Table 2. Serum glucose, triglycerides and insulin concentrations of normal and diabetic Syrian hamsters[a]

	Fed Serum glucose (mg/dl)	Fasted[c] Serum glucose (mg/dl)	Serum triglycerides (mg/dl)	Insulin[d] (μU/ml)
Normal	106±4 (N=34)	109±3 (N=52)	126±89 (N=29)	60±7 (N=48)
Diabetic[b]	412±7 (N=58) p<0.001	329±11 (N=40) p<0.001	478±181 (N=29) p<0.01	25±3 (N=57) p<0.001

[a] Untreated normal and diabetic age-matched male hamsters. [b] All serum values of both groups determined from blood samples collected one month after initiation of streptozotocin injections in the diabetic group. All values are the means of the number of samples given in parentheses ± SEM. [c] Fasted 18 h prior to collection of blood by orbital sinus puncture. [d] Serum insulin concentrations determined by radioimmunoassay as described in the materials and methods section.

levels higher than 55 mg/kg being lethal. In a later report, House and Tassoni[5] found that most (70%) of their 'diabetic' hamsters reverted to normoglycemia after a few days to several weeks even though their definition of diabetes was very liberal (blood glucose > 130 mg/dl = diabetic). Sak and Beaser[6] were able to induce diabetes in hamsters with a much higher i.c. dose (100 mg/kg) of Ax but experienced difficulties with fatalities.

However, by giving multiple injections of Sz, we have been able to consistently induce diabetes in a majority (≅ 80%) of our hamsters. In addition, most of the hamsters (≅ 84%) which fell into the diabetic range 1 week after treatment remained diabetic after 1 month.

To our knowledge, this is the first report of the successful induction of chronic diabetes in a predictable high percent of Syrian hamsters. The value of the hamster cheek pouch for microcirculation studies has long been appreciated and the diabetic Syrian hamster model should gain considerable utility.

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The transfer of juvenile hormone from male to female during mating in the Cecropia silkmoth[1]

P.D. Shirk[2,3], G. Bhaskaran and H. Röller

Institute of Developmental Biology, Texas A & M University, College Station (Texas 77843, USA), 8 August 1979

Summary. The juvenile hormone (JH) stored in the accessory sex glands (ASG) of adult male *Hyalophora cecropia* (L.) originates both from sequestration of circulating hormone and from JH synthesized de novo in the ASG from JH acid taken up from the hemolymph. The secretions present in the lumina of the ASG contain most of the accumulated JH. During mating, endogenous JH, labeled biosynthetically via injected [^{3}H-methyl]-methionine, is transferred along with the other seminal material to the bursa copulatrix of the female. The physiological significance of the JH transfer remains unknown.

The storage of large quantities of juvenile hormone (JH) in adult male Cecropia silkmoths[4] represents an atypical endocrine phenomenon which is known to occur in only 2 other closely related saturniids[5,6]. This phenomenon is based on the ability of the male accessory sex glands (ASG) to act as a repository for JH[7]. These glands contain a JH acid methyltransferase which facilitates the transfer of the methyl group of S-adenosyl methionine to a JH acid, thus forming the respective JH[8-11]. In addition to this process, the ASG are also able to take up JH intact. Once in the ASG, most of the JH stored is found in association with the secretions contained in the lumina of the glands[9]. Since the ASG contribute material to the formation of the spermatophore[12,13], we investigated the possibility of JH transfer to the female during copulation.

Cecropia were obtained from commercial suppliers as diapausing pupae and kept at 4 °C for a minimum of 90 days. Adult development was initiated by exposing the pupae to 27 °C, 70–80% relative humidity and 16–8 h light-dark cycle. [^{3}H-methyl]-methionine (3.7 Ci/mmole) was purchased from Schwarz/Mann. Ether was anhydrous, analytical reagent grade (Mallinckrodt).

Freshly eclosed adult male Cecropia were injected with [^{3}H-methyl]-methionine in Weevers' saline[14] (1 μCi/μl). After 24 h the radiolabeled males were placed with untreated freshly eclosed females. Adult Cecropia mate about 30 min before lights on, continue throughout the light period, and separate shortly after the lights go off again. Mating of all pairs occurred during the following dark phase and the day of copulation is referred to as day 0. The copulation of pair 1 was disturbed prior to the next dark phase and both animals were sacrificed, removing the male ASG and the female bursa copulatrix (BC) separately for processing. Pairs 2 and 3 were allowed to complete copulation; the males were sacrificed at the beginning of day 1 after copulation while the females were sacrificed on day 3 after copulation. The ASG and BC were processed for JH identification by extraction with ether/ethanol (6:1), TLC on silica gel HF_{254} (0.25 mm, methanol washed and activated) with a hexanes/ethyl acetate/acetic acid (70:25:5) solvent system, and high pressure liquid chromatography on μ Porasil with a hexanes/ethyl acetate/2-propanol (96.48:3.5:0.02) solvent system. A mixture of cold JH-I and JH-II was added to the ether/ethanol extracts as an internal standard.

As is evident in the table, JH accumulated in the ASG of adult males is transferred to the BC during copulation. When interrupted during mating, all of the labeled JH was found in the BC (pair 1). When the male was sacrificed after copulation, a small amount of radiolabeled JH was detected in the ASG (pair 2, pair 3), which suggests some post-coital accumulation of JH. It is also interesting to note that even 2 days after mating radiolabeled JH was present in the BC.

JH-I and JH-II in male accessory sex glands (ASG) and in the bursa copulatrix (BC) after copulation. Both juvenile hormones were labelled biosynthetically in the male before copulation

Mating Pair	Organ preparation Type of organ	Time after copulation (days)	JH-I (dpm)	JH-II (dpm)
1	ASG	Copulation interrupted	nil[a]	nil[a]
	BC	Copulation interrupted	9600	1600
2	ASG	0 days	2300	200
	BC	2 days	850	nil[b]
3	ASG	0 days	1000	nil[b]
	BC	2 days	10,100	2500
Control	ASG	Unmated male 2 days old	16,400	1800

[a] nil ≤ 60 dpm total between 2 adjacent fractions; [b] nil ≤ 600 dpm total between 2 adjacent fractions.

The transfer of JH from the male to the female Cecropia during mating is similar in many respects to the transfer of cantharidin between the males and females of *Lytta vesicatoria* (Spanish flies). The adult female *L. vesicatoria* is unable to produce any cantharidin but is known to contain this compound[16]. During copulation the cantharidin in the ASG of the male is transferred with the seminal materials to the female[15]. The function of the transferred JH remains unknown. Allatectomy clearly demonstrates that JH plays no role in reproductive processes of the Cecropia silkmoth[17,18].

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Effect of experimental diabetes on estradiol binding by the anterior pituitary and hypothalamus in ovariectomized rats

H. Coirini, L. Weisenberg, S. Tornello and A.F. De Nicola

Laboratorio de Esteroides, Instituto de Biologia y Medicina Experimental, Obligado 2490, 1428 Buenos Aires (Argentina), *17 August 1979*

Summary. In diabetic rats, ^{3}H-estradiol binding by the cytosol and nuclei of the anterior pituitary was lower than normal. Changes in affinity and receptor numbers were demonstrated by Scatchard analysis. Protein synthesis in diabetic pituitaries, however, was in the normal range.

Induction of diabetes in female rats is followed by abnormalities in their reproductive function, manifested by alterations of the sex cycle, persistent anestrus and impaired fertility[1-3]. These changes were ascribed partly to primary ovarian disease[4], although recent work has focused on alterations in the regulation of ovarian activity by the central nervous system (CNS) and in the central action of steroids in diabetes. In this latter context, Denari and Rosner[5] first demonstrated a reduction in estrogen uptake in the CNS and anterior pituitary (AP) obtained from diabetic animals, whereas Gentry et al.[6] reported decreased nuclear uptake, but no changes in whole tissue uptake or cytoplasmic binding of estrogens in CNS areas and AP from diabetic rats. In this report, we confirm the changes in nuclear uptake[6], and show in addition that cytosol binding of estradiol in vitro is consistently lower in the AP, but not in the hypothalamus, from ovariectomized, streptozotocin-diabetic rats. These changes in AP estrogen binding are not due to impaired protein synthesis.

Materials and methods. Adult Wistar rats were ovariectomized and 1 week afterwards, diabetes was induced by the injection of 90 mg/kg of streptozotocin i.v., prepared as described by Junod et al.[7]. The animals were used 1 month after diabetes induction, at which time glycosuria was > 2% and glycemia measured over 400 mg%.

The estradiol binding assay was performed as described by Weisenberg et al.[8]. After decapitation of the animals, the AP and hypothalamus were homogenized in 0.010 M Tris