Chapter 1

General Introduction
1. Introduction

Feather pecking is still one of the major problems in current housing systems for laying hens, as it poses major welfare problems for the hens, and constitutes an economical burden for the farmer. Hens peck and pull at the feathers of conspecifics, causing damage to the plumage and loss of feathers. This adversely affects the costs of egg production, since loss of feathers results in considerable higher energetic needs and, consequently, higher feed requirements of hens as a result of increased body heat loss (Leeson and Morrisson, 1978; Tullet et al., 1980). Furthermore, feather pecking, which is painful in itself (Gentle and Hunter, 1990), may result in severe damage to the integument of the birds, including wounds of the skin. Wounded birds may become the victim of cannibalism (Allen and Perry, 1975; Blokhuis et al., 2000; Hughes, 1982). This may result in a loss up to 15% of the birds per production cycle (in alternative housing systems) (Keeling et al., 1988). Apart from these serious economic losses, there is a moral aspect to the problem as well. Clearly, feather pecking is detrimental to the welfare of the birds.

Beak trimming, a common and effective precautionary measure practised by poultry farmers to prevent serious feather damage and mortality, might be associated with welfare problems. The procedure involves partial amputation of the beak and behavioural studies indicate that hens may suffer from chronic pain, presumably due to neuroma formation (Duncan et al., 1989; Gentle, 1986). Recent studies showed that neuroma development can be prevented, when debeaking is performed at an early age (1-10 days) (Gentle et al., 1997). Beak trimming however, is without doubt painful in itself and has therefore been prohibited in several European countries (e.g. Norway, Sweden, Switzerland). In the Netherlands beak trimming will be legally banned in 2011.

A widespread introduction of loose-housing systems, specifically designed with the aim to improve poultry welfare, is hampered due to the fact that large outbreaks of feather pecking and cannibalism are more likely to occur in these housing systems than in battery cages (Appleby and Hughes, 1991; Gunnarsson et al., 1999). Most probably this is because the presence of a few feather peckers in a
free housing system has a much greater impact, simply because larger numbers of potential victims are present (Allen and Perry, 1975). Thus, feather pecking represents a serious problem that needs to be solved.

Because feather pecking in poultry practice reveals itself at group level, for practical reasons alleged solutions are always applied on the whole flock. Most of the measures taken involve adjusting management conditions, based on scientific knowledge of environmental causal factors (see also section 3). However, despite over 25 years of research and many efforts in practice to alleviate the problem of feather pecking, an adequate solution to this problem has not been reached so far. This strongly suggests that this type of abnormal behaviour cannot be completely prevented by simply changing the environment of a group of birds. Research has shown that only certain individuals in a flock initiate the feather pecking problem, i.e. are the 'real' feather peckers, whereas most birds receive pecks (Bessei, 1984; Keeling, 1994; Wechsler et al., 1998). Furthermore, there is a growing body of understanding that feather pecking results from a complex interaction between the internal state of an animal and its environment (Blokhuis, 1989; Huber-Eicher and Audigé, 1999; Hughes and Duncan, 1972; Leonard et al., 1995; Nicol et al., 2001; Nørgaard-Nielsen et al., 1993). Feather pecking is generally considered an abnormal behaviour, or even a behavioural pathology (Sanotra et al., 1995). Following this latter interpretation, the aetiology of feather pecking may show analogy to psychopathological disorders described in other species, like e.g. (animal) stereotypies (Jenkins, 2001; Pitman, 1987; Pitman, 1989; Stein, 2000) and self-mutilation disorders (Bordnick et al., 1994; Hartgraves and Randall, 1986; Tiefenbacher et al., 2000; Weld et al., 1998). Despite a large body of scientific knowledge on the causation of psychopathologies, surprisingly few efforts have been made to approach feather pecking from the area of behavioural neuroscience and psychiatry (Bilčík and Keeling, 2000; Kjaer et al., 2002).

Therefore, in this thesis feather pecking will be approached from the assumption that it is a psychopathology, comparable to the ones mentioned above. Current knowledge of the causal role of neurobiological systems in the development of these behavioural disorders in both human and animal will be applied to feather pecking. This implies shifting the focus of attention from purely
studying the effects of environmental changes on a group of birds, towards studying causal relationships between feather pecking and predisposing behavioural or neuroendocrine factors within individual birds. Increased understanding of the interaction between a bird’s environment and neuroendocrine systems in relation to its propensity to perform feather pecking, may lead to new insights and possible solutions to the feather pecking problem in the future.

2. Feather pecking: normal versus abnormal behaviour

Feather pecking in domestic fowl involves pecking at the feathers of conspecifics. Its consequences for the recipient depend on the severity of pecking, i.e. the force and velocity of the pecking movements (see for an overview, page 19; Kjaer, 1999). The most gentle form of feather pecking is characterised by mild (Keeling, 1995), often stereotypic pecks (Kjaer and Vestergaard, 1999), causing no or very little damage to the feathers of the recipient birds. The more severe form of feather pecking, characterised by feather pulling or even removal of feathers (which in itself is painful), can result in serious damage to the feathers and integument of the victims (Hughes and Duncan, 1972; Keeling, 1995). Severe feather pecking can ultimately lead to cannibalistic acts, as wounded birds (i.e. blood) are attractive for others to peck at (Blokhuis and Arkes, 1984; Savory and Mann, 1997).

An important issue for debate among scientists is whether gentle and severe feather pecking are related, i.e. depend on the same underlying mechanisms (Kim-Madslien, 2000; Kjaer and Vestergaard, 1999; McAdie and Keeling, 2002). There is some evidence indicating that gentle and severe feather pecking may have a different origin and may be differentially affected by genetic and environmental factors (Kjaer, 1999; Nicol et al., 1999). Furthermore, it appears that gentle feather pecking at an early age does not always predict severe feather pecking at a later age (Rodenburg et al., 2003). However, within a certain age gentle and severe feather pecking are mostly positively correlated (Kim-Madslien, 2000; Rodenburg et al., 2003; Riedstra, 2003) suggesting that 'both forms do
represent different extremes of the same behavioural continuum’ (Kim-Madslien, 2000). Thus, the exact relationship between gentle and severe feather pecking is still rather unclear.

It can be argued whether gentle feather pecking is of interest for poultry welfare (McAdie and Keeling, 2002) as gentle feather pecking does not result in feather damage. Gentle feather pecking in young chicks has even been regarded part of normal social behaviour, representing social exploration (Riedstra and Groothuis, 2002) or allopreening (Blokhuis, 1986; Harrison, 1965; Riedstra and Groothuis, 2002; Vestergaard, 1994). However, high frequencies of gentle pecks are often directed at the same spot on the body of another bird, giving gentle feather pecking a very abnormal and stereotypic appearance (Kjaer and Sørensen, 1997; McAdie and Keeling, 2002). It has been recently suggested that ‘normal’ gentle feather pecking in young chicks may develop into stereotyped gentle feather pecking and subsequently into severe feather pecking by either increased intensity or increased severity of inter-bird pecking (McAdie and Keeling, 2002).

Neither this stereotyped form of gentle feather pecking nor severe feather pecking has been reported to appear in the behavioural repertoire of hens living under natural conditions and may therefore be considered abnormal behaviours (Kim-Madslien, 2000; Kruijt, 1964; Vestergaard et al., 1993). As stereotypies are generally a sign of maladaptation to the environment (Mason, 1991; Wiepkema and Koolhaas, 1993), the occurrence of gentle feather pecking may be an early sign of reduced welfare in birds.

3. Causation of feather pecking

Many years of research have revealed a wide range of factors influencing the development or performance of (both gentle and severe) feather pecking behaviour. Many of these factors affecting feather pecking are related to the management of the birds (external factors). However, research has also revealed numerous factors related to the nature of the birds (internal factors), including
genetic predisposition, developmental stage, hormonal state, underlying fearfulness, and social motivations.

3.1 External factors

Availability and quality of a floor substrate

One of the first and most important single causal factors associated with the occurrence of feather pecking is the absence of (suitable) floor substrate (Blokhuis and Arkes, 1984; Hughes and Duncan, 1972; Levy, 1938). Provision of litter of a certain type or texture early in the development of the chick substantially reduces feather pecking (Blokhuis and van der Haar, 1992; Huber-Eicher and Wechsler, 1998). The two most influential current theories on the causation of feather pecking assign an important role to this factor. It has been postulated that feather pecking is a form of re- or misdirected pecking, related to the motivational system of either feeding/foraging (Aerni et al., 2000; Blokhuis, 1989) or dustbathing (Vestergaard, 1994). According to these theories, exposing chicks to litter early in life would prevent them from perceiving feathers as a substrate for either foraging or dustbathing. However, feather pecking is not fully eliminated by providing suitable substrates (e.g. Huber-Eicher and Wechsler, 1998; Nicol et al., 2001), suggesting the involvement of other causal factors.

Stocking density and group size

Feather pecking behaviour has been found to increase with group size (Allen and Perry, 1975; Bilčík and Keeling, 1999; Keeling, 1994) and stocking density (Allen and Perry, 1975; Appleby et al., 1988; Koelkebeck et al., 1987; Simonsen et al., 1980) (Savory et al., 1999). Group size appears to be more important than stocking density. However, since group size is often confounded with stocking density, the role of the individual factors is difficult to distinguish (Nicol et al., 1999; Savory et al., 1999).
Light intensity

The intensity of light influences the incidence of feather pecking. It is generally agreed that increasing the brightness of the light increases the level of feather pecking (Allen and Perry, 1975; Hughes and Black, 1974; Kjaer and Vestergaard, 1999). Kjaer and Vestergaard (Kjaer and Vestergaard, 1999) found that gentle and severe feather pecking are differently influenced by light intensity: Birds reared at 30 lux showed less mild stereotypic pecking but more than twice as much severe feather pecking as birds reared at 3 lux.

Diet and food form

Many of the earlier studies on feather pecking were based on the hypothesis that it was related to deficiencies in certain nutritional components. Outbreaks were ascribed to inadequate levels of e.g. calcium and protein (aminoacids), dietary fibre, sodium chloride (for an overview see Hughes and Duncan, 1972). Savory et al. (1999) reported of a suppression of feather pecking damage with dietary supplementation with ‘higher’ doses of the essential aminoacid L-tryptophan.

Apart from the composition of the diet, dietary texture has also been found to be an important factor. Several authors (for an overview see Hughes and Duncan, 1972) report that pecking damage is more common when birds are fed on pellets rather than on mash. In this respect, there might be an interaction between diet and other environmental factors such as litter. For example, Savory and Mann (1997) found that food form (mash or pellets) had no significant effect on feather pecking in pullets kept in pens with litter-covered floors. Significant effects of foraging material on feather pecking were found in studies in which the birds were fed pellets (Huber-Eicher and Wechsler, 1997; Huber-Eicher and Wechsler, 1998). Aerni et al. (2000) confirmed this interaction effect. High rates of feather pecking and severe feather damage were only found in hens housed without access to straw and fed food pellets.
3.2 Internal factors

Genetic predisposition

Large variation in the performance of feather pecking exists between strains of laying hens, even when they are kept in the same environment (e.g. Bessei, 1986; Blokhuis and Beuving, 1993; Craig et al., 1975; Cuthbertson, 1980; Kjaer, 1995). The use of breeding programmes in order to solve the feather pecking problem requires knowledge on heritability of feather pecking and genetic correlations with other traits (for instance production traits). Although some studies indicate possibilities for such breeding programmes (Craig and Muir, 1996; Kjaer and Sørensen, 1997; Muir, 1996), often the results are not consistent. For instance, heritability estimates for feather pecking range from 0.04 – 0.56 (Bessei, 1984; Cuthbertson, 1980; Damme and Pirchner, 1984; Dickerson et al., 1961), depending on age and method of recording (e.g. scoring of plumage condition, direct observations).

Developmental stage

A number of studies indicate that feather pecking can already be observed at a very early age (Hoffmeyer, 1969; Perry and Allen, 1976; Wennrich, 1975a). Gentle feather pecking is performed by most members in groups of young chicks (Kjaer and Sørensen, 1997; Wechsler et al., 1998). Severe feather pecking is mostly observed at a later age (Huber-Eicher and Sebö, 2001), and is performed by only a limited number of group members (Bessei, 1984; Keeling, 1994). Thus, the intensity or severity of feather pecking seems to depend on age (Rodenburg and Koene, 2003), with peaks appearing in different stages of development (Blokhuis and Arkes, 1984; Hughes and Duncan, 1972). The intensity of feather pecking also seems to change upon onset of lay and sexual maturity, possibly under the influence of changes of gonadal hormones (Blokhuis and Arkes, 1984; Hughes, 1973; Hughes and Duncan, 1972)
Furthermore, ontogenetic factors involving the interaction between environmental conditions and birds at a young age have been reported to play an important role in the development or occurrence of feather pecking later in life (Blokhuis and van der Haar, 1992; Huber-Eicher and Wechsler, 1998; Johnsen et al., 1998; Verbeek et al., 1994).

**Hormonal state**

Hughes (1973) tested the role of a range of gonadal hormones on feather pecking, by using hormonal implants in hens at 12 weeks of age. Up to 18 weeks, progesterone produced a moderate but significant increase in feather pecking. Oestrogen and progesterone together resulted in a much greater increase. From 18 to 24 weeks the normal rise in feather pecking around the onset-of-lay was prevented by testosterone. Hughes suggested that the increase in feather pecking around the onset-of-lay is hormonally mediated, and can either be stimulated by administering a combination of oestrogen and progesterone or be blocked by giving testosterone.

**Fearfulness**

The role of fear in feather pecking behaviour is not clear. Some authors have suggested that feather pecking is more likely to be initiated by fearful birds e.g. (Johnsen et al., 1998; Vestergaard et al., 1993). However, most studies indicate that fearfulness is a consequence of feather pecking, induced by feather damage and pain, rather than a cause (Hansen and Braastad, 1994; Jones and Hocking, 1999; Lee and Craig, 1991).

**Social motivations**

Although it appears that (severe) feather pecking is initially performed by a restricted number of birds, feather pecking and particularly cannibalism can escalate by spreading through a flock (McAdie and Keeling, 2000; Siren, 1963).
The simplest mechanism is that birds are attracted to damaged feathers. Damage to the integument or plumage has been found to facilitate and accelerate outbreaks of feather pecking, leading to a domino effect (Allen and Perry, 1975; Freire et al., 1999; McAdie and Keeling, 2000; Savory and Mann, 1997). Social transmission or social learning have also been suggested to be involved in the spread of feather pecking and cannibalism through a flock (Cloutier et al., 2002; McAdie and Keeling, 2002; Zeltner et al., 2000).

Recently, Riedstra and Groothuis (2002) proposed another mechanism underlying the spread of gentle feather pecking in a flock. They argued that gentle feather pecking plays an important functional role in the building (social exploration) and maintenance of social relationships between chicks. Due to the large group size in husbandry conditions, there might be an exponential increase in the need to engage in and maintain such social relationships.

Jones (1995) and Blokhuis (2001) and their colleagues showed that birds of a low feather pecking line displayed more social reinstatement behaviour than birds of a high feather pecking line. They argued that if high feather pecking birds are less socially motivated than low feather pecking birds, this might compromise their ability to interact successfully with their companions and to adapt to large social groups. Social motivation (at 1, 17, 24 and 30 weeks of age) even predicted the likelihood to develop gentle feather pecking (at 24 and 30 weeks of age) (Blokhuis et al., 2001).

4. Summary and conclusion

From section 3 it is clear that research has succeeded in revealing many factors influencing feather pecking. However, neither a single causal factor has been identified so far, nor a combination of factors has resulted in an adequate solution of the problem. It is generally agreed that feather pecking reflects multifactorial processes (Hughes and Duncan, 1972), in which the interaction between external (environmental) and internal (animal-based) factors affects its occurrence.
As mentioned earlier, there is a growing acceptance that feather pecking is a behavioural pathology (Sanotra et al., 1995; Zeltner et al., 2000). However, from a neuroendocrine perspective, hardly any scientific evidence is available to provide a foundation for this classification of feather pecking. To our knowledge, no attempts were made to draw a parallel between feather pecking and behavioural pathologies in other species, (e.g. stereotypies and obsessive compulsive disorders). It is conceivable that neurobiological and neuroendocrine mechanisms known to underlie such disorders may play a role in the development or performance of feather pecking behaviour as well. Therefore, in this thesis, feather pecking will be approached from a neurobiological angle (see also section 1).

5. A new approach to unravel mechanisms underlying feather pecking

5.1 Individual vulnerability

A wide range of studies, both in human and animals, demonstrated that individuals can profoundly differ in their vulnerability for the development of a behavioural pathology. The likelihood for an individual to develop dysfunctional behaviour depends on a complex interaction between a genotypic (pre)disposition and factors like ontogeny, adult life experiences and age. The outcome of this interaction determines the capacity of an individual to cope with various environmental demands.

Behavioural pathologies may develop when an individual fails to adapt to these environmental demands (Koolhaas et al., 2001). Individual difference in vulnerability for the development of behavioural pathology are accompanied by clear differences in neuroendocrine reactivity and neurobiological makeup (Koolhaas et al., 2001). For instance, the functioning of the hypothalamus-pituitary-adrenal (HPA) axis, the serotonergic (5-HT) system and the dopaminergic (DA)
system have been suggested to be divergent in those individuals that are prone to
the development of psychopathological disorders (see also box 1.2, 1.3 and 1.4).

As mentioned in section 3, large individual and strain differences have
been found for the performance of feather pecking as well. One of the first to
recognise that individual vulnerability for the development of feather pecking may
be a very useful tool in discovering underlying characteristics of birds, were
Blokhuis and Beutler (1992). They used two genetic lines of laying hens differing in
their propensity to feather peck. These so-called high (HFP) and low (LFP) feather
pecking lines of laying hens differed, at an adult age, in the level of feather pecking
damage (Blokhuis and Beutler, 1992) and feather pecking behaviour (Blokhuis and
Beuving, 1993). Apart from a marked difference in the level of feather pecking,
other authors showed that HFP and LFP birds also differ in several other
behavioural characteristics, such as fear and sociality (Johnsen and Vestergaard,
1996; Jones et al., 1995).

Korte and his colleagues (1997, 1999) were the first to investigate
physiological characteristics of adult birds of both lines. On the basis of the marked
behavioural differences between HFP and LFP birds in open-field behaviour found
by Jones et al. (1995), they anticipated differences between lines in the
behavioural and physiological reactivity to an acute stressor. In the experiments by
Korte, it was shown that in response to acute stress induced by manual restraint
(i.e. placing a bird on its side for 8 minutes), adult HFP birds displayed more
struggling behaviour, lower heart rate variability, higher plasma noradrenaline and
lower plasma corticosterone levels than LFP birds. Surprisingly, these behavioural
and physiological characteristics of HFP and LFP hens showed considerable
analogy to the characteristics of the proactive and reactive coping strategies,
respectively, previously found in rodents (Koolhaas et al., 2001). Korte (1997)
postulated that the propensity of a bird to perform feather pecking is related to its
coping strategy, i.e., the way it deals with environmental challenges, both
behaviourally and physiologically.
BOX 1.1 Physiological and neuroendocrine characteristics of the proactive and reactive coping strategy in rodents

Cort = corticosterone, NA = Noradrenaline, A = Adrenaline, 5-HT = serotonin, DA = Dopamine

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<td>HPA-axis reactivity (Cort)</td>
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<td>Neurosympathetic reactivity (NA)</td>
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<td>Adrenomedullary reactivity (A)</td>
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(Koolhaas et al., 2001)

5.3 Coping strategy: a guide in feather pecking research

Research in both humans and animals has revealed a number of neurobiological systems involved in the aetiology of psychopathological behaviours. Especially the HPA-axis (box 1.2), the serotonergic (5-HT) system (box 1.3) and the dopaminergic (DA) system (box 1.4) have been implicated in several behavioural pathologies, including depression, excessive aggressive behaviour, (animal) stereotypies, and obsessive compulsive disorders (OCD) (see boxes for references). Notably, these are also the neurobiological systems involved in the differentiation between the proactive and reactive coping strategy in rodents (see box 1.1).

As mentioned above, studies by Korte et al. (1997, 1999) suggest that the neurobiological characteristics of adult birds of the HFP and LFP line are similar to those of rodents displaying a proactive and reactive coping strategy, respectively. Therefore, in this thesis, the concept of coping strategy will be used as a guide in unravelling possible behavioural and neuroendocrine mechanisms underpinning the development and performance of feather pecking.
BOX 1.2  Hypothalamus Pituitary Adrenal (HPA) axis

In birds, as in mammals, high levels of glucocorticoids in the blood plasma can be indicative for a response to acute stressors (Beuving and Vonder, 1978; Gross, 1990). In reaction to an acute stressor, corticosterone, the main corticosteroid in the avian blood plasma, is released from the adrenal cortex, in response to adrenocorticotropic Hormone (ACTH). ACTH is released from the pituitary under the influence of corticotropin-releasing hormone (CRH), which in turn is produced by the hypothalamus. Corticosterone exerts an inhibitory influence on the activity of the hypothalamus-pituitary-adrenal (HPA)-axis (i.e. on CRH and ACTH release) and extra-hypothalamic structures through interaction with corticosteroid receptors. The corticosteroid receptors in the brain consists of two distinct types of receptors; the mineralocorticoid receptor (MR) and the glucocorticoid receptor (GR). Both intracellular receptor types differ in primary structure, localisation and function (de Kloet et al., 1990). MRs bind corticosteroids with a 10-fold higher affinity than GRs (de Kloet et al., 1998). MRs are highly concentrated in the hippocampus and are mainly involved in the organisation of circadian-driven daily activities and the regulation of basal activity of the HPA-axis. MRs determine the threshold or sensitivity for stress-induced activation of the HPA system (de Kloet et al., 1991). GRs are more widely expressed in the brain and are suggested to be involved (in conjunction with MRs) in corticosterone-mediated feedback on stress-induced activation of the HPA axis.

In humans, functional abnormalities of the HPA-axis and disturbances in glucocorticoid regulation via MRs and GRs, have been implicated in the aetiology of several behavioural disorders, such as depression (Heuser, 1998; Holsboer and Barden, 1996; Reus and Wolkowitz, 2001; Seckl et al., 1990; Steckler et al., 1999), anxiety (Korte, 1991; Korte et al., 1995) and aggression (Haller et al., 1998; Haller et al., 2000).

6. Aim and outline of this thesis

The main scientific aim of this thesis is to identify behavioural, neurobiological and neuroendocrine characteristics of laying hens, that may be causally related to feather pecking behaviour. Birds of the HFP and LFP line will be used as a model for high and low feather pecking, as was previously done by other authors (Blokhuis and Beutler, 1992; Blokhuis and Beuving, 1993; Johnsen and Vestergaard, 1996; Jones et al., 1995; Korte et al., 1997; Korte et al., 1999; McAdie and Keeling, 2002). The extreme differences in behaviour and stress physiology between birds of both lines may help to give a better insight in
mechanisms underlying feather pecking behaviour. Neuroendocrine systems associated with the differentiation of coping strategies, and known to be involved in behavioural pathologies in other species are investigated, including the HPA axis, the serotonergic and dopaminergic system.

In the present thesis it will be investigated whether:

1. the concept of coping strategy represents a useful framework for studying the mechanisms underlying feather pecking. It will be investigated whether differences in behaviour, physiology and neurobiology between birds of the HFP and LFP line are consistent with current knowledge of the proactive and reactive coping strategy, respectively.

2. the development and performance of feather pecking can be explained by differences in neuroendocrinology (HPA-axis, 5-HT and DA) between the two lines. In this thesis, emphasis will lie on investigating the (possible causal) role of 5-HT in the development and performance of feather pecking behaviour.

As mentioned earlier, age was found to be an important factor in the occurrence of feather pecking. Furthermore, different motivational systems have been implicated in the development of feather pecking, i.e. feeding/foraging and dustbathing (section 3). Adult HFP and LFP birds have been shown to differ in feather pecking behaviour, however it is unclear at which developmental stage HFP and LFP chicks start to show differences in feather pecking. Furthermore, it is not known which motivational systems are involved in the development of feather pecking in either line. Therefore, in chapter 2, a study is described investigating the development of feather pecking and related behaviours during the first 8 weeks of life of HFP and LFP chicks.

The clear differences between adult HFP and LFP birds in the behavioural and physiological responsiveness were previously interpreted in terms of differences in coping strategy (Korte et al. 1997; 1999). Furthermore, it was postulated that these differences are causally related to the differences in feather pecking between both lines (Korte et al. 1997; 1999). In Chapter 3 it is investigated whether differences between lines in behavioural development (chapter 2) are associated with physiological and neuroendocrine differences as well.
The neurotransmitter serotonin or 5-hydroxytryptamine (5-HT) is found throughout the central nervous system (CNS). Within the CNS, the cell bodies of 5-HT neurons are clustered along the midline (raphe nuclei) of the midbrain and the brain stem of both mammals and birds including chicken (Okado et al., 1992). These neurons send ascending projections to most parts of the forebrain and descending projections to lower brain stem regions and to the spinal cord. Serotonergic neurons in the brain synthesise 5-HT from the amino acid L-tryptophan. The (rate limiting) enzyme, tryptophan-5-hydroxylase, converts L-tryptophan into 5-hydroxytryptophan, which is then decarboxylated to 5-hydroxytryptamine by 5-hydroxytryptophan decarboxylase (Blier and de Montigny, 1998). Dietary L-tryptophan supplementation (Fernstrom, 1983; Harrison and D'Mello, 1986) or depletion (Klaassen et al., 1999; Young and Leyton, 2002) can be used to increase or decrease 5-HT levels in the brain, respectively.

5-HT is synthesised in nerve terminals and stored in vesicles. After release into the synaptic cleft, 5-HT can bind to receptors of different subtypes. Via binding to these different receptors, 5-HT can influence many parts of the brain involved in controlling a variety of physiologic functions, including mood, behaviour, pain, appetite, endocrine secretion and cardiovascular function. After acting on pre- and postsynaptic receptors, 5-HT is transported back into the nerve terminal via uptake carriers, where it may be recycled in storage granules, or destroyed by a mitochondrial enzyme, monoamine oxidase (MAO). The major degradation product, or main metabolite of 5-HT is 5-HIAA (Fuller, 1995).

5-HT neuron dysfunction appears to have an aetiological role in various behavioural pathologies, since drugs that modify serotonin function are potentially useful in treating many psychopathological disorders, including aggression (Chiavegatto et al., 2001; Lesch and Merschdorf, 2000; van der Vegt et al., 2001), depression (Blier and de Montigny, 1998; Lucki, 1998; Schreiber and de Vry, 1993) and (animal) stereotypies (Kolštál and Savory, 1995; Pitman, 1989; Schoenecker and Heller, 2001). Obsessive Compulsive Disorder (OCD) (Blier and de Montigny, 1998; Luescher, 1998; Pigott, 1996; Stein, 2000). The role of 5-HT in aggression has been extensively studied. Low concentrations of 5-HIAA in the cerebrospinal fluid have been consistently found as a trait characteristic in highly violent, aggressive individuals (van der Vegt et al., 2001). In addition, many animal studies show anti-aggressive effects of selective 5-HT1A and 5-HT1B receptor agonists (which lower 5-HT release), supporting a role of the 5-HT system in aggression (Brown and Linnoila, 1990; de Boer et al., 1999; de Boer et al., 2000; van der Vegt et al., 2001).

Treatment of psychiatric disorders like depression and OCD, involves administration of tricyclic antidepressants or selective serotonin reuptake inhibitors (SSRIs), like for instance fluoxetine (‘Prozac’). Chronic administration of SSRIs, increases 5-HT neurotransmission, and alleviates the symptoms of these disorders (Stein, 2000; Vaswani et al., 2003).
5.2 Coping strategies

Coping strategy is defined as the complex of individual behavioural, physiological and neurobiological characteristics that determine how an individual responds to environmental challenges (Koolhaas et al., 2001).

Coping strategies are characterised by noticeable differences in behavioural response to environmental challenges. Generally, two extremes in behavioural coping strategy are distinguished, originally based on differences in the expression of territorial aggression (Benus et al., 1991b; Benus et al., 1987; Koolhaas et al., 1997); i.e. the ‘active’ fight/flight response, first introduced by Canon (1915) and the ‘passive’ conservation-withdrawal response originally described by Engel and Schmale (1972).

Koolhaas and his colleagues (1997) introduced a new terminology for the two coping strategies: ‘proactive’ (previously labelled ‘active’) and reactive (previously labelled ‘passive’) coping strategies. The basis of this new terminology is the consistency in the way high and low aggressive mice behaviourally react in a wide variety of environmental challenges, either proactively (‘first do, then think’) or reactively (‘first think, then do’). A very fundamental difference seems to be the degree in which behaviour is guided by environmental stimuli (Koolhaas et al., 2001). Proactive animals act primarily on the basis of earlier experience and easily forms routines, whereas the reactive copers are more guided by the information actually present in their environment. These differences in behavioural control mechanisms determine the adaptive character of the two coping strategies: proactive animals are better adapted to stable, highly predictable environmental conditions, whereas reactive copers may adapt well in variable and unpredictable environmental conditions (Benus et al., 1990; Koolhaas et al., 1999).

Besides consistent behavioural differences between proactive and reactive rodents, fundamental differences in the functioning of physiological and neurobiological systems (see box 1.1) have been found, underlying the behavioural differences (for a review on these differences see Koolhaas et al., 2001).
As a first step, the development of adrenocortical (re)activity in HFP and LFP chicks during the first 8 weeks of life was studied. Secondly, we studied DA and 5-HT turnover in the brain of HFP and LFP chicks, at the age of 28 days.

The clear differences between adult HFP and LFP birds in the behavioural and physiological responsiveness were previously interpreted in terms of differences in coping strategy (Korte et al. 1997; 1999). Furthermore, it was postulated that these differences are causally related to the differences in feather pecking between both lines (Korte et al. 1997; 1999). In Chapter 3 it is investigated whether differences between lines in behavioural development (chapter 2) are associated with physiological and neuroendocrine differences as well. As a first step, the development of adrenocortical (re)activity in HFP and LFP chicks during the first 8 weeks of life was studied. Secondly, we studied DA and 5-HT turnover in the brain of HFP and LFP chicks, at the age of 28 days.

The 5-HT and DA system have been implicated in the distinction between coping strategy (Koolhaas et al., 2001) and in the aetiology of behavioural pathologies (Brown and Linnoila, 1990; Ellison, 1994; Goodman et al., 1990; McDougle et al., 1994; Stein, 2000). Therefore, we investigated the role of the 5-HT system in the performance and development of feather pecking. The effect of a decrease (chapter 4) and an increase (chapter 5) of 5-HT turnover in the brain of LFP and HFP chicks on feather pecking behaviour are described.

Chapter 6, reports about a study, in which, as a first step of investigating a possible role of the DA in feather pecking behaviour, we investigated the sensitivity of the DA receptor system of HFP and LFP chicks. We examined the effect of acute APO treatment on behavioural responses in an open field, to identify the hypothesised difference in DA receptors sensitivity between chicks of both lines.

In chapter 7 an integrated discussion is given, by raising the main topics and findings of this thesis.
The neurotransmitter dopamine (DA), together with adrenaline and noradrenaline, is called a catecholamine. Dopamine is synthesised from tyrosine through the actions of two enzymes, tyrosine hydroxylase and decarboxylase. Tyrosine hydroxylase, the rate-limiting enzyme in the process, converts L-tyrosine to dihydroxy-L-phenylalanine, or L-DOPA. Then, the aromatic amino acid decarboxylase immediately converts L-DOPA to DA. In some cells, DA is further converted to the neurotransmitter noradrenaline or even further to adrenaline, but in many neurons DA itself serves as an active neurotransmitter. Once DA is released from presynaptic DA containing vesicles in the terminals, it can interact with postsynaptic DA receptors or different types of presynaptic autoreceptors that regulate transmitter release, synthesis, or firing rate. DA receptors have been classified into two major families (including many subtypes): D₁ and D₂ receptors. Interneuronally (i.e. after reuptake into the neuron) and extraneuronally, DA is metabolised (e.g. by MAO) to the substances DOPAC and HVA, respectively (Tzschentke, 2001).

Apomorphine (APO), a DA receptor agonist is frequently used to evaluate DA function or activity in physiological processes or neuropsychiatric disorders (Lal, 1988), or predict individual differences in sensitivity of DA receptors (Surmann and Havemann-Reinecke, 1995). In several animal species, injection with APO induces stereotyped behaviour and increased locomotor activity (Berridge and Aldridge, 2000; Bolhuis et al., 2000; Delius, 1988; Godoy et al., 2000; Surmann and Havemann-Reinecke, 1995; Zarrindast and Amin, 1992).

Dopamine has been implicated in a variety of functions including motor control, cardiovascular regulation, cognition, learning, endocrine regulation and emotion. Like the HPA-axis and 5-HT, DA dysfunction has been implicated in the aetiology of psychopathological disorders, like schizophrenia (Ellison, 1994) and OCD (Goodman et al., 1990; McDougle et al., 1994). For instance, excessive DA neurotransmission in the forebrain is believed to underlie schizophrenia. Drugs blocking postsynaptic D₂ receptors are effective in treating this disorder (Marcotte et al., 2001; Moore et al., 1999). In OCD, augmentation of DA neurotransmission, either through administration of serotonin reuptake inhibitors, via an interaction between 5-HT and DA (Stein, 2000) and/or haloperidol (van Ameringen et al., 1999), is thought to alleviate the symptoms.