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Review article

The role of the hypothalamus-pituitary-adrenal/interrenal axis in mediating predator-avoidance trade-offs

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ABSTRACT

Maintaining energy balance and reproducing are important for fitness, yet animals have evolved mechanisms by which the hypothalamus-pituitary-adrenal/interrenal (HPA/HPI) axis can shut these activities off. While HPA/HPI axis inhibition of feeding and reproduction may have evolved as a predator defense, to date there has been no review across taxa of the causal evidence for such a relationship. Here we review the literature on this topic by addressing evidence for three predictions: that exposure to predators decreases reproduction and feeding, that exposure to predators activates the HPA/HPI axis, and that predator-induced activation of the HPA/HPI axis inhibits foraging and reproduction. Weight of evidence indicates that exposure to predator cues inhibits several aspects of foraging and reproduction. While the evidence from fish and mammals supports the hypothesis that predator cues activate the HPA/HPI axis, the existing data in other vertebrate taxa are equivocal. A causal role for the HPA axis in predator-induced suppression of feeding and reproduction has not been demonstrated to date, although many studies report correlative relationships between HPA activity and reproduction and/or feeding. Manipulation of HPA/HPI axis signaling will be required in future studies to demonstrate direct mediation of predator-induced inhibition of feeding and reproduction. Understanding the circuitry linking sensory pathways to their control of the HPA/HPI axis also is needed. Finally, the role that fear and anxiety pathways play in the response of the HPA axis to predator cues is needed to better understand the role that predators have played in shaping anxiety related behaviors in all species, including humans.

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1. Introduction

Maintaining energy balance and successfully reproducing are not only important components of fitness, but they are enjoyable activities, reinforced through innate neuronal circuitry involving the release of dopamine in the nucleus accumbens (Hernandez et al., 1988; Bassareo and Di Chiara, 1999; Fiorino and Phillips, 1999; Hull, 2011). Yet as sophisticated as the control of reproduction and foraging may be, there are equally sophisticated mechanisms for shutting these activities off completely. One mechanism that has been studied in detail is the hypothalamus-pituitary-adrenal, or HPA, axis (or the hypothalamus-pituitary-interrenal, HPI, axis in fishes and amphibians). Although a considerable amount has been learned about *how* the HPA/HPI axis inhibits foraging and reproduction, we still do not fully understand *why* such mechanisms would evolve in the first place. One possibility is that HPA/HPI axis inhibition of reproduction and foraging is important for allowing animals to survive encounters with a predator. There are hundreds of papers reporting that reproduction and foraging efforts are adjusted in response to predation. In fact, at the heart of optimal foraging and life history theories is the implicit suggestion that somehow animals are able to gauge the costs and benefits of such predator-avoidance trade-offs in some physiologically unknown way, perhaps through the HPA/HPI axis and its hard-wired connections with visual, olfactory, and auditory brain areas detecting predator cues. This is not a trivial point, as some authors have suggested that the HPA/HPI axis role in predator trade-offs is important enough to ultimately influence population demographics (the chronic stress hypothesis, see Creel et al., 2009 for summary; Sheriff and Thaler, 2014), although data supporting such a role are still elusive (Creel et al., 2009).

Have predator avoidance trade-offs influenced HPA/HPI axis regulation of reproduction and foraging? As provocative as this question is, there is no way to answer it without evidence of a causal role for the HPA/HPI axis in mediating predator avoidance trade-offs. Since HPA/HPI axis hormones and feedback pathways have largely been conserved during vertebrate evolution, analysis across vertebrate taxa is required to flush out all of the evidence that may exist supporting such a relationship. To this end we review the literature on this topic across vertebrate taxa by addressing evidence for three main predictions, 1) that exposure to predators, or predator cues, decreases reproductive and feeding behavior and reproductive investment, 2) that exposure to predators, or predator cues, activates the HPA/HPI axis, and 3) that predator-induced activation of the HPA/HPI axis plays a direct role in foraging and reproduction trade-offs. The first prediction has been explored in detail elsewhere but the most comprehensive reviews on this topic were published about 20 years ago (e.g., Lima and Dill, 1990; Lima, 1998b). Thus, in Section 2 we begin by briefly reviewing this topic in order to provide background for the other two predictions and to address new studies in this area. In Section 3, we review the HPA/HPI axis and describe mechanisms by which HPA/HPI axis hormones can regulate foraging and reproduction. The idea that predators cause activation of the HPA/HPI axis is not new, but no review, to our knowledge, has compiled extensive evidence for this topic across vertebrate taxa (but see Hawlena and Schmitz, 2010; Johnstone et al., 2012; Zanette et al., 2014 for discussion of aspects of stress and predators); this topic is covered in Section 4 and includes a table with data from 168 experimental cases. In Section 5 we discuss scenarios in which variation in the HPA/HPI axis response to predators might be expected, and in Section 6 we propose a potential mechanism of how predator-induced HPA/HPI axis activation may modulate response to future predator exposure. Namely, that exposure to predators, and concomitant HPA/HPI axis activation, may produce neuroregulatory changes that promote survival by altering brain regions important for detecting and responding to predators or predator cues. In Section 7 we investigate the strength of the existing evidence supporting a role for the HPA/HPI axis in mediating predator avoidance trade-offs and evaluate ways in which comparative endocrinologists can use their expertise to test this hypothesis experimentally.

2. Adjustments in reproductive and foraging effort in response to predators

In the presence of a predator or predator cues, decreases in foraging and reproductive investment are well documented across taxa (Lima, 1998a; Brown and Kotler, 2004; Hawlena and Schmitz, 2010; Apfelbach et al., 2005; Kavaliers and Choleris, 2001; Lima and Dill, 1990; Hegab et al., 2015). Such decreases are presumably beneficial as they help organisms respond to, and hopefully avoid, predation attempts, and thus increase survival and overall fitness. Predator-induced changes in prey reproduction can include decreases in reproductive physiology (e.g., hypothalamus-pituitary-gonadal, HPG, axis function) and/or changes in reproductive investment and behavior (e.g., courtship and mating, territory choice and defense, fecundity, offspring size, and parental behaviors; see Lima, 1998a,b; Sih, 1994). Reproductive alterations can be a direct response to a predator (e.g., abandoning a courtship attempt or offspring during predator attack) or can be from indirect effects due to a lack of the energy reserves needed to support reproductive functions. Foraging alterations can include various tactics such as increased vigilance, decreased feeding time, or feeding in safer areas that contain food of lesser quality (see Brown and Kotler, 2004; Lima, 1998a). Although it is unknown exactly how predator threat results in decreased reproductive function and altered foraging, a potential mechanism is through activation of the stress response and the resulting increase in glucocorticoid hormone concentrations (see Boonstra, 2013; Lima, 1998b). Before discussing predation and the mechanisms by which HPA/HPI axis activity may influence reproduction and foraging, however, we provide a brief review of elements of reproduction and foraging that are suspected targets of HPA/HPI axis hormones and are known to be influenced by predation.

The cycling of prey species' population size in response to predator threat has been used as a model for addressing various predation-related trade-offs (see Korpimaki and Krebs, 1996). For example, the responses of snowshoe hares (*Lepus americanus*) in Yukon, Canada (see Boonstra et al., 1998; Krebs et al., 1995), elk (*Cervus elaphus*) in Yellowstone Ecosystem (see Creel et al., 2007), and voles (*Myodes* spp., *Microtus* spp., *Clethrionomys* spp.) in Fennoscandia (see Ims et al., 2008; Oksanen and Oksanen, 1992) to predators all have been used as models for addressing predator-prey questions. Therefore, several of the mammalian examples below will discuss data from these model systems.

2.1. Predator-induced adjustments in prey reproduction

2.1.1. Courtship and copulatory behavior

Changes in courtship or copulatory behavior due to predator cues can be driven by both males and females, or primarily by one sex. In response to steppe polecat urine and fecal extract, root vole (*Microtus oeconomus*) pairs delayed breeding and decreased frequency of copulation (Wang and Liu, 2002). Similarly, male rats exposed to a live cat decreased the number of female-directed mounts as compared to mounting behavior prior to cat presence (Blanchard and Blanchard, 1989). Bank voles (*Clethrionomys glareolus*) and field voles (*Microtus agrestis*) both reduced copulations (mainly driven by changes in female receptive behavior) and displayed breeding suppression in the presence of predators or predator odor (summarized in Ylönen et al., 1995). When comparing elk from areas of differing predation pressure, males did not differ in their courting behaviors; all males spent a significant amount of time engaged in attracting mates and did not differ in vigilance behavior (Wolff and Van Horn, 2003). However, female elk from areas of increased predation pressure were more vigilant than females from areas of low predation pressure, suggesting sexes differ in their behavioral response to predation (Wolff and Van Horn, 2003).

In addition to differences between sexes, behavioral responses to a predator may depend on an animal's reproductive state. For example, Cushing (1985) found that wild deer mice (*Peromyscus maniculatus*) in estrus emerged from their burrows earlier than did diestrus mice, making estrus mice active before weasels (a natural predator) began hunting. Additionally, when presented with a weasel, estrus mice fled whereas diestrus mice froze (Cushing, 1985). These strategies could help protect reproductive females from predation attempts.

Displays or calls related to mating make prey more conspicuous to predators and there are numerous examples (reviewed by Zuk and Kolluru, 1998) of predators cuing in on reproductive displays via visual (Moodie, 1972; Promislow et al., 1992; Slagsvold et al., 1995), auditory (Tuttle and Ryan, 1981; Mougeot and Bretagnolle, 2000), or olfactory clues (Ylönen et al., 2003; Hughes et al., 2010). In Lake Eyre dragon lizards (*Ctenophorus maculosus*), predation risk altered the type of rejection behavior females performed, presumably to make them less conspicuous to predators (McLean and Stuart-Fox, 2010). In male broad-headed skinks (*Eumeces laticeps*) the degree to which courtship was sacrificed in response to a predator depended upon the perceived benefit of the mate (Cooper, 1999). In mountain dusky salamanders (*Desmognathus ochrophaeus*), exposure to a predator reduces courtship behavior (Uzendoski et al., 1993; Fonner and Woodley, 2015). Male sand gobies (*Pomatoschistus minutus*) reduced courtship behavior in response to a natural predator (cod, *Gadus morhua*) (Forsgren and Magnhagen, 1993) and reductions in courtship behavior also have been observed in green razorfish (*Xyrichtys splendens*) (Nemtzov, 1994) and Iowa darters (*Etheostoma exile*) (Chivers et al., 1995) exposed to predators. However, Evans et al. (2002) reported that female guppies (*Poecilia reticulata*) exposed to predators underwent courtship behavior normally. In bank voles (*Myodes glareolus*) lactating females adjusted their foraging efforts based on season and manipulated presence of a predator (common shrew; *Sorex araneus*); when predation pressure was high, mothers did not nest guard but instead foraged further from the nest, possibly to limit the accumulation of odor signals near pups (Liesenjohann et al., 2015).

2.1.2. Investment in offspring

The predator-induced breeding suppression hypothesis (BSH; Ronkainen and Ylönen, 1994; Ylönen and Ronkainen, 1994) suggests that in response to the odor of mustelid predators female voles will reduce reproductive output. In this model chronic, as opposed to acute, exposure to predators can alter various aspects of reproduction including litter size, offspring size, offspring sex ratio, and offspring development. These outcomes can be influenced by pre- or post-copulation changes in either the male or the female. In a broader survey of several studies across a variety of mammalian species offspring outcomes were measured, but it is not always clear from these studies if alterations in the male, the female, or both were responsible for the resulting predator-induced changes. In the large data set on the BSH in voles more data exist on specific effects in females, in part due to the higher predation pressure on female voles than males (Norrdahl and Korpimaki, 1998). In general, though, the trend for an emphasis on the response of female prey to predators seems to be borne out in many mammalian studies and less has been done to document predator-induced changes in male prey reproduction.

A long-term field monitoring study of three different vole species (*Clethrionomys glareolus*, *Microtus epiroticus*, *M. agrestis*) found that litter size was negatively associated with mustelid predator density and that removal of weasels increased the number of pregnant bank voles (*C. glareolus*; Korpimaki et al., 1994).

Similar results were noted in elk living in Yellowstone, long-term field studies show that as wolf predation pressure decreases, the calf to female elk ratio increases; this effect does not seem to be due to changes in calf predation by wolves (Creel et al., 2007). Consistent results also were noted in hares. Female hares protected from predators had the highest fecundity in each year and did not experience a decrease over years, whereas unprotected females experienced a consistent decrease in fecundity over years and failed to produce second and third litters in the last year of the study (Hik, 1995). A more controlled, experimental study showed that female root voles exposed to predator (steppe polecat) excreta produced litters that weighed less as compared to litters from females not exposed to predator odor (Wang and Liu, 2002).

Predator impacts on the HPG axis, gametogenesis, and, in females, pregnancy also have been noted. Long-term monitoring of female elk fecal progesterone metabolites (FPM) suggested that circulating progesterone levels were negatively associated with wolf predation pressure (Creel et al., 2007). Additionally, predators have been shown to suppress reproduction in rodents and this effect may be due to disruption to ovarian or testicular function. Compared to control plots, field plots treated with weasel urine contained significantly fewer young and old, reproductively active female grey-sided voles (*Clethrionomys rufocanus*); treatment plots also had a lower juvenile to female ratio (Fuelling and Halle, 2004). Captive female Cambpell's hamsters (*Phodopus cambelli*) exposed daily to ferret urine displayed alterations to estrous cyclicity – cycles were either lengthened or ovulation was disrupted/absent (Sokolskaja et al., 2001). Female common voles (*Microtus arvalis*) exposed to experimentally increased mustelid predation pressure had increased inter-litter intervals, again suggesting predator presence can disrupt estrous cyclicity (Jochym and Halle, 2013). Chronic (34-d) exposure to cat urine resulted in decreased testis and epididymis weights, decreased urinary testosterone concentrations, and increased spermatic chromosomal abnormalities in male hamsters (*Phodopus campbelli*; Vasilieva et al., 2000).

Exposure to steppe polecat excreta decreased sperm count in male root voles and decreased ovary size in females (Wang and Liu, 2002). In a controlled laboratory study on rats (*Rattus norvegicus*), distance of rat cages from cages containing live lynx impacted female reproductive physiology (Naidenko et al., 2003). Specifically, compared to rats housed 25 and 80 m from the lynx cages, when rats were housed within 2 m of the lynx, either with or without direct application of lynx urine to rat-cage bedding, females gave birth to fewer offspring (Naidenko et al., 2003). This difference seems to be due to both pre- and post-implantation losses as the average number of corpora lutea was the same for control (25 and 80 m) compared to experimental (2 m with and without urine) females, and control females had more placental scars (Naidenko et al., 2003). Interestingly, distance to the lynx cage did not alter the percentage of females that became pregnant. An additional study on rats suggests that predator-urine-induced changes in litter size were due to embryo reabsorption during early gestation; these results were not due to elevated corticosterone (Voznessenskaya et al., 2003). In terms of reproductive behavior, surprisingly, exposure of female rats to cat odor on the day of, but not 3 days after, birth resulted in *increased* maternal care of offspring (Mashoodh et al., 2009). While in California mice (*Peromyscus californicus*), exposure to predator odor at 5–7 or 19–21 days post partum did not alter parental behaviors performed by either parent (Chauke et al., 2011). These data suggest that aspects of offspring investment (e.g., physiological or behavioral) can be differentially affected by predator cues, and that timing of predator exposure and prey species is likely important.

Evidence also exists to support a generalized BSH in non-mammalian vertebrates. The presence of a predator (shrike) reduced the probability of initiating a second brood in breeding pairs of tropical stonechats (*Saxicola torquata axillaris*) (Scheuerlein et al., 2001). In pied flycatchers (*Ficedula hypoleuca*), experimental movement of nests to areas of varying predation pressure resulted in smaller chicks in high predation areas (Thomson et al., 2012b). Interestingly, just the perception of predator is sufficient to reduce the number of offspring produced in birds (Zanette et al., 2011). Zanette et al. (2011) eliminated live predators from a study area in the Gulf Islands, B.C., and exposed resident songbirds to predator calls which reduced the number of eggs, hatchlings, and fledglings by 40% (Zanette et al., 2011). This was due partly to a reduced condition in reproducing females, as predator calls reduced egg mass and brood mass (Zanette et al., 2011). Simulated nest predation had similar effects, qualitatively, on this population of song birds (Travers et al., 2010). Predators also can influence brooding behavior, as nuthatches (*Sitta carolinensis*, *S. canadensis*) made fewer visits to their nests when exposed to a simulated predator (hawk) (Ghalambor and Martin, 2000). Similarly, Manx shearwaters (*Puffinus puffinus*) were less likely to return to the colony on moonlit nights when they are more vulnerable to predators (Riou and Hamer, 2008).

Predation as a cost of reproduction has been studied extensively in fish. Fecundity was reduced in killifish (*Rivulus hartii*) exposed to a natural predator (*Hoplias malabaricus*) (Fraser and Gilliam, 1992) while male dollar sunfish (*Lepomis marginatus*) spend less time guarding the nest in the presence of a predator (Winkelman, 1996). Gobies (*Pomatoschistus microps*) stay away from the nest longer in the presence of a predator (Magnhagen and Vestergaard, 1991). Three spined stickle back males suppressed breeding and reduced nuptial pad coloration in the presence of predators (Candolin, 1998) while predation risk changed mate selection behavior in sand gobies (*Pomatoschistus minutus*) (Forsgren, 1992).

Some evidence suggests that predation may influence parental behavior in fish. Convict cichlids (*Cichlasoma nigrofasciatum*) spent more time defending their brood at the cost of reduced foraging in the presence of a predator (Rangeley and Godin, 1992). When offspring are present fish may devote more effort to parental defense behavior. The latency for retreat from a predator is greater in parental male three spine sticklebacks (*Gasterosteusa culeatus*) than non-parental fish (Huntingford, 1976). Similarly, aggression reaches its peak after nest building and when offspring are present in male pumpkinseed sunfish (*Lepomis gibbosas*) (Colgan and Gross, 1977). These findings are in some ways directly opposite of those one would predict from the BSH, pointing out that reproductive stage and value of offspring are critical in assessing risk and applying the BSH across taxa.

2.2. Predator-induced alterations in prey foraging

The potential cost that foraging poses to prey animals, i.e. that prey must balance energy acquisition with safety (Sih, 1982, 1992; Lima and Dill, 1990; Skelly, 1992; Lima, 1998b; Downes, 2001; Brown and Kotler, 2004; Cresswell, 2008; Ferrari et al., 2009; Hebblewhite and Merrill, 2009), has been reported in hundreds of studies across all taxa and is an established part of optimal foraging theory (Schoener, 1971; Pyke et al., 1977; Pyke, 1984; Fraser and Huntingford, 1986; Brown et al., 1999). For example, presence of a predator increased vigilance and decreased overall activity of prey species in many but not all (Eurasian siskins, *Carduelis spinus*, for example, Pascual and Senar, 2014) studies. Vigilance is generally thought not to involve fear, but low levels of anxiety and consequently low levels of HPA/HPI axis activation, as vigilance occurs when threat of a predator contact is low (Perusini and Fanselow, 2015). For example, when wolves, having previously been absent for 50 years, were re-introduced to Yellowstone Park, greater elk (*Cervus elaphe*) and bison (*Bison bison*) females

responded by increasing vigilance (Laundre et al., 2001). This response was especially pronounced in breeding female elk with calves (Laundre et al., 2001; Winnie and Creel, 2007; Lung and Childress, 2007). Additionally, female elk from areas with greater predation were more vigilant and foraged less than females from areas of lower predation pressure; again, the response was more pronounced in females with calves (Wolff and Van Horn, 2003). Wild mule deer (*Odocoileus hemionus*) were more vigilant when at the forest edge than in the open forest, presumably due to higher predation risk by mountain lions at the edge (Altendorf et al., 2001). A study of roe deer (*Capreolus capreolus*) used the hunting season as a proxy for predator threat and found that deer spent more time vigilant and fed in areas of lesser food quality during the hunting season as compared to the non-hunting season; vigilance also decreased as distance from houses increased (Benhaiem et al., 2008).

Predator effects on prey vigilance also have been noted in rodents. Marmots (*Marmota caligata*) increase vigilance behavior when foraging farther from their burrow, and when food supply was equal (due to supplementation) marmots spent more time foraging in low-risk patches compared to high-risk patch (Holmes, 1984). Wild prairie voles (*Microtus ochrogaster*) in areas with predator exclusion had larger home ranges than voles in areas with predators present (Desy et al., 1990), suggesting that predators limit movement and home range size of voles.

Vigilance often is measured in birds and is affected by group size (Elgar, 1989) as well as the resource content in foraging areas. Essentially, in large groups and in high-resource areas both birds and mammals reduce vigilance, although obviously for different reasons (reviewed in Beauchamp, 2014). Nonetheless vigilance increases in response to predator cues in birds as it does in mammals (Powell, 1974; Caraco et al., 1980; Gluck, 1987; Tang and Schwarzkopf, 2013). Much less is known about vigilance behavior in anamniotes (Beauchamp, 2014). In some cases vigilance is used as a catch all term for behavior that is not foraging, since typical measurements of actual vigilance behavior, such as looking, do not really apply to anamniote species. For example, roach (*Rutilus rutilus*) intermission intervals in feeding were greater in the presence of predator odor and this was interpreted as a need for increased predator vigilance (Bartosiewicz and Gliwicz, 2011).

Freezing is a well-documented antipredator behavior that is obviously mutually exclusive with foraging and generally occurs *after* a predator encounter (Perusini and Fanselow, 2015). Freezing has been recognized for decades as the first component of a series of defensive behaviors associated with fear and predation (freeze, fight, flight, immobility; Ratner, 1967) and has been reported to occur in response to predators in all vertebrate taxa (Webb et al., 2010; Binazzi et al., 2011; Crane and Ferrari, 2015; Davis and Gabor, 2015; Wernecke et al., 2015).

Risk of predation can be the largest cost to the forager, and thus optimal foraging theory predicts that foraging organisms should balance food acquisition with risk of predation (see Brown and Kotler, 2004). In primates (Maior et al., 2011, 2012) and non-primate mammals (Weldon et al., 1987; Coulston et al., 1993; reviewed by Kavaliers and Choleris, 2001 and Table 1 in Apfelbach et al., 2005) predator cues decrease prey foraging activity, although there are a few cases where predator cues had no adverse affect on prey foraging (Powell and Banks, 2004). After re-introduction of wolves to Yellowstone Park, elk (*Cervus elaphus*) responded by decreasing foraging; these responses were more pronounced when wolves were present and were more robust in breeding females than in males (Winnie and Creel, 2007; Creel et al., 2005, 2007; Creel and Winnie, 2005). Elk in the Yellowstone ecosystem face almost daily changes in predation risk by wolves. Data from radio-collared elk show that when wolf predation pressure increases, elk shift from their preferred grassland foraging space to covered woodlands (Creel et al., 2005). When food was placed at various distances from dingo urine, semi-wild western grey kangaroos (*Macropus fuliginosus*) ate more of the food located greater than 12 m from the urine source (Parsons et al., 2007).

Snowshoe hares (*Lepus americanus*) exposed to various fecal, urinary, and body predator odors decreased feeding effort (Sullivan et al., 1985). Rodents in semi-natural outdoor enclosures reduced foraging efforts, shifted towards safer feeding areas (more cover), and left more food behind when exposed to a live owl, as compared to control no-owl nights (*Gerbillus allenbyi*, *G. pyramidum*, Kotler et al., 1991, 1992; *Perognathus amplus*, *P. baileyi*; *Dipodomys merriami*, Brown et al., 1988). In a semi-natural laboratory experiment, male and female rats exposed to a live cat spent less time eating, drinking and out in the open as compared to control conditions (Blanchard and Blanchard, 1989). Root vole (*Microtus oeconomus*) pairs housed in a semi-natural enclosure and exposed to steppe polecat excreta decreased food intake and lost weight compared to water-treated controls (Wang and Liu, 2002).

Suppression of foraging in the presence of a predator also has been observed in fishes (Abrahams and Sutterlin, 1999; Elvidge et al., 2014), amphibians (Skelly and Werner, 1990; Sih, 1992; Werner and Anholt, 1993; Crowley and Hopper, 1994; Abrams and Rowe, 1996; Ziemba et al., 2000; Dmitriew, 2011; Alcaraz et al., 2015), reptiles (Anson et al., 2013) and birds (Cresswell, 2008; Tilgar et al., 2011; Meitern et al., 2013) and seems to be a basal vertebrate trait (Lima, 1998a,b), although the physiological mechanisms underlying foraging suppression may differ as we will discuss ahead. An interesting exception to the general observation that prey foraging is reduced in the presence of a predator has been documented in spotted salamander (*Ambystoma maculatum*) larvae that adjust the rate of foraging based on the size of a predator's gape or capture apparatus (so called gape-dependent foraging) (Urban, 2007; Urban and Richardson, 2015). Since the size of a predator's catching apparatus may be limited, prey can avoid predation by increasing foraging rate and thus increasing growth and the likelihood that they become too large to be captured (Urban, 2007).

2.3. Changes in nutrient assimilation caused by predator cues

An interesting phenomenon has recently been reported in Trinidadian guppies (*Poecilia reticulata*; Dalton and Flecker, 2014) related to changes in energy assimilation in the face of predator cues. In response to olfactory cues from a natural predator *Crenicichla alta*, guppies not only showed a predictable and robust decrease in foraging but an increased efficiency in nitrogen assimilation (Dalton and Flecker, 2014). The reduced nitrogen excretion in guppies exposed to predator cues not only reflects the increased efficiency in processing dietary amino acids and proteins, but may deprive the local stream ecosystem of a rate limiting nutrient, thus impacting other stream flora and fauna indirectly. Moreover, the well-established role of glucocorticoids in regulating deamination and gluconeogenesis (Norris and Carr, 2013) suggests a possible role for this steroid hormone in predator-induced shifts in nutrient assimilation.

3. The HPA/HPI axis and its role in regulation of reproduction and foraging

3.1. The physiological stress response and the HPA/HPI axis

Stress is a ubiquitous term but it is often difficult to define. Due to this fact, several authors have proposed definitions and discussions of stress and the stress response can be complicated by

imprecise terminology (see Johnstone et al., 2012). Here, for the purposes of this review, we use the definition by Dhabhar and McEwen which states "Stress is a constellation of events, which begins with a stimulus (*stressor*), which precipitates a reaction in the brain (*stress perception*), which subsequently results in the activation of certain physiologic systems in the body (*stress response*; Dhabhar and McEwen, 1997)," This definition is useful in that it separates the often confusing terms of stress, stressor, and stress response, and highlights that an individual must perceive an event as stressful in order to trigger a physiological response, thus differences in perception can lead to both intra- and inter-individual variation in response. Stressors can be either acute, short-term events less than 24 h in duration (but generally much shorter), or chronic, long-term usually on the timeframe of days to weeks, and each type can result in a physiological response by the organism. Both the sympathetic nervous system (SNS) and the HPA/HPI axis are part of the physiological stress response and are critical for response to and recover from stressors (Sapolsky et al., 2000; Stratakis and Chrousos, 1995). The SNS is the primary mediator of the "fight or flight" response and responds within seconds of the onset of a stressor by releasing catecholamines, epipephrine and norepinephrine. The catecholamines result in increased heart and respiration rate, increased blood pressure, and aid in glucose metabolism (Stratakis and Chrousos, 1995). While the SNS is important and likely plays a role in both physiological and behavioral responses to stressors, it is not the main focus of this review (for information on predators and SNS, see Hawlena and Schmitz, 2010), and here we will focus solely on the HPA/HPI axis.

Although sensory information integrated in various brain regions can activate the HPA/HPI axis (see Section 4.1 below; Fig. 1), by definition this axis begins in the paraventricular nucleus (PVN) of the hypothalamus with relase of corticotropin-relasing factor (CRF) and in some instances, arginine vasopressin (AVP). These peptide hormones travel through the pituitary portal system to stimulate the release of adrenocorticotropic hormone (ACTH) from the cotricotropes in the anterior pituitary. ACTH then travels throughout the systemic circulation and binds to receptors in the adrenal/interrenal gland which results in the synthesis and release of glucocorticoids, namely cortisol or corticosterone, hereafter abbreviated CORT. CORT binds to receptors in the hypothalamus, anterior pituitary, and other brain regions (e.g., medial prefrontal cortex) and can aid in its own regulation using a classical negative feedback pathway. In addition to its role in the stress response, CORT release follows a well-documented circadian rhythm with levels peaking at the time of waking and lowest levels occurring during sleep (Sapolsky et al., 2000; Landys et al., 2006).

CORT generally rises 3–10 min following a stressor (Romero and Reed, 2005), can be measured in a suite of biological substrates (e.g., plasma, serum, saliva, hair, feathers, urine, feces). Given its steroid, hydrophobic nature, the majority of CORT in the blood stream bound to proteins, either plasma albumins or corticosteroid binding globulin (CBG) (Breuner and Orchinik, 2002; Breuner et al., 2013). CORT can be measured as free (not bound to CBG), bound (attached to CBG), or total (free + bound). The role plasma and tissue CBG play in CORT function and regulation is not fully understood and is still debated in the literature (Breuner et al., 2013; Schoech et al., 2013).

For several reasons, including the fact that CORT's conserved structure makes it easily quantifiable using a variety of commercially available kits and antibodies, the most commonly measured marker of HPA/HPI axis function is total CORT concentration (see Section 5 for more on free vs. total). Breuner et al. (2013) argues that assessment of CORT alone is insufficient to fully gauge activity of the HPA/HPI axis and the impact of stress on an organism as total CORT only yields data on one aspect of the HPA/HPI axis and getting at other HPA/HPI markers and downstream affectors is likely necessary to fully understand how stress impacts organisms. Collecting multiple measures is possible in certain scenarios, yet there are many logistic and technical factors that can limit assessment of the HPA/HPI axis in non-model species, and some have even questioned the value of free CORT estimates in blood collected in field studies (Schoech et al., 2013). In addition the lack of suitable homologous antisera for ACTH and CRF, the practical limitation of body size, and missing data on gene or protein structure all can be factors in deciding how best to assess HPA/HPI axis activity. Steroid hormones as a group tend to be more stable than HPA/HPI axis peptides (i.e., ACTH) and can be assayed from samples collected in a non-invasive manner.

Elevation of CORT in response during stress is adaptive as it aids organisms in responding to and coping with stressors (Sapolsky et al., 2000). CORT can influence regulation of roughly 10% of the genome (Le et al., 2005), and can affect multiple processes, such as metabolism, growth, inflammation, gluconeogenesis, cognition, reproduction, cardiovascular function, and behavior (Lupien et al., 2007; Sapolsky et al., 2000; Stratakis and Chrousos, 1995). The most well-known mechanism by which CORT exerts is effects by altering gene transcription and protein synthesis, a process taking 1–2 h (Yamamoto, 1985; Hayashi et al., 2004). These genomic effects occur by activation of nuclear mineralocorticoid receptors (MR) and glucocorticoid receptors (GR), with MR being bound at low and high CORT levels and GR being bound at high levels (Landys et al., 2006; Dallman et al., 2000). However, more recent studies have suggested that CORT also has membrane-bound MR-like receptors (Joëls et al., 2013; Joëls and Baram, 2009); allowing for rapid effects of CORT on neuronal function and organismal behavior (Moore et al., 2005; Groeneweg et al., 2011).

3.2. HPA/HPI axis regulation of reproduction and foraging

In his 1998 review Lima states "Finally, I suspect that research exploring the link between antipredator decision making and the physiological stress response will prove rewarding." (pg. 265; Lima, 1998b). This sentiment may be shared by many comparative endocrinologists, although it is difficult to argue that predator-stimulus-evoked elevations in blood CORT can act via traditional GRs to alter the immediate outcome of a predator-prey encounter. This is because GR activation may take hours, and the non-consumptive part of a predator prey encounter can be over in seconds. On the other hand, neurons in the PVN producing peptides such as CRF and arginine vasopressin (AVP; or arginine vasotocin, AVT in non-mammalian vertebrates) are activated much more rapidly after exposure to a predator and have entirely separate independent actions on reproduction and foraging. It is entirely possible, however, that CORT may affect prey reproduction and foraging prior to and after a predator encounter by activating rapid, membrane-bound receptors or by maintaining or modulating important predator-response neural pathways (see Section 6).

3.3. PVN and extra-PVN CRF and AVP/AVT effects on reproduction and foraging

CRF has been reported to inhibit courtship (Maney and Wingfield, 1998), parental care (Pedersen et al., 1991; Klampfl et al., 2013; Saltzman et al., 2011; Gammie et al., 2004), estrous behavior (Seymour et al., 2005), and reproductive behavior in males (Sirinathsinghji, 1987). CRF can affect the HPG axis at multiple levels including regulation of gonadotropin secretion from the pituitary gland (Kageyama et al., 2003; Kageyama, 2013; Nemoto et al., 2009) and regulation of gonadotropin-releasing hormone (GnRH) secreting neurons (Kinsey-Jones et al., 2006) and the GnRH pulse generator (Li et al., 2010). AVP also has been suspected to

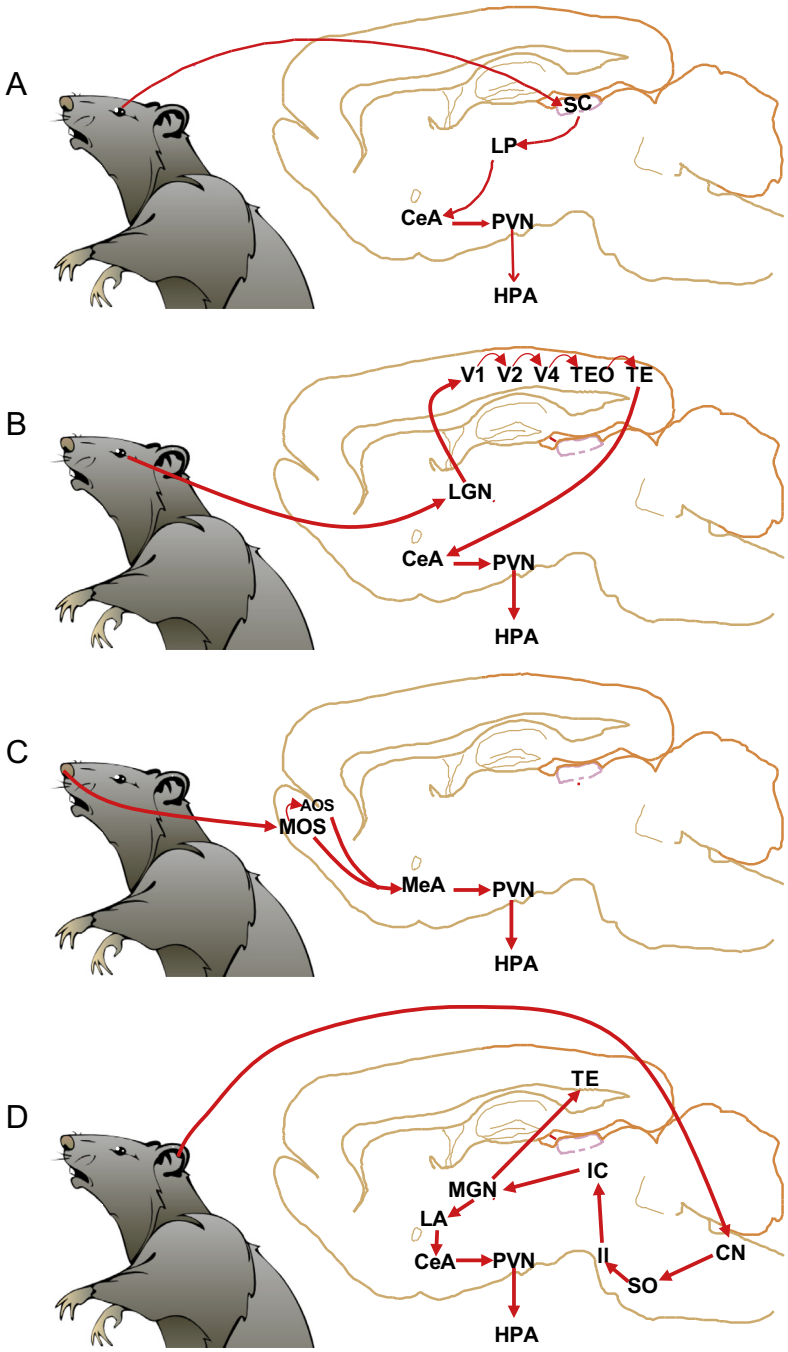

Fig. 1. The hypothalamus-pituitary-adrenal axis is the motor output for the colliculus-amygdala-hypothalamus, cortex-amygdala-hypothalamus, olfactory bulb-amygdala-hypothalamus and cochlear-amygdala-hypothalamus pathways responding to predator cues. There are two possibilities by which visual predator cues are detected and activate HPA axis secretion, a subcortical route (A) and a cortical route (B), both of which relay information to the PVN though the CeA. Olfactory cues are relayed to the PVN through the medial amygdala (C) while acoustic cues require the MGN, LA, and CeA as relays (D). Arrows depict known efferent projections. CeA, central amygdala; CN, cochlear nucleus; IC, inferior colliculus; LA, lateral amygdala; LGN, lateral geniculate nucleus; ll, lateral lemniscus; MGN, medial geniculate nucleus; SO, superior olivary complex; TE, inferior temporal cortex; SC, superior colliculus; TE, inferior temporal cortex; TEO, inferior temporal cortex; V, visual cortex. A and B based on Pessoa and Adolphs (2010) and Carr (2015), while D is adapted from Campeau and Watson (1997).

influence several aspects of reproductive behavior (see Caldwell et al., 2008; Pei et al., 2014; Gesto et al., 2014; Kelly and Goodson, 2014; Benarroch, 2013; Balment et al., 2006).

CRF administration inhibits many aspects of foraging (Crespi and Denver, 2004, 2005), prey capture (Carr et al., 2002; Crespi and Denver, 2004; Matsuda et al., 2010; Morimoto et al., 2011), and food intake (de Pedro et al., 1993; Bernier and Peter, 2001; Zorrilla et al., 2003; Bernier and Craig, 2005; Volkoff et al., 2005; Bernier, 2006; Matsuda, 2013; Ortega et al., 2013; Stengel and Tache, 2014) across all vertebrate taxa. CRF also can induce freezing (Swiergiel et al., 2007). Although most studies involve administration of CRF directly into brain ventricles, peripheral administration of CRF also inhibits food intake (Stengel and Tache, 2014) through a number of potential mechanisms including delayed gastric emptying and increased satiety feedback, hyperglycemia and direct actions of glucose on glucose sensing neurons

in the hypothalamus, or via intrahypothalamic neuronal connections and modulation of anorexia-promoting cell groups outside the PVN (summarized in Stengel and Tache, 2014).

Because responding quickly to a predator cue may be of paramount importance to survival, we began looking at the possibility that CRF and CRF receptors may be located in the optic tectum, a visual area critical for detecting and responding to predators in all vertebrates. We hypothesized that by acting at the site of first-order retinal synapses, CRF may influence visually guided prey capture more rapidly than through deeper hypothalamic pathways and that the increased speed in affecting prey capture might be evolutionarily adaptive. In fact, CRF immunoreactive fibers are extensive in outer layers of the tectum and, in addition, we have identified CRF producing cells in tectal layers 6 and 8, suggesting that interneurons may express CRF as well (Carr et al., 2010). The optic tectum has CRF R1 binding sites and CRF is released by tectal neurons in a calcium dependent manner (Carr et al., 2013), suggesting that this peptide may be an important modulator in tectal prey capture pathways.

In addition to its role in reproduction, AVP (and its non-mammalian homologs, e.g., vasotocin, isotocin) has been implicated in regulation of feeding behavior. For example, in rainbow trout (*Oncorhynchus mykiss*), central infusion of arginine vasotocin resulted in a decrease in feeding (Gesto et al., 2014). Similarly, in mice, activation of paraventricular hypothalamic AVP neurons inhibits food intake, a respond though to be associated with melanocortin signaling (Pei et al., 2014).

In summary, it clear that these same peptides involved in the classical HPA/HPI axis response to stressors are also involved in the regulation of reproduction and feeding and may play a role in predator-induced alterations of these behaviors.

3.4. Melanocortins

The anterior (AL) and intermediate (IL) pituitary lobes produce and release a variety of peptides derived from post-translational processing of pro-opiomelanocortin (POMC). ACTH, β-lipotropin (β-LPH) and β-endorphin are major end products of POMC processing in the AL, whereas the major end products of POMC cleavage in the IL are N-acetylated $ACTH_{1-13}$ amide (α-MSH) and shorter and N-acetylated forms of β-endorphin in most vertebrates that have been examined (Mains and Eipper, 1979; Eipper and Mains, 1980; Dores et al., 1989; Lancha et al., 1994; Norris and Carr, 2013). While these peptides may enter the peripheral circulation after release, and have well-documented central effects after peripheral administration (Banks and Kastin, 1995), their precise role in regulation of reproduction and feeding is unclear. POMC is produced by neurons in just 1–2 brain areas across vertebrate taxa (see Venkatesan and Carr, 2001 for a review), and it is the population of POMC neurons in the mammalian arcuate nucleus that have been reported to mediate the inhibitory actions of leptin on satiety (Cone, 2005). Pituitary corticotropes are believed to be involved in inhibiting gonadotropin production, although through secretion of urocortin-2, not through the secretion of POMC-related peptides (Kageyama, 2013).

3.5. Glucocorticoids

CORT inhibits reproduction at several levels including upstream regulators of GnRH secretion (Kirby et al., 2009; Gojska and Belsham, 2014; Son et al., 2014), GnRH neuronal development and migration (Lim et al., 2014a,b), GnRH synthesis and secretion (Ahima and Harlan, 1992; De Franco et al., 1994; Attardi et al., 1997; Calogero et al., 1999; Dondi et al., 2005; Gore et al., 2006; Wagenmaker et al., 2009b; Li et al., 2010), pituitary gonadotropes (Kamel and Kubajak, 1987; Breen et al., 2008; Pierce et al., 2009; Wagenmaker et al., 2009a; Breen and Mellon, 2014), and gonad steroidogenesis (Bernier et al., 1984; Orr and Mann, 1992; Schultz et al., 1993; Silva et al., 2010) across all taxa (see Fuzzen et al., 2011; Carr, 2011; Tokarz and Summers, 2011; Breuner, 2011; and Uphouse, 2011 for reviews on fish, amphibians, reptiles, birds, and mammals, respectively). While preclinical studies using laboratory rodents seem to emphasize these inhibitory effects of CORT on reproduction (Geraghty and Kaufer, 2015), an examination of non-model vertebrates species seems to reflect a wide variety of effects that can vary depending upon life history patterns, developmental stage, duration of exposure, nutritional status, seasonal status, and sex (Breuner, 2011; Breuner et al., 2008; Bonier et al., 2009; Carr, 2011; Fuzzen et al., 2011; Romero, 2002; Tokarz and Summers, 2011; Uphouse, 2011; Woodley, 2011). The precise role of predator-induced elevations in blood CORT on various aspects of reproduction has yet to be tested (see ahead in Section 7), but it is certainly plausible that CORT may mediate the inhibitory effects of predator cues on courtship and investment in reproduction. CORT rapidly inhibits courtship behavior via actions on spinal pre-motor and motor neurons in newts (Rose et al., 1993, 1998; Moore et al., 1998; Lewis and Rose, 2003; reviewed in Carr, 2011) and decreases female preference for auditory cues in calling green treefrogs (*Hyla cinerea*) (Davis and Leary, 2015).

When considering the effects of CORT on reproduction, it is important to remember that elevated CORT also facilitates certain aspects of reproductive activity, ostensibly because of its role in elevating blood glucose. Elevated CORT levels are associated with amplexus in amphibians (Orchinik et al., 1988; Reedy et al., 2014). Elevated plasma CORT may play an especially important role in supplying energy for calling behavior. Plasma CORT is greater in calling than non-calling male anurans (Mendonca et al., 1985; Leary et al., 2004) and is positively correlated with the rank order of calling effort for a range of anuran species (Emerson and Hess, 2001). To explain the seemingly paradoxical energetic need for CORT during courtship with data showing that CORT can suppress plasma sex steroid levels and courtship, Emerson (Emerson and Hess, 2001) proposed the "Energetic-Hormone Vocalization Model" (reviewed in Carr, 2011), although attempts to validate this model in natural populations of calling amphibians have failed to date (Leary et al., 2015).

As with reproduction, it is difficult to generalize regarding the influence of CORT on foraging across all vertebrate taxa. Administration of CORT has been reported to increase foraging across taxa in rodents (Challet et al., 1995), birds (Astheimer et al., 1992; Breuner and Wingfield, 2000; Kitaysky et al., 2001; Pravosudov, 2003), amphibians (Crespi and Denver, 2004) and fishes (Bernier et al., 2004). However the opposite trend has been observed in some studies. Madison et al. (2015) found that rainbow trout implanted with cortisol releasing osmotic minipumps decreased food intake over a period of 34 d. Likewise, administration of CORT to male penguins (*Pygoscelis adeliae*) reduced foraging (Thierry et al., 2014). In garter snakes (*Thamnophis sirtalis parietalis*) administration of the 11β-hydroxylase inhibitor metyrapone increased the preference for food cues over reproduction cues, consistent with a hypothesis that low CORT is required to switch from seasonal reproductive to foraging modes in this species (Lutterschmidt and Maine, 2014). CORT also has been implicated in freezing behavior (see Section 2) through rapid actions mediated via membrane receptors in the medial prefrontal cortex (Reis et al., 2015). A single dose of CORT has been reported to reduce prey capture behavior in bufonids (Carpenter and Carr, 1996). With respect to vigilance behavior, which is sometimes, but not always (Lima and Bednekoff, 1999), incompatible with foraging, fecal glucocorticoid metabolites (FGMs) are positively correlated with

vigilance levels and CORT administration increases the latency to resume foraging in meerkats after hearing a conspecific alarm call (Voellmy et al., 2014), confirming earlier work reporting that CORT administration reduced foraging in female meerkats (Santema et al., 2013). How these different effects of CORT on foraging relate to differences in plasma CORT levels (e.g., baseline vs. post-stress concentrations), MCBC (max CORT binding capacity), and CORT clearance is an interesting area for future research.

4. HPA/HPI axis response to predators

4.1. Detection of predator cues and getting the signal to the hypothalamus

Quite often predator cues are lumped into the same category with other known 'stressors', with little regard for the pathways linking various sensory modalities to CRF neurons in the hypothalamus, or whether the states of stress, fear, or anxiety are the end result of predator exposure. From the point of view of understanding the downstream physiological consequences of CORT release such considerations may seem unimportant. However, an understanding of how hard-wired connections between sensory structures and the hypothalamus are organized is critical for understanding the neuroendocrine circuits involved in responding to predation, and the potential consequences of CORT action on those circuits (see Section 6 ahead).

Prey can detect predators via one or more sensory systems, including olfactory, visual, auditory, and tactile (e.g., lateral line). If the HPA/HPI axis is involved in the response to predators, then honest predator signals of multiple modalities should all be able to activate the axis (Fig. 1); data seem to support this prediction. The PVN, which houses the hypophysiotropic CRF neurons regulating ACTH secretion, is innervated by the medial amygdala, which in turn is innervated by the main and accessory olfactory systems (Takahashi, 2014). Work in laboratory rodents suggests that multiple parallel pathways for detection of predator odors and activation of the medial amygdala and ventromedial hypothalamus exist (Ferrero et al., 2011; Perez-Gomez et al., 2015). Visual predator cues are conveyed indirectly to the PVN via cortical (retina-lateral geniculate nucleus (LGN)-striate cortex) and subcortical (retina-optic tectum-pulvinar nucleus) pathways which are routed through the central amygdala (CeA) (Carr, 2015) prior to reaching the PVN (Rodrigues et al., 2009). This seems to be the general pattern for tetrapods. In ray finned fishes homologs of the central amygdala and thalamic visual relays (LGN, pulvinar) have not been identified and visual information is relayed to the telencephalon by the preglomerular complex in the posterior tubercle (Carr, 2015). The superior colliculus (optic tectum in non-mammals) is required for processing visual predator cues (Fig. 1), although the relative degree to which cortical and subcortical visual pathways play a role in predator detection in mammals is still a matter of debate (Carr, 2015). However, selective activation of the superior colliculus in mice activates the HPA axis (Wei et al., 2015). Information about audiogenic stressors reaches the PVN via a complex set of connections beginning in the cochlear nucleus and then reaching the medial geniculate nucleus (MGN) in the thalamus, which directs information both to the auditory cortex and the PVN. Lesions of the MGN in rats prevents the rise in CORT and ACTH that normally follows exposure to an audiogenic stressor (Campeau and Watson, 1997). Whether this same pathway is involved in communicating auditory predator cues to the PVN is unknown. While much has been learned about how predators localize prey acoustically, little is known about the mechanisms involved in prey learning to recognize acoustic predator cues, although some degree of certainty exists with respect to the pathway for detecting acoustic stressors (Campeau and Watson, 1997). Understanding how predator cues are registered and processes is important for understanding how predation can influence the HPA/HPI axis and for understanding how these predator-sensitive pathways evolved.

4.2. Assessing HPA/HPI responses to predator cues

We compiled 168 published cases covering data on amphibians, fishes, birds, and mammals in an attempt to systematically assess the impact that predator and predator cues have on HPA/HPI axis activation (Table 1). We included any study that exposed prey animals to some form of predator (e.g., live or stuffed) or predator cue (e.g., odor, auditory, visual) and also measured some aspect of the HPA/HPI axis response. The majority of studies we found measured total CORT, although some measured other HPA/HPI variables, and we noted whether CORT concentration was determined from blood plasma, whole-body tissues, feathers, fecal samples, or as excreted 'CORT' in the tank water. In addition to sensory modality and method of CORT determination, we also noted if data were collected in the laboratory or field, if studied prey animals were male or female, if they were adults or juveniles, and whether predator exposure was acute or chronic, as all of these factors may modulate the HPA/HPI response to predator threat (see Section 5 below). In this section, data are broadly summarized by taxa and we discuss whether predators/predator cues resulted in an increase, decrease, or no change in HPA/HPI axis measures.

As shown in Table 1, the large majority of studies examined report an increase in HPA/HPI activity based on estimates of CORT release, although there are some obvious trends across taxa. The greatest percentage of studies reporting an increase in CORT secretion in response to predator cues, across all ages, was observed in fishes and in mammals. Out of a total of 35 papers on fish representing a total of 16 species, authors in 25 studies (71%) reported that predator cues increased CORT release (Table 1). Similarly, 77% of the mammalian studies reviewed (60 out of 78 studies, across 25 species) reported increased CORT release in response to predator cues, as did 50% (11 out of 22) studies in amphibians. The taxa with the fewest studies reporting an increase in CORT release after predator exposure were reptiles (29% of 7 studies examined) and birds (38% of 25 studies examined).

Two major conclusions can be drawn based upon the striking similarity in the percentage of fish and mammal studies reporting an increase in CORT release upon predator exposure. First, it suggests that such a response may be a basal evolutionary trait, given the persistence of this response for hundreds of millions of years. Secondly, despite the variety of CORT extraction and measurement methods employed (whole body extraction, water extraction, plasma sampling), the same qualitative conclusion is reached. The fact that several of the mammalian studies also report elevated plasma ACTH levels after predator cue exposure (Table 1) suggests that this CORT response is reflective of increased HPA axis activity, although the virtual lack of similar corroboration in non-mammalian groups limits the extrapolation of this to all taxa for the time being.

Obviously not every study has reported an increase in HPA/HPI activity in response to predator cues, with some studies reporting no relationship between predator cue exposure and estimates of CORT release (Table 1). A finding of no change in CORT release in response to predator cue exposure was most common for birds, occurring in well over half (15 out of 26) of the studies examined in this group. It is tempting to rule out a methodological basis for this difference, as the large majority of both mammalian and bird studies examined blood plasma CORT. On possible reason for the lower responses in birds could be the type of experimental design. Out of 26 studies, 15 of them used an indirect measure of

Table 1
Response of the hypothalamus-pituitary-adrenal/interrenal axis to predator cues in various vertebrate taxa.

Prey species	Sex	Age	Predator	Cue	Cue exposure	Lab/field	ΔCORT	Matrix	Other HPA markers	Reference
Fish										
Batrachoididae										
Opsanus beta (Gulf toadfish)	M	?	*Tursiops truncatus* (bottlenose dolphin) sounds	A	Acute	Field	Increase	P	–	Remage-Healey et al. (2006)
Cichlidae										
Oreochromis niloticus (Nile tilapia)	?	J	Conspecific skin extract	O	Acute	Lab	Increase	P	–	Sanches et al. (2015)
Cyprinidae										
Carassius auratus (goldfish)	M&F	J	*Lepomis macrochirus* (Bluegills)	M	Acute	Lab	Increase	P	–	Kagawa and Mugiya (2000)
C. auratus	M&F	J	*Lepomis macrochirus* (Bluegills) water	O	Acute	Lab	No change	P	–	Kagawa and Mugiya (2000)
C. auratus	?	J	*Lepomis macrochirus* (Bluegills)	M	Acute	Lab	Increase	P	–	Kagawa and Mugiya (2002)
Cyprinus carpio (Carp)	?	A	*Lutra lutra* (Eurasian otter)	M	Chronic	Mesocosm	Increase	P	–	Poledník et al. (2008)
Danio rerio (zebrafish)	?	?	*Parachromis managuensis* (Cichlid)	V	Acute	Lab	Increase	WB	–	Barcellos et al. (2007)
D. rerio	?	?	*Parachromis managuensis* (Cichlid)	M	Acute	Lab	Increase	WB	–	Barcellos et al. (2007)
D. rerio	?	?	*Parachromis managuensis* (Cichlid)	M	Acute (after previous predator experience)	Lab	Increase	WB	–	Barcellos et al. (2010)
D. rerio	?	?	*Parachromis managuensis* (Cichlid)	V	Acute (after previous predator experience)	Lab	No change	WB	–	Barcellos et al. (2010)
D. rerio	M&F	?	*Astronotus ocellatus* (tiger Oscar)	M	Acute	Lab	Increase	WB	–	Barcellos et al. (2014)
D. rerio	M&F	?	*Astronotus ocellatus* (tiger Oscar)	V	Acute	Lab	No change	WB	–	Barcellos et al. (2014)
D. rerio	M&F	?	*Astronotus ocellatus* (tiger Oscar)	O + A	Acute	Lab	No change	WB	–	Barcellos et al. (2014)
D. rerio	M&F	?	Chemical cues from conspecifics exposed to *Astronotus ocellatus* (tiger Oscar)	O	Acute	Lab	Increase	WB	–	Barcellos et al. (2014)
D. rerio	M&F	A	Chemical cues from dead conspecifics	O	Acute	Lab	Increase	WB	–	Oliveira et al. (2014)
Fundulidae										
Fundulus majalis (longnose killifish)	?	?	*Cynoscion arenarius* (Sand seatrout)	V	Acute	Lab	Increase	P	–	Woodley and Peterson (2003)
Gasterosteidae										
Gasterosteus aculeatus (threespined sticklebacks)	F	?	Model heron	V	Acute	Lab	Increase	TW	–	Furtbauer et al. (2015a)
G. aculeatus	F	A	Model heron	V	Acute	Lab	Increase	TW	–	Furtbauer et al. (2015b)
G. aculeatus	M&F	A	Dead frozen pike on a stick	M	Acute	Lab	Increase	P	–	Mommer and Bell (2013)
G. aculeatus	M&F	J	Chronic exposure of mother to model pike	V	Chronic	Lab	Increase in laid eggs	E	–	Giesing et al. (2011)
G. aculeatus	?	J (sub adult)	*Exos Lucius* (pike)	M	Acute	Lab	Increase	WB	Increase whole-brain NE	Bell et al. (2007)
Gobiidae										

(*continued on next page*)

Table 1 (*continued*)

Prey species	Sex	Age	Predator	Cue	Cue exposure	Lab/field	ΔCORT	Matrix	Other HPA markers	Reference
Elacatinus evelynae (cleaning gobies)	?	?	Either *Cephalopholis cruentata* (Graysby grouper) or *Gymnothoras moringa* (Spotted Moray)	V	Acute	Lab	Increase	TW	–	Soares et al. (2012)
Pleuronectidae										
Pseudopleuronectes americanus (winter flounder)	?	J	*Carcinus maenus* (green crab)	M	Acute (24 h)	Lab	No change	WB	–	Breves and Specker (2005)
P. americanus	?	J	*Crangon septemspinosa* (sand shrimp)	M	Acute (24 h)	Lab	Increase	WB	–	Breves and Specker (2005)
P. americanus	?	J	*Paralichthys dentatus* (summer flounder)	M	Acute (24 h)	Lab	Increase	WB	–	Breves and Specker (2005)
Poeciliidae										
Brachyrhaphis episcopi	M&F	?	None; fish from high or low predation streams	M	Chronic	Lab	Increase (decreased post-open field test release for high predation)	TW	–	Archard et al. (2012)
Gambusia geiseri (Largespring mosquitofish)	?	?	*Lepomis cyanellus* (Green sunfish)	V + O	Acute	Lab	No change	TW	–	Blake et al. (2015)
Poecilia reticulata (Trinidadian guppy)	M	A	None, high or low predation streams	M	Chronic	Lab	Decrease (baseline in high-predator populations)	TW	–	Fischer et al. (2014)
P. reticulata	M	A	*Crenicichla frenata* (Pike cichlid)	O	Chronic	Lab	Decrease	TW	–	Fischer et al. (2014)
Xiphophorus birchmanni (sheepshead swordtail)	M	A	Decoy heron	V	Acute	Lab	Decrease	TW	–	Boulton et al. (2015)
Pomaceotridae										
Pomacentrus ambionensis (tropical damselfish)	F	A	None; variation with density of egg predators	M	Chronic	Field	Increase with increasing predator density	Ovarian tissue	–	McCormick (1998)
P. ambionensis	F	A	Manipulated predation pressure; Addition of 3–4 egg predators to reef	M	Chronic	Field	Increase	Ovarian tissue	–	McCormick (2009)
Salmonidae										
Oncorhynchus kisutch (coho salmon)	?	J	*Ptychocheilus oregonensis* (northern squawfish)	O	Acute	Lab	Increase	P	–	Rehnberg and Schreck (1987)
O. kisutch	?	J	*Catostomus macrocheilus* (largescale sucker)	O	Acute	Lab	Increase	P	–	Rehnberg and Schreck (1987)
Oncorhynchus mykiss (rainbow trout)	?	?	Plastic heron and trout alarm substance	V + O	Chronic	Lab	No change	P	Increase whole brain *crf* mRNA	Thomson et al. (2012a)
Amphibians										
Pelobatidae										
Pelobates cultripes (Western spadefoot toad)	?	J	*Dytiscus circumflexus* (dytiscid beetle) larvae	M	Chronic	Lab	Decrease	WB	–	Burraco et al. (2013)
Plethodontidae										
Desmognathus ochrophaeus (dusky salamanders)	M	A	*Gyrinophilus porphyriticus* (spring salamander) kairomones	O	Acute	Lab	No change	P	–	Fonner and Woodley (2015)
D. ochrophaeus	M	A	*Gyrinophilus porphyriticus* (spring salamander)	O	Chronic	Lab	No change	P	–	Fonner and Woodley (2015)

Table 1 (*continued*)

Prey species	Sex	Age	Predator	Cue	Cue exposure	Lab/field	ΔCORT	Matrix	Other HPA markers	Reference
			kairomones							
D. ochrophaeus	F	A	*Gyrinophilus porphyriticus* (spring salamander) kairomones	O	Acute	Lab	No change	P	–	Fonner and Woodley (2015)
D. ochrophaeus	F	A	*Gyrinophilus porphyriticus* (spring salamander) kairomones	O	Chronic	Lab	No change	P	–	Fonner and Woodley (2015)
Eurycea nana (San Marcos salamander)	?	?	*Micropterus salmoides* (largemouth bass) kairomones	O	Acute	Lab	Increase	TW	–	Davis and Gabor, 2015
E. nana	?	?	*Lepomis auritus* (redbreast sunfish) kairomones	O	Acute	Lab	Increase	TW	–	Davis and Gabor (2015)
Ranidae										
Platymantis vitiana (Fijian ground frog)	M	A	*Bufo marinus* (cane toad)	M	Acute	Lab	Increase	UGM	–	Narayan et al. (2013)
P. vitiana	F	A	*Bufo marinus* (cane toad)	M	Chronic	Field/ Mesocosm	Increase	UGM	–	Narayan et al. (2015)
P. vitiana	M	A	*Bufo marinus* (cane toad)	M	Chronic	Field/ Mesocosm	Increase	UGM	–	Narayan et al. (2015)
Rana pipiens (leopard frog)	M&F	J	*Aeshna* nymphs (dragonfly)	M	Chronic	Lab	Indirect	–		Hossie et al. (2010)
R. clamitans (green frog)	M&F	J	Conspecific chemical alarm cue	O	Acute	Lab	Decrease	WB	–	Fraker et al. (2009)
R. clamitans	M&F	J	*Anax spp.* (dragonfly) water	O	Acute	Lab	Increase	WB	–	Marino et al. (2014)
R. sylavtica (wood frog)	M&F	J	*Anax* larvae (dragonfly)	M	Acute	Field/ Mesocosm	Decrease	WB	–	Middlemis Maher et al. (2013)
R. sylvatica	M&F	J	*Anax* larvae (dragonfly)	M	Chronic	Field/ Mesocosm	Increase	WB	–	Middlemis Maher et al. (2013)
R. sylvatica	M&F	J	None; tadpoles from high or low predation ponds	M	Chronic	Field/ Mesocosm	Increase	WB	–	Middlemis Maher et al. (2013)
R. sylvatica	M&F	J	*Anax spp.* (dragonfly) water	O	Acute	Lab	Increase	WB	–	Marino et al. (2014)
R. sylvatica	M&F	J	Conspecific chemical alarm cue	O	Acute	Lab	Decrease	WB	–	Fraker et al. (2009)
R. sylvatica	M&F	J	Water from tadpole-fed beetle and dragonfly larvae	O	Chronic	Lab/ Mesocosm	No change	WB	–	Reeve et al. (2013)
R. temporaria (common frog)	?	J	*Aeshna* larvae (dragonfly)	M	Acute (24 h)	Lab	Increase	WB	–	Dahl et al. (2012)
R. temporaria	?	J	*Aeshna* larvae (dragonfly)	M	Chronic	Lab	No change	WB	–	Dahl et al. (2012)
Salamandridae										
Taricha granulosa (rough-skinned newt)	F	A	Tapping back with forceps	T	Acute	Lab	Increase	P	–	Neuman-Lee et al. (2015)
Reptiles										
Iguanidae										
Amblyrhynchus cristatus (marine iguana)	M	A	Human chasing	M	Acute	Field	Increase (only in those animals from predator-present islands)	P	–	Rodl et al. (2007)
A. cristatus	M	A	None; High predation vs. low predation islands	M	Chronic	Field	Decrease (baseline higher on island with no predation)	P	–	Rodl et al. (2007)
A. cristatus	M&F	A	None; High predation vs. low predation islands	M	Chronic	Field	No change	P	Increase[a]	Berger et al. (2007)
A. cristatus	M&F	J	None; High predation vs. low predation islands	M	Chronic	Field	No change	P	Increase[a]	Berger et al. (2007)

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Table 1 (*continued*)

Prey species	Sex	Age	Predator	Cue	Cue exposure	Lab/field	ΔCORT	Matrix	Other HPA markers	Reference
Phrynosomatidae										
Urosaurus ornatus (tree lizard)	M	A	*Crotaphytus nebrius* (collared lizard)	M	Acute	Mesocosm	No change	P	–	Thaker et al. (2009a)
U. ornatus	M	A	*Crotaphytus nebrius* (collared lizard)	M	Acute	Field	Increase	P	–	Thaker et al. (2009b)
Varanidae										
Varanus varius (lace monitor)	?	?	*Vulpes vulpes* (red fox)	M	Chronic	Field	No change (baseline CORT)	P	–	Anson et al., 2013
Birds										
Anatidae										
Somateria mollissima (Eider)	F	A	None; females from high and low predation islands	M	Chronic	Field	Increase, SC	S	–	Jaatinen et al. (2014)
Columbidae										
Columba livia (rock pigeon)	?	A	Various live raptors	M	Acute	Mesocosm	Increase	P	–	Pakkala et al. (2013)
Emberizidae										
Junco hyemalis dorsalis (Grey-headed juncos)	M	A	Manipulated predation pressure	M	Chronic	Field	No change (baseline CORT only)	P	–	Fontaine et al. (2011)
J. hyemalis dorsalis	F	A	Manipulated predation pressure	M	Chronic	Field	No change (baseline CORT only)	P	–	Fontaine et al. (2011)
Melospiza melodia (Song sparrow)	M	A	Manipulated predation pressure	M	Chronic	Field	Increase	P	No change CBG binding capacity; increase in free CORT	Clinchy et al. (2011b)
M. melodia	F	A	Manipulated predation pressure	M	Chronic	Field	No change	P	Decrease CBG binding capacity; increase in free CORT	Clinchy et al. (2011b)
M. melodia	M&F	A	Manipulated predation pressure	M	Chronic	Field	Increase	P	–	Clinchy et al. (2004)
M. melodia	M	A	None; high and low predation populations	M	Chronic	Field	No change	P	Decrease plasma DHEA	Newman et al. (2013)
M. melodia	F	A	None; high and low predation populations	M	Chronic	Field	No change	P	No difference in plasma DHEA	Newman et al. (2013)
M. melodia	F	A	Simulated predation pressure via egg removal	M	Chronic	Field	Increase (baseline CORT in females with higher nest predation)	P	Increase plasma CBG	Travers et al. (2010)
Zonotrichia leucophrys (white-crowned sparrow)	?	J	Parental alarm calls	A	Acute	Field	No change	P	–	Rivers et al. (2011)
Falconidae										
Falco sparverius (American kestrel)	M&F	J	Adult alarm calls	A	Acute	Field	No change	P	–	Dufty and Crandall (2005)
Fringillidae										
Carduelis chloris (Greenfinch)	M	J&A	Photo of an owl	V	Acute	Lab	No change	F	–	Sepp et al. (2014)
C. chloris	M	J&A	Photo of an owl face	V	Acute	Lab	No change	F	–	Meitern et al. (2013)
Muscicapidae										
Ficedula albicollis (collared flycatchers)	M	A	Human approach	M	Acute	Field	No change	FGM	–	Garamszegi et al. (2012)
F. hypoleuca (pied flycatcher)	M&F	A	Stuffed sparrowhawk	V	Acute	Field	Increase	P	–	Tilgar et al. (2010)
Saxicola torquata axillaris (tropical stonechat)	M	A	Territories with and without a (shrike) predator	M	Chronic	Field	Increase (baseline in territories with shrike	P	–	Scheuerlein et al. (2001)

Table 1 (*continued*)

Prey species	Sex	Age	Predator	Cue	Cue exposure	Lab/field	ΔCORT	Matrix	Other HPA markers	Reference
							when fledged young present)			
S. torquata axillaris	F	A	Territories with and without a (shrike) predator	M	Chronic	Field	No change	P	–	Scheuerlein et al. (2001)
S. torquata rubicola (European stonechats)	M&F	A	*Strix aluco* (tawny owl)	M	Acute	Lab	Increase	P	–	Canoine et al. (2002)
Paridae										
Parus major (great tit)	M&F	A	Stuffed Tengmalm's owl	V	Acute	Lab	Increase	P	–	Cockrem and Silverin (2002)
P. major	F	A	Stuffed sparrowhawk	V	Acute	Field	Increase in eggs laid by exposed female	E	–	Pitk et al., 2012
Turdidae										
Turdus merula (common blackbird)	M&F	J	*Pica pica* (magpie) calls	A	Acute	Field	Decrease (in baseline cort; no change in post-exposure CORT	P	–	Ibanez-Alamo et al. (2011)
Vireonidae										
Vireo atricapilla (black-capped vireo)	M&F	A	Decoy crow	V	Acute	Field	No change	P	–	Butler et al. (2009)
V. atricapilla	M&F	A	Multiple offspring-directed threats	M	Chronic	Field	No change	P	–	Butler et al. (2009)
Vireo griseus (white-eyed vireo)	M&F	A	Decoy crow	V	Acute	Field	No change	P	–	Butler et al. (2009)
V. griseus	M&F	A	Multiple offspring-directed threats	M	Chronic	Field	No change	P	–	Butler et al. (2009)
Mammals										
Callitrichidae										
Callithrix jacchus (Common marmoset)	F	A	Simulated hawk	M	Acute	Lab	Increase	P	Increase plasma ACTH	Saltzman and Abbott (2011)
Cerrapithecidae										
Lophocebus albigena (Grey-cheeked mangabey)	M	A	Conspecific alarm call; presence of crowned eagle	M	Acute	Field	Increase	FGM	Increase only in highest ranking males chasing eagle	Arlet and Isbell (2009)
Cervidae										
Cervus elaphus (Elk)	?	A	Wolves	M	Chronic	Field	No change	FGM	–	Creel et al. (2009)
Cricetidae										
Cricetulus triton (Ratlike hamster)	M	A	Anal gland secretions of Siberian weasel smeared on the oronasal groove	O	Chronic	Lab	Increase	P	Increase adrenal weight	Zhang et al. (2003)
Clethrionomys glareolus (Bank vole)	F	A&J	Soiled weasel bedding	O	Acute	Lab	No change	FGM	–	Ylönen et al. (2006)
C. glareolus	F	A&J	Soiled weasel bedding	O	Chronic	Lab	No change	FGM	–	Ylönen et al. (2006)
C. glareolus	M	A	Weasel feces	O	Chronic	Lab	Increase	FGM	–	Tidhar et al. (2007)
C. glareolus	F	A	Weasel feces	O	Chronic	Lab	Increase	FGM	–	Tidhar et al. (2007)
Lasiopodomys brandtii (Brandt's voles)	M&F	A	*Felis catus* (domestic cat) feces	O	Acute	Lab	Increase	P	Increase plasma ACTH	Hegab et al. (2014)
L. brandtii	M&F	A	*Mustela sibirica* (weasel) feces	O	Acute	Lab	Increase	P	Increase plasma ACTH	Hegab et al. (2014)
L. brandtii	M&F	A	*Xenopeltis hainanensis* (snake) feces	O	Acute	Lab	Increase	P	Increase plasma ACTH	Hegab et al. (2014)
Mesocricetus auratus (Golden hamster)	M	A	Anal gland secretions of Siberian weasel smeared on smeared at the oronasal groove	O	Chronic	Lab	Increase	P	Increase adrenal weight	Zhang et al. (2003)

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Table 1 (*continued*)

Prey species	Sex	Age	Predator	Cue	Cue exposure	Lab/field	ΔCORT	Matrix	Other HPA markers	Reference
Microtus arvalis (Common voles)	F	A	*Crocidura russula* (greater white-toothed shrew)	M	Chronic	Mesocosm	Increase (when measured at parturition)	FGM	–	Liesenjohann et al. (2013)
Microtus pennsylvanicus (Meadow vole)	M	A	Soiled weasel bedding	O	Acute	Lab	No change	P	–	Fletcher and Boonstra (2006)
Microtus socialis (Social voles)	M	A	*Tyto alba* (barn owl)	M	Acute	Lab	Increase	P	–	Bodek and Eilam (2015)
M. socialis	F	A	*Tyto alba* (barn owl)	M	Acute	Lab	Increase	P	–	Bodek and Eilam (2015)
M. socialis	M	A	*Strix aluco* (tawny owl) calls	A	Acute	Lab	Increase	P	–	Eilam et al. (1999)
Peromyscus californicus (California mouse)	M	A	Bobcat urine	O	Acute	Lab	Increase	P	–	Harris and Saltzman (2013b)
P. californicus	M	A	Bobcat urine	O	Acute	Lab	Increase	P	–	Harris and Saltzman (2013a)
P. californicus	F	A	Bobcat urine	O	Acute	Lab	Increase	P	–	Harris and Saltzman (2013a)
P. californicus	M&F	A	Fox urine	O	Acute	Lab	Increase	P	–	Harris et al. (2012)
P. californicus	M&F	J	Bobcat urine	O	Acute	Lab	Increase	P	–	Harris et al. (2013)
Lemuridae										
Lemur catta (ring-tailed lemur)	F	A	Aerial and ground predator alarm calls	M	Chronic	Field	No change	FGM	–	Pride (2005)
Leporidae										
Lepus americanus (Snowshoe hare)	M&F	A	Aerial predators only	M	Chronic	Field	Increase	P	Increase free cortisol decrease maximum corticosteroid binding capacity (MCBC)	Boonstra et al. (1998)
L. americanus	F	A	Lynx	M	Chronic	Field; Enclosure	Increase	FGM	–	Sheriff et al. (2009)
L. americanus	F	A	Dog	M	Chronic	Field	Increase	FGM	–	Sheriff et al. (2010a)
L. americanus	F	A&J	Lynx	M	Chronic	Field; Enclosure	Increase in parents and offspring	FGM	MCBC	Sheriff et al. (2010b)
Oryctolagus cuniculus (European rabbits)	M&F	A	*Vulpes vulpes* (red fox) feces	O	Chronic	Lab	Increase	P	–	Monclús et al. (2005)
O. cuniculus	M&F	A	*Vulpes vulpes* (red fox) feces	O	Chronic	Lab	Increase	FGM	–	Monclús et al. (2006)
O. cuniculus	?	A	Manipulated predation pressure	M	Chronic	Field	Increase (baseline in populations with higher predation)	FGM	–	Monclús et al. (2009)
Muridae										
Acomys cahirinus (spiny mouse)	M	A	*Strix aluco* (tawny owl) calls	A	Acute	Lab	Increase	P	–	Eilam et al. (1999)
Apodemus agrarius (striped field mouse)	M	A	*Mustela sibirica* (Himalayan weasel)	O	Acute (repeated)	Lab	No change	FGM	No change in adrenal weight	Wang et al. (2011)
A. agrarius	F	A	*Mustela sibirica* (Himalayan weasel) anal gland odor	O	Acute (repeated)	Lab	Increase	FGM	Increase adrenal weight	Wang et al. (2011)
Apodemus sylvaticus (wood mouse)	M&F	A	Owl (*Tyto alba* and *Bubo bubo*) calls	A	Chronic	Lab	Increase	FGM	–	Monarca et al. (2015)
A. sylvaticus	M	A	*Vulpes vulpes* (red fox) feces	O	Chronic	Field	No change	FGM	–	Navarro-Castilla and Barja (2014)
A. sylvaticus	F	A	*Vulpes vulpes* (red fox) feces	O	Chronic	Field	No change	FGM	–	Navarro-Castilla and Barja (2014)
Mus musculus (House mouse)	M&F	A	Cat odor	O	Acute, after exposure to estrous/non-estrous female odors	Lab	Increase	P	–	Kavaliers et al. (2001)
M. musculus	F	A	Predator odor (TMT)	O	Chronic	Lab	Increase (chronic	P	–	Hacquemand et al. (2010)

Table 1 (*continued*)

Prey species	Sex	Age	Predator	Cue	Cue exposure	Lab/field	ΔCORT	Matrix	Other HPA markers	Reference
							exposure beginning postnatally)			
M. musculus	F	A	Live cat (*Felis domesticus*)	M	Acute (24 h)	Lab	Increase	P	–	Liu et al. (2012)
M. musculus	M	A	Live rat	M	Acute	Lab	Increase	P	–	Amaral et al. (2010)
Rattus norvegicus (HL) (Norwegian rat)	M	A	Towel with cat odor	O	Acute	Lab	Increase	P	–	File et al. (1993)
R. norvegicus (LE)	M	A	Live cat	M	Chronic	Lab	Increase (in basal, pre-stressor levels)	P	Increase adrenal weight	Blanchard et al. (1998)
R. norvegicus (LE)	M	J	Soiled cat bedding	O	Acute	Lab	No change	P	No change in *crf* mRNA in PVN	Wiedenmayer et al. (2005)
R. norvegicus (LE)	M	J	Adult male rat	M	Acute	Lab	Increase	P	Increase *crf* mRNA in PVN	Wiedenmayer et al. (2005)
R. norvegicus (SD)	M	?	Ferret odor	O	Acute	Lab	Increase	P	Increase plasma ACTH Increase *cfos* mRNA in PVN	Campeau et al. (2008)
R. norvegicus (SD)	M	?	Ferret odor	O	Chronic	Lab	No change	P	Increase plasma ACTH Increase *cfos* mRNA in PVN	Campeau et al. (2008)
R. norvegicus (LE)	F	A	Cat-soiled cloth	O	Acute	Lab	No change	P	–	Mashoodh et al. (2008)
R. norvegicus (LE)	M	A	Cat-soiled cloth	O	Acute	Lab	Increase	P	–	Mashoodh et al. (2008)
R. norvegicus (LE)	F	A	Cat-soiled cloth	O	Chronic	Lab	No change	P	No change in hippocampal MR and GR mRNA	Mashoodh et al. (2008)
R. norvegicus (LE)	M	A	Cat-soiled cloth	O	Chronic	Lab	Increase	P	No change in hippocampal MR and GR mRNA	Mashoodh et al. (2008)
R. norvegicus (LE)	M	A	*Felis domesticus* (house cat)	M	Acute	Lab	Increase	P	–	Adamec et al. (2006)
R. norvegicus (Hooded rats)	?	A	*Felis domesticus* (cat)	M	Acute	Lab	Increase (sample taken 8 days after 5-min cat exposure)	P	–	Adamec (1997)
R. norvegicus (LE)	M&F	A	2-propylthietane (2-PT; weasel gland secretion	O	Chronic, two exposures over 2 d	Lab	Increase	P	Increase plasma ACTH	Perrot-Sinal et al. (1999)
R. norvegicus (SD)	M	A	Towel with cat odor	O	Acute	Lab	Increase	P	Increase plasma ACTH	Munoz-Abellan et al. (2009)
R. norvegicus (SD)	M	A	Towel with cat odor	O	Acute	Lab	Increase	P	Increase plasma ACTH	Munoz-Abellan et al. (2008)
R. norvegicus (SD)	?	A	Ferret-soiled cloth	O	Acute	Lab	Increase	P	Increase plasma ACTH	Masini et al. (2009)
R. norvegicus (SD)	M	A	Ferret-soiled cloth	O	Acute	Lab	Increase	P	–	Masini et al. (2010)
R. norvegicus (SD)	M	A	Soiled cat litter	O	Acute	Lab	Increase (in baseline 7 days after exposure)	P	–	Kozlovsky et al. (2008)
R. norvegicus (SD)	M	A	Soiled cat litter	O	Acute	Lab	Increase (in baseline 7 days after exposure)	P	–	Kozlovsky et al. (2007)
R. norvegicus (SD)	M	A	Soiled cat litter	O	Acute	Lab	Increase (in baseline 7 days after exposure)	P	–	Kozlovsky et al. (2009)
R. norvegicus (SD)	M	A	*Felis domesticus* (house cat)	M	Acute	Lab	Increase	P	Increase plasma ACTH	Cohen et al. (2003)
R. norvegicus (SD)	M	A	Ferret-soiled cloths	O	Chronic	Lab	Decrease (habituation)	P	No change in *cfos* mRNA in the PVN or plasma ACTH	Weinberg et al. (2009)
R. norvegicus (SD)	M	A	Ferret-soiled cloths	O	Acute	Lab	Increase	P	Increase *cfos* mRNA in the PVN; Inc. plasma ACTH	Weinberg et al., 2009
R. norvegicus (SD)	M	A	Live cat daily for 7 or 14d	M	Chronic	Lab	Increase	P	Increase plasma ACTH Increase *crf* mRNA in PVN	Figueiredo et al. (2003)
R. norvegicus (SD)	M	A	Towels with ferret odor	O	Acute	Lab	Increase	P	Increase plasma ACTH; Increase *fos* mRNA PVN	Masini et al. (2005)
R. norvegicus (SD)	M	A	Paper soaked with TMT 2,5-dihydro-2,4,5-trimethyl thiazoline	O	Acute	Lab	Increase	P	–	Thomas et al. (2006)
R. norvegicus (SD)	M	A	Live ferret	M	Acute	Lab	Increase	P	Increase *crfbp* mRNA	Roseboom et al. (2007)
R. norvegicus (W)	M	A	Cat-soiled cloth	O	Acute	Lab	Increase	P	Increase plasma ACTH	Cohen et al. (2000)
R. norvegicus (W)	?	A	*Felis domesticus* (cat)	M	Acute	Lab	No change (sample taken 8 days after 5-min cat exposure)	P	–	Adamec (1997)
R. norvegicus (W)	M/A	A	Bobcat urine	O	Acute	Lab	Increase	P	Increase plasma ACTH	Whitaker and Gilpin, 2015

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Table 1 (*continued*)

Prey species	Sex	Age	Predator	Cue	Cue exposure	Lab/field	ΔCORT	Matrix	Other HPA markers	Reference
Rhombomys opimus (Great gerbil)	M	J	Frequency of *Varanus griseus caspius* (monitor lizard) colony visits	M	Chronic	Field	Increase	FGM	–	Rogovin et al. (2004)
Pseudocheiridae										
Pseudocheirus peregrinus (ringtail possum)	M&F	A&J	Manipulated predation pressure; *Vulpes vulpes* (red fox)	M	Chronic	Field	No change (baseline cort only)	P	–	Anson et al. (2013)
Sciuridae										
Marmot faviventris (yellow-bellied marmot)	F	A	None; populations with different predation pressure	M	Chronic	Field	Increase (baseline in populations with higher predation)	FGM	–	Monclús et al. (2011)
Spermophilus beldingi (Belding's ground squirrel)	M&F	A	None; high vs. low predation sites	M	Chronic	Field	Decrease (higher in populations with low predation risk)	FGM	–	Mateo (2007)
Spermophilus columbianus (Columbian ground squirrel)	F	A	Dog, once/wk for 8 wk	M	Chronic	Lab	Increase	P	Increase free CORT	Hubbs et al. (2000)
Spermophilus parryii plesius (Arctic ground squirrel)	M	J	None; high vs. low predation site	M	Chronic	Field	Decrease (baseline CORT in high pred. population)	P	CBG lower in high predation population	Hik et al. (2001)
S. parryii plesius	F	J	None; high vs. low predation site	M	Chronic	Field	Decrease (baseline CORT in high pred. population)	P	CBG lower in high predation population	Hik et al. (2001)
S. parryii plesius	M&F	?	Live dog	M	Acute	Field; Lab	Increase	FGM	–	Sheriff et al. (2012)

Prey species, rat strains: HL, hooded Lister; LE, hooded Long Evans; SD, Sprague-Dawley; W, Wistar.
Sex, age class: M = male; F = Female; M&F = measured both but did not separate results by sex; A = adult; J = juvenile; A&J = measured both but did not separate results by age; NA = not listed.
Predator cues: A, auditory; M, multimodal sensory; O, olfactory; T, tactile; V, visual.
Predator cue exposure: acute, less than 24 h; chronic, greater than 24 h.
Indirect: indirect evidence for change in CORT based on use of metyrapone. SC, stress response CORT test.
Matrix: E, Eggs; F, feathers; FGM, fecal glucocorticoid metabolites; NA, not applicable; P, blood plasma; S, serum; TW, tank water; UGM, urinary glucocorticoid metabolites; WB, whole body.
[a] Increased response to restraint, no difference in basal levels.

predators (e.g., adult bird alarm call, comparison of high vs. low predation populations, manipulation of predation pressure). Of the remaining 11 direct predator/cue exposure studies, 5 (45%) found an increase in CORT in response to predators. Moreover, of the 11 studies that used direct predator cues, two did not report a change in *feather* CORT – due to the timecourse of CORT metabolism and excretion, coupled with pronounced circadian changes, using feathers to measure acute CORT response may not be the best method to gauge predator effects. As to what type of biological factors might explain the lack of response observed in the bird studies is not obvious at present, unless one considers that in none of the bird studies were odors used as a predator cue. With the exception of ground dwelling birds such as the Kiwi, it is generally thought that birds do not rely on olfaction for either prey or predator detection. This difference in the ability to detect olfactory predator cues is a major difference between birds and fishes, larval amphibians, and mammals.

Few of the 168 cases examined across all taxa reported a decline in plasma CORT in response to predator cue exposure, ranging from 18% in amphibians to a high of 8% in non-amphibian groups. A decrease in CORT after predator exposure is not obviously associated with the whole body extraction method that predominates in larval amphibian studies (Table 1), as it is not a trend in the fish studies using whole body CORT measurements (Table 1). Although many of the amphibian studies reporting a decrease in whole body CORT after predator exposure used larvae, similar trends were not apparent in studies in other taxa focusing on juvenile animals (Table 1). In amphibians the initial decrease in whole-body CORT content after predator cue exposure has been hypothesized to facilitate the expression of antipredator behavior in tadpoles (Fraker et al., 2009).

5. Addressing the variability in the HPA/HPI axis response to predator cues

We set out to test the hypothesis that HPA/HPI axis-induced inhibition of feeding and reproduction evolved as part of a predator defense. To do so we used the literature to discuss three main predictions, 1) that exposure to predators, or predator cues, decreases reproductive and feeding behavior and reproductive investment, 2) that exposure to predators, or predator cues, activates the HPA/HPI axis, and 3) that predator-induced activation of the HPA/HPI axis plays a direct role in foraging and reproduction trade-offs. Prediction 1 has been supported numerous times across a variety of taxa as documented in Section 2. After compiling 168 cases which measured the HPA/HPI axis response to predator cues, we found equivocal support for Prediction 2 (Section 4.2 above). In other words, exposure to predators, or predator cues, does not always elicit an HPA/HPI axis response. This finding suggests that HPA/HPI axis activation is not necessary for a response to predators in all cases, but it does seem that the HPA/HPI axis can play a role as some as 108 out of 168 (64%) of studies showed clear HPA/HPI activation. These results mirror equivocal findings of inconsistent antipredator defensive behavior and predator avoidance mechanisms detailed in the ethology and ecological literature (see Kavaliers and Choleris, 2001) and suggest that under certain scenarios the behavioral and (stress) physiological responses to predators are uncoupled or dissociated (Müller et al., 2006). The apparent dissociation of the behavioral and neuroendocrine response to predators leads to an interesting question: Under what specific conditions, then, would we expect the HPA/HPI axis to be associated in the response to predators? Kavaliers and Choleris (2001) proposed several scenarios that should moderate prey behavioral response to a predator. Here, we explore some of these, as well as some additional scenarios with specific reference to the HPA/HPI axis response, and in Section 6 we propose a mechanism by which predation (and/or stress) history may shape future responses to predator cues.

5.1. Acute vs chronic predator threat

The chronic stress hypothesis (Hik, 1995; Boonstra et al., 1998), also known as the predation stress hypothesis (Creel et al., 2009), posits that long-term or chronic exposure to predators should result in (chronically) increased CORT which in turn interrupts foraging and reproduction (Creel et al., 2009). Thus, within this framework, chronic as opposed to acute predator exposure is predicted to play a larger role in the HPA/HPI axis-induced disruption of feeding and breeding. But, given that short-term HPA/HPI activation can be beneficial (see below), coupled with the fact that the HPA/HPI axis is predicted to mediate physiological and behavioral changes induced by predators, acute exposure to predators should, in theory, still result in increased CORT. Thus, we have examined how chronic versus acute predator cue exposure affects the HPA/HPI axis in Table 1. Of the 99 studies examining the HPA/HPI axis response to acute predator cues in Table 1, 72 (73%) reported an increase in HPA/HPI axis activity whereas only 36 out of 68 (52%) studies using chronic predator cue exposure reported the same. These findings are consistent with the idea that acute exposure to predator cues is more likely to activate the HPA/HPI axis than chronic predator exposure, possibly as a result of habituation (see ahead).

With rare exceptions that we know of (e.g., Trinidadian guppies, Galapagos iguanas), animals have evolved with predation threat. This relationship is, in part, the basis for hypotheses addressing whether presumable chronic activation of the HPA/HPI axis in response to chronic predator cues is adaptive (the chronic stress hypothesis, Clinchy et al., 2004; BSH, Ylönen, 1994; Ruxton and Lima, 1997). Short-term, rapid activation of the HPA/HPI axis can, in theory, increase survival-related behaviors and processes, and is thus thought to be adaptive (see Carr and Summers, 2002; Greenberg et al., 2002; Wingfield and Sapolsky, 2003; de Kloet et al., 2005; discussed in Breuner et al., 2008), although there is a paucity of empirical data supporting a causal link between acute HPA/HPI axis activation and survival and/or fitness (Breuner et al., 2008). Long-term or chronic activation of the HPA/HPI axis, however, has primarily been viewed as deleterious as prolonged CORT exposure can detrimentally affect reproduction, immune function, cognition, and metabolism in a variety of species (see Sapolsky et al., 2000; Wingfield and Sapolsky, 2003; McEwen, 1998a,b), but long-term studies on a relationship between elevated CORT and mortality have yielded equivocal findings in humans (Whitehall II studies summarized by Kumari et al., 2011).

Importantly, chronic activation of the sensory pathways that ultimately inform the HPA/HPI axis about predators may result in classical habituation of neuronal pathways and behavioral responses. While in some uses the 'chronic stress hypothesis' may presume that chronic activation of the HPA/HPI axis occurs in response to chronic predation (e.g., studies in hares, see Section 7.1), to our knowledge such a relationship has not been demonstrated across vertebrate species. In fact, it is well known in the mammalian literature that chronic exposure to the same stressor can lead to habituation (Natelson et al., 1988; De Boer et al., 1990 or 'adaptation, as some argue, Rabasa et al., 2015) of the HPA axis and is the basis for using chronic variable stress paradigms (de Jong et al., 2013; Harris et al., 2013).

5.2. Free vs. Bound Glucocorticoid Measures

Several authors have argued that measuring only total circulating or excreted glucocorticoid metabolites is not sufficient for

determining HPA/HPI axis function (see Breuner et al., 2013; Goymann, 2012; Sheriff et al., 2010a), but, despite this, total and excreted CORT are still the main dependent variables in multiple studies and thus the majority of what we know about HPA/HPI axis function on a comparative level comes from assessing studies using these measures. The data we have compiled are no different and two studies measured CBG and free CORT. Thus, we do not have enough data to systematically discuss free vs. total CORT; however, the distinction may be informative and could provide valuable information in future studies.

The role of CBG in the HPA/HPI axis response has been best characterized in birds (Malisch and Breuner, 2010) and may be more important in this taxon as compared to others, however, data on the role of CBG in other taxa is no where near as extensive as the collection of evidence from avian species so it is difficult to conclude this for sure. Data from stonechats and song sparrows (see Section 7.3 below) do suggest that measuring total CORT alone is not sufficient, at least in these species.

Two general hypotheses exist to explain the role of free (not bound to CBG) vs. bound (attached to CBG) vs. total CORT (free + bound). The Free Hormone Hypothesis suggests that only non-bound, free CORT is available to enter cells and is thus the only biologically active form, making measures of total CORT problematic because they overestimate CORT levels and miss subtle changes related to free hormone levels (Malisch and Breuner, 2010; Breuner et al., 2013). The Reservoir Hypothesis states that while free hormone is the only biologically active form, CORT bound to CBG is not cleared by the liver and is thus available as a reservoir for a longer-lasting or localized CORT response (Malisch and Breuner, 2010; Breuner et al., 2013). This hypothesis suggests that total CORT measures may be beneficial as they measure CORT that is currently available as well as CORT that could be available over the course of the stressor (Schoech et al., 2013), but measuring total CORT can still miss important changes as CBG and CORT concentrations can change independently of one another and on different time courses (Breuner et al., 2013).

The debate over measuring free vs. total CORT and the exact role of tissue and plasma CBG is still ongoing and the details surrounding this area are beyond the scope of this review, please see excellent papers by Breuner, Schoech, and colleagues for a thourough discussion (e.g., Breuner et al., 2013; Schoech et al., 2013; Breuner and Orchinik, 2002; Malisch and Breuner, 2010).

5.3. Life history stages and sex

Predation risk and the behavioral and physiological responses to predators likely change with sex, reproductive state, developmental stage, and season and thus each of those factors may moderate the HPA/HPI axis response to predation. A full review of this topic is outside the scope of this paper, but below we provide two examples that we can address with data from Table 1 of how sex and life-history stage could impact HPA/HPI function in response to a predator.

Females are predicted to be more sensitive to predation (see discussion in Kavaliers and Choleris, 2001; Apfelbach et al., 2005; Klein et al., 1994). Sex differences, likely due to HPG axis interaction and reproductive state, in HPA/HPI response have been noted (Viau, 2002; Seale et al., 2004; Kudielka and Kirschbaum, 2005) and could impact response to predators. For example, male mice exposed the scent of a novel estrus female showed a decreased CORT response to predator urine (Kavaliers et al., 2001), and in California mice, virgin males (but not paired males or first-time fathers) tended to increase CORT response to predator urine over trials (Chauke et al., 2011), suggesting that reproductive status can impact the HPA response to predators. We do not have information on reproductive status on animals in Table 1, but we do have data on sex. Based on literature, females may display an enhanced HPA/HPI axis response to predators. When we compare male vs. female data from Table 1 we find that of the 58 cases on males, 39 (67%) found an increase in CORT compared with 62% (22 out of 35) of studies finding an increase in females (studies that lumped males and females for analysis were excluded from the averages). Thus, we did not find that females were more responsive than males.

Age also has well-documented impacts on HPA/HPI axis function and, in general, older individuals are thought to have more responsive axes (Otte et al., 2005). Additionally, young animals often have a period of stress non-responsiveness (Sapolsky and Meaney, 1986; Rensel et al., 2010; Ibanez-Alamo et al., 2011). Thus, we might expect adults to have a more pronounced HPA/HPI axis response to predators as compared to young/juvenile animals. Comparison of adult vs. juvenile data from Table 1 shows that of the studies that were conducted on adults 77 out of 112 (69%) found an increase in CORT, whereas only 44% (17 out of 39) studies in juveniles reported an increase in CORT. Here, it does appear that adults are more responsive than are juveniles.

5.4. Interindividual variation in HPA/HPI axis response

The concept of behavioral syndroms suggests interindividual variability is important and that organisms have ‘personalities’ or that they display sets of correlated behaviors over multiple scenarios (Sih et al., 2004), including those displayed in response to predators (Bell and Sih, 2007). It is predicted that animals should err on the side of too cautious as opposed to too bold, as bold animals are likely more at risk for succumbing to predation. And, if the HPA/HPI axis mediates the response to predators then it would be more advantageous to have a more sensistive (more responsive) axis. However, data from multiple taxa suggest that organism can fall anywhere on a Bold-Shy or Hawk-Dove continuum and this continuum is associated with differences in HPA/HPI function (Korte et al., 2005), stress coping sytles (see Koolhaas et al., 1999) and response to predators and risk taking (see Bell, 2007; Korte et al., 2005). Bold ‘hawks’ are risky, show low anxiety, and have low CORT output whereas shy ‘doves’ are cautious, anxious, and display enhanced CORT output (see Korte et al., 2005). To date, the cause of variation in risk-taking phenotype is unknown, but it is thought to have adaptive value (see Wolf et al., 2007; Korte et al., 2005).

Given that individual differences in HPA/HPI axis response can be meaningful and can be associated with specific predator-related behavioral phenotypes, reporting average CORT data in response to stressors (predators) can be problematic (see Williams, 2008). Unfortunately, we do not have the ability to look at interindividual variation using the data from Table 1, but future studies should look for high and low responders and/or report coefficient of variation data along with mean CORT values (Cockrem, 2013; Cockrem et al., 2009).

5.5. Level of predation risk

Another parameter that may impact the variability in HPA/HPI axis response to predator cues is the level of predation risk encountered by the prey. Due to the energetically costly nature of responding to a predator, prey should assess the risk of predation so as to balance responding with not responding. Being too responsive means loss of energy (from the actual response and from loss of foraging opportunities) and potential loss of reproductive bouts whereas being not responsive enough can mean death and complete loss of fitness. This relationship between high and low risk situations is the rationale behind the predation risk allocation hypothesis (Lima and Bednekoff, 1999) and the threat-sensitive

predator avoidance hypothesis (see Monclús et al., 2009), which both suggest that animals should balance costly antipredator strategies with predation risk. The threat-sensitive hypothesis posits that the HPA/HPI axis activation displayed is proportional to the perceived risk and is supported by data from wild hares (Monclús et al., 2009). Whether we extend the findings from Monclús et al. (2009) broadly across vertebrates is not known, and thus the role that risk perception and assessment plays in the variation of the HPA/HPI axis response to predator cues (Table 1) is not known. The exact mechanism by which prey determine predation risk and how that level is risk translates to HPA/HPI axis function is unknown, but several individual and environmental variables may influence the way in which prey assess and respond to predation threats, examples are discussed below.

5.5.1. Type of predator cue – sensory modality

Predators are often viewed as 'stressors' for prey species, despite the fact that the neuronal predator detection pathways (visual, olfactory, auditory; see Section 4.1) do not fit neatly into recent views of the neuronal pathways mediating anticipatory and reactive stressors (Herman et al., 2003). Direct predator cues (e.g., visual, auditory) should be a more potent and honest signal of threat than indirect cues (e.g., scent). If this risk determination is coded, at least in part, by the HPA/HPI axis then we would expect visual predator cues to provide the most honest signal and thus always promote an increase in HPA/HPI activity with auditory being next and olfaction being last. However, analysis of the studies cited in Table 1 suggests just the opposite. The most effective predator cues were multimodal (including live predators), which caused activation of the HPA/HPI axis in 67% of the studies. The rank order efficacy for individual sensory modalities to elicit HPA/HPI axis activity was: olfactory cues (64%) > auditory cues (57%) > visual cues (50%). By far the most common predator stimuli used in the studies we examined (Table 1) were multilmodal cues (46% of all studies) followed by olfactory cues (39%). These findings seem to bear out the observation by Monclús et al. (2009) that live predators are more effective than individual predator cues in activating the HPA axis.

5.5.2. Prey satiety

Hungry prey take more risks in the presence of a predator (McNamara and Houston, 1992; Heinen, 1994; Horat and Semlitsch, 1994), indicating that satiety state can affect prey/predator interactions. If the HPA/HPI axis is involved in feeding-related changes in response to a predator, then it is reasonable to suspect that the HPA/HPI axis is modulated by satiety and orexigenic peptides that inform the CRF neurons in the PVN about satiety state. For example, neuropeptide Y (NPY), which is orexigenic when administered i.c.v., seems to integrate the activity of hypothalamic appetite control centers and the HPA axis (Hanson and Dallman, 1995). CRF neurons also play a role in appetite regulation apart from their regulation of pituitary corticotropes (Zorrilla et al., 2003; Stengel and Tache, 2014), and PVN CRF neurons interact reciprocally with orexigenic and anorexigenic neurons in the arcuate nucleus and other hypothalamic areas. Thus, it is likely that differences in satiety state might contribute to the variation in the HPA/HPI axis response to predator cues as shown in Table 1, although we have no way to examine this as the satiety state of the prey being monitored cannot be determined in many study situations, such as those involving natural prey populations.

5.5.3. Predictability of predator cues

Predictability of predators and predator cues can be evaluated in a variety of ways. For instance, predictability could mean how likely a prey animal is to encounter a predator at a certain location (e.g., a watering hole, out in an open field, at the forest edge) or at a certain time of day (crepuscular vs. diurnal vs. nocturnal predators). Additionally, predictability could also be interpreted as how likely it is for a prey animal to come across a specific species or type of predator – specifically, is the predator evolutionary/historic or is it novel (i.e., is it an introduced/invasive species)? Here we discuss this later form of predictability. Discrimination between historic and novel predators would suggest that some cues of predation risk assessment and response are learned and are not innate, or that if they are innate they can be down-regulated with repeated exposure. Fonner and Woodley (2015) suggest that sympatic or "known" predators would be more predictable than novel or new predators and would thus be less likely to activate the HPA/HPI axis of prey. However, assuming that predator cues being detected are still honest signals of predation risk, it seems likely that even if a prey species is used to encountering a specific type of predator, appropriate behavioral and physiological responses (HPA/HPI activation) would still be prudent to promote survival and thus innate and/or preserved predator responses would be beneficial.

Data supporting an innate response to predators exists throughout the literature. The discovery of feature detecting cells in the amphibian visual system by Jerome Lettvin and Jörg-Peter Ewert (Lettvin et al., 1959; Ewert, 1980) revealed the fact that animals are born with the innate ability to recognize key visual features of prey and predators. In toads, feature detecting cells ('snake-detector cells') in the lateral thalamus inform the animal about the height and movement of potential predators, with an optimal stimulus configuration (resembling a moving snake) releasing a characteristic behavioral defense posture that presumably reduces the likelihood of being eaten (Ewert, 1980). Such 'snake detector' cells also have been proposed to exist in the pulvinar nucleus of primates (Le et al., 2013, 2014), including humans (Almeida et al., 2015). Whether such feature detecting cells inform the HPA/HPI axis about visual predator cues is not known, but the possibilty certainly exists given functional connections between the superior colliculus and HPA axis (Wei et al., 2015).

Many laboratory rodents have innate behavioral and HPA/HPI axis responses to predator urine (Harris et al., 2012; Apfelbach et al., 2005) despite having never encountered a predator themselves. Additionally, the antipredator behavioral response of rodents to a specific compound, 2,4,5-trimethylthiazoline (TMT), in fox urine suggests that rodent prey may respond generally to canids, regardless of whether the predator is historical or novel (Fendt et al., 2005; Fendt and Endres, 2008; Fendt, 2006; in contrast to see McGregor et al., 2002). Along these same lines, mice showed a decreased anxiety response when presented with feces from a cat fed a vegetarian diet vs. a carnivorous diet (Berton et al., 1998) and hamsters are able to discriminate urine produced from a ferret fed hamster and non-hamster diets (Apfelbach et al., 2015), suggesting that prey may be sensitive to the excreta of any potential canriovours predator, regardless of past experience.

We cannot test predictability using data in our Table 1, but based on the above, we do not expect that novel vs. historic predator cues would play a large role in the HPA/HPI response (but see case study data in Sections 7.4 and 7.5 below), although, this is certainly an interesting area for follow-up in future studies.

5.6. Other potentially important variables

5.6.1. Parasites

In the wild, animals are almost certainly host to several parasites, a feature that is often not shared by their laboratory counterparts. Parasitic infection could play a role in prey species HPA/HPI axis response to predators, as parasites have been known to alter various aspects of prey physiology and behavior, often making the prey species more susceptible to predation (Weinersmith and Faulkes, 2014; Adamo and Webster, 2013). For example, both rats

and mice infected with *Toxoplasma gondii* showed a decreased avoidance of, and even attraction to, cat (predator) urine (Berdoy et al., 2000; Ingram et al., 2013). Moreover, infected rats show decreased baseline and cat-induced levels of CORT (Mitra et al., 2013), suggesting that at least with respect to *T. gondii*, parasites can manipulate host HPA/HPI axis function. We do not have information on parasite load and thus cannot determine if parasites influenced the results in Table 1, but we can compare laboratory vs. field studies as a proxy for parasite presence. Compared to 72% of laboratory studies (81 of 112 total), only 49% of field studies (25 out of 51 total) reported an increase in CORT in response to predator cues. Future studies investigating the relationship between the HPA/HPI axis and predator response should include measures of parasite load and parasite type.

5.6.2. Experimental design and method of CORT analysis

In addition to the influence of various ecological and physiological variables discussed above, choices about experimental design and hormone analysis could impact CORT results. The HPA/HPI axis follows a circadian rhythm with CORT levels generally peaking at the time of waking and then waning throughout the rest of the animal's active period. If predator-presentation studies are conducted during the time when CORT levels are at their peak, as opposed to at their nadir, significant changes in CORT may not be detected (Harris et al., 2012); however, whether non-significant increases in CORT have biological/functional importance is another discussion. Additionally, the sex of the researcher, and possibly of the predator providing the cues, could influence CORT results. A study by Sorge and colleagues (2014) showed that male experimenters, as well as unfamiliar male conspecifics, enhanced CORT secretion in laboratory rats and mice, an effect mediated by androgen-derived olfactory cues (Sorge et al., 2014). Lastly, the CORT analysis method choice could impact results. As mentioned previously (Section 3.1), most studies use measures of circulating or excreted CORT as the endpoint marker in HPA/HPI axis studies. Despite the conserved nature of the CORT molecule, assays, especially for metabolized and excreted form of CORT, must be validated for use in each species. Failure to do so can produce results that are not biologically meaningful and could thus impact conclusions drawn from such studies (Harris et al., 2012; Touma and Palme, 2005).

5.7. Summary

Overall, we found more support for a an increase in CORT secretion following acute predator exposure vs. chronic and in adults over juveniles. Additionally, we found that the type of predator cue also plays a role with multimodal stimuli (including live predators) to be the most effective, followed by olfactory, auditory, and then visual. Sexes did not appear to differ in their HPA/HPI responses to predators while there is a stark difference between HPA axis reactivity to predators in laboratory versus field situations. However, with all of these variables mentioned, there may be interactive effects (e.g., acute stress in males vs. females; stress in young vs. old males and females; odor cues in females; etc.) that could not be detected with this method of review. Thus, a future meta-analysis taking all of these variables into account is warranted and is being conducted by the authors.

6. Potential role of the HPA/HPI axis in gauging future predation risk

6.1. Can CORT modulate brain areas that process predator cues?

As discussed above, the predation risk allocation hypothesis (Lima and Bednekoff, 1999) and the threat-sensitive predator avoidance hypothesis (see Monclús et al., 2009) predict that prey should accurately gauge the risk of a predator encounter in order to maximize foraging and reproduction opportunities. One mechanism that may mediate such internal 'risk management' is the HPA/HPI axis. We hypothesize that predator-cue induced activation of the HPA/HPI axis, and the resulting increase in CORT, can modulate sensory processing areas used in predator detection, thus shaping future response to predators. Before considering this hypothesis though, we must remember that the neuroendocrine circuits that contain the HPA/HPI axis and control CORT secretion do not begin in the hypothalamus. Rather, they begin in first order synapses within sensory or sensory-processing brain areas detecting predator cues such as the optic tectum (visual cues), the main and accessory olfactory areas, and the cochlear nuclei (discussed above in Section 4.1). Indeed GRs are present in brain regions associated with visual (Wiggert and Chader, 1975; Teistma et al., 1998; Yao et al., 2008; Shahbazi et al., 2011), olfactory (Sousa et al., 1989; Gao et al., 1994; Morimoto et al., 1996), and auditory (Rarey and Luttge, 1989; Rarey and Curtis, 1996; Siaud et al., 2006; Stutzmann et al., 1998) processing, and data from a multitude of studies indicate that stress and CORT exposure induce changes in neuroanatomy, neuropeptide expression, and neuronal function in a variety of stress-related and emotional processing areas (McEwen, 2007, 2010; McEwen et al., 2012, 2015; Gray et al., 2013; Vyas et al., 2003; Ahima and Harlan, 1990; Wiedenmayer, 2004; Joëls et al., 2013). If CORT can modulate sensory processing (e.g., whether stimuli associated with a predator, predator cue, or event is interpreted as stressful) and emotionality (e.g., propensity to display fear, anxiety, and vigilance behaviors) then predator-threat-induced increases in HPA/HPI axis activity, and CORT elevation, may induce cellular changes that are beneficial for responding to future predator-related events (Fig. 2). Thus these stress-induced changes may be part of a trade-off to increase survival and overall reproductive fitness.

There are some data to support such a hypothesis although no studies, to our knowledge, have specifically examined neuroendocrine or structural changes in sensory (e.g., auditory, visual, olfactory) brain regions specifically resulting from acute or chronic predator-exposure-induced CORT increases (but see Hegab and Wei, 2014 for discussion on predator odors and immediate early gene activation). However, data from macaques, rats, California mice, fence lizards, and Frillfin gobies suggest that CORT can alter aspects of sensory processing. Treatment of rhesus macaques (*Macaca mulatta*) with the 11β-hydroxylase inhibitor metyrapone decreased the intensity and rate of alarm calls in response to simulated threats (Bercovitch et al., 1995), suggesting that CORT can alter the detection, processing, or response to predator-related cues. Injection of male California mice with CORT did not alter measures of direct paternal behavior (licking, huddling, kyphosis) or overall activity, but compared to control-injected males, CORT-injected males did take longer to contact their pup during a pup retrieval test (although statistical significance did not survive alpha correction; Harris et al., 2011). While sensory processing measures were not collected in this study, the data suggest that CORT-injected males may have perceived and/or responded to pup cues differently than control-injected males. When male rats were given metyrapone before acute exposure to a cat they displayed impaired long-term memory suggesting that CORT elevation following a predator stimulus is important for neural processing (Zoladz et al., 2014). In fence lizards (*Sceloporus undulatus*), treatment with metyrapone prevented beneficial changes in escape behaviors and learning following an encounter with a simulated attacker whereas replacement of CORT restored behaviors and learning, again suggesting that predator-induced CORT increase is important for predator-related memory and learning (Thaker et al., 2010). In Frillfin gobies (*Bathygobius soporator*),

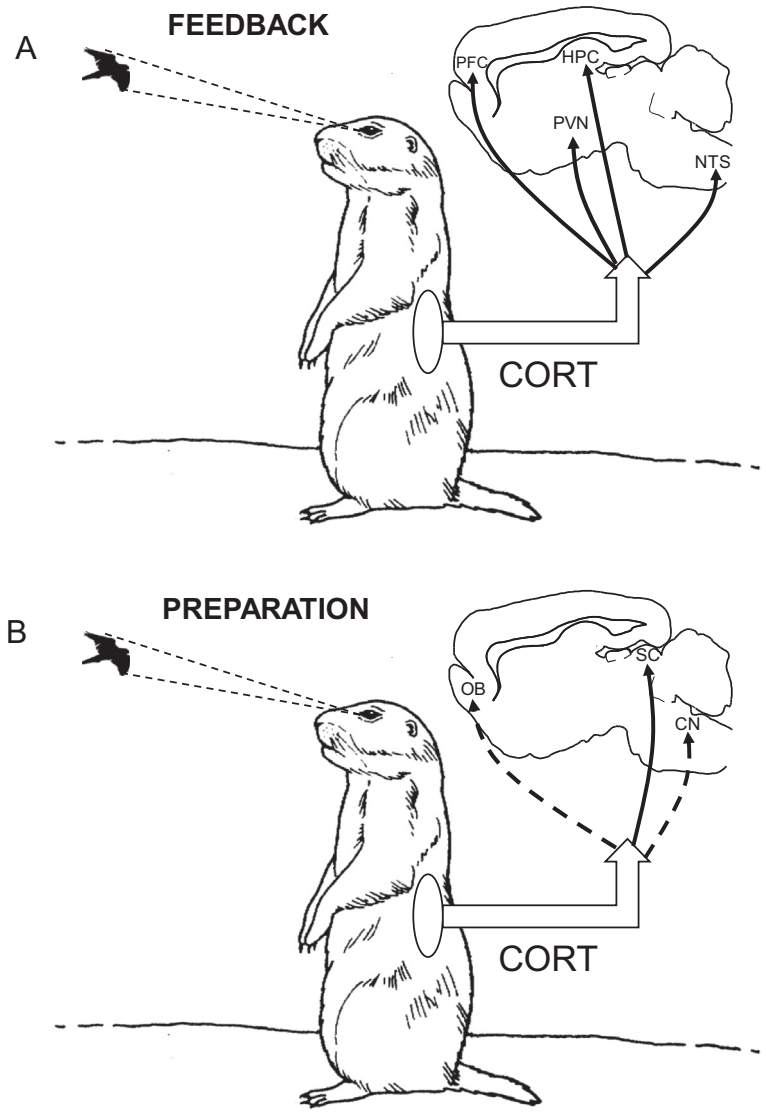

Fig. 2. Glucocorticoids (CORT) carry out feedback and possibly preparative functions in the central nervous system (CNS) after predator exposure. A. Visual predator cues lead to CORT secretion from the adrenal cortex, where CORT enters the CNS and acts on glucocorticoid receptors (GRs) in the prefrontal cortex (PFC), hippocampus (HPC), paraventricular nucleus (PVN) and nucleus of the solitary tract (NTS) to directly (PVN) and indirectly (PFC, HPC, NTS) inhibit corticotropin-releasing factor secretion. B. CORT may act on GRs in brain areas receiving first order sensory signals, in this case the superior colliculus, to modulate sensorimotor processing in the event of future exposure to the same predator cue. Brains areas conveying first order olfactory (olfactory bulb, OB) and auditory (cochlear nucleus, CN) information also are shown receiving CORT modulation by the dashed lines.

pretreatment with CORT enhanced the antipredator behavioral response to a conspecific alarm cue more than either the alarm cue or CORT alone, suggesting that CORT may alter the way a predator-related cue is perceived (Barreto et al., 2014). Although none of these studies looked directly at post-predator-exposure CORT effects on sensory brain regions, the data suggest that CORT likely plays a role, at least in some capacity, of sensory perception and/or processing.

6.2. Translation to preclinical models of fear and anxiety

When a prey animal is exposed to either a live predator or a predator cue, prey show increased fear and anxiety-related behaviors (Clinchy et al., 2011a, 2013; Cohen et al., 2012). Predator-induced fear paradigms using both acute and chronic predator presentation are common in the rodent literature and are used to model human psychopathologies such as post-traumatic stress disorder (PTSD), as these models are a form of psychological stress for the prey (Clinchy et al., 2011a, 2013; Cohen et al., 2012). Predator-induced psychological stress is likely not just an artifact of the laboratory setting, as predators in the wild have profound non-killing, psychological effects on prey (see Preisser et al., 2005 for meta-analysis and review). Moreover, prey that develop an anxious phenotype may be better equipped to deal with future predator threats and thus may have increased survival compared to non-anxious prey (see Korte et al., 2005; Sih et al., 2004; Conrad et al., 2011; Brilot et al., 2012; Fig. 2), suggesting that while this anxious phenotype resembles a clinically diagnosed pathology (PTSD), it may not represent a necessarily maladaptive phenotype under natural conditions (see Diamond and Zoladz, 2015).

The mechanism underlying the observed 'anxious prey' phenotype (Cohen et al., 2012; Clinchy et al., 2013; Brilot et al., 2012) is not entirely known; but, it is presumably induced, at least in part, by predator-related increases in HPA/HPI axis activity. Chronic stress and increased CORT are both associated with an increase in amygdalar CRF concentrations, an alteration which is

hypothesized to underlie fear and anxiety behavior (Schulkin et al., 1994, 2005; Heinrichs and Koob, 2004; Müller et al., 2003; Gross and Canteras, 2012; Diamond and Zoladz, 2015). Additionally, activation of GR, but not MR, enhances the memory for contextual fear conditioning (Cordero and Sandi, 1998).

There are likely trade-offs associated with the brain remodeling that occur in respond to predation threat. For example, an organism would be more anxious and possibly responsive to sensory cues which may aid it in being able to adequately respond to threats, but it also must balance this increased "vigilance" with feeding and reproducing. Not only does this idea integrate how predator-induced HPA/HPI axis activation could lead change in organism behavior and fitness, it would help explain the modulatory effect of satiety peptides on sensory input and feed/flee neural networks (Carr, 2002, 2006; Carr et al., 2002). Balancing feed or flee decisions also is bound to affect reproduction because 1) reproduction is energetically expensive and adequate energy reserves are needed to sustain reproduction, and 2) reproduction is often conspicuous and organisms must balance being eaten with copulatory and parental care behaviors.

7. Weighing the evidence that predator avoidance trade-offs influence HPA/HPI axis regulation of reproduction and foraging

Based on the literature reviewed in the previous sections we can say with certainty that a) animals adjust reproduction and foraging efforts and HPA/HPI activity in response to predator cues, and b) that the HPA/HPI axis can modulate reproduction and foraging at multiple levels. However, the simple fact that predator cues may increase HPA/HPI activity, and that HPA/HPI axis hormones may modulate foraging and reproduction, does not necessarily mean that the HPA/HPI axis mediates predator effects on prey reproduction and foraging. What is the strength of the available evidence linking the HPA/HPI axis to mediation of predator effects on foraging and reproduction? In theory the data supporting a role for the HPA/HPI axis in mediating predator effects on prey reproduction and foraging could span a range from associations between HPA/HPI axis activity (weak) to evidence that selective GR antagonists block predator effects on prey reproduction and foraging, which would be relatively strong evidence for such mediation.

In the final section of this paper we aim to address whether the hormones of the HPA/HPI axis mediate predator effects on reproduction and feeding. We begin by summarizing evidence from studies that have measured predator (predator cue)-induced changes in reproduction and feeding, and have measured some aspect of HPA/HPI activation. We next address the experimental constraints and limitations that exist in addressing the HPA/HPI axis' mechanistic role and offer suggestions on how to more explicitly address this question in both wild and laboratory animals. Lastly, we suggest a new framework in which this question can be viewed.

7.1. Individual-level effects of predator-induced CORT release on reproductive output in snowshoe hares

Snowshoe hares in the Yukon experience 10-yr population cycles that are driven by changes in predator pressure. The hare population decline can be linked to increase in predator number and hare deaths by predation. Hares also experience changes in reproduction over the course of a population cycle: during low predation pressure hares have increased reproductive outcomes as compared to times of high predation pressure. CORT is a likely candidate as for suppressing reproduction levels increase with predation risk (Boonstra et al., 1998). Sheriff and colleagues (Sheriff et al., 2009) used a natural monitoring study and an experimental manipulation to determine if increased CORT is directly related to predator exposure and decreased reproductive output. Wild female hares monitored for three years had increased FGMs at birth of their first litter within each year compared to their second litter, but FGMs did not differ across years; first litters were smaller in number of offspring born and the offspring were smaller in size and lighter than those born in the second litter. Dog-exposed (stressed) pregnant hares had significantly elevated FGMs and had a lower birth rate than the control hares (35% of stressed hares gave birth to stillborn offspring vs. 8% of controls). Stressed hares gave birth to smaller offspring that weighed less compared to control hares (Sheriff et al., 2009). Overall, this study suggests that on an individual level, predator-induced increases in CORT have a direct negative impact on reproduction.

7.2. Population-level changes in FGMs, fecal progesterone metabolites (FPMs), and reproductive output in elk

After 50 yrs without wolves, elk living in the Yellowstone Ecosystem were presented with new predation pressure when wolves were reintroduced. Creel and colleagues (Creel et al., 2009) used this population to test two hypotheses (predator-sensitive food and predation stress, a.k.a chronic stress) to determine the mechanism driving decreases in feeding and reproduction. Contrary to expectations, for the four years of sample collection elk living in lower predation areas produced higher winter FGMs than did elk living in higher predation populations, but these effects seemed to be driven by endocrine changes associated with gestation (higher pregnancy rates in the low predation areas). But, after accounting for this difference, FGMs did not correlate with predation risk. Additionally, two measures of reproduction, calf recruitment and FPMs, were not related to FGMs. This dataset suggests that different levels of predator pressure can alter aspects of feeding and reproduction, but that population level changes in FGMs are not mediating food and reproductive outcomes (Creel et al., 2009).

7.3. Population-level stress and reproductive responses to high vs. low predation pressure in birds

As in other taxa, few studies directly assessing the relationship among predators, HPA axis response, feeding and/or reproduction exist in birds. There are three studies, however, that begin to address this interaction and data suggest that measures other than total CORT concentrations (i.e., free CORT and CBG) are needed (see introduction in Clinchy et al., 2011b). A study on tropical stonechats (*Saxicola torquata axillaris*) related circulating CORT and breeding behavior to presence or absence of a predator (shrike) living on the stonechat territory (Scheuerlein et al., 2001). In stonechats, increased parental care and parental vigilance are associated with greater survivorship of their broods and in stonechat pairs living in territories with shrikes, parents cared for their broods longer and delayed the onset of a second brood (Scheuerlein et al., 2001). Additionally, males of these pairs also engaged in more distraction calls to deter predators from the nest (Scheuerlein et al., 2001). The results of the CORT analysis, however, were mixed: male stonechats from shrike territories had higher CORT than males from non-shrike territories, whereas in females, stonechats from predator and non-predator territories did not differ in circulating CORT (Scheuerlein et al., 2001).

A study by Clinchy and colleagues addressed the interaction of food availability and predator pressure on nest outcomes and measures of chronic stress (including circulating CORT) in song sparrows (*Melospiza melodia*; Clinchy et al., 2004). Free-living sparrows were from either high- or low-predation areas and were either food supplemented, or not, yielding 4 groups; to prevent

nest disturbance, only males (fathers) were blood sampled for analysis of plasma CORT (and other markers of chronic stress). Food availability and predators both affected baseline and post-stress (30-min post holding the bird in a cloth bag) CORT in fathers, with unfed birds having higher values with and without the presence of predators, but fed, low-predator presence males had the lowest CORT while unfed, high-predation males had the highest CORT (Clinchy et al., 2004). Additionally, broods from pairs in unfed and high-predators sites were of poorer quality than their fed and low-predators-site counterparts (Clinchy et al., 2004). These data suggest that presence of a predator can increase markers of chronic stress (CORT), alter parental behavior, and affect nest outcomes, but it is not possible to say whether elevated CORT is directly responsible.

A second study by Clinchy and colleagues set out to examine how differences in predator threat (high vs. low) can affect multiple aspects of baseline glucocorticoid regulation (free, bound, and total) in male and female song sparrows (Clinchy et al., 2011b). They used the same population of sparrows as described above and have previously shown that birds from high- and low-predation areas differ in multiple reproductive outcomes. Males from high-compared to low-predation areas had higher baseline total CORT; females did not differ in circulating total CORT (Clinchy et al., 2011b), which mirrored resulted found in stonechats (Scheuerlein et al., 2001). When comparing levels of CBG, females, but not males, from high-predation areas had lower concentrations than females from low-predation areas (Clinchy et al., 2011b). Thus, males and females had similar changes in *free* CORT, but the mechanism driving changes in free CORT increase differed by sex (higher total CORT in males, lower CBG in females). These data suggest that measuring multiple aspects of CORT regulation is important and highlight between-sex differences.

7.4. Population-level differences in glucocorticoid response to predators in Galapagos iguanas reared in the presence or absence of predators

Marine iguanas in the Galapagos provide a unique system as this species has evolved for up to 15 million yrs without predators (see Rodl et al., 2007). When comparing iguanas from islands with differing levels of predation (none, intermediate, and heavy [due to introduced dogs]), all iguanas showed a CORT response to capture and restraint, but following a simulated attack (chasing by a human) only iguanas from the high-predation island had CORT levels higher than naïve controls (Rodl et al., 2007). These data suggest that the HPI axis response specific to predator threat is acquired after the introduction of predators and that living without predator threat is associated with a decreased ability to recognize potential predation threats (Rodl et al., 2007). Physiological responses appear to be related to behavior as iguanas from low/no predation islands had shorter flight initiation distances (a measure of fear; shorter distances are associated with a less fearful animal) and decreased CORT response to acute stress compared to those from higher predation islands (Berger et al., 2007). To date, no study has linked predators, CORT, and reproduction and feeding directly, but lower levels of baseline and stress-induced CORT was associated with increased reproductive performance and higher body condition in female iguanas (Vitousek et al., 2010), and baseline CORT is likely related to food intake, the tidal cycle, and the light/dark cycle (Woodley et al., 2003). Additionally, baseline and stress-induced CORT levels are associated with decreased survival during an El Niño famine (Romero and Wikelski, 2001, 2010). These data suggest that elevated CORT can be detrimental to iguana reproduction and survival and that with repeated exposures iguanas can process predator threats as stressor that activates the HPI axis.

7.5. Population-level differences in Trinidadian guppies evolving in high- and low-predation areas

Like the Galapagos iguanas, Trinidadian guppy populations provide a unique wild system in which to study the effects of predators. In Trinidad, guppies can either live in high- or low-predation streams (see Reznick and Endler, 1982; Reznick et al., 2001) that are geographically similar (often separated by a fish-impermeable waterfall) making them good populations for studying the long-term effects of predation on the evolution of antipredator mechanisms and the HPI axis response to predator cues. Guppies from high- and low-predation sites differ in several life-history variables (see Reznick and Bryga, 1987; Reznick et al., 2001) and, not surprisingly, the guppies from high-predation areas suffer greater mortality (Reznick et al., 1996). Like the iguanas, no author to date has measured predators, HPI axis activity, feeding and reproduction in a single guppy study, but data from multiple papers can help enhance understanding of this topic.

Guppies exposed to olfactory predator cues (water from guppy-fed pike cichlid housed in the tank sump) early in life had lower metabolic and growth rates compared to guppies raised in the absence of predator cues (Handelsman et al., 2013). Compared to guppies from low-predation sites, guppies from high-predation sites matured earlier, had higher fecundity, and grew faster, changes likely associated with diet selection (Zandona et al., 2011). In commercially purchased guppies, exposure of females to chemical, visual, or chemical + visual predatory cues, compared to control, resulted in increased brood-size and shortened the brood time (duration from breeding to birth); this effect disappeared for the second brood when the predator cues were removed (Dzikowski et al., 2004). Female guppies exposed to either visual or chemical predator cues decreased brood time compared to control females, and offspring born earlier showed decreased swimming speeds suggesting a trade-off between pregnancy duration and offspring quality in the presence of a predator (Evans et al., 2007). Guppies from high-predation sites showed stronger behavioral responses to two predatory fish models than did guppies from low-predation sites, thus suggesting that high-predation guppies are better at visually recognizing predators than are low-predation guppies (Kelley and Magurran, 2003). Wild-caught guppies from low- and high-predation sites did not differ in baseline CORT (measured via tank water samples) on days 1–4 of collection, but by day 5, high-predation fish secreted less CORT than low-predation fish (Fischer et al., 2014; Table 1). Laboratory-reared fish from low- and high-predation sites also differed in (tank water) CORT, again, high-predation fish tanks had lower CORT (Fischer et al., 2014). When laboratory-reared fish were continually exposed to a predator cue (guppy-fed cichlid housed in sump), regardless of predation site, tank water contained less CORT as compared to control tanks (Fischer et al., 2014). Acute exposure to a chemical predator cue did not alter (tank water) CORT levels in low- or high-predation laboratory-reared guppies (Fischer et al., 2014). Contrary to the majority of evidence presented thus far in our paper, it seems that guppies living in areas of high predation have evolved decreased acute and long-term stress responses to predators, and increase reproductive investment in the presence of predators.

7.6. Evaluating the strength of the evidence supporting a causal linkage between predation and the HPA/HPI axis regulation of reproduction and foraging

Although the above case studies begin to address the associations among predators, HPA/HPI axis hormones, and feeding and reproduction, drawing conclusions regarding causal relationships remains problematic as results were not consistent. One of these

studies found no relationship between predation and CORT (Creel et al., 2009) and another found lower CORT in high predator environments (Fischer et al., 2014). These studies point out, implicitly, the fact that realistic constraints exist when answering this question, especially in wild animals.

While a few of the studies have used controlled conditions to show that isolated exposure to predator cues alter CORT release, none have examined whether CORT released as a result of predator cues influences reproduction and/or foraging. To date the strength of the evidence for HPA/HPI mediation of predator effects is limited to associations and correlations, which by their nature cannot address cause and effect. To determine a causal link among predators, the HPA/HPI axis, and behavior, experiments could expose animals to varying levels of predation pressure (cues), alter HPA/HPI axis function (using 11β-hydroxylase inhibitors, adrenalectomy, or GR antagonists such as mifepristone, for example) and measure aspects of feeding and/or reproduction. Dependent variables for these studies should include relevant, species- and sex-specific measures of reproduction and feeding, circulating CORT, CBG levels (see Clinchy et al., 2011a,b), and ideally brain expression of CRF, tissue activity of 11β-hydroxysteroid dehydrogenase, and MR/GR density and activation. Additionally, it is likely the relationship among predators, HPA/HPI axis activation, and feeding and breeding is influenced by multiple variables (see Section 5). Experimenters should also take note of the life history stage of the organism being tested as predator exposure early in life (e.g., during development or the juvenile period) could produce very different effects on reproduction and feeding as compared to predator exposure during adulthood as shown for larval anurans (Table 1). Moreover, duration, acute or chronic, of predator exposure as well as time course from exposure and method of measuring HPA/HPI response are also important.

If the increase in HPA/HPI axis activity following predator (cue) exposure alters reproduction and feeding, then blockade of predator-induced HPA/HPI increase should fail to alter reproduction and feeding in the presence of a predator. Likewise, stimulation of the HPA/HPI axis, without predator exposure, should result in the same reproductive and feeding changes as seen during predator cue exposure. Manipulation of the HPA/HPI axis could be done in a variety of ways but major difficulty lies in the fact that the aim is to suppress activity of a system which is important for not only for dealing with stressors, but is also important for baseline, daily functioning (differences between baseline, circadian changes, and stress-induced changes in HPA/HPI activity). Moreover, the presence of feedback pathways means that manipulations aimed at removing one part of the axis (inhibition of 11β-hydroxylase activity using metyrapone, for example), indirectly elevates other components of the same axis (CRF and ACTH in the case of metyrapone) known to affect the same endpoints (see Table 1 and Section 3). Since no single manipulation method is without criticism, combining multiple approaches may be required to establish causal linkages.

8. Summary

Changes in reproduction and feeding in response to predators has been documented in hundreds of studies. Dozens more studies have shown changes in HPA/HPI axis activity after predator cue exposure, and these studies, along with studies showing that HPA/HPI axis hormones alter reproduction and feeding on their own, have led to the hypothesis that predator-induced changes in reproduction and foraging are mediated by the HPA/HPI axis. There is accumulating evidence that predator-induced changes in HPA/HPI activity are associated, and correlated, with changes in reproduction and foraging, yet cause and effect relationships have yet to be established. More experimental work using species-specific, ecologically-relevant hypotheses and dependent variables (see Sections 5 and 7.6) is needed to show that direct manipulation of HPA/HPI axis hormone secretion results in the same types of changes in reproduction and foraging observed after predator exposure. Future studies should aim to incorporate measures of predation risk, type of predator cue, stressor duration, time of day, parasite load, age, sex, and multiple markers of HPA/HPI axis function, as all of these variables can impact results (see Section 5). Likewise, more experimental work is needed to show that manipulation of the HPA/HPI axis causes predictable changes in reproduction and foraging after predator cue exposure. It is important in future work to include the sensory modalities detecting predator cues into the framework of the HPA/HPI axis response, as this is where the physiological response to predators first begins, and it is plausible, if not likely, that HPA/HPI axis hormones in turn modulate the response of these sensory pathways to subsequent predator exposures. Considerable evidence already exists to support a role for CORT, CRF, and melanocortin peptides acting within the optic tectum/superior colliculus to modulate visuomotor processing in as of yet undetermined ways. While predator cues often are lumped in with other so-called 'stressors', it must be recognized that while stress is an outcome of exposure to visual, olfactory, and/or acoustic predator cues, the sensory pathways regulating the HPA/HPI axis also are involved in emotional aspects (fear and anxiety) of the predator response. Based upon the fact that emotional states are now recognized to have been conserved among all vertebrate taxa (Kalueff et al., 2014; Ogawa et al., 2014; Carr, 2015), more studies need to carefully tease part the contribution of fear and anxiety to predator-induced changes in reproduction and feeding. More elegant behavioral and physiological tools also are needed to discriminate between stress, fear and anxiety in studies using wildlife and non-mammalian models. This would aid comparative and behavioral neuroendocrinologists in unlocking the potential that non-traditional animal models hold for shedding light on the evolutionary basis of anxiety and PTSD in humans.

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