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

Published in:
Physiology & Behavior

DOI:
10.1016/S0031-9384(02)00667-4

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2002

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):

Copyright
Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment.

Take-down policy
If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): http://www.rug.nl/research/portal. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.
Adrenocortical reactivity and central serotonin and dopamine turnover in young chicks from a high and low feather-pecking line of laying hens

Yvonne M. van Hierden a,b,*, S. Mechiel Korte a, E. Wim Ruesink a, Cornelis G. van Reenen a, Bas Engel a, Gerdien A.H. Korte-Bouws a, Jaap M. Koolhaas b, Harry J. Blokhuis a

aDivision of Animal Sciences, Institute for Animal Science and Health (ID-Lelystad), PO Box 65, NL-8200 AB Lelystad, The Netherlands
bDepartment of Animal Physiology, University of Groningen, PO Box 14, 9750 AA Haren, The Netherlands

Received 9 August 2001; received in revised form 26 November 2001; accepted 10 January 2002

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 responsiveness [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 norepinephrine 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
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 corticosterone levels) is suggested to underlie this difference [24]. Due to their lipophlic nature, corticosteroids may readily enter the brain to bind to specific cytoplasmic 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 behavioral 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 pecked [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 (DELFIA), 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 nonbeak-trimmed. 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 precooled 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 μg/ml glutathione and 200 ng/ml N-ß-methylserotonin (internal standard) with a MSE Soniprep 150 ultrasonic tissue processor (Beun de Ronde, NL). Thereafter, 50 μl 2 M HClO₄ 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×g (4 °C). Thereafter, 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 PerkinElmer 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 Na₂HPO₄–2H₂O, 0.26 mM EDTA, 0.356 mM sodium octyl sulphonate, 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 χ² 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).
Calculations 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 (turnover) levels of the neurotransmitters DA and 5-HT and their metabolites (ng/mg brain tissue) after 5 min of manual restraint.

<table>
<thead>
<tr>
<th></th>
<th>LFP (n=15)</th>
<th>HFP (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corticosterone</td>
<td>13.192 ± 1.651***</td>
<td>5.579 ± 0.271</td>
</tr>
<tr>
<td>5-HIAA/5-HT</td>
<td>0.105 ± 0.006**</td>
<td>0.081 ± 0.004</td>
</tr>
<tr>
<td>(DOPAC+HVA)/DA</td>
<td>0.405 ± 0.029***</td>
<td>0.300 ± 0.013</td>
</tr>
<tr>
<td>5-HT</td>
<td>1.705 ± 0.051*</td>
<td>1.911 ± 0.059</td>
</tr>
<tr>
<td>5-HIAA</td>
<td>0.176 ± 0.006*</td>
<td>0.154 ± 0.009</td>
</tr>
<tr>
<td>DA</td>
<td>0.453 ± 0.036</td>
<td>0.492 ± 0.027</td>
</tr>
<tr>
<td>DOPAC</td>
<td>0.087 ± 0.005*</td>
<td>0.072 ± 0.003</td>
</tr>
<tr>
<td>HVA</td>
<td>0.134 ± 0.007**</td>
<td>0.109 ± 0.004</td>
</tr>
</tbody>
</table>

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

Table 2 shows plasma corticosterone levels and (turnover) 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. \( t=3.38, df=17.82 \)).

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
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. Bilčik [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 unsusceptible 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.

Acknowledgments

This study was supported by the Dutch Organisation for Scientific Research (NWO) (grant ALW, PPWZ 805-46.051). Furthermore, this research was partly supported by the European Commission, Directorate General for Agriculture, within the framework of the specific RTD program in the field of Agriculture and Fisheries (Contract FAIR-CT97-3576). We would like to thank Willem Buist for his assistance on the statistical analyses.

References

[20] Jong de IC, van Voorst A, Erkens JHF, Elsharid DA, Blokhuis HJ. Determinations of the circadian rhythm in plasma corticosterone and...
catecholamine concentrations in growing broiler breeders using intra-
[21] Keeling L, Jensen P. Do feather pecking and cannibalistic hens have
behaviour in laying hens (Gallus gallus domesticus). Appl Anim Be-
[23] Kjaer JB, Vestegaard KS. Development of feather pecking in relation
CG, Hopster H, de Jong IC, Ruis MAW, Blokhuis HJ. Coping styles in
animals: current status in behavior and stress-physiology. Neurosci
[25] Korte SM. Corticosteroids in relation to fear, anxiety and psychopa-
[26] Korte SM, Beuving G, Ruesink W, Blokhuis HJ. Plasma catechol-
amine and corticosterone levels during manual restraint in chicks from
a high and low feather pecking line of laying hens. Phys Behav
[27] Korte SM, Meijer OC, de Kloet ER, Buwalda B, Keijser J, Sluyter F,
van Oortmerssen G, Bohus B. Enhanced 5-HT1A receptor expression
in forebrain regions of aggressive house mice. Brain Res 1996;736:
338–43.
[28] Korte SM, Ruesink W, Blokhuis HJ. Heart rate variability during
manual restraint in chicks from high- and low-feather pecking lines
[29] Korte-Bouws GAH, Korte SM, de Kloet ER, Bohus B. Blockade of
corticosterone synthesis reduces serotonin turnover in the dorsal hip-
pocampus of the rat as measured by microdialysis. J Neuroendocrinol
[30] Kostal L, Savory CJ. Serotonergic mechanisms in control of stereo-
[32] Lowry CA, Burke KA, Renner KJ, Moore FL, Orchinik M. Rapid
changes in monoamine levels following administration of corticotro-
pin-releasing factor or corticosterone are localized in the dorsomedial
[33] Mason GJ. Stereotypies: a critical review. Anim Behav 1991;41:
1015–37.
[34] McCullagh P, Nelder JA. Generalized linear models. London: Chap-
man & Hall, 1989.
[35] Meijer OC, de Kloet RE. Corticosterone and serotoninergic neurotrans-
mition in the hippocampus: functional implications of central corti-
[36] Piazza PV, Le Moal M. Glucocorticoids as a biological substrate of
reward: physiological and pathophysiological implications. Brain Res
[37] Rivoit JM, Stinus L, LeMoal M, Mormede P. Behavioral sensitization
to amphetamine is dependent on corticosteroid receptor activation.
[38] Rots NY, Wolkel J, Oitzl MS, Bérod A, William R, Cools AR, de
Kloet ER. Development of divergence in dopamine responsiveness in
genetically selected rat lines is preceded by changes in pituitary–
[39] Ruis MAW, te Brake JHA, van de Burgwal JA, de Jong IC, Blokhuis
HJ, Koolhaas JM. Personalities in female domesticated pigs: behav-
ioral and physiological indications. Appl Anim Behav Sci 2000;
66:31–47.
[40] Sandi C, Rose SPR. Corticosteroid receptor antagonists are amnestic
for passive avoidance learning in day-old chicks. Eur J Neurosci
[41] Savory CJ, Mann JS, Macleod MG. Incidence of pecking damage in
growing bantams in relation to food form, group size, stocking den-
sity, dietary tryptophan concentration and dietary protein source. Br
[43] Thiffault S, Langston JW, Di Monte DA. Increased stratial dopamine
turnover following acute administration of rotenone to mice. Brain
[44] Vegt van der BJ, de Boer SF, Buwalda B, de Ruiter AJH, de Jong JG,
Koolhaas JM. Enhanced sensitivity of postsynaptic serotonin-1A re-
ceptors in rats and mice with high trait aggression. Phys Behav