VOLUME 98 • NUMBER 12 • DECEMBER 2017

ECOLOGY

ECOLOGICAL SOCIETY OF AMERICA

Ecology, 98(12), 2017, pp. 3199–3210

Physiological stress responses to natural variation in predation risk: evidence from white sharks and seals

Neil Hammerschlag,[1,2,6] Michael Meÿer,[3] Simon Mduduzi Seakamela,[3] Steve Kirkman,[3] Chris Fallows,[4] and Scott Creel[5]

[1]*Department of Marine Ecosystems and Society, Rosenstiel School of Marine and Atmospheric Sciences, University of Miami, Miami, Florida 33149 USA*
[2]*Leonard and Jayne Abess Center for Ecosystem Science and Policy, University of Miami, Coral Gables, Florida 33146 USA*
[3]*Branch: Oceans and Coasts, Department of Environmental Affairs, Private Bag X4390, Cape Town 8000 South Africa*
[4]*Apex Shark Expeditions, Shop 3 Quayside Center, Simonstown Cape Town 7975 South Africa*
[5]*Department of Ecology, Montana State University, Bozeman, Montana 59717 USA*

Abstract. Predators can impact ecosystems through consumptive or risk effects on prey. Physiologically, risk effects can be mediated by energetic mechanisms or stress responses. The predation-stress hypothesis predicts that risk induces stress in prey, which can affect survival and reproduction. However, empirical support for this hypothesis is both mixed and limited, and the conditions that cause predation risk to induce stress responses in some cases, but not others, remain unclear. Unusually clear-cut variation in exposure of Cape fur seals (*Arctocephalus pusillus pusillus*) to predation risk from white sharks (*Carcharodon carcharias*) in the waters of Southwestern Africa provides an opportunity to test the predation-stress hypothesis in the wild. Here, we measured fecal glucocorticoid concentrations (fGCM) from Cape fur seals at six discrete islands colonies exposed to spatiotemporal variation in predation risk from white sharks over a period of three years. We found highly elevated fGCM concentrations in seals at colonies exposed to high levels of unpredictable and relatively uncontrollable risk of shark attack, but not at colonies where seals were either not exposed to shark predation or could proactively mitigate their risk through antipredatory behavior. Differences in measured fGCM levels were consistent with patterns of risk at the site and seasonal level, for both seal adults and juveniles. Seal fGCM levels were not correlated with colony population size, density, and geographic location. Investigation at a high risk site (False Bay) also revealed strong correlations between fGCM levels and temporal variation in shark attack rates, but not with shark relative abundance. Our results suggest that predation risk will induce a stress response when risk cannot be predicted and/or proactively mitigated by behavioral responses.

Key words: *antipredator behavior; apex predator; ecology of fear; ecophysiology; glucocorticoid; nonconsumptive effects; predation risk; risk effect; seal; shark; stress.*

Introduction

Apex predators can strongly alter ecosystems by affecting the numbers, distribution and behavior of their prey (Werner and Peacor 2003, Peckarsky et al. 2008). Moreover, such behavioral modifications in response to predation risk can manifest in physiological changes in prey that carry fitness costs and affect population dynamics (Peckarsky et al. 2010, Zanette et al. 2014). Together, these predator effects can have cascading impacts on other species (Schmitz et al. 1997, Brashares et al. 2010).

Two general hypotheses have been proposed for the physiological mechanism underlying predation risk effects on prey populations (outlined in Creel et al. 2009). The predator-sensitive food hypothesis predicts that predators constrain foraging activity or efficiency of their prey, thus increasing energetic or nutritional constraints on prey reproduction or survival (Sinclair and Arcese 1995). Many studies across a broad range of taxa have found empirical evidence in support of this hypothesis (Werner and Peacor 2003). Growing research has revealed that food-mediated reactions of prey to predators (e.g., food–risk trade-offs) are not simply an artifact of predator encounter rates and often depend on predator hunting mode, anti-predator behavior, and on landscape features that can influence the probability of death given an encounter with a predator (Schmitz 2008, Heithaus et al. 2009). The predation-stress hypothesis predicts that exposure to risk causes increased secretion of glucocorticoids (or other physiological stress responses; Romero 2004). However, studies of this hypothesis in natural settings have been limited and the results mixed (Boonstra et al. 1998, Clinchy et al. 2004, Creel et al. 2009, Sheriff et al. 2009). Moreover, most investigations of this hypothesis in the wild have generally employed study designs for monitoring of potential stress changes through time at a single site (Boonstra et al. 1998, Zanette et al. 2014); but few natural study systems have been conducive to investigating the effects of risk on

Manuscript received 28 September 2017; accepted 6 October 2017. Corresponding Editor: John F. Bruno.
[6] E-mail: nhammerschlag@miami.edu

stress responses in prey populations with replication within both a low risk and high risk condition. Additionally, many studies assume that risk effects are mediated by fear of predation, which is manifested in stress; however, anti-predatory behavioral responses to risk can arise through mechanisms that do not involve a stress response. For example, in the greater Yellowstone ecosystem, elk (*Cervus elaphus*) forage in suboptimal habitats to reduce their exposure to predation from wolves (*Canis lupus*; Creel et al. 2005), which incurs physiological costs associated with lower nutritional intake, but does not result in a stress response (Creel et al. 2009). Taken together, a key question remains: under what conditions are risk effects mediated by stress in the wild? This knowledge gap is largely driven by the inherent challenges of working with predators and their prey in nature. Such studies are particularly important at this time given declines in predator populations worldwide (Estes et al. 2011), together with successful predator recovery programs in some ecosystems (Marshall et al. 2016).

The waters off the Western Cape of South Africa provides an opportunity to study predator–prey interactions involving white sharks *Carcharodon carcharias* and Cape fur seals *Arctocephalus pusillus pusillus*, and an ideal system to test the effects of predation risk on prey physiology (Fig. 1A). Here, the population of Cape fur seals in the region is segregated into discrete colonies that inhabit different inshore islands; but the seals from the different island colonies move offshore to feed where they share the same general foraging grounds (Rand 1959, 1967, Oosthuizen 1991). White sharks only actively target certain seal colonies, creating a natural spatial variation in seal colonies to threat of attack (Bonfil et al. 2005, Dudley 2012, De Vos et al. 2015*a*, Andreotti et al. 2016). At these locations, there also exists clear seasonal variation in predation risk, as sharks only actively hunt Cape fur

FIG. 1. (A) Photograph of a white shark lunging toward a Cape fur seal at the edge of Seal Island (background) in False Bay, South Africa. White sharks aggregate during the cool season at specific island colonies to hunt seals when they enter and exit the water to and from offshore foraging. (B) A white shark patrols the border of a kelp bed that surrounds the seal colony at Geyser Rock. The kelp is used by seals as refuge from sharks while traversing to and from the island. (C) Kelp and other high relief landscape features are absent from the waters around the island colony in False Bay, where attack rates on seals are 18 times higher than at Geyser Rock. (D) A large seal bearing a fresh injury from a white shark attack hauls out on to Seal Island in False Bay where it is investigated by a group of pups and juvenile seals. All images by C. Fallows. [Color figure can be viewed at wileyonlinelibrary.com]

seals during winter months (Hammerschlag et al. 2006, Jewell et al. 2013, Towner et al. 2016). However, among the different colonies targeted by sharks, predation risk to seals can vary significantly due to landscape features that either offer protection to seals traversing the gauntlet or leave them exposed to unpredictable risk of attack (Wcisel et al. 2015). For example, white shark attack rates on seals at the seal colony in False Bay (1.97 attacks/h) are 18 times higher than at Geyser Rock (0.1 attacks/h), because the latter is surrounded by dense kelp beds and reefs that serve as a refuge from sharks when the seals traverse from their colony (Wcisel et al. 2015; Fig. 1B). Such landscape features are absent from False Bay (Fig. 1C), where predation rates on seals average 6.7 attacks per day during the cool season, with success rates as high as 0.55 kills per attack (Hammerschlag et al. 2006). Rarely do spatially discrete natural prey populations share the same general food supply, while being exposed to such distinct variation in predation risk as in the current study system.

The purpose of this study was to test the predation-stress hypothesis using this shark-seal system off South Africa. First, we analyzed movement patterns of satellite tagged white sharks to evaluate spatial and temporal patterns of white shark residency and density at six seal colonies that vary in risk from shark attack. Next, we collected seal scat samples from these six seal colonies over multiple years and seasons for measurements of fecal glucocorticoid metabolites (fGCM) to evaluate if concentrations were associated with patterns of spatial and temporal occurrence of white sharks. Finally, we conducted a multi-year study at Seal Island in False Bay (a high risk site), to test if seal fecal cortisol levels were correlated with either relative abundance of white sharks or measured attack rates by sharks on seals. We used these data to (1) evaluate if seal colonies exhibit spatial and seasonal variation in fGCM concentrations consistent with patterns of white shark predation risk, (2) test if fGCM levels in seals correlate with shark relative abundance or measured surface attack rates by sharks on seals in False Bay, (3) test if differences in colony-specific landscape features that influence the type and magnitude of shark predation risk to seals are associated with differences in seal fGCM concentrations, and (4) determine whether fecal cortisol levels differ in in adult seals vs. juveniles under risk of predation. Additionally, we tested the alternative hypotheses that fGCM concentrations were associated with seal colony population size, density and geographic location (and associated variation in environmental factors).

Materials and Methods

Study system

Cape fur seals exhibit high site fidelity to specific inshore island colonies, which therefore remain fairly discrete units (Rand 1959); however, seals from different colonies share the same general offshore feeding grounds as revealed through mark–recapture (Oosthuizen 1991). Previous satellite and acoustic tracking of white sharks within South Africa have demonstrated that sharks aggregate at certain seal colonies during cool months of the year to target Cape fur seals. These "high shark abundance" seal colonies include False Bay, Mossel Bay, and Geyser Rock, where shark habitat use and predation has been relatively well studied (Martin et al. 2005, Johnson et al. 2009, Fallows et al. 2012, Jewell et al. 2013, Kock et al. 2013, Towner et al. 2016, Wcisel et al. 2015). During warm months, white sharks shift the focus of their hunting away from seals at the colonies, presumably to feed on teleosts and elasmobranchs, resulting in lower shark occurrence and predation pressure to seals at these high shark abundance colonies during the summer (Hammerschlag et al. 2006, Kock et al. 2013, De Vos et al. 2015*a*). There are also "low shark abundance" seal colonies within the region that are not targeted by sharks at any time of year (Bonfil et al. 2005, Kock et al. 2013, De Vos et al. 2015*a*). These spatiotemporal patterns of white shark abundance have also been found through region-wide survey data of white sharks through standardized boat-based baited surveys as part of a project to estimate population sizes of white sharks in South Africa (Andreotti 2015, Andreotti et al. 2016).

In this study, we focused our investigation on six seal colonies that varied in exposure to the seasonal presence of hunting white sharks: three high shark abundance colonies (False Bay, Mossel Bay, and Geyser Rock) and three low shark abundance colonies (Bird Island in Lambert's Bay, Jutten Island, and Robbesteen; Fig. 2) during both the "high predation season" (winter, June–September) and "low predation season" (summer, October–May). However, to confirm this previously described spatial and temporal variation in exposure of the six focal seal colonies to white sharks during the study period, we analyzed white shark movement data that were collected as part of a larger collaborative satellite tagging project between the USA-based non-profit OCEARCH and South African researchers.

White shark tracking

Between March and May of 2012, a total of 37 white sharks (14 males, 23 females) were captured and tagged at five different localities across the Western Cape of South Africa: Algoa Bay, False Bay, Gansbaai, Mossel Bay, and Struisbaai. Details on capture and handling methods can be found in Wcisel et al. (2015). Briefly, sharks were captured with baited barbless hooks and carefully lead onto a hydraulic platform. One or two hoses where then inserted into the shark's mouth to pump fresh oxygenated saltwater over the gills. Sharks received antibiotics and electrolyte injections to enhance recovery time. Smart Position-only and Temperature Transmitting tags (SPOT5 tags; Wildlife Computers, Redmond, Washington, USA) were affixed to the dorsal fin of sharks. Depending on their size, sharks were tagged with either a small SPOT tag (2-yr battery life for

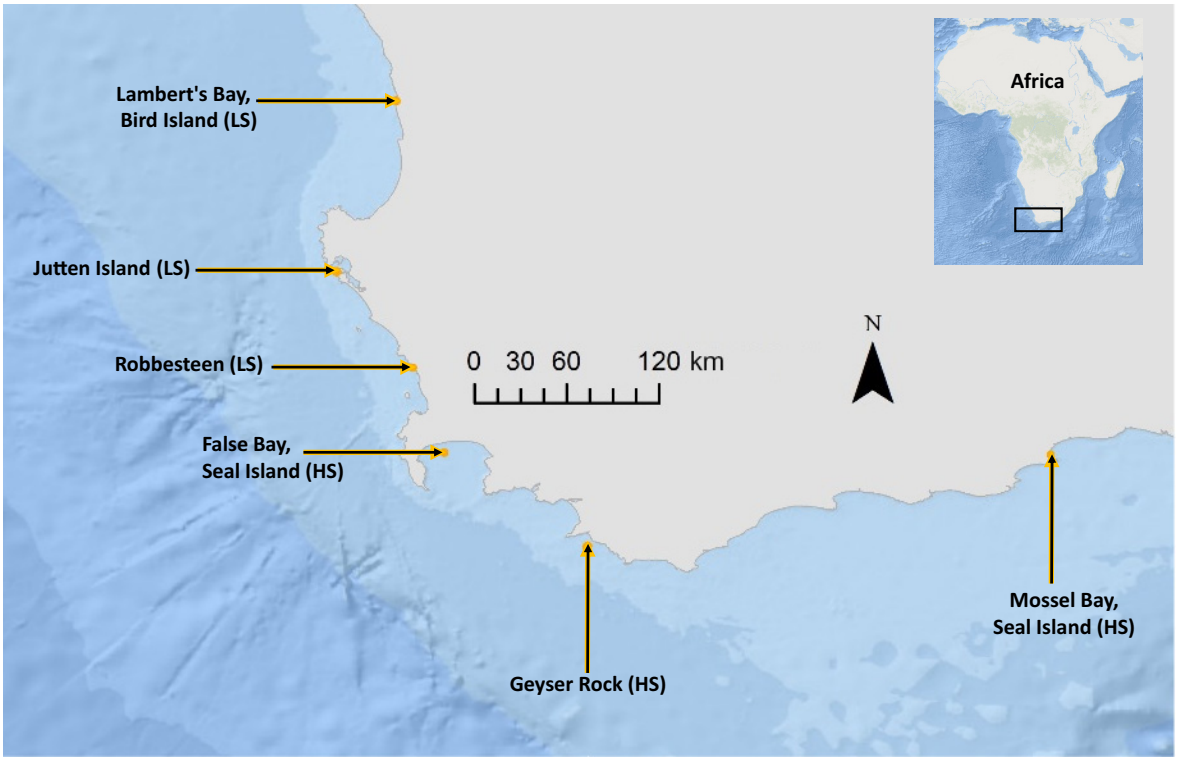

FIG. 2. Locations of six focal colonies off the Western Cape of South Africa (inset). LS, low shark abundance colony; HS, high shark abundance colony. [Color figure can be viewed at wileyonlinelibrary.com]

sharks <3 m total length) or a large SPOT tag (5-yr battery life for sharks >3 m total length).

Locations were acquired whenever the dorsal-fin mounted SPOT tag broke the water surface and transmitted a signal to a passing ARGOS satellite. Geographic locations of SPOT tagged sharks were determined by Doppler-shift calculations made by the ARGOS Data Collection and Location Service (Argos CLS, Toulouse, France). Accuracy of position data is variable depending on number of transmissions received by ARGOS and categorized into location classes (LC) as follows: LC 3 < 250 m, 250 m < LC 2 < 500 m, 500 m < LC 1 < 1,500 m. The median error for LC 0, A, and B ranges from 1 to 3 km. Class Z indicates that the location process failed and estimates of position are highly inaccurate and are removed before spatial analysis.

Seal fecal sample collection

Between 2012 and 2015, a total of 502 fecal samples were collected and analyzed for fGCM, with at least two collection periods in the high predation season and two collection periods in the low predation season for each colony (N = 92 samples from Geyser Rock, N = 44 from Robbesteen, N = 52 from Jutten Island, N = 145 from Lambert's Bay, N = 114 from Seal Island in False Bay, N = 55 from Seal Island in Mossel Bay). At each sampling occasion, ~20 g samples from clearly distinct defecations by single seals were collected and placed in 50-mL screw-lid vials and frozen within 1–2 h of collection. Where possible, fecal samples could be assigned to seal age classes (adult vs. juvenile) based on differences in size (though no effect of age class was detected: see *Results*). The juvenile category also included young-of-the year seals. Based on direct observation, juvenile scats ranged in size from 6 to 10 mm diameter; whereas adult scats ranged in size from 15 to 20 mm diameter. Adult scats could also be further distinguished based on a relatively higher frequency of fish bones in the scat, which are fewer in juveniles. Ambiguous samples and scats between 11 and 15 mm were not assigned to an age class.

Immunoassay

Steroid hormone metabolites were extracted from fecal samples by drying the scat and boiling a known mass of dry feces in ethanol using methods that have been described in detail previously (Monfort et al. 1997, Creel et al. 2009).

We measured glucocorticoid metabolite concentrations in fecal extracts (fGCM) using an enzyme-linked immunoassay with a cortisol antibody (Enzo Life Sciences ADI-900-071, Farmingdale, New York, USA)) that has broad cross-reactivity and has been procedurally and biologically validated for assay of fecal extracts in a broad range of species (Monfort et al. 1997, Creel et al.

2009, 2013). We expressed fGCM concentrations as milligrams of cortisol immunoreactivity per gram of dry feces. Antibody binding was parallel for a dilution series of cortisol standards and seal fecal extracts diluted from 1:1 to 1:256 (log-linear slopes −0.011 and −0.014, respectively). Quantitative recovery of cortisol added to seal fecal samples was highly accurate ($r^2 = 0.997$, $b = 1.00 \pm 0.03$ [mean ± SE]) for a range from 156 to 5,000 pg of cortisol added to fecal extracts at working concentration. Assay sensitivity was several orders of magnitude below the concentration of fecal extracts. Based on preliminary analysis, we assayed extracts at 1:100 dilution to maximize sensitivity, assaying all samples in duplicate with a seven-standard curve, controls and measures of total activity, zero-steroid binding, and non-specific binding on each plate. Intra- and inter-assay coefficients of variation from pooled fecal extracts were 13.36 and 13.71, respectively. The composition of scats did not vary between sites or seasons, and we detected no association between fGCM concentrations and the composition of scats, as measured by the proportions of water and indigestible matter.

Seal population data

Aerial photographs of seal colonies from fixed wing and helicopter aircrafts were taken at the peak of the seal pupping season and the numbers of newborn pups of the year were counted on the photographs. This is because pups are confined to land (during their first month of life) and their numbers can be used to infer total population size of adults and pups, subject to certain assumptions (Kirkman et al. 2011). Because Jutten Island is primarily a non-breeding colony where relatively few pups are currently born, both pups and adult seals were counted.

Aerial seal counts were conducted annually at all six seal colonies between 2011 and 2014, excluding 2012 at Mossel Bay and Lambert's Bay and 2013 at Robbesteen, Geyser Rock, False Bay, and Mossel Bay. Annual seal counts were then averaged across the study period (hereafter referred to as colony population size). We also calculated an estimate of seal density (seals/ha) at each colony by dividing the number of seals counted by the area of the island occupied by the seals (hereafter referred to as colony density).

Shark predation and relative shark abundance in False Bay

Between February 2014 and August 2015, standardized surveys were conducted at Seal Island in False Bay to evaluate for the potential differential effects of shark occurrence vs. hunting behavior (i.e., components of predation risk) on seal fGCM concentrations.

Predation by white sharks on Cape fur seals was recorded daily around the seal island colony in False Bay for one week prior to fecal sample collection, which occured on seven dates: 11 February, 25 April, 30 July, and 12 September 2014 as well as on 19 March, 7 July, and 12 August 2015. Previous laboratory studies that have subjected sea lions (*Eumetopias jubatus*) to an adrenocorticotropic hormone (ACTH) challenge found a lag of up to 4 d between ACTH injection and peak fGCM (Hunt et al. 2004). Accordingly, for one week prior to collecting fecal samples on the dates specified above, standardized observations for shark predation events were performed from a research vessel daily between 07:00 and 09:30, sea conditions permitting. Monitoring of shark predations on seals were conducted following the approach of Martin et al. (2005), and Fallows et al. (2016). Here, sharks primarily attack Cape fur seals at the surface via a vertical breach when seals are surface porpoising to and from the Island. Attacks are concentrated on the southern side of the Island, close to shore (within 2 km). By positioning at the south end of Seal Island where the majority of predatory activity occurs, we were able to survey the waters surrounding the island for predations up to a distance of at least 3 km, although the majority of predations occur within 400 m of southwest end of the island (Martin et al. 2009).

After about 09:30 , our research vessel anchored on the southern side of Seal Island and conducted standardized boat-based baited surveys of white sharks using an approach modified from Hammerschlag and Fallows (2005). Between 10:00 and 12:00, sea conditions permitting, sharks were attracted to the boat using a large tuna head and seal decoy. Individual white shark can be identified based on a combination of visual makers, including unique scarring, presence/absence of claspers, and individual variation in pigmentation patters on the gill flaps, pelvic fins, and caudal fins (Domeier and Nasby-Lucas 2007). The duration of baited surveys were recorded, along with the number of different individual white sharks observed during this period. Using these data, we calculated the number of different white sharks observed per hour of baited survey as a metric of relative shark abundance.

White shark tracking analysis

SPOT satellite tagging data were downloaded from ARGOS CLS. Given variable tag size (and thus battery life) and possible issues related to biofouling with increasing tag age, we included only positions within a year of tagging for each individual shark. Given irregular transmissions and varying levels of position accuracy (see Hammerschlag et al. 2011 for details), all SPOT location data were interpolated and regularized to a constant 6-h interval using a hierarchical, first-difference, correlated, random-walk, switching (hDCRWS), state-space model (SSM) described by Jonsen (2016). This approach accounts for both the variability in geoposition accuracy provided through ARGOS location classes and the irregularity of surfacing. Locations were not

interpolated for data gaps >14 d and for shark tracks with fewer than 20 positions (per instructions by I. Jonsen). The SSM model also estimates a behavioral state (bt) for each position ranging from one (transient, migratory state) to two (resident, area-restricted state; Jonsen 2016). Following Acuña-Marrero et al. (2017), each position was classified as being either predominantly transient (bt < 1.25) or predominantly resident (bt > 1.75). Given we were interested in predation risk to seals from hunting sharks residing at the colonies, subsequent analysis were restricted to 6-h interpolated positions classified as predominately resident (bt > 1.75) and plotted in ArcMap 10.1 (ESRISA).

To examine relative differences in the spatial distribution of white sharks in relation to the focal seal colonies, we applied kernel density analysis to the position data in ArcMap 10.1. This calculated and plotted the number of shark positions per km^2 using a kernel function (Silverman 1986). We then used ArcMap spatial analysis tools to calculate the average kernel densities (KD) of white shark positions within a 2 km radius of the high vs. low shark abundance colonies. This analysis was conducted separately for the warm (October–May) and the cool season (June–September). To evaluate patterns of shark residency at the colonies, we calculated the mean, minimum, and maximum number of days individual white sharks spent within 2 km of the high vs. low shark abundance colonies within each season. We selected a 2 km radius given the spatial accuracy of the SPOT data and also because white sharks patrolling the colonies concentrate hunting effort within 2 km of shore (Johnson et al. 2009, Fallows et al. 2012, Jewell et al. 2013, Towner et al. 2016).

Statistical analysis

In addition to colony-specific and seasonal variation in predation risk to seals, differences in colony population size and/or density could contribute to seal stress levels. Moreover, given that colonies were widely distributed along the coast, differences in their geographic locations (i.e., latitude and longitude) could potentially expose the colonies to differences in environmental factors that could influence seal stress levels. Accordingly, generalized linear models (GLiMs) were employed to test the effects of these explanatory variables on measured fGCM concentrations, which included seal colony (Jutten Island, Lambert's Bay, Robbesteen, Geyser Rock, Mossel Bay, False Bay), season (warm, cool), the interaction between colony and season (colony × season), colony population size, density, latitude, and longitude. GLiMs were constructed with backward and forward stepwise selection, starting with a model containing all the plausible explanatory variables. Retention or removal of variables were based on the Akaike information criterion (AIC), with the lowest AIC suggesting the best fitting model. Based on results of the GLiMs, which revealed that the best explanatory model included only the interactive effects of colony × season, Tukey's standardized range (HSD) tests were used for pairwise evaluations among colonies across both seasons.

For the high shark abundance colonies (False Bay, Mossel Bay, Geyser Rock), analysis of variance (ANOVA) was used to test for potential differences in fGCM between seal age classes (adult vs. juveniles), between seasons, and the interaction of these effects. Tukey's HSD tests were then used for pairwise evaluations between age classes among colonies.

For predation data recorded in False Bay, hourly attack rates by white sharks on seals were averaged across the seven days prior to each fecal sampling. Pearson's correlation was used to test if weekly shark attack rates were correlated with associated fGCM concentrations. Similarly, for shark survey data, hourly relative abundance of white sharks were averaged across the seven days prior to each fecal sampling and Pearson's correlation was used to test if shark relative abundance was correlated with associated fGCM concentrations.

In addition to the GLiMs testing for effects of plausible explanatory variables on measured fGCM concentrations, we used Pearson correlations to separately test for significant relationships between colony population size and density on mean fecal cortisol concentrations. Similarly, we used Pearson correlations to separately test for potential significant relationships between latitude and longitude on seal fGCM values in both the warm and cool season.

All statistical analyses were conducted in SAS statistical software.

Results

Thirty-four tagged white sharks provided geoposition data (three tags failed to report) for spatial analyses of locations regularized to 6-h intervals using SSM. Sixteen individual sharks provided resident positions (bt > 1.75) within 2 km of a high shark abundance colony (Geyser Rock, Mossel Bay, and False Bay) in both the warm and cool season. In contrast, no sharks provided resident positions within 2 km of a low shark abundance colony (Jutten Island, Lambert's Bay, Robbesteen) in either season. In fact, no sharks tracked occurred within 2 km of these colonies; the closest a resident individual came within proximity of a low shark abundance colony was a single individual 44 km off Jutten Island. Within 2 km of the high shark abundance colonies, average density of shark positions were 2.5 times higher in the cool season (KD = 0.35 km^2) than warm season (KD = 0.14 km^2). In terms of residency, sharks were resident 1.5 times more days within 2 km of high shark abundance colonies during the cool season than during the warm season (Table 1). Moreover, the maximum number of days an individual shark was resident at a high shark abundance colony during the cool season was double that of the warm season (Table 1).

Fecal GCM concentrations showed considerable variation, ranging from a minimum of 26.66 mg/g to a

TABLE 1. Mean kernel density (KD) estimates of white shark interpolated position per km^2 and the mean, minimum (min.) and maximum (max.) number of days individual sharks were resident within 2 km of a low shark abundance colony (Jutten Island, Lambert's Bay, and Robbesteen) or a high shark abundance seal colony (Geyser Rock, Mossel Bay, and False Bay) in the warm (low predation) and cool (high predation) season within one year of tagging.

Colony	Season	Mean KD	Mean days	Min. days	Max. days
Low	warm	0	0	0	0
Low	cool	0	0	0	0
High	warm	0.14	7.1	1	22
High	cool	0.35	10.8	1	47

maximum of 3,372.11 mg/g. The best fitting GLiM included only the interactive effects of colony and season (Colony × Season) as an explanatory variable, which was highly significant ($P < 0.0001$; Appendix S1: Table S1). Pairwise comparisons using Tukey's HSD Tests revealed fGCM concentrations were significantly higher in samples from the high shark abundance colonies in False Bay and Mossel Bay (Fig. 3; Appendix S1: Table S2). While fGCM concentrations for these two colonies did not differ from one another in the high predation season, both were significantly higher than from collection made from those same colonies in the low predation season as well as significantly greater than those from any other colony in either season, including the high shark abundance colony at Geyser Rock (Fig. 3). Concentrations of fGCM measured from the island colonies in False Bay and Mossel Bay (1,737.8 ± 689.16 SD mg/g) were on average 2.8 times greater than the mean of all other colonies across both seasons (627.26 ± 523.88 mg/g), including Geyser Rock during the high predation season (620.01 ± 388.19 m/g).

For the high shark abundance colonies, there was a significant interaction between season, seal age class, and individual colony on measured fGCM values (ANOVA, N = 261, df = 7, $F = 10.26$, $P < 0.0001$; Appendix S1: Table S3). At these sites, fecal glucocorticoid metabolite concentrations did not differ between adults and juveniles in either the low or high predation season, but values for both adults and juveniles were significantly higher in the high vs. low predation season (Appendix S1: Table S4). The exception was for Geyser Rock, in which there were no differences in fGCM values between age classes during the high vs. low predation season (Appendix S1: Table S4).

At Seal Island in False Bay, white shark predation rates on seals were recorded on seven occasions during the week prior to fecal sampling. Mean weekly predation rates ranged from 0 attacks/h (4–10 February 2014) to 3.49 attacks/h (5–11 August 2015). Pearson correlation revealed a strong positive linear correlation between mean fGCM concentrations and mean predation rates (attacks/h) over the prior week ($r = 0.96$, $P = 0.0007$; Fig. 4A). In contrast, fGCM concentration were not correlated with shark relative abundance ($r = 0.2$, $P = 0.66$; Fig 4B).

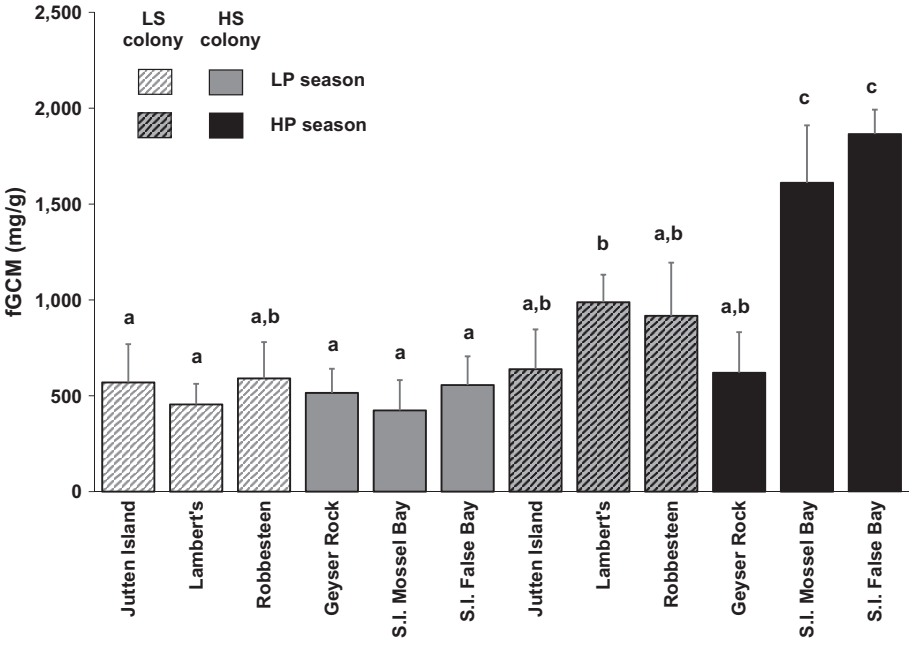

FIG. 3. A comparison of fecal glucocorticoid metabolite concentrations (fGCM) across all six seal colonies in both the low predation season and high predation season. Fecal samples were collected from each colony at least twice per season for at least two years between 2012 and 2015. High shark abundance colonies are solid bars and low shark abundance colonies are vertical patterned bars. Values are means and whiskers are 95% confidence intervals. Values with different letters denote statistical differences. HS, high shark abundance colony; LS, low shark abundance colony; HP, high predation season; LP, low predation season; S.I., Seal Island.

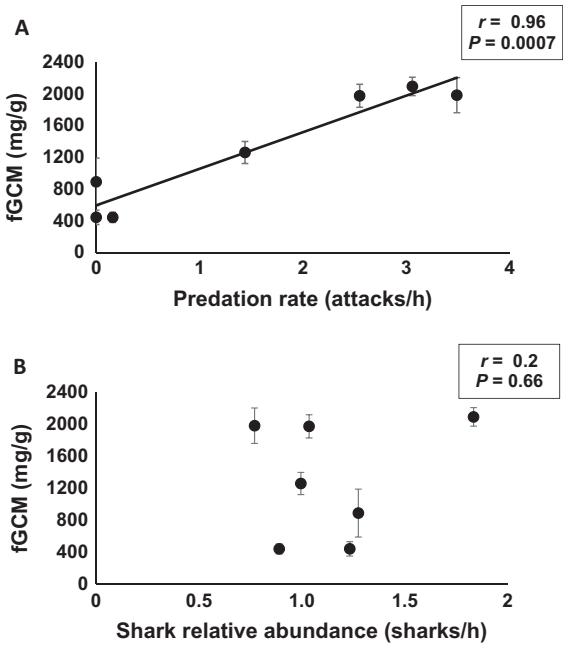

FIG. 4. Correlation between fGCM concentrations and (A) mean weekly predation rates (attacks/h) and (B) mean weekly relative shark abundance (sharks/h) at Seal Island in False Bay. Fecal sampling occurred on seven occasions between February 2014 and August 2015. Values are fGCM means ± SE.

Average counts of seals at each colony across the study period and associated density can be found in Appendix S1: Table S5. Model selection did not include these explanatory variables in the final GLiM, and seal colony population size and density was not correlated with mean fGCM concentrations at that colony (Fig. 5A,B). Moreover, seal fGCM concentrations were not correlated with either colony latitude or longitude in either the warm or cold season (Fig. 5C–F).

DISCUSSION

The predation-stress hypothesis predicts that risk from predators causes activation of physiological stress reactions in prey, which can directly or indirectly reduce reproduction and survival (Romero 2004, Clinchy et al. 2013). Observational and experimental studies have found mixed evidence for such increases in stress hormones of prey in response to predators or predation cues (Boonstra et al. 1998, Clinchy et al. 2004, Creel et al. 2009, Sheriff et al. 2009) and to date there have been too few studies of the predation-stress hypothesis in the wild to infer general relationships (Clinchy et al. 2013). In this study, we found that spatiotemporal patterns of stress in Cape fur seals, measured by fGCM concentrations, were consistent with predation risk from white sharks at the site and seasonal level. In contrast, fGCM levels were not detectably related to seal colony population size, density and geographic location. Moreover, our subsequent study in False Bay, revealed seal fGCM concentrations were strongly positively correlated with temporal variation in shark predation rates, but not with shark relative abundance (Fig. 4). These findings provide empirical support for the predation-stress hypothesis in the wild from a natural experiment involving large long-lived apex predators and their prey. It is worth considering here that seals may also incur costs consistent with the predator-sensitive food hypothesis, which could be assessed through the future research using energetic or nutritional indicators (Gallagher et al. 2017*a*, *b*). The two mechanisms are not mutually exclusive (Creel et al. 2009).

Below we separately discuss plausible alternative explanations for the patterns found; however, it is likely that some unmeasured variables (e.g., storms) are contributing at least partly to the variation in measured fecal cortisol levels, such as the seasonal difference in mean fGCM that were found for the colony in Lambert's Bay. However, the data gathered suggest that these factors are not the primary drivers of the nearly quadrupling in fGCM levels that occurred at the False Bay and Mossel Bay "high risk" seal colonies during the high predation season. In contrast, we believe the majority of this variation is attributable to predation stress. While our data is correlative, our inference is strengthened by the added results from the False Bay study where the magnitude in variations of fGCM levels were associated with recorded predation rates on seals, measured on multiple occasions across different seasons over the course of two years (Fig. 4A).

The occurrence of some white sharks at the high shark abundance colonies during the warm season, albeit lower than during the cool season (Table 1), may seem difficult to reconcile with the low fGCM stress levels found there, which were comparable to the low shark abundance colonies where no tracked sharks occurred. We suggest this is because during the warm season at the high shark abundance sites, white sharks are not actively hunting Cape fur seals (Hammerschlag et al. 2006, Wcisel et al. 2015). Corroborating these data is behavioral information previously gathered from Cape fur seals using acoustic telemetry in False Bay (De Vos et al. 2015*a*, *b*). For example, seals engaged in safer behaviors, such as swimming in larger groups and avoiding deeper water, during the high predation season and not during the low predation season (De Vos et al. 2015*a*, *b*).

Although exposed to hunting white sharks during the cool season, seals at Geyser Rock did not exhibit significant seasonal increases in fGCM levels in a manner similar to False Bay and Mossel Bay. This pattern can be explained by the previously described variation in seals' use of landscape features unique to the waters surrounding Geyser Rock. Specifically, these waters contain a mosaic of structurally complex reef and dense kelp beds that are used by seals as refugia from white sharks and appear to effectively reduce attack rates on seals (Wcisel et al. 2015). So while encounter rates between white sharks and seals are high during the cool season at Geyser Rock, the seals can behaviorally mitigate their exposure to risk by using the reef and kelp as a relatively safe

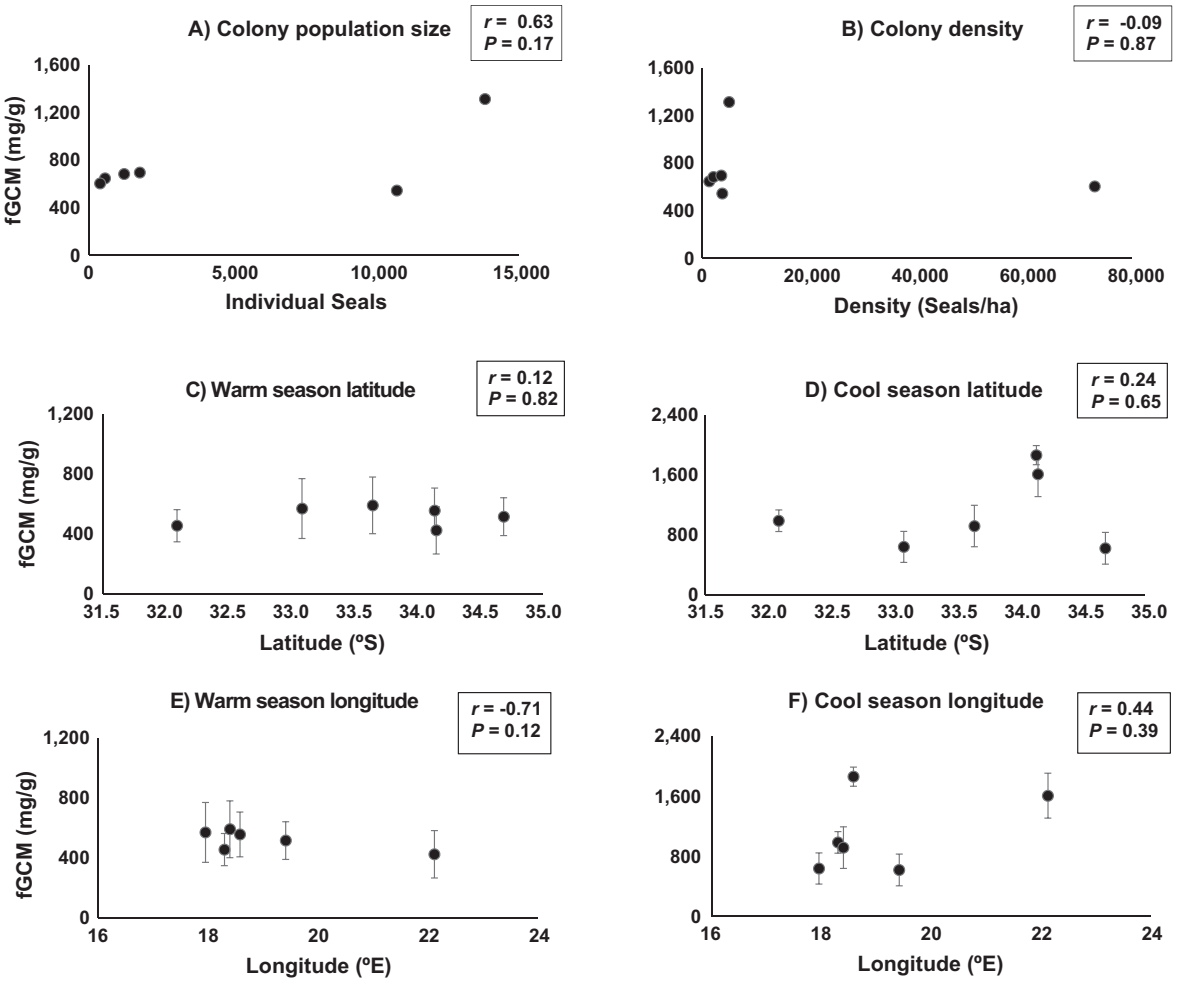

FIG. 5. Plots showing lack of correlation between fGCM concentrations and seal colony (A) population size, (B) density, (C) latitude, with data in the warm season, (D) latitude, with data in the cool season, (E) longitude, with data in the warm season, and (F) longitude, with data in the cool season. Values are fGCM means ± SE.

underwater pathway to escape the immediate vicinity of the colony (Wcisel et al. 2015; Fig. 1B). In comparison, the waters in the immediate vicinity of the two other high shark abundance colonies (Mossel Bay and False Bay) are relatively deep and featureless, lacking landscape characteristics that allow seals to proactively mitigate their risk of attack (Fig. 1C). Indeed, recorded hourly predation rates by white sharks on seals in False Bay are more than 18 times higher than at Geyser Rock (Wcisel et al. 2015). Moreover, the False Bay investigation revealed strong positive correlations between seal fecal stress levels and shark attack rates, but not with shark relative abundance. These results are consistent with growing theoretical and empirical predator–prey research indicating that predator presence alone does not equate to risk, as predator hunting mode, prey anti-predator behavior, and habitat characteristics that mediate both will influence risk (Schmitz 2008, Heithaus et al. 2009).

The finding from False Bay that seal fecal cortisol levels were correlated with attack rates at a weekly scale suggests seals can accurately assess and subsequently mount a stress reaction in relation to the magnitude of actual risk posed from shark predation. The base of information used by seals leading to these responses might be obtained from direct observations of daily shark attack rates around the colony, by in-water encounter rates with attacking sharks, or by on-island encounter rates with conspecifics bearing fresh bite wounds (Fig 1D). It is likely that such information could be socially transmitted at colonies or in water among these highly social mammals.

Alternative explanations

High population density or crowding can impact stress levels in animals and could have contributed in part to some of the variation in glucocorticoid secretion measured among colonies. However, it is unlikely that density differences among colonies or between seasons was the primary driver of fGCM patterns found, for several reasons. First, mean fGCM values were not correlated with our measures of seal

colony density or population size across the study period (Fig. 5A,B). Second, as long-lived, *k*-selected species, female Cape fur seals only give birth to one offspring per year (Kirkman et al. 2011) and established colonies in the study region do not exhibit significant monthly or seasonal fluctuations in population size or density (Huisamen et al. 2011). The only period during which the islands exhibit relatively large changes in seal density is during the breeding season (December); however, we did not collect fecal samples at this time to avoid disturbing breeding animals.

Glucocorticoids often rise in late gestation of mammals due to an increase in binding globulins (see Creel et al. 2009 for a discussion), but this phenomenon cannot explain the observed differences among colonies or the correlation of fGCM with exposure to risk at the weekly time scale in False Bay. Additionally, Jutten Island is not a breeding colony.

While annual or seasonal changes in climate could impact seal stress levels, this is unlikely to be the primary driver of spatiotemporal variation in stress levels measured given that all the study islands occur within the same region. Similarly, changes in food supply could be expected to influence stress levels in the seals, but the colonies examined shared a common food supply (Rand 1959, 1967, Oosthuizen 1991). Although colonies vary geographically and thus could be exposed to an associated gradient in environmental factors that may influence seal stress levels, we found no correlation between latitude and longitude on fGCM levels at the colonies.

Hypothesized conditions leading to stress-mediated responses to risk

It has been previously suggested that differences in the frequency and magnitude of predation risk may affect whether prey mount a physiological stress response (Creel et al. 2009). Seals at the high risk colonies of False Bay and Mossel Bay may behaviorally reduce their individual level of risk by employing grouping when in the water (dilution effect and increased vigilance; Fallows et al. 2012) and/or perhaps by shifting movements to the night (presumably to benefit from the cover of darkness; Johnson et al. 2009, De Vos et al. 2015*b*, Fallows et al. 2016). However, once in the water and traversing the gauntlet, the seals cannot reliably predict or detect a hunting shark prior to the launch of an ambush attack (Martin et al. 2005). For example, shark kill rate averages 0.48 per day in False Bay, with frequency of attacks ranging from 0 to 45 per day (Hammerschlag et al. 2006). Thus, when traversing the gauntlet in False Bay and Mossel Bay, seals are exposed to unpredictable, potentially lethal, and relatively uncontrollable risk of attack, precisely the conditions known to produce physiological stress in controlled biomedical experiments (Weiss 1970, Romero 2004, Sapolsky 2005). As discussed above, despite a high abundance of hunting white sharks at Geyser Rock, relative predation risk to seals is comparatively low due to landscape features permitting seals to proactively mitigate risk of shark attack (Wcisel et al. 2015). Here again, the ability to control exposure to a stressor is known to reduce glucocorticoid responses in biomedical experiments (Weiss 1970). A similar predator–prey scenario to the one at Geyser Rock may exist between wolves and elk in the Yellowstone ecosystem. Specifically, the presence of wolves has not been found to cause activation of the fGCM stress response (Creel et al. 2009), likely because elk can proactively mitigate their risk by altering their patterns of habitat selection, grouping and behavior, and wolves can be detected proactively using a combination of vision, olfaction, hearing, and perhaps social transmission of information (Creel and Winnie 2005, Fortin et al. 2005, Christianson and Creel 2010).

Conclusions

In summary, our study had four primary results. First, differences in measured Cape fur seal fGCM levels were strongly associated with patterns of white shark predation risk at the site and seasonal level, equally for both adults and juveniles, based on multi-year sampling from six populations. Second, seal fGCM levels were not correlated with colony population size or density, nor with colony geographic location (a proxy for geographic gradients in environmental factors that might be expected to influence stress levels). Third, within a high risk site (False Bay), fecal cortisol concentrations across two years were strongly correlated with temporal variation in shark attack rates on seals at the weekly scale, but not with shark relative abundance. Finally, seals from the Geyser Rock colony did not show a pronounced rise in fGCM levels in response to sharks despite being exposed to high encounter rates with sharks. However, in contrast to the other focal colonies targeted by white sharks, seals at Geyser Rock can proactively mitigate their risk behaviorally through use of subsurface habitat refuges (Wcisel et al. 2015). Based on results from the current shark–seal study system and in comparison to other systems (e.g., elk exposed to wolves), we hypothesize that predation risk will produce physiological costs, in the form of a stress response, when risk cannot be adequately predicted or mitigated by behavioral responses.

Glucocorticoid stress responses can carry fitness and reproductive costs to individuals (Weiss 1970, Sapolsky 2005) and previous research has revealed that acute and chronic physiological stress experienced by fur seals can consequently result in mortality (Seguel et al. 2014). Thus, future research is needed to determine the potential differential contribution of various shark effects (e.g., predation mortality, physiological stress, foraging mediated costs) and environmental variables (e.g., food limitation) on seal fitness and population dynamics at the colonies. This is especially important given recent conflicting data on declining white shark populations in the study region and associated concerns for the ecological consequences (Towner et al. 2013, Andreotti et al. 2016).

Understanding the mechanisms and underlying circumstances that can lead to differences in the occurrence,

magnitude, and type of prey response to a predator in the wild is a challenge, but an important one given widespread declines of top predators globally and associated growing conservation recovery efforts and successes (Myers and Worm 2003, Estes et al. 2011, Neubauer et al. 2013, Marshall et al. 2016). It is probable that pathways leading to stress reactions of prey to predation risk will vary among taxa, contexts, and systems, with differing consequences for prey population dynamics, as has been found for food-mediated responses to risk (Schmitz 2008). The hypothesis presented here, describing the conditions leading to physiological stress responses in prey, provides a testable null model that may aid in future empirical investigations of the physiological mechanisms underlying predation risk effects on prey in the wild.

Acknowledgments

For providing satellite tagging data used in this study, we wish to thank OCEARCH and collaborating researchers from the OCEARCH South Africa Expedition, including Chris Fischer, Alison Kock, Dylan Irion, Alison Towner, Enrico Gennari, Ryan Johnson, and Malcolm Smale. For providing white shark survey data throughout the Western Cape province of South Africa, we are thankful to Sara Andreotti. Seal fecal samples were collected under research permits from the South African National Parks and the South African Department of Environmental Affairs (DEA). For assisting with collection and organization of fecal samples, we are grateful to Yves Chesselet (Cape Nature), Deon Kotze, Steven McCue, Laurenne Snyders, Bruce Dyer, Lieze Swart, and several DEA interns. For assisting with predation data and field work, thanks to Monique Fallows and Lacey Williams from Apex Shark Expeditions. Thanks to Rachel Skubel and Emily Nelson for helping with paper formatting. We thank John Bruno, Mark Hebblewhite and the anonymous reviewers of our manuscript for their comments that significantly strengthened our paper.

Literature Cited

Acuña-Marrero, D., A. N. H. Smith, N. Hammerschlag, A. Hearn, M. J. Anderson, H. Calich, M. D. M. Pawley, C. Fischer, and P. Salinas-de-León. 2017. Residency and movement patterns of an apex predatory shark (Galeocerdo cuvier) at the Galapagos Marine Reserve. PLoS ONE 12:e0183669.

Andreotti, S. 2015. The conservation of South African white sharks: population number, genetic distinctiveness and global connections. Dissertation. Stellenbosch University, Stellenbosch, South Africa. https://scholar.sun.ac.za

Andreotti, S., M. Rutzen, S. Van der Walt, S. Von der Heyden, R. Henriques, M. Meÿer, H. Oosthuizen, and C. A. Matthee. 2016. An integrated mark-recapture and genetic approach to estimate the population size of white sharks in South Africa. Marine Ecology Progress Series 552:241–253.

Bonfil, R., M. Meÿer, M. C. Scholl, R. Johnson, S. O'Brien, H. Oosthuizen, S. Swanson, D. Kotze, and M. Paterson. 2005. Transoceanic migration, spatial dynamics, and population linkages of white sharks. Science 310:100–103.

Boonstra, R., D. Hik, G. R. Singleton, and A. Tinnikov. 1998. The impact of predator-induced stress on the snowshoe hare cycle. Ecological Monographs 68:371–394.

Brashares, J. S., L. R. Prugh, C. J. Stoner, and C. W. Epps. 2010. Ecological and conservation implications of mesopredator release. Pages 221–240 *in* J. Terbough and J. A. Estes, editors. Trophic cascades: predators, prey, and the changing dynamics of nature. Island Press, Washington DC, USA.

Christianson, D., and S. Creel. 2010. A nutritionally mediated risk effect of wolves on elk. Ecology 91:1184–1191.

Clinchy, M., M. J. Sheriff, and L. Y. Zanette. 2013. Predator-induced stress and the ecology of fear. Functional Ecology 27:56–65.

Clinchy, M., L. Zanette, R. Boonstra, J. C. Wingfield, and J. N. M. Smith. 2004. Balancing food and predator pressure induces chronic stress in songbirds. Proceedings of the Royal Society B 271:2473–2479.

Creel, S., D. Christianson, and P. Schuette. 2013. Glucocorticoid stress responses of lions in relationship to group composition, human land use, and proximity to people. Conservation Physiology 1:1–11.

Creel, S., J. A. Winnie, and D. Christianson. 2009. Glucocorticoid stress hormones and the effect of predation risk on elk reproduction. Proceedings of the National Academy of Sciences USA 106:12388–12393.

Creel, S., J. Winnie, B. Maxwell, K. Hamlin, and M. Creel. 2005. Elk alter habitat selection as an antipredator response to wolves. Ecology 86:3387–3397.

Creel, S., and J. A. Winnie. 2005. Responses of elk herd size to fine-scale spatial and temporal variation in the risk of predation by wolves. Animal Behaviour 69:1181–1189.

De Vos, A., M. J. O'Riain, M. A. Meÿer, P. G. H. Kotze, and A. A. Kock. 2015*a*. Behavior of Cape fur seals (*Arctocephalus pusillus pusillus*) in response to spatial variation in white shark (*Carcharodon carcharias*) predation risk. Marine Mammal Science 3:1234–1251.

De Vos, A., M. J. O'Riain, M. A. Meÿer, P. G. H. Kotze, and A. A. Kock. 2015*b*. Behavior of Cape fur seals (*Arctocephalus pusillus pusillus*) in response to temporal variation in predation risk by white sharks (*Carcharodon carcharias*) around a seal rookery in False Bay, South Africa. Marine Mammal Science 31:1118–1131.

Domeier, M. L., and N. Nasby-Lucas. 2007. Annual re-sightings of photographically identified white sharks (*Carcharodon carcharias*) at an eastern Pacific aggregation site (Guadalupe Island, Mexico). Marine Biology 150:977–984.

Dudley, S. F. J. 2012. A review of research on the white shark, *Carcharodon carcharias* (Linnaeus). Pages 511–532 *in* M. L. Domeier, editor. Global perspectives on the biology and life history of the white shark. Taylor and Francis Group, Boca Raton, Florida, USA.

Estes, J. A., et al. 2011. Trophic downgrading of planet Earth. Science 333:301–306.

Fallows, C., R. A. Martin, and N. Hammerschlag. 2012. Pages 105–117 *in* M. L. Domeier, editor. Global perspectives on the biology and life history of the white shark. CRC Press, Boca Raton, Florida.

Fallows, C., M. Fallows, and N. Hammerschlag. 2016. Effects of lunar phase on predator-prey interactions between white shark (*Carcharodon carcharias*) and Cape fur seals (*Arctocephalus pusillus pusillus*). Environmental Biology of Fishes 99:805–812.

Fortin, D., H. L. Beyer, M. S. Boyce, D. W. Smith, T. Duchesne, and J. S. Mao. 2005. Wolves influence elk movements: behavior shapes a trophic cascade in Yellowstone National Park. Ecology 86:1320–1330.

Gallagher, A. J., S. Creel, R. P. Wilson, and S. J. Cooke. 2017*a*. Energy landscapes and the landscape of fear. Trends in Ecology & Evolution 32:88–96.

Gallagher, A. J., R. A. Skubel, H. R. Pethybridge, and N. Hammerschlag. 2017*b*. Energy metabolism in mobile, wild-sampled sharks inferred by plasma lipids. Conservation Physiology 5. https://doi.org/10.1093/conphys/cox002

Hammerschlag, N., and C. Fallows. 2005. Galapagos sharks (*Carcharhinus galapagensis*) at the Bassas da India atoll: First record from the Mozambique Channel and possible

significance as a nursery area. South African Journal of Marine Science 101:375–377.

Hammerschlag, N., A. J. Gallagher, and D. M. Lazarre. 2011. A review of shark satellite tagging studies. Journal of Experimental Marine Biology and Ecology 398:1–8.

Hammerschlag, N., R. A. Martin, and C. Fallows. 2006. Effects of environmental conditions on predator–prey interactions between white sharks (*Carcharodon carcharias*) and Cape fur seals (*Arctocephalus pusillus pusillus*) at Seal Island, South Africa. Environmental Biology of Fishes 76:341–350.

Heithaus, M. R., A. J. Wirsing, D. Burkholder, J. Thomson, and L. M. Dill. 2009. Towards a predictive framework for predator risk effects: the interaction of landscape features and prey escape tactics. Journal of Animal Ecology 78:556–562.

Huisamen, J., S. P. Kirkman, L. H. Watson, P. A. Pistorius, and V. G. Cockcroft. 2011. Re-colonisation of the Robberg Peninsula (Plettenberg Bay, South Africa) by Cape fur seal. African Journal of Marine Science 33:453–462.

Hunt, K. E., A. W. Trites, and S. K. Wasser. 2004. Validation of a fecal glucocorticoid assay for Steller sea lions (*Eumetopias jubatus*). Physiology & Behavior 80:595–601.

Jewell, O. J., R. L. Johnson, E. Gennari, and M. N. Bester. 2013. Fine scale movements and activity areas of white sharks (*Carcharodon carcharias*) in Mossel Bay, South Africa. Environmental Biology of Fishes 96:881–894.

Johnson, R., M. N. Bester, S. F. Dudley, W. H. Oosthuizen, M. Meÿer, L. Hancke, and E. Gennari. 2009. Coastal swimming patterns of white sharks (*Carcharodon carcharias*) at Mossel Bay, South Africa. Environmental Biology of Fishes 85:189–200.

Jonsen, I. 2016. Joint estimation over multiple individuals improves behavioural state inference from animal movement data. Scientific Reports 6. https://doi.org/10.1038/srep20625

Kirkman, S. P., W. H. Oosthuizen, M. A. Meyer, S. M. Seakamela, and L. G. Underhill. 2011. Prioritising range-wide scientific monitoring of the Cape fur seal in southern Africa. African Journal of Marine Science 33:495–509.

Kock, A., M. J. O'Riain, K. Mauff, M. Meÿer, D. Kotze, and C. Griffiths. 2013. Residency, habitat use and sexual segregation of white sharks, *Carcharodon carcharias* in False Bay, South Africa. PloS ONE 8:e55048.

Marshall, K. N., A. C. Stier, J. F. Samhour, R. P. Kelly, and E. J. Ward. 2016. Conservation challenges of predator recovery. Conservation Letters 9:70–78.

Martin, R. A., N. Hammerschlag, R. S. Collier, and C. Fallows. 2005. Predatory behaviour of white sharks (*Carcharodon carcharias*) at Seal Island, South Africa. Journal of the Marine Biological Association of the United Kingdom 85:1121–1135.

Martin, R. A., D. K. Rossmo, and N. Hammerschlag. 2009. Hunting patterns and geographic profiling of white shark predation. Journal of Zoology 279:111–118.

Monfort, S. L., K. L. Mashburn, S. K. Wasser, M. Burke, B. A. Brewer, and S. R. Creel. 1997. Steroid metabolism and validation of noninvasive endocrine monitoring in the African wild dog (*Lycaon pictus*). Zoo Biology 6:533–548.

Myers, R. A., and B. Worm. 2003. Rapid worldwide depletion of predatory fish communities. Nature 423:280–283.

Neubauer, P., O. P. Jensen, J. A. Hutchings, and J. K. Baum. 2013. Resilience and recovery of overexploited marine populations. Science 340:347–349.

Oosthuizen, W. H. 1991. General movements of South African (Cape) fur seals *Arctocephalus pusillus pusillus* from analysis of recoveries of tagged animals. South African Journal of Marine Science 11:21–29.

Peckarsky, B. L., C. A. Cowan, M. A. Penton, and C. Anderson. 2010. Sublethal consequences of stream-dwelling predatory stoneflies on mayfly growth and fecundity. Ecology 74: 1836–1846.

Peckarsky, B. L., et al. 2008. Revisiting the classics: considering nonconsumptive effects in textbook examples of predator-prey interactions. Ecology 89:2416–2425.

Rand, R. 1959. The Cape fur seal (*Arctocephalus pusillus*): Distribution, abundance and feeding habits off the south western coast of the Cape Province. Investigational Report No. 34, South African Division of Fisheries, South Africa.

Rand, R. 1967. The Cape fur seal (*Arctocephalus pusillus*): General behaviour on land and at sea. Investigational Report No. 60, South African Division of Fisheries, South Africa.

Romero, L. M. 2004. Physiological stress in ecology: lessons from biomedical research. Trends in Ecology & Evolution 19:249–255.

Sapolsky, R. M. 2005. The influence of social hierarchy on primate health. Science 308:648–652.

Schmitz, O. J. 2008. Effects of predator hunting mode on grassland ecosystem function. Science 319:952–954.

Schmitz, O. J., A. P. Beckerman, and K. M. O'Brien. 1997. Behaviorally mediated trophic cascades: effects of predation risk on food web interactions. Ecology 78:1388–1399.

Seguel, M., E. Paredes, H. Pavés, and N. L. Gottdenker. 2014. Capture-induced stress cardiomyopathy in South American fur seal pups (*Arctophoca australis gracilis*). Marine Mammal Science 30:1149–1157.

Sheriff, M. J., C. J. Krebss, and R. Boonstra. 2009. The sensitive hare: sublethal effects of predator stress on reproduction in snowshoe hares. Journal of Animal Ecology 78:249–1258.

Silverman, B. W. 1986. Density estimation for statistics and data analysis. Chapman and Hall, New York, New York, USA.

Sinclair, A. R. E., and P. Arcese. 1995. Population consequences of predation-sensitive foraging: the Serengeti wildebeest. Ecology 76:882–891.

Towner, A. V., V. Leos-Barajas, R. Langrock, R. S. Schick, M. J. Smale, T. Kaschke, O. J. Jewell, and Y. P. Papastamatiou. 2016. Sex-specific and individual preferences for hunting strategies in white sharks. Functional Ecology 30:1397–1407.

Towner, A. V., M. A. Wcisel, R. R. Reisinger, D. Edwards, and O. J. D. Jewell. 2013. Gauging the threat: the first population estimate for white sharks in South Africa using photo identification and automated software. PLoS ONE 8:6–12.

Wcisel, M., M. J. O'Riain, A. de Vos, and W. Chivell. 2015. The role of refugia in reducing predation risk for Cape fur seals by white sharks. Behavioral Ecology and Sociobiology 69:127–138.

Weiss, J. M. 1970. Somatic effects of predictable and unpredictable shock. Psychosomatic Medicine 32:97–408.

Werner, E. E., and S. D. Peacor. 2003. A review of trait-mediated indirect interactions in ecological communities. Ecology 84:1083–1100.

Zanette, L. Y., M. Clinchy, and J. P. Suraci. 2014. Diagnosing predation risk effects on demography: Can measuring physiology provide the means? Oecologia 176:637–651.

Supporting Information

Additional supporting information may be found in the online version of this article at http://onlinelibrary.wiley.com/doi/10.1002/ecy.2049/suppinfo