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Age, growth and size interact with stress to determine life span and mortality

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ABSTRACT

Individuals in a large experimental field population, of the short-lived perennial species *Plantago lanceolata*, were followed to determine the sources of variation that influence mortality and life span. The design included multiple age groups with initially similar genetic structure, which made it possible to separate age effects from period effects and to identify the genetic component to variation in life span. During a period of stress, individuals of all ages showed parallel increases in mortality but different cohorts experienced this period of high mortality at different ages. This then influenced the distribution of life spans across cohorts. Age and size-age interactions influenced mortality during the period of stress. Smaller individuals died but only if they were old. Additionally, growth and age interacted with stress such that older individuals had negative growth and high mortality whereas younger individuals had positive growth and relatively lower mortality during stress. The results of this study show that it is not simply the environment that can have a major impact on demography in natural populations; rather, age, size and growth can interact with the environment to influence mortality and life span when the environment is stressful.

1. Introduction

We know very little about the causes of variation in mortality and life span in natural populations. For a long time, the general hypothesis has been that life in the wild is short, and hard, and that aging in the wild has little impact on demographic patterns because few survive to old age (Comfort, 1964; Hayflick, 2000; Medawar, 1952). In most experimental laboratory studies of senescence, a single cohort is followed over the course of its life span and environmental influences on mortality are deliberately minimized to focus on intrinsic aspects of mortality. In natural populations, mortality is influenced by environmental processes that fluctuate seasonally or even daily (c.f. Menges and Quintana-Ascencio, 2004; Pico and Retana, 2008; Roach, 2003; Vavrek et al., 1997). These age- and state-independent environmental factors can have major impacts on mortality that may obscure the intrinsic causes of variation in mortality patterns. Changes in external forces that result in shifts in mortality must be distinguished from state-dependent changes in mortality. Moreover, in a situation termed condition-dependent aging, recent studies have shown that extrinsic and intrinsic factors can interact with each other to change the mortality risk for an individual (Reznick et al., 2004; Roach et al., 2009; Williams et al., 2006). It is important that the components of variation in mortality be understood because this variation influences the distribution of life spans and the patterns of mortality both within and among populations.

In addition to variable environmental effects, there are a number of experimental factors that limit our understanding of mortality and life span in natural populations. High levels of environmental mortality can complicate the detection of senescence in natural populations because it decreases population size. Experimentally, even in laboratory studies, it has only been with very large sample sizes that good mortality estimates of the latest ages have been obtained (cf. Carey et al., 1992; Curtsinger et al., 1992). Most studies of mortality have been done in an environment less harsh than that in which the organism's life history evolved (Charlesworth and Partridge, 1997). Finally, we know very little about the genetic basis of mortality and life span in natural populations. Some animal studies have used pedigree analysis to determine the quantitative genetics of reproductive aging in natural populations of animals (for example Charmantier et al., 2006; Wilson et al., 2007). This work has been limited, and more studies using quantitative genetic approaches are needed to explore variation in patterns of aging in natural populations (Wilson et al., 2008).

Senescence in wild animal populations has now been found in a wide range of taxa including ungulates (Loison et al., 1999), grey seals (Bowen et al., 2006), antler-flies (Bonduriansky and Brassil, 2002) and guppies (Reznick et al., 2004). The range of life spans for plant species is larger than for animals, but we only have limited empirical data on their age-specific demography and the determinants of life span (Ehrlén and Lehtilä, 2002; García et al., 2008; Roach, 2003; Silvertown et al., 2001). There are many advantages of using plants to study the demography of aging including the fact that large numbers of individuals can be marked and followed longitudinally for their entire life. Additionally, individuals

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can be followed in their natural habitat and age-specific reproduction, growth and other physiological measures can be quantified with no impact on the individual.

This study investigates how stress interacts with age to determine lifespan and mortality. It makes use of the appearance of an unplanned extrinsic 3-year stress period during an 11-year study of demographic rates in four experimentally created cohorts in the natural environment of the species *Plantago lanceolata*. Previous analysis of this experiment had shown that during high extrinsic mortality there was condition-dependent aging such that older individuals had higher mortality (Roach et al., 2009). The question addressed here is: What determined this age-dependent response to these stressful conditions?

2. Materials and methods

2.1. Experimental protocol

Plantago lanceolata (ribwort plantain) is a short-lived perennial species with a basal rosette. It is widely distributed throughout the world in mown fields and along roadsides. In our field site, located at the Shadwell Preserve of the Jefferson Monticello Foundation in Virginia, USA, it germinates in both fall and spring and remains green all year; thus individuals can be easily followed and an accurate assessment of mortality can be made in every month of the year. A large population of *Plantago* naturally exists at this site and to create seed for this study we isolated random parental genotypes from throughout the 70 m × 35 m research area and performed crosses in the greenhouse (see Roach et al. (2009) for details).

For the genetic analysis, seeds were created for the experiment from a crossing design using parental genotypes isolated from the experimental field site. Individuals were designated to be either sires or dams for the crossing design which was a modified North Carolina II design (Lynch and Walsh, 1998). Distinct 'sets' of sires and dams were used to produce 50-200 seedlings for 80 sire-dam combinations. The crosses included five sets of 'large crosses' where a large cross consisted of four sires crossed to each of two dams resulting in eight sire-dam combinations and 200 offspring from each. This was repeated for five unique sets of sires and dams, for a total of 20 sire genotypes, 10 dam genotypes, 40 sire-dam combinations and 8000 individual offspring. A second set of 'small crosses' were made consisting of two sires to each of two dams with 50 offspring produced from each of those four resulting crosses. Ten unique sets of sires and dams were used in the small crosses, adding 20 sire genotypes, 20 dam genotypes, 40 sire-dam pairs, and 2000 individuals to the experiment. This complete design was used for cohorts 1 and 2 and one-half of the total number of individuals per cross was used for cohorts 3 and 4. No parental genotypes were used in more than one set of crosses and the same parental genotypes were used for all age groups. Most seeds were produced in the initial round of crosses and the seed was stored dry in envelopes until planting. When necessary, the parental genotypes maintained in the greenhouse were crossed again, to attain the required seeds per cross.

Four age groups (cohorts) were planted over 3 years to facilitate separation of age- and environmental-effects. The initial genetic composition of each group was equivalent at the time of planting to minimize genetic differences across ages. We used the same protocol described in Roach (2003) for raising seedlings, planting, and marking individuals. We planted individuals with minimal disturbance to the surrounding community and in a randomized block design with replication of genotypes and different age groups within blocks. Individuals that died within one month of planting, due to transplant shock, were deleted from the study. The planting dates for the different aged groups were as follows: October 2000 (cohort 1); October 2001 (cohort 2); April 2002 (cohort 3); October 2002 (cohort 4).

Mortality and size were measured from November 2000–2011. Mortality was censused monthly and quantified as $q(x)$, the number of deaths between ages x and $x+1$, divided by the number of individuals alive at age x. Data from the monthly census is presented as survival. Q(x) during the period of stress was calculated for each age group as the number of deaths between May 2003 and November 2006 divided by the number of individuals alive at the onset of the stress period. Semi-annually (every May and November), we counted the number of leaves on each individual as a metric of size; we defined growth as the change in size per 6-month period. Details of this field protocol are described by Roach et al. (2009).

2.2. Statistical analysis

The genetic analysis of life span, size and growth was modeled as a function of crossing set, spatial block, sire (set), dam (set) and sire-by-dam interaction (set), with 'set' as a fixed-effect factor and the others as random effects. Proc GLM (SAS v. 9.1) was used to generate significance tests and independent contrasts for each factor, and the significance tests from the Sheffé model for the random-effect factors are reported (Fry, 1992). Proc VARCOMP was used to calculate the percent variance components. A square root transformation was used for life span and size to meet assumptions of normality of the residuals.

Complete information on individuals across their life cycle made it possible to do a retrospective analysis to determine the factors that contributed to differential mortality during the period of stress. For this logistic analysis, Proc GLIMMIX was used and AIC scores were used to compare alternative models. All analyses were run in SAS v. 9.1.

3. Results

3.1. Survival curves and variation in life span

All cohorts show a coordinated pattern of declining mortality when survival is graphed by date (Fig. 1) indicating that some aspect of their shared environment caused this age-independent decline. One of the consequences of these period effects is that the distribution of life spans differs across cohorts because the severe environmental effects influencing survival impacted the younger cohorts at a younger age (Fig. 2). This increasingly earlier age for the onset of high extrinsic mortality resulted in a younger median life span of 37, 27, 17 and 19 months respectively for cohorts 1–4. There was also more variation in life span when the age at the onset of stress was younger (CV = 49.30, 59.62, 73.95 and 81.86 respectively for cohorts 1–4).

In addition to environmental period effects, life span variation was influenced by spatial and genetic components and their interactions.

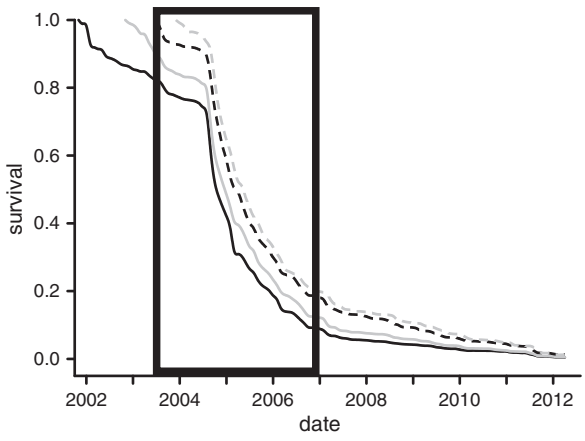

Fig. 1. Survival by date for four cohorts of *Plantago lanceolata* in an experimental field population. Cohort 1 (solid black line), Cohort 2 (solid grey line), Cohort 3 (dashed black line), Cohort 4 (dashed grey line). The boxed window designates the period of stress from May 2003–November 2006.

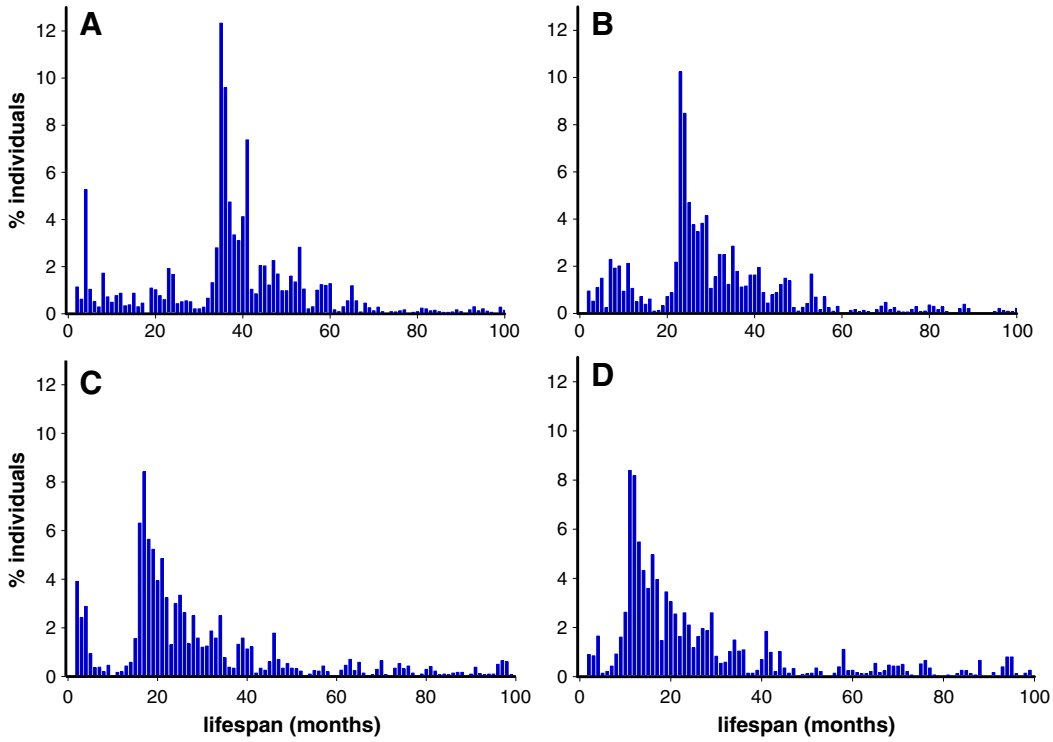

Fig. 2. Life span distributions for each age group: Cohort 1 (A); Cohort 2 (B); Cohort 3(C); Cohort 4(D). Values given are the percent of individuals in a cohort with a particular life span (months) ($n = 5944$, 8771, 4221 and 4286 respectively for cohorts 1–4).

Across the experimental population, age ($F = 82.64_{[3,22.223]}$, $p < 0.0001$) and spatial block ($F = 27.03_{[16,22.223]}$, $p < 0.0001$) influenced life span, as did sire ($F = 4.35_{[27,22.223]}$, $p = 0.0290$) and dam ($F = 5.05_{[17,22.223]}$, $p = 0.0003$). There was a significant interaction between sire and spatial block ($F = 1.24_{[428,436.19]}$, $p = 0.0132$) suggesting that sires did differently across the experimental plot. Life span was also influenced by the non-additive genetic interaction of sire-by-dam-by-cohort ($F = 1.51_{[63,21045]}$, $p = 0.0035$). There were no other significant interactions, nor were there any differences in life span across crossing sets. This genetic model described only 16% of the variation in life span, which is consistent with the very strong environmental period effects.

3.2. Variation in mortality during stress

At one particular point in time (May-November 2003), a high proportion of individuals of all age classes died. This interval marked the onset of an epoch of high mortality which lasted for 3 years (Roach et al., 2009). During this high mortality epoch, 6-month mortality rates averaged 0.29 ± 0.01 so that by November 2006 less than 15% of each cohort was still alive. Mortality during this period was significantly affected by location, size at the beginning of the stressful period, and growth rate during this period (Tables 1 and 2). Models with paternal genotype or early-life reproduction had higher AIC scores and thus did not explain any more of the mortality across this period. Plants that were larger at the beginning of the stressful period survived significantly better than smaller ones, but only if they were older. Individuals that were 2.5 and 1.5 years old (cohorts 1 and 2) at the beginning of the stressful period showed a significant impact of size on the probability of dying during the period of stress. Size marginally did not influence mortality for cohort 3, which was 1 year old at the onset of stress (Table 1). Controlling for plant size by focusing on small plants (<5 leaves) only, we found an age effect during the stressful period: older individuals had a higher mortality than younger individuals (Table 2, contingency test, mortality in cohort 1 > cohort 2 > cohort 3 > cohort 4, $p < 0.02$ for all contrasts).

The oldest individuals were the largest at the onset of the stress ($22.18 + 0.38$, $11.88 + 0.24$, $12.77 + 0.33$ and $4.23 + 0.06$ leaves, mean ± se for cohorts 1–4 respectively; $F = 634.24_{[3,\ 6054]}$, $p < 0.0001$). The oldest were significantly larger and the youngest were significantly smaller than the two intermediate age groups (Independent contrasts, $p < 0.001$ in all cases). In addition to age, spatial block ($F = 39.97_{[16,\ 6054]}$, $p < 0.0001$), dam ($F = 3.16_{[5,\ 19.246]}$, $p = 0.03$) and sire*dam ($F = 2.92_{[15,\ 6054]}$, $p < 0.0001$) contributed to variation in size.

Table 1
Logistic regression of mortality for each age class during the period of stress (May 2003–November 2006). Size is defined as the number of leaves at the onset of stress. Growth is defined as the change in size during the first 6 months of stress. *F*-values are given for each factor and the subscripts in parenthesis are the degrees of freedom. $^{****}p < 0.0001$, $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$, $^{+}p < 0.06$.

	Age at onset of stress			
	2.5 years$_{[1703]}$	1.5 years$_{[1180]}$	1.0 year$_{[756]}$	0.5 year$_{[682]}$
Spatial block$_{[11]}$	1.95*	2.72**	2.32**	1.63^{+}
Size$_{[1]}$	21.62****	9.84**	3.65^{+}	0.05
Growth$_{[1]}$	73.67****	34.20****	44.63****	24.09****

Table 2
Mortality by size class and age at onset of stress. Size class is defined as the number of leaves at the onset of stress. Mortality, $q(x)$, was calculated as the proportion of individuals who died during the period of stress (May 2003–November 2006). Note that for the calculation of mortality, there were a minimum of 60 individuals per size class for each age class. The youngest age class had only a few individuals in the largest size classes.

Number of leaves	Age at onset of stress			
	2.5 years	1.5 years	1.0 year	0.5 year
1–5	95.61%	90.77%	84.78%	77.53%
6–10	94.98%	86.28%	81.76%	82.63%
11–15	89.47%	88.18%	85.00%	
16–20	87.77%	89.16%	77.87%	
21–25	89.66%	81.20%	79.57%	
26–30	87.60%	89.47%	72.22%	
>30	85.07%	85.71%	81.58%	

Individuals who were older at the onset of stress but survived the period of stress shrank (negative growth), while those who were younger grew (positive growth) (Fig. 3). Independent of age, growth of all individuals during the first 6 months of stress was predictive of survival over the whole 3 year period (Table 1). Variation in growth during these 6 months was determined by age ($F = 58.05_{[3,\ 22.013]}$, $p < 0.0001$), spatial location ($F = 4.17_{[16,\ 41.653]}$, $p = 0.0001$), cohort-by-block ($F = 2.49_{[38,\ 1526.9]}$, $p < 0.0001$) and a cohort-by-sire-by-dam interaction ($F = 1.56_{[38,\ 1335.1]}$, $p = 0.0169$). This model explained 54% of the variation in growth and age explained 20% of this variation.

4. Discussion

Understanding the factors that determine life span is central to demography, particularly for a species like *Plantago lanceolata* that can reproduce at the latest ages. The results of this study show that it is not simply the environment that can have a major impact on demography in natural populations; rather, age, size and growth can interact with the environment to influence mortality and life span when the environment is stressful.

Environmental variability under natural conditions is high and through an individual's life span this variability has the largest influence on mortality patterns. In this study, in which individual plants were monitored from 2000 to 2011, there were 3 years, 2003–2006, when ecological conditions were stressful and mortality was high (Roach et al., 2009). Individuals of all ages showed parallel increases in mortality but different cohorts experienced this period of high mortality at different ages. This then influenced the distribution of life spans across cohorts. Not surprisingly, those cohorts that were older when the stressful period occurred lived longer on average, even though older individuals did not survive the stressful period as well as younger ones. There is a difference between the total expected life of individuals from birth and their conditional life expectancy after having reached a particular point in time. These results are consistent with demographic models with temporal environmental variability that show that the average and the variance of life expectancy and survivorship is influenced by the cumulative effects of environmental changes over an individual's life (Tuljapurkar and Horvitz, 2006). Additionally, Steiner et al. (2010) have shown that within a population of the kittiwake (*Rissa tridactyla*) individual variation in lifetime reproductive success can be explained solely by dynamic, environmental, heterogeneity and that intrinsic, fixed, individual differences had no significant influence on the variation among individuals. From an experimental design perspective, the substantial impact of environmental stochasticity on this population

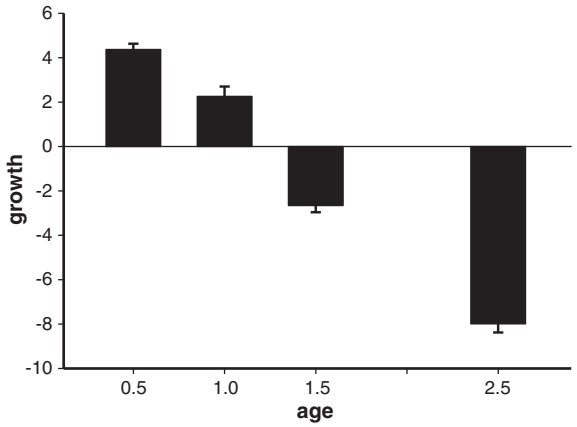

Fig. 3. Growth rate (±SE) during the first 6 months of stress (May–November 2003) for each age class. Age is the age at the onset of stress. Growth is defined as a change in the number of leaves during this period and plants that shrink in size have a negative growth rate. Independent contrasts show that all cohorts are different from each other ($p < 0.0001$ in all cases).

makes it clear that no single cohort could be used to determine age-specific mortality. The results here show that both multiple cohorts and large numbers of individuals per cohort are required to uncover the demographic patterns that are characteristic of a species in its natural environment. From an analytical perspective, our understanding of variation in life histories depends on our ability to separate variation due to stochastic environmental variation and to fixed characteristics of individuals (Coulson et al., 2010; Tuljapurkar et al., 2009).

The influence of environmental effects may include not only the present environment but also the past environment, in other words early-growth conditions (Lindstrom, 1999; Monaghan, 2008; Nussey et al., 2007). In this experimental plant population, each cohort was established at a different time and whereas they shared later-life environmental conditions, they each experienced different environments at their earliest ages. These early-life environmental differences resulted in differences across cohorts in early-life size. Subsequently, some of the variation across cohorts in later-life size and mortality could have been due to carry-over effects from early-life environmental differences. In another study we also found that the frequency and timing of reproduction, for *Plantago*, is cohort dependent and thus due to these carry-over effects (Shefferson and Roach, 2010). Similar early-life carry over effects on adult traits have been found in other studies (Clutton-Brock and Sheldon, 2010; Metcalfe and Monaghan, 2001; Nussey et al., 2007). In a recent field experiment that manipulated early- and late-life environments, it was shown that early individual history can have dramatic effects on adult phenotypes irrespective of adult environment for a small mammal (Helle et al., 2012). These delayed life history effects can also have broader consequences for population dynamics (cf. Beckerman et al., 2002; Shefferson and Roach, 2012).

Among individuals within a population, phenotypic and demographic variation may also be influenced by static traits including additive genetics, non-additive genetics (sire-by-dam interactions), and maternal effects. Unfortunately, little is known about the genetics of aging in the wild in either animals or plants (but see Wilson et al., 2007) A unique aspect of the experimental design of this project is that the same parental genotypes were used for all cohorts and the initial genetic structure of each cohort was identical. The results reported here show that some proportion, albeit small, of the total variation in life span, size, and growth could be explained by these static traits. Maternal effects usually impact the earliest life stages but they can influence later life stages (Roach and Wulff, 1987). In this same experimental population, we found that maternal effects also explained a small but significant component of the variation in the number of reproductive years of *Plantago* (Shefferson and Roach, 2010). In another recent analysis (Shefferson and Roach, 2012) we used life table response experiment (LTRE) analyses to determine the parameters that contribute to changes in population growth rate, λ, in this same experimental population. Our results showed that demographic traits in *Plantago* are strongly genetically variable and plastic. Paternity, time, and their interaction exerted strong influences over λ. Moreover, our results also showed that sire and year-by-sire interactions are strong and varied with cohort (Shefferson and Roach, 2012).

Only a small subset of individuals survived through the 3-year period of high stress to be the oldest-old. On the one hand it is not surprising that an individual's state, in other words its size and growth rate, at the onset of this stressful period were critical to survival. A novel result of this experiment is that there was an interaction between state and cohort such that the state of an individual that yielded the highest probability of mortality was cohort dependent. Previous work with this species has shown that mortality is size-dependent (Roach and Gampe, 2004), but the results here take this one step further to show that the impact of size on mortality depends on age. Differential mortality across size classes was most pronounced for the cohorts that were the oldest at the onset of stress, and within the smallest size class, mortality was highest for the oldest cohorts. Additionally, there were large differences in growth rate across cohorts in the early stages of the stressful conditions such that

the oldest cohorts were shrinking and the youngest were growing. This was most pronounced for oldest cohort that was largest in size and shrank the most; but the growth rates at this time were also different for cohorts 2 and 3, despite the fact that the size at the onset of the stressful period was the same for these two intermediate aged cohorts. Plasticity of growth and shrinkage may be a mechanism to cope with heterogeneous environments in both animals (Marinovic and Mangel, 1999; Wilelski and Thom, 2000) and plants (Horvitz and Schemske, 1995; Morris and Doak, 2004; Salguero-Gomez and Casper, 2010). In the study reported here, variation across cohorts in growth patterns and mortality suggests condition-dependent aging of a plant in its natural environment (see also Roach et al., 2009). Evidence for an age effect on mortality was also found in a demographic analysis of perennial grasses Laurenroth and Adler (2008), but not for a 300-year-old mountain herb (García et al., 2011). Little is known about senescence in plants, and given the plasticity of plants to respond to environmental change, multiple-cohort experiments that can combine both cross-sectional and longitudinal analysis may be the only way to evaluate the impact of age on mortality variation in plants. More broadly, these results show that condition-dependent frailty of the elderly is a general phenomenon for organisms across the spectrum from humans to plants.

5. Conclusions

Life in the wild is "hard" and the environment does dictate the patterns of mortality and life span. The results of this study show that when mortality is high, the intrinsic state of an individual, including its age, size, growth, genetics and interactions between these states can impact mortality and life span in natural populations.

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