Tyr(Me)AVP disrupts the circadian rhythm of food intake when injected into SCN. The disruption seen in this study was transient and was neither photoperiodic nor dose-dependent.

VP has been implicated in many of the central integrative processess[21] in addition to its classical role in water and electrolyte metabolism. Recently it has been suggested that VP may have a role in stress-induced feeding as well[22]. The understanding of its role in the control of circadian rhythms has come far from studies[20,23,24] in brattlebore rats only, which are deficient in VP synthesis. However, the presence of a separate neuroanatomical system responsible for the circadian cerebrospinal fluid VP rhythm, as suggested by Schwartz and Reppert[25], and its effective insulation from osmotic regulation of blood VP[26] makes this peptide important in circadian time-keeping.

In view of the report that ethanol can alter the electrical activity of some brain areas[27,28] it is likely that ethanol injection may also alter the activity of SCN neurons and thereby disrupt the circadian rhythm.

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Embryotoxicity of RU 486 in English albino rabbit, *Oryctolagus cuniculus*

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The synthetic steroid RU 486, when administer orally at daily doses of 6.4 mg, 32.0 mg, and 160.0 m rabbit (low, high and toxic dose respectively) during t period of organogenesis to CDRI colony-bred ad female rabbits, caused 100% resorptions in all treat groups. Control animals had no resorptions.

RU 486, a synthetic progesterone-receptor blocker, c be effectively used for termination of pregnancy[1]. It advisable that a distinction be made between : extended pharmacological effect and its teratoger effect on the embryo[2-7]. This communication repo the results of an embryotoxic evaluation of RU 486.

Colony-bred adult nulliparous female rabbits we mated to bucks of proven fertility. Copulation w observed, confirmed by the presence of sperms vaginal smears and the day when presence of sperm w noticed first was designated day zero of pregnancy. Tl mated rabbits were divided into 4 groups of 5 anima each and the compound was administered orally fro day 6 to day 15 post-coitus as follows: group I, contr 1% gum acacia; group II, low-dose group, contr ceptive dose (CD), 6.4 mg/rabbit/day; group III, higl dose group, CD × 5, 32.0 mg/rabbit/day; group IV, toxi dose group, CD × 25, 160.0 mg/rabbit/day.

Body weight of all animals was recorded on days 1, 14, 21, 28 and 30 post-mating. On day 30 post-coitu caesarian sections were performed on all animals ar the number of corpora lutea; number of implantation implantation sites; number of resorptions; number live/dead foetuses; size, weight and gross abnormality each foetus; and viability, growth and deformities newborns were recorded.

Half of the foetuses were fixed in Bouin's solutic and were examined for visible abnormalities by tl sectioning method[3]. The remaining foetuses we cleared in 1% KOH solution and stained by Dawson Alizarin Red technique for visualization of osseo defects[3].

None of the mothers showed any noticeable deviatic in food intake throughout the experimental perio There was no mortality in any of the groups. There w steady gain in body weight of all animals of all group

Table 1. Effect of RU 486 on pregnancy in rabbits.

	Group I	Group II	Group III	Group IV
Dose	None (1% gum acacia)	6.4 mg/rabbit/day	32.0 mg/rabbit/day	160.0 mg/rabbit/day
Animals (n)	5	5	5	5
Total no. of implantations/implantation sites	26	25	24	19
Average no. of implantations/implantation sites per rabbit	5	5	5	5
Total no. of live births	23	Nil	Nil	Nil
Average no. of live births	5	Nil	Nil	Nil
Total no. of still births	Nil	Nil	Nil	Nil
Average no. of still births	Nil	Nil	Nil	Nil
Total no. of resorptions	3	25	24	19
Average no. of resorptions	1	5	5	4
Average foetal weight (g)	36.83	Nil	Nil	Nil
Average crown–rump length (cm)	5.05	Nil	Nil	Nil

though treated animals gained less weight compared to control animals. Maternal gain in weight in treated animals was dose-related. Whole foetus examination, Alizarin Red preparation for skeletal defect examination, and slicing method of Wilson for visceral defect examination revealed that (i) foetuses were not formed in any of the drug-treated groups, and only implantation sites were seen; and (ii) none of the foetuses of control group showed any gross or visceral defects. The results are summarized in Table 1.

RU 486 is a recently synthesized steroid with potent antiprogesterone properties, and presumably acts as a progesterone antagonist by blocking progesterone receptors. It is a proven effective medication for non-surgical termination of pregnancy. Local action of the compound on the endometrium quickly induces menstruation, though the exact dose regimen has not yet been established. In a clinical trial with women, the dose regimen employed (ranging from 100 mg/day × 7 days to 200 mg/day × 4 days or 400 mg/day × 4 days) terminated early pregnancy. It was found that lower dose for longer duration has a higher success rate than higher dose for shorter period[1].

The reason for lower effectiveness of a high-dose regimen is not known. If there is undue toxicity in early pregnancy, the embryo dies, is resorbed, and only the presence of the site of implantation is indicated[8]. For obvious reasons, this is termed resorption. In our study we observed implantation sites in the uteri of drug-treated rabbits. However, administration of RU 486 in low, high and toxic doses (6.4 mg, 32.0 mg and 160.0 mg/rabbit respectively) during the period of organogenesis produced 100% resorptions in all the animals. We therefore conclude that RU 486 is an effective embryotoxic agent at all the doses used in the present study.

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Effect of 2-deoxy-D-glucose on HeLa cells

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The effect of 2-deoxy-D-glucose (2-DG), an inhibitor of glycolysis and glucose transport, on growth and survival of unirradiated and UV-irradiated HeLa cells was investigated. Addition of 5 mM 2-DG to cultures resulted in reduction of the number of viable cells to 18.5% of that of control. 2-DG (2.5 mM) also increased cell mortality in UV-irradiated cultures.

2-DEOXY-D-GLUCOSE (2-DG) is a known glucose anti-metabolite and an inhibitor of glycolysis[1,2]. 2-DG can act in a number of ways, the chief route of action being in its capacity to inhibit competitively both phosphorylation (hexokinase) and transport of glucose[3–6]. Catabolism of cellular nucleotides (chiefly adenosine) to nucleosides and bases[7] is another route of action of destabilizing the cellular energy system[8–10].

Further, 2-DG has been shown to inhibit repair of