Theoretical and Empirical Support for Bilateral Decrementation

Idan S. Solon

University of Central Florida, Burnett School of Biomedical Sciences

Correspondence: IdanSolon@gmail.com

Abstract

The theory is introduced that individuals in many species practice dual strategies, which are, herein, called amplification and reservation. In amplification, individuals respond to various feedback about their relative fitness (hereafter, "fitness" is used to mean general ability against biotic and abiotic stressors) by adjusting their ability to reproduce in the direction of their relative fitness, such that fitter individuals garner an even greater reproductive share than otherwise. In reservation, fitter individuals, in particular, refrain from functioning maximally against the natural enemies most likely to rapidly evolve a set of mutations that eliminates the practicing allele. This reservation primarily 1) allows the identification of holders of the practicing allele that are fittest against these "lethal enemies," which helps restrict reproduction to these holders and 2) attenuates selection upon holders of mutant alleles and, especially, these lethal enemies by allowing the less fit individuals in these mutant and enemy populations to garner a greater reproductive share than otherwise. That is, the dual strategies of amplification and reservation increase the selection intensity upon the practicing individuals and decrease the selection intensity upon their mutant conspecifics and lethal enemies, which promotes the frequency and persistence of these strategies. At least ten empirical phenomena are characterized by, heretofore, paradoxical decrementations in inclusive fitness and are explained by this theory of bilateral decrementation. Bilateral decrementation offers a nascent example of volitional selection practiced in nature and the prevalence with which it is practiced confers empirical support for robust selection at higher levels than kin (e.g., spatial, group).

Keywords:

1. Introduction

The cluster of natural phenomena reviewed in the present paper is characterized by a common pattern, by which individuals that receive positive feedback about their relative fitness[1] make various (e.g., epigenetic, hormonal, behavioral) adjustments that enhance their ability to gain reproductive share but decrease their ability against the set of natural enemies (such a set is usually a parasite, pathogen or predator) that is particularly likely to rapidly evolve a combination of mutations that eliminates the practicing allele, while individuals that receive negative feedback about their relative fitness make adjustments that exert the reverse effect—diminishing their reproductive ability and augmenting their ability against these "lethal enemies." Herein, the theory is advanced that the modification of reproductive outlook in the direction of relative fitness (hereafter called amplification) and the modification of capacity against lethal enemies in inverse relationship to relative fitness (hereafter called reservation) are dual strategies (hereafter collectively referred to as bilateral decrementation) which have, themselves, been selected to intensify selection upon the individuals that practice these strategies and to attenuate selection upon both mutant conspecifics and, especially, lethal enemies to the practicing individuals. Additionally, it is argued herein that the reproductive forfeitures incurred by less fit individuals and the deficits against lethal enemies incurred by fitter individuals are incompletely recompensed (i.e., that deficits in one area are not made up in the other) and incur decrementations in inclusive fitness upon these practicing individuals. Therefore, they have been selected on higher levels than kin (e.g., spatial, group).

In section 3, it is demonstrated that amplification and reservation occur throughout the life cycle, are observable across a broad range of species (e.g., in birds, insects, mammals, reptiles, amphibians, and plants), are triggered by many kinds of feedback (e.g., degree of nurturing by parents, which partly reflects fitness relative to others in a brood and also reflects the parents' partly heritable fitness; hunger; disease affliction; capacity to survive the effects of reservation; receptivity of mates; performance on a multiple choice test) regarding many fitness facets (e.g., ability to procure and metabolize food; susceptibility to parasites, pathogens or predators; ability to respond to temperature changes; intelligence), and influence a variety of aspects of fitness (e.g., size, strength, aggression, immunity, cognition, reproductive capacity and desire, predator evasion, suicide risk, and signals that attract mates

---

[1] For the remainder of the present paper, "fitness" is used to mean general ability against biotic and abiotic stressors and is not used as a synonym for reproductive success; that is, it is used in essentially the first sense of fitness identified by Dawkins, 1982.

and influence conspecifics). In some cases, reproduction is influenced indirectly, through reductions in size, strength, aggression, or mortality. In other cases, reproductive physiology and/or desire are directly impacted. Either way, fitter individuals that practice bilateral decrementation produce a greater-than-otherwise proportion of the total offspring of individuals that practice bilateral decrementation. (In the case of condition-dependent, offspring sex-ratio variation, it is the proportion of grandoffspring that is influenced.)

Wilson (2014) has used the term "volitional selection" to refer to technology that humans might use to give preference to particular genes and/or characteristics, thereby determining the evolution of their own populations. Sexual selection has been noted to occur in many species and involves preference individuals give to particular characteristics exhibited by prospective mates. However, what sexual selection does not involve, and what technology increasingly offers, is a mechanism that allows a specific individual to exclude elements of his or her own contribution to the next generation's gene pool. While the examples of bilateral decrementation presented herein do not involve the isolation of specific genes or characteristics, they do involve the selected, "volitional" selection of one's own collective of genes on the basis of the relative fitness it has demonstrated. In this sense, bilateral decrementation represents volitional selection, practiced not only by humans but also by many other species.

Bilateral decrementation may also be characterized as a sort of meta-selection: It is a set of practices that has been selected to intensify selection upon practicing individuals and attenuate selection upon mutant conspecifics and lethal enemies. Bilateral decrementation intensifies selection upon holders of practicing alleles (called "wild" alleles below), attenuates selection upon holders of alternative alleles that do not practice reservation (called "mutant" alleles below), and attenuates selection upon lethal enemies in primarily the following ways:

1) Stronger selection upon wild allele holders

Amplification and reservation each increase the selection intensity upon wild allele holders by reducing, or outright preventing, reproduction by individuals that are not among the fittest wild holders. As a result of amplification, less fit individuals forfeit offspring-sustaining resources and reproductive opportunities to fitter individuals. As a result of reservation by fitter individuals in particular, more of these fitter individuals perish than

otherwise and the fittest of these fit individuals are identified via the demonstration of their ability to survive and reproduce despite the reservation they undertake.

Reservation is particularly useful for the identification of specific facets of fitness, since reservation can reveal relative condition or disguise it, depending on the degree to which the facet in question is given to metered measurements and how well the effects of reservation can be taken into account from individual to individual. (This point is developed further in the section on self-handicapping.) For example, it is usually straightforward to determine which of two students has performed better on a multiple choice exam, as the multitude of potential scores makes it highly likely that the two students' scores differ. Alternatively, if the evaluation of a particular task is closer to binary, many individuals may receive the same evaluation, in which case reservation can reveal the most skillful individuals by detracting enough from each individual's performance that only a select few are able to accomplish the task.

In particular, the evaluation of predator evasion is more binary than that of numerous other fitness dimensions. For example, an individual may consume many prey during its lifetime, which allows for a lot of variation in the number of prey captured and metabolized, but an individual has either successfully evaded all predators or has been captured and it may be difficult to determine which of multiple surviving individuals is better at predator evasion. However, if each individual were to reserve ability against predators, more individuals would perish, which allows the identification of a more exclusive set of individuals that are fittest against these predators. The ability to survive parasites or pathogens may also be identified via reservation. In a given generation, a multitude of individuals may be able to withstand these natural enemies without incurring significant impairments. However, if each individual reserves capacity against these natural enemies, more individuals may perish or become significantly impaired. The select set of healthy survivors is identified as the fittest of these reserving individuals against these natural enemies by virtue of their having demonstrated the ability to withstand the reservation.

The identification of the fittest wild holders against lethal enemies helps ensure that reproducing wild holders possess traits that allow them to manage these enemies. As a result, more of the fitter wild holders may succumb to these lethal enemies than otherwise during a particular generation, but since selection upon wild holders is stronger than otherwise for characteristics that defend against these enemies, the wild allele is also more likely to

be possessed in subsequent generations by at least some holders that can survive them, which is particularly critical if the enemies evolve rapidly and/or nonlinearly. (This point is illustrated in section 2.2.)

In addition to using their own resources and reproductive opportunities, the fitter wild individuals that survive the effects of reservation endeavor to also utilize the resources and reproductive opportunities forfeited by wild individuals that refrain from reproduction due to amplification and wild individuals that perish due to reservation. The degree to which fitter wild individuals can exploit resources and reproductive opportunities forfeited by other wild individuals is dependent upon the wild individuals' physiological capacity to produce more offspring to meet environmental capacity. Evidence indicates that reproduction in natural populations is constrained more often by environmental than physiological elements (Williams, 1966), which suggests that fitter individuals would usually have the physiological capacity to recoup reproduction forfeited by less fit individuals. The documented higher fecundity in response to stronger-than-usual threats from parasites (Norris et al., 2010) and predators (Berglund, 1993; Slobodkin, 1957; Stibor, 1992) also indicates that physiological output can be increased to meet environmental capacity. The degree to which fitter wild holders recoup reproduction forfeited by less fit wild holders also depends upon the degree to which wild holders are spatially located in proximity to other wild holders compared to holders of competing alleles (Nowak, 2012).

2) Diminished resistance against lethal enemies

The reservation practiced by fitter wild holders against lethal enemies diminishes the resistance offered against these enemies, which potentially exerts multiple influences. The most secure ramification of this diminished resistance is that it becomes easier for lethal enemies to have success against wild holders, which allows more of the less fit members of these lethal enemy populations to survive and reproduce than otherwise. Thus, reservation exerts the opposite influence upon lethal enemies that amplification and reservation exert upon wild holders: The selection intensity upon the enemy populations becomes weaker. For example, some individuals are sufficiently adept at predator evasion that they can only be captured by fast predators. Slower predators may make many failed pursuits of them before they are captured by faster predators. However, if these individuals reserve capacity against predators by exhibiting a weighty ornament, they become more likely to be captured by slower predators, as

predation success becomes determined less by speed and more by spatial orientation. Therefore, as a result of the handicap, the surviving and reproducing predators are, on average, slower than otherwise.

Additionally, the reservation against lethal enemies may exert a situationally-dependent protective influence upon conspecifics by drawing the enemies toward them and away from the conspecifics. If so, more of the less fit mutant individuals would be able to survive and reproduce, as the selection intensity upon these mutant individuals is attenuated. However, as more wild individuals engage in reservation (which occurs either by increasing wild quantity or an increasing percentage of wild individuals that engages in reservation) and, consequently, as more mutant conspecifics are spared by that reservation, the likelihood increases that the complete elimination of the mutant population (and consequent fixation of the wild allele) is prevented. Reservation that draws the attention of lethal enemies toward reserving individuals may also have the effect of protecting vulnerable holders of the wild allele that do not reserve (including, potentially, females, developing offspring and less fit males) by drawing enemy attention away from these individuals. These less fit wild individuals would, however, be substantially prevented from reproduction due to their amplification, unless an inordinate number of the wild individuals previously identified as fitter are killed, in which case the relative fitness of the surviving individuals would increase and their orientation toward reproduction would increase accordingly. In this manner, these less fit wild individuals can act as insurance. A potentially adverse side effect of this protection afforded to both mutant holders and other wild holders is that the quantity of the reserving individuals in the total population would be inversely related to the intensity of the selection pressure upon each reserving individual, since a greater quantity of reserving individuals would attract more enemy attention from any single reserving individual. An additional consideration is that this protective effect upon conspecifics has been characterized above as "situationally-dependent" because if reserving individuals perish quickly, then more lethal enemy individuals would be in pursuit of the remaining individuals in the population. That is, the selection intensity upon each remaining individual would be stronger than otherwise.

Insofar as it is possible to decouple reservation against parasites, pathogens or predators from reservation against prey, the former practices are more likely for multiple reasons. First, the abilities to withstand parasites, pathogens or predators are more necessary to ensure the persistence of the wild allele than the ability against prey. If certain prey evolve an adaptation that allows them to evade the holders of the wild allele, these holders can often

exploit another prey species, whereas if a parasite, pathogen or predator population evolves an adaptation that gives it an advantage over the holders of the wild allele, the wild allele's persistence in the population is more severely threatened. A related advantage to reservation by wild holders against parasites, pathogens or predators is that reservation by wild holders against these enemies that attenuates selection upon mutant holders for abilities against these enemies is more likely to lead to the elimination of the entire mutant population than if selection were attenuated upon mutant holders for abilities against prey. Second, under circumstances in which prey constitute the factor limiting population growth and wild individuals reserve against prey, mutant holders are capable of exploiting the prey left alive by those reserving wild individuals in order to multiply rapidly. This would both a) significantly contribute to the frequency of mutant holders and b) re-intensify selection pressure upon the prey, thereby nullifying the effects of reservation upon the prey. Third, an individual is only captured and killed once during its life cycle, but commonly captures many prey during that span. Therefore, it is common for a wild allele holder to be engaging in reservation during the only part of its life cycle in which it is captured. This is true even if the individual relaxes reservation at other points of its life cycle, which potentially includes both its developmental period and its adulthood if it is identified as less fit. By contrast, wild individuals that sometimes relax reservation would often capture a multitude of prey while not engaging in reservation. This allows reserving individuals to more effectively attenuate selection upon predator populations than selection upon prey populations. In accordance with these observations, there are many more recorded examples of handicaps detracting from ability against predators than handicaps detracting from ability against prey (Zahavi & Zahavi, 1997).

2. Model

A quantitative demonstration of the merits of practicing amplification is presented in section 2.1. Then, in section 2.2, a second quantitative demonstration is given of the merits of practicing amplification and reservation compared to a strategy of only amplification.

2.1 Hybrid versus Mutant

Consider a haploid population comprised of individuals with either of two alleles: a hybrid allele that practices amplification but not reservation or a mutant allele that practices neither amplification nor reservation.

Each individual in the population is designated a fitness level, which is a number that reflects its general ability against biotic and abiotic factors and which also indicates the quantity of offspring it produces if it reproduces. All hybrid allele holders practice amplification, such that the only reproduction undertaken by hybrid allele holders is by individuals that have the highest fitness level of all hybrid allele holders. (If multiple individuals share this highest fitness level, each of these fittest hybrid holders reproduces.) All mutant allele holders reproduce to their capacity (that is, produce the quantity of offspring indicated by their fitness level). Additionally, both reproducing hybrid allele holders and mutant allele holders utilize for reproduction some fraction of the resources and reproductive opportunities unused by hybrid holders that do not reproduce. All reproduction occurs simultaneously and a generation has passed when reproduction has completed.

Furthermore, consider that the following conditions are met:

1) The weighted average fitness of the reproducing holders of an allele in one generation becomes the mean fitness of all the holders of that allele in the following generation.

2) An allele's quantity is divided among the same number of fitness levels from generation to generation and this number of fitness levels is the same for each allele. Consider that if an allele is represented by a non-zero quantity, this quantity is distributed across at least two fitness levels.

3) The proportion between the level of any particular fitness rank representing an allele during a generation and the mean fitness for the holders of that allele in the same generation is the same for each allele and these proportions are the same across generations. That is, for example, the proportion between the highest fitness level representing an allele for a generation and the mean fitness of the holders of that allele for that generation is constant from generation to generation and from allele to allele. Therefore, the highest fitness level represented by an allele during generation 2 can be determined by multiplying that allele's highest fitness level of generation 1 by the quotient of the allele's generation 2 mean fitness divided by the allele's generation 1 mean fitness.

4) Consistent percentages of an allele's quantity represent particular fitness ranks of the holders of that allele from generation to generation and these percentages are the same for each allele. That is, for example, the same x% of the holders of each allele represent their respective allele's highest fitness level for that generation and the same y% of the holders of each allele represent their respective allele's second-highest fitness level for that generation.

5) In order to represent how non-reproducing individuals' resources and reproductive opportunities are utilized by the reproducing holders of each allele, each allele's quantity is multiplied by a factor that is unique to the holders of that allele, but consistent from generation to generation. That is, the utilization of non-reproducing hybrid holders' resources and reproductive opportunities by hybrid holders is a proportion of hybrid quantity that is consistent from generation to generation and the utilization of non-reproducing hybrid holders' resources and reproductive opportunities by mutant holders is a proportion of mutant quantity that is consistent from generation to generation. These proportions are determined by the spatial relationships between the non-reproducing individuals and the reproducing holders of each allele. Consider that this additional quantity is produced in proportion to fitness and, therefore, does not change the mean fitness for the holders of either allele.

Under these conditions, the factor by which the quantity of an allele increases from one generation, t, to the next is given by $abc^{(t-1)}$, where a is the sum of the products of the starting reproducing fitness levels of the allele and the respective frequencies with which the levels are represented among the holders of the allele (which takes the form of $ux+vy+\ldots wz$, where u is that allele's fittest level during that generation, x is the fraction of that allele's holders that represent that fittest level, v is that allele's 2nd fittest level during that generation, y is the fraction of that allele's holders that represent that 2nd fittest level, w is that allele's least fit level during that generation, and z is the fraction of that allele's holders that represent that least fit level); b is the per generation factor by which the allele's quantity increases as a result of utilization of the resources and reproductive opportunities of non-reproducing hybrid allele holders; and c is the per generation factor by which the mean fitness of the allele increases (which, as a result of the above conditions, is the same from generation to generation); therefore, the quantity of the allele after any particular generation, t, is given by $n(ab)^t\ c^{((1/2)(t^2-t))}$, where n is the allele's starting quantity.

The difference between hybrid allele quantity and mutant allele quantity after any particular generation, t, is $n(ab)^t\ c^{((1/2)(t^2-t))}-m(df)^t\ g^{((1/2)(t^2-t))}$, where n is the starting hybrid allele quantity; a is the product of the starting fittest hybrid allele level and the frequency with which that fittest hybrid allele level is

represented among hybrid allele holders; b is the per generation factor by which the hybrid allele quantity increases as a result of the utilization of the non-reproducing hybrid holders' resources and reproductive opportunities; c is the factor by which the mean fitness of hybrid holders increases per generation (which is the fittest hybrid level in a generation divided by the mean hybrid fitness in the same generation, since, for hybrid holders, the fittest level during one generation becomes the mean fitness during the following generation); m is starting mutant allele quantity; d is the sum of the products of the starting mutant fitness levels and their respective frequencies among mutant holders; f is the per generation factor by which the mutant allele quantity increases as a result of the utilization of the non-reproducing hybrid holders' resources and reproductive opportunities; and g is the factor by which the mean fitness of mutant holders increases per generation, which is the weighted average of the mutant individuals' fitness divided by the previous generation's mean mutant fitness and which, as a result of the above conditions, is the same from generation to generation:

$$((uux+vvy+\ldots wwz)/(ux+vy+\ldots wz))/h,$$ where u is the fittest mutant level, x is the

fraction of mutant holders that represent that fittest mutant level, v is 2nd fittest mutant level, y is the fraction of mutant holders that represent that 2nd fittest mutant level, w is the least fit mutant level, z is the fraction of mutant holders that represent the least fit mutant level, and h is the previous generation's mean mutant fitness). Assuming all values are positive, the limit to this expression approaches infinity as t approaches infinity as long as c, the factor by which mean hybrid fitness increases per generation, is greater than one and exceeds g, the factor by which mean mutant fitness increases per generation, which always occurs under the above conditions. One caveat is that, depending on the values given in the expression, the hybrid allele may incur a quantity deficit to the mutant allele that is very large before mean hybrid fitness grows large enough in comparison to mean mutant fitness that the hybrid quantity increases relative to the mutant quantity. In the above representation, there is no limit to total population size. In nature, by contrast, resources are finite, which imposes a limit upon total population size. If the quantity deficit incurred by the hybrid allele to the mutant allele exceeds a realistic population size, the implication is that, in nature, the hybrid allele would be eliminated before it has the opportunity to recover from the deficit.

2.2 Wild versus Hybrid

Consider a haploid population comprised of individuals with either of two alleles: a wild allele that practices amplification and reservation or a hybrid allele that practices only amplification. Each individual possesses two fitness types: a lethal fitness type, which reflects its ability to survive the lethal enemies, and a reproductive fitness type, which reflects its ability in all other areas and indicates the quantity of offspring it produces if it reproduces. In a particular generation, all individuals that do not exhibit lethal fitness above the "lethal threshold" for that generation perish without reproduction. This lethal threshold is determined by the evolution of the lethal enemies.

Both wild and hybrid allele holders practice amplification and, as a result, only reproduce if they represent the highest reproductive fitness level of the holders of their respective alleles for a particular generation. However, unlike hybrid allele holders, wild allele holders also practice reservation. This reservation is practiced by wild holders that represent the highest reproductive fitness of wild holders. These reserving wild holders reduce their expressed lethal fitness until all reserving individuals have perished (which results when their expressed lethal fitness drops below the lethal threshold) except a fraction that represents the wild holders with the highest lethal fitness level among wild allele holders. These wild holders that survive the effects of reservation are identified as representing both the highest reproductive fitness level among wild allele holders (since these are the individuals that engage in reservation) and the highest lethal fitness level among wild allele holders (since only these individuals survive the reservation). These wild individuals then reproduce. While reservation reduces a reproducing individual's expressed lethal fitness, the offspring lethal fitness levels are proportions of that individual's pre-reservation fitness. That is, reservation does not carry over to offspring. Both reproducing wild individuals and reproducing hybrid individuals utilize some fraction of the resources and reproductive opportunities unused by individuals that do not reproduce because they amplify and/or perish.

Consider that the five conditions outlined above for the hybrid and mutant alleles apply to both the reproductive and lethal fitness levels for both the wild and hybrid alleles, except that the mean lethal fitness of an allele's holders in one generation is only a simple average of the lethal fitness held by the previous generation's reproducing holders of that allele and not a weighted average, since a surviving individual with a higher lethal fitness does not possess a reproductive advantage over a surviving individual with a lower lethal fitness. Furthermore, consider that there is no pleiotropy, so that, among the holders of an allele, a particular reproductive

fitness rank is no more likely to be combined with one lethal fitness rank than any other and a particular lethal

fitness rank is no more likely to be combined with one reproductive fitness rank than any other.

Under these conditions, the mean reproductive fitness of wild allele holders grows under the same selection

intensity as that of hybrid allele holders, but the mean lethal fitness of wild allele holders grows under higher

selection intensity than that of hybrid allele holders (except if the lethal threshold grows so rapidly that only hybrid

holders with highest lethal fitness among hybrid holders are able to survive and reproduce). This gives the wild

allele a better chance of surviving a lethal threshold that grows nonlinearly from generation to generation. For

example, consider that, in a particular generation, both wild and hybrid allele quantities are equally distributed

across five lethal fitness levels, 2.5, 2.25, 2, 1.75, and 1.5. Consider further that the lethal enemies have evolved in

such a way that all individuals with lethal fitness lower than 2 perish without reproduction. (This lethal threshold is

likely lower than otherwise due to the attenuating influence by reserving individuals, in accordance with the above

discussion regarding the influence of reservation upon the evolution of both conspecifics and the lethal enemies.)

For holders of the hybrid allele, reproduction is then undertaken by individuals with lethal fitness of 2.5, 2.25, and 2.

By contrast, wild individuals engage in reservation, leading wild holders with lethal fitness of 2.25 and 2 to exhibit

lethal fitness below the lethal threshold and, therefore, perish. The wild holders with lethal fitness of 2.5 also exhibit

lower lethal fitness because of reservation, but the wild holders with lower lethal fitness perish first. Once only $1/x$

of reserving individuals remains alive, where x represents the number of fitness levels, this $1/x$ can be identified as

having the highest lethal fitness of wild holders during that generation. These wild holders then reproduce. (In this

scenario, these reproducing wild holders represent $1/x^2$ of all wild holders, where, again, x represents the number of

fitness levels, since only the $1/x$ of wild individuals with highest reproductive fitness engage in reservation.) In the

following generation, the mean hybrid lethal fitness becomes 2.25 and each hybrid lethal level is multiplied by

2.25/2, leading the hybrid lethal levels to become 2.8125, 2.53125, 2.25, 1.96875, and 1.6875, while the mean wild

lethal fitness becomes 2.5 and each wild lethal level is multiplied by 2.5/2, leading the wild lethal levels to become

3.125, 2.8125, 2.5, 2.1875, and 1.875. If, between generations, the lethal enemies evolve in such a way that the

lethal threshold grows from 2 in the previous generation to anywhere between 2.8125 and 3.125, then all hybrid

allele holders perish prior to reproduction and the wild allele gains fixation. Otherwise, mean wild lethal fitness

continues to grow in subsequent generations in comparison to mean hybrid lethal fitness, as long as the lethal

threshold spares at least the holders of the 2nd highest lethal fitness among hybrid allele holders. This gives the lethal threshold a larger window into which it can grow in order to lead to the fixation of the wild allele.

In exchange for this faster mean lethal fitness growth, the wild allele sacrifices quantity since fewer holders reproduce than if the hybrid strategy were practiced: While reproduction is undertaken by all hybrid holders with the highest reproductive fitness among hybrid holders, only wild holders with the highest reproductive fitness and the highest lethal fitness among wild holders reproduce. If there is no lethal threshold or one that grows slowly, an allele that does not engage in reservation can still be held by at least some holders each generation that have lethal fitness that exceeds the lethal threshold, as the mean lethal fitness of the holders of such an allele would grow sufficiently rapidly due to the perishing of individuals below the lethal threshold. In such situations, selection may not favor reservation.

Alternatively, if the lethal threshold grows very rapidly, it may benefit the wild allele to have reservation and/or reproduction undertaken by more than the reproductively fittest wild holders, since reservation by a larger percentage of wild holders may help further attenuate the growth of the lethal threshold. Additionally, in a departure from the scenario above, there may be no individuals with the highest lethal fitness among wild holders that also represent the highest reproductive fitness among wild holders. A rapidly growing lethal threshold would select for reproduction by individuals with the highest lethal fitness even if they have a low reproductive fitness, though this would lead to a lower mean wild reproductive fitness. Likewise, if progenitor diversity has an influence upon offspring fitness range, such that a greater number of progenitors increases the offspring fitness range (which leads, ceteris paribus, to greater mean grandoffspring fitness), it may benefit the wild allele for reproduction to be undertaken by more wild individuals with highest lethal fitness, even those that are less reproductively fit. This would hasten mean wild lethal fitness growth at the expense of mean wild reproductive fitness growth and may be selected if the lethal threshold grows with sufficient rapidity.

Additionally, selection may favor the use of reservation against multiple enemies at once. If so, the criteria for reproduction would be the demonstration of the ability to survive despite reservation against each of the enemies.

Individuals that practice bilateral decrementation act based upon feedback about their relative fitness compared to other individuals that practice bilateral decrementation. A wild individual is likely able to determine its

relative fitness compared to other wild holders, even in the presence of conspecifics with an alternative alleles that signal in a selfishly dishonest manner about their own fitness. An individual receives some feedback from its own mother about its relative fitness in comparison to other individuals in the same brood that likely, though not necessarily, share the same allele. The mother is also aware of differential allocation of resources in offspring, since she has primarily done the allocation, and, therefore, she can consider offspring performance in terms of this allocation. Additionally, if mean wild reproductive fitness is comparable to, or exceeds, mean hybrid reproductive fitness and mean mutant reproductive fitness, then it is likely that a wild individual is able to determine whether it has high reproductive fitness compared to other wild holders by evaluating its reproductive fitness compared to the rest of the population. To this end, an individual can utilize observation (some abilities, e.g., food procurement, may be difficult to fake), consideration of competitive outcomes versus conspecifics, and feedback from observers that are in position to evaluate multiple individuals. For example, a student can learn from his or her professor about his or her performance relative to other students in his or her class. For non-humans, a prospective mate can potentially fulfill this function. This can include feedback about lethal fitness if the nature of the reservation is visible by an observer. (Once rejected by prospective mates, the individual would indicate that rejection to future prospective mates.) Moreover, given a lethal threshold that grows sufficiently rapidly and/or nonlinearly, selection on the group level would favor those groups in which wild individuals have the ability to determine their relative lethal fitness and reproduce if they are fit, whether this occurs because the reservation is visible or because hybrid and mutant holders signal honestly: The quantitative exploration above demonstrates that wild alleles in groups so characterized are more likely to be able to survive these lethal enemies in comparison to alternative alleles in alternative groups and that a lethal threshold that grows sufficiently rapidly and/or nonlinearly would lead all individuals to perish if they are not in groups so characterized.

In sum, 1) a strategy of amplification enables its practicing alleles to gain long-term quantity advantages over alleles that practice neither amplification nor reservation and 2) a combination of amplification and reservation compares favorably to the sole strategy of amplification in enabling its practicing alleles to persist against a lethal threshold that grows rapidly and/or nonlinearly. In section 3, it is argued that bilateral decrementation, involving both amplification and reservation, is, indeed, practiced pervasively in the natural world. The following ten empirical phenomena are presented in the approximate order in which they occur in the life cycle. These phenomena are characterized by forfeitures of reproductive share by less fit individuals and/or reservation of

capacity against parasites, pathogens or predators by fitter individuals. An additional critical commonality is that these tradeoffs were evidently not imposed by physical constraints and that mutations have apparently been accessible (that is, obtainable in a viable individual) in evolutionary time that would have either prevented these forfeitures or decoupled inclusive fitness benefits in one area from forfeitures in the other, allowing their holders to experience only the benefits. The absence of these mutations is attributable to countervailing selection for bilateral decrementation. In this analysis, the key consideration is not whether selfish mutations are accessible in extant individuals but whether such mutations have been accessible in evolutionary time. This is because processes involved in bilateral decrementation may have been gradually utilized for additional functions over evolutionary time, such that selfish mutations in an extant individual would also impose the prohibitive cost of interfering with those additional functions. That is, the invasion of an allele by a selfish mutation can be inhibited if it cannot replace the favorable functions in which the allele pleiotropically participates. The prevalence of such pleiotropy in nature would imply the selection of the pleiotropically-preserved allele itself.

3. Empirical support for bilateral decrementation

3.1 Differential allocation

Parental investment of resources in offspring has been demonstrated to vary both within and between broods (e.g., Burley, 1986; Gowaty, 2008; Groothuis et al., 2005). One factor that influences a parent's allocation of resources to offspring is the degree to which that parent prefers the other progenitor of the offspring in comparison to other prospective progenitors. Frequently, some individuals in a population are generally more attractive than others (that is, in these cases, mate preference is not completely self-referential: Gowaty, 2008). Many researchers have found that parents invest more resources in offspring produced with more attractive progenitors, a phenomenon documented in multiple investment forms (e.g., androgens, fecundity, female parental care), for multiple exhibitions of attraction (e.g., ornaments, vocalizations, size), and in a wide range of species, including species of insects, fish, birds, amphibians, and mammals (Burley, 1986; Sheldon, 2000; Stiver & Alonzo, 2009). Since attractive exhibitions are often diminished as a result of stressors, a progenitor's attractiveness signals its predominantly heritable ability to withstand extant biotic and abiotic challenges (Andersson, 1994). Therefore, by investing more

in offspring from more attractive progenitors, parents are investing more in fitter offspring, which reinforces the reproductive advantages of these fitter offspring.

Resources that are allocated differentially both within and between broods are assumed to exist in limited quantities or to have costs associated with their investment. Otherwise, some parents would be unnecessarily investing less in some offspring and across some broods. While many differentially-allocated resources are limited in quantity, androgens (e.g., testosterone) are theoretically available in unlimited quantity, yet many scholars have found that parents invest more androgens in offspring produced with fitter mates (reviewed in Groothuis et al., 2005; Safran et al., 2008). This pattern is consistent with amplification, since androgens confer advantages in reproductive competition by promoting begging, aggression, lower mortality until fledging, growth, and, ultimately, social rank and attractiveness to mates (Folstad & Karter, 1992; Gil, 2008; Groothuis et al., 2005), which induces greater mate fecundity (reviewed in Sheldon, 2000). The differential allocation of androgens has encouraged inquiries about costs associated with androgens, with the most popular explanation being that the reproductive advantages from androgens come with a greater energy burden that is afforded by sacrificing immune development (Folstad & Karter, 1992; Habig & Archie, 2015; Roberts et al., 2004). However, studies examining the influence of androgens upon immunity have reported inconsistent results. While androgens have been reported to detract from defenses against parasites, higher status individuals have been found on multiple occasions to possess stronger defenses against non-infectious diseases (Martinez et al., 1998; Sapolsky, 1998; Sa-Rocha et al., 2006). This differential susceptibility, of fitter individuals to natural enemies (e.g., parasites) and of less fit individuals to diseases that do not involve natural enemies, is consistent with bilateral decrementation.

In a meta-analysis, Roberts et al. (2004) found no effect of androgens upon direct measures of immunity, but they did find androgens to increase ectoparasite abundance in several studies. Additionally, Kankova et al. (2014) found that when Japanese quail offspring were subjected to moderate caloric restriction during development, the offspring with high egg testosterone content retained their growth advantage and did not suffer an immunity disadvantage, which, they concluded, contradicted the hypothesized energetic tradeoff between androgen-mediated growth and immunity. In a larger, more recent meta-analysis, Habig & Archie (2015) replicated the finding that dominant males are consistently more susceptible to a breadth of parasites, but found that result "puzzling," since they could find no significant differences between the immune responses of dominant and subordinate males. They

suggested that dominant males may suppress antibody production in response to antigens, which is an aspect of the less energetically costly Th-2 mediated immunity and which is contrary to how individuals reducing immunity to save energy would be predicted to operate (Lee, 2006). Consequently, Habig & Archie (2015) concluded that their results did not support the hypothesis that dominant individuals sacrifice immune function to engage in greater reproductive effort. They offered that the results might be attributable to the priority of access that dominant males have to food and mates, which may result in differential exposure to parasites. However, testosterone also exerts a suppressive effect upon antibody responses in humans (Furman et al., 2014), despite minimal variation between humans in access to food or mates.

In the continued absence of a well-supported finding that the susceptibility to parasites that results from a greater receipt of androgens is a tradeoff of the androgenic reproductive benefits that is imposed by nature, the differential allocation of androgens is best explained by selection for bilateral decrementation. That is, androgens: a) are invested preferentially in offspring of fitter progenitors; b) confer reproductive advantages to these fitter offspring, apparently without tradeoffs imposed by physical constraints; and c) are associated with greater susceptibility to parasites, even though there were likely mutations in evolutionary time that decoupled these advantages from this susceptibility, since the coupling did not result from physical constraints.

While positive differential allocation (i.e., greater investment in offspring with preferred progenitors) characterizes the substantial majority of documented examples of differential allocation (Stiver & Alonzo, 2009), empirical support has also accumulated for a reverse, "compensatory" pattern, under which parents allocate greater resources to offspring with non-preferred progenitors. These apparently contrasting models have encouraged attempts to identify other factors that might direct parents to invest preferentially in offspring from either preferred or non-preferred progenitors (Harris & Uller, 2009; Ratikainen & Kokko, 2010).

Researchers reporting empirical findings of compensatory investment have most commonly explained them by invoking the reproductive compensation hypothesis (Gowaty, 2003; Gowaty, 2008; Gowaty et al., 2007), which is intended to explain parental allocation under circumstances in which preference for progenitors is self-referential (Gowaty, 2008). Other studies reporting compensatory phenomena are best classified outside of the domain of the reproductive compensation hypothesis because they involve experiments upon males with generally attractive characteristics. While female fitness or attractiveness is seldom reported in studies of differential allocation, the few

compensatory findings in studies upon males with generally attractive characteristics have disproportionately reported a female sample with anomalously high fitness, whether the fitness was indicated by predation avoidance (Byers & Waits, 2006), previous fecundity (Bolund et al., 2009), or size (Braga Goncalves et al., 2010). In consideration of the relative scarcity of both a) studies reporting compensation and b) studies reporting a female sample with anomalously high fitness levels, the multiple reports of both phenomena in combination warrant further investigation. Though it is not necessary to invoke bilateral decrementation in order to explain these findings, the two findings in tandem are consistent with the expectation that even the inferior offspring of those produced by a very fit female would be among the fittest offspring in a group and would reproduce even if they were to practice bilateral decrementation and that, therefore, additional investment in those offspring would be likely to materialize in the production of grandoffspring.

Another pattern (albeit, again, involving a small sample) of compensatory investment appears to involve carotenoid investment. In a departure from the prevalent positive differential allocation, progenitors have been repeatedly demonstrated to invest greater levels of carotenoids in offspring produced with less fit progenitors (Bolund et al., 2009; Navara et al., 2006; Safran et al., 2008; Saino et al., 2002), which is consistent with reservation, since carotenoids support immune functions, particularly against infections (von Schantz et al., 1999). Carotenoids are also utilized in ornaments (see section 3.4). However, while carotenoid supplementation enhances ornamental exhibition (Hill, 2006), it should not be assumed that specifically these compensatory carotenoids influence ornamental exhibition in a linear (or even positive) manner.

In sum, studies on the differential allocation of resources to offspring have, in aggregate, yielded several secure results and a couple of conjectures (pending larger samples) that can be made in favor of bilateral decrementation. Androgens: 1) are preferentially invested in fitter offspring within a brood and also in fitter broods; 2) confer reproductive advantages, apparently without obligate tradeoffs; and 3) are associated with greater susceptibility to natural enemies (e.g., parasites), which is not imposed by physical constraints associated with the advantages they confer. More conjecturally, it may also be that: 1) less fit individuals incur immunity costs relating to non-infectious conditions (i.e., those not involving natural enemies); and 2) carotenoids, which are to be used against infectious diseases (i.e., against natural enemies), are preferentially invested in less fit offspring.

3.2 Maternal nurturing

Maternal nurturing of offspring is another form of differentially-allocated parental investment. In this section, the argument is made that maternal nurturing exerts a positive influence upon offspring fitness across environmental circumstances and that mothers adhere to a regimen of nurturing their offspring a) which is determined by the accumulation of feedback they have received about their relative fitness and their perception of the other progenitor's relative fitness (both of which are indicators of the offspring fitness) and b) which is not restricted by physical tradeoffs. Thus, maternal nurturing of offspring is an example of amplification.

Evidence indicates that in a wide range of species (including rodents and primates), higher levels of maternal nurturing lead to increased brain derived neurotrophic factor (BDNF) expression (Zhang & Meaney, 2010), increased synaptic density (Zhang & Meaney, 2010), increased growth (Denenberg & Karas, 1959; Field, 1998; Field et al., 1986; Schanberg, 1995), increased alertness (Field, 1998; Field et al., 1986), greater behavioral maturity (Field et al., 1986), better memory function (Field, 1998), and lower mortality risk (Denenberg & Karas, 1959; Field et al., 1986), while lower levels (or a complete absence) of maternal nurturing lead to depression (Franklin et al., 2010; Franklin et al., 2011; Weiss, I. et al., 2011), anxiety (Weiss, I. et al., 2011), immune system deficiencies (Schanberg, 1995; Suomi, S., 1995; Weiss, I. et al., 2011), and greater tendencies to resign to stressful sources (Nestler, 2012). Despite fitness costs incurred by offspring from diminished nurturing, mothers that experience chronic stress (an indicator of low relative fitness) exhibit diminished nurturing across the aggregate of their offspring—an effect mediated epigenetically (Mansuy et al., 2013; Zhang & Meaney, 2010). Offspring of socially stressed fathers are more likely to exhibit depression and anxiety when mothers are impregnated naturally than when mothers are impregnated via in vitro fertilization (Dietz et al., 2011; Dietz & Nestler, 2012), which indicates that maternal effects are responsible.

Diminished nurturing does not appear to result from constraints faced by the mother. Even in the rearing absence of the progenitor, a mother adjusts her nurturing levels depending on indications of the progenitor's fitness (Dietz et al., 2011; Dietz & Nestler, 2012; Mashoodh et al., 2012). Additionally, maternal nurturing often occurs in the form of licking and grooming, which incur minimal energy costs, and individual differences in licking and grooming are not associated with differences in litter size or offspring weaning weight (Champagne et al., 2003), indicating that mothers do not refrain from additional nurturing due to insufficient capacity. Furthermore, the close

correlation between licking and grooming levels (Liu et al., 1997) indicates that no tradeoffs are necessary between primary nurturing forms.

Nevertheless, differential levels of maternal nurturing across the aggregate of offspring are preserved between lineages, as a mother's nurturing practices remain consistent throughout her lifetime and her offspring are likely to convey the same levels of nurturing to their offspring that they received from their mother (Mansuy et al., 2013). The rate of maternal rejection and infant abuse is also preserved (Mansuy et al., 2013). This effect is also oriented by paternal nurturing toward both the mother and offspring (Mansuy et al., 2013; Putallaz et al., 1998). The intergenerational persistence of nurturing behavior is mediated epigenetically (Mansuy et al., 2013). Considering the costs associated with diminished nurturing, the conservation, both within a lifetime and intergenerationally, of maternal nurturing differences between lineages is paradoxical (Champagne et al., 2003).

While few (if any) researchers hypothesize any benefits to offspring from outright abuse or neglect, Champagne et al. (2003) offered that moderately reduced nurturing could adaptively lead offspring to be more cautious and more attuned to environmental challenges, primarily predators. One would anticipate the accessibility of a more efficient method of communicating caution that spares the fitness costs of diminished nurturing, but it might be argued that these costs result unavoidably from energy demands associated with vigilance. However, offspring from the same mother (and evidently the same formative environment) are induced via maternal effects to incur costs that are determined by their father's fitness, even in the absence of their father's postpartum presence (Dietz et al., 2011; Dietz & Nestler, 2012; Mashoodh et al., 2012). Nurturing deficits often manifest themselves during formative stages when offspring share an environment with controls and/or are helpless against predators (Hager et al., 2009; Kuhn et al., 1978; Schanberg & Field, 1987). Moreover, exposure to predator odor during gestation has been found to stimulate postpartum maternal affection (McLeod et al., 2007; Mashoodh et al., 2009), which is opposite of the effect exerted by psychosocial stressors upon nurturing (Bosch et al., 2007; Heiming et al., 2011) and which casts doubt upon the hypothesis that diminished nurturing is intended to benefit offspring against predators. Additionally, offspring of preferred males have been reported to exhibit more risk-averting freezing behavior in a predator-avoidance test (Curley et al., 2011), which also detracts from the hypothesis that reductions in nurturing are intended to prepare offspring of non-preferred males against predators.

In sum, more fit offspring receive greater nurturing than less fit offspring, even though nurturing appears to benefit offspring across environmental circumstances and even though the reduction in nurturing toward less fit offspring does not appear to be imposed by physical constraints. These practices amplify reproductive advantages experienced by fitter offspring. Over time, lineages with low nurturing levels are more likely to perish; however, other lineages endure stressors that identify them as less fit and lead to lower nurturing levels, which leads to a persistent population-level presence of low nurturing lineages.

3.3 Handicapping

It has been documented since Darwin (1871) that in many species, individuals (especially fitter males) manifestly exhibit traits and behaviors that give advantages to their enemies (particularly predators and prey) but are attractive to prospective mates. These sorts of exhibitions include bright colors (when camouflage would be favorable), vision obstructions, cumbersome and/or weighty ornaments, loud calls, and demonstrative displays (drawing predator attention) and have been documented in species of amphibians, fish, insects, mammals, plants, and reptiles (Zahavi & Zahavi, 1997). Zahavi (1975) proposed the handicap principle, arguing that such costly exhibitions are fundamental to reliably signaling their exhibitor's intrinsic condition to predators, peers, rival conspecifics and potential mates, since less fit individuals would not be able to overcome the associated expenses. Even though many apparently disadvantageous exhibitions by fitter males have been catalogued, the handicap principle has been subjected to unrelenting criticism on the theoretical grounds that a handicap is not a necessary or sufficient condition for reliable signaling (Getty, 1998; Getty, 2006; Higham, 2014; Szamado, 2011).

If a handicap has been selected merely to be a signal (as is supposed, according to the handicap principle), selection at the kin level would favor a low cost signal. Honest signals with minimal costs are achievable in numerous ways (reviewed in Szamado, 2011). If a lower cost signal is accessible but not selected, the handicap may be attributable to bilateral decrementation, according to which reservation of capacity (e.g., with a handicap) is utilized primarily to a) identify fitness in order to increase the reproductive share of the fittest individuals (especially those that are fittest against the lethal enemies against which the reservation is undertaken) and thereby intensify selection upon the individuals that practice bilateral decrementation and b) offer a more manageable challenge to

lethal enemies in order to attenuate selection upon these enemies. For example, Folstad & Karter (1992) observed that androgens a) help develop characteristics that attract mates and b) are associated with costs against parasites. Their immunocompetence handicap hypothesis holds that androgenic, sexually-selected characteristics signal that their exhibitor has ample fitness against parasites to compensate for the androgens. Evidence has been presented above that a) androgens do lead individuals to decrement fitness against parasites but that b) these costs are not imposed by physical constraints. Additionally, while the androgenic signals are visible to their recipients, the associated handicaps to defenses against parasites are not, making them unlikely index signal candidates (see section 3.4). Therefore, it is likely that selfish mutations decoupling the handicaps against parasites from the androgenic signals have been accessible in evolutionary time. The underrepresentation in nature of mutations decoupling the handicaps from the signals is indicative of countervailing selection in favor of bilateral decrementation, according to which the handicaps would offer reproductive benefits but lead to costs against lethal enemies.

3.4 Honest and uncostly signaling

Scholars are generally in accord that signal honesty can be maintained when there is a sufficient cost for signal dishonesty, such that dishonest signals are too costly to fake (Biernaskie et al., 2014; Higham, 2014). However, empirical inquiries have failed to uncover costs associated with many signals that are exhibited honestly (Hill, 2011; Husak & Swallow, 2011; Kotiaho, 2001; Warren et al., 2013). Scholars have advanced the index hypothesis to account for the honesty with which uncostly signals are exhibited. The index hypothesis states that a signal reporting a characteristic of an individual's condition is reliable if the signal is tied (i.e., indexed) to the characteristic in such a manner that a dishonest signal cannot be made (Maynard Smith & Harper, 1995). Early examples of indexing included spider web vibrations by funnel web spiders to convey information about weight and tree trunk scratching by tigers to communicate size (Maynard Smith & Harper, 1995). These signals are reported outside of the signaller's body and appear to be unalterably indexed to characteristics they represent. In other examples (such as back-arching by cats and leg-raising by dogs to indicate size: Hasson, 1997), the signal directly magnifies the represented characteristic. However, in some instances, the purportedly indexed signal is self-reported, represents a characteristic concealed from the signal's recipient, is directed toward a recipient that does not fully share the reporter's interests, and exhausts negligible resources.

The prevailing assumption is that index signals are static constraints that are not subject to selection (Biernaskie et al., 2014). However, Biernaskie et al., (2014) have argued that the criterion of differential costliness for low-quality individuals, which applies to the selection of other honest signals, also applies to the selection of honest index signals. Accordingly, if there have been mutations accessible in evolutionary time that would have allowed selfishly dishonest signals to replace honest index signals and would have cost less than the cost imposed by honesty, then their underrepresentation in nature implies selection at higher levels than kin (e.g., spatial, group).

For example, scholars have recently argued that in many species, some sexually-selected characteristics (e.g., ornaments, weapons) are indexed to multiple highly conserved biochemical pathways (Emlen et al., 2012; Hill, 2011; Hill, 2014; Hill & Johnson, 2012; Hill & Johnson, 2013; Johnson & Hill, 2013; Kuo et al., 2012; Warren et al., 2013). The honest exhibition of these characteristics is explained by their connections to particular biochemical pathways that, according to costly signaling theory, are unalterable by mutations without incurring costs that exceed the costs of honesty. Since a signal was, by definition, selected strictly for communicating information to others (Maynard Smith & Harper, 2004), there could have been, and ostensibly were, alternative mutations allowing a fully functional biochemical pathway that was decoupled from any signal, which would have allowed their holders to exhibit selfishly dishonest signals. Their absence implies selection for the coupling of the biochemical pathway and the signal, to the detriment of their exhibitor's inclusive fitness. Indeed, evidence indicates that the causal link between pathways (e.g., insulin/insulin-like growth factor) and sexually-selected characteristics is subject to selection (Emlen et al., 2012; Warren et al., 2013). Shared biochemical pathways are unlikely to maintain signal honesty if a selfish mutation is accessible via stepwise pathway evolution (Simons et al., 2015).

Carotenoid-dependent coloration is a widespread, well-studied class of signals used by many species of fish, reptiles, and birds to communicate multiple aspects of fitness (e.g., immunity and oxidative stress state) and is instrumental in repelling enemies and attracting mates (Simons et al., 2014; Svensson & Wong, 2011). The prevailing assumption in recent decades has been that carotenoids are limited and that individuals displaying them in signals forfeit the ability to utilize them in other physiological processes, which ensures the honesty of carotenoid-dependent signals (von Schantz et al., 1999; Svensson & Wong, 2011). However, in some species, individuals display significant variation in carotenoid coloration even when supplemented amply with carotenoids (Hill, 2006; Hill, 2014). In other species, individuals maintain carotenoid coloration despite diets deficient in carotenoids (Hill,

2006; Hill, 2014). Moreover, higher carotenoid levels evolve in response to the evolution of carotenoid-dependent coloration, which challenges the assumption that carotenoids are limiting (Simons et al., 2014). Hill & Johnson (2012) argued that the honesty of carotenoid ornaments is ensured by shared biochemical pathways between vitamin A regulatory mechanisms, redox systems and carotenoid pigmentation. However, Simons et al. (2015) criticized that vitamin A-redox hypothesis, citing empirical findings inconsistent with the hypothesis and proposing mutations that would allow an individual to cheat with a dishonest signal that would cost the cheater less than the cost imposed by honesty.

The zebra finch mating system is also instructive, due not to any anomalous characteristics associated with it, but because it is better known than that of any other bird (Forstmeier, 2004). Nowicki & Searcy (2004) advanced the developmental stress hypothesis to explain why developmental stressors lead to the deterioration of song exhibitions that zebra finch males use to repel other males and attract mates. According to their hypothesis, developmental stressors force zebra finches to forego investing in song in order to address the stressors, which makes song an honest signal of developmental stress levels endured by the zebra finch (Spencer & MacDougall-Shackleton, 2011). Indeed, male zebra finches do learn songs less precisely if they experience developmental stress and those that sing well tend to be more reproductively successful than others, indicating that songs are used by prospective mates as reliable signals of fitness (reviewed in: MacDougall-Shackleton & Spencer, 2012). However, multiple lines of evidence indicate that mutations would have been accessible that would have maximized a finch's song ability, irrespective of developmental stress. First, song deficits last long after the precipitating stressor has ceased, even though a mere elevation in social status can lead other songbirds to experience enhancements to the song control areas in their brains that allow them command over new song types (Voigt et al., 2007) and even though female zebra finches apparently lack the ability to verify the specific model from which the song was copied (Lachlan & Nowicki, 2012), which suggests the potential for post facto recovery. Second, while male zebra finches use song-control nuclei to sing, female zebra finches use song-control nuclei to evaluate male songs, yet developmentally-stressed female zebra finches show no diminished ability to differentiate between male songs (Woodgate et al., 2011), even though the preservation of the ability to differentiate between songs is less important to the reproductive success of the female than song is to the male. Third, stressed birds incur song deficits even after a song allotment demonstrated to be adequate for exact copying in other birds (Brumm et al., 2009). Indeed, in zebra finches (as in humans: see section 3.5), BDNF levels diminish in response to stress and Dittrich et al. (2013)

demonstrated experimentally that BDNF upregulation allows juvenile male zebra finches to copy song with maximal accuracy. In sum, the spontaneous song control augmentation that other songbirds experience, the ability exhibited by developmentally-stressed female zebra finches to differentiate between male songs, and the maximal accuracy with which juvenile male zebra finches copy after BDNF upregulation indicate that a mutation allowing maximally self-promoting signaling, irrespective of developmental stress, would have been accessible for lower costs than those imposed by honest signaling. The absence of such a mutation indicates selection at higher levels than kin exemplified by bilateral decrementation.

Moreover, male zebra finch song amplitude lowers as a result of stress (Ritschard & Brumm, 2012), making song amplitude an accurate indicator of stress experienced. Concurrently, female zebra finches exhibit greater attraction to males that sing with higher amplitude (Ritschard et al., 2010). However, zebra finches are capable of increasing their amplitude in response to background noise via the well-known Lombard effect (Brumm & Slabbekoorn, 2005), which suggests they otherwise refrain from their highest amplitude song, even though it is attractive to mates and even though it does not use appreciably more energy than other song—a phenomenon considered paradoxical (Zollinger et al., 2011). Thus, bilateral decrementation may also explain the signal honesty of song amplitude.

In sum, multiple examples demonstrate that less fit individuals signal honestly, despite the apparent accessibility of mutations that would allow selfishly dishonest signaling. This signaling honesty imposes reproductive costs upon these less fit individuals and constitutes an example of amplification.

3.5 Depressive responses

Depression is characterized by sadness, self-loathing, and anhedonia; impaired cognition, concentration, and memory; changes in psychomotor, sleeping, and eating patterns; and diminished fertility and libido (Anders et al., 2013; Austin et al., 2001; Sun et al., 2013). The prevalence of depression among people of all ages and societies (Andrews & Thomson, 2009) has led some scholars to hypothesize that depression was selected (e.g., Andrews & Thomson, 2009; Raison & Miller, 2013; Watson & Andrews, 2002), while others have disputed these hypotheses on the grounds that depression is maladaptive to the individual (e.g., Hagen, 2003; Nettle, 2004; Varga, 2012). In this

section, the argument is presented that both of these positions have valid elements: Depression is 1) a selected response to stress and 2) deleterious to the afflicted individual's inclusive fitness. In line with amplification, an individual that experiences chronic stress relative to conspecifics is able to infer that it is less fit compared to those conspecifics and the depressive symptoms that result from a diverse variety of chronic stress sources (Nestler, 2012; Sapolsky, 1998) diminish the afflicted individual's reproductive share. In line with reservation, depression is also associated with increased immune activity against parasites and pathogens, which has inspired multiple scholars to hypothesize that depression has evolved as a response to these enemies. The reproductive deficits and the advantages against lethal enemies that are visited upon less fit individuals by depression are consistent with bilateral decrementation.

The prevalence of depression is demonstrated by its inclusion among the top five causes of disability throughout the world (Caspi et al., 2003; Hagen, 2003). Thirty percent of lost productivity worldwide is believed to be caused by stress-precipitated psychiatric pathologies, such as depression, anxiety and schizophrenia (Nestler, 2012). Depression increases the mortality rate for all major disease-related causes of death (Mykletun et al., 2007) and is a significant risk factor for suicide (Hagen, 2003), which claims approximately one million people worldwide each year (Hawton & van Heeringen, 2009). Individuals in many species are susceptible to a condition analogous to depression, which is often referenced as learned helplessness (Seligman, 1976). Learned helplessness is provoked by stress, is characterized by many of the symptoms associated with depression (e.g., anhedonia, psychomotor retardation, reduced self-care, and diminished appetite and libido), and has been induced, via application of uncontrollable stress, in many species, including insects, cats, dogs, rats, birds, fish, and primates (Franklin et al., 2012; Sapolsky, 1998; Weiss, J. et al., 1982). Though there are obstacles in determining the intent, some models of learned helplessness have included suicide and self-mutilation (Crawley et al., 1985; Preti, 2011; Sapolsky, 1998; Seligman, 1976). These symptoms are considered maladaptive and are reversed in animals by the same therapies that are used to treat depression in humans (Russo et al., 2012; Weiss, J. et al., 1982).

Depressive responses to stress show indications of selection. Depression is the leading cause of disability and loss of productivity during peak working and childbearing years (Kinney & Tanaka, 2009), ages at which selection is strong (Medawar, 1952). Moreover, the link between stress and depression (or learned helplessness) is mediated epigenetically. In humans and numerous species of non-human primates and rodents, individuals are more

likely to develop depressive symptoms if chronic stress was imparted: a) to their descendants (Dietz et al., 2011; Franklin et al., 2010; Franklin et al., 2011; Gapp et al., 2014; Weiss, I. et al., 2011); b) to the individuals prenatally (Lupien et al., 2009; Maccari et al., 2003; Vialou et al., 2013; Weinstock, 2005; Weinstock, 2010); or c) to the individuals during their lives (Fuchikami et al., 2010; Nestler, 2012).

Chronic stress consistently leads to an epigenetically-mediated reduction in brain-derived neurotrophic factor (BDNF) expression (Martinowich et al., 2007) and strong empirical support exists for a causal role for reduced BDNF expression in depressive responses (Duman & Monteggia, 2006). Depressed individuals have lower BDNF levels than other individuals (Duman & Monteggia, 2006; Martinowich et al., 2007; Wolkowitz et al., 2011) and virtually every effective antidepressant enhances BDNF expression (Castren et al., 2007; Duman & Monteggia, 2006; Martinowich et al., 2007; Nibuya et al., 1995; Wolkowitz et al., 2011), including chemical antidepressants, electroconvulsive shock treatment, and physical exercise. Additionally, direct infusions of BDNF into the hippocampus have been found to engender antidepressant effects (Martinowich et al., 2007). Furthermore, it does not appear that the depression resulting from reduced BDNF expression is a spandrel (Gould & Lewontin, 1979) of a selected effect, such as energy conservation, since caloric restriction increases BDNF levels (Duan et al., 2001).

While antidepressants increase BDNF levels, reduced BDNF expression does not necessarily induce depression (Groves, 2007; Martinowich et al., 2007; Wolkowitz et al., 2011), indicating that lowered BDNF expression mediates depression in conjunction with other factors, which are also shaped by chronic stress. Critically, the causal pathway between stress and depression is more nuanced than a simple BDNF reduction: While low BDNF expression overall, and in the hippocampus particularly, is caused by stress via epigenetic modifications and leads to depression (Nibuya et al., 1995; Smith et al., 1995), enhanced BDNF expression in the nucleus accumbens is also caused by stress via epigenetic modifications and has also been found to lead to depression (Russo & Nestler, 2013; Walsh et al., 2014). Thus, while BDNF expression has been implicated as a factor through which stress mediates depression, stress has location-specific effects on BDNF expression, each of which results from epigenetic modifications and each of which mediates depression, which indicates strongly that depression does not result from a process gone awry, but rather is a selected response to chronic stress.

Scholars have struggled to offer potential benefits for even mild depressive symptoms (Raison & Miller, 2013). The analytical rumination hypothesis (Andrews & Thomson, 2009) is the most heavily cited of the recent

attempts to explain depression. According to the analytical rumination hypothesis, depression evolved as a mechanism for discouraging distractions and directing an individual's energy into solving the complex problems that are the sources of the individual's stress. Andrews & Thomson (2009) acknowledged the poorer cognitive performance on some lab tasks by people with depression and argued that the poorer performance results from channeling one's focus into solving the problems that are the sources of depression. However, first, it is unclear why self-loathing and its associated costs must be incurred merely to compel an agent to focus upon a source of stress, since people are capable of obsessively contemplating a problem without being depressed. Second, depressive symptoms are common to individuals of many species (e.g., mice, rats, cats, birds, fish) that are not known to ruminate extensively. Third, the deficits in appetite and sleep associated with depression are inconsistent with an individual who requires an abundance of energy for investment into a cognitive task. Fourth, depression is associated with a feeling that one's situation is hopeless and that the precipitating stress sources are uncontrollable (Sapolsky, 1998; Weiss, J. et al., 1982). Either the sources of stress are intrinsically uncontrollable, in which case depressive contemplation is futile and unnecessarily costly, or the feeling of uncontrollability is an effect of depression, in which case it disincentivizes the individual from focusing upon the situation, which is inconsistent with the attention-orienting effect it is hypothesized to have. Fifth, Andrews & Thomson (2009) argued that people experiencing depression perform poorer on cognitive tasks because they are busy directing their mental energy into thinking about the sources of the depression, yet the ability to ponder seemingly unrelated questions often benefits an individual in solving a mental task by allowing the task to be solved by analogy (Dunbar, 2001; Polya, 1957). In sum, depression does not appear to have been selected to orient an individual toward mental focus.

Some scholars have offered that depression may have multiple applications, with each determinant responsible for its own depressive feature. However, while many kinds of stress may lead to depression, the depressive response includes some common, costly effects (e.g., self-hatred, self-neglect, and cognitive impairments) which are difficult to reconcile with any of the prominent evolutionary explanations for depression. Alternatively, the bargaining model of depression, according to which individuals exhibit depressive symptoms in order to communicate a lack of cooperation with current terms (Hagen, 2003), accounts fairly well for these costs, but does not explain well-documented associations between depression and immunity activation. Multiple scholars have cited these associations in introducing their hypotheses that depression evolved to protect hosts and their kin from infection by parasites or pathogens (Anders et al., 2013; Raison & Miller, 2013). These scholars have pointed

to evidence for a mutually causal relationship between immune activation and depression, noting that immune activation leads to depression, whereas depressive symptoms are associated with greater resistance against infections. According to these views, depressive behavioral symptoms have been selected for the purpose of: 1) conserving metabolic resources (e.g., for immune activation, fever generation, and tissue repair); 2) reducing risk of infection for kin by withdrawing socially; 3) inhibiting appetite for foods counterproductive during an infection; and 4) protecting against threats engendered by sickness (e.g., increased risk for predation, reduction in ability to care for offspring, and loss of status and territory to conspecifics) with hypervigilance. Accordingly, the immunity activation and depression that occur in response to a wide range of stressors (including psychosocial stressors) are adaptive, since in evolutionary time, most stressors related to hunting, escaping predators, or fighting conspecifics in dominance hierarchies, and infection risk was increased by wounds associated with these activities.

The hypotheses that suggest that depression was selected on the kin level to fight infections are seriously challenged by the wide range of stressors that lead to depression. Many of these stressors existed in evolutionary time (e.g., competitive losses against conspecifics; rejections by prospective mates; sickness or impairments due to noninfectious defects; adversity in youth, which could have been caused by any of the above afflictions to one's progenitors) and would not have suggested enough of an increase in infection risk to justify the costs imposed by depression. Even if the individual were afflicted with an infection, mutations were presumably accessible in evolutionary time that would have protected kin of the afflicted by imparting a preference for solitude in response to infection and that would have spared the individual the costs associated with self-hatred and self-neglect. Additionally, while depressive symptoms are hypothesized to conserve metabolic resources, the lethargy associated with depression also impairs individuals in procuring metabolic resources. Moreover, if depression were a selected response against infection risks posed by hunting, escaping predators, and fighting conspecifics, one would expect males to be both more susceptible to depression and more oriented toward fighting infection, since males have been, in evolutionary time, more likely to experience those stressors. Instead, males are less susceptible to developing depression (Andrews & Thomson, 2009) or are as susceptible once gender ideals are considered (Martin et al., 2013).

Sapolsky (1998) and Suomi, J. (1997) have documented subsets of, respectively, baboons and rhesus monkeys disposed to inefficient stress responses. These animals apparently do not distinguish in their reactions to

threatening and innocuous situations: Their resting glucocorticoid levels are pathologically higher than the average but their glucocorticoid response to a real stressor is smaller and slower (Sapolsky, 1998). This condition is both a cause and an effect of diminished maternal nurturing and is characterized by social deficiencies. Therefore, Sapolsky (1998) and Suomi, J. (1997) each considered it maladaptive. Sapolsky (1998, 293) found the mechanisms leading to the inefficient glucocorticoid pattern in baboons to be "virtually identical" to those in humans with major depression and noted inefficient stress responses to be common to chronically stressed individuals in many other species (e.g., rats, mice, hamsters, guinea pigs, wolves, rabbits, pigs, fish). Suomi, J. (1997) estimated that this inefficiency characterizes twenty percent of rhesus monkeys, which is roughly the same portion of the human population that experiences major depressive disorder (Andrews & Thomson, 2009). However, in rhesus monkeys (and presumably other species), the inefficient stress response can be prevented by fostering the animals to nurturant mothers (Sapolsky, 1998). Thus, the inefficient stress response is maladaptive, yet is also apparently under maternal influence, which is the same pattern noted above for other species. Since the inefficient stress response is both a cause and an effect of diminished maternal nurturing, it persists within a lineage across generations. Sapolsky (1998) anticipated that after long-term natural selection, the population would be comprised of individuals with efficient stress responses. However, since these animals have already been subjected to natural selection over evolutionary time, the more likely interpretation is that animals with an inefficient stress response are indeed disadvantaged and that their genes are less likely to reach subsequent generations, but that over the generations, a frequency of inefficient stress responses persists within a population as new lineages adopt this characteristic (that is, are decremented) after a history of chronic stress (that is, after an indication that they are less fit).

3.6 Self-handicapping

Self-handicapping is a practice by which people report (or introduce) obstacles that inhibit their own performance (Jones & Berglas, 1978). Multiple forms of self-handicapping have been identified (Leary & Shepperd, 1986). Claimed self-handicapping is characterized by individuals' claims that their performance has been handicapped by factors external to their volition. Behavioral self-handicapping involves individuals' introduction of obstacles that impede their performance (e.g., alcohol and drug use, selection of unattainable goals, taking on too many projects, staying out late the night before a performance, lack of practice and/or effort). While claimed self-

handicapping has generally been interpreted to be a self-serving strategy, behavioral self-handicapping has been viewed as a paradox, since individuals who engage in behavioral self-handicapping incur costs without receiving an obvious benefit.

The most popular explanation for behavioral self-handicapping is that it is practiced by individuals who anticipate a failing performance, in order to prevent an accurate evaluation of that performance (Urdan & Midgley, 2001). This usage of a handicap to prevent accurate evaluation may appear to conflict with the argument made in the present paper that reservation (e.g., with a handicap) is utilized to identify ability, but the contradiction is only apparent. In the present paper, the argument has been made that reservation can either help identify or disguise performance on a task, depending upon whether the task is measured in a binary or metered manner and how well the effects of the reservation are taken into account. Self-handicapping helps prevent accurate appraisal of performance because it is used in situations in which it is usually not difficult to differentiate between the performances of multiple individuals (e.g., standardized exams, athletic performances) and because it is used in ways in which the hindrance to performance from self-handicapping is not carefully measured.

Since individuals engage in behavioral self-handicapping even when it is not observable by others (Jones & Berglas, 1978; Rhodewalt, 2008), researchers have hypothesized that behavioral self-handicapping primarily occurs not to influence others' perceptions but rather to reduce the damage to one's own self-esteem (Kolditz & Arkin, 1982; Zuckerman et al., 1998). This suggests that low self-esteem is an unfavorable reaction to a failing performance, which invites a query about why it wasn't selected against. In consideration of the substantial overlap between depression and low self-esteem (Sowislo & Orth, 2013) and the argument made above that depression has been selected despite being detrimental to the individual, it is possible that self-handicapping was selected on the individual level to prevent against low self-esteem and that a contingency for low self-esteem was selected at higher levels. However, while some scholars allow that behavioral self-handicapping exerts a short-term positive influence upon an individual's self-esteem, the preponderance of studies have found that behavioral self-handicapping is long-term costly (Rhodewalt, 2008; Schwinger et al., 2014; Urdan & Midgley, 2001; Zuckerman & Tsai, 2005) and that behavioral self-handicapping and maladjustment reinforce each other in a self-perpetuating cycle as behavioral self-handicapping leads to performance costs, which leads to further behavioral self-handicapping (Rhodewalt, 2008; Urdan & Midgley, 2001; Zuckerman et al., 1998; Zuckerman & Tsai, 2005). In line with amplification, this cycle

involves a poor performance, which leads to feedback indicating that the poorly performing individual is less fit and then subsequent self-handicapping that detracts from future performance (i.e., influences his or her reproductive outlook in the direction of that feedback).

3.7 Reproductive forfeiture

Reproductive skew, which refers to an imbalance of reproductive output across individuals, commonly occurs in cooperatively breeding species (Clutton-Brock et al., 2010; Johnstone, 2000), which is paradoxical, since subordinates cooperatively care for the offspring of dominants without having their own offspring (Saltzman et al., 2009). In some species, a group's subordinates are completely bereft of reproductive ability; however, their fertility and libido quickly return if the dominant group members depart the group (Sapolsky, 2001). Subordinates' reproductive inhibition (which has been called "socially induced suppression," "physiological suppression," and "social contraception") has been documented in mammals, birds, fish, and invertebrates (Saltzman et al., 2009) and sometimes occurs even though subordinates show comparable body condition to dominants and overlap dominants substantially in age (Harrison et al., 2013; Saltzman et al., 2009). The traditional interpretation of reproductive inhibition is that subordinates reproductively suppress themselves to avoid bullying by dominants and to avoid incurring the costs of producing offspring that would be terminated by dominants (Beehner & Lu, 2013; Saltzman et al., 2009). However, levels of subordinate submissiveness and reproductive inhibition often vary within a group. Ovulatory subordinates are less submissive than anovulatory subordinates and individual differences in aggressiveness and submissiveness reliably predict subsequent social status, supporting the idea that a female's suppression of ovulation is determined primarily by that female's perception of herself as a subordinate, rather than her receipt of agonistic behaviors (Saltzman et al., 2009).

Relatedly, in many cooperatively breeding mammals and birds, females apparently have higher variance in reproductive success than males due to the reproductive inhibition of subordinate females. However, in these species, there appears to be paradoxically little selection among females for size and armaments, despite the advantages they afford females in dominance acquisition and retention (Stockley & Bro-Jorgensen, 2011; Young & Bennett, 2013). Extant evolutionary explanations (Young & Bennett, 2013) are unconvincing. Higher costs of

combat incurred by females do not explain the lack of selection for characteristics that facilitate combat, since if the costs were high enough to preclude combat, dominant females could not enforce their dominance through the threat of combat. The potential for established power asymmetry between females in different stages of development also does not adequately explain the lack of selection for combat characteristics, since, as indicated above, subordinate females are of similar age as dominants in numerous examples of reproductive skew. Finally, lesser variance in inclusive fitness between females does not appear to explain the lack of selection for female combat characteristics, since male-biased size dimorphism evidently still occurs in species in which mean relatedness is higher among competing males (Young & Bennett, 2013).

Moreover, a thorough review revealed that there is little evidence that the behavior of members in cooperatively breeding groups is governed by an evaluation of patterns of relatedness or competitive ability in order to set reproductive skew (Nonacs & Hager, 2011). The review's authors concluded that some individuals lose inclusive fitness by disadvantageously joining, or staying in, groups and they invoked group selection as a primary reason that the data did not match up with prevailing theory (Nonacs & Hager, 2011).

Evidence also suggests that the well-studied zebra finches engage in decrementation of reproductive fitness after experiencing chronic stress. Developmentally-stressed female zebra finches are considerably less active in mate choice trials despite no apparent deficits in their ability to differentiate between male levels of attractiveness (Woodgate et al., 2010; Woodgate et al., 2011). Additionally, Rhainds (2010) has documented that in over 100 species of insects, a significant fraction of females remain unmated through their lifetime, despite being functionally reproductive. In at least 20 species, the percentage of unmated females is greater than 50%. While the lack of female reproductive participation is more paradoxical than that of males, owing to the expectation that females would be more reproductively selective (Trivers, 1972), males in some species also exhibit diminished reproductive interest (though increased homosexual tendency) in response to multiple stress sources (Poiani, 2010; Ward, 1972).

In sum, it appears that chronically stressed females in many species engage in amplification by foregoing reproductive opportunities. This accounts for the observation that members in cooperatively breeding groups do not maximize inclusive fitness and explains the lack of selection for female size and armaments, as the underlying determinant upon female reproductive success is not the ability to combat other females but rather feedback that the female has received about its relative fitness. This influence of relative fitness upon reproductive success may be

exerted through size and armaments which are coupled with, and fluctuate with, relative fitness. Selection would favor this coupling itself—at higher levels than kin (e.g., spatial, group). When female armaments are selected, it may be primarily for use against natural enemies (e.g., against predators: Stockley & Bro-Jorgensen, 2011).

3.8 Foliar anthocyanins

In plants, stressors such as wounding, pathogen attack, nutrient deficiency, UV-B radiation, transplant shock, and high light combined with cold temperatures often lead to leaf reddening, which is proximately attributable to the plant's extra production of anthocyanins and which imposes a photosynthetic cost, as a non-photosynthetic pigment competes with chlorophylls for photon capture (Manetas, 2006). In consideration of the associated photosynthetic cost, the reddening of plant leaves in response to stress has been considered a paradox by scientists for the past century, as many explanations have been offered but none have been accepted without controversy (Kovinich et al., 2014; Manetas, 2006). Since plants produce anthocyanins in response to a wide range of stressors, some plant physiologists have advanced hypotheses explaining anthocyanin production as a reaction to abiotic stressors, such as strong light, UV-B radiation, temperature extremes, heavy metals and drought. However, these hypotheses have been substantially criticized on theoretical grounds (Hatier & Gould, 2008; Manetas, 2006). One popular hypothesis of this kind is that anthocyanins serve as antioxidants that neutralize reactive oxygen species, which arise during stress (Gould et al., 2002). However, other scholars have argued that anthocyanins do not appear to be spatially or temporally oriented toward fulfilling this function, particularly in comparison with phenolics and flavonoids (Hernandez et al., 2009; Manetas, 2006). Hypotheses suggesting that plants signal red leaf color in order to prevent attack by herbivores have also been plagued by conceptual problems (Archetti, 2009). Schaefer & Rolshausen (2006) advanced the defense indication hypothesis, noting that while direct evidence of anthocyanin activity against herbivores is tenuous, the production of defensive compounds and the production of anthocyanins as pigments are connected by a common biosynthetic pathway, leading the anthocyanins to be produced during stress along with defensive compounds. As a result, they argued, herbivores grew to associate the color red with a plant's high defensive commitment and to generally avoid red plants for this reason. However, it is unclear why, according to this hypothesis, the coupling of defensive compounds and anthocyanins should have become widespread in the first place, since this coupling does not appear to have benefited an individual holder.

While numerous alternative mutations were likely accessible in evolutionary time, the common coupling of photosynthetic costs and herbivore defenses accords with the patterns of bilateral decrementation, by which less fit individuals engage in decrementation of reproductive fitness but augment defenses against lethal enemies.

3.9 Heterosis

Heterosis, a phenomenon documented by biologists for hundreds of years, is the general improvement in biological functionality (e.g., fertility, development speed, biomass, height, resistance to stressors, photosynthesis) in a hybrid offspring compared to its best functioning parent (Birchler et al., 2003; Birchler et al., 2010; Chen, 2013). While heterosis has been studied most extensively in plants, it has also been reported in studies on height and, controversially, intelligence and attractiveness in humans; growth and reproductive traits in mice and farm animals; and stress responses in fungi (Chen, 2013). Though heterosis has been subjected to centuries of commercial and scholastic inquiry, little consensus has emerged about its proximate mechanisms or ultimate evolutionary significance (Baranwal et al., 2012; Birchler et al., 2003). Early explanations centered upon the dominance hypothesis, according to which the superiority of hybrids results from the suppression of deleterious recessive alleles from one parent by dominant alleles from the other, and the overdominance hypothesis, according to which hybrids benefit from heterozygous combinations of alleles that would be less favorable if solely expressed. These two explanations are not mutually exclusive and neither implies selection for heterosis itself. That is, they explain how fitness benefits accrue to hybrids but do not imply selection for the accrual of those fitness benefits to hybrids. However, in recent decades, scholars have argued that these two explanations cannot account fully for heterosis, citing the unresponsiveness of the magnitude of heterosis to improvement in inbred lines, the progressive heterosis in tetraploids, and the rapidity in inbreeding decline compared to what would be predicted based on the homozygosis of alleles (reviewed in Birchler et al., 2003). Recently, an epigenetic contribution to heterosis has been established in both plants and animals (Baranwal et al., 2012; Fu et al., 2015; Han et al., 2008), which suggests that heterosis itself may have been selected. Moreover, Semel et al. (2006) noted findings that demonstrated that, in multiple species, heterosis occurs primarily among reproductive traits, which, they argued, constitutes evidence that heterosis itself was selected to promote heterozygosity in populations, in a manner analogous to a plant's self-incompatibility system.

The applicability of heterosis in offering credibility for bilateral decrementation is two-fold:

1) Amplification potentially offers a partial explanation for heterosis, since fitness advantages accrue to offspring from simple complementation and the additional advantages in heterosis that cannot be explained by simple complementation are consistent with amplification.

2) Though selection for heterozygosity does not involve selection that facilitates selection, it offers another example of selection for a mechanism that promotes evolution (e.g., segmentation, self-incompatibility, sex), of which the theory advanced in the present paper would offer another instantiation.

3.10 Condition-dependent, offspring sex-ratio variation

Trivers & Willard (1973) hypothesized that, since condition is both substantially heritable and a determinant of competitive success, parents in good condition would garner more grandoffspring by producing more offspring of the gender that has more variable reproductive success, while parents in poor condition would garner more grandoffspring by producing more offspring of the gender that has less variable reproductive success. Therefore, if offspring sex ratio were characterized by amplification, less fit individuals would decrement their share of grandoffspring by producing more males in species in which the male has more variable reproductive success, since this would lead to their producing a lower quantity of grandoffspring than otherwise. Scholars generally recognize males to have more variable reproductive success in most animal species and there is empirical support for a U-shaped relationship between parental dominance and male births, as more dominant individuals produce more males but socially stressed animals also produce more males (Cameron, 2004). The production of more males by dominant individuals is consistent with the Trivers-Willard hypothesis. However, sex ratio and maternal condition may be related for reasons unrelated to the Trivers-Willard hypothesis (Sheldon & West, 2004). For example, female-biased sex ratios in response to poor condition have been documented most consistently when the source of poor condition is a food supply deficiency (Cameron, 2004), and a female-biased sex ratio during food shortages can be explained by selection on both the individual and group levels (Clutton-Brock et al., 1985). The production of more males by socially stressed animals is, so far, unexplained (Cameron, 2004), but is consistent with amplification: The Trivers-Willard hypothesis predicts that the mother in poor condition obtains more

grandoffspring by producing more females, so the production of more males imposes upon a less fit mother a diminished share of grandoffspring.

4. Summary

The strategy of bilateral decrementation has been introduced, along with a theoretical analysis that shows that it is selectable. Individuals that practice bilateral decrementation make adjustments that lead fitter practicing individuals to 1) enhance their reproductive advantages over less fit practicing individuals (which is called amplification) but 2) incur disadvantages against the lethal enemies in comparison to less fit practicing individuals (which is called reservation). Consequentially, selection intensity increases upon the holders of the allele practicing bilateral decrementation and decreases upon their mutant conspecifics and, particularly, their lethal enemies.

While legions of strategies have been reported in leading biology journals to be theoretically selectable under certain parameters, the salience of bilateral decrementation owes to its ability to explain at least the ten different empirical phenomena documented above, many of which have, heretofore, been considered paradoxical and are uniquely elucidated by bilateral decrementation. In these reviewed cases, at least part of the reproductive forfeitures by less fit individuals are not recompensed as additional ability against lethal enemies and/or at least part of the reservation by fitter individuals is not recompensed as gains in reproductive share. This is most manifestly evident when considering the cases of honest but uncostly signaling, self-handicapping, reproductive forfeiture, and the influence of stress upon offspring sex ratio. These phenomena, which are characterized by forfeitures by less fit individuals to fitter individuals, have seldom, if ever, been explained in terms of gains received against natural enemies. Other phenomena reviewed above have, indeed, been explained in terms of tradeoffs: Androgen elevations (and the associated reproductive advantages) are presumed to be traded off against vulnerability to parasites, lapses in maternal nurturing lead to reproductive deficits but it has been argued that they prepare offspring versus predators, handicapping yields reproductive advantages versus conspecifics but vulnerability against natural enemies, depressive responses incur reproductive deficits but have been argued to be adaptive responses to parasite and pathogen threats, and foliar anthocyanins involve photosynthetic costs but guard against herbivores. In the present paper, it has been argued that these tradeoffs are not obligate. That is, there have been mutations accessible

in evolutionary time that would have prevented the reproductive costs or, where applicable, decoupled the reproductive costs from the benefits against lethal enemies, allowing only the benefits. Rather, it has been argued that the offsetting of the amplification of reproductive advantages experienced by fitter individuals and the reservation against lethal enemies by those fitter individuals has itself been selected.

Amplification and reservation occur in response to feedback about long-term evaluations of fitness. There appears to be evidence in humans of a tendency to act in a manner that redresses the fitness effects of undeserved feedback that is inconsistent with longer-term, more deeply ingrained feedback (Hattie & Timperley, 2007). Inquiry into analogous phenomena in other species may be productive.

Bilateral decrementation involves forfeitures that do not preferentially impart resources to kin. The prevalence with which it is practiced, at ages during which selection is strong, indicates robust selection at higher levels than kin. The issue of selection at higher levels than kin has remained controversial for the past five decades, after work by Hamilton (1964) and Trivers (1971) demonstrated that prosocial behavior could be explained by selection on the kin level and Williams (1966) argued that characteristics traditionally attributed to group selection invariably benefit the individual and its kin as well as the group and that due to the greater strength of kin selection, characteristics that benefit the group at the expense of the individual are, for the most part, not selected. While the denouncement of group selection in the 1960s was largely due to its theoretical improbability (Wilson, 1983; Wilson & Wilson, 2007), contemporary biologists generally agree that group selection is theoretically possible (Nowak, 2012; see also Pinker, 2012 and its invited commentary) and are, rather, more apt to criticize its invocation by questioning its usefulness in explaining natural phenomena. However, the paucity of generally accepted invocations of group selection in accounting for empirical phenomena owes largely to the denunciation of group selection five decades ago and its consequent inadmissibility in evolutionary explanations (Eldakar & Wilson, 2011; Wilson, 1983). While scattered empirical support for selection at higher levels than kin may be dismissed as a result of this catch-22, the instantiations of bilateral decrementation reviewed herein are commonly practiced by individuals in many species, which illustrates robust selection at higher levels.

A related point is that, contrary to prevailing assumptions, selection of strategies at a higher level does not necessarily lead to the optimization of the short-term quantity of individuals at that level. Competition between individuals that enables the identification of the fittest of those individuals can be selected on the group level even if

that practice temporarily reduces group quantity. Analogously, filial cannibalism and parent-sponsored siblicide (Klug, 2007a) have been explained by selection on the individual level even though these practices often reduce a progenitor's lifetime offspring produced (Klug & St Mary, 2005), which Klug (2007b) attributed partly to reduction of filial competition for resources and elimination of poorer quality offspring (Klug, 2007b).

The cases of bilateral decrementation reviewed above indicate that in nature, bilateral decrementation may not be practiced in a perfectly efficient manner. Even the fittest individuals that practice bilateral decrementation may underperform their reproductive capacity, as fitter individuals' reservation against lethal enemies may exert incidental reproductive effects. There is also the potential for a "false start," in which an individual decrements its reproductive ability early in its development in response to unfavorable feedback and subsequently increments its reproductive ability in response to favorable feedback. In this case, the individual may not be able to fully recover the forfeitures associated with the original decrementation. While such occurrences would not interfere with the identification of the genetically fittest individuals that practice bilateral decrementation, these practicing individuals may cede an advantage to mutant conspecifics or to individuals in other species that pursue the same prey. Another potential source of inefficiency is that the manifestations of androgenic deficits, nurturing deficits, depression, self-handicapping, and photosynthetic deficits may impair less fit individuals not just against fitter individuals with the same allele but also against the specific lethal enemies against which fitter individuals reserve capacity. These examples imply competition between different levels of selection: More parsimonious and direct means of amplification or reservation have evidently not been selected due to selection on the individual level. Some exemplifications of decrementation in nature may involve reproduction by less fit individuals, which is also consistent with competition between differing levels of selection, but which may be attributable to selection for diversity (Simmons, 2005), whether on the individual or group level. This may be particularly true under conditions of rapid natural enemy evolution, in which case progenitor fitness is less of a reflection upon offspring fitness.

Certain features of amplification and reservation may be subject to selection, including the intensity of the amplification in response to certain feedback, the intensity of the reservation, and the enemies against which reservation occurs. Selection at the group level would favor minimal variation between individuals in a group, which would allow a more accurate evaluation of each individual's relative fitness. However, there may be variation from population to population. The groups that are most successful at surviving lethal enemies would

figure to reserve in a manner that best accomplishes the objectives of reservation against those enemies. As a result of selection, this reservation may become specifically tailored to the species of the most pernicious enemies facing the group. (If so, reservation would continue to be useful against related enemy species.) This conjures the idea that therapies against infectious diseases (which, in some species, include cancers: Metzger et al., 2016) may be informed by focusing upon the cellular or epigenetic differences in defenses between the fit individuals and less fit individuals of a population (whether that population be comprised of humans or other species), provided that these infectious diseases are sufficiently lethal that reservation occurs. Additionally, the prevalence of bilateral decrementation among species in nature raises the notion that bilateral decrementation is a fundamental property of systems shaped by natural selection, irrespective of level. For example, it is possible that a microcosm of amplification and/or reservation occurs at the level of the immune system within a single individual. Accordingly, it may be productive to train or otherwise modify an immune system facing a foreign enemy to amplify and/or reserve in a manner analogous to the amplification and/or reservation practiced within the immune systems of the individuals in the populations most accustomed to defending against that enemy.

Acknowledgements

References

Anders, S., Tanaka, M. & Kinney, D. 2013. "Depression as an evolutionary strategy for defense against infection." *Brain Behav. Immun.* (31): 9-22

Andersson, M. 1994. *Sexual Selection*. Princeton: Princeton University Press

Andrews, P. & Thomson, J., Jr. 2009. "The bright side of being blue: Depression as an adaptation for analyzing complex problems." *Psychol. Rev.* 116(3): 620-654

Archetti, M. 2009. "Classification of hypotheses on the evolution of autumn colors." *Oikos* 118(3): 328-333

Austin, M., Mitchell, P. & Goodwin, G. 2001. "Cognitive deficits in depression: possible implications for functional neuropathology." *Br. J. Psychiatry* 178(3): 200-206

Baranwal, V., Mikkilineni, V., Zehr, U., Tyagi, A. & Kapoor, S. 2012. "Heterosis: emerging ideas about hybrid vigour." *J. Exp. Bot.* 63(18): 6309-6314

Beehner J. & Lu, A. 2013. "Reproductive Suppression in Female Primates: A Review." *Evol. Anthropol.* 22(5): 226-238

Berglund, A. 1993. "Risky Sex- Male Pipefishes Mate At Random In The Presence Of A Predator." *Anim. Behav.* 46(1): 169-175

Biernaskie, J., Grafen, A. & Perry, J. 2014. "The evolution of index signals to avoid the cost of dishonesty." *Proc. R. Soc. Lond. B.* 281(1790): 20140876

Birchler, J., Auger, D. & Riddle, N. 2003. "In Search of the Molecular Basis of Heterosis." *Plant Cell* 15(10): 2236-2239

Birchler, J., Yao, H., Chudalayandi, S., Vaiman, D. & Veitia, R. 2010. "Heterosis." *Plant Cell* 22(7): 2105-2112

Bolund, E., Schielzeth, H. & Forstmeier, W. 2009. "Compensatory investment in zebra finches: females lay larger eggs when paired to sexually unattractive males." *Proc. R. Soc. Lond. B.* 276(1657): 707-715

Bosch, O., Musch, W., Bredewold, R., Slattery, D. & Neumann, I. 2007. "Prenatal stress increases HPA axis activity and impairs maternal care in lactating female offspring: Implications for postpartum mood disorder." *Psychoneuroendocrinology* 32(3): 267-278

Braga Goncalves, I., Mobley, K., Ahnesjo, I., Sagebakken, G., Jones, A. & Kvarnemo, C. 2010. "Reproductive compensation in broad-nosed pipefish females." *Proc. R. Soc. Lond. B.* 277(1687): 1581-1587

Brumm, H. & Slabbekoorn, H. 2005. "Acoustic communication in noise." *Adv. Study Behav.* (35): 151-209

Brumm, H., Zollinger, S. & Slater, P. 2009. "Developmental stress affects song learning but not song complexity and vocal amplitude in zebra finches." *Behav. Ecol. Sociobiol.* 63(9): 1387-1395

Burley, N. 1986. "Sexual Selection for Aesthetic Traits in Species with Biparental Care." *Am. Nat.* 127(4): 415-445

Byers, J. & Waits, L. 2006. "Good genes sexual selection in nature." *Proc. Natl. Acad. Sci. U.S.A.* 103(44): 16343-16345

Cameron, E. 2004. "Facultative adjustment of mammalian sex ratios in support of the Trivers-Willard hypothesis: evidence for a mechanism." *Proc. R. Soc. Lond. B.* 271(1549): 1723-1728

Caspi, A., Sugden, K., Moffitt, T., Taylor, A., Craig, I., Harrington, H., McClay, J., Mill, J., Martin, J., Braithwaite, A. & Poulton, R. 2003. "Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene." *Science* 301(5631): 386-389

Castren, E., Voikar, V. & Rantamaki, T. 2007. "Role of neurotrophic factors in depression." *Curr. Opin. Pharmacol.* 7(1): 18-21

Champagne, F., Francis, D., Mar, A. & Meaney, M. 2003. "Variations in maternal care in the rat as a mediating influence for the effects of environment on development." *Physiol. Behav.* (79): 359-371

Chen, Z. 2013. "Genomic and epigenetic insights into the molecular bases of heterosis." *Nat. Rev. Genet.* 14(7): 471-482

Clutton-Brock, T., Albon, S. & Guinness, F. 1985. "Parental investment and sex differences in juvenile mortality mammals." *Nature* 313(5998): 131-133

Clutton-Brock, T., Hodge, S., Flower, T., Spong, G. & Young, A. 2010. "Adaptive Suppression of Subordinate Reproduction in Cooperative Mammals." *Am. Nat.* 176(5): 664-673

Crawley, J., Sutton, M. & Pickar, D. 1985. "Animal Models of Self-Destructive Behavior and Suicide." *Psychiatr. Clin. North Am.* 8(2): 299-310

Curley, J., Mashoodh, R. & Champagne, F. 2011. "Epigenetics and the origins of paternal effects." *Horm. Behav.* 59(3): 306-314

Darwin, C. 1871. *The descent of man and selection in relation to sex.* London: John Murray

Dawkins, R. 1982. *The Extended Phenotype.* Oxford: Oxford University Press

Denenberg, V. & Karas, G. 1959. "Effects of differential infantile handling upon weight gain and mortality in the rat and mouse." *Science* 130(3376): 629-630

Dietz, D., LaPlant, Q., Watts, E., Hodes, G., Russo, S., Feng, J., Oosting, R., Vialou, V. & Nestler, E. 2011. "Paternal transmission of stress-induced pathologies." *Biol. Psychiatry* 70(5): 408-414

Dietz, D. & Nestler, E. 2012. "From Father to Offspring: Paternal Transmission of Depressive-Like Behaviors." *Neuropsychopharmacol. Rev.* 37(1): 311-312

Dittrich, F., ter Maat, A., Jansen, R., Pieneman, A., Hertel, M., Frankl-Vilches, C. & Gahr, M. 2013. "Maximized song learning of juvenile male zebra finches following BDNF expression in the HVC." *Eur. J. Neurosci. 3*8(9): 3338-3344

Duan, W., Lee, J., Guo, Z. & Mattson, M. 2001. "Dietary restriction stimulates BDNF production in the brain and thereby protects neurons against excitotoxic injury." *J. Mol. Neurosci.* 16(1): 1-12

Duman, R. & Monteggia, L. 2006. "A Neurotrophic Model for Stress-Related Mood Disorders." *Biol. Psychiatry* 59(12): 1116-1127

Dunbar, K. 2001. "What scientific thinking reveals about the nature of cognition." In Crowley, K., Schunn, C. & Okada, T. (eds.), *Designing for science: Implications from everyday, classroom, and professional settings*, Mahwah, NJ: Lawrence Erlbaum Associates Publishers, pgs. 115-140

Eldakar, O. & Wilson, D. 2011. "Eight criticisms not to make about group selection." *Evolution* 65(6): 1523-1526

Emlen, D., Warren, I., Johns, A., Dworkin, I. & Lavine, L. 2012. "A Mechanism of Extreme Growth and Reliable Signaling in Sexually Selected Ornaments and Weapons." *Science* 337(6096): 860-864

Field, T. 1998. "Touch Therapy Effects on Development." *Int. J. Behav. Dev.* 22(4): 779-797

Field, T., Schanberg, S., Scafidi, F., Bauer, C., Vega-Lahr, N., Garcia, R., Nystrom, J. & Kuhn, C. 1986. "Tactile/Kinesthetic Stimulation Effects on Preterm Neonates." *Pediatrics* 77(5): 654-658

Folstad, I. & Karter, A. 1992. "Parasites, Bright Males, and the Immunocompetence Handicap." *Am. Nat.* 139(3): 603-622

Forstmeier, W. 2004. "Female resistance to male seduction in zebra finches." *Anim. Behav.* 68(5): 1005-1015

Franklin, T., Linder, N., Russig, H., Thony, B. & Mansuy, I. 2011. "Influence of early stress on social abilities and serotonergic functions across generations in mice." *Plos One* 6(7)

Franklin, T., Russig, H., Weiss, I., Graff, J., Linder, N., Michalon, A., Vizi, S. & Mansuy, I. 2010. "Epigenetic transmission of the impact of early stress across generations." *Biol. Psychiatry* 68(5): 408-415

Franklin, T., Saab, B. & Mansuy, I. 2012. "Neural mechanisms of stress resilience and vulnerability." *Neuron* 75(5): 747-761

Fu, D., Xiao, M., Hayward, A., Jiang, G., Zhu, L., Zhou, Q., Li, J. & Zhang, M. 2015. "What is crop heterosis: new insights into an old topic." *J. Appl. Genet.* 56(1): 1-13

Fuchikami, M., Yamamoto, S., Morinobu, S., Takei, S. & Yamawaki, S. 2010. "Epigenetic Regulation of BDNF Gene in Response to Stress." *Psychiatry Investig.* 7(4): 251-256

Furman, D., Hejblum, B., Simon, N., Jojic, V., Dekker, C., Thiebaut, R., Tibshirani, R. & Davis, M. 2014. "Systems analysis of sex differences reveals an immunosuppressive role for testosterone in the response to influenza vaccination." *Proc. Natl. Acad. Sci. U.S.A.* 111(2): 869-874

Gapp, K., Jawaid, A., Sarkies, P., Bohacek, J., Pelczar, P., Prados, J., Farinelli, L., Miska, E. & Mansuy, I. 2014. "Implication of sperm RNAs in transgenerational inheritance of the effects of early trauma in mice." *Nat. Neurosci.* 17(5): 667-669

Getty, T. 1998. "Reliable signalling need not be a handicap." *Anim. Behav.* 56(1): 253-255

Getty, T. 2006. "Sexually selected signals are not similar to sports handicaps." *Trends Ecol. Evol.* 21(2): 83-88

Gil, D. 2008. "Hormones in avian eggs: Physiology, ecology and behavior." In Brockmann, H., Roper, T., Naguib, M., Wynne-Edwards, K., Barnard, C. & Mitani, J. (eds.), *Advances in the study of behavior, Vol*ume 38. San Diego, CA: Academic Press, pgs. 337-398

Gould, S. & Lewontin, R. 1979. "The Spandrels of San Marco and the Panglossian Paradigm: A Critique of the Adaptationist Programme." *Proc. R. Soc. Lond. B.* 205(1161): 581-598

Gould, K., McKelvie, J. & Markham, K. 2002. "Do anthocyanins function as antioxidants in leaves? Imaging of H(2)O(2) in red and green leaves after mechanical injury." *Plant Cell Environ.* 25(10): 1261-1269

Gowaty, P. 2003. "Power asymmetries between the sexes, mate preferences, and components of fitness." In: Travis, C. (ed.), *Women, Evolution, and Rape.* Cambridge, MA: MIT Press, pgs. 61-86

Gowaty, P. 2008. "Reproductive compensation." *J. Evol. Biol.* 21(5): 1189-1200

Gowaty, P., Anderson, W., Bluhm, C., Drickamer, L., Kim, Y. & Moore, A. 2007. "The hypothesis of reproductive compensation and its assumptions about mate preferences and offspring viability." *Proc. Natl. Acad. Sci. U.S.A.* 104(38): 15023-15027

Groothuis, T., Muller, W., Von Engelhardt, N., Carere, C. & Eising, C. 2005. "Maternal hormones as a tool to adjust offspring phenotype in avian species." *Neurosci. Biobehav. Rev.* 29(2): 329-352

Groves, J. 2007. "Is it time to reassess the BDNF hypothesis of depression?" *Mol. Psychiatry* 12(12): 1079-1088

Habig, B. & Archie, E. 2015. "Social status, immune response and parasitism in males: a meta-analysis." *Philos. Trans. R. Soc. Biol.* 370(1669): 20140109

Hagen, E. 2003. "The Bargaining Model of Depression." In Hammerstein, P. (ed.), *Genetic and Cultural Evolution of Cooperation.* Cambridge, MA: The MIT Press, pgs. 97-123

Hager, R., Cheverud, J. & Wolf, J. 2009. "Change in maternal environment induced by cross-fostering alters genetic and epigenetic effects on complex traits in mice." *Proc. R. Soc. Lond. B.* 276(1669): 2949-2954

Hamilton, W. 1964. "The Genetical Evolution of Social Behaviour. I." *J. Theor. Biol.* 7(1): 1-16

Han, Z., Mtango, N., Patel, B., Sapienza, C. & Latham, K. 2008. "Hybrid vigor and transgenerational epigenetic effects on early mouse embryo phenotype." *Biol. Reprod.* 79(4): 638-648

Harris, W. & Uller, T. 2009. "Reproductive investment when mate quality varies: differential allocation versus reproductive compensation." *Philos. Trans. R. Soc. Biol.* 364(1520): 1039-1048

Harrison, X., York, J., Cram, D., Hares, M. & Young, A. 2013. "Complete reproductive skew within white-browed sparrow weaver groups despite outbreeding opportunities for subordinates of both sexes." *Behav. Ecol. Sociobiol.* 67(12): 1915-1929

Hasson, O. 1997. "Towards a General Theory of Biological Signaling." *J. Theor. Biol.* 185(2): 139-156

Hatier, J. & Gould, K. 2008. "Foliar anthocyanins as modulators of stress signals." *J. Theor. Biol.* 253(3): 625-627

Hattie, J. & Timperley, H. 2007. "The power of feedback." *Rev. Educ. Res.* 77(1): 81-112

Hawton, K. & van Heeringen, K. 2009. "Suicide." *Lancet* 373(9672): 1372-1381

Heiming, R., Bodden, C., Jansen, F., Lewejohann, L., Kaiser, S., Lesch, K., Palme, R. & Sachser, N. 2011. "Living in a dangerous world decreases maternal care: A study in serotonin transporter knockout mice." *Horm. Behav.* 60(4): 397-407

Hernandez, I., Alegre, L., Van Breusegem, F. & Munne-Bosch, S. 2009. "How relevant are flavonoids as antioxidants in plants?" *Trends Plant Sci.* 14(3): 125-132

Higham, J. 2014. "How does honest costly signaling work?" *Behav. Ecol.* 25(1): 8-11

Hill, G. 2006. "Environmental Regulation of Ornamental Coloration." In Hill, G. & McGraw, K. (eds.), *Bird Coloration, Volume 1*. Cambridge, MA: Harvard University Press, pgs. 507-560

Hill, G. 2011. "Condition-dependent traits as signals of the functionality of vital cellular processes." *Ecol. Lett.* 14(7): 625-634

Hill, G. 2014. "Cellular Respiration: The Nexus of Stress, Condition, and Ornamentation." *Integr. Comp. Biol.* 54(4): 645-657

Hill, G. & Johnson, J. 2012. "The Vitamin A-Redox Hypothesis: A Biochemical Basis for Honest Signaling via Carotenoid Pigmentation." *Am. Nat.* 180(5): E127-E150

Hill, G. & Johnson, J. 2013. "The mitonuclear compatibility hypothesis of sexual selection." *Proc. R. Soc. Lond. B.* 280(1768): 20131314

Husak, J. & Swallow, J. 2011. "Compensatory traits and the evolution of male ornaments." *Behaviour* 148(1): 1-29

Johnson, J. & Hill, G. 2013. "Is carotenoid ornamentation linked to the inner mitochondria membrane potential? A hypothesis for the maintenance of signal honesty." *Biochimie* 95(2): 436-444

Johnstone, R. 2000. "Models of reproductive skew: A review and synthesis." *Ethology* 106(1): 5-26

Jones, E. & Berglas, S. 1978. "Control of attributions about self through self-handicapping strategies-appeal of alcohol and role of underachievement." *Personal. Soc. Psychol. Bull.* 4(2): 200-206

Kankova, Z., Zeman, M. & Okuliarova, M. 2014. "Selection for high egg testosterone and immune response of young Japanese quail under mild food restriction." *Avian Biol. Res.* 7(1): 25-32

Kinney, D. & Tanaka, M. 2009. "An Evolutionary Hypothesis of Depression and Its Symptoms, Adaptive Value, and Risk Factors." *J. Nerv. Ment. Dis.* 197(8): 561-567

Klug, H. 2007a. "Evolutionary Significance of Filial Cannibalism in Fishes with Parental Care." Doctoral Dissertation (University of Florida)

Klug, H. 2007b. "When to care for, abandon, or eat your offspring: the evolution of parental care and filial cannibalism." *Am. Nat.* 170(6): 886-901

Klug, H. & St Mary, C. 2005. "Reproductive fitness consequences of filial cannibalism in the flagfish, Jordanella floridae." *Anim. Behav.* 70(3): 685-691

Kolditz, T. & Arkin, R. 1982. "An Impression Management Interpretation of the Self-Handicapping Strategy." *J. Personal. Soc. Psychol.* 43(3): 492-502

Kotiaho, J. 2001. "Costs of sexual traits: a mismatch between theoretical considerations and empirical evidence." *Biol. Rev.* 76(3): 765-776

Kovinich, N., Kayanja, G., Chanoca, A., Riedl, K., Otegui, M. & Grotewold, E. 2014. "Not all anthocyanins are born equal: distinct patterns induced by stress in Arabidopsis." *Planta* 240(5): 931-940

Kuhn, C., Butler, S. & Schanberg, S. 1978. "Selective Depression of Serum Growth Hormone During Maternal Deprivation in Rat Pups." *Science* 201(4360): 1034-1036

Kuo, T., Fedina, T., Hansen, I., Dreisewerd, K., Dierick, H., Yew, J. & Pletcher, S. 2012. "Insulin Signaling Mediates Sexual Attractiveness in Drosophila." *Plos Genetics* 8(4)

Lachlan, R. & Nowicki, S. 2012. "How Reliable Is Song Learning Accuracy as a Signal of Male Early Condition?" *Am. Nat.* 180(6): 751-761

Leary, M. & Shepperd, J. 1986. "Behavioral self-handicaps versus self-reported handicaps: A conceptual note." *J. Personal. Soc. Psychol.* 51(6): 1265-1268

Lee, K. 2006. "Linking immune defenses and life history at the levels of the individual and the species." *Integr. Comp. Biol.* 46(6): 1000-1015

Liu, D., Tannenbaum, B., Caldji, C., Francis, D., Freedman, A., Sharma, S., Pearson, D., Plotsky, P. & Meaney, M. 1997. "Maternal care, hippocampal glucocorticoid receptor gene expression and hypothalamic-pituitary-adrenal responses to stress." *Science* 277(5332): 1659-1662

Lupien, S., McEwen, B., Gunnar, M. & Heim, C. 2009. "Effects of stress throughout the lifespan on the brain, behaviour and cognition." *Nat. Rev. Neurosci.* 10(6): 434-445

Maccari, S., Darnaudery, M., Morley-Fletcher, S., Zuena, A., Cinque, C. & Van Reeth, O. 2003. "Prenatal stress and long-term consequences: implications of glucocorticoid hormones." *Neurosci. Biobehav. Rev.* 27(1-2): 119-127

MacDougall-Shackleton, S. & Spencer, K. 2012. "Developmental stress and birdsong: current evidence and future directions." *Journal fur Ornithologie* 153, Supplement 1: 105-117

Manetas, Y. 2006. "Why some leaves are anthocyanic and why most anthocyanic leaves are red?" *Flora* 201(3): 163-177

Mansuy, I., Mashoodh, R. & Champagne, F. 2013. "Transgenerational Inheritance in Mammals." In Sweatt, J., Meaney, M., Nestler, E. & Akbarian, S. (eds.), *Epigenetic Regulation in the Nervous System: Basic Mechanisms and Clinical Impact*. London: Academic Press, pgs. 323-338

Martin, L., Neighbors, H. & Griffith, D. 2013. "The experience of symptoms of depression in men vs women: analysis of the National Comorbidity Survey Replication." *JAMA Psychiatry* 70(10): 1100-1106

Martinez, M., Calvo-Torrent, A. & Pico-Alfonso, M. 1998. "Social defeat and subordination as models of social stress in laboratory rodents: a review." *Aggress. Behav.* 24(4): 241-256

Martinowich, K., Manji, H. & Lu, B. 2007. "New insights into BDNF function in depression and anxiety." *Nat. Neurosci.* 10(9): 1089-1093

Mashoodh, R., Sinal, C. & Perrot-Sinal, T. 2009. "Predation threat exerts specific effects on rat maternal behaviour and anxiety-related behaviour of male and female offspring." *Physiol. Behav.* 96(4-5): 693-702

Mashoodh, R., Franks, B., Curley, J. & Champagne, F. 2012. "Paternal social enrichment effects on maternal behavior and offspring growth." *Proc. Natl. Acad. Sci. U.S.A.* 109(Supplement 2): 17232-17238

Maynard Smith, J. & Harper, D. 1995. "Animal signals: models and terminology." *J. Theor. Biol.* 177(3): 305-311

Maynard Smith, J. & Harper, D. 2004. *Animal signals*. Oxford: Oxford University Press

McLeod, J., Sinal, C. & Perrot-Sinal, T. 2007. "Evidence for non-genomic transmission of ecological information via maternal behavior in female rats." *Genes Brain Behav.* 6(1): 19-29

Medawar, P. 1952. *An Unsolved Problem in Biology*. London: Lewis

Metzger, M., Villalba, A., Carballal, M., Iglesias, D., Sherry, J., Reinisch, C., Muttray, A., Baldwin, S. & Goff, S. 2016. "Widespread transmission of independent cancer lineages within multiple bivalve species." *Nature* (letter published online; DOI: 10.1038/nature18599)

Mykletun, A., Bjerkeset, O., Dewey, M., Prince, M., Overland, S. & Stewart, R. 2007. "Anxiety, depression, and cause-specific mortality: the HUNT study." *Psychosom. Med.* 69(4): 323-331

Navara, K., Badyaev, A., Mendonca, M. & Hill, G. 2006. "Yolk antioxidants vary with male attractiveness and female condition in the house finch (Carpodacus mexicanus)." *Physiol. Biochem. Zool.* 79(6): 1098-1105

Nestler, E. 2012. "Epigenetics: Stress makes its molecular mark." *Nature* 490(7419): 171-172

Nettle, D. 2004. "Evolutionary origins of depression: a review and reformulation." *J. Affect. Disord.* 81(2): 91-102

Nibuya, M., Morinobu, S. & Duman, R. 1995. "Regulation of BDNF and trkB mRNA in rat brain by chronic electroconvulsive seizure and antidepressant drug treatments." *J. Neurosci.* 15(11): 7539-7547

Nonacs, P. & Hager, R. 2011. "The past, present and future of reproductive skew theory and experiments." *Biol. Rev.* 86(2): 271-298

Norris, A., Cockle, K. & Martin, K. 2010. "Evidence for tolerance of parasitism in a tropical cavity-nesting bird, planalto woodcreeper (Dendrocolaptes platyrostris), in northern Argentina." *J. Trop. Ecol.* 26(6): 619-626

Nowak, M. 2012. "Evolving cooperation." *J. Theor. Biol.* 299(2012): 1-8

Nowicki, S. & Searcy, W. 2004. "Song function and the evolution of female preferences-Why birds sing, why brains matter." *Ann. N.Y. Acad. Sci.* (1016): 704-723

Pinker, S. 2012. "The False Allure of Group Selection." Edge. On the web: http://edge.org/conversation/the-false-allure-of-group-selection Last accessed: 7/16/16

Poiani, A. 2010. *Animal Homosexuality*. Cambridge: Cambridge University Press

Polya, G. 1957. *How to Solve It*. Garden City, NY: Doubleday

Preti, A. 2011. "Animal model and neurobiology of suicide." *Prog. Neuropsychopharmacol. Biol. Psychiatry* 35(4): 818-830

Putallaz, M., Costanzo, P., Grimes, C. & Sherman, D. 1998. "Intergenerational Continuities and Their Influences on Children's Social Development." *Soc. Dev.* 7(3): 389-427

Raison, C. & Miller, A. 2013. "The evolutionary significance of depression in Pathogen Host Defense." *Mol. Psychiatry* 18(1): 15-37

Ratikainen, I. & Kokko, H. 2010. "Differential allocation and compensation: who deserves the silver spoon?" *Behav. Ecol.* 21(1): 195-200

Rhainds, M. 2010. "Female mating failures in insects." *Entomol. Exp Appl.* 136(3): 211-226

Rhodewalt, F. 2008. "Self-handicapping: On the self-perpetuating nature of defensive behavior." *Soc. Personal. Psychol. Compass* (2/3): 1255-1268

Ritschard, M. & Brumm, H. 2012. "Zebra finch song reflects current food availability." *Evol. Ecol.* 26(4): 801-812

Ritschard, M., Riebel, K. & Brumm, H. 2010. "Female zebra finches prefer high-amplitude song." *Anim. Behav.* 79(4): 877-883

Roberts, M., Buchanan, K. & Evans, M. 2004. "Testing the immunocompetence handicap hypothesis: a review of the evidence." *Anim. Behav.* 68(2): 227-239

Russo, S., Murrough, J., Han, M., Charney, D. & Nestler, E. 2012. "Neurobiology of resilience." *Nature Neurosci.* 15(11): 1475-1484

Russo, S. & Nestler, E. 2013. "The brain reward circuitry in mood disorders." *Nat. Rev. Neurosci.* 14(9): 609-625

Safran, R., Pilz, K., McGraw, K., Correa, S. & Schwabl, H. 2008. "Are yolk androgens and carotenoids related to parental quality?" *Behav. Ecol. Sociol.* 62(3): 427-438

Saino, N., Bertacche, V., Ferrari, R., Martinelli, R., Moller, A. & Stradi, R. 2002. "Carotenoid concentration in barn swallow eggs is influenced by laying order, maternal infection and paternal ornamentation." *Proc. R. Soc. Lond. B.* 269(1501): 1729-1733

Saltzman, W., Digby, L. & Abbott, D. 2009. "Reproductive skew in female common marmosets: what can proximate mechanisms tell us about ultimate causes?" *Proc. R. Soc. Lond. B.* 276(1656): 389-399

Sapolsky, R. 1998. *Why Zebras Don't Get Ulcers*. New York: W.H. Freeman and Company

Sapolsky, R. 2001. "Physiological and pathophysiological implications of social stress in mammals." In: McEwen, B. (ed.), *Handbook of Physiology: Section 7, The Endocrine System; Volume IV: Coping With The Environment.* Oxford: Oxford University Press

Sa-Rocha, V., Sa-Rocha, L. & Palermo-Neto, J. 2006. "Variations in behavior, innate immunity and host resistance to B16F10 melanoma growth in mice that present social stable hierarchical ranks." *Physiol. Behav.* 88(1-2): 108-115

Schaefer, M. & Rolshausen, G. 2006. "Plants on red alert: do insects pay attention?" *BioEssays* 28(1): 65-71

Schanberg, S. 1995. "The genetic basis for touch effects." In Field, T. (ed.), *Touch in Early Development.* New York: Psychology Press

Schanberg, S. & Field, T. 1987. "Sensory Deprivation Stress and Supplemental Stimulation in the Rat Pup and Preterm Human Neonate." *Child Dev.* 58(6): 1431-1447

von Schantz, T., Bensch, S., Grahn, M., Hasselquist, D. & Wittzell, H. 1999. "Good genes, oxidative stress and condition-dependent sexual signals." *Proc. R. Soc. Lond. B.* 266(1414): 1-12

Schwinger, M., Wirthwein, L., Lemmer, G. & Steinmayr, R. 2014. "Academic self-handicapping and achievement: A meta-analysis." *J. Educ. Psychol.* 106(3): 744-761

Seligman, M. 1976. *Helplessness: On Depression, Development, and Death.* San Francisco: W.H. Freeman and Company

Semel, Y., Nissenbaum, J., Menda, N., Zinder, M., Krieger, U., Issman, N., Pleban, T., Lippman, Z., Gur, A. & Zamir, D. 2006. "Overdominant quantitative trait loci for yield and fitness in tomato." *Proc. Natl. Acad. Sci. U.S.A.* 103(35): 12981-12986

Sheldon, B. 2000. "Differential allocation: tests, mechanisms and implications." *Trends Ecol. Evol.* 15(10): 397-402

Sheldon, B. & West, S. 2004. "Maternal Dominance, Maternal Condition, and Offspring Sex Ratio in Ungulate Mammals." *Am. Nat.* 163(1): 40-54

Simons, M., Groothuis, T. & Verhulst, S. 2015. "An appraisal of how the vitamin A-redox hypothesis can maintain honesty of carotenoid-dependent signals." *Ecol. Evol.* 5(1): 224-228

Simons, M., Maia, R., Leenknegt, B. & Verhulst, S. 2014. "Carotenoid-Dependent Signals and the Evolution of Plasma Carotenoid Levels in Birds." *Am. Nat.* 184(6): 741-751

Smith, M., Makino, S., Kvetnansky, R. & Post, R. 1995. "Stress and glucocorticoids affect the expression of brain-derived neurotrophic factor and neurotrophin-3 mRNAs in the hippocampus." *J. Neurosci.* 15(3, part 1): 1768-1777

Simmons, L. 2005. "The Evolution of Polyandry: Sperm Competition, Sperm Selection, and Offspring Viability." *Annu. Rev. Ecol. Evol. Syst.* 36(1): 125-146

Slobodkin, L. 1957. "A laboratory study of the effect of removal of newborn animals from a population." *Proc. Natl. Acad. Sci. U.S.A.* 43(8): 780-782

Sowislo, J. & Orth, U. 2013. "Does low self-esteem predict depression and anxiety? A meta-analysis of longitudinal studies." *Psychol. Bull.* 139(1): 213-240

Spencer, K. & MacDougall-Shackleton, S. 2011. "Indicators of development as sexually selected traits: the developmental stress hypothesis in context." *Behav. Ecol.* 22(1): 1-9

Stibor, H. 1992. "Predator induced life-history shifts in a freshwater cladoceran." Oecologia 92(2): 162-165

Stiver, K. & Alonzo, S. 2009. "Parental and Mating Effort: Is There Necessarily a Trade-off?" *Ethology* 115(12): 1101-1126

Stockley, P. & Bro-Jorgensen, J. 2011. "Female competition and its evolutionary consequences in mammals." *Biol. Rev.* 86(2): 341-366

Sun, H., Kennedy, P. & Nestler, E. 2013. "Epigenetics of the Depressed Brain: Role of Histone Acetylation and Methylation." *Neuropsychopharmacol. Rev.* 38(1): 124-137

Suomi, S. 1995. "Touch and the immune system in rhesus monkeys." In Field, T. (ed.), *Touch in Early Development*. New York: Psychology Press

Suomi, J. 1997. "Early determinants of behavior: evidence from primate studies." *Br. Med. Bull.* 53(1): 170-184

Svensson, P. & Wong, B. 2011. "Carotenoid-based signals in behavioural ecology: a review." *Behaviour* 148(2): 131-189

Szamado, S. 2011. "The cost of honesty and the fallacy of the handicap principle." *Anim. Behav.* 81(1): 3-10

Trivers, R. 1971. "The Evolution of Reciprocal Altruism." *Q. Rev. Biol.* 36(1): 35-57

Trivers, R. 1972. "Parental investment and sexual selection." In Campbell, B. (ed.), *Sexual selection and the descent of man.* Chicago: Aldine

Trivers, R. & Willard, D. 1973. "Natural selection of parental ability to vary the sex ratio of offspring." *Science* 179(4068): 90-92

Urdan, T. & Midgley, C. 2001. "Academic self-handicapping: What we know, what more there is to learn." *Educ. Psychol. Rev.* 13(2): 115-138

Varga, S. 2012. "Evolutionary psychiatry and depression: testing two hypotheses." *Med. Health Care Philos.* 15(1): 41-52

Vialou, V., Feng, J., Robison, A. & Nestler, E. 2013. "Epigenetic Mechanisms of Depression and Antidepressant Action." *Annu. Rev. Pharmacol. Toxicol.* (53): 59-87

Voigt, C., Leitner, S. & Gahr, M. 2007. "Socially induced brain differentiation in a cooperatively breeding songbird." *Proc. R. Soc. Lond. B.* 274(1626): 2645-2652

Walsh, J., Friedman, A., Sun, H., Heller, E., Ku, S., Juarez, B., Burnham, V., Mazei-Robison, M., Ferguson, D., Golden, S., Koo, J., Chaudhury, D., Christoffel, D., Pomeranz, L., Friedman, J., Russo, S., Nestler, E. & Han, M. 2014. "Stress gates neural activation of BDNF in the mesolimbic reward pathway." *Nature Neurosci.* 17(1): 27-29

Ward, I. 1972. "Prenatal stress feminizes and demasculinizes the behavior of males." *Science* 175(4017): 82

Warren, I., Gotoh, H., Dworkin, I., Emlen, D. & Lavine, L. 2013. "A general mechanism for conditional expression of exaggerated sexually-selected traits." *BioEssays* 35(10): 889-899

Watson, P. & Andrews, P. 2002. "Toward a revised evolutionary adaptationist analysis of depression: the social navigation hypothesis." *J. Affect. Disord.* 71(1): 1-14

Weiss, J., Bailey, W., Goodman, P., Hoffman, L., Ambrose, M., Salman, S. & Charry, J. 1982. "A model for neurochemical study of depression." In Spiegelstein, M. & Levy, A. (eds.), *Behavioral Models and the Analysis of Drug Action*. Amsterdam: Elsevier Scientific Publishing Company.

Weiss, I., Franklin, T., Vizi, S. & Mansuy, I. 2011. "Inheritable effect of unpredictable maternal separation on behavioral responses in mice." *Front. Behav. Neurosci.* 5(3)

Weinstock, M. 2005. "The potential influence of maternal stress hormones on development and mental health of the offspring." *Brain Behav. Immun.* 19(4): 296-308

Weinstock, M. 2010. "Intrauterine factors as determinants of depressive disorder." *Isr. J. Psychiatry Relat. Sci.* 47(1): 36-45

Williams, G. 1966. *Adaptation and Natural Selection*. Princeton: Princeton University Press.

Wilson, D. 1983. "The Group Selection Controversy-History and Current Status." *Annu. Rev.Ecol. Syst.* 14(1): 159-187

Wilson, D. & Wilson, E. 2007. "Rethinking the theoretical foundation of sociobiology." *Quart. Rev. Biol.* 82(4): 327-348

Wilson, E. 2014. *The Meaning of Human Existence*. New York: Liveright Publishing Corporation.

Wolkowitz, O., Wolf, J., Shelly, W., Rosser, R., Burke, H., Lerner, G., Reus, V., Nelson, J., Epel, E. & Mellon, S. 2011. "Serum BDNF levels before treatment predict SSRI response in depression." *Prog. Neuro-Psychopharmacol. Biol. Psychiatry* 35(7): 1623-1630

Woodgate, J., Bennett, A., Leitner, S., Catchpole, C. & Buchanan, K. 2010. "Developmental stress and female mate choice behavior in the zebra finch." *Anim. Behav.* 79(6): 1381-1390

Woodgate, J., Leitner, S., Catchpole, C., Berg, M., Bennett, A. & Buchanan, K. 2011. "Developmental stressors that impair song learning in males do not appear to affect female preferences for song complexity in the zebra finch." *Behav. Ecol.* 22(3): 566-573

Young, A. & Bennett, N. 2013. "Intra-sexual selection in cooperative mammals and birds: why are females not bigger and better armed?" *Philos. Trans. R. Soc. Biol.* 368(1631): 20130075

Zahavi, A. 1975. "Mate Selection—A Selection for a Handicap." *J. Theor. Biol.* 53(1): 205-214

Zahavi, A. & Zahavi, A. 1997. *The Handicap Principle: A Missing Piece of Darwin's Puzzle*. Oxford: Oxford University Press

Zhang, T. & Meaney, M. 2010. "Epigenetics and the Environmental Regulation of the Genome and Its Function." *Annu. Rev. Psychol.* (61): 439-466

Zollinger, S., Goller, F. & Brumm, H. 2011. "Metabolic and respiratory costs of increasing song amplitude in zebra finches." *Plos One* 6(9)

Zuckerman, M., Kieffer, S. & Knee, C. 1998. "Consequences of self-handicapping: Effects on coping, academic performance, and adjustment." *J. Personal. Soc. Psychol.* 74(6): 1619-1628

Zuckerman, M. & Tsai, F. 2005. "Costs of self-handicapping." *J. Personal.* 73(2): 411-442

57