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Review

Social status, glucocorticoids, immune function, and health: Can animal studies help us understand human socioeconomic-status-related health disparities?

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ABSTRACT

This article is part of a Special Issue "Neuroendocrine-Immune Axis in Health and Disease."

For humans in developed nations, socioeconomic status (SES)—relative income, education and occupational position in a society—is a strong predictor of morbidity and mortality rates, with increasing SES predicting longer life span (e.g. Marmot et al., 1991). Mechanisms underlying this relationship have been examined, but the relative role of each mechanism still remains unknown. By understanding the relative role of specific mechanisms that underlie dramatic health disparities between high and low social status individuals we can begin to identify effective, targeted methods to alleviate health disparities. In the current paper, we take advantage of a growing number of animal studies that have quantified biological health-related correlates (glucocorticoid production and immune function) of social status and compare these studies to the current literature on human SES and health to determine if and how animal studies can further our understanding of SES-associated human health disparities. Specifically, we compared social-status related glucocorticoid production and immune function in humans and animals. From the review, we show that our present understanding of the relationships between social status and glucocorticoid production/immune function is still growing, but that there are already identifiable parallels (and non-parallels) between humans and animals. We propose timely areas of future study focused on (1) specific aspects of social status that may influence stress-related physiology, (2) mechanisms underlying long-term influences of social status on physiology and health, and (3) intervention studies to alleviate potentially negative physiological correlates of social status.

Contents

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1. Introduction

Animals that live in social groups tend to establish and maintain social ranks within their groups (e.g. social status, dominance rank, socioeconomic level; Rowell, 1974; Wilson, 1975). Evolutionary theoreticians have proposed that for group-living individuals to survive, it is helpful for group members to develop stable social relationships to minimize energy committed to fighting for resources within a group (e.g., Drews, 1993). Psychological theory further posits that complex social animals (e.g. humans) have brains that are particularly good at identifying differences among individuals and assigning relative value to individual traits and status (Cummins, 2000). For example, a recent study has shown that the synaptic strength of cells in the medial prefrontal cortex of mice differs according to the individual's social status (Wang et al., 2011), suggesting neurological programming that supports the ability to make social comparisons.

In humans, social status has often been evaluated using the concept of 'socioeconomic status' (SES). SES, usually quantified as a combination of income, education, and occupation, accounts for notable health disparities in developed societies (Adler and Ostrove, 1999; Adler et al., 1994). The earliest of these findings from the 'Whitehall' studies in Britain showed a clear SES-related gradient in life span and health among civil servants, with lower SES individuals living shorter lives than higher SES individuals (e.g. Marmot et al., 1984, 1991). Many follow-up studies have shown the ubiquity of the socioeconomic health gradient and that health-related behaviors (e.g. smoking, diet, medical visits, etc.) only partially explain this gradient. Other proposed mechanistic pathways include stress-related biological processes (Fein, 1995; Kristenson et al., 2004; Stringhini et al., 2011). The goal of the current paper is to determine if and how data from animal societies can help us understand mechanisms that underlie SES-related human health disparities. We review studies that compared stress-related biological function among high- and low-status humans and animals. Specifically, we review the following in both human and animal studies: (1) What is the relationship, if any, between social status and glucocorticoid production?, (2) What evidence is there that one's social status is related to immune function?, (3) How do the human and animal results compare and contrast?, and (4) What information from animal studies can be used to inform human studies on SES-related health disparities and associated physiological stress processes?

2. Human socioeconomic status vs. animal dominance/social status

The current work builds on prior reviews that identified parallels and differences between human socioeconomic status and animal dominance rank (Sapolsky, 2004, 2005). These reviews also showed evidence of increased glucocorticoid production (and biases in other physiological systems) in low- vs. high-status humans and animals, which could account for a portion of differential status-related health outcomes. There are some clear parallels between the human and animal status concepts, and at least one major difference. First, in both human and animal societies, it is possible for social rank (SES or dominance) to be inherited, although in animals this is more often seen among female primates than in males or other species. Second, in both human and animal societies, social status is flexible or can change across the life span. Thus, status is not necessarily inherent to the individual but involves a complex interaction of individual characteristics relative to current social contexts and norms. A major difference between human SES and animal dominance is in the methods that are used to estimate social status and the consequential association between an individual's social status and their daily social interactions. Human SES, as discussed above, is estimated based on relative income, education, and job status. Thus, SES is often compared among humans that have never interacted with one another. This is different from methods used to estimate animal dominance status, where status is assessed from repeated social interactions (aggression, displacement, and/or submission) within individual dyads and a matrix developed based on these dyadic interactions to identify individual ranks within a social group (e.g. Martin and Bateson, 1993). Thus, in animal groups, social status is specifically based on the quality of face-to-face interactions among individuals, which is different from SES determination in humans. However, low and high SES humans do come into contact with one another and subtle social comparisons that occur during these interactions may be an important psychological component of human SES. Overall, both conceptions of social status (human SES and animal dominance) have been developed as simplified estimates of individual group members' access to resources and exposure to challenges (both physical and social).

3. Context and dynamics of social status

Depending on the species or societal structure, status within a society comes with different corollaries. Social status (whether socioeconomic status in humans or dominance rank in other animals) may be fixed or flexible over time, may be earned through repeated social interactions or inherited from a parent's status, can involve either a high or low degree of agonistic interactions, might come with high or low levels of social support, might be associated with either high or low access to resources or exposure to environmental dangers, and might involve frequent or infrequent encounters among individuals at different ends of the status spectrum (reviewed in Goymann and Wingfield, 2004; Sapolsky, 2005). For example, female baboons and macaques inherit their social status based on their mother's rank whereas female ring-tailed lemurs must establish and maintain their rank through repeated aggressive attacks (e.g. Jolly, 1998; Silk et al., 1999). In another example, some human societies exhibit a relatively large degree of variance from their most affluent to least affluent members, while others exhibit a more egalitarian distribution of wealth. There also seem to be specific health implications associated with each of these social systems (Wilkinson, 1999). Finally, across species, access to escape, protection, or support during aggressive interactions may be more or less available depending on characteristics of the living space (e.g. open vs. closed), exposure to agonistic partners, and levels of social support. These context-specific aspects of social status are key in determining the relative stress and the different health outcomes associated with each social position. In this review, we attend to how these contextual variables predict endocrine, immune and health implications of social status.

To best determine what it is about high or low social status that may influence our bodies and our health, researchers have begun to dissect social status into its different correlates, such as access to resources, exposure to danger, and specific social interactions related to status (e.g. aggression, defeat/submission, grooming, social support). A few key correlates of social status have been associated with physiological and health outcomes: (1) stability of one's social environment, (2) initiation of or exposure to aggression, and (3) access to social support (Abbott et al., 2003; Adler and Ostrove, 1999; Goymann and Wingfield, 2004; Gust et al., 1993; Wittig et al., 2008). We will focus on these aspects throughout this review, but acknowledge that there are many other components of social status that may be key mediators of its relationship to biology and health (e.g., access to key non-social resources like food and shelter, protection from harm or trauma; Cheney and Seyfarth, 2009).

4. Glucocorticoid production and immune function

Glucocorticoid production has become a common estimate of physiological stress. In addition, recent methodological advances have led to an exponential increase in studies that measure glucocorticoids. Glucocorticoids are steroid hormones produced in the cortex of the adrenal gland and represent the 'final' hormone in the hypothalamic–pituitary–adrenal (HPA) cascade. Glucocorticoids are involved in glucose metabolism and

play an important role in moderating pro-inflammatory responses (Sapolsky et al., 2000). They can have both stimulatory and suppressive influences on the different branches of the immune system (e.g., Besedovsky et al., 1986; Dhabhar and McEwen, 1999; Sapolsky et al., 2000). However, there is presently ample evidence that chronic elevated glucocorticoid exposure is implicated in dampened cell-mediated immune responses and immune cell glucocorticoid resistance which can allow 'minimally-regulated' or 'run-away' immune reactivity (e.g. Avitsur et al., 2001; Dhabhar and McEwen, 1999; Biddie et al., 2012; c.f. Roberts et al., 2007). This influence of chronic glucocorticoid overproduction may be most relevant when considering the biological pathways by which social status—which tends to represent a chronic condition—is associated with immune and health outcomes. Admittedly, only reviewing glucocorticoid production presents a limited estimate of physiological stress and excludes any review of sympathetic activity which also influences immune function (Sanders and Straub, 2002; Sloan et al., 2007). Due to methodological limitations in measurements of sympathetic activity and hormones higher up in the HPA cascade, there are more studies relating social status to glucocorticoid production than these other aspects of physiological stress, limiting the current review to glucocorticoid production.

For the literature search on social status and glucocorticoid production we used the following search terms in PubMed and Web of Science databases: (social status or social rank or dominance or socioeconomic status) and (glucocorticoid or corticosterone or cortisol). To mimic human social conditions, we limited our review of animal studies to those conducted with free-ranging subjects and subjects living in large, complex enclosures (e.g., Cheney and Seyfarth, 2009). We did not include studies conducted with animals living in confined spaces because (a) confined space limits an individual's ability to escape stimuli associated with status and (b) there has been a recent surge of studies conducted in free-ranging animals. Finally, we limited this search to studies published since 2000 and made use of recent reviews that summarized earlier studies. For the literature search on social status and immune function, we used the following search terms in PubMed: (social status or social rank or dominance or socioeconomic status) and (pathogen burden or virus or immune function or splenocyte proliferation or antibody or NK-, T-, or B-cell or lymphocyte). To investigate relationships between social status and C-reactive protein, a meta-analysis (Nazmi and Victora, 2007) adequately described previous research. For this relationship, only papers published after 2007 were reviewed in addition to this study. No other time constraints were placed upon search terms. Because of methodological limitations, there are fewer studies that have been conducted on social status and immune function in free-ranging animals as compared to studies on status and glucocorticoid production. Thus, for the immune function review, we used studies conducted with both free-ranging and caged animals, and noted differences in results between free-range and caged animal studies. Pertinent references cited in papers from the above searches were also incorporated into the review.

5. Social status and glucocorticoid production

5.1. Humans

A variety of studies document higher psychological stress in low- vs. high-SES humans, and this seems particularly true in societies with large SES variance (reviewed in Seeman and Crimmins, 2001). However, there is little evidence that this increased psychological stress mediates the relationship between SES and health (Baum et al., 1999; Matthews et al., 2010). Although physiological stress may be another mediator of SES-related health differences, there are still relatively few studies that have compared glucocorticoid production across levels of SES.

A recent review of studies comparing SES to glucocorticoid production indicates that the relationship is not consistent (Dowd et al., 2009b). Of 21 studies summarized in this review, seven found greater cortisol production in low- vs. high-SES individuals, while the remaining 14 showed either no relationship (n = 8), a mixed relationship (n = 4), or an opposite relationship between SES and cortisol production (n = 2). There was no evidence that countries with greater income disparity (for example USA or Taiwan) were more likely to show elevated cortisol levels in low-SES individuals compared to more egalitarian income societies (e.g. Canada, Sweden, or Germany). There was no clear difference between men and women in how SES related to glucocorticoid production, although one data set suggested that males may more readily show the expected relationship of elevated glucocorticoid production in low-SES individuals compared to high-SES. The authors attributed this variance in study results to the number of different methods used to measure cortisol production across studies. The most consistent relationship between glucocorticoid production and SES was found when measures of cortisol production were taken multiple times per day for each participant, allowing for the calculation of slopes and 'area under the curve' (AUC) for daily cortisol measures. These measures provide a rough estimate of cortisol/HPA regulation over time, and studies that used these measures indicated greater cortisol production across the day (i.e. unregulated cortisol production) in low- vs. high-SES individuals (Cohen et al., 2006a, 2006b; Li et al., 2007b). Studies since the Dowd et al. (2009b) review that have used repeated cortisol measures have produced some similar results, but also some exceptions (e.g. Agbedia et al., 2011; Hajat et al., 2010). More research is necessary on long-term cortisol regulation in low- vs. high-SES individuals. Another area to examine in future studies is based on recent findings associating low parental SES with significant increases in offspring cortisol production during childhood that continues into adulthood, suggesting a long-term influence of 'inherited' status (Chen et al., 2010; Gustafsson et al., 2010; Miller et al., 2009).

The consistency of circadian rhythm or AUC cortisol estimations with respect to SES better accommodates for the pulsatile nature of hormone release and the relative noise involved in any single measure of hormone production within an individual (Lightman et al., 2008). Repeated measures are necessary to estimate both glucocorticoid reactivity to specific stressors and to estimate circadian glucocorticoid production/regulation in one individual vs. another (Hellhammer et al., 2007; Hruschka et al., 2005). This issue is particularly true for glucocorticoid hormones that are highly responsive to environmental perturbations from minute to minute. Although the relationship between SES and cortisol production may be highly sensitive to measurement methods, there is suggestive evidence of an underlying relationship between SES and circadian glucocorticoid regulation.

5.2. Other animals

Compared to human studies, animal research often involves multiple measures of glucocorticoid production within the same individual at different times of the year. Given this difference, we might expect more clarity about how social status in animal species may relate to subtle differences in mean glucocorticoid production or regulation over time—a characteristic of glucocorticoid production that may have significant consequences on immune function, health, or life span. In addition, the human literature suggests that SES is most often related to cortisol when multiple cortisol measures are used. Therefore, a review of animal studies that use these measurement methods is appropriate.

Initial and recent studies comparing physiological stress in low- vs. high-status caged animals (usually males) confirm a long-standing assumption that low status is associated with elevated activation of physiological stress-related systems (e.g. rats: Barnett, 1955; McKittrick et al., 1995; mice: Bronson and Eleftheriou, 1964; Davis and Christian, 1957; monkeys: Gust et al., 1993; Manogue et al., 1975; reviewed in Sapolsky, 2004; c.f. hamsters: Chelini et al., 2011). However, recent findings from both males and females across a greater variety of species

Table 1
Recent studies comparing animal social status to glucocorticoid production in non-caged animals (i.e. free-ranging/wild and/or in relatively large or complex enclosures that allow for more natural social interactions) published after 2000 (see Abbott et al., 2003; Creel, 2001; Sapolsky, 2004 for studies published before 2000.) When a study included data from both stably and unstable social periods, the relationship between dominance status and glucocorticoid (CORT) levels is separately reported for the two periods. The experimental group(s) that exhibit greater relative glucocorticoid levels are designated by bold-faced characters. See Key below for further explanation of columns and information within columns and see text for further description of literature search methods and criteria.

Species	Sex	Environment/ housing	No. of animals/ groups	Social structure (dominance)	Medium	Period of CORT sampling	Reproductive state	Samples per ind'l (range)	Social/ enviro. stability	Dom. vs. sub. CORT	Authors (year)
Primates											
Chimpanzees	M	Free-range	11/1	MMF (non-inherited)	Urine	12 mo	Full-year	AM: 31 PM: 15 (6–56, 5–30)	Stable	AM: D = S PM: **D** > S	Muller and Wrangham (2004)
Chimpanzees[a]	M, F	Outdoor enclosure	23/10	MMF peer groups (non-inherited)	Urine	7 mo (across 2.5 yr)	Summer–autumn	23 (12–?)	Unstable?	D = S	Anestis (2005)
Chimpanzees	F	Free-range	18/1	MMF (non-inherited)	Urine	5 yr	Non-estrus, estrus, lactation	67 (8–174)	Stable UNSTABLE Stable?	D = S **S** > D **S** > D	Thompson et al. (2010)
Chacma baboons	M	Free-range	13/1	MMF (non-inherited)	Feces	14 mo	Multiple	37 (7–56)	Stable Unstable	**S** > D **D** > S	Bergman et al. (2005)
Chacma baboons	F	Free-range	10/1	MMF (inherited)	Feces	17 mo	Multiple	26 (10–47)	?	D = S	Weingrill et al. (2004)
Chacma baboons	F	Free-range	21/1	MMF (inherited)	Feces	16 mo	Multiple	30	Unstable	D = S	Engh et al. (2006)
Chacma baboons	F	Free-range	22/1	MMF (inherited)	Feces	6 wk	Multiple	24	Stable Unstable	D = S **S** > D	Wittig et al. (2008)
Chacma baboons	F	Free-range	18/1	MMF (inherited)	feces	8 mo	Multiple	31	Stable Unstable	D = S D = S	Crockford et al. (2008)
Savannah baboons	M	Free-range	125/5	MMF (non-inherited)	Feces	9 yr	Multiple	36	Stable Unstable	**D,S** > I **D,S** > I	Gesquiere et al. (2011)
Japanese macaques	M	Free-range	6/1	MMF (non-inherited)	Feces	6 mo	Mating	42 (34–55)	Stable	**D** > S	Barrett et al. (2002)
Assamese macaques	M	Free-range	6/1	MMF (non-inherited, but tolerant)	Feces	5 mo	Gestation and mating	35 (23–41)	Stable Unstable	D = S **S** > D	Ostner et al. (2008)
Rhesus macaques	F	Free-range	70/6	MMF	Serum (stimulated)	5 mo (across 2 yr)	Multiple	1–2	?	D = S	Hoffman et al. (2010)
Long-tailed macaques	M	Free-range	16/1	MMF	Feces	4.5 mo	Mating	15	Unstable	D = S	Girard-Buttoz et al. (2009)
Cynomolgus macaques	M	Indoor cages	20/5	MM (normally MMF, non-inherited)	Plasma (basal and stimulated)	3 days (across 4 mo)	na	B: 1–3 S: 4	Stable Unstable	B: **D** > S S: **S** > D B: **S** > D	Czoty et al. (2008)
Mandrills	F	Semi-free-range	19/1	MMF (inherited)	Feces	12 mo	Multiple	18 (3–36)	Stable	D = S	Setchell et al. (2008)
Sykes' monkeys	F	Free-range, provisioned	11/1	MMF (non-inherited)	Feces	16 mo	Gestation and mating	165	Stable Unstable	D = S **S** > D	Foerster and Monfort (2010)
Common marmoset	F	Free-range	6/3	MMF (non-inherited, high rep. skew)	Feces	10–16 mo	Multiple	69 (49–102)	Stable Unstable	D = S **S** > D	Sousa et al. (2005)
Golden lion tamarin	M	Free-range	24/14	MMSF (non-inherited, high rep. skew)	Feces	16 mo	Lactation and mating	8	?	D = S	Bales et al. (2006)
Tufted capuchins	M	Free-range	6/1	MMF (non-inherited)	Feces	12 mo	Multiple	> 14	Stable Unstable	D = S D = S	Lynch et al. (2002)
Verreaux's sifakas	M	Free-range	10/5	MMF (non-inherited)	Feces	5 mo	Birth and mating	32 (21–40)	Stable Unstable	D = S **D** > S	Fichtel et al. (2007)
Ringtailed lemurs	F	Free-range and semi-free-range	39/8	MMF (non-inherited, no repro skew)	Feces	2–3 wk	Lactation	6 (1–13)	Stable Unstable	D = S **D** > S	Cavigelli et al. (2003)
Ringtailed lemurs	F	Free-range	45/7	MMF (non-inherited, no repro skew)	Feces	4–12 mo	Lactation, weaning, gestation	~10 (3–21)	?	D = S	Pride (2005)
Ringtailed lemurs	M	Free-range	14/3 13/3	MMF (non-inherited, no repro skew)	Feces	4–5 mo (across 3 yr)	Gestation and mating	(5–6) (10–12)	Stable Unstable	D = S D = S	Gould et al. (2005)
Ringtailed lemurs	M, F	Semi-free-range	32/3	MMF,SMMF (non-inherited, no repro skew)	Feces	51 mo (across 7 yr)	Multiple	43	Stable Unstable	D = S **I** > S,D (F)	Starling et al. (2010)
Carnivores											
Spotted hyenas	F	Free-range	110/8	MMF (non-inherited)	Feces	3–8 yr	Multiple	2	Stable	**S** > D (if not lactating)	Goymann et al. (2001)
N. American wolves	M, F	Free-range	?/3	MMF family groups (non-inherited, med. rep. skew)	Feces	19 mo	Non-breeding, breeding	4	Stable Unstable	**D** > S **D** > S	Sands and Creel (2004)

Table 1 (*continued*)

Species	Sex	Environment/ housing	No. of animals/ groups	Social structure (dominance)	Medium	Period of CORT sampling	Reproductive state	Samples per ind'l (range)	Social/ enviro. stability	Dom. vs. sub. CORT	Authors (year)
Iberian wolves	M, F	Free-range	?/1	MMF family groups (non-inherited, high rep. skew)	Feces	15 days (across 15 mo)	Non-breeding, breeding	?	?	**D**>S	Barja et al. (2008)
Ungulates											
Rock hyraxes	M	Free-range	22/6	MMF (non-inherited)	Hair	10 mo (across 5 yr)	Premating	5	Stable?	**D**>S	Koren et al. (2008)
Bison	M	Large refuge	87/?	MMF	Feces	5 wk (across 3 mo)	Pre/post-rut, rut	3	Stable Unstable	D=S? **D**>S	Mooring et al. (2006)
Marsupials/rodents											
Common brushtail possum	M	Outdoor enclosure	20/10	MMSF (normally solitary)	Plasma (basal?)	5 mo	Non-breeding, hierarchy formation	14 6	Stable Unstable	D=S D=S	Wehi et al. (2006)
Laboratory rats (Long–Evans)	M	Visible burrow system	?	MMF (non-inherited)	Serum (basal?)	2 wk	na	1 1–2	Stable Unstable	D=S **S**>D	Hardy et al. (2002)
Rat-like hamsters	F	Single-caged	48/na	Daily staged fights (normally solitary)	Serum (basal?)	1 day	Non-breeding, breeding	1	Stable Unstable	D=S D=S	Wang et al. (2006)
Birds											
Ring-necked pheasants	M	Outdoor enclosure	13/1	MMF (non-inherited)	Plasma (basal?)	1 day	Mating	1	Unstable?	**D**>S	Mateos (2005)
Mallard ducks and Pintail ducks	M, F	Aviary	33/2	MMF	Plasma (basal and stimulated)	1 day 1 day	Pre-mating	B: 1 S: 2	Stable	B: D=S S: **D**>S	Poisbleau et al. (2005)
Superb starlings	M, F	Free-range	257/9	MMF family groups (low rep.skew)	Plasma (basal and stimulated)	4 mo? (across 4 yr)	Pre-breeding	B: 1+ S: 3–4	Stable Unstable	B: D=S S: D=S **S**>D	Rubenstein (2007)
Mountain chickadees	M, F	Pair-caged	24/12	?	Plasma (basal and stimulated)	2 day (across 3 mo)	?	B: 2 S: 3	Stable	B: D=S S: **D**>S	Pravosudov et al. (2003)
Fish											
Brown trout	M, F	Free-range	21/3	?	Plasma (basal)	1 wk?	?	1	Stable?	**D**>S	Sloman et al. (2008)

Key:
Environment: refers to housing conditions. 'Free-range' groups did not receive human food supplementation, all other groups received some form of food/water/shelter supplementation.
Animals/groups: refers to the total number of animals/total number of social groups in the study.
Social structure (dominance): codes in this column indicate—MMF (multi-male, multi-female), SMMF (single-male, multi-female), MMSF (multi-male, single-female). Information in parentheses indicate how dominance is established in the species/groups (inherited vs. non-inherited) and whether reproductive skew is high or low. In groups with high reproductive skew, dominant individuals are the only breeding animals.
Medium: refers to the biological sample type used for glucocorticoid measures. For fecal and urinary measures, the idea is that these measures most likely include an average estimate of both basal and stimulated glucocorticoid production averaged over time (i.e. metabolic rate and gut transit time). For blood measures (plasma, serum), we indicate whether basal (undisturbed) or stimulated levels were measured. Estimates of basal levels come from blood samples collected within minutes of animal disturbance whereas most stimulated measures come from blood samples collected 20–60 min after capture (Hoffman et al., 2010 represents an exception to this in that their stimulated blood samples were collected 24 h following capture).
Period of CORT sampling: refers to how many days, weeks (wk), months (mo), or years (yr) samples were collected. This column provides an indication of whether the sampling represents mean glucocorticoid production over a relatively long or short period.
Reproductive state: refers to reproductive state of study animals and/or group mates if study animals were males.
Samples/individual: 'B' refers to basal glucocorticoid measures, 'S' refers to stimulated glucocorticoid measures.
Social/enviro. stability: these ratings were based primarily on descriptions of the social conditions in each paper. Mating periods in seasonally-breeding species with multiple breeding males (either as members or visitors) were considered to be inherently unstable social periods. In addition, new group formations and periods/groups/seasons that showed increased aggression were considered unstable.
[a] All studies were conducted with adult animals (i.e. beyond the earliest known reproductive age), with the exception of Anestis (2005) which was conducted with juvenile/adolescents.

and environmental contexts (e.g. caged vs. free-ranging individuals) suggest a more complicated relationship between social status and stress physiology. Two review articles investigating the social rank–glucocorticoid production relationship across many species concluded that subordinate individuals do not always have higher glucocorticoid production than dominants. Specific circumstances associated with social status, like reproductive effort, degree of exposure to aggression, and level of social support, have to be considered to best determine the relationship between social status and glucocorticoid production (Abbott et al., 2003; Creel, 2001). These reviews stimulated a series of follow-up studies in free-ranging animals

The studies that have been published on the relation between social status and glucocorticoid production primarily in free-ranging animals are summarized in Table 1. Many of the reviewed studies were conducted across long periods of time (months to years) which provides important power to these naturalistic studies. Of the

32 studies conducted with free-ranging or complexly-housed animals, the majority were conducted with primates (22), with just a handful of studies on carnivores (3), ungulates (2), marsupials/rodents (2), birds (2), and fish (1). Studies were evenly split between male and female subjects.

Similar to human studies, there is a lot of variance in how dominance status relates to glucocorticoid production (Table 1). However, in free-ranging animals there is rather good evidence that social status is related to glucocorticoid production most often during periods of social instability (e.g. during the mating season in seasonal breeders when aggression levels increase, or during periods of social group change like immigration events, or other periods of increased aggression). Specifically, status was related to glucocorticoid production in 15 of 21 cases of instability, but only 8 of 24 cases of stability. Interestingly, when there was a relationship between status and glucocorticoid production there was no consistent direction to this relationship; i.e., elevated glucocorticoid production was observed among low-ranking subjects in nine studies and among high-ranking subjects in twelve studies. However, the sex of the study animals revealed a consistent pattern. Most studies with males showed greater glucocorticoid production in dominant compared to subordinate animals (10 of 13 studies; with exceptions to this trend in one study of male chacma baboons, and in Assamese macaques and superb starlings), while a majority of studies with females showed greater glucocorticoid production in subordinate vs. dominant animals (6 of 10 studies; with exceptions being in female N. American and Iberian wolves, ringtailed lemurs, and brown trout). (Studies in which no relationship was observed between status and glucocorticoid production were equally split between the sexes: 13 male studies, 12 female studies.) Duration of the study and the average number of samples collected from each individual did not influence the above results. At a gross level, these results suggest that dominance rank or social status are not clearly related to glucocorticoid production, but when social groups are unstable there is the greatest divergence in glucocorticoid production between high- and low-status individuals. Furthermore, low rank is most often associated with elevated glucocorticoid production in females, whereas high rank is more often associated with elevated glucocorticoid production in males.

These disparate findings suggest that specific correlates of animal social status, such as exposure to or initiation of aggression, access to resources, or protection from danger, may be more closely associated with glucocorticoid production than social rank alone (Abbott et al., 2003; Creel, 2001, 2005; Goymann and Wingfield, 2004). Of the 12 studies that investigated the relationship between dominance-related aggression and glucocorticoid production, five showed a significant positive relationship between aggressive behavior and glucocorticoid levels (Cavigelli et al., 2003; Crockford et al., 2008; Gesquiere et al., 2011; Mateos, 2005; Muller and Wrangham, 2004), three showed a significant positive relationship between aggression received and glucocorticoid levels (Crockford et al., 2008; Ostner et al., 2008; Thompson et al., 2010), and seven showed no relationship between aggression initiated or received and glucocorticoid levels (Anestis, 2005; Creel, 2005; Girard-Buttoz et al., 2009; Lynch et al., 2002; Pride, 2005; Sands and Creel, 2004; Weingrill et al., 2004). This diversity of results could not be attributed to sex- or species-specific effects. Thus, although it has been suggested that social status must be dissected into its component parts to best understand how social status relates to glucocorticoid production, it is clear that this dissection must take into account more than just rates of aggression initiated or received (e.g. Abbott et al., 2003; Cavigelli et al., 2003; Creel, 2001, 2005; Goymann and Wingfield, 2004).

Because social status is necessarily related to its component parts and status seems to relate to glucocorticoid production only at times of instability or high conflict, it is possible that a portion of the relationship between social status and health may result from status-specific glucocorticoid production. If an individual within a group maintains relatively stable status over a life time and no long-term consequential alterations in glucocorticoid production, it may still experience shorter intervening periods of social unrest during which its social status predicts its glucocorticoid regulation, which may be enough to influence immune function during a critical period of disease susceptibility. Such complex relationships would be difficult to identify and would also involve other health-related systems (e.g. neurological systems, cardiovascular systems, etc.).

In the above studies, there is an unstated, subtle assumption that social status affects activation of the physiological stress response systems instead of the reverse. However, there is little longitudinal or experimental work to confirm the direction of causality between social status and glucocorticoid production. In the 1970's Rowell (1974) theorized that social-living animals with particularly sensitive physiological responses to environmental challenges would be more dominant in their social groups because of their increased reactivity. Although it is easy to assume that social status will influence stress physiology, the opposite is also possible. A handful of studies that have measured glucocorticoid reactivity prior to and following establishment of social rank suggest that dominance status seems to influence glucocorticoid production and that the reverse seems less likely (Czoty et al., 2008; Gust et al., 1993; Øverli et al., 2004; Roberts et al., 2007; c.f. Tuchscherer et al., 1998).

Similar to some human studies, animal research shows that maternal social status at the time of offspring conception or birth predicts adult offspring glucocorticoid production. In free-ranging baboons, a mother's social rank at the time of conception predicted sons' fecal glucocorticoid levels 6 years later as they reached adulthood (Onyango et al., 2008). In fact, mother's rank was a better predictor of male offspring glucocorticoid production than the offspring's own rank at the time of hormonal measurement. This idea of long-term 'glucocorticoid programming' based on maternal social status warrants further investigation.

5.3. Human–animal comparison

The diverse results reviewed above serve to caution researchers that are interested in modeling the human condition in an animal system. If the goal is to model the human condition, it is important to identify an appropriate animal species living in the appropriate context (e.g. free-ranging vs. cage, stable vs. unstable, etc.). Specifically, it would be ideal to use a species in which low social status has been associated with heightened circadian glucocorticoid production (or decreased glucocorticoid regulation), since at present there is more evidence to indicate that humans with lower SES have heightened glucocorticoid production. Table 1 provides a comprehensive list of which species may be the most appropriate models.

In animal studies, low status was more often associated with elevated glucocorticoid production in females, while high status was more often associated with elevated glucocorticoids in males. These sex differences in the glucocorticoid–social status relationship have not been extensively studied in humans. There is some indication that sex differences in humans may be the opposite of what we identified in the current review of animal studies—males of low status may produce more glucocorticoids than high status males, whereas females of low status may produce less glucocorticoids than high status females (e.g. Kunz-Ebrecht et al., 2004; Steptoe et al., 2003; c.f. Wright and Steptoe, 2005). These sex differences in the relationship between social status and glucocorticoid production require further investigation in both humans and animals.

The final difference between animal and human studies comes from methodology. The majority of animal studies in the natural habitat involve fecal or urinary measures of glucocorticoid production whereas human studies rely on salivary or blood measures. These differences are important because the two kinds of measures (feces/urine vs. saliva/blood) capture different aspects of glucocorticoid production. Salivary and blood measures capture momentary levels of glucocorticoid

production while fecal and urinary samples provide an estimate of the average level of glucocorticoid production over several hours (sometimes as long as 24 h). Blood and saliva measures, if done rapidly and during periods of low activity or stress can represent an estimate of basal glucocorticoid production whereas fecal and urinary measures should represent an average of both basal (unstimulated) and stimulated production over a significant portion of time. This seemingly technical methodological distinction is important when comparing human and animal studies. Furthermore, stronger SES–glucocorticoid relationships in both human and animal studies that used repeated glucocorticoid sampling methods support the use of these methods in future studies.

6. Social status and immune function

6.1. Humans

Trends in disease with respect to SES are well documented, but an understanding of the inner mechanisms involved is limited. There is a clear trend toward greater prevalence of asthma, cardiovascular disease, diabetes, cancers, and infectious illness in lower socioeconomic classes (Chen et al., 2003, 2006; Cohen, 1999; Steenland et al., 2002; Steptoe et al., 2007; van Rossum et al., 2000). In many of these relationships, immune function alterations and consequent pathogen burden may be compelling factors in driving health outcomes (Steptoe et al., 2007).

In humans, there is a strong relationship between SES and pathogen burden. Pathogen burden is the measure of produced and circulating antibodies specific to a number of generally latent antigens, such as Cytomegalovirus (CMV), Herpes Simplex Virus type 1 (HSV-1), and Epstein–Barr virus (EBV). This measure reflects the continued seroprevalence of targeted antigens in an individual. When an individual's cell-mediated immunity is suppressed, antibodies must be produced specific to these latent viruses to combat their resurgence within the body. Thus, higher levels of detected antibodies suggest dampened cell-mediated immune response (Dowd et al., 2009a; Glaser and Kiecolt-Glaser, 1997; McDade et al., 2000). Numerous studies have suggested an inverse relationship between an individual's SES and circulating antibodies for these pathogens (Dowd et al., 2007, 2009a, 2009c; Simanek et al., 2008; Steptoe et al., 2007; Zajacova et al., 2009). In an investigation of the Whitehall II cohort, individuals in lower grades of employment display evidence of heightened HSV-1 burden (Steptoe et al., 2007). In another study, children in lower SES families exhibit higher levels of CMV antibodies, implying that the relationship may arise as early as childhood (mean age: 11 years, Dowd et al., 2009c). In these studies, SES is determined by education level, occupational grade, family income, or some combination of these components.

Separately, education, income, and race/ethnicity can be strong, independent predictors of pathogen burden (Aiello et al., 2009; Dowd and Aiello, 2009; Dowd et al., 2007; Zajacova et al., 2009). Children belonging to impoverished families show an inverse relationship between income and CMV antibodies only until income crosses above the poverty line (Dowd et al., 2012). By contrast, an inverse relationship lacking such a threshold effect is observed when education is incorporated into the primary SES measure (Dowd et al., 2007, 2009c; Simanek et al, 2008; Zajacova et al., 2009).This simple difference between SES components elucidates the need to consider income, education, and occupational grade as separate entities when comparing them to immune measures.

Of the eleven studies surveyed for relationships between CMV burden and SES, 10 suggested an inverse relationship in which individuals with lower SES had higher CMV levels (Dowd and Aiello, 2009; Dowd et al., 2007, 2009a, 2009c, 2012; Mustakangas et al., 2000; Shen et al., 1992; Simanek et al., 2008; Steptoe et al., 2007; Zajacova et al., 2009). Of these studies, three involved children as young as 11 years old and one studied pregnant women (Dowd et al., 2009a, 2009c, 2012; Mustakangas et al., 2000). Five studies used education level as a primary SES component (Dowd et al., 2007, 2009a, 2009c; Simanek et al., 2008; Zajacova et al., 2009), and two used only income measures (Dowd et al., 2009a, 2012). Only one study was conducted outside of western populations. Conducted on Taiwanese mothers and children, the study failed to produce a significant relationship between CMV and SES. In this population, the majority of pregnant women were seropositive for CMV, suggesting that CMV transmission to children could be influenced by infected breast milk and the genital tract instead of social status related factors (Shen et al., 1992). Of six studies relating SES and HSV-1, another latent virus, all suggest an inverse relationship between education level of the individual and HSV-1 seroprevalence (Bünzli et al., 2004; Dowd et al., 2007, 2009c; Simanek et al., 2008; Steptoe et al., 2007; Zajacova et al., 2009).

This trend may be partially due to the interplay between the HPA axis and the immune system. In response to stressful stimuli, heightened glucocorticoid levels can suppress cell-mediated immune function. With this process inhibited, dormant antigens replicate, forcing the humoral immune system to accommodate for increased virus levels with heightened antibody production (Dowd et al., 2007; Glaser and Kiecolt-Glaser, 1994, 2005; Sorensen et al., 2009).

The presence of antibodies reflects the failure of cell-mediated processes to control the infection. Reactivation of CMV due to poor cell-mediated immunity has also been shown to contribute to T-cell immunosenescence, further damaging the cell-mediated response (Dowd and Aiello, 2009; Dowd et al., 2007; Koch et al., 2006). Furthermore, CMV has been detected in the tissues of individuals with inflammatory disorders, suggesting a link to the inflammatory aspect of the immune system as well (Dowd et al., 2009a; Söderberg-Nauclér, 2006).

Inflammatory responses are another aspect of immunity that can be influenced by stressors potentially originating from social status (Owen et al., 2003). C-reactive protein (CRP) is a pro-inflammatory biomarker associated with coronary heart disease and other syndromes (Libby et al., 2002; Owen et al., 2003; Rosvall et al., 2008). Thirteen of 14 studies surveyed confirmed an inverse relationship between SES and CRP (Demakakos et al., 2008; Friedman and Herd, 2010; Gimeno et al., 2007, 2008; Gruenewald et al., 2009; Hagger-Johnson et al., 2012; Maksimović et al., 2008; McDade et al., 2011; Nazmi and Victora, 2007; Petersen et al., 2008; Saijo et al., 2008; Schafer et al., 2011; Seeman et al., 2008; Tabassum et al., 2008). Two of these studies only found a relationship between income and CRP, one of which was only significant in males, and another three studies found a link between CRP levels and education only (Demakakos et al., 2008; Friedman and Herd, 2010; Gimeno et al., 2008; Maksimović et al., 2008; McDade et al., 2011; Petersen et al., 2008). One of the above studies was a meta-analysis that found 19 inverse relationships between CRP levels and SES out of 20 studies prior to 2006 (Nazmi and Victora, 2007). While there is some conflict among papers regarding SES measures, it is clear that low SES is associated with higher levels of this pro-inflammatory biomarker. Low SES has also been linked to high levels of pro-inflammatory cytokine IL-6 and fibrinogen suggesting that social status may influence multiple components of the inflammatory response (Friedman and Herd, 2010; Gruenewald et al., 2009; Petersen et al., 2008; Pollitt et al., 2008).

SES early in life can be a powerful influence into adulthood regardless of socioeconomic changes. Adults that had a low SES background during childhood display up-regulation of genes involved in leukocyte signaling. Genes containing glucocorticoid receptor response elements are down-regulated in the same group of individuals, perhaps allowing for increased pro-inflammatory immune signaling (Miller et al., 2009). Despite their current SES, adults who spent their childhood in a low SES environment also display higher cortisol levels and increased IL-6 expression, suggesting a greater stress response, glucocorticoid resistance, and consequent iterations in pro-inflammatory signaling (Miller et al., 2009; Packard et al., 2011; Saxton et al., 2011; Zhang et al., 2006).

Table 2
Studies comparing animal social status to immune function in both caged and non-caged animals. The experimental group(s) that exhibit greater levels of the designated immune measure are displayed in bold. Key below provides further explanation of columns and information within columns. The text provides information on literature search procedures and criteria.

Species (strain)	Sex	Environment/housing	Primary dominance measure	Antigen/immune challenge	Medium	Immune measure	Social stability	Dom. vs. Sub.	Authors (year)
Primates									
Chimpanzees	M/F	Group-housed (mixed sex)	Aggression/submission	None	Blood	IgG, IgM	Stable	**S**>D	Masataka et al. (1990)
Chimpanzees	M	Group-housed (9)	Aggressive vocalization	Epstein–Barr virus chimp	Blood	Viral antibodies	Both	D=S	Yamamoto et al. (2010)
						Viral reactivation	Unstable	[**D**>S]	
							Stable	D=S	
Yellow baboons	M/F	Free-range	Aggression	Intestinal parasites	Feces	Parasite egg count	Stable	[**D**>S] (M) D=S (F)	Hausfater and Watson (1976)
Olive baboons	M	Free-range	Aggression/submission	None	Blood	Lymphocyte count	?	**D**>S	Sapolsky (1993)
Olive baboons	M/F	Free-range	Aggression/submission	Helminth parasite	Feces	Parasite infection frequency	Stable	D=S	Müller-Graf et al. (1996)
Savannah Baboons	M	Free-range	Aggression/submission	Naturally-occurring injury/illness	Skin/other	Incidence	?	**D**>S	Archie et al. (2012)
						Healing rate		**D**>S	
Cynomolgus macaques	M	Group-housed	Aggression/submission	Tetanus toxoid	Blood	IgG	Both	**D**>S (marginal)	Cunnick et al. (1991)
		(5) (1/3 SDR)				Tetanus antibodies (1° response)	Both	**S**>D	
						Tetanus antibodies (2° response)	Both	D=S	
Cynomolgus macaques	M	Group-housed (5) (1/2 SDR)	Aggression/submission	Adenovirus	Blood	NK, T-cell counts	Both	D=S	Cohen et al. (1997)
						B cell count		**S**>D	
					Nasal secretion	Viral intranuclear inclusion (2, 4 pi)		[**S**>D]	
				ConA, PHA	Blood	T-cell proliferation		D=S	
Rhesus macaques	F	Free-range	Aggression/submission	None	Blood	IL-6, IL-8	Stable	D=S	Hoffman et al. (2011)
Mandrills	F	Semi-free-range	Avoidance behavior	Intestinal parasites	Feces	Parasite prevalence	Stable	D=S	Setchell et al. (2007)
Red-fronted lemurs	M	Free-range	Aggression/submission	Intestinal parasites	Feces	Nematode infection intensity	Unstable	D=S	Clough et al. (2010)
						Protozoan infection intensity		[**D**>S]	
Ungulates									
Pigs	F	Group-housed (3) (1/2 ship-stressed)	Aggression	K562	Blood	NK cytotoxicity before stress	Stable?	D=S	McGlone et al. (1993)
						NK cytotoxicity after stress	Unstable?	**D**>S,I	
Pigs	M/F	Group-housed (3) (1/2 heat-stressed)	Aggression/submission	SRBC	Blood	IgG, SRBC antibodies	Both	D=S	Morrow-Tesch et al. (1994)
						Monocyte/neutrophil count when no stress	Stable	D=S	
							Unstable	**S**>D	
						Monocyte/neutrophil count during heat stress	Stable	D=S	
							Unstable	**D**>S	
				ConA, PHA		T-cell proliferation	Both	D=S	
				PWM		B-cell proliferation	Both	**I**>D,S	
Pigs	M/F	Group-housed (3) (ship-, heat-, cold-stressed)	Aggression	None	Blood	Total WBC count, differentials	Stable	D=S=I	Hicks et al. (1998)
				K562		NK cytotoxicity after cold stress		**D**,**I**>S	
						NK cytotoxicity after heat and ship stress and no stress		D=S=I	
				Staphylococcus aureus		Neutrophil chemotaxis/phagocytosis		D=S=I	
				PHA		T-cell proliferation		D=S=I	
				SRBC		IgG, SRBC antibodies		D=S=I	

Pigs	M/F	Group-housed (9)	Aggression/submission	ConA, PHA	Blood	T-cell proliferation	Unstable	**D**>S	Tuchscherer et al. (1998)
				PWM		B-cell proliferation		**D**>S	
						IgG		**D**>S	
						IgM, C3c, alpha2-macroglobulin		D=S	
Pigs	M/F	Group-housed (3) (heat-, crowding-stressed)	Aggression/submission	SRBC	Blood	Total WBC count	Unstable	**D**>S	Sutherland et al. (2006)
						Neutrophil, lymphocyte, monocyte, eosinophil counts		D=S	
						IgG, SRBC antibodies		D=S	
				K562		NK cytotoxicity		**D**>S	
				ConA, LPS		Lymphocyte proliferation		D=S	
				hC5a, pIL-8		Neutrophil chemotaxis		D=S	
						Neutrophil phagocytosis		**D**>S	
Pigs	M/F	Group-housed (9)	Aggression and food competition	ADV	Blood	Ex-vivo lymphocyte proliferation	Stable	**D**>S,I	Hessing et al. (1994)
						ADV antibodies		**S**>D,I	
						Leukocyte/neutrophil counts		**S**>D	
						Morbidity, mortality		[**S**>D,I]	
Pigs	M/F	Pair-housed	Aggression	ADV	Blood	Ex-vivo lymphocyte proliferation	Stable	**D**>S	de Groot et al. (2001)
							Unstable	**S**>D	
						Antiviral lymphocyte proliferation after vaccination	Both	**D**>S	
						IFN-γ (20 pi)	Both	**D**>S	
						IL-10	Both	D=S	
						IFN-γ: IL-10 (TH1:TH2 ratio)	Stable	D=S	
							Unstable	**S**>D	
						IgG1, IgG2	Both	D=S	
						IgG1:IgG2 ratio	Both	**D**>S (marginal)	
						IgM	Both	D=S	
						Fever response	Both	D=S	
						Virus excretion	Stable	D=S	
							Unstable	**D**>S	
Rodents									
Syrian hamsters	F	Group-housed (10)	Post-stress displacement and walking over	Malignant melanoma tumor	Under skin	Tumor growth	Stable	D=S	Temoshok et al. (1987)
							Unstable	[**S**>D]	
Brandt's voles	M	Group-housed (3)	Food competition	Human IgG	Spleen	Human IgG antibody	Stable	**S**>D	Li et al. (2007a)
						Spleen weight		**S**>D	
Rats (Long–Evans)	M	C: single-housed D, S: pair-housed	Aggression/submission During 10-d resident–intruder cohabituation	None	Thymus	Thymus weight	Stable?	**D**>S	Raab et al. (1986)
				ConA	Spleen	T-cell proliferation		D=S=C	
				PHA		T-cell proliferation		**D,C**>S	
				PWM		B-cell proliferation		**D,C**>S	
Rats (Long–Evans)	M	Group- (3 M, 1 F) to single-housed	Aggression toward group-intruder on d10	Seoul virus	Blood	Seoul virus antibodies, shedding (10, 15, 20, 30, 40 pi)	Stable?	D=S	Hinson et al. (2006)
					Spleen, thymus	Spleen, thymus weight (40 pi)		D=S	
Rats (Sprague–Dawley)	M	Pair-housed	Food competition	None	Hypothalamus	IL-1 levels after restraint stress with shaking	Stable	**S**>D	Barnum et al. (2008)
						IL-1 levels after foot shock		S=D	
Mice (Balb/c)	M	Group-housed (5)	Aggression/submission when exposed to aggressive intruder (SDR)	Herpes simplex virus—type 1	Eye	Viral shedding/reactivation (~50 pi)	Unstable	[**D**>S]	Padgett et al. (1998)
Mice (Balb/c)	M	Group-housed (5)	First copulation when female introduced	*Trypanosoma cruzi* parasite	Blood	Parasite burden	Stable	[**S**>I>D]	Schuster and Schaub (2001)
Mice (Balb/c ByJ Ico)	M	Group-housed (5) (1/2 SDR)	Food competition	KLH	Blood	Anti-KLH antibodies (IgG)	Both	D=S	Merlot et al. (2004)
				LPS	Spleen	B-cell proliferation	Stable	**I**>D,S	
							Unstable	D=S	
						IFN-γ, IL-6, IL-10	Both	D=S	
				none		IFN-γ, IL-10	Stable	D=S	
				BCG and TB		Speed of IFN-γ, IL-10 responses	Stable	**D**>S	

(continued on next page)

Table 2 (*continued*)

Species (strain)	Sex	Environment/housing	Primary dominance measure	Antigen/immune challenge	Medium	Immune measure	Social stability	Dom. vs. Sub.	Authors (year)
						IFN-γ:IL-10 ratio	Stable	D = S	
						Bacterial load	Stable	D = S	
Mice (Balb/c OlaHsd)	M	Single-housed	Aggression/submission to repeated exposure to aggressive intruder	None	Spleen	Spleen weight	Unstable?	D = S = C	Savignac et al. (2011)
					Blood	IL-1β, IL-6, chemokine mKC increase after social stress		**S** > D	
			(C57BL/6)			TNF-α, IFN-γ, IL-12p70, IL-10 increase after social stress		D = S	
Mice (C3H/HeJ)	M	Single-housed	Submission to repeat	None	Spleen	Spleen weight	Unstable	**S** > D	Hardy et al. (1990)
			Exposure to foreign	YAC-1		NK cytotoxicity		D = S	
			Mouse in new arena	ConA, PHA		T-cell proliferation		**D** > S	
				STM		B-cell proliferation		D = S	
Mice (C57BL/6J)	M	Pair-housed	Aggression/submission	SRBC	Spleen	Rosette-forming cell count	Stable	**D** > C > S	Devoino et al. (2003)
							Unstable	**DD, SD** > SS,DS	
Mice (C57BL/6)	M	Pair-housed (with barrier)	Aggression/submission	SRBC	Spleen	Rosette-forming cell count	Stable	**D,C** > S	Idova et al. (2007)
Mice (C57BL/6)	M	Pair-housed	Aggression/inspection response to intruder after SDR	None	Spleen	Spleen weight, splenocyte count	Stable	D = S	Avitsur et al. (2007)
							Unstable	**S** > D,C	
				LPS	Spleen	TNF-α, IL-6	Stable	D = S	
							Unstable	**S** > D,C	
Mice (CD-1)	M	D,C: single-housed S: group-housed (4)	Submission when exposed to novel	None	Blood	IL-1β: low CORT mice after repeated social encounters	Unstable	**S** > D,C	Audet et al. (2010)
			Aggressive resident			IL-1β: high CORT mice, low CORT mice after single encounter		D = S = C	
						IL-6: low CORT mice		**S** > D,C	
						IL-6: high CORT mice after single social encounter		**D,S** > C	
						IL-6: high CORT mice after repeated social encounters		D = S = C	
						IL-10: all mice		D = S = C	
					Brain (PFC)	IL-1β mRNA: low CORT mice		**S** > D,C	
						IL-1β mRNA: high CORT mice		**D** > C	
						IL-6 mRNA: low CORT mice		D = S = C	
						IL-6 mRNA: high CORT mice after single social encounter		**D** > S,C	
						IL-6 mRNA: high CORT mice after repeat social encounters		D = S = C	
						TNF-α mRNA: all mice		D = S = C	
Mice (Swiss CD-1)	M	Group-housed Siblings (3–7) and pair-housed non-siblings	Aggression/submission to intruder	ConA	Blood	IL-2, IL-4, IL-10, IFN-γ	Stable(sibs)	D = S	Bartolomucci et al. (2001)
						IL-4, IFN-γ	Unstable	D = S	
						IL-2, IL-10		**DD,SD, SS** > DS	
					Spleen	T-cell proliferation	Both	D = S	
Mice (CFLP)	M	Group-housed (6)	Aggression	*Babesia microti*	Blood	IgG: decline during group-living	Stable?	D = S	Barnard et al. (1993)
						Infection clearance rate		**S** > D	
Mice (CFLP)	M	Group-housed (3, 6, or 10)	Aggression	None	Blood	IgG: decline during group-living	Stable?	[**D** > S]	Barnard et al. (1994)
				Babesia microti		Infection clearance rate		**S** > D	
Mice (DBA/2)	M	Group-housed (3 or 9)	Aggression/submission	Moloney leukemia virus	Thymus	Development of leukemia	Stable	[**S** > D]	Ebbesen et al. (1991)

Mice (DD/S)	M	Group-housed (3 or 5)	Aggression/submission	Shionogi mouse mammary carcinoma	Peritoneum	Tumor growth	Stable Unstable	[**S**>D] [**D**>S]	Grimm et al. (1996)
Mice (OF1)	M	Pair-housed	Aggression/submission	SRBC	Spleen	IL-1 IL-2	Stable Unstable Both	D=S **D**>S D=S	Fano et al. (2001)
Birds									
House finches	M/F	Group-housed (4 M, 4 F) (low-, high-competition)	Food competition	SRBC	Blood	SRBC antibodies	Stable	**D**>S	Hawley et al. (2006)
House finches	M/F	Group-housed (5 M, 5 F)	Food competition	SRBC PHA MG	Blood	SRBC antibodies T-cell proliferation MG infection severity (eye inflammation level)	Stable	**S**>D **D**>S [**S**>D] (M) D=S (F)	Hawley et al. (2007)
Greenfinches	M	Group-housed (4)	Food competition	Sindbis virus	Blood	Rate of virus clearance	Stable	**D**>S	Lindström (2004)
Fish									
Zebrafish	M/F	Group-housed (2 M, 2 F)	Aggression	None	Spleen	tnf-α expression ifng1-1 expression il-1β expression ifn1 expression	Stable	**D**>S (M) **D**>S (F) **S**>D (M) **D**>S (F) **S**>D (M) **D**>S (F) D=S (M) **D**>S (F)	Filby et al. (2010)

Key:
Environment/housing: refers to housing conditions. If group-housed, the number of individuals in each group is indicated in parentheses. Other manipulations of environmental conditions are included in this column as well (SDR stands for social disruption paradigm—e.g. Merlot et al., 2004).
Primary dominance measure: refers to the primary measure used to assess social rankings.
Antigen challenge: refers to the antigen used to stimulate an immune response in subjects. Codes include ADV for Aujeszky disease virus (also known as pseudorabies), BCG for Calmette–Guérin Bacilli, ConA for concanavalin A, DNP-Asc for dinitrophenyl coupled-ascaris extract, K562 for human chronic leukemia tumor cells, KLH for keyhole-limpet hemocyanin, MG for *Mycoplasma gallisepticum*, PHA for phytohaemagglutinin, PWM for pokeweed mitogen, SRBC for sheep red blood cell, STM for *Salmonella typhimurium*, TB for tuberculin, YAC-1 for T-cell lymphoma derived from Moloney leukemia virus.
Medium: refers to the cell-type collected/used for the immunological measure.
Immune measure: refers to the parameter(s) used to assess immune function. pi refers to when the measure was collected, in days, after inoculation.
Social stability: was inferred based on (1) the amount of time animals had lived with their social partners (<1 week = unstable, > week = stable), (2) whether manipulations of social structure were conducted (social manipulations = Unstable), and (3) indication by the authors of the relative consistency of individual dominance ranks over time.
Dom vs. Sub: identifies the relationship between social status and immune response. Codes to indicate social status include DD for consistently dominant, SS for consistently subordinate, DS for role reversal from dominant to subordinate, SD for role reversal from subdordinate to dominant, and C for controls. Codes outside of these specific contexts are S for subordinate, I for intermediate, and D for dominant. Brackets [] surrounding the letters indicates that the immune measure was an estimate of poor immune function (i.e., parasite load or tumor growth) and thus the direction of the dominance relationship should be considered reversed in these cases.

These residual effects from early life experiences suggest an early life programming of immune and endocrine transcription mechanisms for later life. Interestingly, these effects can be partially rectified by early life support and maternal warmth (Chen et al., 2011). Such psychosomatic effects imply that this reprogramming is at least partially stress-mediated and psychological. Furthermore, in a study comparing EBV antibody levels, SES, and social support in adult females diagnosed with breast cancer, social support was only 'protective' in more educated women, suggesting that the mediating effect of social support is wider-reaching in developing children than in stressed adults (Fagundes et al., 2012).

There is some suggestion that low SES may also be associated with increased antibody responses. Among children between ages 5 and 14, individuals in low social classes have heightened secondary immune biomarkers, like IgG and IgA, and appear less susceptible to severe respiratory infections than individuals in higher classes (Bolte et al., 1999). It is possible that increased frequency of lesser infections bolsters a low SES child's humoral immunity to more severe infections. Furthermore, it is possible that a spike in humoral activity (IgG and IgA) is merely an attempt to combat an infection that the suppressed cell-mediated immune system cannot (Dowd et al., 2009a; Glaser and Kiecolt-Glaser, 1997; McDade et al., 2000).

Social and environmental context has received limited attention due to the focus of research on fixed, relatively stable populations. Interestingly, there are a few studies that have been conducted in less stable societies, which may be a human parallel to unstable animal social systems discussed by Sapolsky (1983). In humans, the effect of unstable social status has been modeled using lifestyle incongruity (LI), a measure of disparity between SES and material lifestyle (i.e. current wealth—as measured by ownership of consumer goods and luxury items, Sorensen et al., 2009). This measure has been developed to account for different estimates of social status observed in changing social systems (e.g. when government shifts from a socialistic to a capitalistic system). In such societies, an individual with high SES (e.g. high education or job status) but relatively low access to material resources is indexed at a low LI score, whereas an individual with low SES but relatively high, or on par, access to resources is indexed as having a high LI score. Thus low LI is associated with a certain loss of status whereas high LI may be associated with a gain in social status. These LI scores provide a method to study the 'mismatch' between an individual's traditional SES and their relative access to material resources. In a study of an indigenous Siberian population undergoing rapid economic and cultural change, individuals with low LI had greater EBV antibody levels which were interpreted as indicative of dampened cellular-mediated immune function (Sorensen et al., 2009). By contrast, people who had higher LI scores had lower EBV antibody counts. However, another study in socially-involved Samoan adolescents showed that individuals with high LI had poorer cell-mediated immunity than individuals with low LI (McDade, 2001). This reversal may occur because an adolescent does not generally contribute to a family's socioeconomic status. Higher social integration and less influence over financial means may instead emphasize the cultural aspect of social instability over the risk of losing SES. In such a case, the conflict between the mingling traditional Samoan and western lifestyles may cause stress-induced reduction of cell-mediated immunity (McDade, 2002). The differential effects of cultural and social instability may prove an illuminating route for further investigation.

From the papers surveyed there appears to be an overall trend toward greater pathogen burden—or antibody activity to latent viruses—and greater inflammation in humans of lower SES. Even as an individual grows into adulthood, studies suggest that residual effects of low SES during childhood may remain regardless of adult SES. These trends point predominantly toward an inefficient, dampened cell-mediated response, causing a greater dependency on humoral immunity. While cell-mediated immunity is dampened, the humoral side of immune function seems to operate at higher levels, especially against severe infections. Interestingly, high SES individuals at risk for losing status in a socially unstable population may take on the characteristics of low SES individuals in stable social situations. Further research into lifestyle incongruity may shed light on the extent of these differences. The finding that social support and maternal warmth can alleviate potential SES-related programming of the immune system suggests a strong mediating effect of the psychological stress inherent in lower SES.

6.2. Other animals

Studying social rank and immune function in animals may yield information that is otherwise difficult to obtain through human subject research. For instance it is possible to test immune response following exposure to a specific virus and to simulate unstable, ever-changing hierarchies that may be used to tease apart the physiological mechanisms at work in shifting human SES (Devoino et al., 2003; Hawley et al., 2006; Hessing et al., 1994). Furthermore, it allows for more invasive investigations of tissues such as the thymus and spleen. Due to the range of opportunities with animals, studies have used a variety of immune challenge procedures and measures with a variety of species—from primates (11 studies) to pigs (7 studies), rodents (19 studies) and even birds (3) and fish (1) (Table 2 summarizes all the studies identified). A near majority of studies (18 of 41) were conducted with rodent males housed in controlled cage situations. In this section we review animal studies to identify potentially universal associations between social status and immune function. These studies can be categorized according to immune measures used—which range from natural disease susceptibility to measure of inflammatory processes, antibody production, and *in vitro* mitogen-stimulated lymphocyte proliferation.

At the disease resistance level, dominant pigs, primates, greenfinches and mice show greater evidence of viral resistance and clearance than subordinates. Of the six studies that measured initial viral resistance/clearance, five revealed this trend (Cohen et al., 1997; de Groot et al., 2001; Ebbesen et al., 1991; Hessing et al., 1994; Lindström, 2004). However, two of four studies that considered viral shedding/reactivation as their primary measure suggested that dominant males in relatively aggressive social systems (mice and chimps) were more susceptible to viral reactivation than subordinate males (Padgett et al., 1998; Yamamoto et al., 2010). Three studies measured tumor resilience, and they all indicated that dominant individuals were more resilient to tumor growth and risk of leukemia development than subordinates (Ebbesen et al., 1991; Grimm et al., 1996; Temoshok et al., 1987). On the other hand, no conclusive evidence was found for a relationship between parasite load and social rank (Clough et al., 2010; Hausfater and Watson, 1976; Müller-Graf et al., 1996; Schuster and Schaub, 2001; Setchell et al., 2007). Importantly, these last studies on parasite load were all conducted with free-ranging (or semi-free-ranging) primates where parasite exposure and resilience were not separately assessed (i.e. parasite exposure was not experimentally-controlled). The above studies provide a relatively strong indication that dominant individuals tend to be more resistant to viruses and cancer development compared to subordinate individuals, but unveil no clear status influence on susceptibility to parasites. To understand these trends, specific aspects of the immune system must be discussed.

One vital branch of the immune system that can be influenced by social rank is cell-mediated immunity. This subset of the immune response combats infection without the use of antibodies, but rather mobilization of cytotoxic T-cells, natural killer (NK) cells, and phagocytes such as macrophages (Janeway et al., 2005). One way to quantify a subset of cell-mediated immunity is with *in vitro* NK cytotoxicity assays, in which NK lysis of abnormal tissue (like tumor cells) is assessed (Hardy et al., 1990; Janeway et al., 2005). From the four studies that investigated NK cell activity, there was no clear relationship between status and cytotoxicity among pigs

and mice that were exposed to no stress or a brief period of a mild stressor (e.g. interaction with an intruder for one period of 10 min, Hardy et al., 1990; Hicks et al., 1998; McGlone et al., 1993). However, when a potentially severe or chronic stressor—like cold stress, crowding, or frequent social disruption—was involved, dominant animals tended to have greater NK activity than subordinates (Hicks et al., 1998; McGlone et al., 1993; Sutherland et al., 2006). Interestingly, no study was discovered that suggested greater NK activity in subordinates than dominants. These findings may help explain the above-mentioned increased cancer resistance in dominant individuals, since the ability of NK cells to recognize and kill abnormal tumor cells may play a role in preventing tumor growth (Janeway et al., 2005).

One aspect of immune function that is important for viral resistance is the ability to produce IFN-γ—a cytokine that helps fight viral infections by activating macrophages, a group of phagocytes that are also crucial to cell-mediated immunity (Janeway et al., 2005). Three of four studies surveyed indicated that dominant animals had greater IFN-γ responses to antigen than subordinates (de Groot et al., 2001; Filby et al., 2010; Merlot et al., 2004; c.f. Bartolomucci et al., 2001). These results may explain increased viral resistance among dominant vs. subordinate animals. IL-2, a cytokine that plays a role in T-cell activation, and IL-8, a cytokine that recruits neutrophils to inflammation sites, also provide a measure of cell-mediated immunity (Janeway et al., 2005). However, the three studies that investigated these cytokines failed to show a consistent relationship with social status (Bartolomucci et al., 2001; Fano et al., 2001; Hoffman et al., 2011).

Cytokines are not only involved in activation of specific cell-mediated responses, but also in pro-inflammatory responses. Of the different immune responses reviewed here, pro-inflammatory cytokine production (e.g. IL-1β, IL-6, TNF-α) was one of the few responses that was stronger in subordinates than dominants (see Table 2). In stable groups of mice and rhesus macaques, social rank did not relate to splenic or circulating IL-6 levels (Avitsur et al., 2007; Hoffman et al., 2011; Merlot et al., 2004), but in three studies that subjected animals to social disruption or otherwise unstable social hierarchies, submissive mice exhibited greater splenic or circulating IL-1β and IL-6 levels most likely mediated by glucocorticoid resistance (Audet et al., 2010; Avitsur et al., 2007; Savignac et al., 2011). Anti-inflammatory cytokine production was not found to relate to social status (Bartolomucci et al., 2001; de Groot et al., 2001; Merlot et al., 2004; Savignac et al., 2011). Despite this, a study investigating the dynamics of IL-10 levels over time indicated that dominant mice had faster rises and falls in this anti-inflammatory cytokine after exposure to an infectious, live antigen, demonstrating the importance of measuring these biomarkers at multiple time points to best estimate potentially relevant temporal dynamics of immune responses (Merlot et al., 2004). There have been a few studies that investigated brain levels of inflammatory cytokines with respect to social rank, but too few to make any clear conclusions (e.g., Audet et al., 2010).

One of the methods frequently used to measure immune cell activity—i.e. in vitro mitogen-stimulated T- and B-cell proliferation—is not clearly related to dominance status in animals. Mitogens are foreign substances, such as Concanavalin (ConA), Phytohemagglutinin (PHA), Pokeweed mitogen (PWM), and Lipopolysaccharide (LPS), that stimulate specific lymphocyte proliferation in tissues but do not replicate within the organism. ConA, PHA, and PWM each induce T-cell proliferation while PWM and LPS stimulate B-cells (Janeway et al., 2005). In theory, differential proliferation rates should reflect a subject's immune reactivity to a variety of immune challenges. However, conflicting results across many studies suggest that mitogen-stimulated lymphocyte proliferation is either not affected by social status, or that the proliferation measures may not provide accurate estimates of immune function specificity. Because mitogens are unable to replicate and incapable of simulating a true infection, they may not adequately simulate an immune system's response to live or naturally-occurring antigens. Of the ten studies that measured T-cell proliferation after mitogen stimulation, four studies showed heightened T-cell proliferation in dominant vs. subordinate animals (Hardy et al., 1990; Hawley et al., 2007; Raab et al., 1986; Tuchscherer et al., 1998) and six studies found no relationship between social rank and mitogen-stimulated effects (Bartolomucci et al., 2001; Cohen et al., 1997; Hicks et al., 1998; Morrow-Tesch et al., 1994; Raab et al., 1986; Sutherland et al., 2006). No studies suggested elevated in vitro T-cell proliferation in subordinate vs. dominant animals or any distinct effect for each individual mitogen. In two studies, ConA stimulation increased T-cell levels more in dominants vs. subordinate animals, but in another four, no relationship was suggested (Bartolomucci et al., 2001; Morrow-Tesch et al., 1994; Raab et al., 1986; Sutherland et al., 2006). Similarly, no conclusive results were found when PHA was used to stimulate cells (Hardy et al., 1990; Hawley et al., 2007; Hicks et al., 1998; Morrow-Tesch et al., 1994; Raab et al., 1986). Sometimes these conflicting results were observed within studies, with one mitogen revealing a relationship while another did not (Beitia et al., 2005; Raab et al., 1986). Results from T-cell proliferation studies lead to a similar conclusion made by Sapolsky (2004)—there is no clear evidence that social status in animals is related to an individual's ability to proliferate T-cells in the face of mitogenic stimulation. Similar findings arise from studies that measured mitogen-stimulated B-cell proliferation. Of six studies that measured this, four found no difference between dominant and subordinate B-cell proliferation (Hardy et al., 1990; Merlot et al., 2004; Morrow-Tesch et al., 1994; Sutherland et al., 2006) and two found that dominants produced approximately 30% more B-cells in response to pokeweed mitogen compared to subordinates (Raab et al., 1986; Tuchscherer et al., 1998). Interestingly, of the four studies that showed no difference between dominants and subordinates, only one used pokeweed mitogen while two used LPS. B-cells are responsible for antibody production, filling an intermediate role in the humoral response (Janeway et al., 2005). Thus, this measure of B-cell proliferation tells us about the first necessary steps involved in antibody production, but represents an early and potentially noisy aspect of B-cell function.

Seventeen studies were found that investigated levels of antibody activity in response to a variety of antigens. When antigens were used that the study organism would not normally encounter (e.g. non-replicating mitogens like keyhole limpet hemocyanin and sheep red blood cells), there were no consistent associations between social status and antibody production (Hawley et al., 2006, 2007; Hicks et al., 1998; Li et al., 2007a; Merlot et al., 2004; Morrow-Tesch et al., 1994; Sutherland et al., 2006; Tuchscherer et al., 1998). For example, in a pair of studies with greenfinches, antibody production induced by sheep red blood cells was three times greater in dominant birds compared to subordinate birds in one study and three times greater in subordinates compared to dominants in a follow-up study (Hawley et al., 2006, 2007). Because sheep red blood cells are not live, relevant antigens, they may yield inconclusive results like the above-mentioned mitogens. Focusing on immune challenges that mimic those found in the natural setting (e.g. bacteria and viruses) revealed clearer relationships. In six studies with bacterial, viral, or no challenges (*Babesia microti*, *Mycoplasma gallisepticum*, Aujesky disease virus, and Seoul virus) subordinate primates, pigs, mice, and finches showed greater primary antibody production than their dominant group-mates (Barnard et al., 1993, 1994; Cunnick et al., 1991; Hawley et al., 2007; Hessing et al., 1994; Masataka et al., 1990) while three studies in rats, pigs and chimps showed no significant difference between dominant and subordinate antibody production (de Groot et al., 2001; Hinson et al., 2006; Yamamoto et al., 2010). These data suggest greater humoral immune responses in subordinate animals compared to dominants, which may be a compensatory response to dampened cell-mediated activity (Dowd et al., 2009a; Glaser and Kiecolt-Glaser, 1997; McDade et al., 2000).

Studies that investigated social status–immune relationships in animals are briefly summarized in Table 2. Overall, there is a general

trend toward reduced cell-mediated immunity in subordinate animals, as indicated by an approximate two-fold decrease in NK cell cytotoxicity and IFN-γ production. On the other hand, compared to dominants, subordinate animals exhibited heightened antibody responses to bacterial and viral antigens that are representative of the kind of immune challenges that would occur in the natural environment. Evidence also suggests a stress-dependent effect of social status on pro-inflammatory signaling, with subordinates showing increased inflammatory response in situations of chronic or potent stress. There was limited evidence that social group stability affected these findings. Furthermore, the above-mentioned findings were primarily derived from caged animals. Results from free-ranging animals often showed no difference in immune measures between low- and high-status animals, primarily when unstimulated systemic measures were used (e.g. Hoffman et al., 2011; Müller-Graf et al., 1996; Setchell et al., 2007).

6.3. Human–animal comparison

Trends in some immune measures appear to be consistent across both humans and other species. Low SES has been shown to relate to heightened risk for a variety of diseases, cancers, and infections, implying a protective effect of higher SES (Chen et al., 2003, 2006; Cohen, 1999; Steenland et al., 2002; Steptoe et al., 2007; van Rossum et al., 2000). Similarly, studies conducted in laboratory animals suggest increased viral and cancer resistance in dominant animals (Cohen et al., 1997; de Groot et al., 2001; Ebbesen et al., 1991; Grimm et al., 1996; Hessing et al., 1994; Lindström, 2004; Temoshok et al., 1987). However, in neither human nor animal studies were there any consistent, conclusive trends between social status and parasite resistance. These general parallels between animals and humans regardless of the way social status is defined for each suggest some level of similarity between ultimate immune-related outcomes.

While different measures were used between human and animal subjects to assess cell-mediated and humoral activity, the overall effects were similar across species—lower-status individuals seemed to have dampened cell-mediated immunity and heightened humoral responses compared to higher-status individuals. In humans, pathogen burden was used to determine an overall cell-mediated effect across social ranks. Lower SES was associated with higher levels of CMV and HSV-1 specific antibodies (Bünzli et al., 2004; Dowd and Aiello, 2009; Dowd et al., 2007, 2009a, 2009c, 2012; Mustakangas et al., 2000; Shen et al., 1992; Simanek et al., 2008; Steptoe et al., 2007; Zajacova et al., 2009). Humans with low SES also displayed elevated secondary immune biomarkers (IgG, IgA) to severe infections (Bolte et al., 1999). Similarly in other animals, antibody responses to relevant infectious antigens were greater in subordinates (Barnard et al., 1993, 1994; Cunnick et al., 1991; Hawley et al., 2007; Hessing et al., 1994; Masataka et al., 1990). These studies describe relative humoral activity across social ranks which may represent an indirect assessment of cell-mediated functionality (Dowd et al., 2009a; Glaser and Kiecolt-Glaser, 1997; McDade et al., 2000). In animals, more direct measures of cell-mediated function further showed that dominant animals tended to have greater IFN-γ production, a crucial cytokine involved in fighting viral infections (Bartolomucci et al., 2001; de Groot et al., 2001; Filby et al., 2010; Merlot et al., 2004). Suppressed cell-mediated immunosuppression in low-status individuals may explain increased risk for infection in low-status humans and animals. NK activity in animals was also greater in dominants compared to subordinates, but only under some stressful situations (Hicks et al., 1998; McGlone et al., 1993; Sutherland et al., 2006). Overall, lower-status individuals show cell-mediated immunosuppression and humoral activation across species, and some studies suggest that these relationships could be partially stress-mediated.

In inflammatory responses, similar trends were observed in humans and animals as well. CRP and IL-6 levels were elevated in lower SES humans (Demakakos et al., 2008; Friedman and Herd, 2010; Gimeno et al., 2007, 2008; Gruenewald et al., 2009; Hagger-Johnson et al., 2012; Maksimović et al., 2008; McDade et al., 2011; Nazmi and Victora, 2007; Petersen et al., 2008; Saijo et al., 2008; Schafer et al., 2011; Seeman et al., 2008; Tabassum et al., 2008), while circulating and splenic IL-6 and IL-1β levels were elevated in subordinate mice living in unstable social groups (Audet et al., 2010; Avitsur et al., 2007; Savignac et al., 2011). These trends suggest that this pro-inflammatory bias may only be observed in subordinates during unstable social situations or periods of chronic social stress. The increased pro-inflammatory signaling of low-SES humans in the absence of specific stressors suggests that status itself may represent a chronic stressor potent enough to produce a trend in humans that is otherwise absent from animals in controlled and stable environments. Further research into perceived social stress among lower SES individuals may grant further insight into the similarities or differences among human and animal inflammatory biomarker levels due to social status and stress.

7. Conclusions and future studies

Results from the current review suggest that status-related glucocorticoid and immune function show some distinct similarities (as well as differences) between human and animal societies. At present, the similarities between human and animal social status-related immune disparities appear stronger than social status-related glucocorticoid function (see the two Human–animal comparison sections above). This may be a result of the methods used in the literature review, since the glucocorticoid review used studies of free-ranging animals while the immune review relied on more controlled laboratory studies. The diversity in relationships between stress-related physiology and social status in animals suggests that we need to look at specific components of social status in both humans and animals to better understand the biological mechanisms underlying social status-related health disparities.

The current review identified several important areas for future investigation. First, there is good evidence that instability in a social system accentuates or alters the relationship between social status and glucocorticoid production (supporting earlier work by Sapolsky, 1983, 2004, 2005). In addition, there is a high degree of variability in how social status relates to glucocorticoid production; sometimes the highest-status individuals in a group have heightened glucocorticoid production, sometimes the reverse is true, and in other situations no clear relationship exists between social status and glucocorticoid production. This variability in how social status relates to glucocorticoid production begs further inquiry into the specific components or corollaries of social status. In other words, what aspects of social status lead to elevated glucocorticoid production in some individuals within the social hierarchy but not others? When considering elevated glucocorticoid production in low-status individuals, is the experience of social defeat or threat the major factor that accounts for heightened glucocorticoid production (e.g. Dickerson and Kemeny, 2004; Razzoli et al., 2007, 2009; Trainor et al., 2011)? Or do other factors, like poor access to food and other resources (Champoux et al., 1993, 2001; Conn et al., 1995) issues of environmental controllability and predictability (Dickerson and Kemeny, 2004; c.f. Michaud et al., 2008), or non-social threats in the environment (Apfelbach et al., 2005; Roseboom et al., 2007), account for the elevated glucocorticoid production in low-ranking individuals? If so, to what extent do each of these other factors mediate glucocorticoid production (e.g. what proportion of the variance in glucocorticoid production is accounted for by these different social status correlates)? The same can be asked when considering elevated glucocorticoid production in individuals with high status, as well as the sex difference noted in the relationship between social status and glucocorticoids. Although not perfect models of the human social system, social animals do provide the opportunity to experimentally disentangle these factors and determine the relative influence of each factor on short- and long-

term glucocorticoid production. Whatever the specific components of status that might account for glucocorticoid production, the fact that social status may only relate to glucocorticoid production during truncated periods of instability does not minimize the relative importance of stress-related biological mechanisms in social-status-related health outcomes. Even brief periods (days or weeks) of elevated glucocorticoid production (or altered cardiovascular or immune function) may have a lasting impact on an individual's ability to ward off disease and maintain mental and physical health. Given these findings, future research should always take into account the relative stability of the social group(s) being studied, and take on the greater challenge of identifying specific aspects/correlates of social status that may explain altered glucocorticoid production, immune function, and other health-related physiological processes.

Another important area for future research is to better quantify the influence of childhood vs. adult social status on stress-related biological pathways and long-term health, as well as identify the mechanisms by which early/childhood social status may have long-term influences on health-related biological processes. Evidence from classic and current endocrine and developmental psychobiology research indicates several mechanisms by which early/childhood social status may have long-term impacts on adult health (e.g. organizational endocrine influences of early glucocorticoid exposure). These mechanisms are particularly important given findings that low-status females are more likely to experience elevated glucocorticoid production than low-status males. If low social status leads to elevated glucocorticoid production in gestating females, this altered physiology can have long-term influences on glucocorticoid receptors (through epigenetic processes) and alter the developing offspring's brain (and other organs) in such a way that leads to changes in glucocorticoid regulation and other health-related processes well into adulthood (e.g. 'glucocorticoid programming'; Räikkönen et al., 2011; Harris and Seckl, 2011; Seckl, 2004). This is particularly important given the human and animal findings that glucocorticoid regulation over time is the most likely aspect of glucocorticoid regulation that may relate to social status. Alternatively, maternal social status effects may be transmitted to offspring through mother–offspring behavioral interactions early in life which can have long-lasting influences on offspring anatomy and physiology (again through epigenetic mechanisms—e.g. Robinson et al., 2005; Weaver et al., 2004). Future research on the physiology of social status should focus on the different pathways by which maternal social status influences developing fetuses and children. This area of work is particularly important if we consider the potential social implications of permanent alterations in offspring development based on maternal social status.

The final area for future research is to design creative interventions that could moderate the influence of social status on health-related physiology. Social status is not easily changed, but if we identify key components of status that negatively (or positively) influence stress-related biological processes, then we can intervene at these levels. For example, if low access to nutritious food presents a significant stressor for low-status individuals, then studies could be designed to evaluate the moderating influence of early-life nutritional supplementation or even food stamp programs on stress-related physiological processes like glucocorticoid production, cardiovascular function, immune function, etc. Again, although social animal societies are not the same as human societies, they do provide an experimental testing ground for pre-clinical intervention ideas. Experimental intervention studies in animals showing similar results as correlational human studies suggest that pre-clinical intervention studies with animals may not be far-fetched (e.g. Schanberg and Field, 1987; Stern, 1997).

Based on the literature reviewed herein, we identify one methodological point to consider in future studies—specifically, the methods used to assess social-status-relevant biological processes. The current palette of research methods is diverse and continuously growing, and we have seen a powerful expansion of research in the past decade that borrows methods from various disciplines. Because physiological processes are temporally dynamic (as are social processes), and because these dynamics are important in affecting health maintenance, we must sample these processes in such a way that documents this variability over time and across contexts. For example, frequent, diverse, and repeated sampling of an individual's glucocorticoid production will provide a better estimate of their overall glucocorticoid regulation than a single measure, and this overall level or change in production may be a better predictor of health. There is a cost–benefit analysis that must be done in terms of whether to sample little from many individuals, or to sample a lot from fewer individuals. In the case of tracking social and biological processes associated with stress and health, because these processes are dynamic over time and because change may be a key factor in understanding health-related outcomes, we advocate a sampling bias toward frequent sampling from a smaller (although adequate) sample of individuals rather than vice versa.

Animal models provide us with the ability to test experimentally the causal influence of social status and its component parts on physiological stress processes, helping clarify pathways by which social status affects health (e.g. Lindström et al., 2005). Furthermore, many animals can be tracked from birth to death to document longitudinal influences of social status on biology and health, to identify periods during which social status is particularly influential, and to find points when age-specific interventions are most beneficial in neutralizing the effects of social status. For example, appropriate animal models allow us to determine if and how childhood social status can influence adult physiology, health and life span. Finally, free-ranging animals can provide a naturalistic complement to experimentally-based laboratory studies to best study the influences of social status-related real-life dangers, challenges, and benefits on physiology and health. Based on this review, selection of an appropriate animal model is essential, particularly if the goal is to model the human situation.

Social status, in humans and animals, has been associated with health outcomes. Our current understanding of the biological mechanisms underlying this relationship is still relatively weak although we have made great advances in the past two decades. To keep this positive trajectory in motion, we suggest the following areas of focus for future studies: (1) identification of social status correlates that causally affect stress-related physiological processes, (2) identification of the pathways by which parental or childhood social status can have long-term influences on stress-related physiology, and (3) identification of early interventions that may moderate the impacts of social status on stress and health. With an advanced understanding of how social status relates to biological processes and health we can begin to identify targeted interventions to alleviate stress-related health issues at different levels of the socioeconomic status gradient.

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