GENERAL CONSIDERATIONS

It was as early as in 1563 that Bartolomeo Eustacchio first described the adrenal gland which he termed glandulae Renibus incumbentes although his Tabulae Anatomicae were left unpublished at his death. In the years to follow, adrenal glands unlike gonads were left undisturbed by the bothersome investigations of medical men. It was only a century later that the distinction between the adrenal cortex and the medulla was recognized by the early anatomists, such as Casper Bartholinus the Elder (1611) who described the adrenal gland as Capsule atrabilariae, a capsule filled with black fluid (this was due to rapid post-mortem autolysis of the medulla).

The beginning of any real scientific history of the adrenal has to be dated from the concise and admirably written monography by Thomas Addison in 1855. Historically, our understanding of adrenal function began with the British physician Addison's description in 1855 of a disease that now bears his name Addison's syndrome which is now recognised as generalised insufficiency of adrenocortical function and was associated with lesions in adrenal glands. Although the discovery of the adrenal gland dates back to the 16th century, meaningful work regarding the functional status of adrenal

gland was assessed and, consensus achieved for the fact that adrenal cortex was essential for life only by 18th century. To examine adrenal function experimentally, adrenalectomy was attempted by Brown Sequard in 1856. The great rejuvenator found that death followed upon removal of both adrenal glands or even one gland and concluded that adrenals were essential for life. It seems more likely that the animals died from surgical trauma rather than from adrenal insufficiency.

It is still enigmatic as to how the two type of tissues having an all together separate embryological origin have come together to lie in such close association so much so that it is sometimes mistaken to be one single entity. The cortical portion is the first to be differentiated embryologically. The primordia bud off from the coelomic epithelium, ventrally and medially to the mesonephros. Soon after their formation, the prospective cortical cells leave the epithelium, and move dorsally to form paired masses of scattered cell groups in the mesenchyme on each side of the aorta. During their further development, the cortical cells become arranged in cords, and blood cells and vessels appear between the cell cords. Medullary (chromaffin) cells, appear several days later,

arising from the primordium of the sympathetic nervous system. These cells migrate singly, ventrally between the aorta and, groups of cortical masses collect and tend to arrange themselves in cords between the cortical tissue, before separating into groups of variable size around blood vessels (Romanoff, 1960)

The name adrenal or suprarenal, implies a close anatomical and embroyonic relation with the kidneys. In birds and mammals the adrenals are located bilaterally at the anterior poles of metanephric kidneys. In teleost fishes, the anatomic association is more intimate and the adrenal tissues are embedded in the renal structure. In forms such as elasmobranch fishes and the anuran amphibians, the adrenal tissues are often found outside the elongated kidney masses and may themselves be elongate structures on the surface of the adult mesonephric kidneys. In reptiles, the adrenals are located anterior to the kidneys often at little distance from the adult mesonephric kidney.

In general, it is difficult to attribute any functional significance to the close association between interrenal and chromaffin tissues. Recently, it has been found that adrenaline stimulates the 17 and 21 hydroxylation of progesterone in bovine adrenal preparation. Chromaffin may affect

steroid biosynthesis by interrenal tissue. In mammals, Benteley (1974) pointed out that adrenaline causes constriction of medullary arteries and a diversion of blood into the cortical sinusoids, allowing an increased output of cortical hormones. This may be the significance of morphologic association, except among teleosts, where most of the blood supply is from the renal portal veins.

Number of experiments with mammalian ACTH or avian pituitary extracts have suggested the occurrence of a classic pituitary-adreno-corticotropic control scheme in birds as in other vertebrates; this control differing in some respects from that of mammals, as the level of adrenal-cortical function of hypophysectomized birds differs from that in hypophysectomized mammals. Whereas in the latter, hypophysectomy leads to a drastic atrophy of the adrenals, and an associated fall of 70-80% in plasma corticosterone, in hypophysectomized birds, adrenal atrophy is variable and moreover, the plasma corticosterone levels remain at about 40-60% of the control. This could be as a result of lowered metabolic clearance rate as observed in hypophysectomized ducks which exhibited an 83% increment in the half life of corticosterone and a correlative 42% reduction in metabolic clearance rate (Bayle et al., 1971). However a 50% increase in the half

life of corticosterone in hypophysectomized rats has also been reported (Miahle-voloss et al., 1965). Thus the obvious discrepancy in plasma corticosterone in either birds or mammals may more likely be due to higher residual corticosterone secretion rather than to some quantitative difference in peripheral metabolism of the hormone. To account for these differences, two theories have been proposed (1) occurrence of an autonomous adrenal cortex secretory activity, independent of pituitary ACTH (Newcomer, 1959; Nagra et al., 1963) and (2) the possible action of an extrahypophyseal corticotropic substance, either ACTH or an ACTH analogue (Miller, 1961; Resko et al., 1964; Frankel et al., 1967a,b; Peczely, 1969). In the case of pigeons, ACTH-like activity has been demonstrated in the median eminence of the hypothalamus. Peczely et al. (1970) have shown that this ACTH like activity in the median eminence of the pigeon, 1 month after hypophysectomy, is still as high as in the control animals. Furthermore, hypophysectomized pigeons respond to formalin stress by morphological activation of cortical tissue. On the other hand, Frankel et al. (1967) have recorded in hypophysectomized fowl a drop in plasma corticosteroid to nearly zero after injection of dexamethasone. It is therefore suggested that in the studies on the effect of adrenal corticosteroids on general body metabolism as well as on the

reproductive physiology of any species, care must be exercised in interpreting the data as the results could be due to alterations in the circulating titer of the hormone rather than due to complete abolishment of the function of the gland. It would therefore be advisable to use dxm or other synthetic corticosteroid known to inhibit ACTH synthesis, thereby rendering the animal depleted of corticosterone to evaluate the adrenal cortical function.

The occurrence of diurnal fluctuations in adrenal-cortical function as revealed by plasma or urinary corticosteroids has been extensively investigated in the Japanese quail. Quails reared in isothermic rooms but under natural light conditions, exhibited a marked daily rhythm for both adrenal-cortical paramaters studied, very similar to that of diurnal mammals, with a steep increase during the second half of the night, a peak towards the end of the night hours, and a progressive lowering during day hours. Chan and Phillips (1970, 1973a,b) found peaks in the rate of <u>in vitro</u> synthesis of aldosterone and corticosterone at dawn and dusk. Further, Boissin and Assenmacher (1968) have reported the corticosterone content of adrenal glands and peripheral blood plasma to exhibit circardian rhythm; plasma corticosterone being maximum at the end of the dark period (5.00 hrs),

equivalent to 5µg/100ml and minimum at 21.00 hrs. equivalent to 1/4th of the maximum value. However, Joseph and Meier (1973) reported that pigeons maintained on daily 8:16 hours photoperiodic schedule exhibited increased plasma corticosterone content few hours after the onset of the dark period, reaching peak in 4-8 hrs. During the light hours, a gradual reduction in concentration was observed except for a secondary peak late in the day. However, the rhythmicity is lost in pigeons kept in continuous light for 15 days. Jones (1974) reported the existence of circardian rhythm in the ability of natural and synthetic corticosterone to suppress ACTH secretion. In man and rat, maximum inhibitory effect is exerted when these substances are administered 4-8 hrs. before the circadian peak. There is therefore a sound argument for restricting corticosterone administration in the morning thereby limiting the dangers of adrenal suppression. Thus for experiments involving pituitary ACTH, late morning injections of the synthetic drug will be most effective as it would diminish the secondary peak during the day as well as the next surges on continued administrations of dexamethasone.

Apart from this diurnal rhythmicity, in the past years, a number of investigators have claimed the occurrence of seasonal variations in histomorphology of the avian adrenal. In most species, visible histological activation of adrenal

cortical tissue has been noticed during the breeding season e.g., in the duck (Hohn, 1947; Phillips and Van Tienhoven, 1960; Höhn et al., 1965) in the hen (Riddle, 1923; Legait and Legait, 1959), the brown pelican (Knouff and Hartman, 1951), the European Starling, Sturnus vulgaris (Burger, 1938), the European Black bird, Turdus merula (Fromme-Bouman, 1962), and the house sparrow, Passer domesticus (Bhattacharyya and Ghosh, 1965; Moens and Coessens, 1970). However in the duck, a second period of histological activation has been observed during the fall and winter (Höhn et al., 1965). On the other hand, Lorenzer and Farner (1964) observed in the white crowned sparrow, Zonatrichia leucophrys that the adrenal-cortical tissue showed maximum activity during the quiescent phase of testicular cycle and vice versa. Adrenalectomy has been recorded to induce pronounced atrophy of the testis (Henrick and Finerty 1941; Hewitt, 1947) and to inhibit the development of right gonad in overiectomised chickens (Taber et al., 1956). On the other hand, testicular atrophy following the administration of cortisone acetate and depression of egg laying by corticosterone administration have been reported in chickens by Selye and Friedman (1941) and Flickenger (1966) respectively. An androgenic effect of deoxycorticosterone acetate on comb size and stimulation of testicular functions have also been reported (Hooker and Collins, 1940; Boas, 1958). However, Bhattacharyya and Ghosh (1970) have reported

induction of pathomorphic changes in the reproductive system of pigeons under conditions of hyper and hypo adrenocorticalism. These conflicting reports have provided the motivation to undertake the present study on Indian subtropical feral blue rock pigeons, Columba livia. The results accrued have thrown light on the importance of adrenal corticosteroids as a positive modulator of gonadal cyclicity and functioning. The importance of adrenal steroids in gonadal functioning in mammals has also been reported. It has been suggested that a slow rise in progesterone level in the plasma preceeding the LH surge might be due to adrenal secretion and that the adrenal source of progesterone could be important for determining some of the manifestations of reproductive behaviour (Davson and Segal, 1980). Infact, based on their work in a four day cycling rat, Mann and Barraclough (1973) concluded that the morning peak of progesterone secreted by the adrenals might as well be the trigger for the preovulatory rise in LH secretion. Feder et al. (1971) have shown in this connection that the slow rise in plasma progesterone was associated with the corresponding rise in corticosterone and that when the animal was dosed with dxm, ovulation was blocked which could be reversed by administration of either ACTH or progesterone. Though the role of adrenal progesterone to act as a final trigger for LH surge must remain an open question, injection of ACTH into oestrogen primed ovariectomised rats could

induce mating behaviour (Feder and Ruf, 1969; Davson and Segal, 1980), whilst adrenalectomy was shown to delay lordosis (Nequin and Schwartz, 1971). On the other hand, Davidson et al. (1968) found that the oestrogen induced lordosis occurred in ovariectomised rats in spite of adrenalectomy. Most of these reports reviewed and, the findings of the present work taken together emphasize the effectiveness of adrenocortical hormones in general on reproductive physiology of higher vertebrates to a greater or lesser extent. Although, three types of correlations between adrenocortical cycle and breeding activities i.e. positive, negative or indifferent, could be considered feasible, these correlations pin point the species specific interactions between the two glands. The present study has also revealed a parallel adrenal-gonad axis with the adreno-cortical activity exhibiting parallel increase along with the activation of gonads. Increased adrenocortical activity remains operative through out the breeding phase. Experimental manipulation of adrenocortical activity in the form of suppression during its active phases and activation during its reduced activity phase has induced remarkable gravimetric changes in various organs, histological alterations in gonads, adrenal and thyroid and, biochemical changes in liver, adrenal and gonads indicating the importance of corticosteroids in the various

seasonal modulations associated with breeding. Although the exact mode of action of adrenal steroids is still obscure, the present study has revealed a parallel adrenal-gonad and inverse thyroid-gonad axes to be operative in wild pigeons based on gravimetric changes and histological features of the three glands. Literature scan on birds shows reports indicating both parallel as well as inverse adrenal-gonad interrelationships (Höhn, 1947; Knouff and Hartman, 1951; Legait and Legait, 1959; Lorenzen and Farner, 1964; Bhattacharyya and Ghosh, 1965; Moens and Coessens, 1970; Bengt, 1975). Similarly, synergistic and antagonistic interrelationships between the thyroid and gonads have also been reported (Jaap, 1933; Aron and Benoit, 1934; Woitkewitch, 1940; Vaugien, 1954; Thapliyal and Garg, 1969; Jallageas and Assenmacher, 1974). However, the present study has shown the thyroid-gonad relationship to be more precisely antagonistic and has also been inferred so by some previous workers from this laboratory (Patel et al., 1985). An apparent inverse thyroid-adrenal axis can also be presumed from the present study. Based on the reported reciprocal relationship between steroids and lymphoid organs (Glick, 1967; Glick and Dreesen, 1967; Glick, 1972), and the status of uropygial gland as a target organ for steroids (Gupta and Maiti, 1983; Amet et al., 1983), the currently observed loss in weight of spleen and increased weight of uropygium during the

breeding season or under the influence of ACTH/corticosterone during the non-breeding season are understandable. The favourable influence of adrenal steroids on functional status of gonads is clearly indicated by the observed gonadal regression in dexamethasonised birds during the breeding phase and the gonadal enlargement with histologically visible signs of functional recovery induced by administration of ACTH/corticosterone during the non-breeding phase. Though gonadal enlargement and signs of recovery of gametogenic function were clearly noticeable in ACTH/corticosterone treated birds, full spermatogenic activity as marked by appearance of spermatids and spermatozoa or formation of fully mature ova could not be achieved. However, it has to be recalled that the schedule of hormonal treatment was of a short duration of ten days and as such may be insufficient to activate the gonads completely, from a fully regressed slumberous state. It would be therefore interesting to test the possibility of establishing complete gametogenic activity in the quiescent gonads by administering ACTH/corticosterone for a slightly more prolonged period of time. The fallacy of increasing the dose is well exemplified by the herein observed reduced activational changes in the testes with a higher dose of corticosterone. However, a probable sexual dimorphism in this respect is indicated by the more significant changes in ovarian weight obtained herein with the higher dose of

corticosterone. Moreover, a diurnal specificity is also indicated by the observed relatively better effectiveness of the morning schedule of corticosterone treatment as compared to the evening treatment.

The effects of glucocorticoids are more wide spread than those of other steroid hormones in terms of diversity of effects produced and the number of tissues affected. These effects are reviewed by Schulster et al. (1976) and, their mode of actions by King (1976). Riddle (1937) was the first to show the hyperglycaemic effects of adrenal extracts on hypophysectomised, thyroidectomised or partially adrenalectomised pigeons. Later, several others demonstrated hyperglycaemia and hepatic glycogenic effects of cortisone and hydrocortisone in the domestic fowl (Golden and Long, 1942; Stamler et al., 1954; Brown et al., 1954; Brown et al., 1958; Greenman and Zarrow, 1961; Snedecor et al., 1963). Glucocorticoids oppose the action of insulin and tend to increase the concentration of glucose in the blood; the increase in the glucose concentration occurs due to gluconeogensis in which precursors of glucose are made available to liver from muscle and adipose tissue coupled with an increased amino transfer (Wicks, 1974). An adequate supply of carbohydrates specifically glucose is considered to be an essential requirement for gonadal functioning. Due to the generally

low store of carbohydrates and their uneven distribution, the testis tissue is probably dependent on a constant exogenous supply. In this light, the presently observed gonadal shrinkage in adrenal suppressed birds during the reproductively active phases could be due to the hypoglycaemic condition induced by adrenocortical insufficiency. In this context, the report of Mancine et al, (1960) who observed extensive injury to the germinal epithelium, congestion and oedema in the interstitium and vacuolization of sertoli cells in adult rats after insulin induced hypoglycaemia is relevant. Along the same lines, Warren and Lecompte (1952) have also reported atrophic changes in the testis of untreated Diabetic Mellitus individuals due to inefficient utilization of blood glucose. Both these reports tend to suggest the definite influence of blood glucose on gonadal functioning. The lowered blood glucose level observed during the non-breeding season in the present study corresponding to regression of the gonads may have some relevance in this perspective. This is further emphasized by the recorded elevation in blood glucose level along with gonadal enlargement in ACTH/corticosterone treated birds in the non-breeding season.

The effects of glucocorticoids on intermediary metabolism and growth are multifaceted and very often

Chickens. The glucocorticoids act by increasing the flow of amino acids to the liver and also stimulate gluconeogenesis at relatively slower rate than by glucagon or catecholamines. Changes in gluconeogenesis are noted to be very important in the metabolic derangement occurring in diabetes and disorders associated with glucocorticoid secretion (Exton, 1972). In this behest, the importance of adrenocortical activity to bring about the right changes in general body physiology so as to create a correct " milieu internae" required for gonadal functioning cannot be overlooked and as such related favourable changes have been observed in the present study.

Glucocorticoids profoundly exert influence on protein metabolism to a greater or lesser extent. Despite a significant hyperphagia, corticosterone or hydrocortisone treated chikens exhibited marked inhibition of growth (Dulin, 1956; Baum and Meyer, 1960 : Nagra and Meyer, 1963). Further, stimulation of increased adreno-cortical activity in adult drakes caused them to lose body weight. Although treatment of 6-7 weeksold cockerels with corticosterone stimulated them to eat more food; their rate of growth was still retarded (Nagra and Meyer, 1963). But, even when the administration of corticosterone did not affect the growth

rate, the disposition of protein, glycogen and lipids in the body were changed significantly compared to that of untreated birds (Nagra and Meyer, 1963). Changes in body weight and body protein suggest that an overall negative nitrogen balance occurs in the presence of high circulating levels of corticosteroids. The present study examplifies an inverse relationship of tissue protein content with that of breeding cycle. Recrudescent and breeding phases were marked by reduced protein content while the inverse relationship holds true during the non-breeding phase. The catabolic effects of glucocorticoids are further emphasized by the observed decrease in protein content in liver, muscle and gonads coupled with increased transaminase activity together with increased gluconeogenesis during gonadal activity. The quiescent phase of gonads is marked by increased protein content as a result of decreased catabolic activity together with low activity levels of transaminases and gluconeogenic enzyme, which could be explained on the basis of lowered adreno-cortical activity. The observations of Nagra and Meyer (1963) of decreased rate of transformation of injected (14_C) glucose into proteins in growing chickens after corticosterone treatment is worth mentioning. The role of glucocorticoids in accelerating protein breakdown could be a necessary factor for optimal gametogenic activity as the

process necessarily involves synthesis of many proteins requiring a free pool of amino acids. Incidentally, the report of Setchell et al. (1967) of increased concentration of glutamate, glutamine, glycine, alanine and aspartate in the testicular fluid of ram suggesting an especially favourable environment for nucleic acid synthesis gains credence. The similar changes observed in the hepatic tissue seems to further underscore the increased demand for amino acids in relation to gonadal functioning and also as substrate for increased gluconeogenic activity in the liver observable during the same period. Loss of these functional modulations in adrenal suppressed birds may therefore be responsible for the loss of functional capacity of the gonads and further elucidates the role of corticosteroids in supporting a normal process of oogenesis and spermatogenesis.

The glucocorticoids on the other hand accelerate the hepatic uptake of amino acids produced as a result of its catabolic action in the peripheral tissues for the synthesis of albumin. Apart from this, some of the amino acids are deaminated to form urea and substrates for energy metabolism. The basic feature of the approach of breeding season is the increased demand of energy placed upon the body. To meet this increased demand the metabolic activities of the tissues are geared up as exemplified by

increased SDH and LDH activities in liver and gonads during the breeding phase. The increased energy expenditure is revealed by increased ATPase activity in the tissues during the same phase. All these enzymes depicted a low profile of activity during the non-breeding phase. The activity pattern of the enzymes closely parallels the adreno-cortical activity. The relative importance of steroids elaborated by adrenals becomes more clear from the observations of low levels of enzyme activity in the adrenal suppressed birds. On the other hand, birds administered with ACTH/corticosterone revealed significantly increased enzymic activity corresponding to the reawakening of gonads. These observations point towards the functional involvement of glucocorticoids by regulating enzyme systems and the resultant effect on reproductive activity. Uncompensated adrenalectomy causing a reduction in ATPase activity of the intestine has been reported by Luthy and Verzar (1954). Administration of DOCA and cortisone restored the activity within 24 hrs. The decrease in ATPase activity may be one of the causes of reduced metabolic and transfer functions displayed by enterocytes in uncompensated adrenalectomy. The same argument may hold true in the present case too. However, it is enigmatic as to whether these changes resulted in gonadal inactivation (which seems to be the correct reasoning) or the inactivation of gonad due to the adrenal

manipulation led to these changes. Both SDH as well LDH activity have been reported to undergo alterations in the rat jejunum and colon under the influence of ACTH and cortisone in the form of decreased activity as reported by Kilkowska and Kozlowska (1969). These studies definitely reveal the effects of glucocorticoids on reactions of intermediary metabolism.

Lipids too have been shown to undergo seasonal fluctuations in several tissues associated with breeding cycle (Patel, 1982). As the adrenal also depicted seasonal alterations in its functional status, and as adrenal steroids profoundly affect the gonadal functioning, it was thought pertinent to follow up the changes in lipid contents in the adrenals and gonads of normal and adrenal manipulated birds on a seasonal basis. Seasonally breeding vertebrates depict a season specific lipid-cholesterol cycle in the form of decreased lipid content during the active phase of gonads and its subsequent increase during the quiescent phase (Johnson, 1970; Lofts and Lam, 1973; Skinner et al., 1973; Mcpherson and Marion, 1982). Similar trend of changes have been noted to occur in the present study. The striking feature of the present study is that, although the alterations in the total cholesterol content of adrenals depicted marginal fluctuations, the cholesterol ester content

was found to undergo significant fluctuations in association with adrenal functioning. Lipid fractions and total lipid content tended to be high in the inactive gonads and low in the active gonads, except for free fatty acids which tended to exhibit a reverse set of changes. The reports of Hafiez and Bartke (1972) of hypophysectomy induced increase in cholesterol and cholesterol ester contents in rats and mice and the reports of Johnson (1970) of a similar trend in lipid fractions (phospholipid, cholesterol and triglyceride) under the influence of hypophysectomy in the gonads of birds is worth mentioning. Further, Mukherjee and Bhattacharyya (1982) too reported alterations in total free and esterified cholesterol with respect to breeding cycle in Channa punctatus. Whether these effects of adrenal steroids on gonadal lipid content are a direct one or are manifested through the hypothalamo-hypophysial axis is as yet a problem to be resolved and needs further investigative work. However, whatever be the mode or the route of actions, the present study does underscore the relative importance of the adrenal cortical secretions in gonadal physiology.

Like in mammals, the kidney is a major target organ for corticosteroids and, aldosterone is equally potent in causing Na^+ retention in aves. The predominant

physiological effects of adrenal steroids found in birds do not permit them to be classified into discrete categories such as"glucocorticoid" and "mineralocorticoid." This is particularly true with respect to the control of electrolyte balance in birds. Adrenalectomy in ducks causes increased sodium loss in the urine, while large doses of aldosterone or corticosterone reduce sodium loss (Phillips et al., 1961). High K^+ content and low Na^+ content in tissues have been generally associated with tissue functioning and the currently observed high K^+ content and low Na^+ content in gonads during the recrudescent and breeding phases and the reversed state during the non-breeding phase seem to favour this concept. These changes in tissue ionic contents were accompanied by definite changes in serum ionic contents as well. Moreover, the experiments on adrenal manipulation could effectively mimic these changes in tissue and serum ionic contents. Apparently, corticosterone seems to have the ability to induce seasonal alterations in tissue and serum ionic contents in relation to gonadal recrudescence and quiescence either in a cause or effect mode.

The depletion of ascorbic acid (AA) from mammalian adrenocortical tissue has long been considered as a specific

index of stimulation by corticotrophin and is still widely used as a bioassay for corticotrophic activity. Studies on the immature and adult intact chicken (Jailer and Boas, 1950; Elton and Zarrow, 1955) and isolated studies on the intact bob-white Quail, (Zarrow and Baldini, 1952) have failed to show any depletion of adrenal ascorbic acid following cold stress or treatment with ACTH. Subsequently, it was shown that injections of ACTH did cause a depletion of adrenal ascorbic acid in adult chicken but not in the immature birds. Further, it has now been shown that the maxmimal depletion of adrenal AA from the adrenals of immature chickens exposed to stress or injected with ACTH occurs within 20 minutes and repletion is complete within one hour (Freeman, 1967; 1969). Hence, earlier failures to detect this response in immature chickens may have been due to the fact that the adrenal AA concentrations were not measured until atleast one hour after the onset of stress or the injection of ACTH by which time depletion and repletion must have both occurred. Clearly, the phenomenon of adrenal AA depletion should now be re-investigated, since, age, sexual maturity and pituitary activity at the time of experiment and, the mode and frequency of ACTH administration can all significantly affect what may be an extremely

rapid response. Further, the degree of AA depletion obtainable in birds seems to be much less than in the rats subjected to similar cold stress or treatment with ACTH (Sayers and Sayers, 1948; Greenman et al.,1967). This relative insensitvity alone may mitigate against the use of this index as a reliable measure of adrenocortical function. However, the depletion of adrenal AA along with other parameters can reliably indicate the functional status of the gland. If the experimental conditions are kept relatively constant the changes in the AA content would surely give us some idea about the prevailing levels of corticosterone and the functional aspect of the gland itself. In the present study, breeding phase was marked by maximal adrenal-cortical activity and reduced AA content which got further depleted in the regression phase and then increased to the maximal level during the recrudescence phase. These changes could denote increased mobilisation and/or synthesis between regression and recrudescence; in which case the reduced AA content during regression can be considered to be due to both decreased utilization as well as decreased mobilization in keeping with the reduced adreno-cortical activity. In this light, adrenal suppression(during recrudescence and breeding) can cause stockpiling of adrenal AA due to the sudden arrest in utilisation, while ACTH or corticosterone administration during the regression phase can bring about

a further depletion in adrenal AA content due to a sudden stimulation of utilization without any increase in mobilization, changes which were actually noticed during the present course of study. It is however interesting, that high corticosterone dose elicited an increased adrenal AA content, which could presumably be due to inhibition of endogenous ACTH release.

Another main steroidogenic center of vertebrates is the gonads, and the depletion of gonadal AA content is considered to be a major criterion in judging the functional status of the gonads. AA content in relation to gonadal functioning has been reported by many workers (Biswas, 1969; Prabhakar et al., 1975; Chand et al., 1978). The present study has noted definite alterations in gonadal AA content during its annual cyclicity. The changes are indicative of reduced AA content in conjunction with gonadal functioning and increased AA content in association with gonadal quiescence. These normal seasonal alterations could be mimicked by ACTH/corticosterone administration during the regression phase or by adrenocortical suppression by dxm in the recrudescent and breeding phases respectively, thereby suggesting the involvement of corticosterone either directly or indirectly in inducing gonadal recrudescence. Specific alterations in hepatic AA content as well, noted under

altered functional status of the adrenals, is suggestive of the modulatory influence of corticosterone in regulating tissue AA turnover on a seasonal basis.

In conclusion, the conundrum of changes when reviewed as a whole does project certain salient features. The most outstanding feature that gets established is the existence of a parallel adrenal- gonad axis in feral blue rock pigeons, Columba livia. Adrenal manipulation in the form of suppression during its active phases and activation in its relatively inactive phase led to definite alterations in histomorphology and bio-chemistry of the gonads in both the sexes. Although the axis as such has gained validity, the mode of action and the type of inter-relationship between adrenal and gonad are still obscure. It is quite likely that corticosterone may exert a short loop action in the sense that it may have a direct action on the gonads especially in the light of reported presence of glucocorticoid receptors in the gonads (Ballard et al., 1974) or may have a long loop action via the hypothalamo-hypophysial axis involving the gonadotrophins. If the latter be true, it would be logical to identify the gonadotrophic component (FSH or LH) that could be regulated by corticosterone. Further work involving hypophysectomised birds