroid-binding globulin; DHEA: dehydroepiandrosterone; SHBG: sex hormone-binding globulin.