Oviposition Delays Induced by Social Stress Are Reversed by Treatment With the \(\beta\)-Adrenergic Blocking Agent Propranolol

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ABSTRACT Contact with unfamiliar conspecifics (social stress) caused hens to delay oviposition. These delays were prevented by propranolol (10 mg/kg), suggesting that \(\beta\)-adrenergic receptors are involved in their physiological causation.

(Key words: hen, stress, propranolol, \(\beta\)-adrenergic receptor, oviposition delays)

INTRODUCTION

Environmental stressors, such as relocation, exposure to unfamiliar conspecifics, and removal of nest sites, can cause hens to delay oviposition (Duncan, 1970; Hughes, 1979; Hughes et al., 1986; Watt and Solomon, 1988). As these delays cause characteristic eggshell abnormalities that result in financial loss for commercial egg producers, and also provide an index of stress in hens (Hughes et al., 1986), it is of interest to understand their physiological causation. It has been suggested that adrenalin released in response to stress may delay oviposition by suppressing uterine contractions (Weiss and Sturkie, 1952; Sykes, 1955; Hughes and Black, 1976).

Within the smooth muscle of the hen’s oviduct there are \(\alpha\)- and \(\beta\)-adrenergic receptors, stimulation of which causes contraction and relaxation, respectively (Verma and Walker, 1974). As adrenergic receptors in the uterus are predominantly \(\beta\), adrenergic stimulation causes relaxation (Verma and Walker, 1974; Verma et al., 1977; Crossley et al., 1980; Wechsung and Houvenaghel, 1987). Consequently, exogenous adrenalin causes hens to delay oviposition (Sykes, 1955; Crossley, 1983; Hughes and Gilbert, 1984). These delays can be prevented by including propranolol, a \(\beta\)-adrenergic receptor blocker, in the diet (Crossley, 1983). It has been speculated that adrenalin released in response to stress may act through \(\beta\)-adrenergic receptors to suppress contractions of the uterus and thereby delay oviposition (Crossley, 1983). Such a mechanism may underlie oviposition delays caused by saline injection, as these are prevented by propranolol (Crossley, 1983).

The aim of the following study was to investigate the role of \(\beta\)-adrenergic receptors in the causation of oviposition delays induced by an environmental stressor—contact with unfamiliar conspecifics (social stress). If oviposition delays are caused by \(\beta\)-adrenergic receptor-mediated suppression of uterine contractions, then blocking these receptors with propranolol should reduce the duration of such delays.

MATERIALS AND METHODS

ISA Brown hens \((n = 31)\), aged 24 wk and weighing 1.7 to 2.3 kg \((\text{mean} = 1.9 \text{ kg})\), were housed individually in alternate cages \((0.29 \text{ m wide}, 0.46 \text{ m deep}, 0.43 \text{ to } 0.51 \text{ m high})\) of a battery. Lights were on from 0900 to 2300 h and feed and water were provided for ad libitum consumption. From a record of daily oviposition times it was possible to predict the time of each hen’s next oviposition (“predicted oviposition time”).

Hens were randomly divided between four treatments: 0.9% saline injection \((n = 7)\), 0.9% saline injection followed by social stress \((n = 8)\), 10 mg/kg propranolol injection followed by social stress \((n = 8)\), or 10 mg/kg propranolol injection \((n = 8)\). Propranolol \((\pm \text{ propranolol hydrochloride}^1)\) was dissolved in 1 M acetic acid and then diluted with 0.9% saline to a concentration of 20 mg/mL. Both the propranolol and 0.9% saline solutions were adjusted to pH 6.0 by titration with sodium hydroxide and acetic acid. Injections of 0.5 mL/kg were made into the pectoral muscle, 40 min before predicted oviposition time. Immediately after injection, each hen selected to receive social stress was placed in a group cage \((1.16 \text{ m wide}, 0.46 \text{ m deep}, 0.50 \text{ to } 0.58 \text{ m high})\) together with three unfamiliar hens \((57\text{-wk-old ISA Browns})\). These injected hens were returned to their original cages once they laid or after a period of 240 min if laying did not occur.

As hens were tested in the middle of egg sequences, it could be assumed that oviposition time would advance
by a similar increment (this was up to 40 min for the hens in the present study) each day (Etches, 1990). Therefore, expected oviposition time for the day of treatment (Day n) was retrospectively calculated as the mean of the oviposition times on the 2 adjacent d (Days n – 1 and n + 1). For the day of treatment, duration of oviposition delay was calculated by deducting expected oviposition time from actual oviposition time. It should be noted that expected oviposition time is a more precise estimate of when hens would have laid (had there been no treatments) than is predicted oviposition time.

One hen receiving propranolol plus stress and another receiving propranolol alone laid just before they were about to be injected and so were excluded from the analysis. Four hens, all receiving saline plus stress, did not lay during social stress and were considered to have oviposition delays of 180 min for the analysis. To allow for these maximum values, a nonparametric statistical analysis (Mann-Whitney) was used.

**RESULTS**

Mean duration of oviposition delay for each treatment and the statistical comparisons between these are shown in Figure 1. A comparison of saline alone and saline plus stress treatments showed that social stress had a significant delaying effect on oviposition time. Propranolol prevented these stress-induced oviposition delays. Propranolol alone caused ovipositions to occur earlier than expected, an effect that was significant when compared with all other treatments.

**DISCUSSION**

That relocation and contact with unfamiliar conspecifics (social stress) caused hens to delay oviposition is consistent with previous reports (Hughes, 1979; Watt and Solomon, 1988; Reynard and Savory, 1995). The major finding of the present study is that propranolol prevented these oviposition delays. As adrenergic receptors in the hen’s uterus are mainly β, adrenergic stimulation causes relaxation and so delays oviposition (Crossley et al., 1980; Crossley, 1983). Hence, it has been speculated that oviposition delays induced by environmental stress may be caused by the release of adrenalin, which acts through β receptors to relax the uterus (Crossley, 1983). The present study suggests that β-adrenergic receptors are involved in the physiological mechanisms that delay oviposition during social stress and so supports this hypothesis. By comparison, β-adrenergic receptors have also been implicated in the mechanisms underlying stress-related disruptions to parturition in mammals (Bostedt and Rudloff, 1983). Propranolol acts to reduce anxiety in humans (Bowman and Rand, 1980) and could presumably have a similar tranquillising effect in hens, which may contribute towards its ability to prevent stress-induced oviposition delays.

As adrenergic receptors in the utero-vaginal sphincter are predominantly α, adrenergic stimulation causes contraction (Sykes, 1955; Verma et al., 1977). Presumably adrenalin released in response to stress could cause contractions of the utero-vaginal sphincter that contribute towards the delaying of oviposition by obstructing the egg’s passage.

Although it has been reported that saline injections cause oviposition delays (Crossley, 1983), this was not observed in the present study. Possibly the larger injection volume (4 mL per hen) and subcutaneous route described by Crossley (1983) was more stressful than the intramuscular injection of about 1 mL per hen in the present study.

In hens not exposed to social stress, propranolol caused oviposition to occur earlier than expected, an effect not observed when propranolol is included in the hens diet (Crossley, 1983). As propranolol blocks β receptors, adrenergic stimulation of the uterus will be by α receptors only and so will cause contraction (Crossley et al., 1980). Perhaps circulating adrenalin, the concentrations of which may be elevated by stress associated with the injection procedure or the imminent oviposition, acts through α receptors to cause contractions of the uterus that result in premature oviposition. Consistent with this suggestion, it has been reported that propranolol promotes uterine contractions in hens that are not carrying eggs (Verma and Walker, 1974; Murayama et al., 1980).

In conclusion, the present study demonstrates the involvement of β-adrenergic receptors in the causation of stress-induced oviposition delays in hens. This finding is of interest because eggs with shell abnormalities caused by oviposition delays are downgraded and can be used as an index of stress in hens (Hughes et al., 1986).
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REFERENCES


