PGE_2 alone was, respectively, 94±4% and 93±7% of the uptake observed in the absence of added test substance. When ouabain was also present in the incubation medium, ^{86}Rb uptake decreased to 57±7% and to 57±6% with PGE_1 and PGE_2 (fig. 2).

Discussion. The results indicate that although both isoproterenol and prostaglandins E_1 and E_2 increase cAMP formation in rat submandibular acini, only the former substance increases ouabain-sensitive K (^{86}Rb) uptake by the Na, K pump. Thus, our findings indicate that cAMP is not the mediator in the enhancement of pump activity induced by β-adrenergic agonists in salivary cells. Such an effect is likely related to other effects, distinct from the formation of cAMP. Preliminary experiments suggest that this may involve differential effects on Na entry into the cells, which is enhanced by isoproterenol but not by the prostaglandins.

As cAMP does not mediate either ouabain-sensitive K uptake (this study) or Cl efflux in salivary acini[13], the question arises which role, if any, does this nucleotide fulfill in fluid secretion induced by β-receptor stimulation in salivary glands[2]. Both activation of the Na, K pump and of Cl efflux into the lumen through channels presumably localized in the apical cell membrane are believed to be important elements of the ionic mechanism underlying saliva secretion[2, 3, 11]. A recent finding that cAMP enhances tight junctional permeability[14] may provide an answer to this question. An important difference between isoproterenol and the prostaglandins, both of which increase cAMP formation in salivary acini, is that the former also increases Na accumulation in the intercellular space as a result of increased pump activity. This should provide a supply of osmotically active ions to move across the more permeable tight junction to provide a driving force for fluid secretion. In the case of the prostaglandins, tight junctional permeability may be enhanced by the increased formation of cAMP, but the failure to activate the Na, K pump would not provide a source of Na in the intercellular spaces for movement across the tight junction.

The effect of PG on cAMP in submandibular acini suggest that these cells have functional receptors for these substances. It remains to be seen if these receptors are directly coupled to adenylate cyclase or to some other aspect of cAMP metabolism, such as inhibition of phophodiesterase. Although rat submandibular cells may thus have PG receptors coupled to cAMP, the inhibitory effects of PG on fluid secretion in vivo[15] do not seem to be related to effects on ion transport systems[9]. Thus, these receptors may fulfill other cAMP-mediated roles in salivary acini, such as those related to protein synthesis and secretion. However, the inhibitory effects of PG on fluid secretion may still be related to other effects shared with β-adrenergic agonists, as previous exposure to isoproterenol also inhibits cholinergically-induced salivation in vivo[16].

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Casein and lactose concentrations in milk of 31 species are negatively correlated

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Summary. Casein and lactose concentrations in milks of various species vary by at least 1–2 orders of magnitude, yet no single species secretes both components at either the high or the low end of the ranges. This pattern of variation could result from evolutionary adaptations in a single secretory mechanism.

Key words. Casein; lactose; milk composition; milk secretion.

The mechanisms of milk secretion have been studied in detail in only a few species[1–3] most of which are eutherians. Consequently generalizations about the formation of milk in the approximately 4000 extant species must necessarily be circumspect. Nevertheless, the means by which proteins for export are synthesized and secreted by the secretory cells of the mammary gland appears to have much in common with the mechanism used in the export of proteins by other eukaryotic cells fulfilling many diverse functions[4–7]. Milk proteins are packaged for export in vesicles derived from the Golgi apparatus of the mammary secretory cell. The vesicles move to the apical membrane and pass their contents into the alveolar lumen by an exocytotic mechanism. Secretion of milk fat is by a separate process and mixing of the fat and aqueous phases occurs only in the alveolar lumen. The principal difference between the mammary gland and other protein-exporting cells is the synthesis of low molecular-weight carbohydrates such as lactose[8] in the Golgi vesicles of the former. This is thought to influence the volume of aqueous fluid secreted per unit time[9].

Taylor and Husband[10] were the first to recognise clearly the role of lactose synthesis in determining the daily yield of milk from cows. As originally envisaged, synthesis of lactose draws in water osmotically to increase the volume of the aqueous secretion. Since the synthesis of lactose could be accompanied by a compensating loss of an osmotically equi-

Average casein and lactose concentrations in the milks of 31 species

Species	No. in fig.	Animals	Samples	Casein g/kg H_2O	Lactose g/kg H_2O	Reference
Quokka *(Setonix brachyurus)*	1	4	4	25.4	39.3	16
Man *(Homo sapiens)*	2	compilation		4.6	79.9	17
European Beaver *(Castor fiber)*	3	4	7	81.7	34.2	18
Allegheny Wood Rat *(Neotoma floridana)*	4	8	8	60.0	46.8	19
Golden Hamster *(Mesocricetus auratus)*	5	6	6	86.5	63.3	16
Norway Rat *(Rattus norvegicus)*	6	8	8	81.0	32.9	13
House Mouse *(Mus musculus)*	7	5	5	115.5	49.4	16
Coypu *(Myocastor coypus)*	8	?	9	159.0	10.2	20
Guinea Pig *(Cavia porcellus)*	9	?	?	81.5	35.6	22
Spotted Dolphin *(Stenella graffmoni)*	10	7	7	73.4	17.2	23
Dog *(Canis familiaris)*	11	6	12	69.6	37.3	19,29
Black Bear *(Ursus americanus)*	12	6	6	146.2	9.3	25,26
Grizzly Bear *(Ursus arctos)*	13	10	10	103.7	14.8	25
Polar Bear *(Ursus maritimus)*	14	7	7	135.2	5.7	25
Sea Otter *(Enhydra lutris)*	15	3	3	68.3	15.0	27
N. Fur Seal *(Callorhinus ursinus)*	16	5	5	133.7	2.8	28
Weddell Seal *(Leptonychotes Weddelli)*	17	8	8	264.0	23.3	16
N. Elephant Seal *(Mirounga angustirostris)*	18	7	7	97.9	9.4	16
Indian Elephant *(Elephas maximus)*	19	7	7	27.9	69.0	29
Horse *(Equus caballus)*	20	?	231	14.6	69.8	30
Ass *(Equus asinus)*	21	6	12	9.4	69.7	19,31
Pig *(Sus scrofa)*	22	70	535	44.0	66.1	16,32
Dromedary *(Camelus dromedarius)*	23	?	15	31.2	57.9	33
Red Deer *(Cervus elaphus)*	24	4	12	81.0	57.5	34
Reindeer *(Rangifer tarandus)*	25	5	5	125.7	45.3	35
Eland *(Taurotragus oryx)*	26	?	51	75.4	48.9	36
Water Buffalo *(Bubalus bubalis)*	27	compilation		38.6	58.0	37
Cow *(Bos taurus)*	28	compilation		32.1	55.0	17
Musk Ox *(Ovibos moschatus)*	29	6	6	104.9	49.0	19
Goat *(Capra hircus)*	30	?	2662	28.8	47.2	38
Sheep *(Ovis aries)*	31	18	90	40.0	55.8	39

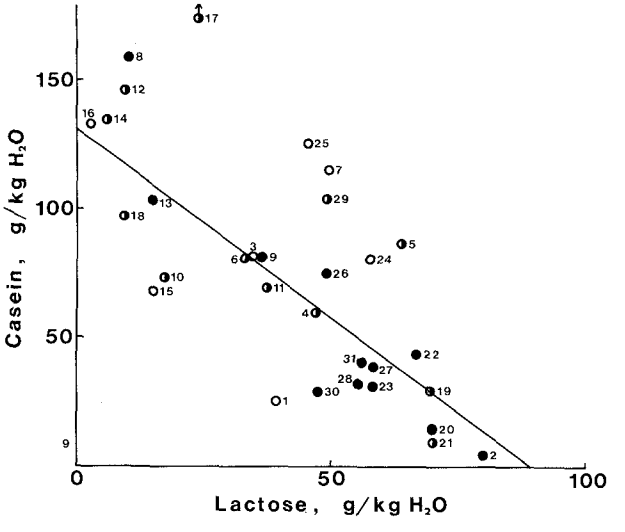
? Not known but assumed to be > 9.

valent amount of salt, the hypothesis has been modified to allow for a coupling of lactose synthesis to the net transport of anions[11]. According to this more recent view, casein is at a relatively high concentration in the Golgi apparatus of the secretory cell but becomes diluted during its transport via Golgi vesicles to the apical membrane while lactose is being synthesized within the vesicle. At any time during the translocation of the vesicle, the casein and lactose concentrations ([C] and [L] respectively) are related by[12]:

$$[C] = [C]_u(1 - (1 + \Omega)[L]/\Pi) \quad (1)$$

where $[C]_u$ is the casein concentration in the Golgi apparatus, Ω is a constant describing the coupling of lactose synthesis to the net transport of anions and Π is the milk osmolarity, taken here to be 0.3 osmol/kg H_2O. The appropriate concentration units for equation (1) are amount per kg H_2O to allow for the excluded volume of co-solutes and the fat phase in influencing the osmotic activity of solutes.

The hypothesis advanced here is that evolutionary adaptations in this mechanism are mainly responsible for the interspecific differences in the concentrations of casein and lactose in the aqueous phase of milks. Specifically, the change from an unswollen Golgi vesicle containing a high concentration of casein and little lactose to a swollen vesicle with a lower casein concentration and more lactose can be terminated at any stage to create many intermediate milk compositions. It follows from equation (1), therefore, that if the concentration of casein in the Golgi apparatus or unswollen Golgi vesicle does not vary overmuch from species to species, then the casein and lactose concentrations in the aqueous part of milks should be negatively correlated. We previously examined milks from six species[12] and with this restricted range of readily available milks found the results to fit equation (1) with $[C]_u = 142$ g/l skim-milk and $1+\Omega = 1.32$ ($r^2 = 0.964$). We have now surveyed literature data on milk composition for over 200 species, as a result of which, reliable values for the casein and lactose concentrations in 29 species have been compiled. Also new results for two species and supplementary results for two others are reported. Values were selected according to the following criteria: 1) milk samples should be from a minimum of three normal, healthy, animals in mid-lactation, 2) lactose concentration should be determined directly, rather than by difference, and on the same sample as was used for the measurement of the casein concentration and 3) sufficient other information about the milk should be available to allow the concentrations of casein and lactose to be expressed as amounts per kg H_2O. An



Interspecific variation in the casein and lactose concentrations of milk from 31 eutherian species. Numbers on the figure identify the species listed in the table and three different symbols are used to indicate the number of animals of each species used to derive the average concentrations: ○, 3–5; ◑, 6–8; ●, 9 or more animals.

exception to (2) was made for milk of the Norway rat[13] where the given lactose values appear to be typical of the species as judged by other work[14, 15].

The data on 31 species, representing 9 mammalian orders, are summarized in the table. Where appropriate, an average value from two or more sources is given, weighted according to the number of samples used in each of the original references, except for the guinea pig where this was not possible and a simple average was taken of results from the two sources. For the most part the analytical methods used were adaptations of the ones used for cows' milk but may be subject to a number of systematic errors. For example, lactose determinations by reducing power or optical rotation are reliable only if lactose is the only sugar present. Likewise, cow casein is most completely precipitated from the milk at around pH 4.6 but in human milk some lactoferrin may co-precipitate. In general, it is impossible to estimate the magnitude of any corrections that might be required and the authors' original values have been used irrespective of the analytical methods. Furthermore, the small numbers of animals and samples taken for some species means that the values given in the table may not be a good approximation to the averages for those species. Nevertheless, apart form the results for the Weddell seal, which appear anomalous and have been excluded from subsequent analysis, a clear negative correlation of casein and lactose concentrations is apparent in the figure. The data were fitted to equation (1) by linear least squares analysis employing sample weighting. A weighting coefficient equal to the square root of the number of animals used to form the averages in the table was calculated for each species except for the species with a large number of samples. Here, a weighting coefficient of 10 was used to avoid excessive bias towards the fitting of points for some ruminant species, horse, pig and man. The derived parameters were then $[C]_u = 135$ g/kg H_2O and $1 + \Omega = 1.20$ ($r^2 = 0.661$) which are only a little different from the values derived in the earlier examination of results from only six species. Without sample weighting, least squares analysis gave the very similar results $[C]_u = 133$ g/kg H_2O and $1 + \Omega = 1.11$ ($r^2 = 0.557$).

The figure shows that no species examined secretes milk with low concentrations of both lactose and casein and, likewise, no species was found to produce milk with high concentrations of both these components. In general, the pattern of interspecific variation is consistent with the hypothesis of a regulation of casein concentration by an osmotic mechanism involving the low-molecular weight milk sugars[11, 12]. More particularly, interpreted in terms of equation (1), most of the points fall in a fairly narrow band indicating that osmotic dilution by lactose synthesis coupled to net anion transport is the major factor governing milk casein concentration, but some variability in the casein concentration in the Golgi apparatus or unswollen vesicle can also occur. Interspecific variation in the concentration of casein in the Golgi apparatus or unswollen vesicle together with the systematic errors in casein and lactose determinations could account for the scatter of points about the fitted line. Interestingly, however, of the points falling farthest from the fitted line, most are from species that inhabit a cold environment and secrete milk with a high fat content[16]. Although no data on the milk of any lagomorph met our criteria, an analysis of a single sample of milk of the domestic rabbit[40] had casein and lactose contents of 84.5 and 24.5 g/kg H_2O respectively which falls close to the regression line in the figure.

Equation (1) was derived to describe the process of maturation of Golgi vesicles within mammary secretory cells. It is easily envisaged that this process provides a means by which milks of very different composition can be produced and that this mechanism could have been used in the evolutionary pathways that have led to some of the interspecific differences in milk composition. Moreover, the implications of this relationship are not confined to the concentrations of casein and lactose alone. For example, if it is advantageous for a particular reproductive strategy to secrete milk with a high content of fat, this can be achieved in two distinct ways. One involves an enhanced rate of secretion of fat in relation to casein and lactose secretion and the other a decreased rate of lactose synthesis in relation to casein and fat secretion. In seals, dolphins, bears, the sea otter, and the coypu, carbohydrate is present in only small amounts in the milks; it can be concluded that the latter mechanism is mainly responsible for the high fat concentrations of these species. In certain terrestrial mammals, such as the red deer, reindeer and musk ox, whose milks have high fat contents, the carbohydrate content is also high, suggesting that a more equal combination of the two mechanisms is used to produce these milks.

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Chemical sympathectomy reveals pre- and postsynaptic effects of neuropeptide Y (NPY) in the cardiovascular system

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Summary. Intravenous injection of neuropeptide Y (NPY) caused short-lasting dose-dependent pressor responses in anesthetized rats. NPY was equipotent with noradrenaline in producing proportional pressor effects. Chemical sympathectomy, following the administration of 100 mg/kg 6-hydroxydopamine (6-OHDA), significantly potentiated the systemic pressor effects elicited by NPY or noradrenaline. Pretreatment with 2 nmol NPY enhanced the noradrenaline-induced pressor response in control rats. NPY did not change the basal tension of isolated rat aortic strips but significantly potentiated the contractile activity induced by 16 nM noradrenaline. This effect of NPY was not observed in aortic strips from rats pretreated with 6-OHDA. The presence of pre- and postsynaptic sites of action for NPY in the cardiovascular system of the rat is discussed.
Key words. Vascular system; NPY responses; NPY-pressor responses; NPY-induced supersensitivity; aortic contractions.

Neuropeptide Y (NPY), a 36-amino acid peptide with structural homology with avian pancreatic polypeptide, is frequently found co-stored with noradrenaline in discrete brain nuclei including the human brain medulla[1] and a variety of sympathetically innervated organs such as the heart, spleen, vas deferens and blood vessels[2,3]. Although the physiology of NPY remains to be established, because of its co-distribution with noradrenaline in central and peripheral synapses, particularly in nerve terminals around blood vessels[4,5], it is anticipated that the peptide plays a role in the noradrenergic transmission in the vascular system. In fact, it is known that NPY contracts blood vessels, increasing systemic blood pressure via a non-adrenergic mechanism[6,10]. NPY enhances the contractile effects of noradrenaline and other pressor substances in isolated vascular smooth muscles[11,12] and potentiates the hypertensive response caused by preganglionic sympathetic nerve stimulation[13]. The aim of this study was to document further the pre- and postjunctional effects of NPY on the cardiovascular system. To distinguish between pre- and postsynaptic sites of action, we sympathectomized rats using 6-hydroxydopamine (6-OHDA) and examined the pressor activity of NPY. In addition, we used isolated aortic strips from normal and 6-OHDA-treated rats to examine whether NPY interacts with noradrenaline in this vessel. Results discuss the presence of pre- and postsynaptic NPY sites in the vascular territory.

Materials and methods. Blood pressure monitoring. Male Sprague Dawley rats (250–280 g) were anesthetized with 40 mg/kg sodium pentobarbital i.p.; systemic blood pressure was recorded continuously via a catheter inserted into the carotid artery. The catheter was connected to a strain gauge transductor; continuous blood pressure recordings were inscribed on a Grass polygraph (for details see Mabe et al.[10]). Drugs were administered i.v. via a cannula placed on the femoral vein. Groups of 6 rats each were used; one group was treated with 100 mg/kg 6-OHDA i.v. 48 h prior to blood pressure monitoring; the control group was injected i.v. with the solvent (0.3 ml of 1% ascorbic acid dissolved in saline). Results compare the mean increase in systolic or diastolic blood pressure induced by NPY and noradrenaline in control and 6-OHDA-treated rats.

In a second series of experiments, noradrenaline pressor dose-response curves were performed in 10 normal rats before and 10 min after a bolus dose of 10 µg NPY/rat (2 nmol). Results plot the increase in systolic blood pressure caused by noradrenaline before and after treatment with NPY.

Isolated aortic strips preparation; noradrenaline-NPY interactions. Adult male Sprague-Dawley rats were sacrificed by cervical dislocation. The thorax and abdominal cavity were opened; the aorta was dissected from the heart to the mid-abdomen. The tissue was placed on a Petri dish containing Tyrode solution to prepare spiral strips of the aorta 2–3 cm long. Special precaution was taken not to damage the aorta endothelium during the tissue handling. The artery was immediately mounted on a 30-ml organ bath to record isometric contractions. The buffer was maintained at 37 °C and bubbled with 95% O_2/5% CO_2 to maintain the pH close to 7.4. Tissues were given a resting tension of 5 mNewton (mN) that was maintained throughout the experiment. The tissues were washed with fresh oxygenated Tyrode solution every 20 min to prevent accumulation of toxic metabolic end products[14]. The composition of the Tyrode solution was as follows (mM): NaCl 118; KCl 5.4; $CaCl_2$ 2.5; KH_2PO_4 1.2; $MgSO_4$ 1.2; glucose 11.1 and $NaHCO_3$ 23.8. The recording of isometric contractions was done with a Grass FT-3C force displacement transducer coupled to a Grass polygraph.

Tissues were equilibrated for 90 min prior to drug additions. Different concentrations of noradrenaline were tested; 16 nM noradrenaline provided the most consistent and reproducible contractile responses to the application of the catecholamine. The noradrenaline-induced aortic contractions were examined in strips from control rats before and after tissue incubation with 10 µg NPY (190 nM) for 3 min.

In a parallel series of experiments, aortic strips from rats pretreated 48 h before with 100 mg/kg 6-OHDA i.v. were prepared (these rats were not the same as those used in blood pressure monitoring). Groups of aortic strips were challenged with 16 nM noradrenaline, while another group was tested with 16 nM noradrenaline in the presence of 190 nM NPY. Results compare the contractile activity of noradrenaline in control and sympathectomized rats and the effects of NPY in the two populations of rats.