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RESEARCH ARTICLE

The Andean bear alopecia syndrome may be caused by social housing

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Abstract

The Andean bear alopecia syndrome is a progressive and chronic condition documented in ex situ populations. Recent advances focus on treating symptoms, not preventing future cases. We therefore explored the epidemiology of this syndrome through an analysis of husbandry and veterinary conditions of 63 Andean bears (26M:37F) housed in North and South American zoos and other ex situ circumstances. We had the most complete information for the North American population and found that 29% of females ($n = 24$) were affected. No males ($n = 26$) were affected. An analysis of generalized linear models indicated that three models were competitive in describing the occurrence of the condition (i.e., $\Delta AIC_c \leq 2$): the model including only the individual's sex ($\chi^2 = 13.41$, df = 1, $p < .001$), the model including both individual sex and social housing status ($\chi^2 = 1.36$, df = 2, $p < .001$), and the model including both individual sex and the expression of stereotypical behaviors ($\chi^2 = 13.82$, df = 2, $p = .001$). Stereotypical behaviors were common among both males (50%, $n = 26$) and females (51.9%, $n = 27$) whether or not they were affected, but the syndrome was seen only in females who had been socially housed. Therefore, we suggest that the Andean bear alopecia syndrome is a symptomatic response to the long-term social housing of bears that would otherwise not live socially. To prevent new cases, we recommend that female Andean bears be housed with adult conspecifics only when females choose to cohabitate.

KEYWORDS
alopecia, captivity, epidemiology, ex situ, Tremarctos ornatus

1 | INTRODUCTION

Modern zoological institutions manage their collections in pursuit of several objectives which require resources and face constraints: education, outreach and engagement, sustainable management of the ex situ population, and support for in situ research and conservation (e.g., Gilbert & Soorae, 2017; Jensen, Moss, & Gusset, 2017). Unfortunately, six of the eight extant bear species are of conservation concern (IUCN Operations Intelligence Center, 1964; www.iucn.org), thus zoo-housed bears—charismatic and popular among zoo visitors—may be effective conservation ambassadors for their wild counterparts. The Andean bear (*Tremarctos ornatus*) is among these vulnerable species, being of conservation concern both *in situ* (Velez-Liendo & Garcia-Rangel, 2017) and ex situ (e.g., Silver, Connolly, & Groome Bryan, 2017). Although Andean bears have been of global conservation concern since 1964 (IUCN Operations Intelligence Center), and despite decades of field research (e.g., Grimwood, 1968; Mondolfi, 1989; Peyton, 1980, 1984; Suárez, 1988), relatively few

quantitative field data exist to guide husbandry for bears in human care. Husbandry and collection professionals must therefore sometimes use trial and error to develop best management practices (Alroy, 2015; Bowkett, 2014; Fa, Gusset, Flesness, & Conde, 2014; Keulartz, 2015; Martin, Lurbiecki, Joy, & Mooers, 2013).

Health problems, ranging from subtle to obvious, have been documented in zoo-housed bears. (e.g., Borbón-García, Reyes, Vives-Flórez, & Caballero, 2017). Skin pathologies of various kinds are frequent (11% of 512 bears) among such bears, including Andean bears (Blake & Collins, 2002), but their etiology is often unknown (29% of 54 cases). Chronic progressive hair loss, or Andean bear alopecia syndrome, has several externally visible characteristics: pruritus, alopecia that frequently involves the face and periocular region as well as the caudal lumbar region and the limbs, progressive hair loss that is generally bilaterally symmetric, mucopurulent ocular discharge or conjunctivitis and changes to the epidermis (Nicolau et al., 2018) that can include thickening, roughness, and increased pigmentation. Prevalence appears much higher in females than males (Jäger et al., 2013), with no clear association to female reproductive status (Langguth, Schachtner, Eulenberger, Kolter, & Bernhard, 2010). Obvious symptoms are usually mild during the initial episodes, which often begin in spring/summer and abate in fall/winter, at which point some hair regrowth may occur. Eventually, however, hair loss appears permanent. Veterinary staff at different zoological institutions have diagnosed this syndrome in different ways (e.g., Langguth et al., 2010): hypersensitivity dermatitis and folliculitis, hypothyroidism, hypoestrogenism, allergic dermatitis but not atopy, all of which are mechanistic explanations that do not address the underlying causation. This syndrome has been observed in Andean bears in human care in Asia (Ueda et al., 2004), Europe (Drake et al., 2017), North America (Owen, Shanks, Sutherland-Smith, Thomas, & Van Horn, 2008), and South America (Restrepo Valencia, 1989). Histological evidence is consistent with an immune response triggered by an unknown cause (Nicolau et al., 2018), but given the global distribution and relatively common occurrence of this syndrome, it is unlikely to arise from a specific allergic reaction, a common source of infection, or simple hypothyroidism.

Variation in the social environment can have complex and subtle effects on the physiology of non-social mammals (e.g., Creel, Danzer, Goymann, & Rubenstein, 2013), so the social environment of bears in human care may affect their physiology. For diverse reasons, adult bears are sometimes housed socially. However, adult wild bears, including Andean bears (Appleton et al., 2018), are thought to directly interact primarily for reproductive purposes, except for brief periods of social interaction when localized resources are abundant (American black bears, *Ursus americanus*: Herrero, 1983; Rogers, 1987; brown bears, *Ursus arctos*: Clapham & Kitchin, 2016; Gill & Helfield, 2012; Peirce & Van Daele, 2006; Penteriani et al., 2017; and polar bears, *Ursus maritimus*: Herreman & Peacock, 2013). Thus, social stress experienced by a typically non-social bear species has been suggested as one possible risk factor for the Andean bear alopecia syndrome (Drake, Bechstein, & Kolter, 2016; Jäger et al., 2013). Stress in zoo-housed polar bears may be correlated with stereotypies (Shepherdson, Lewis, Carlstead, Bauman, & Perrin, 2013), so stereotypic behavior and the alopecia syndrome in Andean bears could be indirectly associated, as two indicators of stress. Kinship or heredity has also been suggested to play a role in the syndrome's occurrence (Drake et al., 2016; Jäger et al., 2013; Leclerc et al., 2015). This syndrome often affects a large portion of an individual's body, making it easy to detect visually, but to our knowledge it has never been reported to occur in wild Andean bears, which are often photographed by camera traps (e.g., Molina, Fuller, Morin, & Royle, 2017; Reyes et al., 2017). Its prevalence in captivity, yet scarcity or absence in the wild, suggests that it results from the ex situ environment.

To alleviate these symptoms, numerous pharmacological treatments have been attempted with little or no long-term efficacy, though recent developments are promising (Drake et al., 2017). However, even if effective treatments are found for affected individuals, the underlying cause of the syndrome has not been identified (Nicolau et al., 2018), meaning that additional individuals may be affected. In an attempt to identify factors that influence the occurrence of this syndrome, suggest hypotheses for its expression, and thereby stimulate evaluation of practices that would prevent its development or reduce its expression, we undertook an analysis of husbandry practices associated with chronic alopecia among Andean bears housed in North and South America.

A chronic syndrome such as this might indicate that environmental or physiological conditions have altered the affected individuals' physiology in ways that would have subtle but important effects. For example, in other species, maternal condition or physiology may alter birth sex ratio, as suggested by evolutionary theory predicting that females will invest in offspring pre- and post-parturition based on interactions between a female's condition and the probability of reproductive success by the offspring (e.g., Cameron, 2004; Trivers & Willard, 1973; Veller, Haig, & Nowak, 2016). In addition, in other species, there can be a causal link between maternal corticosteroids and offspring sex ratio (e.g., Bonier, Martin, & Wingfield, 2007). Perhaps coincidentally, over time the sex ratio of Andean bear cubs born in North America has shifted from an even sex ratio to being male-biased (Faust & Thompson, 2000; Garshelis, 2004). To assess whether the dermatological symptoms might be associated with a skewed primary sex ratio, we examined the sex ratio of cubs. We also evaluated individuals' lifespan, as this is another factor that could be influenced by the unidentified processes producing outwardly obvious symptoms of alopecia.

2 | MATERIALS/METHODS

To collect information on the occurrence of this syndrome, in 2008 we sent email queries to husbandry, animal management, and veterinary medicine staff at 50 zoos in North America (e.g., Connolly, 2007) requesting that staff complete an online questionnaire. We further investigated the initial responses with phone calls and emails,

obtaining complete husbandry data for bears at 13 facilities and complete veterinary information at 17 facilities. In addition, we sought information from field researchers on the occurrence of this syndrome in wild bears. From participant responses and studbook information (Connolly, 2007) we compiled data on the following husbandry variables: ID, sex, age, sire, dam, provenance, past history of mating and cub birth, reproductive status (e.g., fertile, contracepted, etc.), diet, whether the individual was housed alone or with conspecifics, and the identities of any cohabitant. We also compiled data on two indicators of behavior that might be related to stress: whether the bear displayed behavioral stereotypies such as pacing or headtossing, and the general compatibility of cohabitants ranked on a Likert scale from 1 (generally incompatible) to 5 (highly compatible). In addition, we collected veterinary information on the clinical symptoms, their age at first appearance, and the treatment history of affected individuals. When we realized the high prevalence of the occurrence among Andean bears in North American zoos, we pursued information on its occurrence among female Andean bears in South America, where we obtained information solely on affected individuals. We constructed candidate general linear models (GLM) with binomial responses and Firth's bias correction to predict the occurrence of symptoms using the rule of thumb of ≥10 data points for every predictor variable. Our candidate model set included 14 models that included ≤3 predictor variables, the intercept INT, and ≤1 interaction term: individual sex SEX, whether the individual was housed socially SOC, whether the individual expressed stereotypic behaviors STE, the interaction term SEX × SOC, the interaction term SEX × STE, and the interaction term SOC × STE. We used an information theoretic approach (Burnham & Anderson, 2002) to compare the candidate models using the Akaike information criteria corrected for small sample sizes (AIC_c) as the criterion for model selection (Burnham & Anderson, 2002), with $\Delta AIC_c \leq 2$ as the threshold for a model to be competitive. We used χ^2 and p to assess the effectiveness of the "best" model for describing the occurrence of the syndrome and we used full-model averaging to estimate parameters and parameter weights (Symonds & Moussalli, 2011).

To assess whether the syndrome might be associated with subtle but important impacts on reproductive investment and lifespan, we used a two-tailed t test to compare the proportion of males among known-sex cubs born to females with and without obvious symptoms. Small sample sizes precluded conducting age-specific or cohort analyses, so we compared the lifespan of individuals with and without the alopecia syndrome.

Unless noted otherwise, all quantities are expressed as $\bar{x} \pm$ SE and statistical significance refers to two-tailed $p = .05$. Statistical analyses were conducted in JMP 13.2 (SAS Institute Inc., Cary, NC).

3 | RESULTS

We obtained information on husbandry conditions and the occurrence of the syndrome for 63 bears (26M:37F): 26 males and 24 females housed at 24 institutions in North America and 13 females at seven institutions in South America (Table 1). We were able to obtain additional information on some cases from Figueroa and Stucchi (2005). We had information on both affected and unaffected individuals from the North American population. Seven of 24 females had the syndrome (29%), but none of the 26 males were affected.

TABLE 1 Occurrence of Andean bear alopecia syndrome among bears in ex situ conditions in the Americas

	North America (24 institutions)		South America (7 institutions)		
	Affected	Unaffected	Affected	Unaffected	Total
Male	0	26	-	-	26
Female	7	17	13	-	37
Total	7	43	13	-	63

All 24 females in North America for which we have information were captive born. Affected females in South America were captive ($n = 4$) and wild born ($n = 9$). We were able to collect data, or estimate, the age of onset of symptoms for only a subset of females ($n = 11$, $\bar{x} = 12.99 \pm 1.94$ years, range 3–24.5). The lifespan of wild Andean bears is unknown, but we know the lifespan of 18 females in our data set ($\bar{x} = 25.96 \pm 5.45$ years, range 16.38–37.87). Thus, symptoms became first apparent across much of these bears' lifespans. Only 2 females developed symptoms when < 6 years of age; the other affected individuals were > 8.5 years of age.

Stereotypical behaviors were common among both males and females (13 of 26 males, 14 of 27 females), however we were not able to obtain information on stereotypical behavior for 10 females. Compatibility of males with their cohabitants and females with their cohabitants was generally rated as good ($n = 22$, $\bar{x} = 3.73 \pm 0.21$, range 2–5; $n = 20$, $\bar{x} = 3.5 \pm 0.26$, range 1–5, respectively, where compatibility of 3 = some aggression and 4 = generally compatible). Only 3 (of 26) males and 4 (of 22) females were rated as being generally incompatible or having moderate aggression with their cohabitant(s). We lacked information on the compatibility of 15 females with their cohabitant(s), so we excluded this as a potential predictor variable. Unfortunately, we were also unable to quantitatively evaluate relatedness as an indicator of whether there might be a genetic component to this syndrome. Only 11 of the affected females in our data set had any female kin in our data set (Table 2): 4 females had either a sister or female cub that was not affected, 2 had sisters that were not affected but female cubs that were affected, 4 had a sister or a female cub that was also affected, and one affected female cub of an affected female also had an affected female cub.

TABLE 2 Occurrence of Andean bear alopecia syndrome among close female kin (i.e., sister or female cub) of the 11 affected females for which data were available

	Affected	Unaffected
Sister	2	3
Female cub	2	3
Female cub of female cub	1	0

TABLE 3 Estimates (95% CI), full model averaged estimates (95% CI), and Akaike weights within the 95% confidence set of general linear models describing the occurrence of Andean bear alopecia syndrome among 52 captive Andean bears (26M:26F) in the Americas, ranked by ΔAICc

Model	INT	SEX	SOC	STE	SEX × SOC	SEX × STE	SOC × STE[a]	df	χ^2	p	AIC_c	ΔAIC_c	w_i	Σw_i	ER
1	2.29	−1.68						1	13.41	<.001	38.76	0	0.41	0.41	
	(1.21, 4.73)	(−4.12, −0.59)													
2	2.68	−1.64	0.54					2	1.36	<.001	40.07	1.31	0.21	0.62	1.92
	(1.11, 5.57)	(−4.08, −0.55)	(−0.78, 3.03)												
3	2.26	−1.68		0.27				2	13.82	.001	40.61	1.85	0.16	0.79	2.52
	(1.18, 4.70)	(−4.12, −0.59)		(−0.51, 1.07)											
4	2.65	−1.62	0.56	0.28				3	14.78	.002	41.99	3.23	0.08	0.87	5.03
	(1.10, 5.55)	(−4.06, −0.54)	(−0.77, 3.05)	(−0.50, 1.09)											
5	2.03	−0.98	−0.12		0.68			3	13.63	.004	43.15	4.39	0.05	0.91	8.96
	(0.95, 3.70)	(−0.26, 0.47)	(−1.58, 1.45)		(−0.77, 2.25)										
6	1.94	−1.36		0.13		0.13		3	12.97	.005	43.81	5.05	0.03	0.95	12.48
	(1.06, 3.42)	(−0.28, −0.47)		(−1.22, 1.49)		(−1.22, 1.49)									
$\bar{\beta}$	2.37	−1.6	0.17	0.08	0.04	0.01	−0.00								
	(0.08, 4.66)	(−3.53, 0.33)	(−1.36, 1.69)	(−0.63, 0.79)	(−0.58, 0.66)	(−0.27, 0.28)	(−0.27, 0.27)								
$\bar{w}$	1	0.99	0.39	0.33	0.06	0.05	0.02								

Abbreviations: AIC, Akaike information criteria; CI, confidence interval; ER, evidence ratio; INT, intercept; SOC, social behavior; STE, stereotypic behavior.

[a]The interaction term SOC × STE did not appear in the 95% confidence set of models.

We had complete data on all potential predictor variables for 52 bears (26 M, 26 F). The model which best fit these data predicted the occurrence of symptoms from only SEX ($n = 52$, $\chi^2 = 13.41$, df = 1, $p < .001$) but there were two other competitive models (i.e., $\Delta AIC_c \leq 2$): the model that included both SEX and SOC ($\chi^2 = 1.36$, df = 2, $p < .001$) and the model that included both SEX and STE ($\chi^2 = 13.82$, df = 2, $p = .001$; Table 3). The magnitude of the estimated parameters suggested that, among the variables measured, SEX may have the greatest impact on the likelihood of an individual's being affected by the syndrome, followed in possible influence by SOC, then by STE: $\bar{\beta}_{SEX} = -1.6$ (95% CI = −3.53–0.33), $\bar{\beta}_{SOC} = 0.17$ (95% CI = −1.36–1.69), $\bar{\beta}_{STE} = 0.08$ (95% CI = −0.63–0.79). The estimated parameter weights also suggest that SEX is the most likely predictor of Andean bear alopecia symptom, followed by SOC and STE: $\bar{w}_{SEX} = 0.99$, $\bar{w}_{SOC} = 0.39$, $\bar{w}_{STE} = 0.33$. However, we cannot clearly differentiate among the three competitive models based on the relative parameter estimates, the relative parameter weights, and the evidence ratios (ER; Table 3).

We have very limited data on the sex ratio of cubs of dams who gave birth both before and after developing symptoms, possibly due to the ages at which females developed symptoms. One female gave birth to three males and two females before developing any symptoms and later gave birth to two males and one female after beginning to develop mild symptoms; another female gave birth to 1 female before developing symptoms and then gave birth to three males and three females after beginning to develop mild symptoms. Neither female gave birth after developing serious chronic symptoms. Thus, we were unable to carry out an individual-based analysis of the effect of symptoms on cub sex ratio. There were 46 litters of known-sex cubs for which we knew whether mother had the syndrome at time of birth. The proportion of males among known-sex cubs was no different among litters born to females without symptoms at parturition ($n = 40$ litters, 0.63±0.08) than to litters born to females with symptoms ($n = 6$ litters, 0.5±0.22; $t = -0.528$, df = 1, $p = .616$).

Although 18 females for which we have information were still alive at the end of data collection, we do know the lifespan of seven females who never developed symptoms (27.12±2.54 years, range 16.38–37.87) and 12 females who developed symptoms at some point in their life (25.28±1.37 years, range 19.47–35.91). There is no significant difference in lifespan between females depending on whether they ever exhibited symptoms (log-rank $\chi^2 = 1.83$, df = 1, $p = .16$).

4 | DISCUSSION

By including information previously reported from Europe (Kolter et al., 2014; Rodriguez Barbon et al., 2017) with our data, we estimate the approximate Northern Hemisphere prevalence of the Andean bear alopecia syndrome to be 2.7% among males and 36.4% among females. This syndrome is, therefore, a relatively common and serious health issue for female Andean bears in human care. Given that the syndrome occurred in both wild born and captive born females, and thus among females that began developing under very different social and ecological conditions, we suggest that the syndrome does not reflect conditions in early development. Although some affected females were close relatives, we lack the balanced data to assess whether this may result from a genetic predisposition or from shared or similar husbandry conditions.

Our statistical analysis does not clearly differentiate among the three competitive models. However, even though not all socially housed females were affected by the syndrome, the only characteristics shared by all affected bears is that they were all females, socially housed with conspecifics. We therefore hypothesize that the alopecia syndrome in Andean bears may be only the most obvious symptom of endocrine abnormalities or autoimmune responses caused by the long-term social housing of bears that would not otherwise live in groups. As best as we know, adult Andean bears socialize only for mating (Appleton et al., 2018). Other bears are also solitary, but they nevertheless may congregate for short periods of time at natural or artificial food sources or other resources. To date, this has been observed in American black bears (Herrero, 1983; Rogers, 1987), brown bears (e.g., Clapham & Kitchin, 2016; Gill & Helfield, 2012; Peirce & Van Daele, 2006; Penteriani et al., 2017), and polar bears (Herreman & Peacock, 2013). Although it thus seems probable that wild Andean bears may congregate under certain conditions, wild bears may avoid the close proximity of others if they choose. Bears in human care cannot, and thus the inability for a female to move away from a larger individual, with potentially greater combative ability, may be a source of chronic stress. Zoo professionals have suggested for decades that pregnant female Andean bears or females with young cubs should not be housed with even a familiar male, because this is likely to disrupt maternal behavior or induce aggression in the female towards the male (Aquilina, 1981; Bloxam, 1977; Kühme, 1991; McDonald, 1989; Peel, Price, & Karsten, 1979; Restrepo Valencia, 1989; Rosenthal, 1989; Weinhardt, 1989). Thus, the response of an individual female towards a male has long been suggested as a guide for whether to house a female with a male for a period of potential mating activity (e.g., Moseley & Carroll, 1992; Restrepo Valencia, 1984). Although a female may not exhibit an obvious negative behavioral response to a male at other times, stressed females may experience subtle physiological responses. Until clearer information is available on the physiological changes that result in this syndrome and the physiological changes that result from social housing, regardless of whether effective pharmacological treatments are discovered, we suggest that adult Andean bear females not be routinely housed with other bears. Rather, we suggest that husbandry conditions more closely mimic the social conditions to which Andean bears may be adapted. In other words, give females the option of whether to live socially.

If the alopecia syndrome occurs in some female Andean bears constrained to live in undesired cohabitation, it is puzzling that individuals of other bear species with similar nonsocial

lifestyles can seemingly be housed socially (e.g., polar bears, sun bears) without obvious signs of stress or compromised health. However, even in these other species, the reproductive success of socially housed individuals may be compromised (Garshelis, 2004). In addition, perhaps those other bear species rely more on aggressive resolution of agonistic encounters than on dispersive resolution. Bears which fight when placed together would not be housed together in a modern zoo, so zoos may already be using the social preferences of individual bears of some bear species to determine their housing conditions. Perhaps Andean bears would more often resolve conflict with dispersion rather than aggression if they could, but in captivity these bears have nowhere else to go.

5 | CONCLUSIONS

1. Because all affected individuals were females, regardless of provenance, with no observed cases in cubs, we believe this syndrome arises from an endocrine response among reproductively mature females to ex situ conditions.
2. Because all affected females were socially housed, we propose that the critical husbandry practice underlying this syndrome is the long-term cohabitation of females with conspecifics.
3. We were unable to evaluate whether heritability was associated with the syndrome and social compatibility did not vary enough to be informative. There was no clear association of the syndrome with skewed sex ratios of cubs, or with female lifespan.
4. Research on endocrine levels of affected females, unaffected females housed alone, and unaffected females housed socially, should be informative.
5. Regardless of the efficacy of pharmacological treatments, to avoid affecting additional individuals, we recommend that reproductively mature female Andean bears be housed with adult conspecifics only briefly unless the females choose cohabitation.

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