Marmoset wasting syndrome in common marmosets (*Callithrix jacchus*): developments in aetiology and diagnosis of captive individuals

Investigates recent developments regarding the aetiology and diagnosis of marmoset wasting syndrome in common marmosets (Callithrix jacchus).

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Introduction

Common marmosets (*Callithrix jacchus*) are new world monkeys of the Callitrichid family, originating from eastern and northern Brazil. Their small body size, high reproductive success, ease of handling and lack of severe zoonotic diseases have made them a popular species for both research facilities and zoological parks (Niimi, Morishita et al. 2019). A prominent welfare issue associated with common marmosets in captivity is marmoset wasting syndrome (MWS). This syndrome incorporates a variety of clinical signs across Callitrichids, likened to those of inflammatory bowel disease (IBD) in humans, including chronic diarrhoea, anaemia, muscular atrophy, hypoalbuminaemia and histopathological findings of lymphocytic enteritis. This results in a decreased condition score and failure to thrive, with a poor prognosis once severe clinical signs are observed (Cabana, Maguire et al. 2018, Douay, Maguire et al. 2019, Niimi, Morishita et al. 2019). As MWS is multifactorial, the aetiology and pathogenesis are poorly understood. Researchers have hypothesised that MWS may be exacerbated by nutritional and infectious factors, poor husbandry practices and environmental stress (Cabana, Maguire et al. 2018). The syndrome is a significant welfare issue in marmosets; MWS has never been observed in wild individuals, however captive groups experience rates as high as 60%, with up to 44% of captive marmoset deaths being attributed to the disease (Niimi, Morishita et al. 2019). This essay investigates recent advances in the aetiology and diagnosis of MWS in common marmosets.

Discussion

In the decades since discovery of MWS in 1976, volumes of research have focused on the nutritional aspect of MWS, with the overriding notion that the syndrome is due to nutritional (specifically protein) deficiencies (King 1976, Cabana, Maguire et al. 2018). Cabana et al. (2018) investigated these hypotheses via surveys and Zoological Information Management System (ZIMS) database records, returning information on a total of 1,218 Callitrichid individuals. Crude protein was not found to be a significant predictor in the development of MWS. Additionally, Cabana et al. (2018) noted the disconnect between captive fed diets and wild diets. Wild marmosets typically spend around 70% of their foraging time consuming tree gum, which has a crude protein content significantly lower (4−7%) than previous captive suggestions (25−30%). Cabana et al. (2018) therefore stipulate that reaching crude protein contents of 25% would be highly unlikely in the wild, therefore the likelihood of protein deficiencies being related to MWS is low.

Conversely, a study aiming to detect the underlying causes of hypoalbuminaemia in MWS-affected marmosets via the development of an ELISA test found that individuals with MWS typically had a protein-losing gastroenteropathy (Niimi, Morishita et al. 2019). A study by Niimi et al. (2019) investigated the concentrations of α1-proteinase inhibitor (α1-P1) in both serum and faecal concentrations to determine whether this could be used as an indicator for hypoalbuminaemia, and subsequently MWS. α1-P1 was used as a proxy for albumin; it has a similar molecular weight, thus passing into the lumen of the gastrointestinal tract under similar conditions. It is resistant to enzymatic digestion and bacterial degradation in the gut, making it suitable for faecal testing. The study found that increased levels of α1-P1 in faeces signified protein loss via the GIT due to intestinal damage, thereby indicating the potential presence of MWS (Niimi, Morishita et al. 2019).

This provides a contradiction to the Cabana et al. (2018) study in the role, or lack thereof, of protein deficiency in MWS. It is possible that while increasing crude protein intake does not act as a protective factor against MWS (Cabana, Maguire et al. 2018), supplementing with protein once an animal is already affected may alleviate signs by replacing protein as it is lost through the GIT (Niimi, Morishita et al. 2019).

As MWS is in many ways comparable to IBD in humans, Douay et al. (2019) investigated the potential of using commercial human IBD rapid tests to detect MWS in common marmosets via the faecal biomarkers of calcoprotectin and lactoferrin, both of which are found in high faecal concentrations of human patients with IBD. Lactoferrin was found to be negative in all Callitrichid samples, likely indicating the absence of a cross-reaction between Callitrichid and human lactoferrin with the mouse monoclonal antibodies upon which the test depends. Calcoprotectin tested positively in 64.9% of marmoset cases, however, displayed low specificity (Douay, Maguire et al. 2019). As such, commercial human tests were found to be unsuitable in the detection of MWS in common marmosets. However, the use of faecal biomarkers has excellent potential if adequate tests are developed.

Stress is a crucial factor in evaluating the aetiology of MWS (Cabana, Maguire et al. 2018, Douay, Maguire et al. 2019). Preventing environmental stressors by grouping individuals with only family members, providing private areas away from visitors, and housing marmosets out of visual, olfactory and auditory range of predators all have protective factors against the development of MWS (Cabana, Maguire et al. 2018, Douay, Maguire et al. 2019). Further research is required to establish the role of stress in MWS, as it is not known whether chronic stress initiates MWS, or merely worsens signs in sub-clinical individuals.

As with many wildlife studies, the sample size was relatively small in both the Niimi et al. (2019) and Douay et al. (2019) studies. It is also difficult to collect consistent data when relying on records from a multitude of organisations, as in the Cabana et al. (2018) study. While environmental stressors are labelled as being a key factor in the development of MWS, the variation between captive environments makes it difficult to pinpoint exact triggers. As such, the results should be viewed with caution and used as building blocks for further research.

Conclusion

It is evident that MWS is still poorly understood despite its prevalence and negative welfare implications on captive common marmosets. While positive progress has been made in identifying risk and protective factors, diagnosis and subsequent treatment is still problematic. Further studies are therefore required to establish methods of early detection and appropriate treatment to improve the welfare of individuals and groups of common marmosets.

References

Cabana, F, Maquire, R, Hsu, C & Plowman, A 2018, 'Identification of possible nutritional and stress risk factors in the development of marmoset wasting syndrome', *Zoo Biology,* vol. 37, no. 2, pp 98-106.

Douay, G., Maquire, R, Cabana, F, Berthet, M, Lim, T & Meredith, A 2019, 'Assessment of the utility of a commercial calcoprotectin and lactoferrin rapid test in diagnosis of marmoset wasting syndrome', *Journal of Zoo and Aquarium Research*, vol. 7, no. 2, pp 67-73.

King, G 1976, 'An investigation in wasting marmoset syndrome at Jersey Zoo', *Annual Report of the Jersey Wildlife Preservation Trust*, vol. 5, pp 97-107.

Niimi, K, Morishita, H, Usui, M, Ito, R, Kurata, S, Mataga, N & Takahashi, E 2019, 'Measurement of the α1-proteinase inhibitor (α1-antitrypsin) of common marmoset and intestinal protein loss in wasting syndrome', Bioscience reports, vol. 39, no. 7, BSR20190562.