free lipid fraction is lost during alcohol and acetone treatments.

In moribund parathion- and carbamate-treated bugs a few, and in endrin-treated bugs most of the cells of the pars intercerebralis lack SBB and AH positive material. The increased level of SBB and AH positive material in the corpora cardiaca and allata coincides with the degree of its loss from the pars intercerebralis. Since more of the material accumulates in endrin-treated bugs than in parathion- and carbamate-treated ones, it appears that the degree of release of this material is dependent upon the nature and chemical composition of the insecticides.

In roaches under electrically induced stress, Hodgson and Geldaiy[11] reported an insignificant change of AF positive material in the cells of the pars intercerebralis and its decrease in the corpora cardiaca. In the light of the observations of Hodgson and Geldaiy[11] and of our observations, it appears that the proteins and lipids represent 2 fractions of brain hormone and behave independently under different conditions. The release of SBB and AH positive material from neurosecretory cells might be a response to stress situation and may be involved in some way in stimulating the corpora cardiaca and allata for the release of their neuroactive hormones in the blood; high titers of these have been estimated by Davey[12,13], Karter[14] and Colhoun[15] after either stimulating roaches electrically or treating them with DDT and TEPP.

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Influence of somatostatin on serum prolactin concentrations of cows during rest and milking

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Summary. Administration of somatostatin (SRIF) to lactating cows significantly increased basal, premilking serum concentrations of prolactin (PRL), and potentiated PRL release in response to milking and significantly reduced resting concentrations of growth hormone (GH).

Brazeau et al.[2] reported that purified extracts from sheep hypothalami inhibited GH release in rats. A peptide was isolated from these extracts, purified and subsequently named somatostatin (SRIF). SRIF inhibits basal concentrations of GH, as well as GH release in response to a variety of stimuli[3]. SRIF does not influence basal concentrations of PRL in man, but in patients displaying pathologically high levels of GH and PRL, SRIF appears to suppress both hormones[3]. Gala et al.[4] demonstrated that SRIF did not block PRL release in monkeys induced by perphenazine, TRH or serotonin. In fact, SRIF potentiated PRL release induced by perphenazine and TRH, but not that induced by serotonin.

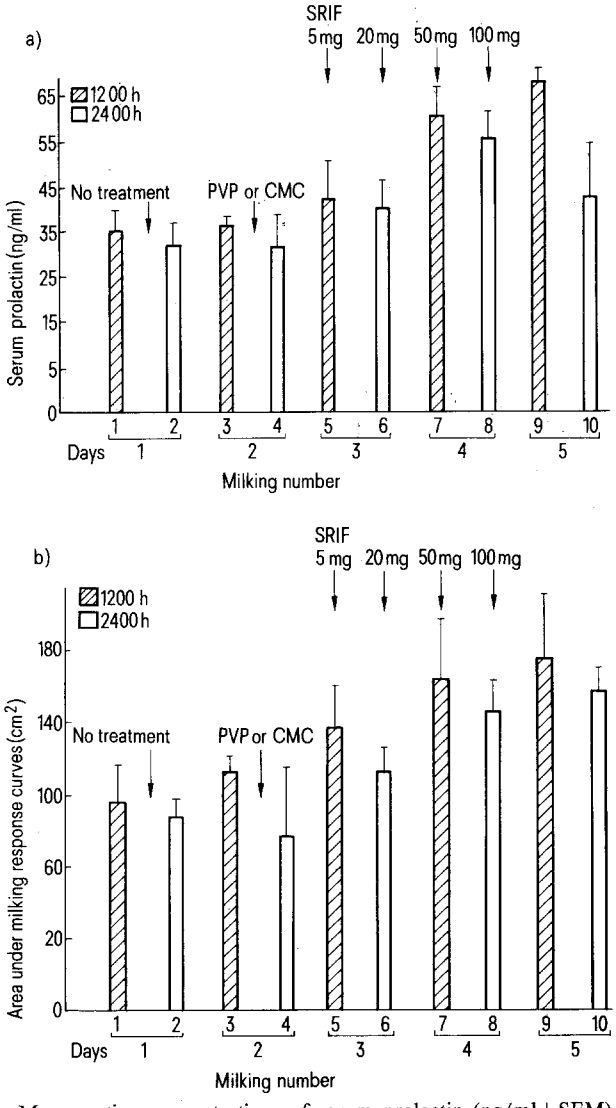
PRL is believed to play an important role in mammary development and milk secretion. Milking or suckling is a potent natural stimulating mechanism for the release of PRL in cows and other lactating animals. We therefore investigated whether SRIF would alter basal serum concentrations of PRL in cows and the milking-induced rise in hormone normally observed in these animals. Changes in resting concentrations of GH after SRIF treatment were also noted.

Serum growth hormone (ng/ml ± SEM)

Milking	No treatment (bleeding)	PVP or	CMC	SRIF* (mg)				No treatment	
				5	20	50	100		
1**	8.5 ± 1.1								
2	6.8 ± 0.8								
3		7.5 ± 1.2							
4			6.9 ± 2.0						
5				6.0 ± 0.6					
6					5.5 ± 0.8				
7						3.0 ± 0.7			
8							1.8 ± 0.5		
9								5.8 ± 0.2	
10									8.0 ± 0.2

* Mean serum concentrations of GH were significantly ($p < 0.05$) reduced when compared with mean serum concentrations of GH after injection of saline, PVP or CMC, or no treatment ($n = 4$).

** Odd numbers i.e., 1, 3, 5, etc. indicate that milking was carried out at 12.00 h. Even numbers indicate that milking was carried out at 24.00 h.



a Mean resting concentrations of serum prolactin (ng/ml ± SEM) for 4 cows analyzed at 30 and 10 min before milking over 5 days. SRIF significantly ($p < 0.05$) elevated resting concentrations of prolactin when overall means were analyzed and compared with controls. Doses of SRIF did not significantly ($p > 0.05$) differ from one another with regard to their effect on PRL release. *b* The area (cm^2) under the prolactin response curves for 5 days during the milking interval, which consisted of the following sampling times: 0 (start of milking), 5 min after commencement of milking and at 10 and 30 min after commencement of milking. SRIF significantly ($p < 0.05$) affected the response of prolactin to milking stimuli. Doses of SRIF did not significantly ($p > 0.05$) differ from one another with regard to their effect on PRL release during the milking interval.

Materials and methods. 4 lactating Holstein cows (3 years of age) entering their 2nd lactation, were fitted with indwelling jugular cannulae. Blood samples were collected and discarded at 15-min intervals for 2–3 h before, during and after 2 milkings (12 h apart) to acclimate animals to the experimental routine. On day 1 of experimentation, blood samples were collected and saved for assays at −30, −10, −5, 0 (milking), +5, +10, +30, +60, +120, +240, +360, and +480 min around 2 milkings over 24 h. On day 2, 2 cows were given a 5.0-ml s.c. injection of 0.85% saline in polyvinyl pyrollidone (saline: PVP, 50v/50v) and the other 2 cows were given a 5.0-ml s.c. injection of 0.85% saline in carboxymethyl cellulose (saline: CMC, 50v/50v) 30 min prior to each of the 2 milkings. Blood sampling continued as above for 24 h. At 12.00 h of day 3, 30 min prior to milking, all cows received a s.c. injection of 5 mg of SRIF in either PVP or CMC. PVP and CMC were used in order to determine which vehicle might be most effective in delivering SRIF. On 24.00 h of day 3, 30 min prior to milking, all cows received 20 mg SRIF. 30 min prior to milking, at 12.00 h of day 4, all cows received 50 mg SRIF, as above. On 24.00 h of day 4, 30 min prior to milking, cows received 100 mg SRIF. On day 5, blood samples were collected through 2 milkings as described above, but no treatments were given. Blood samples (10 ml) were allowed to clot in ice and serum concentrations of PRL and GH were quantified by radioimmunoassay[5,6]. Data from all cows were pooled with vehicles for statistical analyses. Baseline concentrations of PRL and GH were quantified for samples before milking (−30 and −10 min). These data were subjected to analysis of variance to determine the significance of treatments. The milking interval included the following sampling times: 0 (start of milking), +5 (end of milking), +10 and +30 min after milking. Optimum PRL release occured during this interval, peaked from +5 to +10 min and was at baseline concentrations +160 min post milking. The concentration of PRL in ng/ml for all animals was plotted against the times stated above for each day and each milking. The area under each of these graphs was then integrated, using a planimeter, giving a single value for serum PRL released in response to milking for each day and each milking. These values were used to calculate a mean value for all animals for the response of PRL to milking. These means were used in an analysis of variance to determine the significance of treatments.

Results. Serum PRL concentrations are shown before each of a total of 10 milkings for 5 days in the figure, a. SRIF significantly increased premilking concentrations of serum PRL from an average of 31.3 ± 5.1 ng/ml, pretreatment, to 53 ± 6.2 ng/ml during SRIF treatment. Irrespective of the applied treatments, baseline concentrations of PRL were higher during the day when compared with night samples. SRIF did not block the milking induced rise in serum PRL, but rather appeared to potentiate the response (figure, b). The area under the milking response curve for PRL averaged 95 ± 11 cm^2 pretreatment and increased to an average of 145 ± 14 cm^2 after SRIF treatment (figure, b).

Serum GH was assayed in all samples in order to test the biological effectiveness of SRIF on inhibiting GH release. The table shows mean basal serum GH concentrations in 4 cows after PVP or CMC treatment, SRIF treatment and no treatment. GH concentrations in sera were significantly reduced from an average concentration of 7.65 ng/ml and 7.2 ng/ml after bleeding and PVP or CMC injection, respectively to an average of 3.8 ng/ml during SRIF administration. GH concentrations rebounded to an average of 6.1 ng/ml after the last injection of SRIF. GH concentrations were slightly higher at 12.00 h when compared to concentrations at 24.00 h, irrespective of the applied treatment, except for the last 2 milkings (table).

Discussion. Baseline concentrations of serum PRL were elevated in all cows while under the influence of SRIF. To the author's knowledge, this is the first report that SRIF is capable of increasing basal PRL levels in cows. Consistent with the observations of Chen et al.[7], obtained in rats, the suckling-induced release of PRL was not blocked by SRIF, even despite GH suppression. The present study indicates potentiation of milking-induced PRL secretion in cattle by SRIF. Swano et al.[8] observed that chlorpromazine-induced increases in serum PRL of rats were not blocked by SRIF and were in fact slightly potentiated by the combined treatment. The fact that PRL concentrations were elevated after SRIF and that GH concentrations were reduced,

suggests that the endocrine control of these hormones, in cattle, may be a reciprocal system.

This study describes the first attempt in defining the role of SRIF in the endocrine control of mammotropic hormones of cows. The control mechanism of PRL release is at present unknown, although a consensus prevails that PRL is tonically inhibited by prolatin inhibiting factor (PIF). One might speculate that the action of SRIF on increasing basal serum PRL and potentiating the milking induced rise in serum PRL may be via interaction with PIF release, perhaps by inhibiting it. Alternatively, SRIF may directly promote the release of PRL at the pituitary.

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Anti-androgenic action of cyproterone acetate in the epididymis of the rhesus monkey[1]

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Summary. Cyproterone acetate (1 μM) inhibited the binding of labeled testosterone (T) and dihydrotestosterone (DHT) in the caput and cauda epididymides of the rhesus monkey. The same concentration of the anti-androgen failed to inhibit androgen binding in the ductus deferens, prostate and seminal vesicles.

In a study aimed at the evaluation of androgen action in various mammalian tissues, Prased et al.[3] showed that in the rat, hamster and monkey, the epididymis exhibited a higher androgen threshold requirement in comparison to the accessory glands. The fact that the spermatozoa undergo morphological and physiological changes leading to their maturation within the epididymal canal points up the organ as a potential target site for male fertility control. It was suggested that since the epididymis shows a higher androgen threshold requirement, an anti-androgen at a dose level too low to alter the functional integrity of the accessory glands should be able to cause selective inhibition of the epididymal function. An experimental approach to this concept showed that microquantities of the anti-androgen cyproterone acetate (CA) released from s.c. implanted silastic capsules in the rat inhibited epididymal function while not affecting the physiology of the accessory glands[4]. In a companion report[5], we have shown that the caput epididymidis of the rhesus monkey differed from the accessory glands in molecular mechanisms fundamental to androgen action; namely, the hormone-receptor interaction. The present report is an extension of this study where it is demonstrated that the mode of action of microquantities of CA in the epididymis is different from that in the accessory glands.

Materials and methods: The details regarding experimental animals, sources of labeled androgens, preparation of cytosol fractions and the analysis of radioactivity are given in the companion paper[5]. Equilibrium dialysis, for the study of in vitro hormone binding was carried out as follows: Polypropylene 'wells' (2 cm in height and 1.5 cm in diameter), fixed on a glass plate, were partitioned in the middle by dialysis membranes. Cytosol (0.5 ml) was added to 1 chamber and 0.5 ml of TEM buffer containing labeled T or DHT, with or without 1 μM CA was placed in the opposite chamber. The 'wells' were sealed on top and agitated in a metabolic shaker at 4 °C for 40 h. At the end of dialysis, the volume of the fluid on either side of the

Effect of 1 μM CA, in vitro on testosterone (T) and DHT (D) binding to the cytosol macromolecules of ductus deferens (DD), seminal vesicle (SV) and prostate (PR) (expressed as nmoles/mg protein). Method: equilibrium dialysis

Total hormone in the medium (nM)	Androgen	DD		SV		PR	
		−CA	+CA	−CA	+CA	−CA	+CA
2.22	T	0.540	0.714	0.281	0.481	0.490	0.657
	D	0.709	1.111	0.310	0.396	0.408	0.581
4.35	T	0.909	1.111	0.490	0.809	0.800	0.991
	D	1.087	1.333	0.478	0.725	0.571	0.806
9.09	T	0.990	1.428	0.757	1.186	0.990	1.428
	D	1.369	1.886	0.709	1.052	0.704	1.075
12.50	T	1.250	2.006	0.900	1.250	1.098	1.538
	D	1.562	2.325	0.800	1.250	1.333	2.080
16.70	T	1.333	1.219	1.612	1.333	1.666	2.222
	D	1.624	2.357	0.909	1.408	1.624	2.083
22.22	T	1.428	2.400	1.383	1.718	1.428	1.923
	D	1.685	2.425	0.920	1.416	1.639	2.120