The effect of sodium nitrate on escaping behaviour – rapid feather release regulation in Japanese quail (*Coturnix coturnix japonica*)

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The effect of sodium nitrate (0.87 g per kg of food, 14–30 d before testing) on feather shedding in male quails evoked by the dopamine agonist apomorphine (0.5–0.75 mg/kg) and the cholinergic muscarinic receptor antagonist atropine (6.7––20.0 mg/kg) administered separately and in combination with progesterone (5 mg per bird) was investigated.

Apomorphine-induced feather release tended to be enhanced by nitrate. The effect of apomorphine was more pronounced under pretreatment with clonidine (0.005 mg/kg), PCPA (120 mg/kg) and diazepam (6.7 mg/kg) in particular. Moreover, nitrate diminished the negative effect of stress on apomorphine feather release, although yohimbine-(3–4 mg/kg)-induced inhibition of apomorphine effect was not influenced by nitrate.

Sodium nitrate had not enhanced physostigmine (0.075–0.1 mg/kg) and neostigmine (0.075 mg/kg) evoked suppression of apomorphine-induced feather release. However, sodium nitrate diminished the atropine-induced feather release. Augmentation of apomorphine-induced feather release by progesterone was attenuated under sodium nitrate treatment as well. Suppression of atropine effect by sodium nitrate was not abolished by progesterone, it was only diminished.

Thus, the results have shown that neural regulation of extreme escaping behaviour in male quails can be affected by small amounts of sodium nitrate in food. The results have revealed two opposite effects of sodium nitrate on separate links of the regulatory mechanism in the reaction of rapid feather shedding.

Key words: bird, rapid feather release, $NaNO_3$, apomorphine, atropine

INTRODUCTION

Animal behaviour is susceptible to various changes in the environment. As have been shown in our previous investigation, small amounts of nitrate in food, which had no any noticeable deleterious effect on bird's postnatal development, induced some changes in its social and individual behaviour [17] and changed the influence of neurotransmitters on the activity of separate behavioural expressions [18].

The rapid feather shedding reaction is one of the extreme escaping reactions. It occurs some bird species to escape a predator which is in close contact with the pray. This reaction is called fright molt and is a form of protective adaptation [9]. It can be attributed to the phenomenon of autotomy. This reaction we have observed in Japanese quail as well, but it was displayed in small number of birds only. In our recent works [20] the role of dopaminergic and cholinergic neurotransmission in rapid feather release has been revealed and the influence of the hormone progesterone on the effect induced by these neurotransmitters systems has been shown [19].

The objective of the present study was to estimate the possibility of small amounts of nitrate to influence the extreme escaping reaction in birds – rapid feather shedding in Japanese quail, evoked by the dopamine receptor agonist apomorphine and cholinergic muscarinic receptor antagonist atropine under their separate action and in combination with progesterone and other neuroactive agents.

MATERIALS AND METHODS

Subjects. Adult male Japanese quails (*Coturnix coturnix japonica*) weighing 120–170 g served as subjects. The quails were hatched from eggs obtained from the commercial suppliers. The birds were housed in groups, 5–7 per cage ($0.62 \times 0.54 \times 0.66$ m) and had free access to food and water.

Compounds. Sodium nitrate (0.87 g per kg of food) was mixed with fowl food. Quails received sodium nitrate 14–30 days before testing feather shedding.

Apomorphine hydrochloride (0.5–0.75 mg/kg), physostigmine salicylate (0.075–0.1 mg/kg), neostigmine methylsulphate (0.075 mg/kg) (each of these drugs was immediately dissolved before each injection), clonidine (Haemiton, 0.005 mg/kg), yohimbine HCl (3–4 mg/kg), diazepam (Seduxen, Gedeon Richter, 6.7 mg/kg), atropine sulphate (6.7–20.0 mg/kg), parachlorphenylalanine (PCPA, Serva, 120 mg/kg), progesterone (Jelfa, Poland; Monte Farmaco, Italy, 5 mg per bird). The drugs were dissolved in physiological saline and injected subcutaneously in a volume 0.25–0.5 ml.

To investigate the effect of a single drug, apomorphine was administered 5–15 min, atropine 9–15 min, clonidine 10–30 min, yohimbine 60 min, physostigmine and neostigmine 7–13 min, diazepam 10–20 min, progesterone 2 h before testing.

Investigating these compounds in combination, the time between pretreatment of separate drugs and apomorphine injections was as follows: by pretreatment with yohimbine 20–60 min, with physostigmine 17–24 min, with neostigmine 26–32 min, with clonidine 48–60 min, with diazepam 10–20 min, with PCPA 24 h. The length of time between pretreatment with progesterone and apomorppphine or atropine was 2 h. To investigate the effect of yohimbine, physostigmine and saline after apomorphine injection, apomorphine was injected 10–25 min, yohimbine 5–20 min, physostigmine 5–15 min, saline 5–20 min before testing.

Feather shedding test. The test were carried out between 10.00 a.m. and 2.00 p.m.

The bird was hold in human hand for 1 min. The movements of the birds were unrestricted. The feather shedding was tested by running the observer's hand lightly over the quail's feathers. The number of feathers shed by a separate quail was counted.

The results were analyzed by the Mann–Whitney U-test. The value of $P < 0.05$ was considered to indicate a statistically significant difference.

RESULTS

The influence of sodium nitrate on the dopamine agonist apomorphine-induced feather release in quail. Sodium nitrate alone failed to induce any noticeable effect on feather shedding, but under nitrate treatment, a tendency of increase in apomorphine feather release was observed (Table 1). This increase was more pronounced in quail groups treated with a higher doses of apomorphine and in the groups in which saline was injected before apomorphine treatment. Injection of saline before apomorphine markedly suppressed the apomorphine-induced feather release. Under nitrate treatment, the stressogenic effect of this injection was attenuated.

Thus, the results showed that sodium nitrate was able to enhance the apomorphine-evoked feather shedding in quails.

The influence of sodium nitrate on apomorphine feather release under pretreatment with noradrenergic, serotonergic and GABAergic agents.

In order to elucidate the possible mechanism underlying the nitrate effect on apomorphine-induced feather release, the role of other neurotransmitter systems in these processes was investigated. In this study the noradrenergic activity was decreased by clonidine and activated by yohimbine. The results demonstrated (Table 2) that apomorphine-induced feather release was statistically significantly augmented by nitrate only when noradrenergic activy was attenuated by clonidine. This nitrate + clonidine effect on apomorphine was reflected in the amount of feathers shed by quails that were investigated last in a quail cage group. The amount of feathers shed by those quails was similar to the amount of feathers shed by quails investigated as the first. In the group of apomorphine alone the amount of feathers shed by the quails investigated as the last was usually decreased. In the nitrate + apomorphine and clonidine + apomorphine groups an increase in the number of feathers shed by the quails investigated as the last was also observed, but it was less pronounced. Therefore, it seems that the augmentation of feather shedding in the group of nitrate + clonidine + apomorphine could be accounted for by a simple

Table 1. **Effect of sodium nitrate on the amount of feather shed by quails after apomorphine treatment (estimation was made after tonic immobility, TI)**

Dose mg/kg	Apomorphine			Nitrate + apomorphine		
	N	X ± m	Min–Max	N	X ± m	Min–Max
0	18	0.7 ± 0.3	0–3	15	0.6 ± 0.3	0–3
0.01	8	2.2 ± 1.5	0–11	8	1.1 ± 0.5	0–4
0.05	8	3.2 ± 1.2	0–10	8	4.0 ± 1.3	0–10
0.06*	10	3.3 ± 0.9	0–8	8	4.2 ± 1.9	0–16
0.1	6	10.8 ± 6.5	0–32	8	11.9 ± 4.2	0–30
0.6*	16	10.2 ± 3.6	0–55	16	23.5 ± 6.4	1–66
0.7	18	28.3 ± 6.6	3–80	18	34.5 ± 7.5	0–121
0.75	10	40.5 ± 17.5	3–170	10	54.8 ± 16.5	2–140
0.75*	16	11.5 ± 3.9	0–60	13	21.5 ± 5.9	2–70

* Apomorphin injection was preceded by saline injection.

Table 2. **Effect of sodium nitrate on the amount of feather shedding induced by apomorhpine under clonidine, yohimbine, PCPA and diazepam treatment**

	Groups, drugs	Dose, mg/kg	Without $NaNO_3$			With $NaNO_3$			Notes
			N	X ± m	Min–Max	N	X ± m	Min–Max	
I	Saline	0	18	0.7 ± 0.3	0–3	15	0.6 ± 0.3	0–3	After TI
	Apomorphine	0.5	16	5.2 ± 1.9	0–24	16	8.2 ± 2.2	0–29	After TI
	Clonidine + apomorphine	0.005 0.5	16	6.5 ± 1.0	0–27	16	9.6 ± 2.8[a]	0–34	After TI
II	Yohimbine	3.0	7	1.3 ± 0.5	0–3	8	1.7 ± 0.9	0–6	After TI
	Saline + apomorphine	0.75	16	11.3± 3.9	0–60	13	21.7 ± 5.9	2–77	After TI
	Yohimbine + apomorphine	3.0 0.75	15	4.6 ± 1.7[b]	0–23	16	4.7 ± 1.6[c]	0–24	After TI
III	Apomorphine + saline	0.6–0.7	7	35.8 ± 7.0	1–53	14	43.3 ± 7.7	18–102	
	Apomorphine + yohimbine	0.6–0.7 0.4	14	14.8 ± 3.9[d]	1–54	13	21.0 ± 7.3[e]	1–67	
IV	Apomorphine	0.7	18	20.0 ± 5.1	1–88	18	27.9 ± 5.2	1–70	After TI
	PCPA + apomorphine	120.0 0.7	18	26.6 ± 5.3	3–70	18	35.1 ± 8.3[f]	3–114	After TI
V	Apomorphine	0.75	10	40.5 ± 17.5	1–170	10	51.4 ± 12.5	2–140	After TI
	Diazepam + apomorphine	6.7 0.75	10	38.3 ± 9.2	2–73	10	67.5 ± 14.7[g]	8–130	After TI

Mann–Whitney U test:
a – with respect to apomorphine, $P < 0.05$, one-tailed; b – with respect to saline + apomorphine, $P < 0.05$, two-tailed; c – with respect to $NaNO_3$ + saline + apomorphine, $P < 0.05$, two-tailed; d – with respect to apomorphine + saline, $P < 0.05$, two-tailed; e – with respect to $NaNO_3$ + apomorphine + saline, $P < 0.05$, two-tailed; f – with respect to apomorphine, $P < 0.05$, one-tailed; g – with respect to diazepame + apomorphine, $P = 0.05$, two-tailed.

addition of the individual effect of nitrate and clonidine on apomorphine.

Table 2 shows that nitrate has neither attenuated nor increased the suppression of apomorphine-induced feather release, caused by yohimbine injected before or after apomorphine.

When treating with nitrate, PCPA (an inhibitor of serotonin synthesis) and apomorphine in combination, there was a synergistic action of nitrate and PCPA on apomorphine-induced feather release (Table 2). In the nitrate + PCPA + apomorphine group the percentage of birds showing more abundant feather shedding (>40) was 38.9%, in nitrate + apomorphine group 27.8%, and in PCPA + apomorphine group 16.7%.

Apomotrphine-induced feather release was significantly enhanced by nitrate under treatment with the GABA-benzodiazepin receptor agonist diazepam (Table 2).

Thus, apomorohine influence on feather release was increased under attenuation of noradrenergis or serotonergic activity and by activation of GABA benzodiazepine neurotransmission.

The influence of sodium nitrate on cholinergic regulation of feather release. As is seen in Table 3, the activation of cholinergic system by the acetylcholinesterase inhibitors physostigmine or neostigmine have evoked a marked decrease in apomorphine-induced feather release. The effect of physostigmine on apomorphine was weaker when the former drug was injected after apomorphine treatment. Some attenuation of physostigmine effect on apomorphine by nitrate was observed in groups treated with a high dose of apomorphine (0.75 mg/kg): under nitrate treatment 30% whereas without nitrate only 4.2% of quails shed more than 20 feathers. However this effect of nitrate seems to be independent on the attenuation of physostigmine influence, because at lower apomorphine doses this effect of nitrate was not observed. On the other hand, the results did not show nitrate to enhance the effect of physostigmine or neostigmine.

A rapid feather shedding induced by atropine, an antagonist of cholinergic muscarinic receptors, was reduced by sodium nitrate. Table 4 shows that sodium nitrate significantly diminished the atropine-induced feather release at all atropine doses investigated.

Thus, the results did not show that nitrate suppressed or activated the cholinergic influence on apomorphine feather release, but the atropine-induced feather release was reduced under sodium nitrate treatment.

Table 3. **The effect of sodium nitrate on the amount of apomorphine-induced feather shedding under physostigmine and neostigmine treatment**

	Groups, drugs	Dose, mg/kg	Without $NaNO_3$			With $NaNO_3$			Notes
			N	X ± m	Min–Max	N	X ± m	Min–Max	
I	Saline	0	10	0.9 ± 0.5	0–4	11	0.4 ± 0.2	0–2	After TI
	Apomorphine	0.75	23	48.4 ± 10.9	0–183	22	54.3 ± 8.7	2–140	After TI
	Physostigmine + apomorphine	0.1 0.75	18	6.9 ± 1.8[a]	0–23	20	15.1 ± 4.1[b]	0–64	After TI
II	Saline + apomorphine	0.6	16	10.4 ± 3.8	0–50	16	23.5 ± 6.6	1–54	After TI
	Neostigmine + apomorphine	0.075 0.6	15	2.3 ± 0.8[c]	0–10	16	3.4 ± 1.1[d]	0–8	After TI
III	Apomorphine + saline	0.6	7	35.8 ± 7.0	1–53	7	41.7 ± 13.1	18–102	
	Apomorphine + physostigmine	0.6 0.08	7	16.4 ± 5.6[e]	3–42	7	17.4 ± 7.2[f]	4–51	

Mann–Whitney U test, two tailed:
a – with respect to apomorphine, $P < 0.05$; b – with respect to $NaNO_3$ + apomorphine, $P < 0.05$; c – with respect to saline + apomorphine, $P < 0.05$; d – with respect to $NaNO_3$ + saline + apomorphine, $P < 0.05$; e – with respect to apomorphine + saline, $P < 0.05$; f – with respect to $NaNO_3$ + apomorphine+ saline, $P < 0.05$.

Table 4. **Effect of sodium nitrate on the amount of feather shed by quails after treatment with atropine**

Groups, drugs	Dose, mg/kg	Without $NaNO_3$			With $NaNO_3$		
		N	X ± m	Min–Max	N	X ± m	Min–Max
Saline	0	22	1.3 ± 0.6	0–13	24	1.4 ± 0.5	0–2
Atropine	6.7	6	7.3 ± 2.1	1–14	6	2.3 ± 1.2[a]	0–7
Atropine	13.3	10	18.1 ± 6.3	3–67	12	6.3 ± 2.3[b]	1–26
Atropine	20.0	6	16.1 ± 5.8	1–38	6	7.3 ± 0.8[c]	4–9

Mann–Whitney U test:
a – with respect to atropine 6.7 mg/kg, $P < 0.05$; b – with respect to atropine 13.3 mg/kg, $P < 0.05$; c – with respect to atropine 20 mg/kg, $P < 0.05$.

Table 5. **Effect of sodium nitrate on the amount of feather shed by quails under progesterone with apomorphine or atropine simultaneously treatment**

Groups, drugs	Dose, mg/kg	Without $NaNO_3$			With $NaNO_3$		
		N	X ± m	Min–Max	N	X ± m	Min–Max
Saline	0	10	0.6 ± 0.3	0–14	12	1.2 ± 0.5	0–15
Progesterone + saline	5 mg/bird	12	0.2 ± 0.2	0–2	13	0.4 ± 0.3	0–4
Apomorphine	0.75 mg/kg	14	28.9 ± 9.0	1–98	14	39.4 ± 12.4	1–134
Progesterone + apomorphine	5 mg/bird 0.75 mg/kg	14	1020.0 ± 16.3[a]	3–196	14	88.2 ± 19.3[b]	19–282
Atropine	13.3 mg/kg	10	18.1 ± 6.3	3–67	12	6.3 ± 2.3[c]	1–26
Progesterone + atropine	5 mg/bird 13.3 mg/kg	12	28.2 ± 7.2	0–74	13	10.6 ± 3.7[d]	1–36

Mann-Whitney U test:
a – with respect to apomorphine, $P < 0.05$, two tailed; b – with respect to $NaNO_3$ + apomorphine, $P < 0.05$, two tailed; c – with respect to atropine, $P < 0.05$, two tailed; d – with respect to progesterone + atropine, $P < 0.05$, one tailed.

Effect of sodium nitrate on feather release under treatment with progesterone alone and in combination with apomorphine or atropine. Progesterone alone as well as concomitantly with sodium nitrate treatment failed to induce any feather shedding (Table 5). But, as seen in Table 5, progesterone significantly aug-

mented the apomorphine-induced feather release. Under nitrate treatment the effect of apomorphine was enhanced by progesterone as well, but to a less extent than in progesterone + apomorphine group. In progesterone + apomorphine group, feather shedding was 3.5 times greater than in apomorphine group, whereas in nitrate + progesterone + apomorphine group it was only 2.2 times greater than in nitrate + apomorphine group.

As is seen in Table 5, feather release induced by atropine was facilitated by progesterone. But suppression of atropine effect by sodium nitrate was not abolished, but only diminished by progesterone. It seems that the extent of the effect of progesterone on atropine-induced feather release under sodium nitrate treatment was dependent on changes in atropine effect induced by sodium nitrate.

Thus, the influence of progesterone on apomorphine-induced feather release was attenuated under sodium nitrate treatment. Suppression of atropine effect under sodium nitrate treatment was not abolished, but only diminished by progesterone.

DISCUSSION

The results have shown that sodium nitrate which by itself has no effect on feather shedding had influenced the intensity of this reaction induced by the dopamine receptor agonist apomorphine and the cholinergic muscarinic receptor antagonist atropine. The influence of nitrate was also revealed in progesterone-evoked changes in atropine and apomorphine effect. However, the effect of these feather relaxation agents was changed by sodium nitrate in different way: the effect of apomorphine was enhanced, of atropine diminished and of progesterone attenuated. These variations allowed to suggest that the study compounds affect feather release by different mechanisms.

In the present as well in our previous investigation (20) it has been demonstrated that noradrenergic and cholinergic systems suppress apomorphine-evoked feather shedding. Therefore it could be suspected that the effect of nitrate may be mediated by attenuation of cholinergic and noradrenergic activity. In the investigation of nitrate influence on apomorphine-induced feather release under activation of cholinergic system by physostigmine or neostigmine, there was some cotradiction in results, so it was impossible to judge about the attenuating effect of nitrate on the cholinergic system. However, sodium nitrate suppressed atropine-induced feather release, implying that nitrate was not able to reduce cholinergic muscarinic activity in this reaction. On the other hand, the findings indicate that sodium nitrate has not augmented the activity of cholinergic muscarinic system in feather shedding reaction. The findings also demonstrated that apomorphine feather release suppression evoked by the enhanced noradrenergic activity was neither activated nor attenuated under sodium nitrate treatment. Apomorphine feather release augmentation under simultaneous treatment with nitrate and clonidine, which attenuate noradrenergic activity, could be accounted for by a simple addition of the individual effect of nitrate and clonidine. Therefore it is more likely that the effect of nitrate on apomorphine-induced feather release and on injection stress was not mediated by attenuation of noradrenergic and cholinergic activity.

The effect of apomorphine on feather release was significantly enhanced by nitrate + diazepam treatment. Therefore, it can be supposed that the GABA–benzodiazepine system could take part in the nitrate-elicited attenuation of stress influence on apomorphine feather shedding.

By treating the quails with sodium nitrate not only the level of NO^{-}_{3}, but also of Na^{+} and nitrogen metabolites could be elevated in the bird's organism. Therefore alterations in feather release regulation under sodium nitrate treatment could be dependent on the effect of all these substances. Since sodium nitrate and neuroactive agents were administered systemically in the present study, it is not possible to conclude which neuroanatomical site or feather myocytes by themselves mediated the observed effects.

Sodium nitrate as well other nitrites has been used as a donor of nitric oxide (NO) [2]. In addition, nitrites are able to increase the level of NO by their action on methemoglobin formation, and as a consequence the oxyhemoglobin level may be decreased. It is known that the latter is a potent scavenger of NO [5]. We have no data about the mechanisms underlying the effect of NO or of the other substances investigated in our study on feather smooth muscle relaxation, but there are investigations of vascular, airway and uterine smooth muscles.

It is known that NO induces hyperpolarization and hence the relaxation of vascular smooth muscle cells; NO is capable of activating Ca^{2+}-dependent K^{+} channels directly or indirectly by increasing the intracellular concentration of cyclic guanosine monophosphat (cGMP) [8]. It is believed that cGMP-dependent phosphorylation of the myosin light chain is responsible for mediating the vasodilating effect of NO [12]. It has been shown that in smooth muscles of the airway two redox forms of NO induce relaxation in different ways. NO(+) causes a relea-

se of internal Ca^{2+} in an cGMP-independent fashion, leading to activation of Ca^{2+}-dependent K^+ channels and relaxation, whereas NO relaxes the airway through a cGMP-dependent, Ca^{2+}-independent way [11]. Thus, it could be expected that the threshold of smooth muscle relaxation could be lowered by nitrate group of sodium nitrate, and as a consequence the effect of apomorphine and atropine could be increased. However, such influence evoked by nitrate was observed only in apomorphine-induced feather relaxation. There is evidence that dopamine-induced vascular relaxation opened the big Ca^{2+} activated K^+ channels (BKCa) [7]. Therefore some common effects of NO and dopamine on smooth muscle hyperpolarization by a simultaneous action of these substances are able to induce an increase in feather release response.

An increase in the cytoplasmic Ca^{2+} concentration is generally considered the major event of the activation processes of the contractile apparatus in the smooth muscle; cholinegic agonists evoke an increase in cytoplasmic Ca^{2+} concentration from extracellular space and from intracellular Ca^{2+} stores [16]; atropine, a cholinegic muscarinic receptor antagonist, decreases the intracellular Ca^{2+} concentration. Progesterone causes coronary smooth muscle relaxation and decreases uterine contractility in gestation, as well as decreases the tonus of internal smooth muscles mainly by inhibiting Ca^{2+} entry from extracellular space, but not Ca^{2+} release from intracellular stores [4, 15]. Concomitant decrease in intracellular Ca^{2+} and increase in NO concentration could be able to evoke a greater relaxation of smooth muscle. However, our findings have shown that under sodium nitrate treatment the effect of progesterone on apomorphine-induced feather release tended to be diminished, and in atropine-induced feather release the influence of progesterone was not entirely displayed, although nitrates have markedly facilitated apomorphine feather release under diazepam treatment. There is an evidence that benzodiazepine reduces the frequency of Ca^{2+} oscillation in smooth muscle cells along with inducing its relaxation [16]. The role of chloride currents in sodium nitrate effect could not be neglected, either, as it is known that Ca^{2+}-activated Cl^- current is participating in smooth muscle membrane depolarization [1]. Therefore, it can be presumed that several mechanisms in sodium nitrate effect on feather release are involved.

An increase in the concentration of Na^+ in bird's organism can be considered as well. The changes in external and hence internal Na^+ concentration might account for the exchanges in K^+ conductance and intracellular Mg^{2+} concentration. Ca^{2+}-activated K^+ channels could be inhibited by intracellular Na^+ in smooth muscle cells [13] and so myocyte hyperpolarization could be influenced. Moreover, the intracellular Mg^{2+} concentration is reduced, while the extracellular Na^+ concentration is simultaneously augmented [10]. Since Mg^{2+} is able to compete with Ca^{2+} at protein binding sites, intracellular Mg^{2+} might have a modulating effect on intracellular Ca^{2+}-regulating processes [6]. In addition, nitrogen metabolism derivatives in birds also could increase the intracellular Ca^{2+} level [14].

Nitrate and nitrite ingestion has been linked to adverse effects on reproductive efficiency. However, experimental studies show heterogeneous results. Animal experimental data have shown the reproductive toxicity to be associated with high exposure levels to nitrates or nitrites that are not likely to be encountered in drinking water [3]. In the organism, nitrites and nitrates are found as metabolites of NO. NO inhibits uterine contractility during pregnancy [21]. In pregnant sheep, the arterial plasma nitrate and urinary nitrate concentrations were significantly increased. Fetal plasma nitrate levels were ninefold higher than maternal, whereas amniotic fluid concentrations were extremely high [22]. These data led to a suggestion that nitrates and nitrites have not any unfavorable effect on uterine smooth muscle relaxation evoked by NO and progesterone. However, our findings have shown that the effect of progesterone on feather relaxation induced by apomorphine tended to be diminished by sodium nitrate. Also, in atropine-induced feather release the influence of progesterone was not fully displayed.

Taken all these aspects together, it is possible to hypothesize that sodium nitrate is able to influence the feather smooth muscle mechanism in several cells processes having the opposite effect on smooth muscle relaxation. Considering the different effects of sodium nitrate, the reaction of feather shedding induced by apomorphine could not be always expressed distinctly.

CONCLUSIONS

Treatment of quails with small amounts of sodium nitrate in food has elicited opposite changes in feather shedding evoked by apomorphine and atropine administration. Feather release evoked by apomorphine had a tendency to be increased under sodium nitrate treatment. Moreover, the effect of sodium nitrate on apomorphine was elevated by attenuation of noradrenergic activity by clonidine or by the serotonin synthesis inhibitor PCPA, as well

by the benzodiazepine receptor agonist diazepam in particular. In addition, nitrate has diminished the negative effect of stress on the apomorphine-induced feather release. The results have not shown that the effect of nitrate on apomorphine feather release and on injection stress is mediated by attenuation of noradrenergic activity.

Feather release induced by the cholinergic muscarinic receptor antagonist atropine was markedly reduced by sodium nitrate. This effect of sodium nitrate was not exorted by means of enhancing the cholinergic activity. The activating effect of progesterone on apomorphine-induced feather release tended to be diminished under sodium nitrate treatment. Suppression of atropine effect by sodium nitrate was not abolished but only diminished by progesterone.

Thus, sodium nitrate was able to affect the regulation of feather shedding, one of the extreme forms of escaping behaviour in birds, although sodium nitrate showed the opposite action on separate regulating links of this reaction.

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A. Šveistytė

NATRIO NITRATO ĮTAKA JAPONIŠKOSIOS PUTPELĖS (*COTURNIX COTURNIX JAPONICA*) IŠVENGIMO APSAUGINĖS ELGSENOS – GREITO PLUNKSNŲ ATPALAIDAVIMO REGULIAVIMUI

S a n t r a u k a

Buvo tirta natrio nitrato, esančio maiste (0,87 g/kg pašarui 14–30 dienų laikotarpiu), įtaka putpelių patinėlių plunksnų išmetimui, sukeltam dopamino agonisto apomorfino (0,5–0,75 mg/kg) ir cholinerginių muskarininių receptorių antagonisto atropino (6,7–20,0 mg/kg), injekuotų pavieniui ar kartu su progesteronu (5 mg/paukščiui).

Apomorfino sukeltas plunksnų išmetimas buvo linkęs didėti veikiant nitratams. Apomorfino poveikis labiau išryškėjo, kai tuo pačiu metu buvo injekuoti klonidinas (0,005 mg/kg) ar PCPA (120,0 mg/kg), ypač diazepamas (6,7 mg/kg). Be to, nitratai sumažino neigiamą streso įtaką apomorfino sukeltam plunksnų išmetimui, nors apomorfino effekto sumažėjimas, sukeliamas yohimbinu, nebuvo nitratų paveiktas.

Natrio nitratas nestiprino slopinančio fizostigmino (00,075–0,1 mg/kg) ir neostigmino (0,075 mg/kg) poveikio apomorfino sukeltam plunksnų atpalaidavimui. Tačiau jis sumažino atropino sukeltą plunksnų išmetimą. Veikiant natrio nitratui, progesterono aktyvinantis poveikis apomorfino plunksnų atpalaidavimui buvo linkęs mažėti. Progesteronas nepanaikino natrio nitrato įtakos atropinui, tik ją sumažino.

Taigi rezultatai parodė, kad ekstremalios išvengimo – plunksnų išmetimo elgsenos nervinė reguliacija gali būti paveikta mažais natrio nitrato kiekiais pašare. Taip pat buvo išryškinti du priešingi natrio nitrato poveikiai atskiroms greito plunksnų išmetimo reguliacinio mechanizmo grandims.