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Adrenocortical reactivity and central serotonin and dopamine turnover in young chicks from a high and low feather-pecking line of laying hens

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Abstract

Feather pecking in domestic fowl is a behavioral abnormality that consists of mild or injurious pecking at feathers of conspecifics. Previously, it was shown that chicks from a high feather-pecking (HFP) and low feather-pecking (LFP) line of laying hens already differ in their propensity to feather peck at 14 and 28 days of age. As a first step in investigating a possible relationship between the development of feather pecking and physiological and neurobiological characteristics of laying hens, two subsequent experiments were carried out. Firstly, we investigated the development of adrenocortical (re)activity in HFP and LFP chicks during the first 8 weeks of life. Secondly, we studied dopamine (DA) and serotonin (5-HT) turnover in the brain of 28-day-old HFP and LFP chicks. In both experiments, chicks were exposed to manual restraint (placing the chicks on its side for 5 min). Plasma corticosterone levels were lower (baseline on Days 3 and 56; restraint-induced on Days 3, 14 and 28) in HFP chicks. Both brain DA and 5-HT turnover were lower in the HFP chicks, as well. Possible consequences for the observed differences in (stress) physiology and neurobiology between the two lines in relation to the feather pecking are discussed. © 2002 Elsevier Science Inc. All rights reserved.

Keywords: Feather pecking; Chicks; Development; Stress response; Corticosterone; Dopamine; Serotonin

1. Introduction

Feather pecking behavior consists of mild pecking (gentle feather pecking) or vigorous pulling at the feathers of conspecifics (severe feather pecking). The latter can especially cause damage to the plumage and loss of feathers, which increases susceptibility to further injury, like wounds of the skin. At worst, injured birds may be pecked to death (i.e., cannibalism). Thus, feather pecking behavior negatively affects poultry welfare and is a serious problem in poultry practice that needs to be solved [5].

Until now, no single causal factor has been identified that induces feather pecking. There is general acceptance that the development of feather pecking reflects multifactorial processes [17]. Some investigators have stressed the relevance of environmental factors (e.g., housing conditions) [4], while others have implicated animal-related factors (e.g., genetics, hormones) [22] and animal–environment interactions (e.g.,

ontogenetic factors) [18]. Lately, studies of feather pecking behavior are broadening to include animal characteristics [21,26,28].

Previously, it has been shown that two strains of laying hens that differ in their propensity to feather peck [6,7] also show differences in open-field reactions [19], social motivation [19] and behavioral and physiological stress responsivity [26,28]. More specifically, it was shown that in response to acute stress induced by manual restraint, adult birds of the high feather-pecking line (HFP) displayed more struggling behavior, lower heart rate variability, higher plasma noradrenaline and lower plasma corticosterone levels than birds of the low feather-pecking line (LFP).

The behavioral and physiological characteristics of birds of the HFP and LFP line show considerable analogy to the characteristics of respectively the proactive (active) and reactive (passive) coping strategy, known to exist in other species like rodents [2] and pigs [9,39]. In mice, it has been shown that proactive copers are more intrinsically driven. This means that their behavior is less guided by environmental stimuli but more by internal mechanisms. They

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easily develop routines, a rather rigid form of behavior. In contrast, reactive copers are more flexible and react more to environmental stimuli [2,24]. There is a growing body of evidence that adopting a proactive coping strategy makes an individual more vulnerable to develop behavioral abnormalities than a reactive individual (see, for a review, Ref. [24]). A differential HPA axis (re)activity between the two coping strategies (reflected in plasma corticosteroid levels) is suggested to underlie this difference [24]. Due to their lipophilic nature, corticosteroids may readily enter the brain to bind to specific cytoplasmatic receptors [25]. Consequently, corticosteroids may alter neural transmission in the serotonergic (5-hydroxytryptamine, 5-HT) [35] and dopaminergic (DA) system [32]. Indirectly, it has been shown that 5-HT as well as DA neurotransmission is altered in adult proactive individuals compared to adult reactive individuals [27,38].

DA and 5-HT are known to be involved in the expression of (environmentally induced) behavioral disorders (e.g., stereotypies, obsessive compulsive disorder) in adult individuals of several species [2,9,30]. Stereotypies are generally defined as unvarying, repetitive behavior patterns that have no obvious goal or function [33]. It has been suggested that gentle feather pecking (usually performed in long bouts) has stereotypic characteristics [23], as its motor patterns closely resemble drug-induced stereotypic pecking in chickens [3]. Severe feather pecking may have a less clear stereotypic nature. The number of severe pecks per bout is rather low compared to gentle feather pecking, as its performance often evokes a flight reaction of the peckee [23]. It can, however, be described as abnormal behavior with repetitive c.q. routine-like characteristics.

The available data suggest a possible causal role of DA and 5-HT neurotransmission in the development of feather pecking, possibly modulated by corticosteroids. Therefore, it could be hypothesized that the difference in the level of feather pecking behavior between birds of the HFP and LFP line reflects a difference in sensitivity of the DA and 5-HT system in the brain, possibly through interaction with corticosterone.

As mentioned earlier, adult HFP and LFP birds show a consistent difference in feather pecking behavior [6,7]. Recently, we [16] showed that already at an early age, HFP and LFP chicks show clear differences in feather pecking and related behaviors. On Days 14 and 28 (but not on Days 41 and 56) posthatching, HFP chicks showed significantly higher levels of feather pecking than LFP chicks [16]. However, there is no knowledge on physiological and neurobiological characteristics of HFP and LFP birds at a young age. The aim of the present study is to investigate whether the differences in behavioral development between the two lines go parallel with physiological and neurobiological differences.

Therefore, as a first step in investigating the question of a possible relationship between corticosteroids, 5-HT and DA turnover and the development of feather pecking, two subsequent experiments were carried out. Firstly, we investigated the development of adrenocortical (re)activity in HFP and

LFP chicks during the first 8 weeks of life. Secondly, we studied DA and 5-HT turnover in the brain of HFP and LFP chicks on 28 days of age. In the present experiments, feather pecking behavior was not studied. In our indirect approach, we used the manual restraint test, an acute stressor, as a model for coping with environmental challenges.

2. Methods

2.1. Experiment 1. Adrenocortical (re)activity

2.1.1. Birds and housing

In this study, 480 White Leghorn chicks from two strains were used: 240 HFP chicks and 240 LFP chicks [26,28]. All birds were female and nonbeaktrimmed. Chicks arrived on the day of hatching and were housed in litter-floor pens (0.75 × 1.0 m) with four animals per line (60 pens per line). The pens were placed in six identical climate-controlled rooms and the lines were randomly assigned to the pens within the rooms. Visual contact between chicks in adjacent pens was prevented by hardboard separations between the pens. The environmental temperature was lowered from 34 °C on Day 1 to 18 °C at 8 weeks of age. On Days 1 and 2 of age, the light regime was alternately 4 h light and 4 h dark. From 3 days to 8 weeks of age, the light regime decreased from an 18-h light to a 10-h light period. A standard vaccination program was applied during rearing.

All groups had access to three drinking cups and one square feeding trough placed along one of the walls of the pen. Water and a standard rearing feed (mash) were provided *ad libitum*.

2.1.2. Manual restraint and blood sampling

On Days 3, 14, 28, 41 and 56 of age, chicks were killed by rapid decapitation. Trunk blood was collected and blood samples were analysed for plasma corticosterone. Half of the birds (six pens per line per age) were decapitated immediately (within 2 min) after removal from the pen; the other half was manually restrained (i.e., placed on its side) for 5 min before decapitation. Chicks from the same pen were removed, tested and decapitated simultaneously. Treatments (line/age/restraint) were randomly assigned to the pens within the rooms. Decapitation was always carried out between 9.00 and 12.00 h.

2.1.3. Corticosterone measurement

The blood samples were immediately transferred to chilled (0 °C) Lithium–Heparin-coated centrifuge tubes. Blood was centrifuged for 10 min at 3000 rpm at a temperature of 4 °C. Plasma samples for corticosterone analysis were stored at 4 °C in the presence of 0.1% (w/v) sodium azide. Corticosterone concentrations were determined in unextracted, enzymatically pretreated plasma (DELFI), as described earlier [20]. The detection range of the corticosterone assay was 0.2–44 ng/ml.

2.2. Experiment 2. DA and 5-HT turnover in the brain

2.2.1. Birds and housing

In this study, 15 LFP and 15 HFP chicks were used. All birds were female and nonbeaktrimmed. Chicks arrived on the day of hatching and were housed in litter-floor pens (0.75×1.0 m) of four animals per line. The pens were placed in a climate-controlled room. Chicks were reared under the same environmental and management conditions as in Experiment 1.

2.2.2. Measurement of corticosterone levels and DA and 5-HT turnover

On 28 days of age, the chicks were manually restrained for 5 min and killed by rapid decapitation. Blood samples were collected and analysed for corticosterone (see Section 2.1.3).

The brains were immediately frozen in a dry ice pre-cooled tube containing *n*-heptane and stored at -70°C until the assays were performed. For the assay, a brain was transversally cut rostrally to the midbrain 5-HT neurons [31] (see Fig. 1). Thereafter, the rostral brain sections were used for the measurement of 5-HT and DA and the 5-HT metabolite 5-hydroxyindoleacetic acid (5-HIAA) and the DA metabolites 3,4-dihydroxyphenylacetic acid (DOPAC) and homovanillic acid (HVA). Previously, it has been shown that 5-HT turnover is indicated by the 5-HIAA/5-HT ratio [29] and DA turnover by the (DOPAC+HVA)/DA ratio [43]. In order to measure these neurotransmitters and their metabolites, the brain samples were homogenized in ice-water in a 1000- μl solution containing 5 μM clorgyline, 5 $\mu\text{g/ml}$ glutathione and 200 ng/ml *N*- ω -methylserotonin (internal standard) with a MSE Soniprep 150 ultrasonic tissue processor (Beun de Ronde, NL). Thereafter, 50 μl 2 M HClO_4 and 40 μl 2.5 M potassium acetate were added to 200 μl of the homogenate. After 15 min, the tissue samples were centrifuged for 15 min at $15\,000\times g$ (4°C). There-

after, 30 μl of the supernatant was diluted with 450 μl HPLC grade water.

The samples were injected onto a reverse-phase/ion-pair high performance liquid chromatography (HPLC) setup with electrochemical detection for the measurement of 5-HIAA, 5-HT, DA, DOPAC and HVA. The chromatographic system consisted of X-Act degassing unit (Jour Research, Sweden), a Perkin-Elmer series 410 HPLC pump (USA), a Perkin-Elmer ISS 101 autosampler (USA) with a 100- μl loop, the INTRO combined column oven, electrochemical detector (Antec Leyden, NL) and a column (150×4.6 mm i.d.) packed with Hypersil ODS, 5 μm particle size (Alltech Associates, USA).

The mobile phase consisted of 0.051 M citric acid monohydrate, 0.051 M $\text{Na}_2\text{HPO}_4\cdot 2\text{H}_2\text{O}$, 0.26 mM EDTA, 0.356 mM sodium octyl sulphate, 0.265 mM di-*n*-butylamine, 2.0 mM NaCl and 13% methanol. This buffer was filtered through a 0.22- μm membrane filter (Schleicher & Schuell, Germany). Separation was done at 25°C using a flow rate of 1 ml/min.

Detection of the 5-HT and 5-HIAA was performed using an electrochemical detector (Antec, Leiden, the Netherlands) with a glassy carbon working electrode set at -0.611 V versus an in situ Ag/AgCl reference electrode. The data were recorded with a chart recorder (Model BD112, Kipp and Zn., the Netherlands), and peak heights of samples were compared with those of standards determined each day for quantification. The limit of detection (signal/noise ratio 3:1) was 9.5 fmol/100 μl .

2.3. Statistical analysis: Experiments 1 and 2

The data of Experiment 1 were analysed with an analysis of variance model with main effects and interactions for the factors line (HFP/LFP), restraint stress (yes/no) and age. Data were checked for normal distribution and homogeneity of variances. Preliminary analyses of the corticosterone data showed that the variance increased with the mean. Corticosterone levels were log-transformed prior to analysis (averages per pen were analysed). For corticosterone, the log-transformation (in order to obtain normal distribution), appropriate when the variance is proportional to the square of the mean, was not satisfactory. The assumption that the variance was proportional to the mean fitted the data better and the analysis was performed accordingly. Statistical inference was based on maximum quasi-likelihood. A multiplicative overdispersion parameter in the variance was estimated from Pearson's χ^2 statistic. Significance tests were based on the quasi-likelihood ratio statistic. Technical details may be found in McCullagh and Nelder [34].

In Experiment 2, the variances for the two lines differed significantly for some of the variables. Line means were compared with a *t* test for unequal variances employing Satterthwaites approximation [42]. Incidentally, no marked differences with results from the ordinary *t* test (based on an equal variances assumption) were found. All statistical cal-

Fig. 1. Image of the chicken brain. The diagonal line represents the position at which the brain was cut. The right (rostral) brain section was used for 5-HT and DA turnover measurements (Cb: cerebellum, CO: chiasma opticum).

Fig. 2. Baseline corticosterone levels (ng/ml) and corticosterone levels (ng/ml) after manual restraint (5 min) in LFP and HFP chicks on Days 3, 14, 28, 41 and 56. Levels are expressed as means±S.E.M. *** $P<.001$, ** $P<.01$, * $P<.05$, # $.05<P<.08$.

culations were performed with Genstat 5 [12,13]. P values below .05 were considered significant.

3. Results

3.1. Development of adrenocortical (re)activity

Fig. 2 shows the dynamics of baseline and restraint-induced corticosterone levels for the LFP and HFP line during the first 8 weeks of life. No significant interactions between Restraint, Line and Age were found. There were significant effects of Restraint [$F(1,106)=678.6$, $P<.001$], Line [$F(1,106)=32.2$, $P<.001$] and Age [$F(4,106)=92.2$, $P<.001$] on corticosterone levels.

On 3 and 56 days of age, HFP chicks showed significantly lower baseline corticosterone levels than LFP chicks. In the LFP line, baseline levels of corticosterone decreased significantly from 14 to 28 days of age, maintaining the

same level on subsequent days. In the HFP chicks, baseline corticosterone levels also decreased during aging (although less evident): Day 14 was significantly higher than Days 41 and 56.

After manual restraint, corticosterone levels were lower in the HFP line compared to the LFP line on Days 3, 14 and 28 of age. Similar to baseline levels, stress-induced corticosterone levels also decreased during aging. Corticosterone levels of LFP chicks declined significantly from Days 14 to 41 of age and remained constant. HFP chicks showed a similar pattern, with corticosterone levels significantly declining from 14 to 41 days of age.

3.2. DA and 5-HT turnover

Table 1 shows plasma corticosterone levels and (turn-over) levels of the neurotransmitters DA and 5-HT in the brain of 28 days old LFP and HFP chicks, that had been exposed to restraint stress. Corticosterone levels were significantly lower in HFP chicks (approx. $t=4.72$, $df=12.64$) than in LFP chicks. The 5-HIAA/5-HT ratio and the (DOPAC+HVA)/DA ratio are considered markers of the 5-HT and DA turnover, respectively. Both 5-HT and DA turnover (Table 1) were significantly lower in the HFP line compared to the LFP line (approx. $t=3.42$, $df=1.69$ resp. approx. $t=3.38$, $df=17.82$).

Table 1

Levels of corticosterone (ng/ml) and the neurotransmitters DA and 5-HT and their metabolites (ng/mg brain tissue) after 5 min of manual restraint

	LFP ($n=15$)	HFP ($n=15$)
Corticosterone	$13.192 \pm 1.651^{***}$	5.579 ± 0.271
5-HIAA/5-HT	$0.105 \pm 0.006^{**}$	0.081 ± 0.004
(DOPAC+HVA)/DA	$0.405 \pm 0.029^{***}$	0.300 ± 0.013
5-HT	$1.705 \pm 0.051^*$	1.911 ± 0.059
5-HIAA	$0.176 \pm 0.008^{\#}$	0.154 ± 0.009
DA	0.453 ± 0.036	0.492 ± 0.027
DOPAC	$0.087 \pm 0.005^*$	0.072 ± 0.003
HVA	$0.134 \pm 0.007^{**}$	0.109 ± 0.004

Levels expressed as means±S.E.M. *** $P<.001$, ** $P<.01$, * $P<.05$, # $.05<P<.08$.

4. Discussion

4.1. Development of adrenocortical (re)activity

The main finding of this experiment is that young chicks of the HFP line are characterized by lower adrenocortical

(re)activity than LFP chicks. HFP chicks showed lower baseline as well as restraint-induced levels of corticosterone compared to LFP chicks on Days 3 and 56, respectively, on Days 3, 14 and 28 of age. These findings are in agreement with previous findings of Korte et al. [26] in adult hens of these lines and strengthens the idea that the HFP and LFP line are representatives of respectively the proactive and reactive coping style.

Corticosteroids are of crucial importance for the regulation of adaptive behavior, learning, memory and neural plasticity [25,40]. In several species, including birds, it has been shown that circulating corticosteroids enter the brain, where they bind to intracellular mineralocorticoid (MR) and glucocorticoid (GR) receptors, e.g., in the hippocampus and amygdala (mammals) c.q. archistriatal complex (birds) [25,31]. A disturbed balance in MR/GR function is believed to alter responsiveness to the environment, promote susceptibility to stress, alter behavioral adaptation [25], and influence learning and memory processes [40].

Chicks are precocial to ensure their survival, therefore, they need to learn rapidly about the properties of their environment and retain this memory [40]. Recently [16], it was shown that LFP and HFP chicks differ in the way they 'experience' environmental stimuli and interact with it. This was reflected in the different ways pecking behavior was targeted in both lines. LFP chicks showed more interest in exploring and pecking at nonanimate environmental stimuli, i.e., are more engaged in pecking feed and litter. In contrast, HFP chicks showed more interest in pecking at animate stimuli, i.e., showed higher levels of feather pecking and preening (which also includes pecking at feathers). It was hypothesized that differences in learning processes may have lead to the involvement of different underlying motivational systems (respectively, preening and feeding behavior) in the development of feather pecking in both lines.

In accordance with that hypothesis, we suggest here that the differences in the development and performance of feather pecking between LFP and HFP chicks are associated with (1) differences in behavioral and physiological (coping) response to environmental stimuli and (2) differences in learning processes, during early development. Furthermore, we hypothesize that (3) a different MR/GR balance in the brain of LFP and HFP chicks may be underlying these differences.

In future experiments, it is necessary to further investigate whether physiological and behavioral differences between LFP and HFP chicks arise from differences in occupancy of MR and/or GR receptors (MR/GR balance).

4.2. DA and 5-HT turnover, coping and feather pecking

Previously, it has been suggested that proactive individuals, behaviorally characterized by low behavioral inhibition, high routine formation, low cue dependency and low flexibility, are more vulnerable for the development of behavioral abnormalities than their reactive counterparts

[24]. There is accumulating evidence that this difference in vulnerability may be a consequence of the differences in DA and 5-HT neurotransmission (e.g., turnover levels, receptor expression levels and receptor sensitivity) between proactive and reactive copers [9,27].

For instance, in rodents and pigs [1,9], the DA receptor agonist apomorphine produced a greater enhancement of stereotyped behavior in proactive coping individuals than in reactive coping individuals. Furthermore, it was shown that proactive mice have lower 5-HT neurotransmission [27] and (possibly), consequently, a more sensitive (postsynaptic) 5-HT receptor system as compared to reactive mice [44]. A difference in sensitivity of (postsynaptic) 5-HT receptors are suggested to play a role in the differences in behavioral repertoire between proactive and reactive individuals [27,44].

The lower DA and 5-HT turnover in chicks of the HFP line as compared to the LFP line found in the present study are in agreement with above findings in adult pigs and rodents and support the assumption that the HFP and LFP lines are representatives of the respectively proactive and reactive coping strategy.

Both DA and 5-HT have been shown to play a role in the expression of oral stereotypies in fowl [30]. Several DA receptor agonists, e.g., CQP201-403 [11], apomorphine [15] and amphetamine [14], induce stereotyped pecking responses in birds, suggesting a possible involvement of the DA system in the development of stereotypic gentle feather pecking. Bilcık [3] investigated a possible involvement of DA neurotransmission in the expression of feather pecking. His findings were inconclusive as to whether DA plays a role in feather pecking. He did not find a difference in DA sensitivity in young chicks, that were later (at an adult age) identified as feather peckers and nonfeather peckers. However, they did find some minor differences in binding and densities of D1 and D2 dopamine receptor subtypes in specific brain regions, between feather peckers and nonpeckers.

Interestingly, increasing brain 5-HT levels by dietary supplementation with L-tryptophan (precursor of 5-HT) suppressed feather pecking damage in growing bantams [41]. In line with these results, it did not come as a surprise that LFP chicks were characterized by a higher 5-HT turnover. Self-mutilating feather pecking disorder (FPD) in birds is a stereotypy that seems under the control of 5-HT mechanisms. Clomipramine, a tricyclic antidepressive drug inhibiting the reuptake of 5-HT and noradrenaline, was effective in alleviating severe FPD in psittacine birds (parrots and parakeets) [10]. In a study of Blokhuis et al. [8], adult HFP and LFP hens, when housed on battery cages, showed marked differences in the type of stereotypy performed. Almost 60% of the observed HFP birds showed pecking at own feathers, whereas only 6% of the observed LFP birds showed this kind of stereotypy. It is tempting to hypothesize that the higher levels of self-mutilating pecking, found in the experiment of Blokhuis et al. [8] and the higher levels of feather pecking in HFP chicks found in our recent study [16] may be

associated with lower 5-HT turnover in the birds of the HFP line compared to the LFP line.

In view of the above findings, we hypothesize that a lower DA and 5-HT turnover in HFP chicks compared to LFP chicks predispose them to more easily develop a stereotypy like (gentle) feather pecking. Further research is necessary to investigate this possible relationship between DA and 5-HT neurotransmission and the development of feather pecking in the HFP and LFP line.

4.3. Interaction of corticosteroids with DA and 5-HT pathways and feather pecking

Another possible way in which corticosterone may play a role in the development of feather pecking is through interaction with DA and 5-HT pathways.

It is known that corticosteroids stimulate DA release in the brain and that a corticosterone-induced increase in extracellular DA levels results in psychomotor activation [36]. Furthermore, it was shown that corticosteroids via GRs may play an important role in the sensitization of the DA system [37]. Interestingly, the development of divergence in DA responsiveness in apomorphine susceptible and unsuspensible rat lines, that also differ in coping strategy, is preceded by changes in pituitary–adrenal activity [38].

Corticosteroids also stimulate 5-HT synthesis at the level of the raphe nuclei, probably via glucocorticoid receptors, and this results in increased extracellular 5-HT levels in limbic forebrain areas [29]. Consequently, low corticosteroid levels in proactive individuals via these mechanisms may play an important role in the increased vulnerability of these individuals for the developing stereotypies [25].

In conclusion, young chicks of the HFP line are characterized by lower plasma corticosterone levels, and both lower 5-HT and DA turnover as compared to LFP chicks. To our knowledge this is the first time it has been shown that chicks, that are known to differ in feather pecking, also differ in both stress physiology and neurobiology.

Further research is needed to investigate whether a difference in binding of corticosterone to corticosteroid receptors in the brain of HFP and LFP birds is responsible for the differences in the development and performance of feather pecking in both lines. Or, whether a difference in sensitivity of the DA system and 5-HT system, possibly under influence of corticosterone, may be the underlying mechanism in the development of feather pecking.

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