Overall, when (gross) intake rates were high (mean intake rate = 1.14 mg/s, SE = 0.11, $n = 4$ populations) females rarely fed fledglings and crossbills continued to nest. When intake rates were lower, such as near the end of extended nesting periods (mean intake rate = 0.84 mg/s, SE = 0.09, $n = 5$ populations) or when individual crossbills nested successfully once (mean intake rate = 0.88 mg/s, SE = 0.08, $n = 3$ populations), both males and females fed fledglings. This pattern of parental care where females desert fledglings from early nests (but not late nests) occurs in other passerine species (Oring 1982), including other cardueline species (Newton 1972).

These data are consistent with the hypothesis that female crossbills desert their fledglings to renest, but direct evidence of females renesting was not obtained. Desertion to renest has been reported in other species (e.g. Grant and Grant 1987). Nevertheless, if female crossbills did not attempt to renest, there is no compelling reason why females rather than males should consistently desert. On the other hand, if there is renesting, then the female instead of the male should desert. This follows because only females build nests, females alone are capable of incubating eggs in crossbills (Newton 1972), and deserting females should be able to find another mate, because males generally outnumber females (Newton 1972, pers. obs.). For the same reason, males are unlikely to find another mate.

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An Effective Anti-estrogen for Feral Birds

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Estrogens in the plasma of both sexes of birds correlate with a variety of reproductive and behavioral functions (e.g. Lehrman and Brody 1957, Hinde 1965, Wingfield and Farner 1978, Moore 1983, Pröve 1983, Hutchison et al. 1984, Marler et al. 1988). To elucidate the functional role of estrogens (in particular, 17β-estradiol), endogenous levels in both sexes can be supplemented through estradiol-filled silastic implants. Conversely, estradiol titers can be reduced by gonadectomy, although this procedure does not completely suppress estradiol levels in males (Marler et al. 1988).

Anti-estrogenic drugs are an alternative method to eliminate the effects of estradiol in both sexes. I suppressed estradiol-induced oviduct growth in non-

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TABLE 1. Oviduct mass (g) and plasma estradiol levels (pg/ml; before and after implants) in female Song Sparrows ($n = 5$ in each group) treated with tamoxifen ($\bar{x} \pm$ SE).

	Group 1	Group 2	Group 3	Group 4
Oviduct mass	26.2 ± 3.2	22.0 ± 2.5	416.3 ± 56.0	24.8 ± 5.9
Estradiol				
Before	216[a] ± 37	243 ± 53	179 ± 27	265 ± 44
After	565 ± 112	479 ± 70	385 ± 29	165 ± 37

[a] $n = 4$.

domesticated adult passerine females using the anti-estrogen tamoxifen. I used females in this experiment because there is not yet any readily accessible male characteristic, either behavioral or physiological, which is known to be estradiol dependent.

Twenty female Song Sparrows (*Melospiza melodia*) were used in the experiment. Seventeen were captured in the wild between 3–15 April 1988 in grain-baited potter traps ($n = 16$) or in Japanese mist nets ($n = 1$). The remaining 3 females had been hand-reared from the egg the previous year. All birds were kept in individual cages on natural photoperiod, and wild-caught birds were housed for at least 1 month prior to the experiment.

I divided the birds into four groups of five birds. A small blood sample (ca. 200 μl whole blood) was taken from the wing vein of each bird in heparinized capillary tubes. The tubes were sealed and centrifuged at 2,000 rpm for 5 min, and the plasma was removed and stored at −20°C until assayed.

The following day all birds in Group 1 were given a 12-mm implant of silastic tubing (Dow Corning; i.d. = 0.147 cm, o.d. = 0.196 cm) packed with estradiol and sealed at each end with medical adhesive (Dow Corning, Silicone Type A). Reproductive tract development in female birds often is adversely affected by captivity (e.g. King et al. 1966), and the estradiol-filled implants were given to insure that suppression of oviducal mass was not due simply to a lack of sufficient hormone. The following morning injections of the anti-estrogen were begun and continued daily for 10 days. Birds in Group 1 received a dose of anti-estrogen of 25 mg/kg body mass. The day after the injection period, a second blood sample was taken from each bird. The birds were sacrificed immediately and the wet oviducal mass determined to the nearest 0.1 mg.

Group 2 was treated the same as Group 1, except that the daily injected dose of tamoxifen was 50 mg/kg. Group 3 was also treated like Group 1, except that these birds received no anti-estrogen, only the estrogen implant and injections of vehicle. Finally, Group 4 birds received empty silastic implants plus injections of vehicle.

For the injections, the anti-estrogen tamoxifen citrate was weighed out daily and dissolved in methanol, with a final volume of 20 μl methanol per injection. The tamoxifen-methanol solution was injected from a 100 μl fixed-needle glass syringe (Hamilton, 710N). Injections initially were intramuscular; however, because of suggestions of vehicle-induced tissue damage, injections were made subcutaneously after the fourth day. This reduced (but did not eliminate) the irritation caused by the methanol. Tamoxifen is not miscible in water, but will dissolve in organic solvents such as methanol. Methanol can cause tissue damage which could, over time, become a health problem to the birds. Tamoxifen is known to be free of side effects (Furr et al. 1979). Studies have shown that a smaller absolute volume of solvent and multiple injection sites further reduce tissue damage (S. Peters pers. comm.). In addition, an increase in the time interval between injections may be possible, because the half-life of tamoxifen in mammalian species ranges from 24–53 h (Furr et al. 1979).

I measured plasma levels of estradiol in the blood samples in a single radioimmunoassay after partial purification on Celite:glycol microcolumns (Wingfield and Farner 1975). Intra-assay controls were within acceptable limits: Two water blanks each measured <2 pg/ml estradiol; and an accuracy tube, to which 250 pg of estradiol had been added, measured 223 pg. One of the pre-implant samples from Group 1 was lost during the assay.

The control females (Group 4), given neither estradiol nor tamoxifen, exhibited low estradiol levels and showed correspondingly minor oviducal development (Table 1). In contrast, birds in Group 3, given estradiol-filled silastic implants (but no tamoxifen), exhibited extensive oviduct growth. This demonstrated that captive female Song Sparrows retain the capacity to respond to exogenous estradiol. Tamoxifen completely suppressed estradiol-induced oviduct growth (Table 1). Oviduct mass in groups 1 and 2 were similar to those in Group 4. These three groups had significantly lower oviduct mass than Group 3 birds given estradiol alone ($H = 11.07$, 3 df, $P < 0.02$, Kruskal-Wallis).

Tamoxifen competes with estradiol for access to receptors in estrogen-sensitive peripheral target tissue (for references, see Furr et al. 1979). I found suppression of estradiol-induced oviducal growth in the two tamoxifen-injected groups despite the fact that they, like the Group 3 females, exhibited signif-

icant increases in plasma estradiol levels over the course of the experiment because of the hormone implants (Table 1; Mann-Whitney $U = 0$ for Group 1, $P = 0.016$; Group 2: $U = 2$, $P = 0.032$; Group 3, $U = 0$, $P = 0.008$; Group 4: $U = 5$, P = NS).

Tamoxifen may well prove to be a useful research tool in manipulating the effects of endogenously produced estradiol in feral birds, although its central effects may differ from those in the periphery (Mathews et al. 1988). Its value in studies with females will be manifest when ovariectomy is impractical or undesirable. But tamoxifen's greatest value may be in studies with males, where the source of estradiol secretion is still unknown but may be nontesticular (Marler et al. 1988). Tamoxifen may facilitate the suppression of estradiol effects in males without having to remove its source.

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