Nutritional Effects on Serotonin and Behavioural Aggression in Dogs

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Introduction

Each year millions of dogs worldwide are abandoned, relinquished to animal shelters, and euthanazed because of behavioural problems (Bosch *et al*., 2007). Veterinary behaviourists have reported that aggression is the main reason dogs are referred to their clinics (Cakiroglu *et al*., 2007). Aggression is a killer and nutrition is rarely considered as one possible contributing factor to canine behaviour (Lofflin, 2007; Bosch *et al*., 2007). Behaviour is regulated by neurotransmitters and hormones, and thus may be influenced by changes in the availability of their precursors (Bosch *et al*., 2007). This has important implications for animal welfare as fixing aggression issues takes time and owners of aggressive dogs frequently opt for euthanasia (Lofflin, 2007; Cakiroglu *et al*., 2007). Animal welfare will greatly benefit if aggressive behaviour can be moderately controlled by changes in diet.

Discussion

Serotonin or 5-hydroxytryptamine(5-HT) is a neurotransmitter in the central nervous system with an important role in regulating many physiological functions within the body, including sleep, appetite, memory, learning, mood, sexual behaviour, endocrine regulation and depression (Bosch *et al*., 2007). In a study conducted by Cakiroglu *et al*. (2007), 23 dogs characterised by the research group as aggressive and 18 normal dogs, of both sexes and of various breeds, were examined for serum concentrations of serotonin. The results of this study found that the mean serum concentration of serotonin was significantly lower in aggressive dogs. This corresponds to previous studies that linked decreased serotonergic function to behavioural aggression in dogs (Reisner *et al*., 1996; Badino *et al*., 2004).

A study by Jacobs *et al*. (2007) aimed to quantify neurons expressing the serotonin-1B receptors in the basolateral nuclear group (BNG) of the canine amygdala. This enabled an enumeration of differences in normally behaving (n=11) and pathologically aggressive dogs (n=11) to assess whether this receptor is involved in canine aggression. Results confirmed the BNG of the aggressive dogs contained a significantly higher number of serotonin-1B receptor-expressing neurons compared with the controls. This is an apparent contradiction of the aforementioned findings of Reisner *et al*. (1996) and Cakiroglu *et al*. (2007). A possible explanation could be that lower serotonergic activity is present in aggressive dogs because stimulation of pre-synaptic serotonin-1B autoreceptors causes a reduction of serotonin release (Jacobs *et al*., 2007).

Badino *et al*. (2004) suggested that drugs targeting 5-HT receptors might be useful in a wide range of behavioural canine disorders, especially in modulating aggressive behaviour. Serotonin-1B receptors act pre- and post-synaptically and function as autoreceptors regulating serotonin release at the serotonergic nerve terminal. Some serotonin-1B agonists have anti-aggressive properties, possibly because they mimic a highly active serotonergic system (Jacobs *et al*., 2007). This was demonstrated by Dodman *et al*. (1996) in fluoxetine trials that significantly reduced canine aggression. However, the effects of serotonin-1B receptor agonists on aggression depend on whether they act pre- or post-synaptically, because stimulation of the pre-synaptic autoreceptor will reduce serotonergic neurotransmission.

Conversely, in studies explored by Bosch *et al*. (2007) the focus shifted away from pharmaceutical dependency and onto the effects of dietary supplementation of tryptophan (precursor of serotonin) and its potential to curb aggressive behaviour. Hydroxylation of tryptophan to 5-HT by the enzyme tryptophan hydroxylase is the first, and rate limiting, step in serotonin synthesis. As tryptophan hydroxylase is normally only half saturated with tryptophan, an increase in central tryptophan can maximally double serotonin synthesis and thus increase serotonergic neurotransmission. However, the availability of tryptophan to the brain largely depends on the composition of the ingested food.

Tryptophan is found in nearly all foods containing protein, but in lower concentrations compared to other large neutral amino acids (LNAAs). To access the brain through the blood-brain barrier, tryptophan shares the same carrier as other LNAAs. Due to these lower tryptophan concentrations, consumption of a high-protein meal will decrease the ratio of tryptophan to other LNAAs, potentially lowering serotonin synthesis as a result of carrier competition.

Exploring this concept in relation to canine aggression seems limited to one study. DeNapoli *et al*. (2000) formulated diets with high- or low-protein content (approximately 310 or 190g crude protein/kg respectively) and with or without tryptophan supplementation (1.45g/kg) in order to vary tryptophan content and tryptophan:LNAA ratios. Each of the four diets was fed in random order for 1 week to 33 privately owned dogs that displayed high territorial aggression, dominance aggression or hyper activity (n=11 per mutually exclusive group). When analysed as one study population, it was found that territorial aggression significantly decreased in dogs fed the high-tryptophan diet compared with dogs fed the low-tryptophan diet, but only when fed a low-protein diet. Also, dogs fed the high-protein diet without tryptophan supplementation showed a higher dominance aggression score compared with dogs on other dietary treatments. This correlates to findings from Mugford (1987), who reported that low-protein diets decreased aggression in dogs. Unfortunately, this study was not performed under controlled conditions and so could not irrefutably conclude that the result was due to protein concentration.

Most dog foods currently conform to the Association of American Feed Control Officials (AAFCO) guidelines, which set the minimal tryptophan requirements at 0.0669 g/1000 kJ metabolisable energy with a tryptophan:LNAA ratio of 0.061:1 (AAFCO Dog Food Nutrient Profiles, 2008). Animal behaviour is not taken into account as a criterion for passing an AAFCO maintenance feeding protocol. It is currently unknown whether this minimum tryptophan level meets the requirements of the wide variety of dogs, from emotionally stable to anxious individuals (Bosch *et al*., 2007).

Conclusion

Presently, few studies have been conducted to evaluate the role of nutrition in (problem) canine behaviour through the abovementioned mechanisms. As diet composition, nutrient availability and interactions affect the availability of this serotonin precursor in the brain, it is highly probable that nutrition is a contributor to aggressive behaviour. Furthermore, estimates of the maximum amount of tryptophan in canine food are currently not required and it remains to be determined how high-tryptophan diets affect the health of dogs and their behaviour in the long term (Bosch *et al*., 2007). Studies that explore this relationship may greatly help to improve the welfare of dogs and their owners.

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