

**Workshop on Evolutionary Demography**  
6-8 June 2012 in Rostock, Germany

Max Planck Institute for Demographic Research, Rostock, Germany

**Presentation Abstracts**

**WEDNESDAY JUNE 6th**

<p><b>Alberts, Susan</b> <b>Wednesday 9:10-9:50</b></p>	<p><b>Reproductive cessation patterns in primates reveal that humans are distinct</b></p> <p><i>Susan Alberts, Jeanne Altmann, Diane K. Brockman, Marina Cords, Linda M. Fedigan, Anne Pusey, Tara Stoinski, Karen B. Strier, William F. Morris, Anne M. Bronikowski</i></p> <p>Human females experience relatively early reproductive cessation and a long post-reproductive life. Whether this pattern is unique to humans or shared with other primates or mammals has been the subject of much discussion. Here we compared human schedules of reproductive cessation and mortality with those of seven nonhuman primate species, using long-term, individual-based data on mortality and fertility in wild primate populations. We asked, for each species, whether reproductive cessation occurred at the same pace as general senescence (i.e., mortality), or whether we could detect an acceleration of reproductive cessation relative to mortality in any species. As predicted, we found no evidence in any of the nonhuman primates that reproductive cessation experienced higher age-based acceleration than mortality, as is seen in humans.. This result provides strong support for the hypothesis that humans have experienced a different selective regime on reproductive cessation than other primates.</p>
<p><b>Westendorp, Rudi</b> <b>Wednesday 9:50-10:30</b></p>	<p><b>Survival under adverse conditions in contemporary Africa</b></p> <p><i>Rudi Westendorp</i></p> <p>We studied post-reproductive survival from an evolutionary perspective and specifically tested the hypothesis that men and women after age 50 are able to contribute to their fitness either directly by continued reproduction, or indirectly through improving the reproductive success of subsequent generations. In a prospective full kin analysis in the Upper East Region in Ghana, we studied the effect of different kin members on offspring reproduction, survival and early growth patterns in a polygamous population that lives in an environment that could reflect our recent evolutionary past. The consequences of these selection pressures on immune genotypes are being discussed.</p>

<p><b>Mace, Ruth</b> <b>Wednesday 10:30-10:50</b></p>	<p><b>Reproductive competition between females in Gambian families</b></p> <p><i>Ruth Mace, Alex Alvergne</i></p> <p>Here we seek evidence for female reproductive competition in a rural Gambian population. We find a small effect of female co-residency within the same compound, although it was asymmetric, in that older women appear to do better when with larger numbers of unrelated co-residents, whereas younger women suffered costs. We also found evidence of reproductive competition between mothers and daughters of reproductive age although it was in the opposite direction, in that the younger woman (the daughter) did not suffer from reproductive overlap with her mother, but the older woman (the mother) did, and the older she was the stronger the effect. This effect exists even though the cultural practice of virilocality ensures mothers and daughters are not generally co-resident. A cultural norm of late male marriage has the effect of reducing paternal grandmother/daughter-in-law reproductive overlap almost to zero. Cultural practices surrounding marriage norms combined with the existence of menopause could be considered as adaptations to reduce reproductive conflict between generations.</p>
<p><b>Vindenes, Yngvild</b> <b>Wednesday 11:10-11:30</b></p>	<p><b>Effects of temperature on evolutionary demography of pike (<i>Esox lucius</i>)</b></p> <p><i>Yngvild Vindenes, Eric Edeline, Jan Oehlberger, Øystein Langangen, Ian Winfield, Nils Chr. Stenseth, Asbjørn Vøllestad,</i></p> <p>Climate variables often affect vital rates differently, making it hard to predict resulting effects on population-level processes. Based on a long-term study of pike from lake Windermere (UK), we construct a stochastic IPM to evaluate how the demography and population dynamics respond to changes in mean annual lake temperature. Using generalized linear mixed models, vital rates are estimated as functions of length and temperature, within each of six age classes. To estimate environmental stochasticity including correlation between vital rates, year is included as random effect. The model predicts an overall positive effect of temperature on population growth, as well as several other changes in age/length-specific properties with temperature (stable distribution, reproductive values, mean length at age, reproduction function, etc). Furthermore, based on this IPM we calculate age- and temperature-specific selection differentials for viability and fecundity selection on length, and discuss their potential implications for temperature-induced evolutionary responses in the population.</p>

<p><b>Buckley, Yvonne</b>  <b>Wednesday 11:30-11:50</b></p>	<p><b>Increased population growth rate in invasive polyploid <i>Centaurea stoebe</i> provides evidence for rapid evolution</b></p> <p><i>Yvonne Buckley</i></p> <p>Biological invasions are inherently demographic processes, but trait differences between native and introduced genotypes are rarely linked to population growth rates. Native European <i>Centaurea stoebe</i> occurs as two cytotypes with different life histories (monocarpic diploids and polycarpic tetraploids); however, only tetraploids have been found in its introduced range in North America. Both pre-adaptation of tetraploids to reduced specialist herbivory and subsequent rapid evolution may explain invasiveness. We tested these hypotheses in a 3-year common garden experiment using periodic matrix models and life table response experiments. We found no difference in population growth rate between the two European cytotypes and little effect of herbivory in all geo-cytotypes. However, there was a pronounced increase in population growth rate for North American tetraploids compared to European tetraploids due to increased seed production and juvenile establishment. We conclude that rather rapid post-introduction evolution than pre-adaptation through polyploidy may explain the success of North American tetraploids.</p>
<p><b>Jongejans, Eelke</b>  <b>Wednesday 11:50-12:10</b></p>	<p><b>Phenotypic plasticity and rapid evolution in plant population models</b></p> <p><i>Eelke Jongejans</i></p> <p>Hierarchical population models include the impact of environmental drivers, traits and vital rates on population dynamics. This allows for the study of the population-level consequences of plastic and evolutionary trait changes in response to environmental changes (cf. Ellner et al. 2011). Field studies that measure the dynamics of traits and structure of plant populations at the same time are still very rare. Instead I will discuss ways to explore this framework by combining disparate data sets or using simulations.</p>
<p><b>Childs, Dylan Z.</b>  <b>Wednesday 12:10-12:30</b></p>	<p><b>The evolutionary demography of lay date in the Wytham Great tit</b></p> <p><i>Dylan Z. Childs</i></p> <p>I present a demographically informed model of lay date dynamics in the Wytham Great tit. By combining this with an appropriate ‘animal model’ characterising the quantitative genetics of this age-structured trait, I predict the expected response to selection in the face of a temporally stochastic environment. Finally, I apply standard tools from evolutionary demography to understand how changes in the mean and variance of survival and recruitment impact these predictions.</p>

<p><b>Pelletier, Fanie</b>  <b>Wednesday 12:30-12:50</b></p>	<p><b>Population growth and fluctuating opportunities for selection on phenotypic characters</b></p> <p><i>Fanie Pelletier, Tim Coulson</i></p> <p>Recent studies have revealed that evolutionary changes in natural populations can occur on ecological time scales. This realization has generated a lot of interest in characterising short-term fluctuations in selection pressure, often on time scales shorter than the generation; identifying the circumstances under which the opportunity for selection is largest; and whether or not this opportunity is realized. In this talk, I will introduce new metrics to explore how the opportunity for selection on the mean of a phenotypic character varies with time. I then use data from two long-term studies of marked individuals to examine how the opportunity for, and the selection on a character mean and variance covaries with population growth. Empirical results support those obtained through simulation: the classical metric – the opportunity for selection – is not useful for determining how much selection could potentially alter the distribution of phenotypic characters. The new metrics however suggest that selection is strongest in declining populations and is greatest when the opportunity for selection on a character mean is large. All together these results support the contention that selection should be strongest in bad environments.</p>
<p><b>Caswell, Hal</b>  <b>Wednesday 14:30-15:10</b></p>	<p><b>A model for the grandmother effect</b></p> <p><i>Hal Caswell</i></p> <p>Selection gradients on age-specific mortality decline with age after maturity, and drop to zero at the age of last reproduction. This has left post-reproductive survival, as occurs for example in females of humans and toothed whales, as a problematic phenomenon in evolutionary demography. There exist several reasons why post-reproductive survival might be selected for; the most obvious and venerable of these being that post-reproductive mothers might benefit their daughters and grand-daughters in ways having nothing to do with reproduction per se. This talk will present a model for this "grandmother effect" and use it to explore how selection gradients on mortality are affected by the pattern, the magnitude, and the duration of maternal and grand maternal benefits to progeny.</p>
<p><b>Archie, Elisabeth</b>  <b>Wednesday 15:10-15:30</b></p>	<p><b>Social connectedness and longevity in wild baboons</b></p> <p><i>Elisabeth Archie, Jeanne Altmann, Susan Alberts</i></p> <p>Social bonds play an important role in the daily lives of many social species, and the strength and quality of these bonds is expected to influence individual health and longevity. Using long-term data from the wild baboons living in the Amboseli ecosystem, we compare changes in social connectedness over the course of individual life histories, and investigate the implications of social connectedness for individual survival. We discuss the implications of our results for human health and the evolution of sociality.</p>

<p><b>Moorad, Jacob</b>  <b>Wednesday 15:30-15:50</b></p>	<p><b>Contextual analysis as a method for measuring socially derived sources of natural selection for longevity</b></p> <p><i>Jacob Moorad</i></p> <p>Contextual analysis is a variance-partitioning approach used by evolutionary biologists to quantify sources of selection that act through social interactions. However, this method has yet to be applied to address evolutionary demographic questions. I will demonstrate how this approach can be applied to the study of phenotypic selection in age- and family-structured populations. Using longitudinal data from the Utah Population Database, I show how contextual analysis reveals how selection for age-specific survival is modified by the longevity of family members.</p>
<p><b>Roach, Deborah</b>  <b>Wednesday 16:10-16:50</b></p>	<p><b>Joint modeling of reproduction and size in <i>Plantago</i>: do we need to consider history?</b></p> <p><i>Deborah Roach, Jutta Gampe</i></p> <p>We will report on the results from a large, multiple cohort, study which has followed approximately 30,000 individuals, of <i>Plantago lanceolata</i>, over 12 years. Previous analysis on this data has shown that demographic traits in this natural population may be influenced by both short-and long-term environmental fluctuations as well as age-dependent changes. We will present our analysis from joint modeling of reproduction and size to determine whether these demographic patterns may also be influenced not only by an individual's current environment and state (size and age) but also by its history.</p>
<p><b>Steiner, Ulrich</b>  <b>Wednesday 16:50-17:10</b></p>	<p><b>Life histories and variance of individual fitness components in <i>Plantago lanceolatum</i>: genetics, environments and stochasticity</b></p> <p><i>Ulrich Steiner, Deborah Roach</i></p> <p>Understanding and predicting individual life histories and their responses to ecological and evolutionary change requires the quantification of genetic, environmental, and neutral (stochastic) processes. We present a variance decomposition of individual fitness components, using an age-stage structured integral projection model on an experimental cohort study of <i>Plantago lanceolatum</i>. Variance among genotypes in generation time and net reproduction was larger compared to the variance in population growth rates, suggesting some evolutionary buffering between fast and slow life histories. But such buffering is not necessarily due to the classical tradeoff between reproduction and survival. Despite the variance in population growth among genotypes, the total variance in individual fitness components was dominated by non-genetic effects, i.e. environmental variability and other stochastic processes. Genetic drift may play a dominant role in the system and adaptive dynamics with respect to plant size are likely to be slow.</p>

<p><b>Salguero-Gomez, Roberto</b>  <b>Wednesday 17:10-17:30</b></p>	<p><b>Old and new axes of plant demography</b></p> <p><i>Roberto Salguero-Gomez</i></p> <p>Life-history theory predicts that the variation in demographic strategies displayed by the immense majority of organisms can be explained with a single axis: the so-called fast-slow continuum. This life-history axis displays a trade-off between reproduction and future survival, such that individuals that start reproducing earlier in life, live faster and die younger compared to those that delay the first reproductive event. This theory has been largely explored in unitarian organisms (most animals), and remains to be tested in other architectural forms and kingdoms. Here a large database of projection matrices (COMPADRE) was used to explore the existence of life-history axes in ca. 400 plant species ranging from annuals to herbaceous perennials, shrubs, succulents, palms, lianas and trees. Two axes were found to explain most of the demographic variation. The first axis depicts the fast-slow continuum, and is strongly affected by species covariates such as phylogeny, habitat and growth form. The second axis, a novel one, is related to reproductive investment and is not driven by phylogenetic signal or environmental filtering, suggesting that it is universal. The implications of these findings are discussed in the light of life-history theory in modular vs unitarian organisms. I suggest that the lack of maternal care in plants as compared to, for instance, mammals, might explain the emergence of a new axes of life history.</p>
<p><b>Rees, Mark</b>  <b>Wednesday 17:30-17:50</b></p>	<p><b>Growth: an evolutionary perspective</b></p> <p><i>Mark Rees</i></p> <p>Growth is a key process which determines changes in size, survival and fecundity. However, our understanding of the evolution of growth rates is limited. Reasons for this will be presented, dealing with the description of growth processes, compensatory growth, seed-seedling growth relationships, and the cost and benefits of fast/slow growth.</p>
<p><b>Tuljapurkar, Shripad</b>  <b>Thursday 9:10-9:50</b></p>	<p><b>Phenotypic Variability and life History Evolution</b></p> <p><i>Shripad Tuljapurkar</i></p>

<p><b>Dieckmann, Ulf</b>  <b>Thursday 9:50-10:30</b></p>	<p><b>Modern life-history theory and the crucial role of density regulation</b></p> <p><i>Ulf Dieckmann</i></p> <p>Twenty years ago, classical life-history theory was firmly put on the map by two seminal textbooks, written by Stephen Stearns and Derek Roff, respectively. Since then, this approach has enabled a plethora of innovative studies, explaining how life-history events and their schedules are shaped by natural selection. Questions regarding the timing of growth, maturation, reproduction, and mortality, as well as the allocation of resources to the underlying physiological processes, thus became amenable to theoretical analyses within a coherent and unified framework. In particular, the principle of fitness maximization promised to explain how empirically observed life-history characteristics vary with empirically observed environmental characteristics. Despite these successes, two fundamental ecological features were largely left out from this approach: realistic density regulation and frequency-dependent selection. It turns out that, from an evolutionary perspective, these are just flipsides of the same coin: in fact, observed life histories usually do not maximize any absolute measure of fitness; instead, they must be understood as attractors of frequency-dependent evolutionary dynamics determined by the interplay of density-dependent and phenotype-dependent aspects of ecological processes. The resultant challenges for modernizing classical life-history theory are met by the theory of adaptive dynamics, of which this presentation will provide a brief outline.</p>
<p><b>Kirkwood, Tom</b>  <b>Thursday 10:30-11:10</b></p>	<p><b>Connecting ultimate and proximate causes of ageing</b></p> <p><i>Tom Kirkwood</i></p> <p>Understanding why and how senescence evolved is of great importance in investigating the multiple, complex mechanisms that influence the course of ageing in humans and other organisms. Compelling arguments eliminate the idea that death is generally programmed by genes for ageing, but there is still a widespread tendency to interpret data in terms of loosely defined age-regulation which does not usually make either evolutionary or mechanistic sense. This talk will critically address the role of natural selection in shaping ageing within the life history and examine the implications for research on the mechanisms and genetic pathways that influence the lifespan.</p>

<p><b>Baudisch, Annette</b>  <b>Thursday 11:30-11:50</b></p>	<p><b>Explaining Species Diversity in Pace and Shape of Aging</b></p> <p><i>Annette Baudisch</i></p> <ol style="list-style-type: none"> <li>1. Humans age, but how much more or less do we age compared with other species? Do humans age more than chimps, birds more than fish or sheep more than buffalos? In this article, I argue that current methods to compare patterns of ageing across species are limited because they confound two dimensions of age-specific change – the pace and the shape of ageing.</li> <li>2. Based on the two axes of pace and shape, I introduce a new conceptual framework to classify how species age.</li> <li>3. With this method, I rank species according to how strongly they age (shape) and how fast they age (pace). Depending on whether they are ranked by pace or by shape, species are ordered differently.</li> <li>4. Alternative pace measures turn out to be highly correlated. Alternative shape measures are also highly correlated. The correlation between pace and shape ranking is negative but weak. Among the examples here, no species is long lived yet exhibits negligible ageing – contrary to the commonly held view that long-lived species are good candidates for negligible ageing.</li> <li>5. Analysis of species in pace–shape space provides a tool to identify key determinants of the evolution of ageing for species across the tree of life.</li> </ol>
<p><b>Wrycza, Thomas</b>  <b>Thursday 11:50-12:10</b></p>	<p><b>Measuring the Pace and Shape of Aging</b></p> <p><i>Thomas Wrycza</i></p> <p>The concept of the Pace and Shape of Aging tries to disentangle two distinct dimensions of (demographic) aging: one that captures the speed of life (Pace) and one that captures the change over life (Shape). The presentation aims at demonstrating how this concept is realized mathematically. This includes a discussion of why and how to (Pace-)standardize mortality curves, and I will use several examples to illustrate this process. Also, I will present measures for Shape and discuss both the problems and perspectives that arise from their use.</p>
<p><b>Wensink, Maarten</b>  <b>Thursday 12:10-12:30</b></p>	<p><b>Senesce to live long</b></p> <p><i>Maarten Wensink</i></p> <p>In my presentation I combine insights about mortality sources, mortality trajectories and evolution to propose an alternative perspective from which to look at the evolution of senescence.</p>



<p><b>Kozlowski, Jan</b>  <b>Thursday 12:30-12:50</b></p>	<p><b>Mutation accumulation may be a minor force in shaping life history traits</b></p> <p><i>Jan Kozlowski, Maciej Jan Dańko, James Walton Vaupel, Annette Baudisch</i></p> <p>Is senescence the adaptive result of tradeoffs between younger and older ages or the nonadaptive burden of deleterious mutations that act at older ages? To shed new light on this unresolved question we combine adaptive and nonadaptive processes in a single model. Our model uses Penna's bit-strings to capture different age-specific mutational patterns. Each pattern represents a genotype and for each genotype we find the life history strategy that maximizes fitness. Genotypes compete with each other and are subject to selection and to new mutations over generations until equilibrium in gene-frequencies is reached. The mutation-selection equilibrium provides information about mutational load and the differential effects of mutations on a life history trait - the optimal age at maturity. We find that mutations accumulate only at ages with negligible impact on fitness and that mutation accumulation has very little effect on the optimal age at maturity. These results suggest that life histories are largely determined by adaptive processes. The non-adaptive process of mutation accumulation seems to be unimportant at evolutionarily relevant ages.</p>
<p><b>Scheuerlein, Alexander</b>  <b>Thursday 14:10-14:30</b></p>	<p><b>The Varieties of Aging: Mortality and Fertility schedules across the Tree of Life</b></p> <p><i>Alexander Scheuerlein, Owen R. Jones, Roberto Salguero-Gómez, Carlo Giovanni Camarda, Vinicius Lucio, Oskar Burger, Hal Caswell, Daniel Levitis, Barbara Pietrzak, Felix Quade, Maren Rebke, Felix Ringelhan, Ralf Schaible, Annette Baudisch, Jim Vaupel</i></p> <p>Modern humans are characterized by their mortality and fertility trajectories. Mortality follows a Gompertz or a Gompertz – Makeham model, starting low at the onset of sexual maturity until late ages, where high mortality rates are reached. In contrast, fertility (=realized reproduction) rises steeply and drops off sharply, leaving a large proportion of the population to experience reproductive senescence. Biodemography strives to understand patterns of mortality and fertility across the tree of life, and in particular to what extent phylogenetic history has shaped these patterns. Here, using data on mortality and fertility trajectories from iteroparous organisms, we illustrate the great diversity of mortality and fertility patterns across the tree of life. Strikingly, many of the patterns we find here are at odds with classic evolutionary theories of aging, which predict increasing mortality and decreasing fertility after the onset of sexual maturity.</p>

<p><b>Cohen, Alan</b>  <b>Thursday 14:30-14:50</b></p>	<p><b>Why physiology matters in evolutionary demography</b></p> <p><i>Alan Cohen</i></p> <p>Evolutionary demography presumes that selection acts on demographic parameters so as to optimize life history trade-offs. At the same time, there is increasing evidence that for taxon-specific complex interactions across physiological systems. Much as genetic and developmental mechanisms impose constraints on the free hand of selection to shape phenotype, these taxon-specific physiological regulatory systems will constrain the influence of selection on demographic parameters. For example, taxa with strong resistance to oxidative stress may show greater ability to evolve long lifespans as they move into environments with highly variable oxygen supply. Many such constraints will arise not from single physiological systems but from the interactions of many, and we are still far from understanding the specifics of when and how they operate. As a temporary solution, EvoDemo models should take into account phylogenetic structure (formally or informally) as a way to incorporate the inertia of physiological constraints on demographic parameters.</p>
<p><b>Tung, Jenny</b>  <b>Thursday 14:50-15:10</b></p>	<p><b>Gene regulation and aging across the life course: exploring the mechanistic underpinnings of social and life history effects on health and physiology</b></p> <p><i>Jenny Tung</i></p> <p>Biodemographic studies of aging have revealed a number of robust patterns, including sex differences in morbidity and mortality rates, persistent effects of early life on later life history, and a role for the social environment in influencing aging outcomes. These phenomena must ultimately have an underlying physiological basis. However, a mechanistic understanding of how such patterns arise has lagged behind. Here I draw from work on two nonhuman primate systems, captive rhesus macaques and wild yellow baboons, to discuss potential genetic and genomic contributions to these effects. These studies demonstrate a strong effect of social environment on gene regulation, including early life effects on adult gene expression, and epigenetic changes that may help explain the persistence of these effects over the life course. Further, the gene regulatory signature of social environment recapitulates, in some biological pathways, gene regulatory signatures of aging, suggesting how social interactions, life history, and aging may be linked.</p>

<p><b>Jones, Owen</b> Thursday 15:10-15:30</p>	<p><b>Coping with phylogeny and uncertainty in the association between life span and reproductive investment in British birds</b></p> <p><i>Owen R. Jones and Fernando Colchero</i></p> <p>The question of why some species live a long time, and others only a short time, has fascinated scientists for centuries. Birds are an ideal group to investigate this topic because they inhabit a wide range of ecological niches and exhibit a wide range of life histories and life expectancies. Here we use maximum-recorded life span data obtained from the ringing records of the British Trust for Ornithology (BTO) to investigate the association between reproductive effort and life span in approximately 200 British bird species. Maximum-recorded life span has previously been criticized for being sensitive to sample size variation; greater sample sizes lead to greater apparent life spans. However, we use a phylogenetically corrected state-space modeling approach that accounts for this problem and other data issues including truncation (unknown birth date) and censoring (unnatural death) while simultaneously controlling for phylogenetic relatedness. We show that the inherent problems of using the sample-size-sensitive measure of maximum-recorded life span can be overcome, along with other data quality issues. We demonstrate that accounting for phylogenetic relatedness weakens the association between reproductive strategy and life span, implying that inherited characteristics play a dominant role. In addition, our model infers that we consistently underestimate life span in some species groups.</p>
<p><b>Gaillard, Jean-Michel</b> Thursday 15:30-15:50</p>	<p><b>What factors shape actuarial senescence in captive populations of large mammalian herbivores?</b></p> <p><i>Jean-Michel Gaillard, Jean-Francois Lemaitre</i></p> <p>From life tables collected on captive populations of large mammalian herbivores, we performed capture-recapture analyses to model age-specific variation in survival. Gompertz models from a threshold age provided a suitable description of age-specific changes of survival in most species. We thus estimated sex- and species-specific estimates of both baseline mortality and rates of actuarial senescence from the best Gompertz model. In most cases, we found clear statistical support for senescence. Baseline mortality and aging rate were higher in males than in females in most species, independently of the intensity of sexual selection. Among ecological variables, only diet influenced senescence, with browser species suffering more than grazers. There was a positive co-variation between baseline mortality and aging rate, but contrary to the theoretical expectation, the onset of actuarial senescence often occurred after the age at first reproduction. In general, large herbivores in captivity showed reduced senescence compared to their wild counterparts.</p>

<p><b>Carey, James</b>  <b>Thursday 16:10-16:50</b></p>	<p><b>Evolutionary demography of the Mediterranean fruit fly from six world regions</b></p> <p><i>James Carey</i></p> <p>In a common garden environment, we examined whether medfly populations obtained from six global regions [Africa (Kenya), Pacific (Hawaii), Central America (Guatemala), South America (Brazil), Extra-Mediterranean (Portugal), and Mediterranean (Greece)] have evolved different survival and reproductive schedules. Whereas females were either short-lived [life expectancy at eclosion (<math>e_0</math>) 48-58 days; Kenya, Hawaii, and Guatemala] or long-lived (<math>e_0</math> 72-76 days; Greece, Portugal, and Brazil), males with one exception (Guatemala) were generally long-lived (<math>e_0</math> 106-122 days). Although males universally outlived females in all populations, the longevity gender gap was highly variable (20-58 days). Lifetime fecundity rates were similar among populations. However, large differences were observed in their age-specific reproductive patterns. Short-lived populations mature at earlier ages and allocate more of their resources to reproduction early in life compared to long-lived ones. In all populations, females experienced a post reproductive life span with this segment being significantly longer in Kenyan flies. The results of this comparative demographic study reveal the different ways in which medflies evolved different life history strategies to cope with local environmental conditions.</p>
<p><b>Colchero, Fernando</b>  <b>Thursday 16:50-17:10</b></p>	<p><b>Hierarchical Bayesian methods to improve inference on vital rates across the tree of life</b></p> <p><i>Fernando Colchero</i></p> <p>Unraveling how mortality and fertility rates change in wild populations is fundamental to understand the evolution of these life history traits. Unfortunately, little is still known about the demographic rates of most species of plants and animals. Of the fraction of those species for which we have some information, the data available are commonly very patchy. For example, in most field studies age information is normally partially or entirely missing and, as a result, researchers have strong reservations to draw inference on age-specific mortality and fertility rates. Failing to recognize such age effects can strongly bias, for instance, the way we understand the evolution of senescence, or the implementation of conservation plans for endangered species. Hierarchical Bayesian models combined with state-space and imputation methods can provide modeling alternatives for inference on vital rates despite missing data on response or explanatory variables. These methods provide an appropriate framework to disentangle different sources of variability and uncertainty, while allowing researchers to explore more complex process based models. With the strong interest on unraveling the evolution of age related processes across the tree of life and its applications to conservation, these methods can become a fundamental tool for demographers, ecologists and evolutionary biologist.</p>

<p><b>Rebke, Maren</b>  <b>Thursday 17:10—17:30</b></p>	<p><b>A hierarchical Bayesian model of the steps leading to reproduction</b></p> <p><i>Maren Rebke, Fernando Colchero, Peter H. Becker, Tim Coulson</i></p> <p>In a monogamous species two partners contribute to the breeding process. How are partners chosen? Does pair bond play a role for reproductive performance? Is there an additional effect of age? Do males and females show different patterns? Is there heterogeneity between individuals? We use a dataset of the long-lived seabird common tern (<i>Sterna hirundo</i>), marked with automatically read transponders, to address these questions. Despite the high quality of the data, it is not always obvious if unmarked partners are new or not. Further, for unmarked partners, and also for some of the marked individuals, age, sex and time of death are unknown. Handling these complications and modelling the complicated process driving reproduction requires a complex model. We use a hierarchical Bayesian model of the steps that lead to the number of fledglings, which includes processes operating at the individual level and the pair level.</p>
<p><b>Conde-Ovando, Dalia</b>  <b>Thursday 17:30-17:50</b></p>	<p><b>Conservation Demography</b></p> <p><i>Dalia Conde-Ovando</i></p> <p>Climate change, habitat loss, and invasive species, among other consequences of human activities, have dramatic effects on species' vital rates (i.e., survival and fertility). Decreasing survival and fertility can ultimately drive species to extinction. Consequently, predicting future demographic trends for wild populations and triaging our conservation efforts is essential for the management of threatened species. Population Viability Analyzes (PVA) are widely used to predict species population trends and manage at risk taxa. Previous research has show that for PVA to be accurate, they need to be parameterized with at least basic demographic data. However the lack of data is a major constraint, for example in the form of life tables there is only published data for approximately 0.5% of the world's vertebrates. This lack of data makes species management extremely challenging. However, zoos have an extensive amount of data at least for ~15% of the world's threatened species. Therefore, it is essential to estimate for which species and with which confidence we can parameterize PVA using data from captive populations to manage wild ones. By comparing how mortality trajectories change between wild and zoo populations and by understanding how these changes are related to either species with similar life history strategies or taxonomic relatedness, it will be possible to contribute to resolve one of the key issues to manage some of the species at risk of extinction.</p>

<p><b>Levitis, Daniel</b>  <b>Friday 9:10-9:30</b></p>	<p><b>Ontogenescence: What is it, who does it, why doesn't selection eliminate it, and how to investigate it?</b></p> <p><i>Daniel Levitis, Paul Dunn, Barbara Pietrzak, Tuuli Pajunen</i></p> <p>Ontogenescence, the decline in cohort mortality risk over age during ontogenesis, is among the most widely observed life-history traits, occurring broadly across taxa, life-histories, causes of death and environments in senescing and non-senescing populations. While work explaining variation in the pattern or level of ontogenetic mortality is common, few researchers have examined the question of why the general pattern of ontogenescence is so broadly observed, and most of these propose hypotheses that cannot apply as broadly as the pattern is observed. MPIDR's ontogenescence group uses experimentation and comparative and demographic analysis to test hypotheses linking ontogenescent mortality to heterogeneity, to the acquisition of robustness, to the timing of developmental transitions and to genetic recombination. Projects include,</p> <ol style="list-style-type: none"> <li>1. The comparison of ontogenescence between sexually and asexually produced offspring of Hydra.</li> <li>2. The experimental examination of changing robustness to insult over age and across developmental transitions in barnacles.</li> <li>3. Examining the relationship between developmental mortality and developmental transcriptional dynamics in Daphnia.</li> <li>4. Analysis of human demographic data to examine the role of inter-individual heterogeneity in human ontogenescence.</li> </ol>
<p><b>Metcalf, Charlotte</b>  <b>Friday 9:30-9:50</b></p>	<p><b>Ignoring the microbe in the room: infectious diseases and the evolution of demographic age trajectories</b></p> <p><i>Charlotte Jessica Metcalf</i></p> <p>Infectious diseases are a leading cause of human mortality. The evolution of our age trajectory of mortality must have occurred partly in response to this pressure. Because pathogen generation times are generally much shorter than ours, allowing rapid rates of evolutionary change, developing control mechanisms for pathogens is a complex problem. The innovation developed deep in evolutionary history to meet this challenge is the adaptive immune system. This comes with particular costs and outcomes for the right hand side of the mortality trajectory; but also has surprising implications for the left hand side of the mortality trajectory. I outline some of these patterns, and discuss evolutionary constraints on maternal immunity.</p>

<p><b>Pavard, Samuel</b>  <b>Friday 9:50-10:10</b></p>	<p><b>Are old ages useless with respect to selection? A reconsideration of the common variants/common diseases hypothesis</b></p> <p><i>Samual Pavard, Charlotte Jessica Metcalf, BrunoToupance</i></p> <p>Alleles involved in late mortality are considered neutral because no reproduction occurs after menopause. However, most susceptibility alleles to late onset diseases are at low frequency: a characteristic of alleles under negative selection. Three phenomena may explain this: (i) men can reproduce with younger women until old ages; (ii) parental care enhance offspring fitness even during post-reproductive period; and (iii) variance in age of onset means that disease may occur during reproductive ages even for late-onset diseases. Here, we modelled (i) and (ii) for a different demographic regimes, and (iii) for a large parameter space of age of onset. We incorporate these three phenomena into a population genetic framework to calculate selection coefficients for susceptibility alleles to late onset diseases. Results show that negative selection occurs for most of known late-onset genetic diseases and overcome genetic drift for an effective population size compatible with most human populations.</p>
<p><b>Stearns, Steve</b>  <b>10:10-10:30</b></p>	<p><b>Oncogenes and the costs of reproduction in the Framingham Heart Study population</b></p> <p><i>Steve Stearns</i></p> <p>The tradeoff between reproduction and survival is central to the evolution of aging, but the mechanisms that mediate it, and the genes that influence those mechanisms, are not yet well understood. In the Framingham Heart Study population, for women born before 1930, there is are significant negative phenotypic and genetic correlations between lifetime reproductive success (children ever born) and lifespan. A genome-wide association study found 3 genes that influence that relationship: a protein, a transcription factor, and RAS, an oncogene. Other studies have found that P53 and BRCA1/2 share this characteristic: the variants that increase reproductive performance early in life also elevate risk of death from cancer late in life. Cancer did not become a significant cause of death until populations moved through the demographic transition. Prior to that, such genes had predominantly positive effects. Whether other oncogenes share this type of antagonistic pleiotropy is a question well worth pursuing.</p>

<p><b>Lummaa, Virpi</b>  <b>Friday 10:30-10:50</b></p>	<p><b>Sexual conflict over post-reproductive lifespan in human</b></p> <p><i>Virpi Lummaa, Sandra Bouwhuis, Jenni Pettay</i></p> <p>Women outlive men in nearly all contemporary human populations. Such dimorphism may have resulted from sexually antagonistic selection, as recent studies have reported positive selection on female and stabilising selection on male lifespan in humans. We assess whether selection on lifespan differed between the sexes in pre-industrial Finnish populations, and estimate sex-specific heritabilities and genetic correlations for lifespan, post-reproductive lifespan and lifetime reproductive success. Our analyses confirm sexually antagonistic selection for lifespan and post-reproductive lifespan. All traits in both sexes displayed significant heritability. The genetic correlation between post-reproductive lifespan and reproductive success, however, differed between the sexes, being negative in men, but positive in women. The intersexual genetic correlation for post-reproductive lifespan was positive, constraining the predicted response to selection in women but allowing near-perfect predicted response in men. These results provide evidence for strong sexual conflict over post-reproductive lifespan in humans, which should have genetically constrained the evolution of sexual dimorphism.</p>
<p><b>Vaupel, James</b>  <b>Friday 11:10-11:50</b></p>	<p><b>The rates of aging</b></p> <p><i>James Vaupel</i></p> <p>The so-called rate of aging is the relative derivative with age of the force of mortality: it describes how quickly mortality increases. The rate of aging may be a fundamental human invariant. The longevity revolution has been caused not by a slowing of the aging process but by a postponement of mortality (and ill health) to higher ages. Evidence about the rate of human aging will be presented as well as some evidence for non-human species.</p>
<p><b>Missov, Trifon</b>  <b>Friday 11:50-12:10</b></p>	<p><b>Implications of Mortality Plateaus in Fixed-Fraily Proportional Hazards Framework</b></p> <p><i>Trifon Missov</i></p> <p>In a fixed-frailty proportional hazards framework mortality plateaus imply a gamma-Gompertz model at the oldest-old ages. Baseline mortality must converge with age to a Gompertz curve and the frailty distribution among survivors to each subsequent age must approach gamma.</p>



<p><b>Yuhui, Lin</b>  <b>Friday 12:10-12:30</b></p>	<p><b>The Rate of Aging in Smokers</b></p> <p><i>Lin Yuhui, Kaare Christensen, James Vaupel</i></p> <p>A substantial number of epidemiological studies have discussed changes in mortality risk by life history exposures, and yet few have investigated the rate of aging. The rate of aging has been widely accepted as age-related mortality and the decline in physiological function. For example, smoking has been associated with an increased risk for morbidity and mortality. Hence, it has been assumed that smokers experience a higher rate of aging than never smokers. In this study, we address whether this assumption can be casually accepted by investigating the rate of aging in current and never smokers. Here, we illustrate the force of mortality by smoking status and define the rate of aging as the relative change in mortality risk. As expected, smokers experience a higher risk for mortality than never smokers. Interestingly, their relative risk converged with increasing age, a phenomenon due to unobserved heterogeneity during survival time analysis. Our results also demonstrate that the force of mortality on the arithmetic scale offers a limited understanding of the rate of aging. We propose the application of a frailty-based Gompertz model and the relative change in mortality risk to interpreting the rate of aging. This study is motivated by the increasing popularity in population-based studies and research in global aging. All analyses discussed are illustrated using the Danish Twins 1966 study.</p>
<p><b>Burger, Oskar</b>  <b>Friday 12:30-12:50</b></p>	<p><b>Seventy-six is the new thirty: A comparative baseline for evolutionary biodemography</b></p> <p><i>Oskar Burger</i></p> <p>Placing human mortality reduction in biodemographic context requires an appropriate comparative baseline. Such a baseline should approximate the average age-specific trends in mortality as they were experienced through most of human evolution and should not be limited to a specific calendar year or country. This baseline is provided by the average mortality profile of ethnographically observed hunter-gatherers. Comparing this evolutionary baseline to a range of human populations reveals many striking patterns in the pace and timing of mortality improvement. For instance, the mortality difference between Japanese women and the evolutionary pattern is much greater than the difference between the evolutionary pattern and chimpanzees, but this has only been the case for the last 3 or 4 generations. Comparison with model organisms reveals that humans have made more progress per generation than laboratory selection experiments and have made more total progress than lifespan-extending genetic mutations.</p>

<p><b>Andersen, Ken Haste</b> Friday 14:10-14:50</p>	<p><b>Why do individuals have a digestive overcapacity?</b></p> <p><i>Ken Haste Andersen</i></p> <p>The growth rate of an individual is limited by either the available food or the digestive capacity of their gut. It is often observed that individuals have a digestive overcapacity such that the availability of food is limiting growth. The question is why individuals have such a digestive overcapacity? I will demonstrate the digestive overcapacity emerges as the evolutionary optimal solution to a competition between individuals with different digestive capacities. The effect is demonstrated based on data on fish. Finally I will show how the results of the simple calculation compare with a full dynamic simulation of a fish community. The simulations further demonstrate that the digestive overcapacity is increasing with size of the individuals.</p>
<p><b>Lee, Who Seung</b> Friday 14:50-15:10</p>	<p><b>The impacts of early growth trajectory on reproduction and lifespan</b></p> <p><i>Who Seung Lee</i></p> <p>Changes in environmental conditions can cause changes in the tempo and pattern of growth, development and reproductive investment in animals. An episode of poor conditions (i.e. reduced nutrition, and/or low temperatures in the case of ectotherms) is generally linked to a slowing of growth. If adequate conditions are restored after this episode, growth rate is often accelerated and normal adult size can be reached; in other words, ‘compensatory’ growth occurs. Little is known about the long-term effects of this accelerated growth on reproductive performance and lifespan. In this talk, illustrated with data from manipulation experiments using three-spined sticklebacks, I will show how different early growth trajectories affected long-term fitness (= reproduction and lifespan). Additionally, I will illustrate how dynamic-state dependent models suggest that these effects could be due to the accumulation of physiological damage, and that this trade-off between growth tempo and damage level can influence optimal life-history strategies.</p>
<p><b>Gage, Timothy</b> Friday 15:10-15:30</p>	<p><b>Heterogeneity Identified at Birth, and Cardiovascular Risk at 45</b></p> <p><i>Timothy Gage, Erin O’Neill, Fu Fang, Greg DiRienzo</i></p> <p>Diastolic blood pressure (dBp) is a more significant cardiovascular risk factor prior to age 50-60 than systolic blood pressure (sBP). This paper determines if the latent heterogeneity identified in infant mortality using finite mixtures of regression (FMR) also predicts cardiovascular risk at 45 in the 1958 British birth cohort. FMR divides the cohort into “normal”, and “compromised” subpopulations. “Compromised” births display higher infant mortality, faster growth, and higher obesity. Analysis indicates that this latent heterogeneity significantly affects dBp and sBP. In females, the RR of high dBp for “compromised” births is 6.4 (hypertension) and 32.1 (stage two hypertension). Comparable results for sBP are 0.1, and almost 0.0. Results are consistent in males. Consequently “compromised” births are associated higher early and lower late cardiovascular risks compared to “normal” births. Selection on these subpopulations with respect to age and the historical decline in mortality influences the dynamics of cardiovascular disease.</p>

**Davison, Raziel**  
**Friday 15:30-16:10**

**Contributions of covariance: decomposing the components of stochastic population growth**

*Raziel Davison, H. Jacquemyn, F. Nicolè and S. Tuljapurkar*

Correlations between vital rates are common in nature and can have important effects on both evolution and demography. We develop new methods that precisely quantify contributions made by: (1) mean vital rates, (2) differential variability driven by environmental fluctuations, (3) correlations reflecting demographic tradeoffs and synchrony in stage transitions and (4) vital rate elasticities describing local selection pressures. We analyze empirical data on the endangered Lady's Slipper orchid *Cypripedium calceolus* growing in Poland and *Anthyllis vulneraria* (Kidney vetch) growing in Belgium. In *Cypripedium calceolus* we found that stochastic population dynamics were driven largely by differences in the survival payoffs to dormancy. Strong negative correlations imposed by the algebra describing dormancy transitions resulted in conjugate pairing of stochastic contributions that balanced one another and produced stationary population growth rates despite observed differences in the vital rates. In *Anthyllis vulneraria* we found that costs of reproduction reflected in negative correlations between survival and fecundity differed between populations and that the effects of these differences were mediated by local environmental conditions. In particular, we found that deeper soils buffered populations by decoupling negative correlations between survival and reproduction.

**Pringle, Anne**  
**Friday 16:30-17:10**

**Life and Death in a Petersham Cemetery: The Demography of Potentially Immortal Organisms**

*Anne Pringle*

Global change is reshaping the geographic ranges of organisms. Demographic processes will affect how fungi move and establish across landscapes, and data to parameterize basic models of population biology are critically needed. In October 2005 I began a survey of *Xanthoparmelia* lichens growing on tombstones of a New England Cemetery. Each year I recorded births and deaths, along with growth rates of established individuals. I am currently tracking near to 1,000 individuals. Lichens are an ideal demographic model because thalli are visible and easily counted. Although lichens are symbioses of fungi and photosynthetic microbes, in this work I am focused on the filamentous fungus enclosing the photobiont. I am using data to explore a series of fundamental questions, including: are birth rates equivalent each year, or are births more common in a subset of particularly favorable years? Is a fragmenting lichen senescing? How is reproduction apportioned across the lifespan of a lichen? Is the probability of death equivalent across years, and does the probability of death increase with age or size? I am also using genetic data to explore past demographic shifts in the populations of these species. Preliminary analyses suggest *Xanthoparmelia* lichens experienced a massive increase in numbers in the recent past, coincident with the advent of intensive farming across New England and construction of miles of stone walls. Births are not equivalent across years, and smaller lichens easily recover from mechanical damage and fragmentation. Reproduction increases with the size and age of an individual. Although lichens experience “infant” mortality, as lichens grow larger and older the probability of death decreases. Data suggest humans have influenced the population biology of *Xanthoparmelia* lichens since at least the 18th C, and that life history patterns of these modular, indeterminate organisms may be poorly served by traditional demographic models.

**Schaible, Ralf**  
**Friday 17:10-17:30**

**Hydra - life in the absence of senescence**

*Ralf Schaible, Alexander Scheuerlein, Daniel E. Martinez and Jim Vaupel*

We report results of a large-scale, rigorously standardized experiment consisting of two laboratory Hydra populations (~2000 individuals) that have been followed for about 6 years: Despite differences between cohorts the mortality in hydra is extremely low with annual average mortality ranging from 0.010 to 0.0018, meaning that only about one out of 160 individuals died in a given year. Additionally we observed no depression in budding indicating a constant and non-aging asexual reproductive rate. Drawing on published data that show instances of high mortality in hydra under specific environmental conditions we argue that the unique life history processes in hydra can be understood in terms of both the genet and ramet level. We argue that the specific environmental conditions at the ramet level promote the evolution of traits that overcome classical physiological constraints such as Hayflick limits to cell divisions, and Muller’s ratchet of increasing mutational load to generate an organism with negligible senescence.

**Danko, Maciej**  
**Friday 17:30-17:50**

**What makes Hydra immortal? A theoretical approach**

*Maciej Danko, Jan Koziowski, Ralf Schaible*

In comparison with other metazoans, hydras do not appear to undergo senescence. Senescence results from an increasing-with-age imbalance between damage and repair. We propose that hydras control their damage accumulation mainly by apoptosis and cell sloughing. Hydra can be treated as a large single pool of three types of stem cells with some features of differentiated cells. Elimination of damaged cells from a body reflects the relative dynamics of cell proliferation, migration, differentiation and cell death. The large pool size of stem cells in hydras prevents from a “cellular drift”. Such “drift” is inevitable in complex metazoans that can be viewed as conglomerates of differentiated cells and numerous but small and isolated pools of stem cells. We examine our hypothesis with a model that combines cellular damage with stem cells renewal and differentiation. Results favor the view that non-senescence is possible only in simple hydra-like metazoans.

## Poster Abstracts

<b>Thursday 1750-1850</b>	<b>Posters are displayed in the Institute's lobby</b>
<b>Castilho, Lucio Vinicius</b>	<p>Poster: <b>Environmental and genetic manipulations of rates of aging in model systems.</b></p> <p><i>Lucio Vinicius Castilho</i></p> <p>Environmental and genetic manipulations modulate longevity in model systems, but their effect on rates of aging is still controversial. Widespread assumptions are that (i) temperature changes rate of aging in all examined species; (ii) dietary restriction changes baseline levels of mortality and produce fully reversible effects, but leaves the aging process unaltered; (iii) insulin mutants mostly mimic the effects of dietary manipulation. Here I define a framework for comparative analyses of rates of aging under environmental and genetic manipulation and exemplify its use through a review of studies of model systems. Against common generalizations, results show that environmental and genetic effects on rates of aging are strongly dependent upon species, sex, mutations and experimental design. I conclude that claims (frequently based on data from other mammalian systems) about possible outcomes of factors such as dietary restriction on human aging must be relativized.</p>

Mumby, Hannah

Poster:  
**Effects of climate on survival of Asian elephants (*Elephas maximus*)**

*Hannah Mumby, Alexandre Courtiol, Khyne U. Mar, Virpi Lummaa*

Climate change has intensified interest in understanding how climatic variability affects animal life histories. Despite this, little is known of their effect on survival in those species. Asian elephants (*Elephas maximus*) are endangered across their natural distribution, and inhabit regions often characterized by high seasonality of both temperature and rainfall. We investigated the effects of monthly climatic variation on survival and causes of death in Asian elephants of all ages and both sexes, using a unique demographic dataset of over a thousand semi-captive longitudinally monitored elephants from four sites in Myanmar between 1965 and 2000. Temperature had a pronounced effect on survival, with the lowest predicted survival during the hottest and coldest months in both sexes across all ages. Because during a year the elephants spent twice as long in temperatures higher than their optimum (24°C) rather than temperatures below it, most deaths occurred during the “too hot” rather than the “too cold” period. Decreased survival at higher temperatures resulted partially from increased deaths from heat stroke and infectious disease, whilst the lower survival in the coldest months is associated with an increase in non-infectious diseases or poor health in general. Variation in survival was also related to rainfall with the best survival rates during the wettest months. Our results show that even the normal-range monsoon variation in climate can exert large impact on elephant survival in Myanmar leading to large absolute differences in mortality, particularly among the youngest age classes. The persistence of a long-term trend towards higher global temperatures combined with the possibility of higher variation in temperature between seasons may pose a growing challenge to the survival of species such as the endangered Asian elephants.

<p><b>Pietrzak, Barbara</b></p>	<p>Poster:  <b>Does the water flea age faster under perceived predation risk?</b></p> <p><i>Barbara Pietrzak, Piotr Dawidowicz, Maciej Dańko</i></p> <p>Several environmental interventions have been shown to affect Daphnia lifespan, biotic interactions being the most interesting. Quantity of food available affects lifespan in a hormetic manner, with animals mildly restricted living longest. Not only do the food conditions experienced by focal individuals matter, but also those experienced by their mothers. Then, Daphnia living under the conditions of perceived predation pressure (chemical cues) live shorter than Daphnia not experiencing that risk. The shortening of lifespan under the above mentioned conditions is seen as the consequence of different patterns of energy allocation early in life. It is often assumed that this shortened lifespan is a result of faster aging, although no formal analysis has previously been performed to test this. We present novel analysis of new and published data to test the hypothesis that Daphnia living under different biotic conditions age at different rates. Mortality data are fitted to Gompertz models and the best fit values of parameters a and b are statistically contrasted.</p>
<p><b>Ringelhan, Felix</b></p>	<p>Poster:  <b>Aging in a metagenetic basal metazoan – a biodemographic study with eleutheria dichotoma</b></p> <p><i>Felix Ringelhan</i></p> <p>Basal metazoans show large variations in life-cycle patterns, offering great opportunities to study the evolution of aging from a biodemographic point of view. Comparing age-specific survival and reproduction in experimental studies and analyzing demographic data across various basal metazoans, this dissertation project aims to shed light on different resource allocation strategies regarding aging. Laboratory experiments were conducted with Eleutheria dichotoma (Cnidaria: Hydrozoa: Cladonematidae), a metagenetic hydrozoan with a crawling medusa, to test the resource allocation flexibility of a remarkably plastic and variable marine organism. For the first time ever in a longitudinal experiment, the survival, size, and reproduction of isogenic Eleutheria were measured at different feeding regimes for both polyp and medusa stages. Eleutheria medusae have the ability to reproduce both via asexual budding and sexual self-fertilization. The polyps stay asexual, growing as stolonial colonies with the ability to form medusa as buds. Hence, Eleutheria clonal or inbred lines may reach substantial longevities over evolutionary time scales. It will be most interesting to compare polyp with medusa demography and to see if senescence is induced via sexual reproduction in medusae or if polyp colonies of this species senesce at all. The results of this project may broaden our understanding of the genet/ramet complex and its evolutionary demographic implications for the diversity and evolution of aging patterns in metazoans.</p>



<p><b>Wisser, Oliver</b></p>	<p>Poster:  <b>The risk taking pensioner</b>  <b>A modeling approach on how behavior shapes mortality patterns at old ages</b></p> <p><i>Oliver Wisser</i></p> <p>In 1825, Benjamin Gompertz developed the law of mortality, <math>\mu(x)=aebx</math>. This function describes the accelerating damage process by every increment of age. In biodemography, the parameter a has been commonly referred as the extrinsic risk of mortality, and the parameter b as the rate of aging that is due to deterioration of physiological function. Although the law of mortality appears to fit human mortality very well, it has its limitations in capturing mortality that is not due to aging. Consequently, in 1867 William Makeham added a constant parameter c to the law of mortality. I will argue that c is not constant over age. Furthermore it interacts with the aging process and affects the mortality trajectory at old ages. Traffic accident data from Germany for calendar years 1991 to 2009 is used to show that the risk of being involved in an accident decreases exponentially over age. The current model provides a good explanation of each parameter. In a future perspective it could shed light on and how the rate of aging might be different between sexes, countries or over time.</p>
<p><b>Zarulli, Virginia</b></p>	<p>Poster:  <b>Mortality shocks and the human rate of aging</b></p> <p><i>Virginia Zarulli</i></p> <p>Investigating the effect of mortality shocks on humans is difficult in the absence of the possibility to set up laboratory experiments. However, some events in the human history serve as natural experiments. This paper aims to analyze whether sudden changes in external conditions affect the slope of the mortality curve or shift the curve upwards proportionally at all ages. Two cases of natural mortality experiments are presented and used for the analysis: Australian civilian prisoners during WWII in a Japanese camp and the Ukrainian Famine in 1933. The death rates of the POWs were higher during the imprisonment but the slope of the curve appeared not to have changed compared to the normal mortality regime. During the Ukrainian Famine, instead, the curves for different years of famine converged at old ages. The results found evidence that selection could be the cause of convergence. The analysis suggests that sudden, not selective and transitory exposure to severe conditions shifts the mortality curve upward proportionally at all ages.</p>

<p><b>Salguero-Gomez, Roberto and Jones, Owen</b></p>	<p>Poster:  <b>Life and death in the garden: trajectories of mortality and recruitment in plants.</b></p> <p><i>Roberto Salguero-Gomez and Owen Jones</i></p> <p>One group of 'classical' theories of senescence focus on the existence of trade-offs between early and late life performance. This literature, initiated by Hamilton and later supplemented by Williams, Medawar and Kirkwood, all have unitarian (non-modular) organisms in mind. These theories are usually interpreted to predict that, after the age of maturity, survival will decline alongside reproductive output. Using data derived from the COMPADRE (COMparative Plant &amp; Algae Demographic REsearch) database, we examine age-specific survival and reproduction trajectories for ~400 plant species using a matrix modeling approach. We demonstrate the existence of an astonishing variety of demographic trajectories, the majority of which do not adhere to the 'classical' Hamiltonian predictions of inevitable senescence. We argue that environmental filtering and phylogenetic ancestry explains much of the observed variation, and that the 'classical' theories of senescence are not sufficient to explain demographic trajectories in non-unitarian organisms.</p>
<p><b>Levitis, Daniel</b></p>	<p>Poster:  <b>Human post-fertile lifespan in comparative perspective</b></p> <p><i>Daniel Levitis, Oskar Burger and Laurie Bingaman Lackey</i></p> <p>There has long been interest in comparing the extent of women's post-fertile survival to that experienced by other species. This has classically been done by taking the subset of each population that survives well past her last birth, measuring the length of her individual post-fertile period, and taking a mean for each population. This method has many flaws, particularly in a comparative context, and has impeded understanding of the evolution of post-fertile survival. We employ instead a measure called Postreproductive Representation (PrR), that proportion of cohort adult-years lived which are post-fertile. PrR allows for direct comparative examination of post-fertile survival across populations and species. We find that while nearly all species have the capacity for significant PrR (more than expected based on demographic stochasticity) most express this capacity only in artificially safe environments. Human experience higher PrR under even unnaturally harsh environments than do any other primates even in unnaturally safe environments. While most species possess post-fertile viability, humans are the only primate to exhibit a post-fertile stage of life across environments.</p>

<p><b>Danko, Maciej</b></p>	<p>Poster:  <b>The b-hypothesis: Do humans share the same rate of aging?</b>  <i>Maciej Danko, Jutta Gampe and Jim Vaupel</i></p> <p>The Gompertz hazard <math>\mu(x) = a \exp(bx)</math> fits human mortality well up to about age 85, and deceleration in death rates at higher ages can be captured by introducing individual heterogeneity. Heterogeneity in <math>a</math> leads to a mortality plateau at extreme ages, which is suggested after age 110 from analysis of data on supercentenarians, while heterogeneity in the rate of aging <math>b</math> cannot produce a mortality plateau. Here we estimate a model with individual heterogeneity in <math>a</math> and <math>b</math> to identify the amount of variation in both parameters that is compatible with mortality data from several countries in the Kannisto-Thatcher database.</p>
<p><b>Davison, Raziel</b></p>	<p>Poster:  <b>Allocation drives senescence in a model butterfly</b>  <i>Raziel Davison</i></p> <p>Mortality across the life cycle may be influenced by environmental and genetic factors as well as physiological and demographic tradeoffs at different ages. We develop a state-based nutrient and behavioral model that examines the mortality effects of allocation decisions across the adult life cycle of the butterfly <i>Speyeria mormonia</i>. Adults forage on nectar to obtain carbon required for reproduction and maintenance but fecundity is strongly limited by nitrogen stores accumulated during the larval stage. With the assumption that daily nutrient dynamics depend on foraging return rates and reproductive investments, we predict optimal foraging and reproductive behavior for model cohorts that begin adult life with different carbon and nitrogen stores and that experience different mortality responses to starvation (in our model, carbon depletion). We then predict mortality rates across the adult life cycle for cohorts optimizing their allocation schedules. Even without age-dependence imposed in the model, optimal allocation decisions produce a signal of senescence as individuals discontinue foraging to invest heavily in late-life reproduction despite high survival costs. Our findings suggest that allocation decisions can be strong drivers of observed age profiles of mortality, especially in short-lived animals with high costs of reproduction.</p>

**Schaible, Ralf**

Poster:

**Demography of the long-lived Ocean Quahog *Arctica islandica***

*Ralf Schaible, Eva Philipp, H. Gruber, Alexander Scheuerlein, Fernando Colchero*

The ocean quahog *Arctica islandica* is the longest-lived complex animal reported so far, which makes it a unique model to understand life-history patterns in long-lived organisms. We examined differences in size, age and growth patterns between living females and males of the ocean quahog in two natural habitats - Baltic Sea and Iceland - with extremely different environmental conditions (salinity, temperature). Random samples of nearly 600 individuals of both populations were collected in February and March 2010. *A. islandica* from the Baltic Sea population showed a shorter lifespan of 36 years, whereas an age of 241 years was found for the Iceland population. In both populations, males and females became mature at the same age. However, older ages in the Iceland population were dominated by females. The results of the growth rate analysis indicated that individuals in the Baltic Sea incur in trade-offs between investment in shell weight and shell height.