Contents

VOLUME 143, NUMBER 1, SEPTEMBER 1991

SJÖSTRÖM, A., ABRAHAMSSON, M., NORRSELL, K., HELGASON, G. & ROOS, A. Flashed pattern induced activity in the visual system: I the short latency evoked response for the cat visual cortex

RYDQVIST, B. & SWERUP, C. Stimulus response properties of the slowly adapting stretch receptor neuron of the crayfish

Swerup, C., Purali, N. & Rydqvist, B. Block receptor response in the stretch receptor neuron of the crayfish by gadolinium

Schieppati, M., Gritti, I. & Romano, C. Recurrent and reciprocal inhibition of the human monosynaptic reflex shows opposite changes following intravenous administration of acetylcarnitine


Persson, K., García-Pascual, A. & Andersson, K. E. Difference in the actions of calcitonin gene-related peptide on pig detrusor and vesical arterial smooth muscle

Hjelmqvist, H., Ullman, J., Gunnarsson, U., Lundberg, J. M. & Rundgren, M. Haemodynamic and humoral responses to repeated hypotensive haemorrhage in conscious sheep

Bell, I., Zaret, B. L. & Rutlen, D. L. Influence of α-adrenergic receptor stimulation on splanchnic intravascular volume in conscious humans

Oien, A. H. & Aukland, K. A multinephron model of renal blood flow autoregulation by tubuloglomerular feedback and myogenic response

Alving, K., Matran, R. & Lundberg, J. M. The possible role of prostaglandin D₁ in the long-lasting airways vasodilation induced by allergen in the sensitized pig

Bair, R., Hostmark, A. T., Newsholme, E. A., Grønnerød, O. & Sjøersted, O. M. Effects of exercise on recovery changes in plasma levels of FFA, glycerol, glucose and catecholamines

Nylander, O., Sababi, M. & Bark, J. Characterization of ⁵¹Cr-EDTA as a marker of duodenal mucosal permeability

Rapid Communications

Suzuki, H., Tsuzimoto, H., Kasuga, N., Taguchi, S. & Ishihara, S. Effect of endurance training on the oxidative enzyme activity of soleus motoneurons in rats

Yndgaard, S., Schifter, S., Perko, G., Matzen, S. & Secher, N. H. Calcitonin gene-related peptide (CGRP) during head-up tilt in man

Takahashi, H., Wada, M. & Katsuta, S. Expressions of myosin heavy chain IId isoform in rat soleus muscle during hind limb suspension

Lerner, U. H. Parathyroid hormone and transforming growth factor β synergistically stimulate formation of prostaglandin E₂ in neonatal mouse calvarial bones

Nilsson, B.-O., Rosengren, E. & Ekström, J. In vivo inhibition of parasympathetic nerve induced increases in ornithine decarboxylase activity of the rat sublingual gland by α-difluoromethylornithine
ALVING, K., MATRAN, R., FORNHEIM, C. & LUNDBERG, J. M. Late phase bronchial and vascular responses to allergen in actively-sensitized pigs 137

HU, P.-S., JIN, S. & FREDHOLM, B. B. 4-Aminopyridine-induced noradrenaline release from the rat hippocampus depends on the activation of glutamate receptors of the non-NMDA type 139

INSTRUCTIONS TO AUTHORS 141

PROCEEDINGS FOR THE SCANDINAVIAN PHYSIOLOGICAL SOCIETY MEETING IN UPPSALA, 24–26 MAY 1991

VOLUME 143, NUMBER 2, OCTOBER 1991

DUNNING, B. E., KARLSSON, S. & AHRÉN, B. Contribution of galanin to stress-induced impairment of insulin secretion in swimming mice 145

ISLIN, H., CAPITO, K., HANSEN, S. E., HEDESKOV, C. J. & THAMS, P. Ability of omega-3 fatty acids to restore the impaired glucose tolerance in a mouse model for type-2 diabetes. Different effects in male and femalae mice 153

NILSSON, B.-O., ROSENGREN, E. & EKSTRÖM, J. Effects of stimulation of the parasympathetic and sympathetic innervations in bursts on the syntheses of polyamines, DNA and protein in salivary glands of the rat: non-adrenergic, non-cholinergic responses 161

EDIN, B. B. The ‘initial burst’ of human primary muscle spindle afferents has at least two components 169

HATHER, B. M., TESCH, P. A., BUCHANAN, P. & DUDLEY, G. A. Influence of eccentric actions on skeletal muscle adaptations to resistance training 177

SELIGSOHN, E. E. & KOSKINEN, L.-O. D. Effects of alpha_2-adrenoceptor blockade and thyrotropin-releasing hormone (TRH) on the cardiovascular system in the rabbit 187

PAULSSEN, E. J., PAULSSEN, R. H., HAUGEN, T. B., GAUTVIK, K. M. & GORDELADZE, J. O. Regulation of G protein mRNA levels by thyroliberin, vascoactive intestinal peptide and somatostatin in prolactin-producing rat pituitary adenoma cells 195

KUPENOVA, P., VITANOVA, L., MITOVA, L. & BELCHEVA, S. Participation of the GABAergic system of the turtle retina in the light adaptation process 203

FARSTAD, B. S., SUNDREHAGEN, E., OPDAHL, H. & BENESTAD, H. B. Pulmonary, hepatic and splenic sequestration of technetium-99m labelled autologous rabbit granulocytes: scintigraphic cell distributions after intravenous and intraarterial injections, exsanguination and intraarterial injection of cells passed through an intermediary host 211

Rapid Communication


BLOMSTRAND, E., HASSMÉN, P., NEWSHOLME, E. A. Effect of branched-chain amino acid supplementation on mental performance 225

VOLUME 143, NUMBER 3, NOVEMBER 1991

SLØRDAL, S. A., PIENE, H., LINKEJ, D. T. & VIK, A. Segmental aortic wall stiffness from intravascular ultrasound at normal and subnormal aortic pressure in pigs 227

HARALDISSON, B., JOHNSON, E. & RIPPE, B. A note on the errors of using venous congestion in intact rats for determinations of microvascular permeability 233
Contents

CONSTANTIN-TEODOSIU, D., CARLIN, J. I., CEDERBLAD, G., HARRIS, R. C. & HULTMAN, E.
Acetyl group accumulation and pyruvate dehydrogenase activity in human muscle during incremental exercise 367

MALMQVIST, U., ARNER, A. & UVELIUS, B. Mechanics and Ca-sensitivity of human detrusor muscle bundles studied in vivo 373

HENRIKSEN, E. J. & HOLLOSY, J. O. Effect of diffusion distance on measurement of rat skeletal muscle glucose transport in vitro 381

MATRAN, R., ALVING, K. & LUNDBERG, M. Differential bronchial and pulmonary vascular responses to vagal stimulation in the pig 387

HEMSEN, A., GILLIS, C., LARSSON, O., RAEGERSTRAND, A. & LUNDBERG, J. M. Characterization, localization and actions of endothelins in umbilical vessels and placenta of man 395

CERVIN, A., LINDBERG, S. & MERCKE, U. Sympathetic nerve stimulation influences mucociliary activity in the rabbit maxillary sinus 405

MALM, D., GIIVER, A., VONEJ, B., BURIOL, P. G. & FLORHOLMEN, J. Somatostatin inhibition of phospholipase C activity in isolated rat pancreatic islets 413

VEEL, T., VILLANGER, O., HELTHE, M. R., SKORTEN, F. S. & RAEDER, M. G. Intravenous bilirubin infusion causes vacuolization of the cytoplasm of hepatocytes and canalicular cholestasis 421

BUGGE, J. F., STOKKE, E. S. & KIIL, F. Effects of bradykinin and papaverine on renal autoregulation and renin release in the anaesthetized dog 432


Rapid Communications

GÜR, H. & LARSSON, L. Regional differences in the influence of the interval between removal and freezing of muscle samples on muscle fibre size 445

MEISTER, B., HOLGERT, H., APERIA, A. & HÖKFLERT, T. Dopamine D1 receptor mRNA in rat kidney: localization by in situ hybridization 447

WEITZBERG, E., RUDEHILL, A., ALVING, K. & LUNDBERG, J. M. Nitric acid inhalation selectively attenuates pulmonary hypertension and arterial hypoxia in porcine endotoxin shock 451

Author and Subject Index 453
Effects of the nitric oxide synthase inhibitor \(N^G\)-nitro-L-arginine on the erectile response to cavernous nerve stimulation in the rabbit

F. HOLMQUIST, C. G. STIEF*, U. JONAS* and K.-E. ANDERSSON
Department of Clinical Pharmacology, Lund University Hospital, Lund, Sweden and
*Department of Urology, School of Medicine, Hannover, Germany

Using a rabbit model, the involvement of the L-arginine/nitric oxide pathway in penile erection was investigated. The mean basal intracavernous pressure was 21 cm H\(_2\)O. Cavernous nerve stimulation (4–8 V, 20–30 Hz) increased the pressure to approximately 130 cm H\(_2\)O. This response was highly reproducible and usually associated with full penile erection. The pressure increase could be quantified in terms of: (1) the slope of the initial, ascending part of the pressure increase; (2) \(\Delta P\), which was defined as the maximal pressure obtained by the stimulation minus the basal pressure before the stimulation; (3) \(T_{90}\), which was defined as the time to reach 90 per cent of \(\Delta P\). Intrapenile administration of the L-arginine/nitric oxide synthesis inhibitor \(N^G\)-nitro-L-arginine had no effect on systemic arterial blood pressure. However, \(N^G\)-nitro-L-arginine (0.22 and 2.19 mg), administered via the same route, abolished the erectile response induced by cavernous nerve stimulation; \(T_{90}\) increased and slope and \(\Delta P\) decreased significantly. \(N^G\)-nitro-D-arginine (2.19), on the other hand, had no inhibitory effect. L-arginine (21.07 mg), given either directly or after \(N^G\)-nitro-L-arginine had no consistent effect on the functional response to cavernous nerve stimulation.

The results suggest that pharmacologically induced effects on intracavernous pressure in the rabbit can be described quantitatively, and that this model may be useful to study the mechanisms controlling penile erection in vivo. The pronounced inhibitory action of \(N^G\)-nitro-L-arginine demonstrates the important role of the arginine/nitric oxide pathway in mediating relaxation of penile smooth muscles necessary for erection.

Key words: nitric oxide, penile erection, rabbit.

For erection to be induced, the penile arteries and sinusoids have to dilate, thereby decreasing the resistance to penile blood flow (Andersson & Holmquist, 1990). However, the mechanism of penile smooth muscle relaxation has not been fully elucidated. Nitric oxide (NO), which is believed to account for the biological actions of endothelium-derived relaxing factor (for review; Ignarro 1990, Marin & Sánchez-Ferrer 1990), was recently suggested to be of importance in the regulation of penile smooth muscle tone, both in the flaccid state (Holmquist et al. 1991 b) and during erection (Ignarro et al. 1990, Holmquist et al. 1991 a, b). This was based on experiments utilizing isolated preparations of human and rabbit corpus cavernosum. For instance, in both human (Holmquist et al. 1991 a, b) and rabbit (Ignarro et al. 1990, Holmquist et al. 1991b)
preparations, N\textsuperscript{\textit{\textperiodcentered}}-nitro-L-arginine (\textit{l}-NOARG), an inhibitor of the synthesis of NO from \textit{l}-arginine (Moore et al. 1989, Mülisch & Busse, 1990), almost abolished the relaxations elicited by electrical field stimulation. Furthermore, \textit{l}-NOARG produced a tension-increase when given to preparations contracted by noradrenaline (Holmquist et al. 1991b). However, to the best of our knowledge, the possible involvement of the \textit{l}-arginine/NO pathway in the control of penile blood flow has never been investigated \textit{in vivo}.

It has previously been shown that the rabbit is a useful model for the study of erectile mechanisms in the intact animal (Sjöstrand & Klinge 1979, Stief et al. 1990). Using the experimental set-up previously described (Stief et al. 1990), we wanted to investigate the effect of NO synthase inhibition on the erectile response induced by electrical stimulation of the cavernous nerve in the rabbit.

**MATERIALS AND METHODS**

**Animals.** Eleven rabbits (New Zealand White) weighing 4–5 kg were used for the investigation. After sedation with i.m. ketamine (10 mg), the animals were anaesthetized with i.v. pentobarbital (15 mg kg\textsuperscript{-1}) through a 21-gauge needle introduced into an ear vein. Anaesthesia was maintained with 3 mg kg\textsuperscript{-1} i.v. bolus injections of pentobarbital as needed. During the course of the experiment, the rabbits also received warm saline (2–3 ml kg\textsuperscript{-1} h\textsuperscript{-1}) and 10% glucose in saline (0.5 ml kg\textsuperscript{-1} h\textsuperscript{-1}) i.v. The animals breathed spontaneously.

The rabbits were placed in a supine position on a thermoregulated operating table (model 11A, Hugo Sachs Elektronik, Germany). Additional heat was provided with a heating lamp. The abdomen was opened by a midline incision, and the bladder was emptied. The rectum was tied, and the intestines were put in a pad soaked with saline and placed in the upper abdomen. By gentle dissection, the cavernous nerves were exposed in the dorso-lateral aspect of the prostate on both sides.

The penile skin was removed by blunt dissection and a 21-gauge needle was inserted into the left corpus cavernosum for pressure recording. The needle was connected to a fluid line via a threeway stopcock, which allowed for intracavernosal application of drugs. To prevent clotting, 50 I.U. heparin was given through this route every 2–3 h. This dose of heparin is well below the doses needed to induce changes in penile haemodynamics (Kirkey et al. 1990). In some experiments, arterial blood pressure was recorded from one of the femoral arteries. Pressure was measured using Statham transducers (model P23XL) connected via DC Bridge Amplifiers Type 660 to a Watanabe Mark VII recorder (Hugo Sachs Elektronik, Germany).

**Experimental procedure.** The cavernous nerve on one side was stimulated electrically using a movable contact electrode. Square wave pulses were delivered by a Stimulator IZ (Hugo Sachs Elektronik, Germany). Upon stimulation, the intracavernous pressure increased rapidly, and the penis usually became tumescent or erect. The stimulation was continued for 60 s or until a maximal, stable intracavernous pressure had been obtained. The increase in intracavernous pressure during cavernous nerve stimulation was described in terms of: (1) the slope of the initial, ascending part of the pressure increase; (2) \( \Delta P \), which was defined as the maximal pressure obtained by the stimulation minus the basal pressure before the stimulation; and (3) \( T_{90} \), which was defined as the time to reach 90% of \( \Delta P \) (Fig. 1). After stimulation and the pressure had returned to baseline, 1 ml saline was given intracavernosally in order to flush drugs away and to avoid clotting. The time interval between stimulations was approximately 15 min.

In every animal, different stimulation parameters were investigated in a randomized manner to obtain the optimal functional response. This response was reproducible for several hours and used as control. To study the effects of a drug on the functional response to cavernous nerve stimulation, the aorta and v. cava were clamped (30 s) while the drug (dissolved in 1 ml saline) was applied intracavernously through the 21-gauge needle. An incubation-time of at least 10 min was then allowed until the next stimulation was conducted. This incubation time was chosen on the basis of previous studies showing that the maximal effect on arterial blood pressure after intravenous administration of different NO synthase inhibitors is obtained within 5–10 min (Rees et al. 1989, 1990, Persson et al. 1990). The lowest concentration of a drug was always given first.

**Drugs.** The following drugs were used: Ketamine (Parke-Davis, USA), pentobarbital (WDT, Germany), heparin (Roche, Switzerland), d-NOARG (Bachem, Switzerland), \textit{l}-NOARG and \textit{l}-arginine hydrochloride (Sigma, USA). When appropriate, the drugs were dissolved in saline and stored at \(-70\) °C.

**Calculations.** When appropriate, results are given as mean values ± standard error of the mean (SEM) or 99% confidence intervals (as specified). Statistical determination of the effect of a drug on penile erection was performed by using the confidence intervals of the quotients of the slope, \( \Delta P \) and \( T_{90} \) before and after application of the drug. Since the actions of all the drugs were compared with the same control response,
Involvement of NO in penile erection

60 s.

Fig. 1. Schematic drawing showing the increase in intracavernous pressure induced by unilateral cavernous nerve stimulation in the rabbit. The increase in pressure was described in terms of: (1) the slope of the initial, ascending part of the pressure increase (y/x); (2) ΔP, which was defined as the maximal pressure obtained by the stimulation minus the basal pressure before the stimulation; and (3) T_{90}, which was defined as the time to reach 90 per cent of ΔP.

RESULTS

The baseline intracavernous pressure recorded on 64 different occasions was $21 \pm 1$ cm H$_2$O. Stimulation of the cavernous nerve induced a rapid intracavernous pressure increase, usually associated with tumescence or full penile erection. In half of the animals used, the intracavernous pressure did not fall directly after cessation of the stimulation, but declined gradually until it suddenly dropped to baseline (Fig. 2). The slope, ΔP and T_{90} were dependent on the voltage and frequency of stimulation (Table 1), whereas the pulse width (0.5–2.0 ms) seemed to be of less importance (1 ms was chosen for the investigation). Optimal functional responses were obtained with 4–8 V and 20–30 Hz.

L-NOARG (2.19 mg), D-NOARG (2.19 mg) and L-arginine (21.07 mg) had no effect on systemic arterial blood pressure when applied intracavernosally ($n = 3$). L-NOARG (2.19 mg), but not D-NOARG (2.19 mg), decreased the intracavernous basal pressure from $21 \pm 3$ to $16 \pm 2$ cm H$_2$O ($n = 7$). However, this effect was not significant. Pretreatment with D-NOARG (2.19 mg) before cavernous nerve stimulation had no effect on ΔP or T_{90}, but significantly increased the slope compared to control responses (Fig. 3 & Table 2). L-NOARG (2.19 mg), on the other hand, decreased ΔP and increased T_{90} and the slope significantly, and abolished the erectile response (Fig. 3 & Table 2). The effect of L-NOARG was long-lasting and persisted for at least 60 min. The functional response to cavernous nerve stimulation was also impaired by L-NOARG at a lower dose (0.22 mg), although the effect was less pronounced (Fig. 3 & Table 2). L-arginine (21.07 mg), given either directly or after 2.19 mg L-NOARG, had no

99\% confidence intervals were chosen. Student's two-tailed t-test was used to evaluate the drug-effects on basal intracavernous pressure. A probability level < 0.05 was regarded as significant.

Fig. 2. Tracing showing the increase in intracavernous pressure induced by unilateral cavernous nerve stimulation (4 V, 20 Hz) in the rabbit. In this case, the pressure did not fall immediately after the stimulation was stopped, but declined gradually until it suddenly returned to baseline.

---

Table 1. Effect of different stimulation frequencies on the increase in intracavernous pressure induced by unilateral cavernous nerve stimulation at 4 V. Results are given as mean values ± SEM

<table>
<thead>
<tr>
<th>Frequency (Hz)</th>
<th>Maximal pressure (cm H$_2$O)</th>
<th>Pressure increase, ΔP (cm H$_2$O)</th>
<th>T_{90} (S)</th>
<th>Slope</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5</td>
<td>78 ± 16</td>
<td>60 ± 14</td>
<td>106.5 ± 20.9</td>
<td>0.42 ± 0.05</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>86 ± 19</td>
<td>66 ± 20</td>
<td>55.7 ± 9.6</td>
<td>1.18 ± 0.53</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>118 ± 10</td>
<td>98 ± 10</td>
<td>35.6 ± 7.1</td>
<td>3.64 ± 1.36</td>
<td>4</td>
</tr>
<tr>
<td>20</td>
<td>123 ± 6</td>
<td>103 ± 5</td>
<td>23.7 ± 3.0</td>
<td>4.51 ± 0.54</td>
<td>15</td>
</tr>
<tr>
<td>30</td>
<td>130 ± 11</td>
<td>107 ± 7</td>
<td>15.8 ± 0.2</td>
<td>5.31 ± 1.51</td>
<td>4</td>
</tr>
</tbody>
</table>
consistent effect on the functional response to cavernous nerve stimulation ($n = 6$).

**DISCUSSION**

The present study confirms and extends previous findings *in vitro*, suggesting that NO, released either directly from nerves or from the endothelium via the action of some yet unidentified transmitter, is involved in the control of penile smooth muscle tone (Ignarro et al. 1990, Holmquist et al. 1991a, b). Indeed, the pronounced inhibitory effect of L-NOARG on the functional response induced by cavernous nerve stimulation clearly demonstrates the crucial role of the L-arginine/NO pathway in the mechanisms leading to penile smooth muscle relaxation necessary for erection. At the doses used, intrapenile administration of L-NOARG did not cause any pressor effects. This is in agreement with previous investigations where intravenous injections of low doses of L-NOARG methyl ester had no or only minor effects on blood pressure and heart rate (Gardiner et al. 1990, Rees et al. 1990). It is therefore reasonable to assume that the local penile effect of L-NOARG observed in the present study was not influenced by any systemic haemodynamic changes. D-NOARG had no effect on ΔP and $T_{90}$ thus confirming the enantiomer-specific nature of the action of L-NOARG (Ignarro et al. 1990, Holmquist et al. 1991a & b). However, D-NOARG significantly increased the slope of the intracavernous pressure increase evoked by cavernous nerve stimulation. The reason for this is unknown.

Also the basal intracavernous pressure was decreased by L-NOARG, although this effect was not significant. Previous results *in vivo* indicate that there is a continuous release of NO, or a NO-containing compound, modulating vascular tone and thereby the systemic blood pressure (Vallance et al. 1989, Rees et al. 1989, 1990, Gardiner et al. 1990, Persson et al. 1990). Based on experiments in isolated preparations, a similar mechanism, opposing the effect of noradrenaline and other possible contractant factors during the flaccid state (Andersson & Holmquist 1990), was proposed to be of importance also in the penis (Holmquist et al. 1991 b). However, considering that electrical stimulation of the sympathetic trunk (L₄–S₁) induced an intracavernous pressure increase in the rabbit (Stief et al. 1990), it can be questioned whether or not the pressure
decrease observed with L-NOARG reflects an impaired synthesis of basally released NO. The possible involvement of the L-arginine/NO pathway in regulating penile blood flow in the flaccid state remains to be established.

Since L-arginine given intravenously had no direct effect on blood pressure (Rees et al. 1989, 1990, Gardiner et al., 1990, Persson et al. 1990), and since the concentration of endogenous L-arginine in endothelial cells was as high as 0.8 mM (Gold et al. 1989), it was concluded that the enzymatic conversion of L-arginine to NO is saturated and not rate limiting under normal conditions (Gold et al. 1989, Rees et al. 1989, 1990). However, high concentrations of L-arginine could reverse the haemodynamic changes induced by inhibition of the NO synthesis (Rees et al. 1989, 1990, Gardiner et al. 1990, Persson et al. 1990). In accordance with previous findings, intracavernous administration of L-arginine had no effect per se on the penile response induced by cavernous nerve stimulation. In addition, however, L-arginine also failed to reverse the inhibitory effect of L-NOARG. It may be speculated that under the present experimental conditions, the L-arginine dose used, which was 10 times higher than that of L-NOARG, was not sufficient to induce any measurable effects. One must also keep in mind that since the penile blood flow reduction, using the present experimental design, is not complete during drug administration, and since the cavernous bodies constitute an unknown volume, it is difficult to determine the actual intrapenile concentration of a drug injected intracavernosally. Furthermore, L-NOARG, but not L-arginine, is known to act in an irreversible manner (Mülsch & Busse 1990). Thus, quantitative comparisons regarding the different drug doses used cannot be done.

Despite the difficulties in estimating intrapenile drug concentrations, the present results further emphasizes the rabbit as an appropriate model for the study of penile erection. Upon electrical stimulation of the cavernous nerve, the intracavernous pressure increased rapidly until it reached a plateau, which was maintained during the whole period of stimulation. In some cases, the pressure did not fall to baseline immediately after the cessation of stimulation, but declined gradually until it suddenly dropped. During erection, the venous blood flow from the penis is greatly reduced due to compression of the subalbugineal venular plexus and postcavernous venules against the relatively indistensible tunica albuginea. As the intracavernous pressure declines in the detumescent phase, the penile veins become open, with a subsequent increase in venous blood flow. In speculation, it is possible that the different patterns of pressure decrease after electrical stimulation, as observed in this study, reflect interindividual variations in the intracavernous pressure at which the penile veins are opened.

The response to cavernous nerve stimulation in the rabbit is reproducible for several hours using the optimal stimulation parameters. The increase in intracavernous pressure can be described in terms of ΔP, T₉₀ and slope, all of which reflect the erectile response fairly well. By doing so, the effects of different drugs interfering with the erectile mechanism can easily be described and quantified. Future studies will show if this model also can be used to characterize agents of potential use in the treatment of erectile dysfunction.

We would like to acknowledge Dr A. Taher for his technical assistance. This work was supported by the Swedish Medical Research Council (grant no 6837), the Faculty of Medicine, University of Lund, and by grants of the Deutsche Forschungsgesellschaft (DFG Sti 96/2-1 and 96/2-2).

REFERENCES


