FERTILITY RESTORATION IN THE CYTOPLASMIC GENETIC MALE STERILE COTTON (*GOSSYPIUM HIRSUTUM* L)

M. V. THOMBRE
Department of Botany, Mahatma Phule Agricultural University, Rahuri 413 722, India

THOMBRE and Mehetre[1] reported cytoplasmic genetic male sterility in the Okra leaf *G. hirsutum* Cotton GH 572/76. It was indicated that the sterility in this source was due to two duplicate recessive genes. This male sterile based on the *G. hirsutum* cytoplasm has been maintained at the Cotton Project, of this University. Male sterility from this source has been transferred to a number of *G. hirsutum* lines. The original male sterile as well as the converted lines are not influenced by the environmental variations. It is thus similar to the cytoplasmic genetic sterility in *G. hirsutum* cotton on *G. harknessii* cytoplasm reported by Rosales and Davis[2] and Stith[3]. The sterility from GH 572/76 transferred in the first back-cross becomes stable. The flowers in all the male sterile lines are normal in all respects except that their stamens are consistently reduced.

During the course of testing different American cotton genotypes for fertility restoration on these lines, an exotic strain of *G. hirsutum* cotton, Ath 1/82 restored full fertility in the converted line ms Laxmi. The F_1s of this cross Laxmi × ms Ath 1/82 were selfed in 1984 and 14 F_2s were raised in 1985 to observe segregation for fertility and sterility. Seven of these lines showed segregation 3 fertile : 1 sterile and the remaining seven lines showed segregation 15 fertile : 1 sterile. This needs to be confirmed in F_3 but the present results indicate that the fertility obtained in these plants is also due to two duplicate dominant genes. The *P* value ranged between 0.90 and 0.95 confirming the earlier observation in this regard. All the converted male sterile lines showed 50–70 % setting as that of the normal plants, when alternated with fertility restoring lines. Insects appear to play a major role in the natural cross-pollination in this case as reported by Davis[4]. The present cytoplasmic genetic male sterility system on *G. hirsutum* cytoplasm is considered a good addition to the existing two gene-controlled *G. harknessii* male sterility system as it offers scope to reduce dependence on a single cytoplasm source.

The work on testing performance of F_1 hybrids developed by the use of Ath 1/82 pollen and transfer of the fertility restoration factors to the prospective male parents of *G. hirsutum* hybrid combinations is in progress.

The author thanks Sarvashri R. B. Sonawane, D. B. Shete and V. T. Jarad of the Cotton Project for their help.

20 July 1985

1. Thombre, M. V. and Mehetre, S. S., *Curr. Sci.*, 1979, **48,** 112.
2. Rosales, F. E. and Davis, D. D., *Crop, Sci.*, 1976, **16,** 99.
3. Stith, L. S., *Proc. Beltwide Cotton Prod. Res. Conf. Natl. Cotton Conf.*, 1974, 145.
4. Davis, D. D., *Adv. Agron.*, 1979, **30,** 129.

EFFECT OF PROLACTIN ON NUTRITIONAL STRESS-INDUCED IMPLANTATION FAILURE IN LABORATORY MICE

S. C. SAHU and C. J. DOMINIC
Department of Zoology, Banaras Hindu University, Varanasi 221 005, India.

IT is well established that the pre-implantation stage of pregnancy is extremely vulnerable to a variety of stimuli, both spontaneous and induced[1–4]. Nutritional stress during the pre-implantation period causes the death of blastocysts in mice[5–8]. The hormonal changes during nutritional stress resulting in implantation failure have not been elucidated, although it is suggested[6] that food deprivation may contribute to a decrease in hypophysial gonadotrophin secretion. In the present study, the ability of exogenous prolactin to maintain pregnancy in newly inseminated mice deprived of food for 48 hr was evaluated.

All mice used in this investigation were albinos belonging to the Parkes (P) strain. They were housed under standard laboratory conditions and maintained on pelleted food (Hindustan Lever Ltd) and water *ad libitum*. Females were 10-week-old virgins; they were paired with males and on finding the vaginal plug (day 1 post coitum) separated from the latter and housed individually in cages, 40 × 15 × 10 cm. Twenty four hours later (day 2) they were divided into three main groups and treated as follows: *Group I*: Food deprivation for 48 hr beginning at 10.00 hr on day 2 and injection of prolactin (NIH-ovine), 2 mg/female/day (Group IA) or normal saline, 0.1 ml/female/day (Group IB) on days 2 to 6. *Group II*: Food deprivation for 48 hr beginning at 10.00 hr on day 4 and injection

of prolactin, 2 mg/female/day (Group IIA) or normal saline, 0.1 ml/female/day (Group IIB), on days 4 to 8. *Group III*: Left undisturbed after separation from the stud males.

All injections were given intramuscularly. During food deprivation water was available *ad libitum* to the females. Vaginal smears were examined daily from all females up to day 9 (group I) or day 11 (group II and III) post coitum and a return of vaginal cornification within these periods was taken to indicate an implantation failure. Females which failed to exhibit vaginal cornification were observed up to day 21 post coitum; those which did not deliver young were presumed to be pseudopregnant.

The results are presented in table 1. Prolactin administration effectively prevented the implantation failure that normally follows food deprivation; females in which fasting was initiated on day 4, prolactin administration provided complete protection against the nutritional stress-induced implantation failure. By contrast, food-deprived females treated with saline (groups IB and IIB) exhibited a high rate of implantation failure. The length of gestation and litter size in prolactin-treated females (groups IA and IIA) were comparable to those in untreated females (group III).

It is obvious that administration of exogenous prolactin prevents implantation failure in food deprived mice. It is not known whether this effect of prolactin is mediated through alterations in the levels of FSH and LH. However, it should be noted that treatment with chorionic gonadotrophin does not succeed in maintaining pregnancy in fasted female mice beyond day 15 post coitum[6]. This suggests that depression of hypophysical gonadotrophic activity may not play a significant role in fasting-induced implantation failure. Bruce[7] reported a slight decrease in implantation failure when newly inseminated mice were housed in groups during food deprivation. Since female mice in unisexual groups mutually stimulate hypophysical prolactin secretion[9-11], it appears probable that the decrease in the incidence of implantation failure in females housed in groups during food deprivation may be due to the increased luteotrophic activity of the pituitary. The induction of implantation and maintenance of pregnancy to term in prolactin-deficient genetically dwarf mice by exogenous prolactin[12] further emphasize the key role of prolactin in implantation and pregnancy in mice. It thus appears reasonable to conclude that depression of hypophysical prolactin secretion may play an important role in fasting-induced implantation failure in mice. It is of interest to note that depression of hypophysical prolactin release is the primary endocrine factor involved in the male-induced implantation failure (the Bruce effect) in newly inseminated mice[13].

Table 1 *Effect of Prolactin on fasting-induced implantation failure*

Group and treatment*	Proportion and % of females		
	With implantation failure**	Remaining pregnant	Remaining pseudo-pregnant
IA. Fasting started on day 2 + proclatin	1/20 (5.0)	18/20 (90.0)	1/20 (5.0)
IB. Fasting started on day 2 + saline	16/21 (76.2)	5/21 (23.8)	Nil
IIA. Fasting started on day 4 + prolactin	Nil	22/22 (100.0)	Nil
IIB. Fasting started on day 4 + saline	20/21 (95.2)	1/21 (4.8)	Nil
III. Untreated and undisturbed	Nil	18/18 (100.0)	Nil

* For details see text
** Significance of differences: IA *vs* IB, $P < 0.001$; IIA *vs* IIB, $p > 0.001$.

The investigations were supported by grants from the ICMR and UGC, New Delhi.

1 June 1985; Revised 1 October 1985

1. Loeb, L., *Biol. Bull.*, 1917, **33,** 91.
2. Jonen, P., *Z. Geburtsch. Gynak.*, 1931, **101,** 50.
3. Leathem, J. H., In: *Recent progress in the endocrinology of reproduction*, (ed.) C. W. Lloyd, Academic Press, New York, 1959, p. 179.
4. Blandau, R. J., In: *Sex and internal secretions*, 3rd edition, Vol. 2, (ed.) W. C. Young, Williams and Wilkins Co, Baltimore, 1961, p. 797.
5. McClure, T. J., *Nature* (*London*), 1958, **181,** 1132.
6. McClure, T. J., *J. Reprod. Fert.*, 1962, **4,** 241.
7. Bruce, H. M., *J. Reprod. Fert.*, 1963, **6,** 221.
8. Sahu, S. C. and Dominic, C. J., *Adv. Biosci.*, 1985, **4,** 9.
9. van der Lee, S. and Boot, L. M., *Acta Physiol. Pharmacol. Neerl.*, 1956, **5,** 213.
10. Ryan, K. D. and Schwartz, N. B., *Endocrinology*, 1980, **106,** 959.
11. Gangrade, B. K., *Pheromonal influences on the oestrous cycle of the laboratory mouse*, Ph.D. Thesis, Benaras Hindu University, 1983.
12. Bartke, A., *Biol. Reprod.*, 1973, **9,** 373.
13. Dominic, C. J., *J. Reprod. Fert.*, 1966, **11,** 415.