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## Environmental Risk

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## Synonyms

[Environmental adversities](#); [Environmental threats and challenges](#)

## Definition

Within the life history theoretical framework, environmental risks refer to depleting or low levels of resource and high rates of extrinsic threats, such as predation, famine, disease, and intraspecific violence, that are insensitive to the survival effort of the individuals and that cause mortality and morbidity of the population, as well as stochastic variations of the resource levels and mortality–morbidity rates.

## Introduction

Hence, as more individuals are produced than can possibly survive, there must in every case be a struggle for existence, either one individual with another of the same species, or with the individuals

of distinct species, or with the physical conditions of life. (Darwin 1859/1979, p. 117)

In the chapter entitled “Struggle for Existence” of *The Origin of Species*, Darwin points out a critical assumption about evolution – that there will always be more lives than there are resources to support them. This results in constant intra- and interspecies competition in which only the fittest survive. The competition or struggle for existence occurs at the individual or organism level and, fundamentally, at the genetic level. However, advancements in knowledge since Darwin’s seminal work was published have shown further evidence that an individual’s life is composed of characteristics called life history (LH) traits. Limited resources and resource competition manifest as a limited energy budget that cannot support all LH traits, and the strategic allocation of limited energy results in trade-offs between different LH traits. Similarities and patterns derived from LH trade-offs form LH strategies, which are aimed at optimizing the struggle for existence at the individual level. One such broad pattern of LH trade-offs is the fast–slow LH strategic continuum (Promislow and Harvey 1990). A fast LH strategy involves trade-offs characterized by early maturation and reproduction, squandering rather than conserving energy and resources, and high mating and low parenting. By contrast, a slow LH strategy involves late maturation, delayed reproduction, slow development, and a longer lifespan, all of which facilitate amassing and conserving

resources, learning and developing skills, and parenting and training fewer, high-quality offspring who are equipped for intra- and interspecies competition in the struggle for existence. The fast–slow continuum and other intraindividual strategic LH trade-offs respond to and are shaped by the struggle for existence at the individual level. This chapter addresses the relationship between the environmental risks or constraints that make individuals' lives a struggle and the intraindividual LH strategic trade-offs individuals employ to optimize the conditions of the struggle for existence.

## Environmental Harshness

What is essential for life and subsequently responsible for the struggle for existence? The answer must be food and safety, the levels and variations of which also shape LH trade-off strategies. For most species and for humans of the evolutionary past, the main safety concern is predatory risk, which is the main cause of mortality. Other extrinsic causes of mortality include famine, disease, and conspecific violence. Because these causes may also lead to injury, illness, and disability, in addition to death, Ellis et al. (2009) coined the term “mortality–morbidity” to encompass all of these negative consequences of such extrinsic risks as predation and disease. Food, or more generally, resources are essential for life. Their shortage and scarcity is responsible for the struggle for existence. In the evolution literature, resource scarcity and mortality–morbidity are defined as constituting the harshness dimension of the environment that regulates fast–slow LH strategies (Ellis et al. 2009).

## Resource Scarcity

Resources were first studied as a density-dependent selection condition in relation to population density and competition (MacArthur and Wilson 1967). When resources are sufficient to carry or support organisms living in a habitat, organisms tend to freely exploit them for fast development and early reproduction, producing high numbers of offspring who receive little

parental investment in teaching and learning about survival because they can survive and thrive in resource-rich and competition-free environments (Pianka 1970). This is known as *r* selection, where *r* represents the maximal reproductive rate of a species (MacArthur and Wilson 1967). When the high reproductive rate increases the population density to the point that resources become depleted, species adopt slow strategies, also known as *K* selection, where *K* represents the carrying capacity of the environment. Slow LH strategies trade fast development and mating for slow development and parenting; they raise few, high-quality offspring who can compete for and monopolize the depleting resources of their environment (Pianka 1970). The *K*-slow strategy is particularly relevant to humans who rely on skills and knowledge to monopolize resources in contest competition, where resources are unevenly distributed to *K* strategists; this is in contrast to scramble competition, where resources are evenly distributed, leading to fast LH strategies (Rogers 1992). Cross-cultural data on preindustrial societies suggest that resource scarcity arising from high population densities and related energy limitations favor the development of slower LH strategies involving paternal presence and comparatively high biparental care of offspring (Hewlett 1992; Katz and Konner 1981).

Rushton (1985) was among the first to apply the density-dependent *r*–*K* framework to the study of human LH strategies. However, research on resource scarcity as a type of environmental harshness has not flourished partly because other factors, such as age-specific mortality and environmental unpredictability (discussed in the following section), play more important roles than resources alone in shaping LH traits (Ellis et al. 2009). Another reason is that there is no reliable measure for resource availability or scarcity at the individual level. In modern life, the putatively representative indicator of available resources is family income or socioeconomic status (SES) at the individual level and per capita income at the population level. However, SES is an inadequate indicator of the evolutionary concept of resource availability or scarcity for several reasons. It has the floor effect in that even a low

SES in resource-rich, developed societies does not approach the resource depletion point below the environment's carrying capacity. It is also potentially confounded with two other LH concepts. First, in most metropolitan areas, a low SES is associated with an environment that is high in crime and violence, which represents a separate mortality–morbidity component of environmental harshness and unpredictability. Because of this potentially confounding measurement, low rather than high SES has been shown to be associated with fast LH strategies (e.g., Belsky et al. 1991) against LH predictions. Second, it is potentially confounded with intraspecific competition, with higher SESs representing success in intraspecific competition. The opposite effects of competitiveness and resource abundance on LH strategies nullify the potential effect of SES.

### **Mortality–Morbidity**

Whereas resource levels seem to influence LH strategies uniformly across the lifespan, the effect of mortality–morbidity on LH is age and stage dependent (Ellis et al. 2009): “Indeed, extrinsic morbidity–mortality is not defined by the source of death or disability but rather by which members of the population are affected” (p. 219). In general, if extrinsic causes of mortality–morbidity are insensitive to the survival efforts of the able adults of a population, high frequencies of such extrinsic risks lead to fast LH strategies because natural selection favors accelerated development and reproduction before extrinsic mortality–morbidity strikes (Promislow and Harvey 1990). If such mortality–morbidity as that derived from endemic disease exposures mainly affects the young and weak of the population, natural selection tends to select slow LH strategies because LH trade-offs that favor body repair and maintenance, including immune competence over mating, should enable individuals to escape disease attacks (Ellis et al. 2009). In species where juveniles suffer from high rates of mortality–morbidity, the affected fast–slow LH strategies depend on whether mortality–morbidity responds to parental investment, as well as juvenile investment (Ellis et al. 2009). If parental investment reduces extrinsic risk for the juveniles, natural selection should

favor slow LH strategies by increasing physical development to build stronger disease or predatory defense mechanisms. If mortality–morbidity is insensitive to parental investment, natural selection should favor fast LH to accelerate growth and development to outlive the age-specific extrinsic risks.

Low levels of extrinsic mortality–morbidity, however, do not necessarily lead to slow LH strategies (Ellis et al. 2009). Under the conditions of low environmental harshness that characterize human evolution, density-dependent factors, such as resource and intraspecies competition, become more relevant determinants of LH strategies (Pianka 1970). Low mortality–morbidity combined with low resource availability and high competition should lead to slow LH strategies, whereas low extrinsic risks accompanied by high resource availability and low competition should lead to fast LH strategies. However, few human LH studies have examined resource levels or competition. Conducted primarily in the West, extant studies have examined exposure to violence, crime, and antisocial activities (Brumbach et al. 2009) and rundown neighborhood conditions (Crowder and Teachman 2004), including the presence of gangs, abandoned cars, and graffiti (Upchurch et al. 1999), as indicators or proxies of environmental harshness. These proxies generally correlate with fast LH strategies.

### **Environmental Unpredictability**

Stochastic spatiotemporal variations in resource levels and mortality rates are defined as environmental unpredictability, which shapes fast–slow LH strategies (Ellis et al. 2009). These random fluctuations between good times and bad times are caused by such uncontrollable factors as predatory behavioral idiosyncrasy, weather change, or pathogenic breakout and disappearance. Unpredictable environmental fluctuations increase the variance and decrease the mean of fitness (Phillippi and Seger 1989). Because the geometric mean is negatively affected by the variance, reducing the variance increases the geometric mean despite it also reducing the arithmetic

mean. In fluctuating environments where fitness is calculated with the geometric rather than the arithmetic mean, phenotypes with reduced variance are favored over those of higher variance and higher mean fitness (Phillipi and Seger 1989). This trade-off between having a more variable number of offspring across time (with a higher arithmetic mean) and having a more constant number of offspring over time (with a higher geometric mean) is called “bet-hedging” (Phillipi and Seger 1989), for which there are two types: conservative and diversified bet-hedging. In conservative bet-hedging, animals reduce the variance of fitness across time and on specific occasions by decreasing the number of offspring in good times or in stable environments so that the number of offspring is closer to the mean. In diversified bet-hedging, animals reduce the fitness variance across time and space by increasing the number of offspring during bad times or in unpredictable environments. The two types of bet-hedging achieve the same end result, which is reduced variance of fitness across the lifespan or across generations (Phillipi and Seger 1989). Because conservative bet-hedging trades off offspring quantity for quality, it can be regarded as a slow LH strategy. Diversified bet-hedging, which increases offspring quantity at the cost of quality, can be considered a fast LH strategy.

According to Ellis et al. (2009), short-term environmental unpredictability (e.g., within generations) that causes variations in juvenile mortality predicts diversified bet-hedging to reduce fitness variance. Fitness variance is reduced by producing more offspring, reproducing with different partners, or extending the age schedule of reproduction, all three of which potentially diversify offspring phenotypes so that some offspring may survive environmental adversity. When variations in juvenile mortality are due to long-term environmental fluctuations across generations, natural selection favors conservative bet-hedging. Fitness variance is reduced by reducing the quantity and improving the quality of offspring so that, despite the overall number of offspring being lower than what would be optimal in good times or stable environments, the improved quality promotes offspring survival

across the range of environmental fluctuations from good times to bad times. When environmental unpredictability affects adult mortality and morbidity, natural selection favors fast LH strategies: “Both high absolute levels of adult mortality (harshness) and high variation in adult mortality (unpredictability), therefore, select for fast LH strategies” (Ellis et al. 2009, p. 229).

Central to the discussion of environmental unpredictability is the age of a population in which large variations of mortality–morbidity occur. Age-specific mortality–morbidity implies different effects or extents of an effect depending on the age or developmental stage of individuals when they experience environmental unpredictability. A robust and consistent finding from numerous human LH studies is that early childhood experiences of unpredictable environments lower the age of sexual maturation and increase the frequency of sexual activity. In such studies, early environmental unpredictability was represented by microenvironmental proxies from the childhood or early childhood environment. These include family SES (e.g., Belsky et al. 1991), residential mobility (e.g., Crowder and Teachman 2004), family and parental change (e.g., Belsky et al. 2010), disruptive and coercive family relationships (Byrd-Craven et al. 2007), maternal history of psychopathology (e.g., Ellis and Garber 2000), divorce (Mendle et al. 2006), paternal absence (e.g., Belsky et al. 1991), stepfather presence (Ellis and Garber 2000) and the presence of stranger males in the family (Ellis et al. 2003).

The results support the LH prediction that these indicators of environmental unpredictability are either directly or indirectly related to fast LH characteristics such as early menarche (e.g., Belsky et al. 1991), early commencement of sexual activity (e.g., Byrd-Craven et al. 2007), high frequency of sexual activity (e.g., Belsky et al. 2010), teen pregnancy (e.g., Ellis et al. 2003), sexual risk taking (Belsky et al. 2010), and early age at first birth (Nettle et al. 2011). The effect of paternal absence on menarche seems to be most evident when fathers leave their daughters before the age of 7 years (Belsky et al. 1991; Ellis et al. 2003). For

example, women whose parents separated before they were 6 years old were shown to mature earlier than girls whose parents did not separate (Quinlan 2003), and exposure to family conflicts assessed when the daughters were 7 years old was shown to correlate negatively with self-reported menarcheal age (Moffitt et al. 1992). The effect also seems to be quantitatively accumulating, as indicated by several studies that have found that both the length of paternal absence and the amount of time that girls are exposed to unrelated adult males at home correlate with the timing of menarche (e.g., Moffitt et al. 1992).

Moreover, childhood experiences of both environmental harshness and unpredictability have the LH-predicted lasting effect on adult behavior. Self-reports of early childhood experiences of environmental harshness and unpredictability were shown to be related to the anxious style of attachment (Barbaro and Shackelford 2016), which, compared with the secure style of attachment, represents a fast LH strategy. A study across neighborhoods in England showed that adult women who grew up in low-SES neighborhoods gave birth to their first child much earlier than women who grew up in high-SES neighborhoods (Nettle et al. 2011). The same finding was reported in an earlier study (Wilson and Daly 1997); women who grew up in neighborhoods where life expectancies were shorter (as a result of extrinsic risks such as homicide) were shown to give birth at a younger median age than those who grew up in neighborhoods where life expectancies were longer.

In numerous studies on adult populations, experimentally induced mortality–morbidity has not been shown to have the intended main effect, but interacted with childhood harshness and unpredictability in predicting LH strategic outcomes. In such studies, participants have been asked about their childhood SES and then experimentally primed with stimuli suggesting threats of mortality–morbidity (e.g., news stories about rising homicide rates and future uncertainty). Among people reporting low-SES childhoods, induced mortality was associated with the desire to reproduce early and have more children, even at the cost of delaying one’s education and career

development; by contrast, these fast LH effects were shown to be absent in people who reported having grown up under favorable resource conditions (Griskevicius et al. 2011). Induced mortality threats have also prompted participants with low-SES childhoods to opt for diversified bet-hedging by choosing different and riskier stocks over safer and less diversified options (White et al. 2013). Participants with low-SES childhoods have responded to induced environmental harshness with lowered resistance to temptation, high impulsivity, increased risk-taking, and shorter time orientation (e.g., spending more now and saving less for the future), whereas no priming effect has been observed for participants with high-SES childhoods (Griskevicius et al. 2013; Hill et al. 2014). Perceptions of personal control mitigate the psychological effects of resource scarcity threats only for people reporting high-SES childhoods (Mittal and Griskevicius 2014; Griskevicius et al. 2013). Collectively, this stream of research underscores the importance of early life experience in affecting human LH strategic changes.

## Conclusion

Thus, the struggle for existence that Darwin described 150 years ago is currently understood to be powered by strategic allocations of the limited energy budget through LH trade-offs. Environmental cues keep active the contingent response between the interindividual struggle for existence and the intraindividual LH trade-offs. If there were unlimited resources and no obstacles to acquiring them, there would be no struggle for existence or need for the corresponding strategic LH trade-offs. Therefore, environmental cues relevant to LH trade-offs are all negative. They are referred to as environmental risks, which are further divided into environmental harshness and environmental unpredictability. Harshness refers to any physical or psychological strain on an individual organism. Possible contributors include resource scarcity, predatory risk, pathogens and disease, natural disasters, and interspecies violence – all of which are independent of an

individual's effort for survival and cause mortality and morbidity. Among the elements of environmental harshness, probably most relevant to the survival of most nonhuman animals are levels of resources and predation, which can also be inter-related because one individual's food may be another's predation. Because humans are the highest predators who have long had no rivals other than themselves, resources and intraspecific competition to monopolize resources – both of which are density dependent – become the major strain on survival and cause the struggle for existence. For both human and nonhuman animals, density-dependent resource limitations and intraspecific competition drive slow LH strategies by which individuals invest in growth and development and in their children's growth and development so that they become capable competitors for the limited resources. Other harshness elements causing extrinsic mortality–morbidity lead to fast LH strategies because fitness is more efficiently attained in a harsh environment by accelerating development and moving to early reproduction before extrinsic mortality or morbidity strikes.

Published studies based on human populations generally support this theorizing about the role of environmental harshness in regulating human LH trade-off strategies. However, extant human LH studies have generally not pursued a density-dependent approach (Ellis et al. 2009), nor have they examined intraspecific competition, particularly in predicting human LH strategies. The ever-increasing and complex social groups of human evolution render most selection pressures density dependent. Resource scarcity and abundance are density dependent, as are mortality and morbidity arising from such extrinsic risks as diseases, intraspecific violence, and even natural disasters; the effects of all of these factors can be converted into winning versus losing in intraspecific competition. Competition that involves garnering resources and avoiding mortality–morbidity risks therefore captures both spectra of the harshness dimension of the environment.

Humans had in some unique fashion become so ecologically dominant that they in effect became their own principal hostile force of nature [and] nothing would select more potently... than a

within-species co-evolutionary arms race in which success depended on effectiveness in social competition. (Alexander 1990, pp. 4–7)

One direction for future research is to operationalize and investigate intraspecific competition at both the individual and population levels to determine how it affects LH strategies directly and mediates the relations between environmental harshness and individuals' behaviors and LH strategies.

Whereas environmental harshness refers to the absolute levels of morbidity and mortality, environmental unpredictability refers to the random or stochastic variations of morbidity and mortality. Stochastic variations rather than high frequencies of extrinsic risks are probably more relevant to human evolution because it presents a more formidable task in mastering these two hostile forces of nature. LH theory distinguishes between adult and juvenile populations that will be affected by unpredictable mortality and morbidity. When the adult population is the target of unpredictable mortality–morbidity, species adopt a fast LH strategy to allocate energy and resources for fast development and reproduction if there is adequate bioenergy, as determined by the harshness dimension of environmental risk. When juveniles are the target of mortality, species respond either with conservative bet-hedging (a type of slow LH strategy) or diversified bet-hedging (a type of fast LH strategy), depending on whether juvenile mortality and morbidity respond to parental investment. Published human LH studies do not distinguish between these two types of population mortality because they are highly correlated (Ellis et al. 2009) and because, with high parenting and child care, the child–adult mortality differential never reaches a scale such as those of many other animals (e.g., turtles) that require different LH strategies to respond to high juvenile mortality. However, it is also shown that childhood experiences of both harshness and unpredictability have a lasting effect on adult LH strategies, whereas – at least for some experimental studies – induced harshness and unpredictability of adulthood do not have the expected main effect on behavior. These findings

suggest the need for further considerations when applying LH theory to humans as a species-general theoretical framework.

One consideration is the distinction between uncontrollability and unpredictability (Brumbach et al. 2009). Uncontrollability refers to the complete separation between an event's happening and an individual's effort to influence the event, whereas unpredictability includes the additional inability to anticipate and avoid the event. Because LH theory is species general, many mortality and morbidity risks that are defined as unpredictable for animals in general may be uncontrollable but predictable for humans because of the large human brain. The fact that hominin populations (e.g., *Homo erectus*) migrated from Africa as early as 2 million years ago to populate the rest of the world suggests that at least some of the uncontrollable events driving intensive human evolution in the Pleistocene must often have been predictable, because the primary reasons for migration are to purposefully seek resources and avoid risks. In fact, famine, disease, and intraspecific conflict and violence, which are all defined as unpredictable by LH theory, may be uncontrollable but predictable because ancestral humans can anticipate their coming and make adjustments to avoid them; this includes migrating from Africa multiple times during human evolution. The evolution of the large human brain, which is itself a slow LH trait, results from environmental variations (e.g., glacial and interglacial temperature variations had a greater role than the mean cooling of the ice age in effecting brain enlargement; Ash and Gallup 2007) that are certainly uncontrollable but may be predictable because they would otherwise not have led to the large human brain as a slow LH trait. It is therefore possible that many of the environmental unpredictabilities defined by LH theory are predictable to humans who respond not with fast but with slow LH trade-off strategies or with a mixture of both. An apt example of a mixture of the two strategies is the human practice of weaning offspring early and shortening interbirth intervals (fast strategies), which are accompanied by feeding offspring with prepared foods and extensive child care by kin through alloparenting and

siblings helping at the nest (slow strategies). Future human LH studies can hypothesize mixed LH strategies, especially in response to uncontrollable but predictable environmental risks.

## Cross-References

- ▶ [Environmental Harshness](#)
- ▶ [Environmental Harshness/Mortality](#)
- ▶ [Environmental Unpredictability](#)
- ▶ [Environmental Unpredictability and Bet-Hedging](#)
- ▶ [Environmental Unpredictability and Brain Complexity](#)
- ▶ [Harsh Environments](#)

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