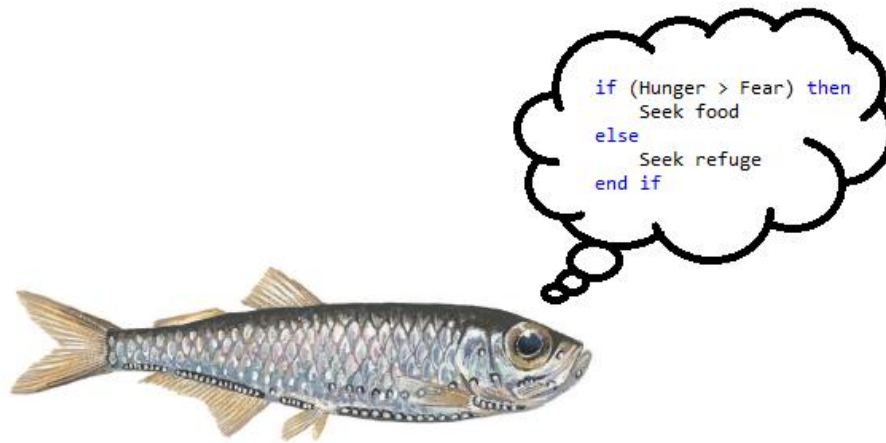


Effects of genetic complexity and time resolution in an individual-based model of mesopelagic fish with hunger and fear



Master of Science in Biology: Biodiversity, Evolution and Ecology

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Takk til

Først av alt vil jeg få takke min veileder Jarl Giske for en fantastisk innsats. Du har vært en inspirasjon til å stå på og du har gitt meg utrolig mange gode tilbakemeldinger. Jeg setter enormt pris på alle timene du har brukt på å lære meg fortranprogrammering og svare på mine utallige spørsmål.

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Sammendrag

Studier har vist at emosjonssystemer brukes for å avgjøre adferd, noe som nylig har blitt undersøkt i en individbasert, matematisk modell for vertikalvandring i planktivor, mesopelagisk fisk. Individene i modellen veksler mellom å være i tilstandene “Redd” eller “Sulten”. Tilstanden avgjør hvordan de evaluerer omgivelsene sine og dermed også adferden. For å undersøke gyldigheten av resultatene fra denne modellen har jeg lagt til muligheten for økt presisjon i de nevrale responsfunksjonene ved å legge til flere gener, samt utført en sensitivitetsanalyse på tidsoppløsningen.

Resultatene mine viser at nivået av frykt ikke stabiliserer seg selv i veldig lange simuleringer eller simuleringer med økt tidsoppløsning og at simuleringer som er kjørt med de eksakt samme parameterne ikke konvergerer når det gjelder nivå av frykt. I tillegg finner jeg at den totale eggproduksjonen i modellen er følsom for tidsoppløsningen i hver døgnsyklus, men ikke av antall døgnsykluser. Når tidsoppløsningen økes, veksler individene oftere mellom å være “Redd” og “Sulten” og har kortere kontinuerlige perioder med frykt i forhold til den totale livslengden sin. Alle egenskaper (kroppsmasse, mortalitet, fekunditet og dybdevalg) utenom frykt var stabile etter bare 2000 generasjoner.

Når den genetiske kompleksiteten i modellen økes, utnytter fiskene dette i særdeles liten grad. Det eneste unntaket er under predatorangrep. Denne muligheten for mer avanserte responser på ytre stimuli gav ingen økning i den maksimale eggproduksjonen eller hvor fort denne ble oppnådd. Hvor mye den økte genetiske kompleksiteten ble utnyttet varierte mye mellom populasjonene og var ikke konsistent for de forskjellige genene i modellen. I likhet med tidligere versjoner av denne modellen ble det evolvert flere ulike løsninger i samme populasjon.

Modellen kan derfor kjøres med høyere tidsoppløsning per døgnsyklus for å forbedre resultatene, men færre døgnsykluser for å spare regnetid. Antall generasjoner kan også reduseres kraftig, med mindre det er nivå av frykt som skal undersøkes. Økningen i genetisk kompleksitet viser seg å være uproblematisk for antall generasjoner som trengs for å finne frem til en god løsning, men er ikke nødvendig før arkitekturen i modellen og dens miljø er gjort mer komplisert.

Abstract

Many studies have shown that emotion systems are in use to direct behavior, which has also been utilized in a recent mathematical model. To investigate the general validity on the results from this model for vertical migration in planktivorous, mesopelagic fish, I increased the potential for precision in the neuronal response functions (by adding more genes) and executed a sensitivity analysis of the temporal resolution. I found that many aspects of the model were sensitive to the temporal resolution, especially when this was decreased. With fewer generations, it is possible to shorten simulation time by more than a factor of 10, but higher temporal resolutions gave better results. More complex neuronal responses did not seem to improve growth or survival.

The increased genetic complexity did not incur longer time to find adaptive solutions in the model. One reason for this may be that the extra genes were only partly utilized to perform more complex neuronal responses by the simulated populations. It was found that for almost all stages in life, the increased genetic complexity was not needed. The usage of more genes to make more complex response curves to their perceptions varied between populations and was not consistent for the genes involved. Complex response curves were mostly used for increased ability to have gradually increasing responses at either very low or very high perception strengths, while still maintaining a sigmoid shape of the curve. As for previous versions of the model, several solutions evolved in the same population.

Adding more time steps in each diel cycle gave a significantly higher population egg production at the highest resolution, while adding more diel cycles in a generation did not yield any differences. At the lowest temporal resolution, reducing the number of diel cycles gave lower population egg production. Total amount of fear during a lifetime varied considerably among simulations in each experiment, but showed no specific pattern in regard to temporal resolution. However, the relative length of continual periods of fear were longer in simulations with low resolution in each diel cycle and decreased as the resolution increased. The levels of fear in the different experiments did not stabilize or converge between simulations, opposite to the case for all other traits. Not even after 120,000 generations did levels of fear stabilize or converge between simulations.

Contents

1	Introduction.....	1
1.1	Background.....	1
1.2	Emotions, global organismic states and survival circuits	3
1.3	Comparison of human and non-human animals.....	6
1.4	Approach through modeling	8
1.5	Hypotheses.....	9
2	Methods	12
2.1	The model.....	12
2.2	Changes to the previous version of the model	14
2.2.1	Sensitivity analysis of temporal resolution	14
2.2.2	Complex neuronal responses	17
2.3	Programs and language.....	19
2.4	Data processing	19
2.5	Statistical analysis.....	19
3	Results	21
3.1	How does the theory of emotion systems apply to fish? - Literature review on the emotion system in fish.....	21
3.2	Sensitivity analysis of temporal resolution	24
3.2.1	Hypothesis 1: Sensitivity after 1000 generations.....	24
3.2.2	Hypothesis 2: Convergence of fear between experiments	30
3.2.3	Hypothesis 3: Egg production insensitive to temporal resolution	36
3.2.4	Hypothesis 4: Duration of fear periods insensitive to temporal resolution.....	44
3.3	Complex neuronal responses	46
3.3.1	Hypothesis 5: Complex neuronal responses improve fitness	46
3.3.2	Comparison of all simulations	49
3.3.3	Comparison of five simulations through life	52
3.3.4	How the increased genetic complexity are utilized by the populations	58
3.3.4.1	Choosing GOS – Emotional appraisal	58
3.3.4.2	Choosing depth.....	60
4	Discussion	63
4.1	Optimization, adaptation and emotion systems.....	63

4.2	Stabilization of GOS levels	65
4.2.1	Natural instability	65
4.2.2	Model-dependent instability	66
4.3	Increased genetic complexity	66
4.4	Emotion systems in fish	68
4.5	Sensitivity analysis of temporal resolution	69
5	Recommendations	72
6	References	73
7	Appendices	82

1 Introduction

1.1 Background

Modeling behavior in ecology goes all the way back to Lotka (1907; 1925). He introduced an equation for fitness maximization whereby anatomy, life history and behavior can be evaluated with a common currency. This modeling paradigm assumed that organisms make optimal decisions and was developed further into Life History Theory by Fisher (1930). In the 1960's this paradigm gave rise to Optimal Foraging Theory (Emlen, 1966, MacArthur and Pianka, 1966) and further developments of Life History Theory (Murdoch, 1966, Williams, 1966). Optimization is a method for mathematical simplification of a problem, based on that natural selection will evolve a solution towards an optimum, although the optimum itself may never be reached (Fisher, 1930). However, while alternate forces operate on the shaping of the gene pool, organisms themselves are triggered by proximate events and processes. In the following quote from Lotka's book, to illustrate that humans are not guided by rate of increase in body mass, he pointed out:

“What guides a human being, for example in the selection of his activities, are his tastes, his desires, his pleasures and pains, actual or prospective.” (Lotka, 1925, p.352)

McNamara and Houston (1986) and Mangel and Clark (1986) stressed that the temporal resolution in the early models was too coarse and that other methods were needed to study dynamic variation in behavior. Their methods, based on dynamic optimization, further developed the concept of a common currency to compare costs and benefits from actions of different impacts on fitness. However, these mathematical methods still did not take the proximate constraints of observation and decision making into account (Fawcett et al., 2013). Also, dynamic optimization models do not include density-dependent or frequency-dependent aspects of fitness (Giske et al., 1998). While ecologists separate age-dependent (Lotka, 1925, Fisher, 1930), state-dependent (Houston and McNamara, 1986, Mangel and Clark, 1986), density-dependent (Fretwell and Lucas, 1970) and frequency-dependent (Maynard Smith and Price, 1973) motivations for behavior by different methodologies, the instantaneous combination of these motivations seems

possible for the animals themselves. Mendl et al. (2009) pointed out that the emotion system which uses reward/punishment mechanisms in directing behavioral decisions, is the way evolution developed an operational common currency for animals. Based on this knowledge, modeling behavior by use of an emotion system is reasonable. Exactly what kind of emotion system a certain animal possesses and how these are to be modeled, are yet not well known.

The assumption of optimality in the early models was a way to simplify the reality, as pointed out by Lotka in the opening quote, in order to find an analytical or simple numerical solution. To find optimal solutions by use of the Euler-Lotka equation is a simplified method for finding the best life history that evolution over time will arrive at, but unlike the assumptions of these early models of animal behavior, all solutions evolved in nature have been under some proximate constraints. In a complex environment, a very large brain would be needed to encounter all situations in an optimal way, and this would come at great expense (Giske et al., 1998, Nilsson, 2000). It has also been shown that animal behavior is not always optimal (Pompilio and Kacelnik, 2005, Henly et al., 2008, Pavlic and Passino, 2010), thus deviating from the predictions of rational choice theory (Fawcett et al., 2014), a phenomenon that has been termed outcome bias (Marshall et al., 2013). This makes much more sense in an evolutionary perspective since it is impossible for an animal in a partly stochastic world to have inherited an ability to foresee the optimal response to every situation it can possibly encounter (McNamara and Houston, 2009, Goldstein and Gigerenzer, 2011).

‘Rules of thumb’ are therefore much more realistic to have evolved to handle the large amount of incoming information. The behavior may not be optimal, but will work more like a general rule and do well over a large range of situations, including those never encountered before (Hutchinson and Gigerenzer, 2005). Individual differences in behavior have also been observed to be consistent over time and situations. This has been referred to as syndromes, personalities and behavioral types (Sih et al., 2004, Bell, 2007, Dingemanse et al., 2010).

An emotion system is thus an evolved version of a ‘rules of thumb’ or a ‘common currency’ system when evaluation outcome from behavior (Cabanac, 1979, Leknes and Tracey, 2008). It describes the path from receiving information, through motivation and state of the individual, to a

physiological and behavioral outcome (Panksepp, 2005, de Waal, 2011). The role of an emotion system is to help an organism in surviving and reproducing by serving different adaptive purposes (LeDoux, 2012b). The exact understanding of an emotion in an emotion system is, on the other hand, more uncertain. It is important to note that the use of the term ‘emotion system’ in this text does not refer to a physical part of a brain. Rather, an emotion system is a system for adaptive responses to stimuli at the organismal level.

1.2 Emotions, global organismic states and survival circuits

Emotions can be explained as activity in the brain that takes over the usual perceptions of an individual’s surroundings (Panksepp, 1998) or a state of the brain that changes the evaluation of stimuli (Kalueff et al., 2012). While the attempts are numerous, there is no objective definition or consensus of the term emotion (Izard, 2010a, LeDoux, 2012a). This has led to different usage of the term emotions in the scientific literature. Some use it almost as a synonym for feelings (Kittilsen, 2013). This assumes a degree of consciousness, i.e. awareness of own thoughts (Mendl and Paul, 2004). Consciousness in different groups of animals is a widely discussed topic, but it is not necessary to have conscious experience to possess an emotion system (Mendl et al., 2011, Rose et al., 2014). The two major competing theories in the field of emotions disagree in this aspect (Panksepp, 2003). While the theory of Panksepp (1998) states that consciousness is obligatory for emotions, the theory of LeDoux (2012b) is independent of consciousness. This means that unconscious animals can have emotions, according to LeDoux, while Panksepp presupposes affect, the ancient form of consciousness that at least is shared by all mammals, an aspect that LeDoux think is a minor detail of the field of emotion research.

The word emotion, a keyword in modern psychological theory, suffers from having many different meanings in the scientific literature (Izard, 2010b). Through the history of the word, starting in the 17th century, it has been associated with the entire aspect of human mental life (Dixon, 2012). This broad use is carried on by psychologists and behavioral neuroscientists today and it is not likely that it can be defined in a way that is adequate for all fields where it is currently used (Izard, 2010a). Some have suggested to avoid the word and some try to distinguish between the many usages of it (Dixon, 2012).

In a theory for fear LeDoux (2012b) does not use the word ‘emotion’ due to these historical constraints (see Dixon, 2012). Instead he introduces the concepts of ‘survival circuits’ and ‘global organismic states’ (GOS). These concepts do not require emotions and hence the problem of confusion is avoided. He points out that reactions by bacteria to both beneficial and harmful stimuli (Macnab and Koshland, 1972) are evolutionary precursors and the neurobiological foundation for what humans today experience as emotions. Our experiences of these emotions and our consciousness have evolved gradually since early forms of life. Similarities in neurochemistry and behavior between vertebrates and invertebrates are indications of the early origin of brain systems that are functionally much alike when it comes to processing stimuli. The arguments for this will be laid out below.

The theory by LeDoux (2012b) does not need any conscious experience by the organism (LeDoux, 2014) and emotions are not necessary when addressing survival circuits and GOS. I am not stating that one of the theories of LeDoux (2012b) or Panksepp (1998) are more correct than the other, but my work is more consistent with that of LeDoux (2012b) which has been expanded by Giske et al. (2013) to also include a circuit for hunger, not just fear.

The GOS in the theory by LeDoux (2012b) is a state of the whole organism where the individual allocates the resources of its brain, and hence also its other physical resources, towards a challenge or opportunity. By entering the GOS, attention towards relevant stimuli are enhanced while other less relevant stimuli are more or less ignored (LeDoux, 2012b). An example of the consequences of attention can be lower feeding efficiency (Lastein et al., 2008), but better chances of survival when in an afraid GOS (Ashley et al., 2009, Braithwaite and Boulcott, 2007).

A survival circuit, as one of the core components of the theory by LeDoux (2012b), is an important factor that contributes to activate the GOS. A survival circuit is a process that serves an adaptive purpose by restricting attention and arousing the relevant parts of the brain and body (LeDoux, 2012b). There exist survival circuits for all basic life-sustaining mechanisms. Some of these are feeding (Figure 1), thermoregulation and reproduction (LeDoux, 2014). Survival circuits, as with most other neurological mechanisms, are most widely studied in mammals, were they are highly conserved between species. At least to some extent, precursors to survival circuits

with similar functions exist in invertebrates (Bateson et al., 2011), but the organization may be different (LeDoux, 2014).

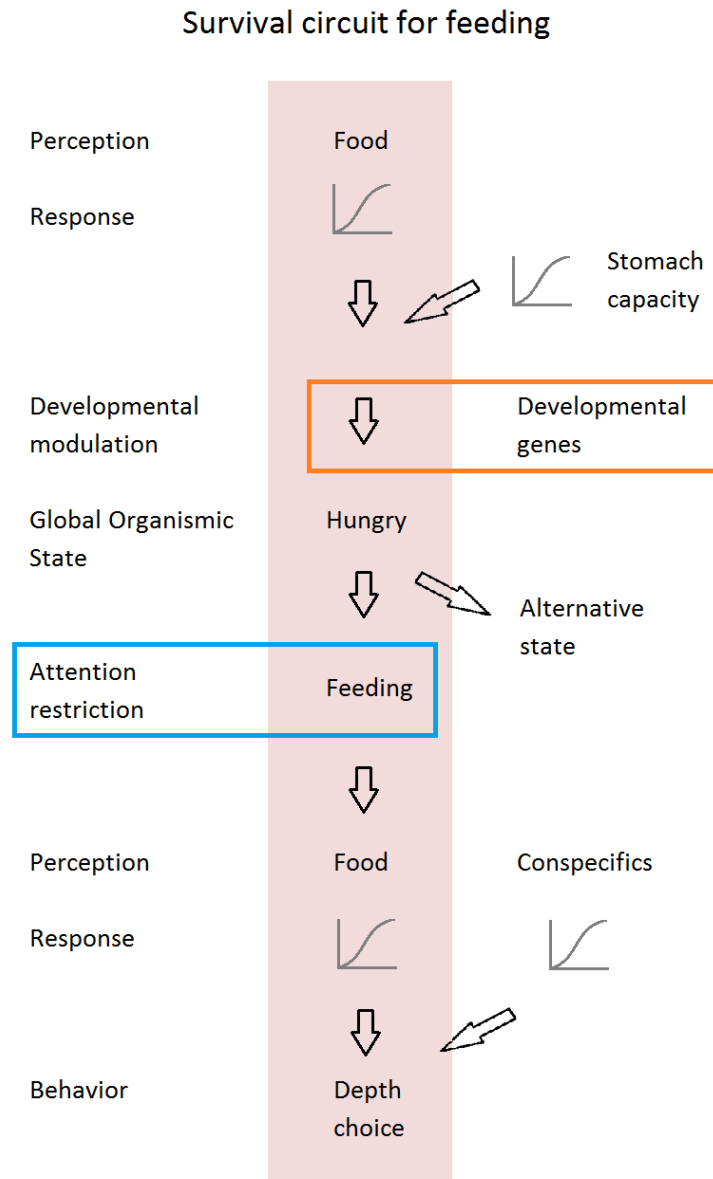


Figure 1: The survival circuit for acquiring nutrition as it is represented in the model used in this study. Area shaded in red represents the survival circuit. The perception of food is processed twice in the model, both when going into a GOS and when producing behavior. When going from perception to behavior, the circuit interacts with responses from other perceptions (stomach capacity and conspecifics), developmental genes, competing GOS and restriction of attention set by the chosen GOS. In the GOS ‘Hungry’ the attention is towards feeding while the attention towards predators is low, hence increasing the risk of being eaten. An explanation for the stages in the survival circuit is to the left in the figure and interactions with other circuits are to the right.

1.3 Comparison of human and non-human animals

The concept of emotion is described from the human experience, but it is difficult to account for emotions in other animals even though this system originated from a more basic form already in bacteria (Macnab and Koshland, 1972). There is good evidence that mammals are in possession of an emotion system (Bekoff, 2000, Cabanac et al., 2009, LeDoux, 2012b) and that our behavior is primarily controlled by this (Panksepp, 2005). Several types of evidence support this.

Brain anatomy is important for comparison of human and non-human animals. The size of the brain in relation to body size, the encephalization quotient, is a good indicator of the mental capacity of a species (Herculano-Houzel, 2007, Cabanac et al., 2009). When looking at this quotient, there is a large difference between ectotherms and endotherms, as warm blooded animals have a brain that is about 10 times larger (Cabanac et al., 2009). This leaves a distinction between Amniota (mammals, reptiles and birds) and Lissamphibia (modern amphibians). In the amniotes, a new structure also emerges in the brain, the cortex (Aboitiz and Zamorano, 2013). This new component gave a new level of complexity to the animal brain. Humans share this brain structure with the rest of the amniotes, although there are large differences in architecture between reptiles and mammals (Aboitiz et al., 2003), making it more plausible that these animals experience emotions in a similar way as humans. The emergence of emotions in the transitions between amniotes and Lissamphibia is also supported by Rial et al. (2008).

The discovery of the limbic system in non-human animals is important for comparing emotions in humans and non-human animals (Kittilsen, 2013). This is due to the function of the limbic system in motivated behavior and emotions in primates (Ono et al., 2000). The olfactory limbic system, which is tightly coupled with the emotion system (Reep et al., 2007), has been suggested to be the driving force of vertebrate brain evolution (Jacobs, 2012). This is due to the importance of odors when navigating in space, a property found in organism of all complexities, including insects (Strausfeld et al., 2009) and fish (DeBose and Nevitt, 2008).

In terms of behavior, both mammals and birds produce many of the same responses when handled as humans do when experiencing an emotion (Rial et al., 2008). Among these responses are tachycardia and fever. The same responses are not observed in fish or amphibians according

to Cabanac et al. (2009). Other behaviors that are coupled with emotion states are observed in several other mammals, e.g. facial expressions (Evers et al., 2014) and vocal expressions (Paul et al., 2005).

Another way to compare mental capacities is to look at neurochemical similarities between species (O'Connell and Hofmann, 2011). Dopamine, serotonin and opioids are associated with emotion systems in humans and are highly conserved in evolution (Mustard et al., 2005, Iliadi, 2009, Curran and Chalasani, 2012). These chemical components are used in punishment and reward in the brain and function as a way of modifying behavior. The presences of these substances in other species are at least an indication of the presence of an emotion system in the form of utilizing a common currency.

A major difference between emotion research in humans and non-human animals is that humans can verbally communicate their experience (Braithwaite and Boulcott, 2007). One recent example of this is a study by Nummenmaa et al. (2014) where they found bodily sensations to have a consistent pattern when experiencing a basic emotion (referring to the theory by Ekman (1992)). This is an indication from human research that such emotions have distinct representations in the body, that emotions (or emotion systems) activate certain physiological bodily components.

Most of the research done in the field of emotions is conducted on mammals. Due to the large phylogenetic divergence between mammals and fish it is not likely to see the same structures (which we do not) of survival circuits. On the other hand, the principles of organization may be preserved. This is at least the thought behind survival circuits, they do not have to be looked at in a different way when working with different animals (LeDoux, 2012b). How the theory of emotion systems apply to fish will be treated independently as a literature review in the results section.

Experiments with honeybees have revealed that these invertebrates display a cognitive bias (Bateson et al., 2011), meaning that their behavior indicates an effect of an emotional state preventing the most rational behavior (Mendl et al., 2009). This evidence for emotional states in invertebrates is as good as those for vertebrates (Mendl et al., 2011), where the number of studies

on different species are numerous (Enkel et al., 2010, Doyle et al., 2011). It is important to note that this does not refer to conscious emotions, but behavioral and physiological changes due to the possession of an emotion system (Paul et al., 2005, Mendl et al., 2009). Even though the brain of an invertebrate is very small compared to a vertebrate, recent studies show that the cognitive abilities of some insect species and other arthropods go beyond what previously acknowledged (Giurfa, 2013). This includes highly contingent integration of multiple sensory inputs in crabs (Hazlett and McLay, 2000), attention direction by fruit flies (van Swinderen and Greenspan, 2003), application of general rules to new situations by honey bees (Srinivasan, 2010) and trade-offs between speed and accuracy in vision of bumblebees (Dyer, 2012), suggesting sophisticated cognitive processes. In this text, cognition refers to the whole process of acquisition, processing, storing and acting on information (Shettleworth, 2009).

1.4 Approach through modeling

To investigate effects of an emotion system on behavior, a model including an emotion system has been developed for fish (Giske et al., 2013). The model represents the ocean's midwater planktivore *Maurolicus muelleri*, Müller's pearlside, but the focus here is not on the specific species. This species was originally chosen because of the knowledge of the behavior and life history of the organism based on good field observations (e.g. Rasmussen and Giske, 1994, Kristoffersen, 2007) and theoretical models (Giske and Aksnes, 1992, Rosland, 1997, Strand et al., 2002).

However, the limitations to the Giske et al. (2013) model are many. One is that each individual is modeled to live for only seven days with 200 time steps in each day. In contrast, *Maurolicus muelleri* may live for up to five years (Gjøsæter, 1981, Kristoffersen and Salvanes, 1998) and can make instantaneous decisions (Balino and Aksnes, 1993, Staby et al., 2011).

The phenotypes produced by the Giske et al. (2013) model are very consistent between simulations. Movement in the water column, body mass, death rates and fecundity show very little variation between independent simulations. On the other hand, the genetic composition and the distribution between the emotional states vary a lot among simulations (Giske et al., 2013).

This is due to the evolution of different shapes of the neuronal response functions (illustrated as sigmoid curves in Figure 1) among simulations. Hence, another limitation could be the shapes of these neuronal response curves, for which to our knowledge there exist no research. Therefore, the complexity of the neuronal response functions was increased. The genomic complexity of the organisms is thereby increased, measured by the amount of information that the genome encodes (Adami et al., 2000). It would then be interesting to see if there are evolved neuronal responses that utilize the possibilities of the increased complexity. Also, if there are changes in the consistencies of genotypes or phenotypes among the populations.

The type of fish (planktivorous mesopelagic) this model is built for have an estimated combined biomass of 1000 million tons and is a potential unused food resource (Kaartvedt et al., 2012, Irigoien et al., 2014). To have good models for this type of fish is of great importance for investigating both how to develop an economically profitable fishery and the ecological implications of such activity.

1.5 Hypotheses

The purpose of the simulation experiments in this study is to investigate the sensitivity of the results obtained by Giske et al. (2013) to the temporal resolution of the model, as well as to the formulation of the neuronal response function.

Giske et al. (2013) investigated emergent dynamics through 30 replicate simulations over 50,000 generations, each resolved into 7 diel cycles with 200 time intervals in each cycle. Each simulation lasted approximately 2 weeks at the FIMM cluster at *Parallab*, the supercomputer facility of the University of Bergen. The sensitivity analyses performed here will investigate whether it is possible to obtain reliable results faster. Since the model describes an evolving population, the ultimate measure of performance is the fitness obtained. However, fitness is a measure at the level of the individual or even the allele, so the total egg production of the population is the nearest available measure of the performance of the evolving population. The question is if population egg production can be found in experiments with shorter simulations and also whether the egg production is sensitive to the resolution of days and time steps within days.

The model used here is scaled so the amount of food and the risk of being eaten are the same when the number of time steps in a generation is adjusted.

The time an individual spends in the GOS 'Afraid' has a fitness cost in lost feeding and growth. Therefore, it would be beneficial to reduce any unnecessary fraction of time steps spent in this GOS. It is possible that the simplification of seven diel cycles and only 200 time steps per cycle is an artificial limitation that forces individuals to be afraid for longer periods at the time than if the resolution of the model had been higher. This is based on the thought that the individuals do not need to be hungry so often when they can make decisions more frequently. I will therefore investigate this by both a decrease and increase in the number of diel cycles and the number of time steps in each cycle from the previous standard.

Giske et al. (2013) also found that the level of fear in populations did not converge. They claimed that this was a consequence of the evolutionary freedom given by the architecture, which means by many different possible pathways from sensory perceptions to behavior that can make the individual either move up or down. However, there are also alternative explanations to the phenomenon. One is that the temporal resolution was not good enough, another that simulation time was not long enough, and a third that the mathematical formulation of the neuronal response was too coarse, so that the organisms were not always able to make the best decisions. Both Giske et al. (2013) and my initial experiments showed rapid stabilization of population egg production. With a more accurate mathematical formulation of the neuronal response, it is possible that population egg production and other population traits will stabilize after fewer generations.

The neuronal response from a perception in Giske et al. (2013) was made by using one pair of genes producing one neuronal response function (examples of functions given in Figure 1). In their experiments, Giske et al. (2013) found large variation in the neuronal responses between individuals, but also variation between the different simulations. The reason could be that the best response was not possible to evolve with only one pair of genes available, i.e. the mathematical formulation of the response was not accurate enough. Therefore, the number of genes was increased from one to three gene pairs for each perception. More complex neuronal response

functions are thereby allowed to evolve in the populations. The question is whether more complex functions will evolve and how this will affect the ultimate measure of performance: population egg production.

To investigate these topics, I have therefore modified the code to allow other temporal resolutions and more precise neuronal response functions, and formulated these five testable hypotheses:

H1: The level of both fear and egg production in the population will be stable after 1000 generations in all experiments.

H2: The level of fear in a population will converge between simulations in all experiments.

H3: The population egg production is independent of the resolution of time.

H4: The duration of a continual period of fear is independent of the resolution of time.

H5: When individuals may use more complex neuronal response functions, they will tend to do so, with higher population egg production and shorter time to evolve it, as the result.

2 Methods

2.1 The model

The model used for the simulations is modified from Giske et al. (2013). Their model follows the concept of survival circuits and GOS of LeDoux (2012b), but excludes learning and enables the organisms to enter a GOS of either hunger or fear. When in one of these states, an individual concentrates its attention towards certain perceptions, but always maintains a minimum attention towards predators and food. Each perception P an individual receives of food, conspecifics, light, predators and its own stomach fullness is modulated into a neuronal response R by two genes, x and y in the 0.1 – 10.0 range:

$$R = \frac{(P/y)^x}{1 + (P/y)^x} \quad [1]$$

For each type of perception there is an own x , y and R . This makes 18 genes for the nine responses. The responses have an additive effect on one of the GOS and their collective strength is further modulated by genes for development, D . Each individual have four D -genes with values ranging between 0 and 1. A value of 1 means full focus on food and a value of 0 means full focus on fear. Which gene that is in use is determined by body mass. Gene D_4 is used at maximum body mass and gene one when the mass is zero. Genes D_2 and D_3 are used at 1/3 and 2/3 of maximum mass, respectively. D -values of body masses in between two of these four are found by linear interpolation. Which one of the GOS an individual enters, is determined by the stronger of Equations 2 and 3.

$$Hunger = D \times (R_{A_{stomach}} + R_{A_{food}}) \quad [2]$$

$$Fear = (1 - D) \times (R_{A_{light}} + R_{A_{predators}} + R_{A_{conspecifics}}) \quad [3]$$

R indicates a neuronal response for stomach capacity, food, light, predators and conspecifics. The subscript A indicates that it is used in emotional appraisal, which is the calculation of Equations 2

and 3. Fish evaluates the available depths according to the attention in their current GOS. They can choose from depths ranging from two depths above to two depths below the current depth. The depths are evaluated by two neuronal responses, based on GOS, and it moves to the depth that maximizes the net neuronal response (Equations 4 and 5).

$$\max_{z-1,z,z+1}(R_{H_{food}} - R_{H_{conspecifics}}) \quad [4]$$

$$\max_{z-1,z,z+1}(R_{F_{conspecifics}} - R_{F_{light}}) \quad [5]$$

In Equation 4, H indicates the GOS ‘Hungry’ where the response from food has a positive effect and conspecifics have a negative effect when evaluating depths. The F in Equation 5 indicates that the individual is frightened (GOS ‘Afraid’) where the response from conspecifics has a positive effect and light has a negative effect when evaluating depths.

For description of environment and reproduction, see Appendix A.

If the number of individuals in a population goes beneath a certain level (1500), a restart is initiated. The restart creates some random individuals and some copies of the remaining ones. This is to avoid a population to die out and is often necessary in some of the early generations of a simulation.

In this text, the word fecundity is used as the total egg production of the population in a generation.

2.2 Changes to the previous version of the model

2.2.1 Sensitivity analysis of temporal resolution

In earlier versions of this model, each generation has gone through seven diel cycles consisting of 200 time steps, 1400 time steps in total. By varying the number of diel cycles and number of time steps per cycle, the life length and the time resolution are adjusted. An increase in the parameters means longer computation time and a decrease can alter the results from the model. Both parameters are increased and decreased in a set of experiments to investigate the effects.

Predation risk due to predatory attacks had to be modified so it would be possible to scale the length of the attacks between the versions with different amount of time steps in each generation. Instead of having an attack increasing the risk in two time steps and then give full risk for a specified number of time steps, the model was adjusted to calculate the length of an attack based on the total number of time steps (for code see Appendix B.2). The length now varies from one to 36 time steps. The risk for an attack occurring and the background predation risk are also scaled to the number of time steps per generation.

Other parameters in the model are also scaled to the number of time steps per generation. These include the amount of food available, the energetic costs of living and the rate of digestion. For food, energetic costs, digestion and predation risk, this is done by dividing their value by the total number of time steps in a generation (*'span'* in the Fortran equations below). This results in an equal amount of predation risk and food in the different experiments along with a scaled metabolic rate. How these four variables are scaled is presented in the following four equations from the actual Fortran code (Equations 6, 7, 8 and 9):

Food encounter rate:

$$\text{didgain}(\text{dep}) = \text{parta}/\text{span} \quad [6]$$

Energetic costs:

$$\text{periodcost} = \text{fbodymass}(\text{ind}) * \text{flivingcost}/\text{span} \quad [7]$$

Rate of digestion:

$$\text{digestion} = \text{fstomach}(\text{ind}, 0) * 100./\text{span} \quad [8]$$

Predation risk:

$$\text{didrisk}(\text{dep}) = \text{autorisk} * \text{visr}/\text{span} \quad [9]$$

Here, *ind* is the current individual and *dep* is the current depth. *Didgain(dep)* and *didrisk(dep)* are the density independent gain and predation risk in the depth at this time step, respectively. The unscaled food encounter rate is *parta*, *periodcost* is the energetic costs for one time step, *fbodymass(ind)* is the body mass of the individual, *flivingcost* is a fixed parameter for the metabolic costs, *digestion* is the reduction in stomach content in one time step, *fstomach(ind,0)* is the stomach content of the individual at the start of a time step, *autorisk* is a fixed parameter for the level of predation and *visr* is the current visual range of the fish.

60 initial simulations with three different combinations of number of diel cycles and time steps were run over 20,000 generations to find out how long the simulations in the main experiments had to be to reach a stable solution. These were combinations with lower or equal number of time steps and diel cycles as the previous standard. There was little change in fecundity, mortality and level of fear after 3000 generations and therefore 4000 generations was set as length of the main experiments (Figure 2).

The data used for all analyses were only from the ‘Normal’ generations. This is the most common of the nine generation long fluctuation pattern (occurring in 20 % of the generations) and have both intermediate food concentration and predation risk in addition to a fixed number (four) of predator attacks starting when a fixed fraction of the time steps have passed. The standardization of the predator attacks in time is done make accurate comparison of simulations possible.

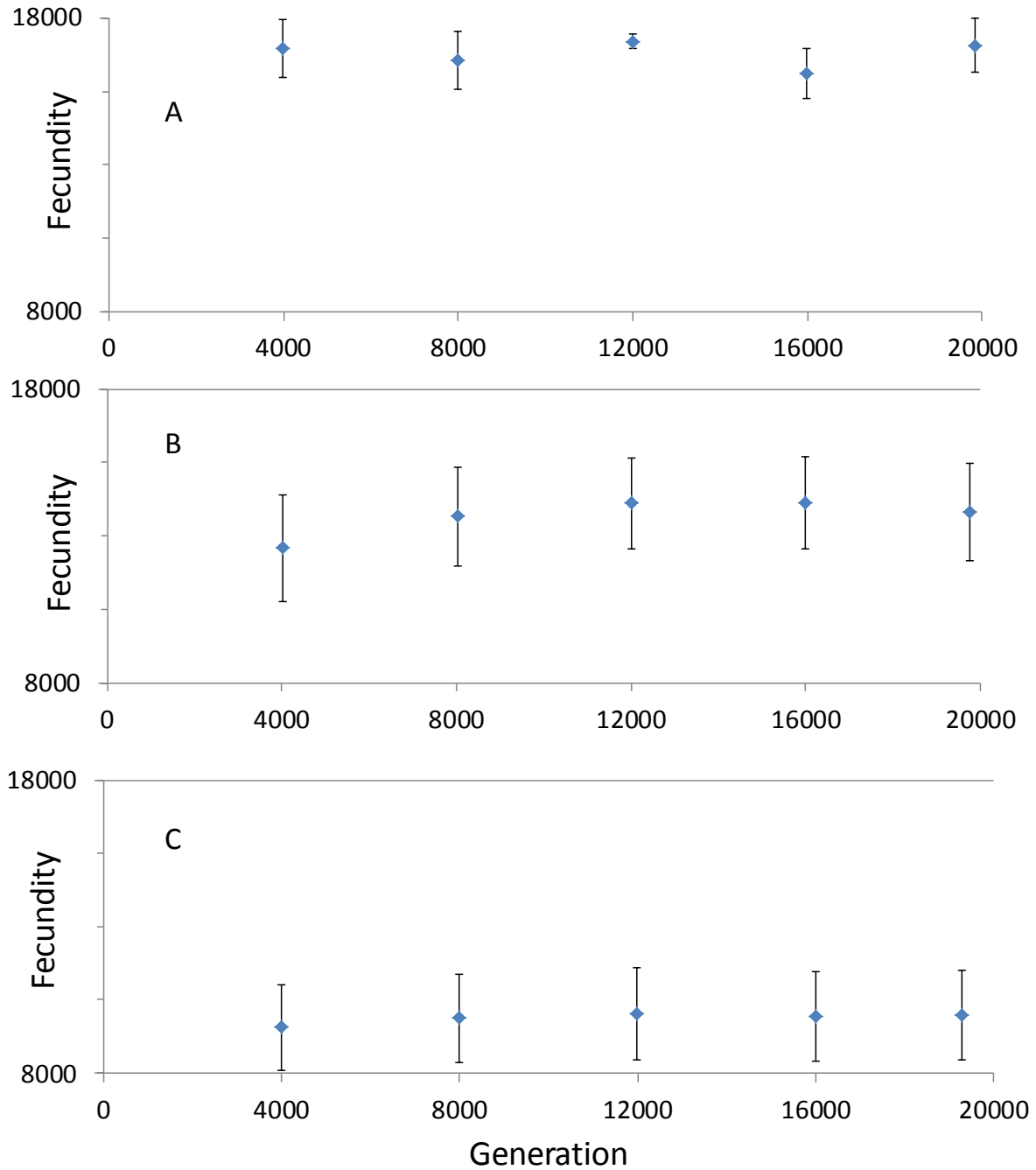


Figure 2: Fecundity in initial simulations. These simulations were executed to determine how long the main simulations had to be to achieve a stable solution. Data points show the average values of 20 simulations at intervals of 4000 generations. Horizontal error bars indicate a 95 % confidence interval of the mean. A) 7 diel cycles and 200 time steps per cycle (previous standard). B) 7 diel cycles and 100 time steps per cycle. C) 4 diel cycles and 100 time steps per cycle. Only 'Normal' generations were used for comparison of fecundities. 'Normal' generations are explained in Appendix A.1.

Nine experiments were then executed on the FIMM cluster at Parallab, the UiB center for high performance computing, operated by Uni computing. 50 simulations were run for each of 4, 8 and 16 diel cycles, each with 67, 200 and 600 time steps per diel cycle, making nine experiments with a total of 450 simulations. Later in this text, the experiments are referred to with an abbreviation, e.g. D4 TS200 for the experiment with four diel cycles and 200 tie steps per cycle. These nine experiments were run with three additive components of each neuronal response, see below. There were also two extra experiments with the same number of diel cycles and time steps as in Giske et al. (2013) (7 diel cycles and 200 time steps per cycle), but where one had only one additive component for each neuronal response and the other had three as in the nine other experiments. Each simulation is based on a different random number sequence, generated by a random seed generator at the start of a simulation, thus giving all simulations different initial gene values and different random fluctuations. The random number is saved for the opportunity to restart a simulation in the case of a crash, yielding the exact same result. All output files from all simulations are stored (3.5 TB).

In addition to these new experiments, some already available data from the old version of the model were used to investigate the stabilization and convergence of fear in longer simulations (hypothesis 1 and 2). The data are for 10 simulations that run for 125,000 – 180,000 generations. These simulations had seven diel cycles and 200 time steps per cycle and only one component for every neuronal response.

2.2.2 Complex neuronal responses

The responses produced by the three single neuronal response functions are added together (Equation 10), making up a new and possibly more complex function.

$$R = \frac{(P/y_1)^{x_1}}{1 + (P/y_1)^{x_1}} + \frac{(P/y_2)^{x_2}}{1 + (P/y_2)^{x_2}} + \frac{(P/y_3)^{x_3}}{1 + (P/y_3)^{x_3}} \quad [10]$$

The differences in the new and old neuronal responses are demonstrated in Figure 3. There are now responses available that was impossible to produce before, e.g. low but significant response

at low perceptions in combination with a highly increasing response when exceeding a certain value of perception (Figure 3, left).

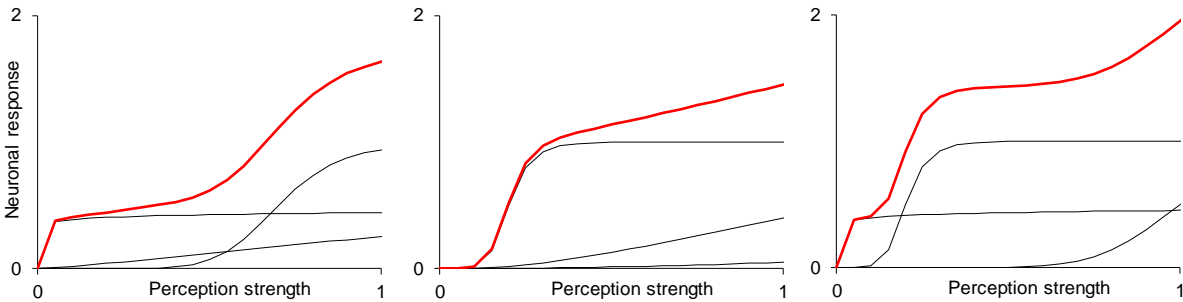


Figure 3: Examples of complex neuronal response functions (red) that can be produced by the new version where three single response functions (black) are added together.

After a simulation, the strength of each of the three components of each of the nine complex responses is ranked for each individual due to their strength through life. This is done by reading individual gene data and perceptions from the last generation and calculating the strongest of each component of each response of the individual in every time step. Each component is added up through life and ranked from strongest to weakest. The strongest component of each complex neuronal response function is denoted NR1. The way NR1 is calculated means that it does not have to be the function that gives the highest maximum response at all possible perception values. There could be ranges of perception where the generally weaker components give a higher response than NR1, e.g. at very low perceptions. Code for the calculation of NR1 is found in Appendix C.1.

Later in this text, the term ‘set of neuronal response components’ is used to indicate how many of the three components of the complex neuronal responses (Equation 10) that is utilized to determine the GOS. One set of neuronal response components refers here to the strongest components of all the five complex neuronal responses that contribute to the choice of a GOS. Two and three sets then refer to when the second and third components are included, respectively. (Code for these calculations is found in Appendix C.1.)

2.3 Programs and language

The code is written in Fortran 90 (.f90) and for debugging and compiling Parallel Studio XE with VS2010 was used. SSH Secure Shell and FileZilla 3.4.7.1 were used in transferring files from FIMM to own locations. Scripts for efficient transferring processes to FIMM by creating the required DOS code and altered parameter files, were written in MATLAB code (for code see Appendix H) using MATLAB R2012b. MATLAB code was also written and used in statistical analysis. Excel was used for some statistical analysis and for making most of the figures.

2.4 Data processing

When processing the data, some output files were run through a second Fortran program. The data used here were only from the “Normal” generations. A new subroutine had to be added to this program for calculations on the complex neuronal response functions and another had to be modified (for code see Appendices C.1 and C.2). The output from this program is designed to be inserted into Excel. One spreadsheet was created for each of the nine experiments in the sensitivity analysis, in addition to one for experiment comparison.

In the second Fortran program, the sensory information for all individuals in the last generation are read and the average strength of the strongest of the three neuronal response components for the five neuronal responses that determine GOS was calculated. The strongest neuronal response component (NR1) was determined by sorting them by strength due to the sensory input to that individual at its depth over its life time. For five of the 50 simulations the fraction of the complex neuronal response that was coming from NR1 was calculated for all nine neuronal responses. The fraction of total hunger or fear and fraction of the response towards the depth that were chosen due to the strength of the different neuronal responses, were also calculated.

2.5 Statistical analysis

When comparing the different experiments, a one-way ANOVA ($\alpha = 0.05$) was applied to data for mortality, fecundity, fear and body mass from the second Fortran program. Then a multiple comparison procedure was applied (multcompare), using Tukey’s honestly significant difference

criterion ($\alpha = 0.05$). These tests were done in MATLAB and produced the plots in the Figures 4, 5, 14, 15 and 16. The script can be found in Appendix G.2, procedure 1. The function 'multcompare' produces plots that compare the confidence intervals of different data sets with the response variable on the x-axis. The values in Table 1 are obtained by calculating linear least squares lines (lsline) (Appendix G.2, procedure 2).

95 % confidence intervals of the mean of all simulations were calculated at each 1000th generation for mortality, fecundity, fear and body mass by the second Fortran program (see Appendix C.2). These values were used to plot the mean and 95 % confidence intervals of the mean in Figure 8.

3 Results

3.1 How does the theory of emotion systems apply to fish? -

Literature review on the emotion system in fish

In an evolutionary perspective, emotions are likely to be present in fish as a common currency mechanism because of the advantages such a system gives in coping with the wide range of situations that an individual encounters during a lifetime (Braithwaite et al., 2013). It is also likely that emotion system that we see in mammals had a precursor in early vertebrates (Kalueff et al., 2012), indicated by the homologies in brain anatomy and neurochemistry (Chandross et al., 2004). A study on Atlantic salmon, similar to the one done on honeybees by Bateson et al. (2011), showed results of cognitive bias and hence indicating effects of an emotion state (Vindas et al., 2012). The last shared ancestor of honeybees and salmon is the last common protostome-deuterostome ancestor (PDA). The divergence of these two lineages have been estimated based on conservation of developmental regulatory principles and paleontological evidence to have started about 550 million years ago, but this estimate is very uncertain (Erwin and Davidson, 2002). The organismic complexity of the PDA varies depending on what the estimate of time of divergence is. Some argue for a more complex expression of genes (for use in more complex nervous system) (Hui et al., 2009), while others argue for a simpler construction of the PDA (Erwin and Davidson, 2002).

The size and structure of the brains of amniotes and other vertebrates, e.g. fish, are very different (Kotrschal et al., 1998). Some studies therefore state that fish are not in possession of an emotion system (Rose, 2002, Cabanac et al., 2009). There is an ongoing discussion on both whether and which parts of fish brains are homologous to the parts of the mammalian brain needed for an emotion system (Mok and Munro, 1998). An example of this is the mammalian amygdala, which is highly involved in the emotion system (LeDoux, 2000, LeDoux, 2003) and the reward system in the brain (Parkinson et al., 2001, Paton et al., 2006, Bermudez et al., 2012). There are different views on homologies to the amygdala in the brain of teleosts (Jesuthasan, 2012). One problem with identifying homologous brain structures between mammals and fish is that their

telencephalic development happens through inversion and eversion, respectively. This places similar structures in different locations in the adult brain (Yamamoto et al., 2007, Maximino et al., 2013). Despite of this, Maximino et al. (2013) summarizes the evidence for the teleost ventral subpallium and the dorsomedial pallium as homologous structures to the mammalian basolateral and central amygdala, respectively. These are mammalian structures processing information about fear and pain and expresses innate behavior and anxiety (Cheng et al., 2014). Structures like the extended amygdala have not been shown to have any homologies in teleosts (O'Connell and Hofmann, 2011).

While there are some differences in the anatomy and neurochemistry, the functional role of the emotion system can still be comparable to that of mammals (Vargas et al., 2009, Panula et al., 2010). The similarities implicate that fish also maybe utilize its emotion system as a common currency to guide the behavior when evaluating widely different perceptions thus simplifying and speeding up the decision process (Mendl et al., 2009). Fish brains produce many of the substances associated with the mammalian emotion system, like dopamine, serotonin and oxytocin/isotocin (Winberg and Nilsson, 1993, Thompson and Walton, 2004, Kittilsen, 2013). These neurochemical substances are involved in systems for reward and punishment in the brain and seem to be strongly conserved in evolution (Bonga, 1997, Tognoli et al., 2010, Sørensen et al., 2013). When treated with drugs that affect the emotion system, the effects in the form of behavior and physiological changes are quite similar between mammals and teleosts (Maximino and Herculano, 2010). However, this is not the case for all drugs tested (Sackerman et al., 2010), but when summing up results from different studies, Lillesaar (2011) concludes that for substances acting on the serotonergic system (relating to fear and anxiety behavior), the effects on related behavior were similar between mammals and fish.

When arguing for emotion systems in fish, it is also natural to look into implications for global organismic states (GOS). For example attention towards predators and prey are key components of survival and will most definitely be evaluated different (or ignored) when in different GOS. There are very few studies investigating GOS since this is still an emerging topic, but there are indications in some studies. Teleosts evaluate predation risk differently when either in pain (Ashley et al., 2009) or in a specific reproductive stage (Lastein et al., 2008), indicating a GOS.

Gravid female three-spined sticklebacks differ significantly from non-gravid females in their anti-predator behavior (Frommen et al., 2009). When directing attention towards one perception, attention to others must decline. This has been demonstrated by Braithwaite and Boulcott (2007) by showing that motivation towards feeding was low when the fish had just experienced pain.

In species where courting activity decides mating success, there is a trade-off between mating and predation risk (Magnhagen, 1991). This is the case for the two-spotted goby where males have to balance this trade-off throughout the breeding season. Early and in the middle of the season, competition between males is fierce and they exhibit bold behavior. At the end of the season there are much more females than males due to high male mortality and females starts competing for males (Forsgren et al., 2004). This shifts the trade-off for the males and they do not have to be bold to achieve mating (Myhre et al., 2012, Magnhagen et al., 2014). This change in boldness can be seen as a change in the state of the organism, but as stated above, this was not the aim of these researchers and therefore not emphasized in the studies. In this specific case it is not the aim of the organism (to reproduce) that is changing with its state, but how it balances its chances of survival while achieving reproduction.

Another aspect of emotion systems is how they manifest themselves in terms of behavior. The result from an emotion system is an advanced form of ‘rules of thumb’ that works well for many situations (Hutchinson and Gigerenzer, 2005). The behavior can be consistent over time and situations and include individual differences in a population. This is then referred to as behavioral syndromes (Sih et al., 2004), but has also been given other names in the scientific literature. Examples are coping styles (Koolhaas et al., 1999), temperament (Budaev, 1997) and animal personalities (Dingemanse et al., 2010). Behavioral syndromes have been found in a large range of animal groups, including teleosts, and that is important in this context (Sørensen et al., 2013) because behavioral syndromes imply limited behavioral plasticity in individuals and hence constraining behavior from being optimal (Conrad et al., 2011). The constraints can be genetic, but they can also be psychological mechanisms, e.g. learning (Fawcett et al., 2013). To determine whether it is genetic or physiological traits that gives a behavioral syndrome of a fish in nature is very hard to measure. When using models, all details are available. Therefore, models of high complexity that focuses on the mechanisms behind behavioral syndromes can be useful.

3.2 Sensitivity analysis of temporal resolution

The level of fear did not stabilize after 1000 generations in any of the nine experiments. The level of fecundity was much more stable, although in most experiments there was a significant difference between generations 1000 and 4000. Despite these statistically significant differences, the biological differences between generations 1000 and 4000 are very small. Levels of fear did not converge within experiments. Neither stabilization of fear level nor convergence between simulations were evident when examining data from simulations over 120,000 generations. Fecundity is found to depend on the resolution of time in each diel cycle. The length of continual periods of fear also becomes shorter when increasing this resolution.

3.2.1 Hypothesis 1: Sensitivity after 1000 generations

The percentage of time that females spend afraid show a larger change between early and late in evolution than fecundity did (Figures 4 and 5). Even though only two of the experiments do not have significantly different total population fecundity when comparing generations 1000 and 4000, the changes between these two stages in evolution are very small. This is due to the small variation between populations in this trait. When comparing fear at each 1000th generation, all experiments show significant difference between generations 1000 and 4000 despite a much larger confidence interval.

To compare change in fecundity and fear in females the percentage increase between generation 1000 and 4000 was calculated (Table 1). The percentage increase differs with about one order of magnitude between these two traits in most of the experiments. This shows that there is much more change in fear than in fecundity after generation 1000, even though fecundity also shows significant difference between generation 1000 and 4000 in most cases.

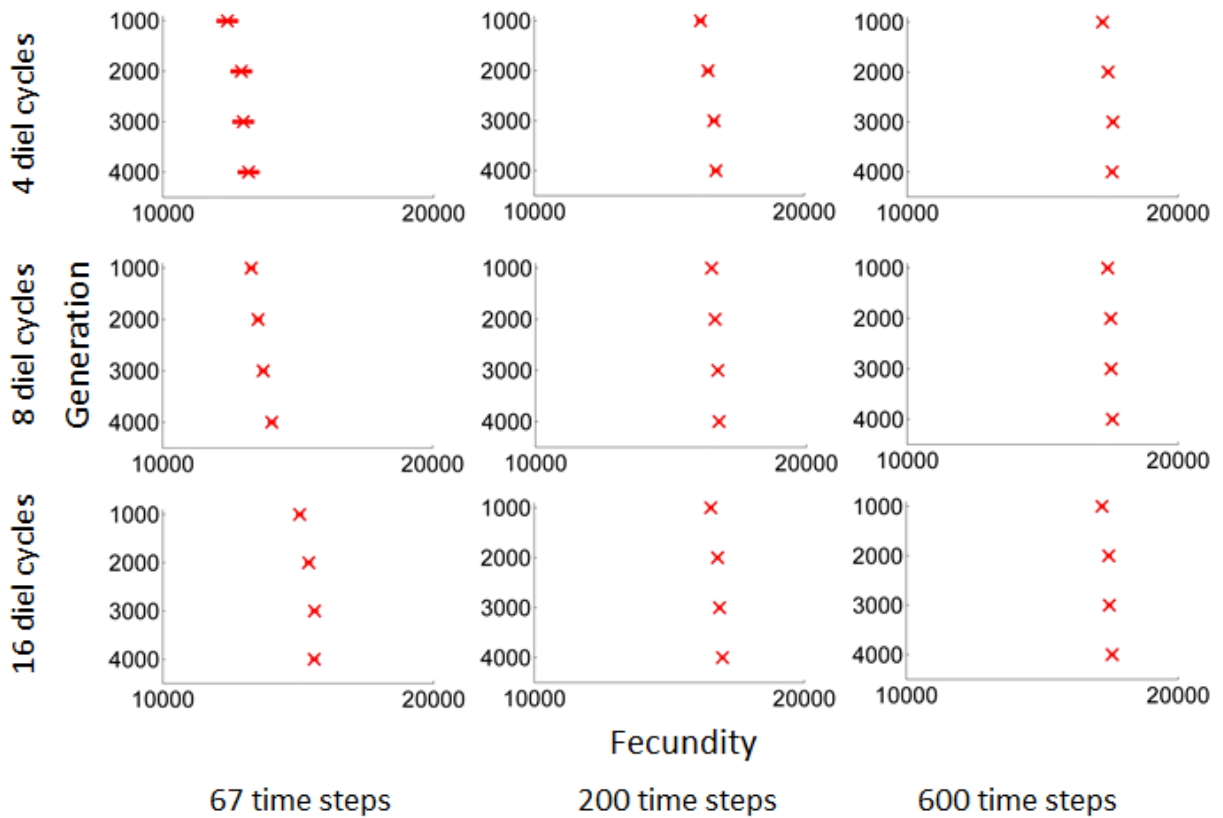


Figure 4: Statistical comparison within each of the nine experiments of total population fecundity after 1000, 2000, 3000 and 4000 generations ($\alpha = 0.05$). Horizontal lines indicate a 95 % confidence interval of the mean. These are present in all panels, but so small that they are hard to see. Only the experiments with four diel cycles and 67 time steps per cycle (top left) and 16 diel cycles and 200 time steps per cycle (bottom middle) do not have a significant difference between their fecundity after 1000 and 4000 generations. All other series show significant difference and increase in fecundity. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

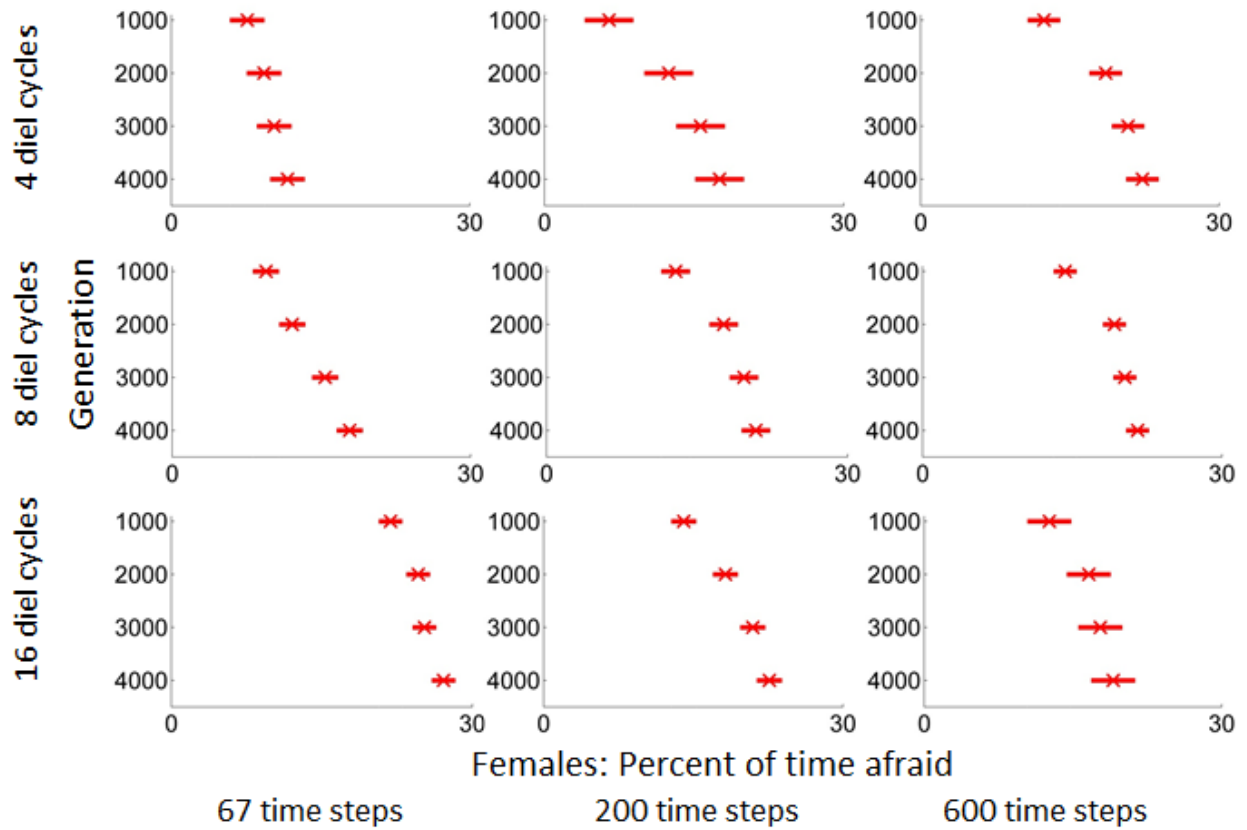


Figure 5: Statistical comparison within each of the nine experiments of percent of time in the state afraid after 1000, 2000, 3000 and 4000 generations ($\alpha = 0.05$). Horizontal lines indicate a 95 % confidence interval of the mean. All series show significant increase in fear between generation 1000 and 4000. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

Experiment	Mortality	Fecundity	Females: Percent of time afraid	Females: Final body mass
D4 TS67	1.175	5.820	50.834	1.175
D8 TS67	0.060	5.591	91.054	0.060
D4 TS200	-4.456	3.591	144.329	-4.456
D16 TS67	2.901	3.619	22.221	2.901
D8 TS200	-5.127	1.680	56.229	-5.127
D4 TS600	-5.084	2.154	69.694	-5.084
D16 TS200	-2.545	2.487	58.173	-2.545
D8 TS600	-4.410	0.924	44.407	-4.410
D16 TS600	-1.301	1.989	45.743	-1.301

Table 1: Comparison of change in mortality, fecundity, fear (females) and final body mass (females) after 1000 generations. Percent increase in average value among the 50 simulations from generation 1000 to 4000 in each of the nine experiments. Values are the percent increase according to the linear least squares line from generation 1000 to 4000 divided by the intersection of this line at generation 1000. The linear least squares line is based on the 50 observations at 1000, 2000, 3000 and 4000 generations. The values in this table therefore represent the relative change after 1000 generations and until the end of the simulations. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

These results show that fecundity is more or less stabilized after 1000 generations and at least after 4000 generations. This is also the case for the mortality rate and final body mass for females (Table 1). The level of fear in females does not stabilize to the same degree. The 10 simulations that were run for 125,000 - 180,000 generations (Figures 6 and 7) show that the levels of fear did not even stabilize after >120,000 generations.

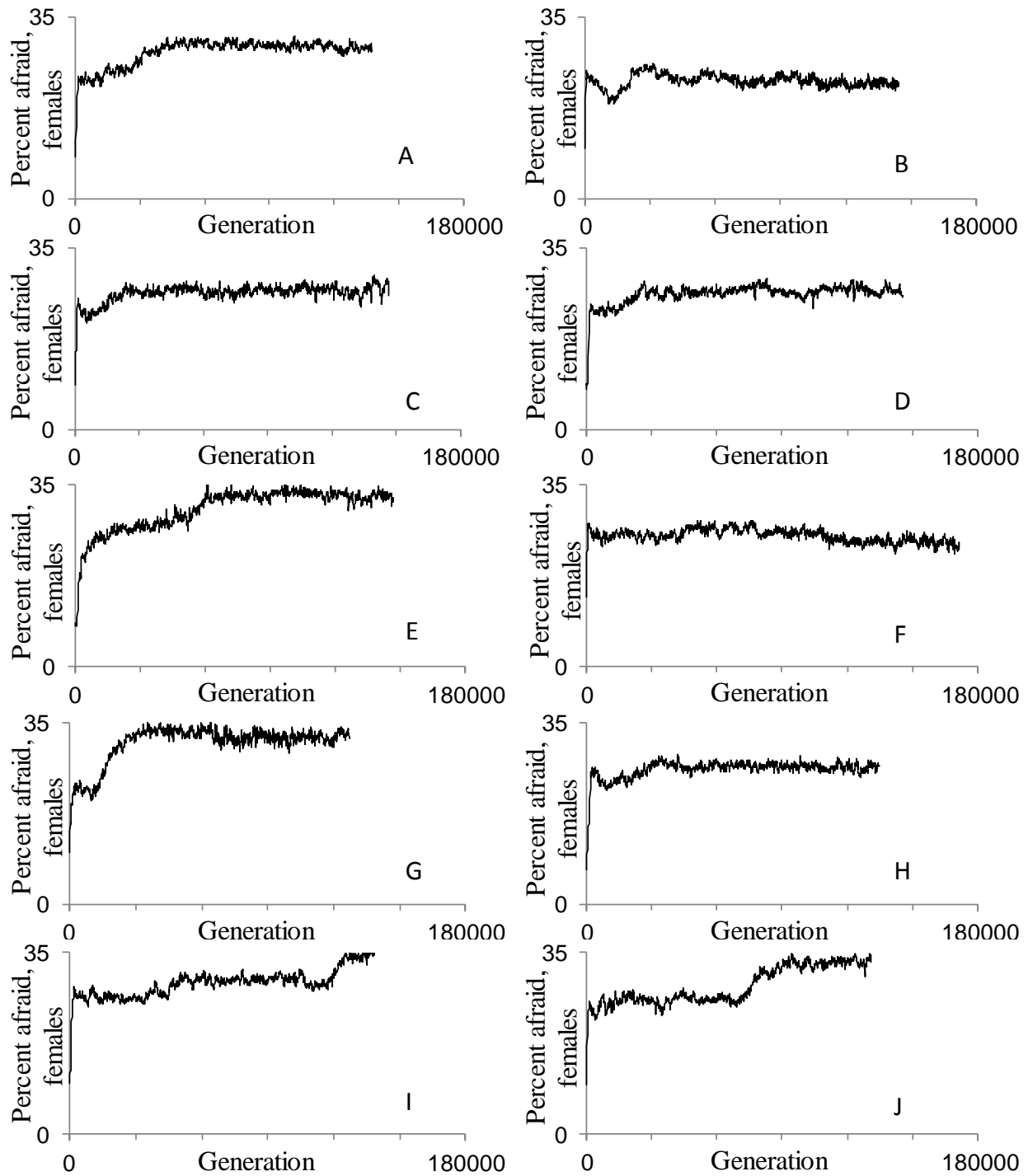


Figure 6: Percent afraid females in 10 simulations (denoted A-J) over >120,000 generations. These are the long simulations run with the Giske et al. (2013) version of the model. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

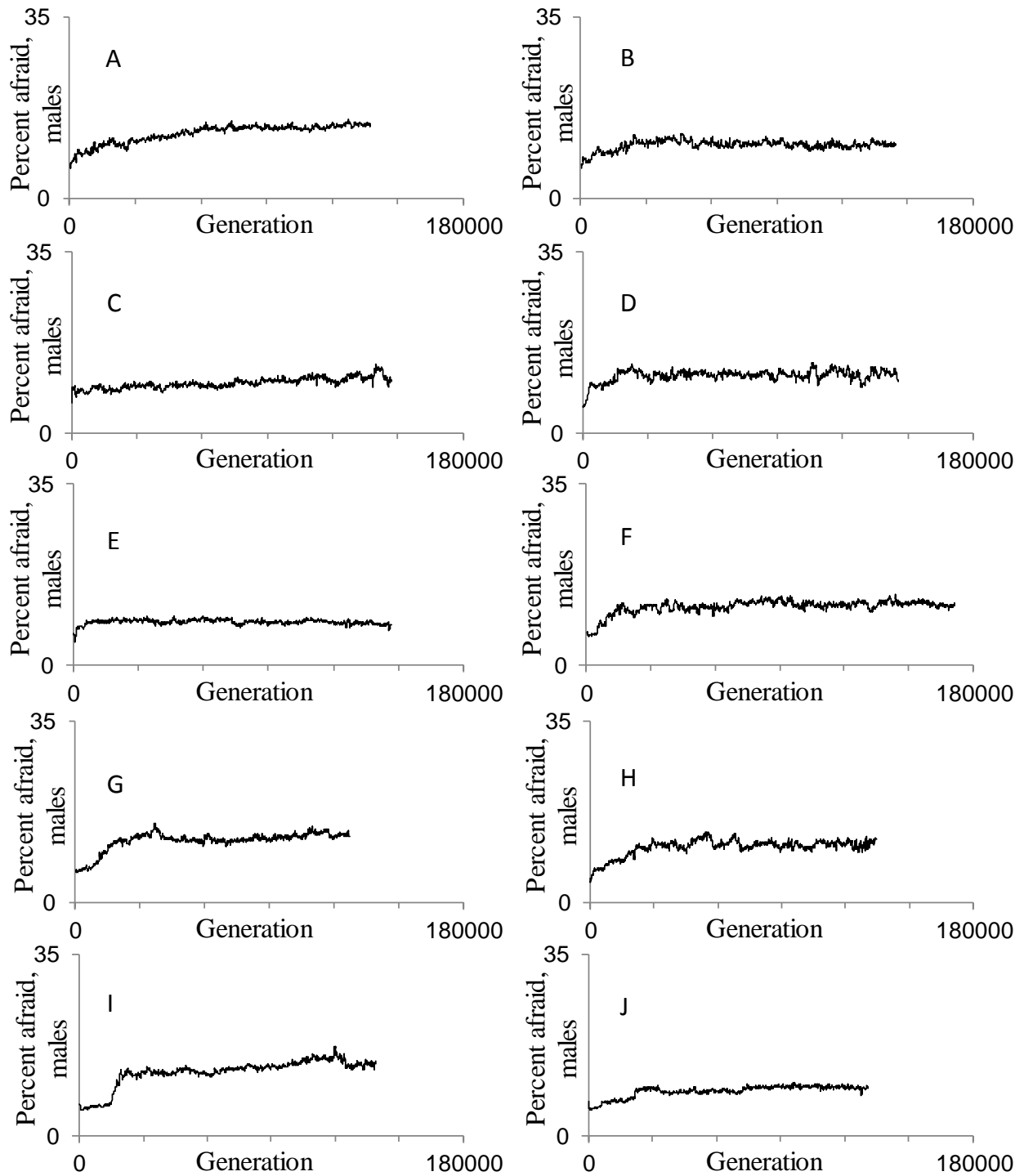


Figure 7: Percent afraid males in 10 simulations (denoted A-J) over >120,000 generations. These are the long simulations run with the Giske et al. (2013) version of the model. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

3.2.2 Hypothesis 2: Convergence of fear between experiments

The amount of variation of mortality, fecundity, fear and body mass within the nine series are very consistent through life. Horizontal bars (Figure 8) indicate 95 % confidence intervals of the mean. For females, the percent of time afraid (Figure 8 C) varies a lot between simulations. This variation diminishes through time, but is still much larger than for all the other traits. All eight other experiments show very similar patterns in both variation and time of stabilization of all traits (for equivalent figures to Figures 8 and 9 for all experiments, see Figures A5-A19 in Appendix E).

The distribution of death rate through life, final body mass and depth at reproduction for both females and males are very consistent between simulations after 4000 generations (Figure 9). However, the distribution of the frequency of the fraction of time spent afraid, varies much more between simulations (Figure 9 C). The level of fear does not converge between simulations in any of the nine experiments. Most of the other nine experiments show more or less the same pattern in regard to less consistency between simulations for time spent afraid than for other traits (for figures, see Appendix E). This lack of convergence of fear level between simulations can be due to the length of these experiments.

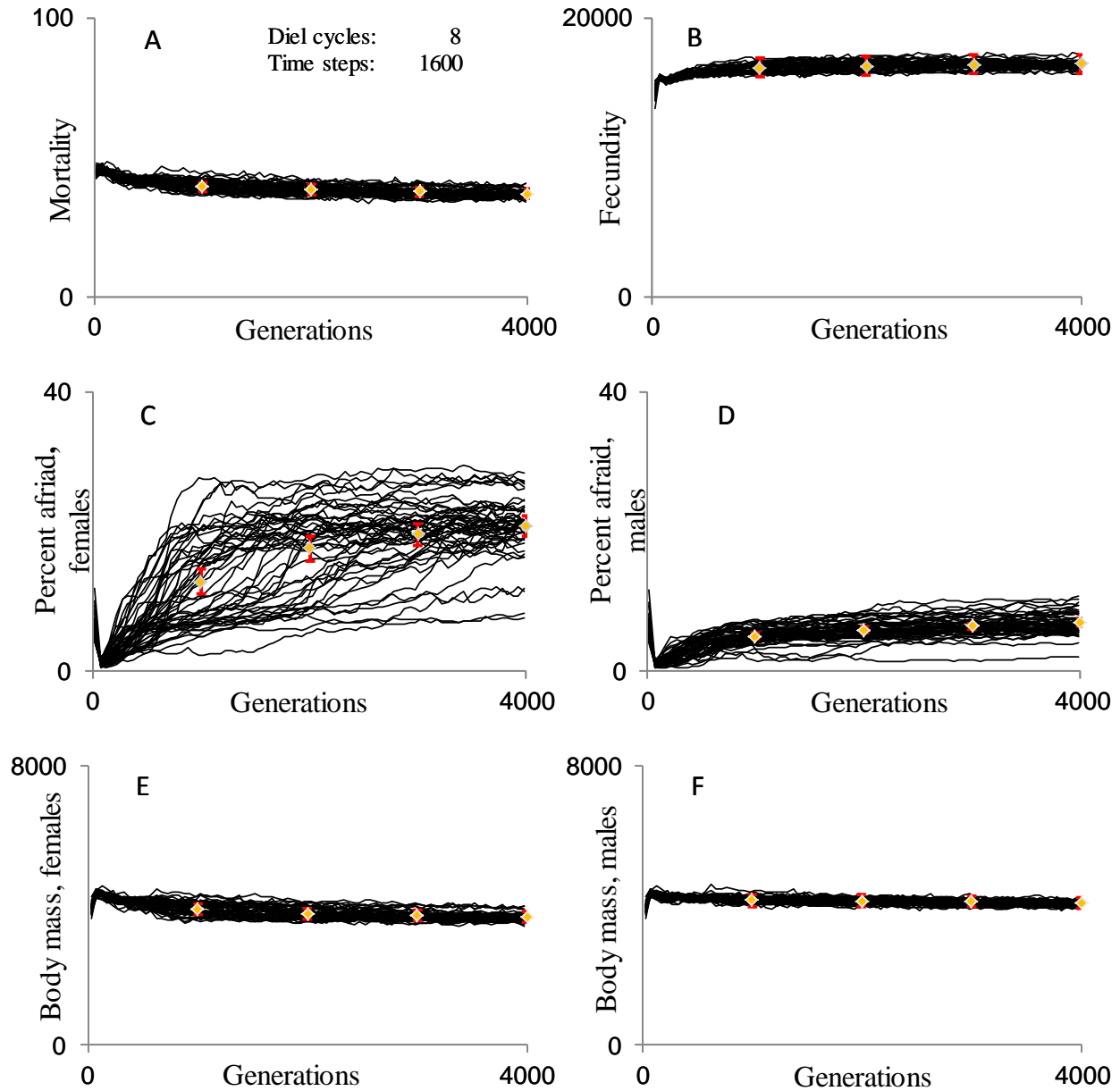


Figure 8: The average in each generation of mortality (A), fecundity (B), percent of time steps in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F) for all 50 simulations with 8 diel cycles and 200 time steps per cycle. The orange markers indicate the mean of all 50 populations at 1000, 2000, 3000 and 4000 generations. If this generation was not a 'Normal' generation, the first one after this was used. 95 % confidence intervals of the mean are marked by red bars at these four points. In some plots (e.g. D), these bars are almost covered by the orange marker. 'Normal' generations are explained in Appendix A.1.

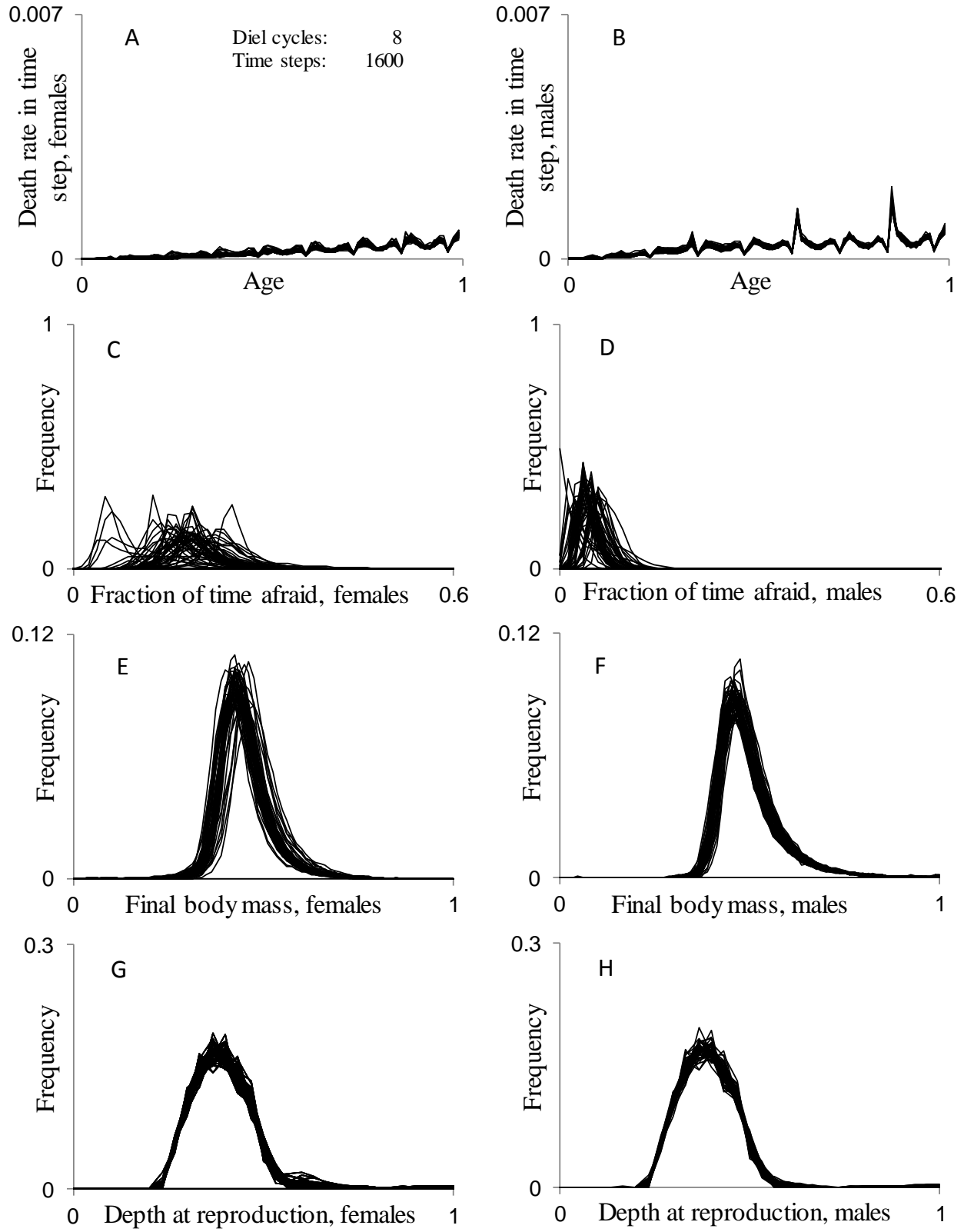


Figure 9, previous page: Comparison of the last 20 'Normal' generations in the 50 simulations in the series with 4 diel cycles and 67 time steps per cycle. A- B) Death rates through life for females and males, respectively. C- D) The frequency of individuals and what fraction of the time they spent in the GOS 'Afraid'. E-F) Body mass at the end of a generation for females and males, respectively. G-H) The depth at which individuals reproduce (i.e. are located in the last time step) for females and males, respectively. 'Normal' generations are explained in Appendix A.1. X-axes in the panels A and B, E and F and G and H are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively.

One experiment that stands out from the others when it comes to stabilization and variation, is the one with the fewest time steps in total: four diel cycles and 67 time steps per cycle (Figure 10). This experiment has a much less stable level of fecundity and mortality than the other eight. Many of the drops in fecundity represent the same simulation, but about half of the simulations have at least one drop. The simulations that have this sudden decrease in fecundity struggle at low fecundity for a few generations before a restart is initiated. This is not always the case, sometimes high fecundity is achieved again without a restart. The fact that there are such a large number of restarts late in evolution means that many of the simulations never become adapted. This result is expected because the same pattern of frequent restarts and drops in fecundity was seen in the initial experiments in the simulation series with 4 diel cycles and 100 time steps per cycle.

Since the levels of fear did not converge between simulations in any of the nine experiments, available data from longer simulations were used to compare levels of fear (Figure 11). After 120,000 generations there is still not convergence in level of fear between the 10 simulations studied (Figure 11 E and F). These data are from the version of the model used in Giske et al. (2013) and hence have only one component for each neuronal response function. However, this does not seem to affect the stabilization or convergence of fear levels. I therefore assume that longer simulations with three neuronal response components not would have given more stable or converging levels of fear either.

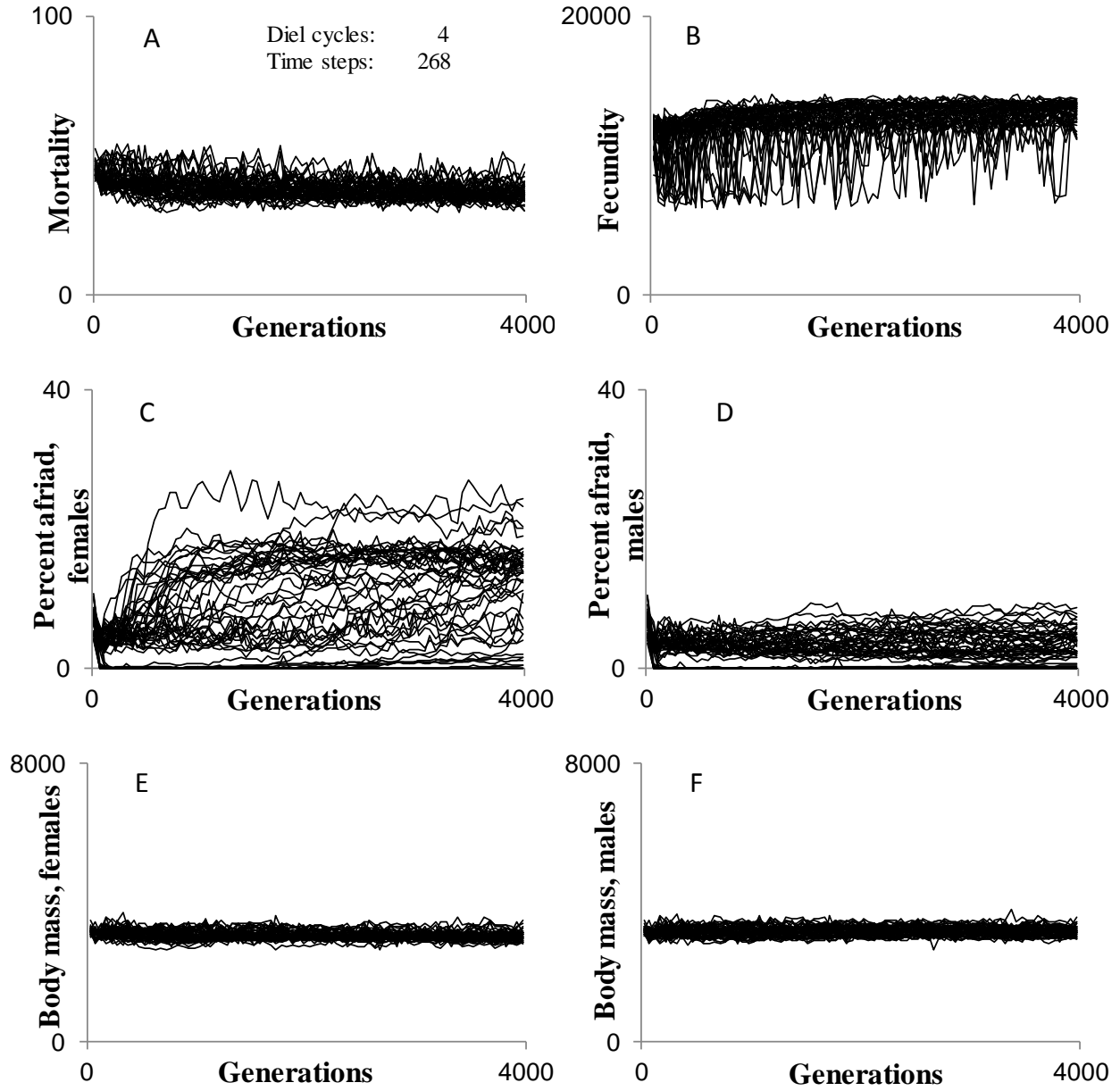


Figure 10: The average in each generation of mortality (A), fecundity (B), percent of time steps in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F) for all 50 simulations with 4 diel cycles and 67 time steps per cycle. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

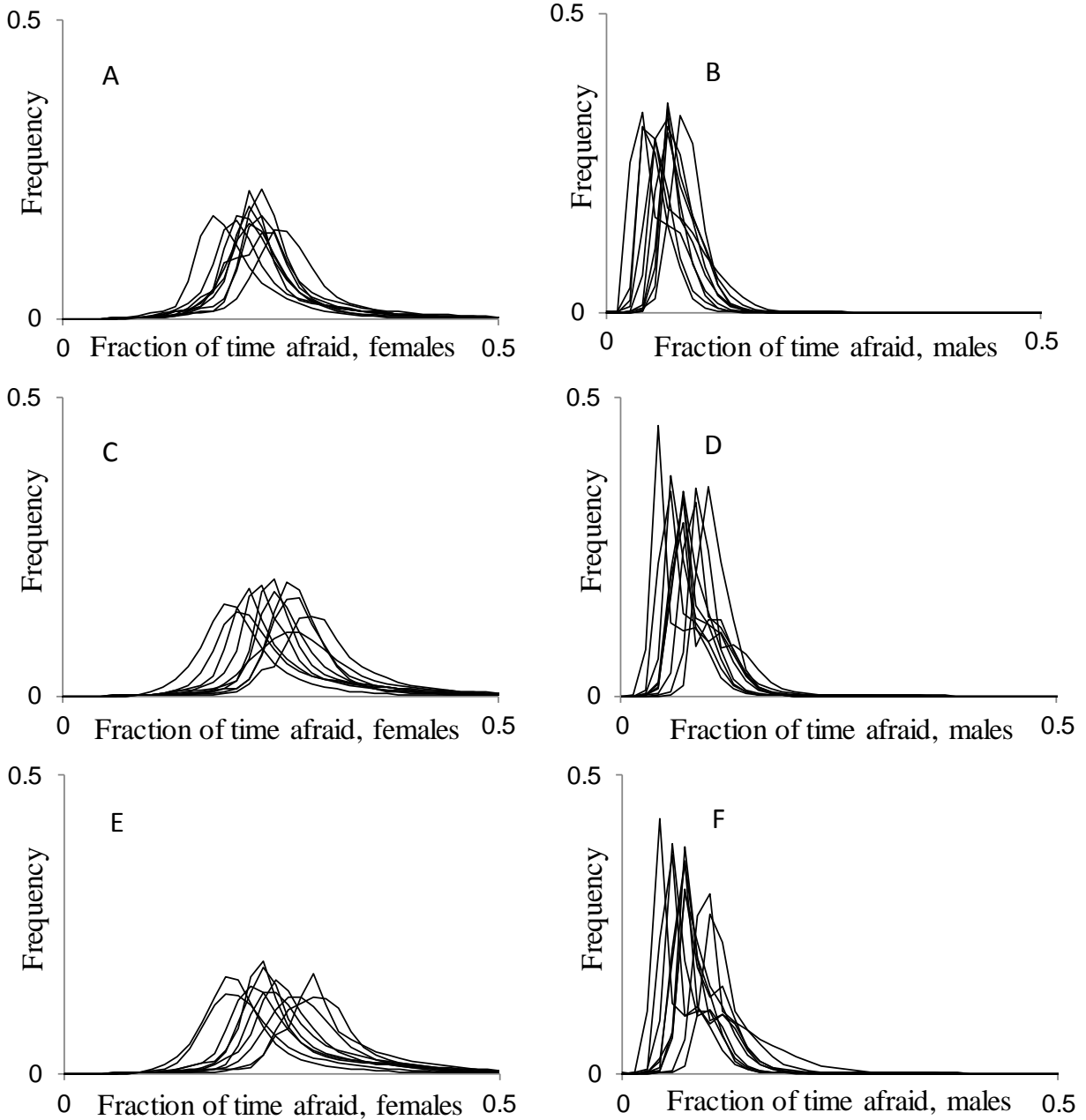


Figure 11: Frequency distribution of fraction of time spent in the GOS 'Afraid'. Each curve represents one of 10 simulations. A-B) The distribution for the last 20 'Normal' generations before generation 20,000 for females and males. C-D) The distribution for the last 20 'Normal' generations before generation 80,000 for females and males. E-F) The distribution for the last 20 'Normal' generations before generation 120,000 for females and males. 'Normal' generations are explained in Appendix A.1. These are the long simulations run with the Giske et al. (2013) version of the model.

3.2.3 Hypothesis 3: Egg production insensitive to temporal resolution

When comparing the mean of all simulations between the nine experiments through evolution, most traits seem to stabilize relatively fast, but more importantly, they appear to stabilize at different levels (Figure 12). Both mortality, fecundity and body mass show little change after 2000 generations. The exception is fear, especially for females, where there is still an increase towards the end of the experiments. For most traits, graphs with the same color (indicating the same amount of time steps per diel cycle) group together after reaching stable values. Again, the exception is fear. The blue graphs, experiments with 67 time steps per diel cycle, do not show the same degree of clustering as the red and green graphs.

For final body mass, experiments with the same number of time steps per diel cycle show a similar distribution (Figure 13 A and B). The exception is the experiment with 16 diel cycles and 67 time steps per cycle. This experiment shows a higher final body mass than the other two experiments with 67 time steps per cycle. The depths where individuals reproduce, i.e. their depth in the last time step of a generation, follow more or less the same pattern as the distribution of body mass (Figure 13 C and D). Individuals in experiments with more time steps per diel cycle stay deeper at time of reproduction and have a higher body mass.

The individual mortality risk in the model is dependent on the body mass in two ways. Larger fish needs more food and is more easily detected by predators. This means that water with higher light intensities is much more dangerous for a large fish, but there is also where the higher food concentrations are and more importantly higher food encounter rate which is dependent on light intensity and food concentration. The green graphs show that fish with higher body mass stay deeper in the water column at the time of reproduction (Figure 13).

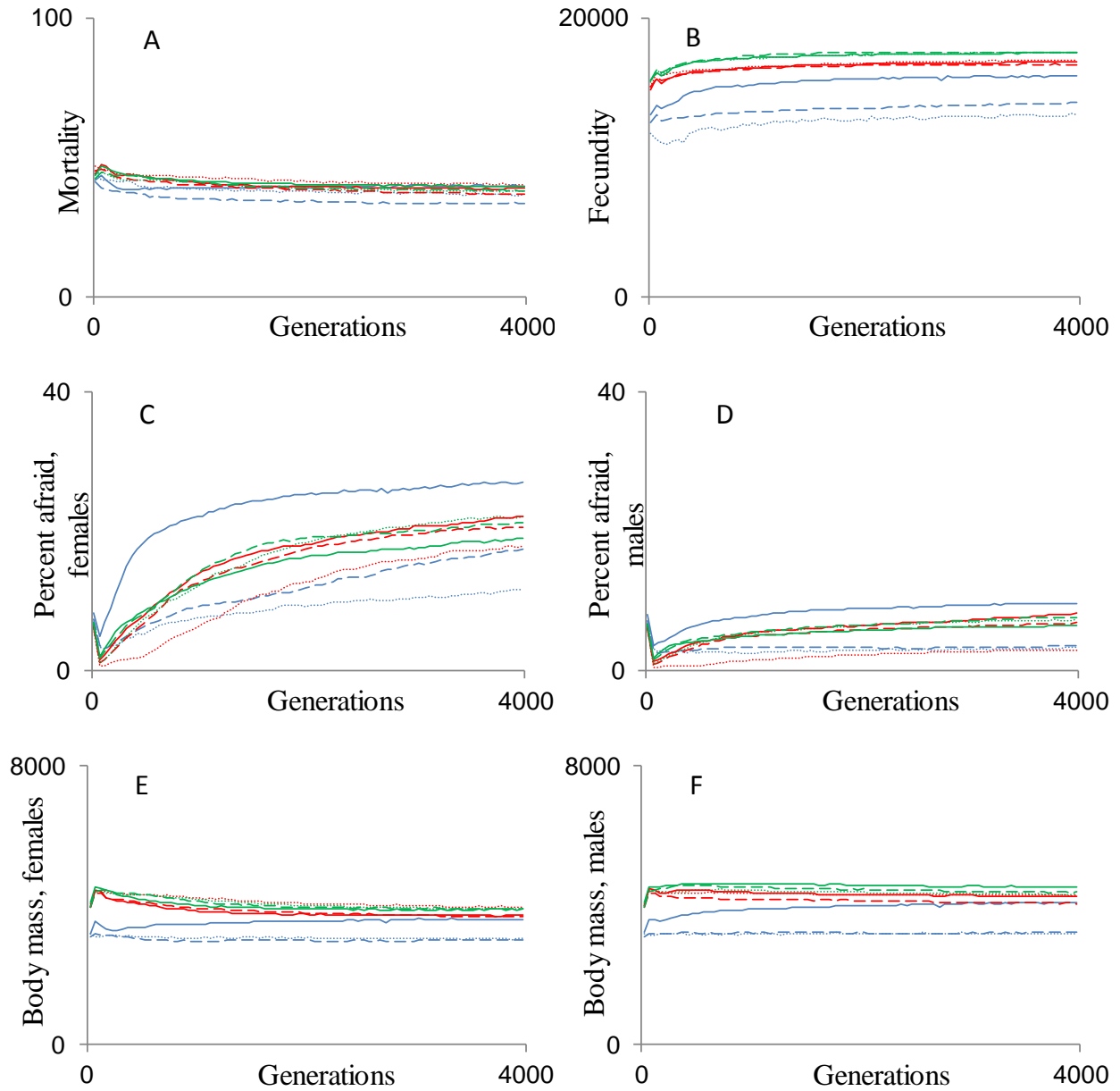


Figure 12: Comparison of the nine experiments. Each graph represents the average of all simulations in one experiment through life for mortality (A), fecundity (B), percent of total time spent in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F). Blue graphs indicate 67, red 200 and green 600 time steps per diel cycle. Dotted graphs indicate 4, dashed 8 and solid 16 diel cycles per generation. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

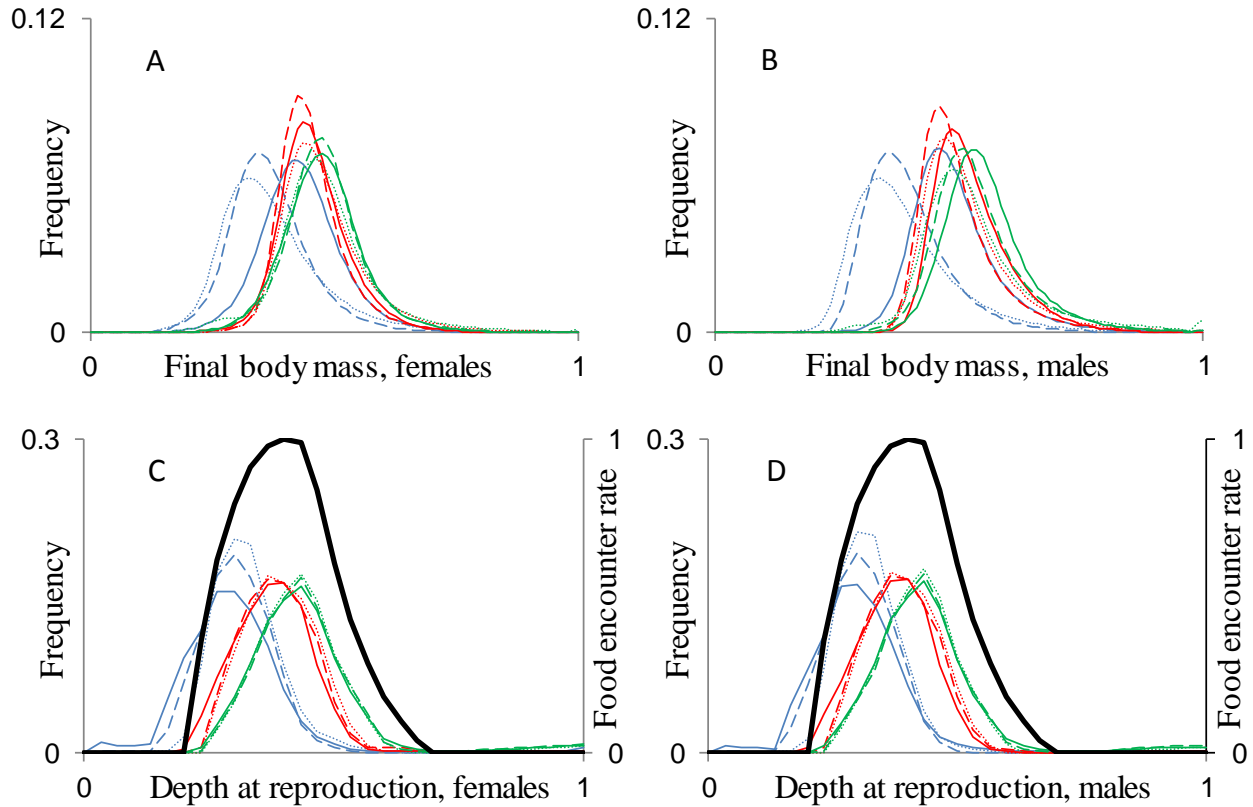


Figure 13: Comparison of the nine series. Each graph represents the average of all simulations in one experiment from the last 20 ‘Normal’ generations for frequencies of final body mass for females (A) and males (B) and frequencies for depth at reproduction for females (C) and males (D). ‘Normal’ generations are explained in Appendix A.1. Blue graphs indicate 67, red 200 and green 600 time steps per diel cycle. Dotted graphs indicate 4, dashed 8 and solid 16 diel cycles per generation. The black graph in C and D indicate the food encounter rate at the last time step and is given as a fraction of the maximum food concentration in that time step. The x-axes in A and B are given as a fraction of the largest possible body mass and the x-axes in C and D ranges from the surface (0) to the bottom (1).

As body mass increases, the organisms change which developmental genes they utilize. A general trend for these genes is that the fourth gene has a smaller value than gene two and three (see figure 3 in Giske et al. (2013)). This makes it more likely for larger individuals to become afraid and will keep the mortality risk down by moving to darker depths and larger groups. The experiments with 200 time steps per diel cycle (red graphs) have a depth distribution that appear to be best according to the food encounter rate. Experiments with the highest temporal resolution in each diel cycle (green graphs) distribute deeper in the water column and this may be due to their larger size and hence higher risk in depths with higher light intensities.

Distribution of final body mass and depth at reproduction show a very similar pattern between males and females when comparing the nine experiments, but males arrive at a slightly higher body mass (Figure 13). Males are chosen as mates by the females dependent on their body mass (see Appendix A.2). This makes it more important for males to become larger than their competitors and results in higher body mass (Figure 16) and higher mortality (Figure 15) than for the females.

Fecundity is the only evolutionary measure of the quality of these simulations. When statistically comparing the fecundity in the last generation of the nine experiments, a clear pattern emerges. Experiments with 600 time steps per diel cycle have a significantly higher fecundity than all other experiments, but are not significantly different from each other (Figure 14, top). Also, experiments with 200 time steps per diel cycle have significantly lower fecundity than those with 600, but significantly higher fecundity than those with 67 and are not significantly different from each other. Experiments with 67 time steps per diel cycle have the lowest fecundities and they also have significantly lower fecundity with fewer diel cycles. The populations will according to this achieve increasing fecundity with shorter time steps, while increasing the number of diel cycles is not important. However if the number of time steps is strongly reduced, then the number of diel cycles matter for the fecundity.

For the level of fear in the last generation, the pattern is not the same as for the fecundity (Figure 14, middle and bottom). Experiments with both 4 and 8 diel cycles per generation show a trend of increasing percent of fear with increasing number of time steps per generation. The three experiments with 16 diel cycles show the opposite trend, decreasing percent of fear in the population with increasing number of time steps per diel cycle. This pattern is the same for both females and males.

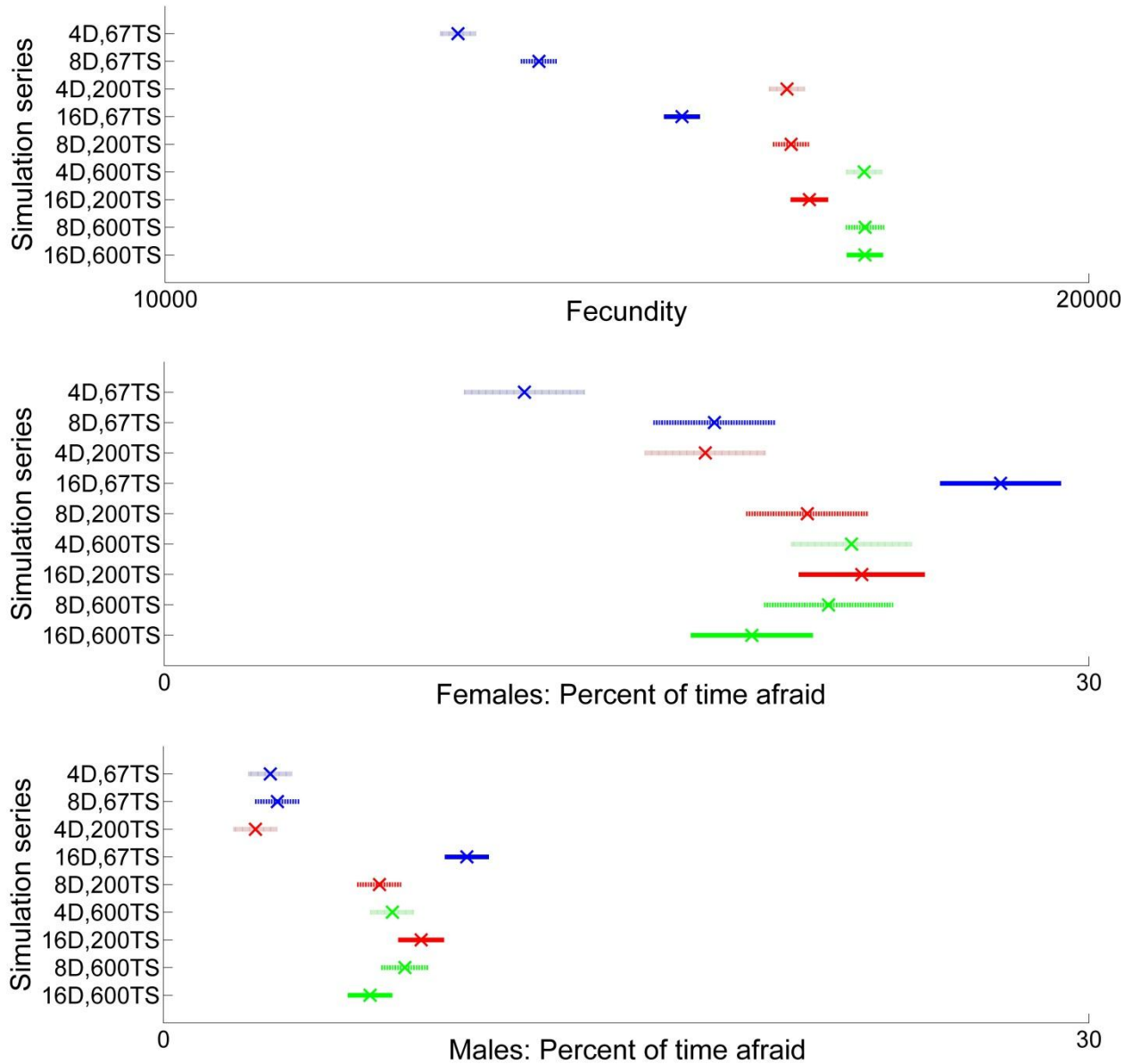


Figure 14: Statistical comparison of the nine experiments at the last generation. The Y-axes specify the experiment where D is the number of diel cycles and TS is the number of time steps in each diel cycle. The series are sorted by increasing number of time steps in a generation from top to bottom. For each series, the 'X' indicates the mean of fecundity (top), percent of time afraid for the females (middle) and percent of time afraid for the males (bottom). The horizontal lines are the 95 % confidence interval of the mean for the respective series. Blue series represent those with 67 time steps in each cycle, red those with 200 and green those with 600. Solid lines for the confidence interval represent series with 16 diel cycles, dashed lines 200 and shaded lines 67. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

The increase in fecundity when increasing the number of time steps per diel cycle can be due to an increase in body mass or a decrease in mortality, or a combination of the two. When increasing the number of time steps per diel cycle from 200 to 600, but keeping the number of diel cycles, there are no significant changes in mortality for females (Figure 15). However, there is significant increase in the final female body mass in the two cases with 8 and 16 diel cycles (dashed and solid lines) (Figure 16). This explains the increase in fecundity. In the case with four diel cycles, there is only a slight increase in body mass while there is also an increase in mortality. The increase in body mass then outweighs the increase in mortality and results in a higher fecundity. Females are the gender of interest here since it is their body mass that gives the fecundity. Male body mass is interesting only in respect to competition for females.

When increasing the number of time steps per diel cycle from 67 to 200, the mortality for females decreases and the final body mass increases for all three cases. Both factors contribute to the large increase in fecundity that we see between these experiments (Figure 14, top). Again, it is the mortality and body mass of the females that is of interest here. Males show in most cases the same trends as the females, but not in all (Figures 14-16).

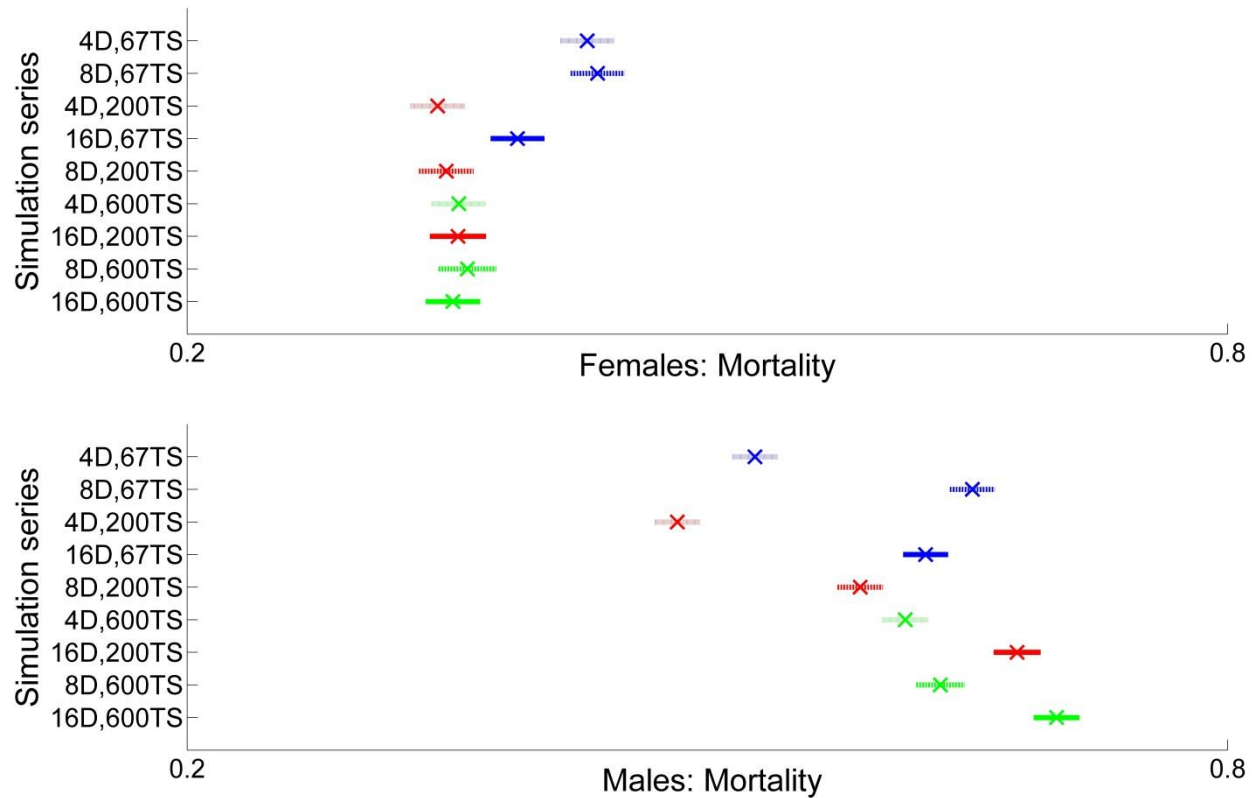


Figure 15: Statistical comparison of the nine experiments at the last generation. The Y-axes specify the experiment where D is the number of diel cycles and TS is the number of time steps in each diel cycle. The series are sorted by increasing number of time steps in a generation from top to bottom. For each series, the 'X' indicates the mean of fraction dead before reproduction (mortality) in the last 20 'Normal' generations for females (top) and males (bottom). The horizontal lines are the 95 % confidence interval of the mean for the respective series. Blue series represent those with 67 time steps in each cycle, red those with 200 and green those with 600. Solid lines for the confidence interval represent series with 16 diel cycles, dashed lines 200 and shaded lines 67. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

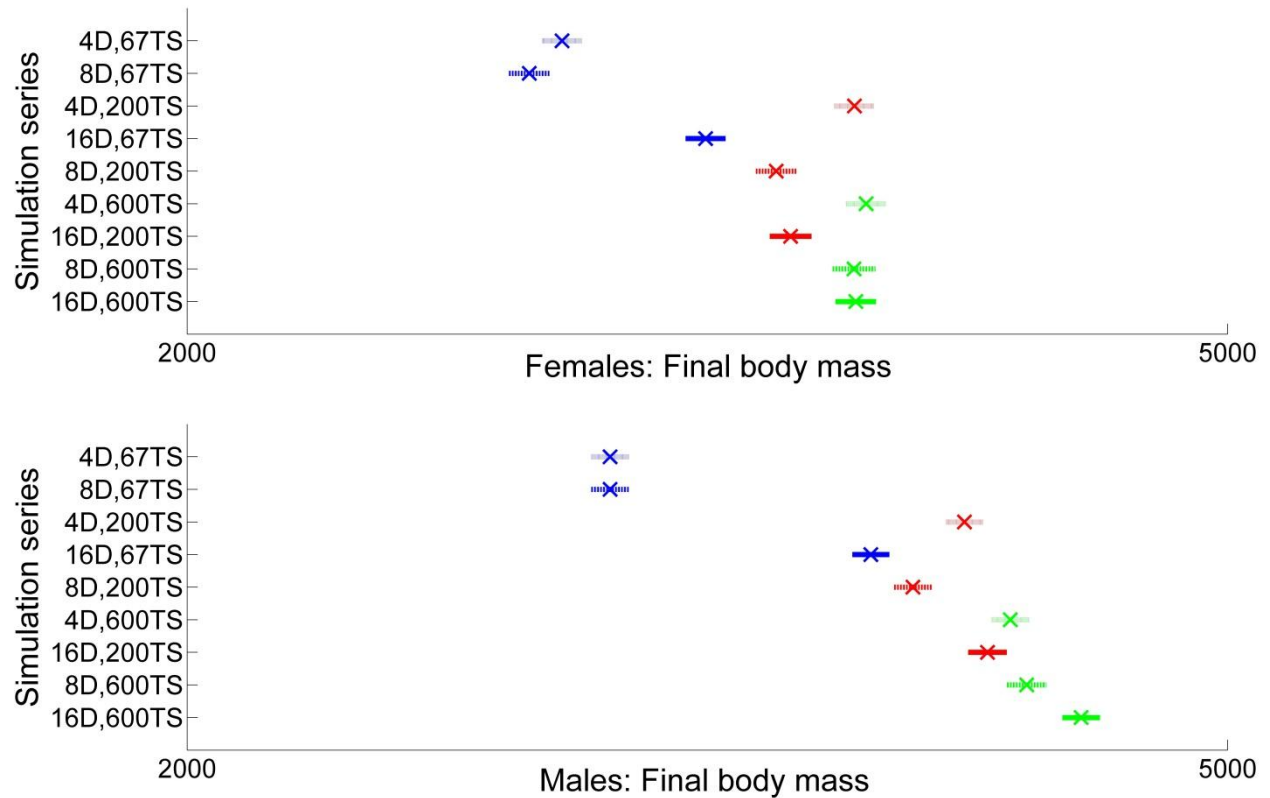


Figure 16: Statistical comparison of the nine experiments at the last generation. The Y-axes specify the experiment where D is the number of diel cycles and TS is the number of time steps in each diel cycle. The series are sorted by increasing number of time steps in a generation from top to bottom. For each series, the 'X' indicates the mean of final body mass for females (top) and males (bottom). The horizontal lines are the 95 % confidence interval of the mean for the respective series. Blue series represent those with 67 time steps in each cycle, red those with 200 and green those with 600. Solid lines for the confidence interval represent series with 16 diel cycles, dashed lines 200 and shaded lines 67. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

3.2.4 Hypothesis 4: Duration of fear periods insensitive to temporal resolution

Even though there is no clear pattern between the different experiments in the level of fear, there is a much clearer pattern when looking at the length of the periods of fear (Figure 17). The simulations with few time steps per diel cycle (blue dots) have a much higher frequency of longer periods of fear. The length of a period is scaled to the length of the generation. To give an example: Experiment D4 TS200 and D16 TS600 have very similar levels of fear (Figure 14). However, when individuals in D16 TS600 are afraid, they are so on average for a much shorter period of their life than individuals in D4 TS200 (comparing red dots in top panel and green dots in bottom panel in Figure 17). When having the possibility of choosing more often, the chain of time steps in the GOS 'Afraid' will be interrupted more often relatively to the total number of time steps in the generation. Higher resolution in the model in the form of more time steps per diel cycle may not result in less fear in the population, but the relative length of the continual periods of fear will decrease. Continual periods of fear are longer in the experiments with four diel cycles, than in those with eight and 16.

In all simulations series there are individuals that stay afraid their entire lives (for illustration see Figure A20 in Appendix F, bar to the right in all panels). The number of individuals that display this behavior ranges from two (D4 TS67) to 94 (D8 TS200) in the last generation in all simulations in each experiment. There are much fewer that are afraid half their life, in most series none. The individuals that display the behavior of always being afraid are both males and females, but all end up at very low body masses (about 10 % of average final body mass). These individuals almost never produce offspring in the next generation due to this low body mass. It is likely that this life strategy is the result of a recent random mutation (or a combination of mutations) that makes the individuals choose the GOS 'Afraid' all the time and hence have good chances of surviving to the end of the generation, but arriving at a small size and therefore most likely leave no offspring.

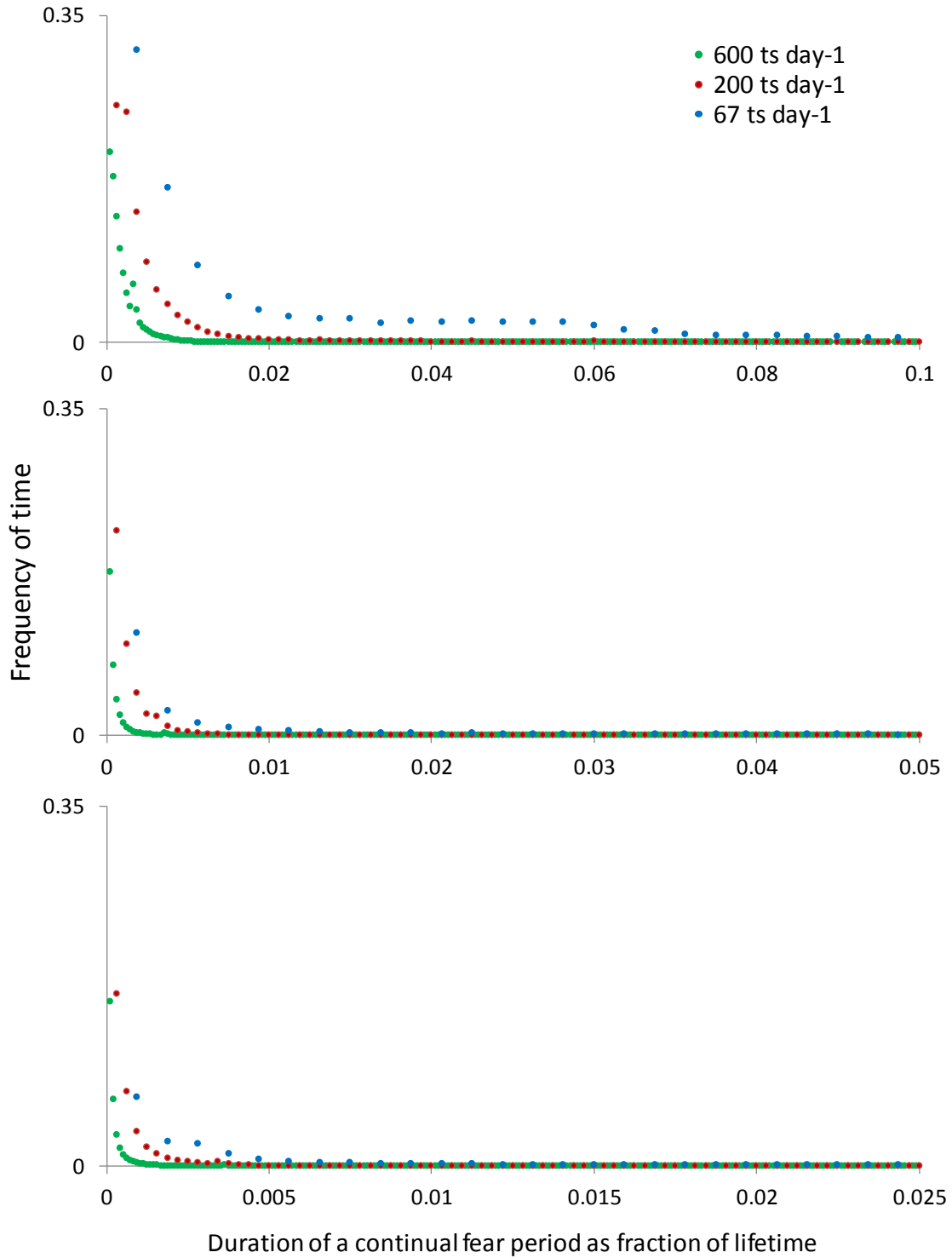


Figure 17: Frequency of time spent in continual periods of fear. The three panels represent experiments with 4 (top), 8 (middle) and 16 (bottom) diel cycles per generation. Blue series represent those with 67 time steps in each cycle, red those with 200 and green those with 600. The duration of a continual fear period is scaled to the number of time steps per diel cycle. There are therefore nine green markers for

each blue marker on the x-axes since one time step in the green series equals only one ninth of the time in a generation compared to the blue series. Because behavior is generally repeated when number of diel cycles are increased (and hence each time step represents less of the total life length), the length of the x-axes are decreased accordingly. The x-axes are cut off at 0.1 or lower because periods longer than this rarely occur. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

3.3 Complex neuronal responses

All analyses are done on the last generation in the simulations in the experiment with eight diel cycles and 200 time steps per cycle (D8 TS200), except for the comparison of levels of fecundity. To what degree the different populations evolved a complex neuronal response function where more than one of the three components had substantial influence, showed consistency for some of the neuronal responses, but not all. The second and third set of components of Equation 10 were almost never needed to determine the GOS, the only exceptions were under predator attacks. When more than one component was utilized, this was to achieve a response function that yielded a still increasing response even at high perception levels or to have a graded response at very low perception levels, and in both cases keep the sigmoid shape of the response function. Also, I found that there is more than one way to become afraid, even when having the opportunity to evolve a more complex neuronal response.

3.3.1 Hypothesis 5: Complex neuronal responses improve fitness

The average between all the 50 simulations of the frequency of how many sets of neuronal response components that they need to pick the same GOS as they did in the real simulation showed that more than one set was almost never needed (Figure 18). The four peaks of use of two sets of components indicate the occurrence of predator attacks. During a predator attack, the perception strength of the predation risk increases tenfold. This indicates that the responses to predators are largely comprised of one single component up to a certain point where from a second (and sometimes third) component also contributes to make the individual choose the GOS 'Afraid'. An example of how such a complex response function looks is given in later in the text (Figure 27).

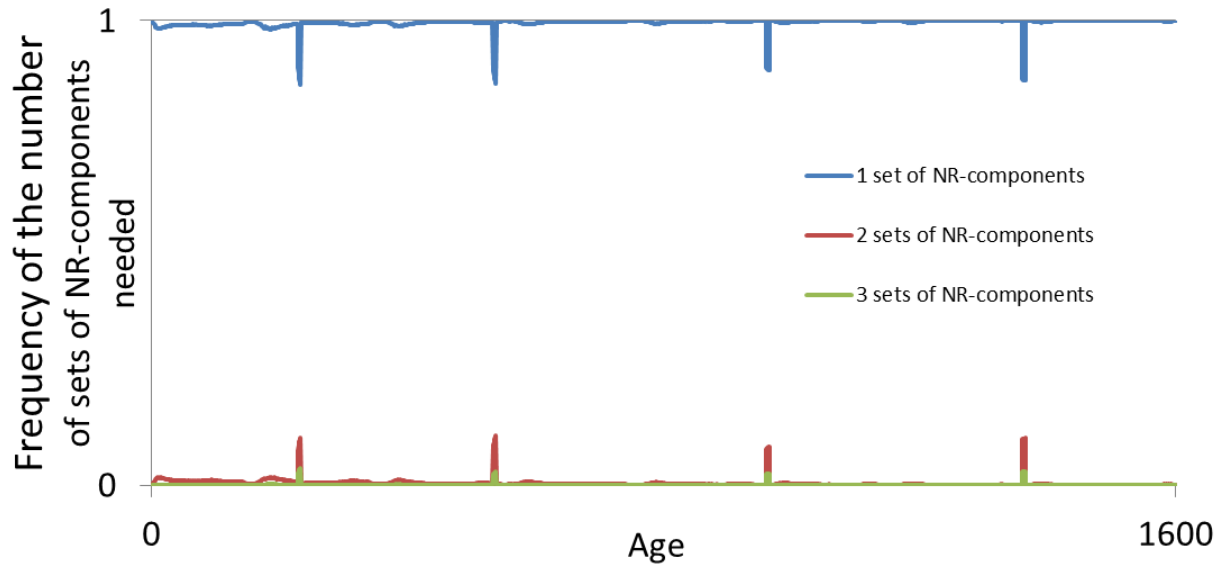


Figure 18: The number of components from each complex neuronal response, starting with the one giving the strongest response, which is needed to find the right GOS through life. The right GOS is the one that the individual chose in the actual simulation. The four peaks in use of the second and third set of responses are at the time of a predator attack. All individuals in the last generation ('Normal') in all simulations from the D8 TS200 experiment are used here.

In 95.3 % of the cases when an individual needed more than one component to find its right GOS, this GOS was 'Afraid'. Since most of these cases are during a predator attack, this is not very surprising. The second and third components are hence generally used to ensure that an individual becomes afraid at the time of a predator attack.

If looking at which one of the three contributors to fear (light and predators increasing fear and conspecifics decreasing it) that contributed most with its second and third component and hence was the factor that gave the choice, there is no consistency between simulations (Figure 19). However, in most of the simulations one of the three contributed with more than 90 %. This illustrates that it is possible to achieve the same GOS, but by utilizing the neuronal responses in different ways.

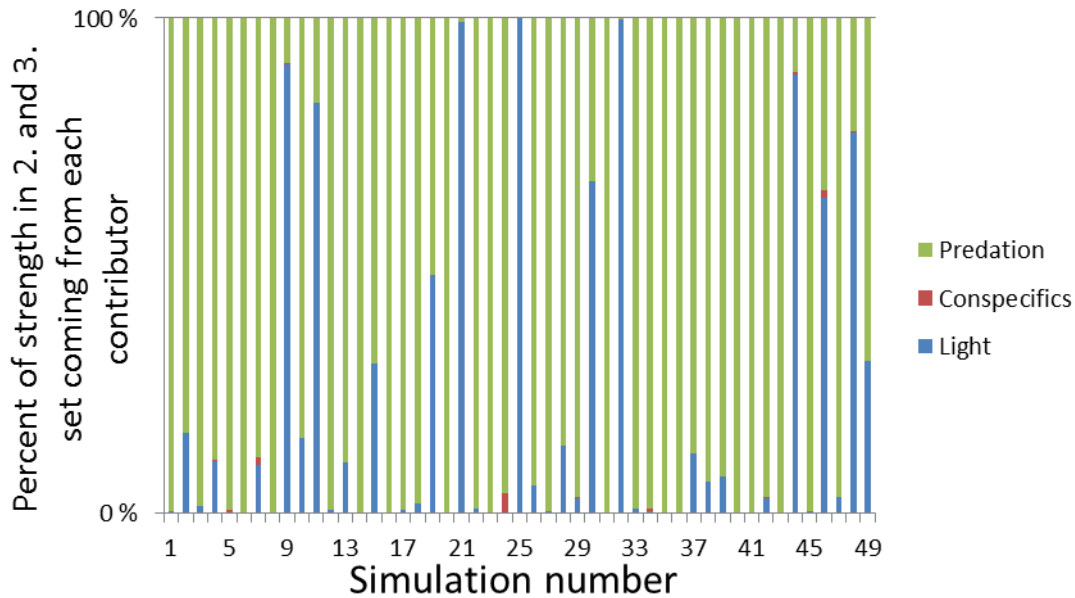


Figure 19: The percent of the strength of the neurobiological response to fear by the two weakest sets of neuronal response components that comes from each of the three contributors. The histogram presents average values in the last generation ('Normal') for all simulations in the D8 TS200 experiment.

When more than one set of neuronal response components are needed, it is the responses from predation and light that are dominating (predation has a negative contribution to fear). In most simulations, one of the two stood for >90 % of the response from the second and third set. For eight of the simulations light was the dominating factor. The rest of the simulations had predation as the dominating factor when they needed more than one set of components.

The maximum level of fecundity and how fast this level was reached did not differ between experiments that only differed in the number of components in their complex neuronal response functions (Figure 20). These two experiments were run with seven diel cycles and 200 time steps per cycle, the previous standard for this model and the settings that were used in Giske et al. (2013).

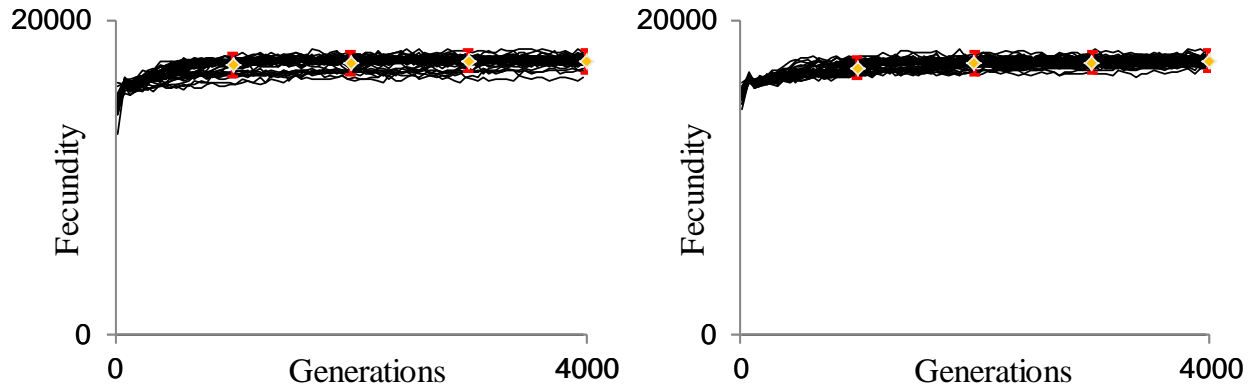


Figure 20: Comparison of mean fecundity in two experiments. These experiments were run with the Giske et al. (2013) standard (7 diel cycles and 200 time steps per cycle), but differed in respect to one (left) and three (right) components of the neuronal responses. The orange markers indicate the mean of all 50 populations at 1000, 2000, 3000 and 4000 generations. If this generation was not a ‘Normal’ generation, the first one after this was used. 95 % confidence intervals of the mean are marked by red bars at these four points. ‘Normal’ generations are explained in Appendix A.1.

3.3.2 Comparison of all simulations

I have now presented the results that directly relate to hypothesis 5. In the following sections I will go further into how the different simulations vary and what the more complex neuronal response functions look like. For all five neuronal responses that contribute to deciding the GOS, the strongest component comprised $> 90\%$ of the response in most simulations (Figure 21). However, there were also some simulations which deviated from this trend. In most cases the average relative strength of the strongest neuronal response (NR1) equals one, meaning that the entire complex response function is made up of one single component. None of the neuronal responses have average relative strengths that is less than half, indicating that there always is one component that is much stronger than the other two and not two or three almost equally strong components.

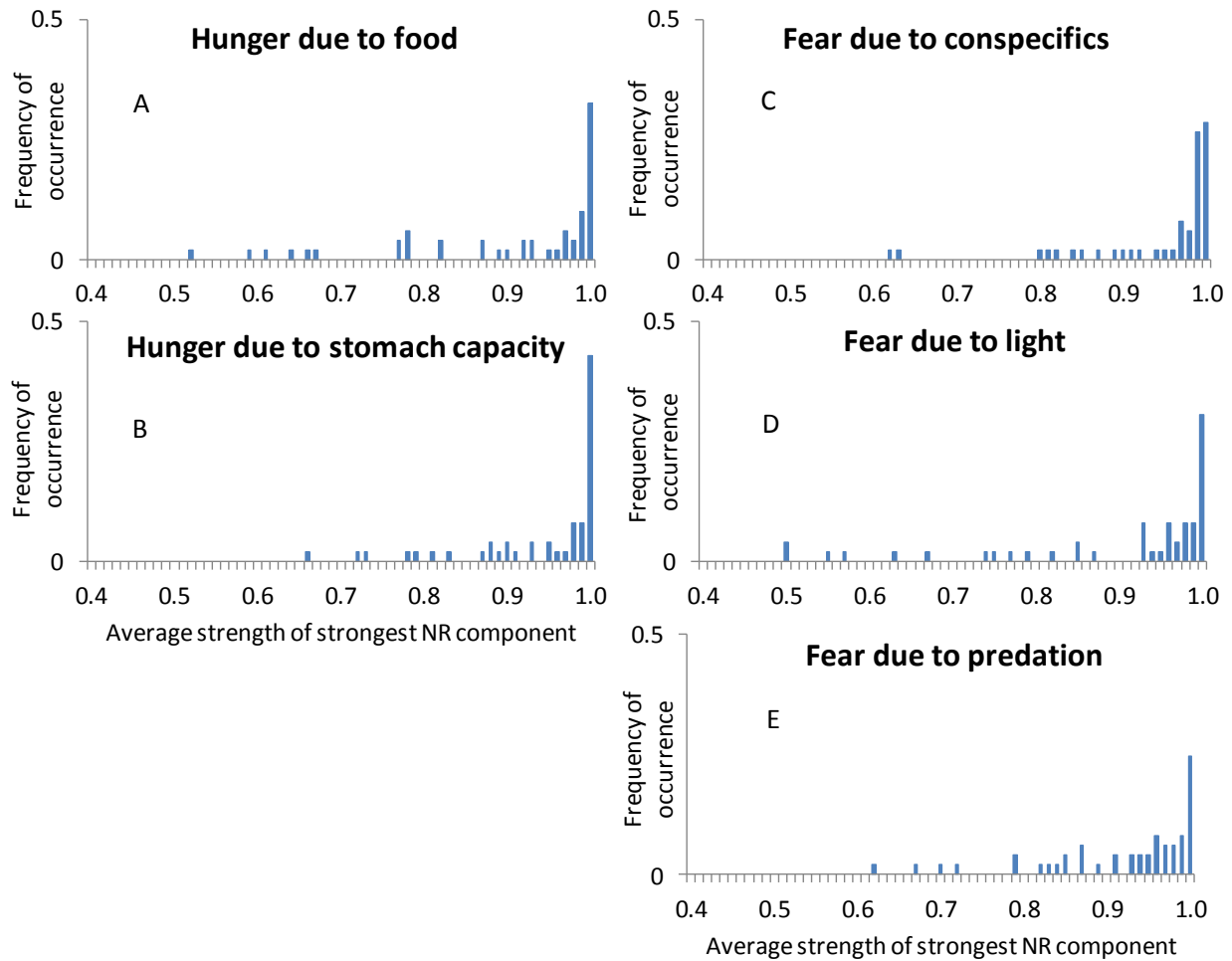


Figure 21: Frequency distribution among 50 simulations of average relative strength through life of the strongest of the three components in a complex neuronal response function. High average strengths for the strongest component indicate that the population only utilizes one neuronal response function. The histograms show frequencies for the neuronal responses that are a part of emotional appraisal, either towards hunger (A-B) or fear (C-E). Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1. Here, the last generation ('Normal') in all simulations in the D8 TS200 experiment is presented.

For the four neuronal responses that determine depth choice when individuals have entered a GOS, there is a lot of variation in the distribution of the average strength of strongest neuronal response component between simulations (Figure 22). This means that the different populations differ in to what extent they utilize multiple components when choosing a new depth. The utilization of a complex neuronal response function was more frequent during the emotional response (Figure 21) than when choosing depth. When doing this, hungry individuals benefited

from the second, and maybe third, component when evaluating food concentration (Figure 21 A), while frightened individuals had a tendency to use a complex function both when evaluating conspecifics (Figure 21 C) and light (Figure 21 D). However, the use of multiple components varied a lot between simulations for the frightened individuals. In comparison to response to food when hungry, frightened individuals used only one component in a substantial fraction of the simulations.

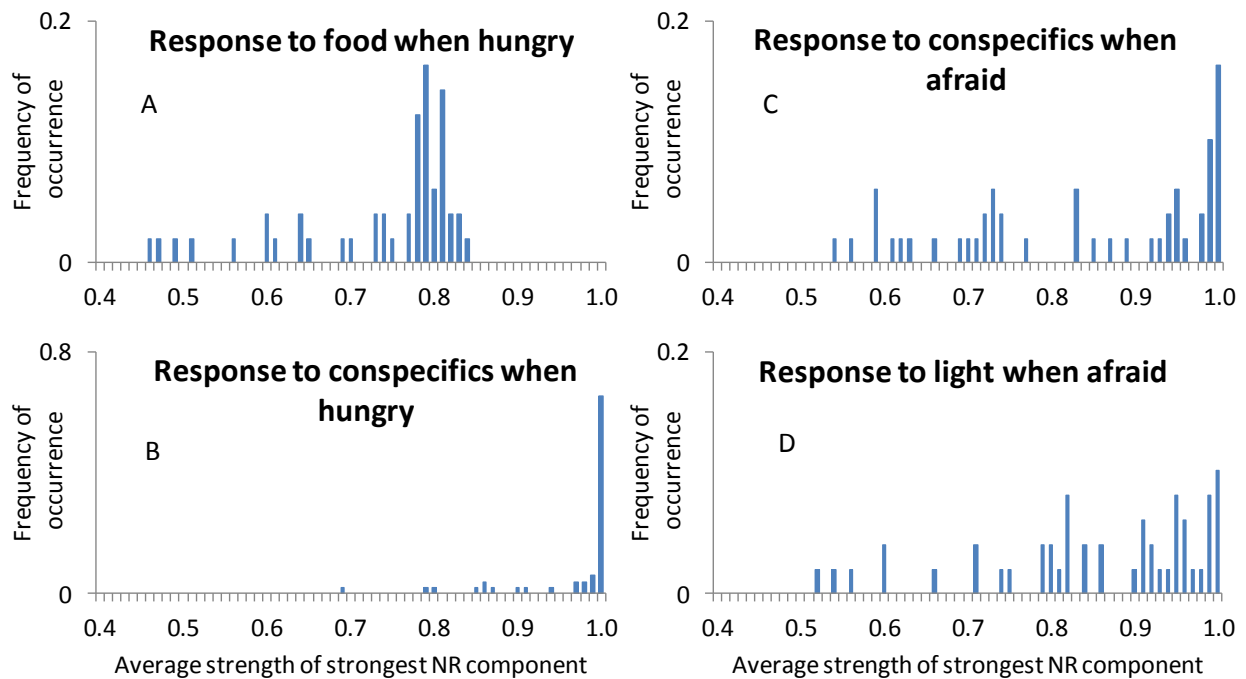


Figure 22: Frequency distribution among 50 simulations of average relative strength through life of the strongest of the three components in a complex neuronal response function. High average strengths for the strongest component indicate that the population only utilizes one neuronal response function. The histograms show frequencies for the neuronal responses that are a part of depth choice when hungry (A-B) or afraid (C-D). Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1. Here, the last generation ('Normal') in all simulations in the D8 TS200 experiment is presented.

All neuronal responses do not display the same pattern of variation. The response to conspecifics when in the hungry state shows that almost all simulations only use one component (Figure 22 B). Here, there was much less variation between simulations.

3.3.3 Comparison of five simulations through life

The fraction of the total response that comes from the strongest neuronal response component (NR1) does in some cases show similar patterns between simulations through life (Figure 23). When this is to only use NR1, it indicates that all populations have evolved a complex response consisting of only a single component (Figure 23 C). The other two are then evolved to a level where they do not contribute. Some simulations have an opposite pattern when it comes to use of other components than NR1 (Figure 23 D) and some have just very different use of NR1, both in the overall level and the variation of this level through life. Equivalents for simulations 6-10 for the Figures 23-26 can be found in Appendix D.

Whether one, two or three components are in use in a complex neuronal response, is not very important if the strength of that response is negligible. When choosing a GOS, the sums of two and three responses are calculated, for hunger and fear respectively where conspecifics have a negative value for fear. The strength of the neurobiological state of hunger (Equation 2) comes almost entirely from the stomach (Figure 24 A). For the fear response (Equation 3), the response from conspecifics is the most dominant one except during a predator attack (Figure 24 B). Then the fear response changes to consist of almost only the response from predation (Figure 24 D).

Not all simulations have evolved the same mechanism for becoming afraid. Among these five simulations, there is one that stands out with a totally different pattern (blue graph in Figure 24 B-C). In this particular simulation, the response to light (Figure 24 C) has a much stronger effect on fear than the other two. This again illustrates that the architecture of the model gives the opportunity to evolve different solutions to the same problem.

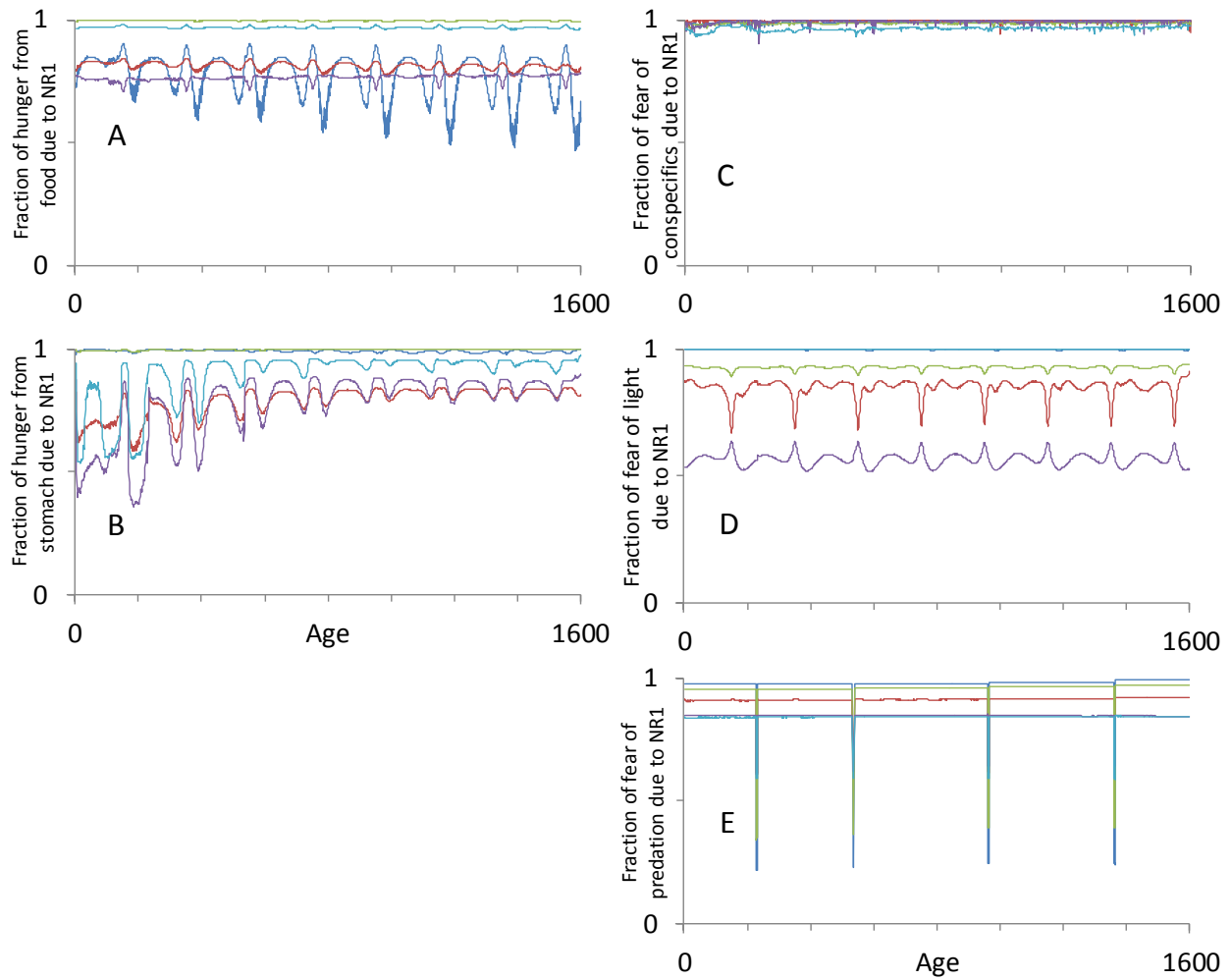


Figure 23: Relative contribution from components in complex neuronal response functions: Comparison of the last generation of five simulations from the D8 TS200 experiment, shown with same color in all panels. A-B) The fraction of the total neuronal response contributed by the stronger (NR1) of the two contributors to hunger: food (A) and available stomach capacity (B) through life. C-E) The fraction of the total neuronal response contributed by the stronger (NR1) of the three contributors to fear: reduction by conspecifics (C) and increase of fear by light (D) and predation (E) through life.

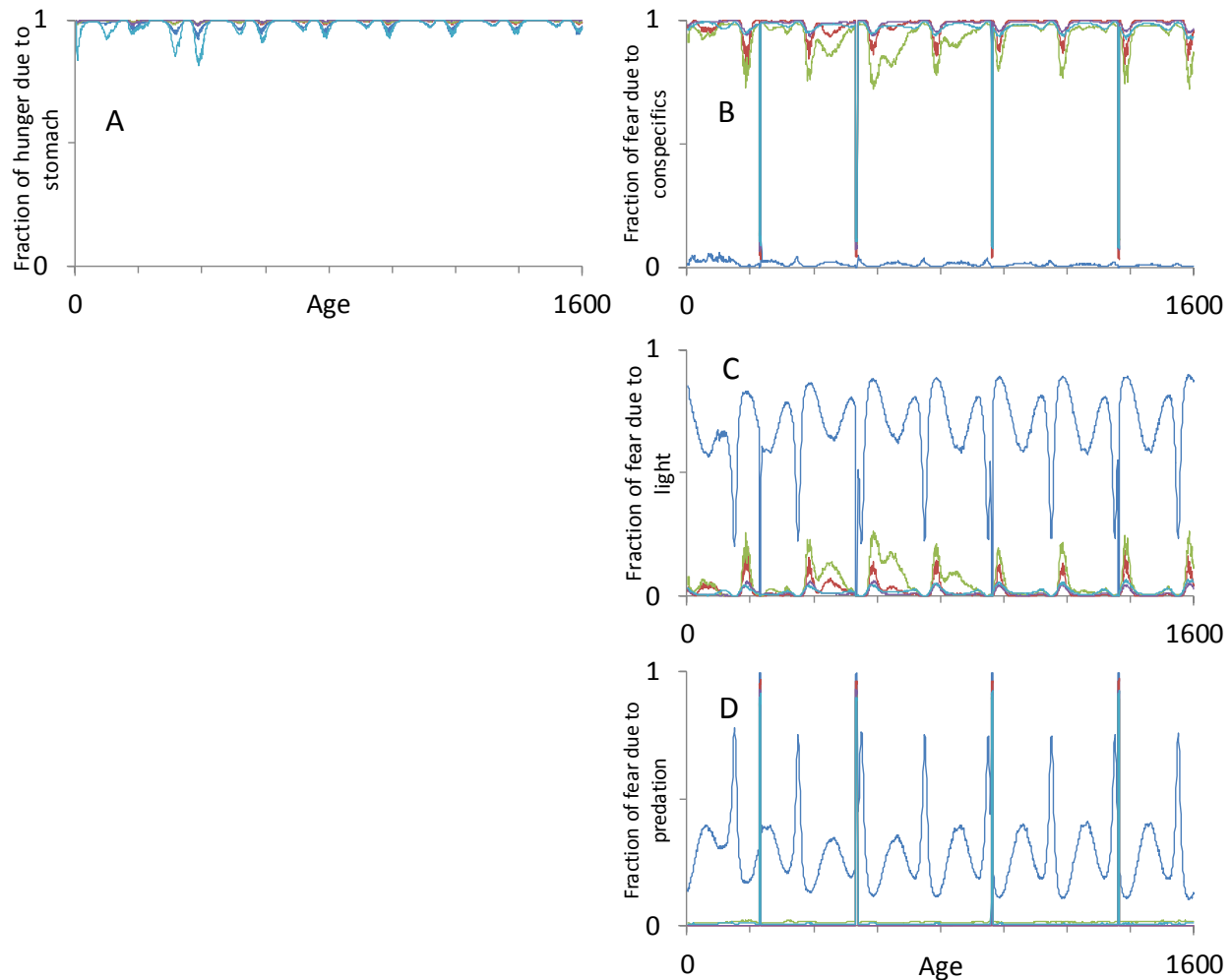


Figure 24: Relative contribution from the complex neuronal response functions when selecting GOS: Comparison of the last generation of five simulations from the D8 TS200 experiment, shown with same color in all panels. A) The relative strength of the neuronal response from available stomach capacity impacting hunger through life. B-D) The relative strength of the three neuronal responses impacting fear: conspecifics (B), light (C) and predation (D) through life. Data are from all individuals in the last generation of the simulations.

The consistency in use of multiple components varies a lot between the different neuronal responses that is used to choose depth. The fraction of the complex neuronal response that comes from strongest neuronal response component is very consistent between simulations in the response to conspecifics when in the GOS ‘Hungry’ (Figure 25 B). All simulations utilize only one component in this case. The response to food in this state is also consistent between simulations in regard to using more than one neuronal response (Figure 25 A). The degree of use varies, but the patterns through life are very similar.

For other neuronal responses, the use of multiple components varies much more between simulations, exemplified by the use of strongest neuronal response component in the complex response to both conspecifics and light when in the GOS 'Afraid' (Figure 25 D and E). Some of the simulations used only one and some used multiple components.

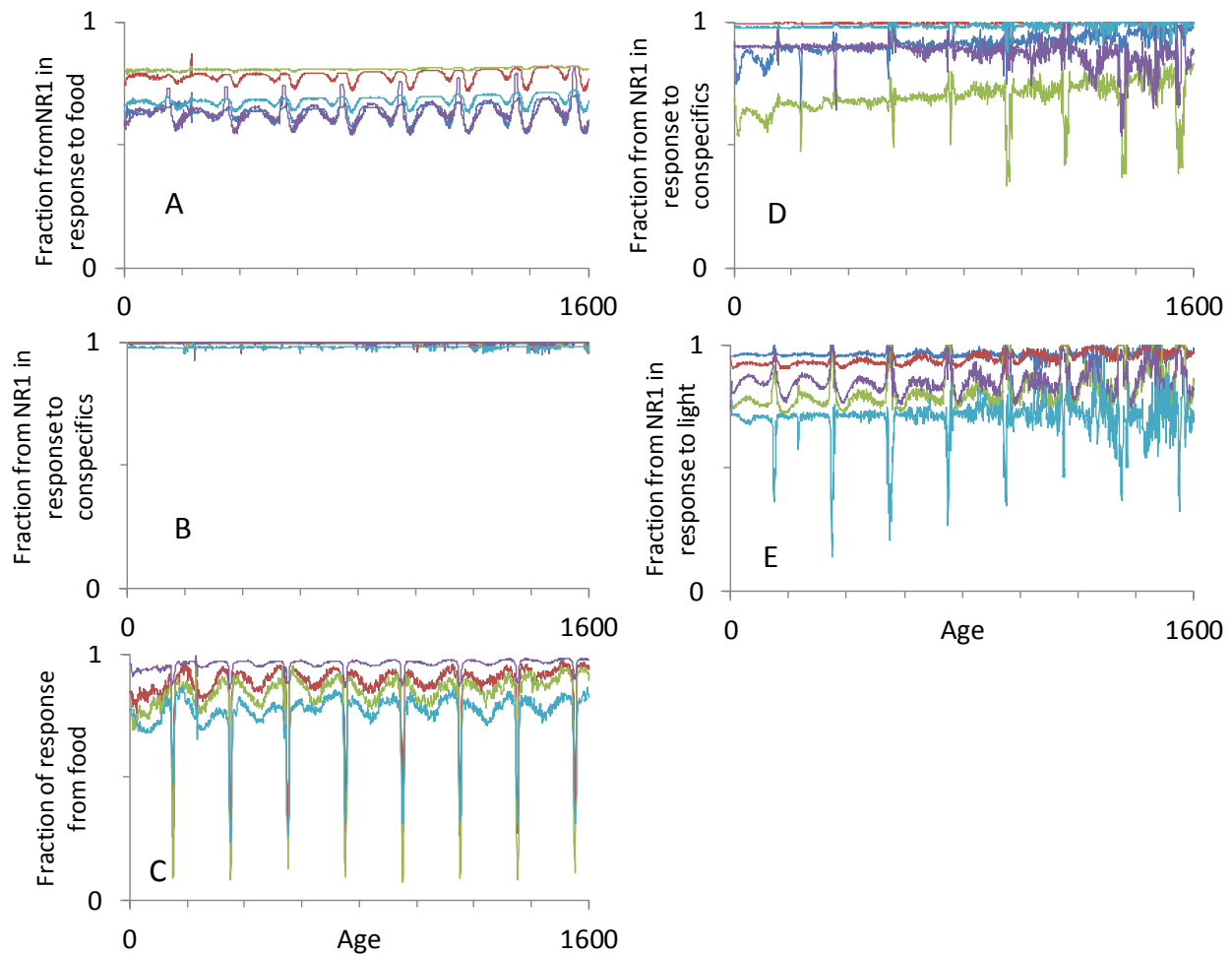


Figure 25: Relative contribution from components in complex neuronal response functions and contribution from the complex neuronal response functions when selecting depth: Comparison of the last generation of five simulations from the D8 TS200 experiment, shown with the same color in all panels. A-B) The fraction of the total neuronal response contributed by the stronger (NR1) of the three components through life when hungry for attraction from food (A) and repulsion from conspecifics (B). C) The fraction of the attraction towards a depth coming from food when in the GOS hungry through life. D-E) The fraction of the total neuronal response contributed by the stronger (NR1) of the three components through life when afraid for attraction from conspecifics (D) and repulsion from light (E).

When in the GOS 'Hungry', the fraction of the response that comes from food is generally much larger than the fraction from conspecifics (Figure 25 C). However, eight times during the last generation the fraction shifts to be almost entirely dominated by the response from conspecifics. This is due to the eight diel cycles in the simulation. The perception of food is determined by the amount of food and the visual range of the fish. The visual range decreases quadratically with the ambient light intensity (see Equation A2 in Appendix A.1). Therefore, when the light intensity drops to a certain level, the response from food becomes much smaller than the one from conspecifics which is not dependent on light.

The fraction of the evaluation of a depth that comes from the response to light when in the GOS 'Afraid' varies a lot between simulations (Figure 26). Some do not consider light at all when choosing a depth, and those that do varies a lot in how strong this response is compared to the competing one (conspecifics).

It does not appear to be consistency in the way different populations utilize the increased number of neuronal response components, except in the response to conspecifics when in the hungry state. Here almost all populations utilize only one neuronal response, although the fraction of the total evaluation of a depth that it contributes to, still varies a lot between populations (Figure 25 C).

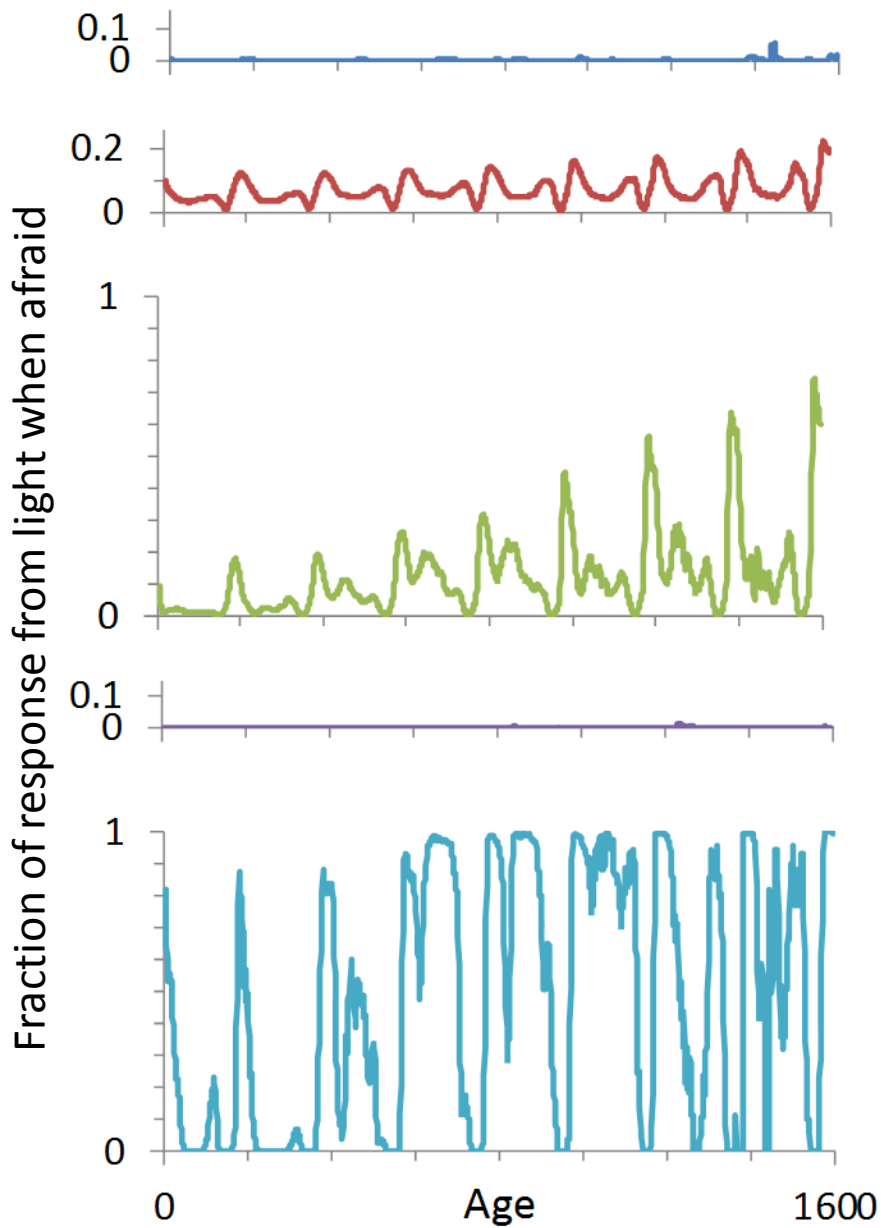


Figure 26: Relative contribution from light when selecting depth: The fraction of the repulsion from a depth coming from light when in the GOS afraid through life. Data are from all individuals in the last generation of simulations 1 (top) to 5 (bottom). The fraction at all 1600 time steps in each panel are the average of that age \pm 4 time steps, i.e. the average of 9 time steps. This is to make graphs easier to interpret. The graphs represent the last generation ('Normal') in the first five simulations in the D8 TS200 experiment.

3.3.4 How the increased genetic complexity are utilized by the populations

What kind of neuronal responses that are evolved and how the ability to add responses together to achieve new forms in these simulations is presented below. All examples are taken from simulation number two in the D8 TS200 experiment if not stated otherwise. This was picked because it was not among the few that deviated a lot from the rest according to the amount of fear in the population and had a pattern in the usage of multiple components in the complex neuronal response that were similar that of the majority of the other simulations.

I will go through how the different responses are used in the order that they appear in the model. First, the five neuronal responses that are used in emotional appraisal. This means that they together with the developmental modulation genes determine the GOS. Last, the four neuronal responses (for which only two are used dependent on GOS) to choose between the possible depths. All complex neuronal response functions that are presented in the Figures 27-30 are function that have evolved in simulation number two in the D8 TS200 experiment and where a considerable amount (>30 %) of the population have this (or a very similar) response function.

3.3.4.1 Choosing GOS – Emotional appraisal

One way to utilize multiple neuronal responses is to have a graded response even at high and low perceptions, i.e. discriminating at the extremes. This form is found for the complex neuronal response function for light when choosing a GOS and is not possible to make with only a single response function (Figure 27, left). Another example of an evolved function that has a similar shape, discriminating even at high perceptions, is one for the response to predators (Figure 23, right). The shape of this function serves to make sure that the organism enters the GOS ‘Afraid’ during a predator attack and the addition of the second component further ensures this at the highest perceptions without increasing response at low perceptions. Both complex function presented here only utilize two components, the gene values of the third component have evolved to values where it does not give any response.

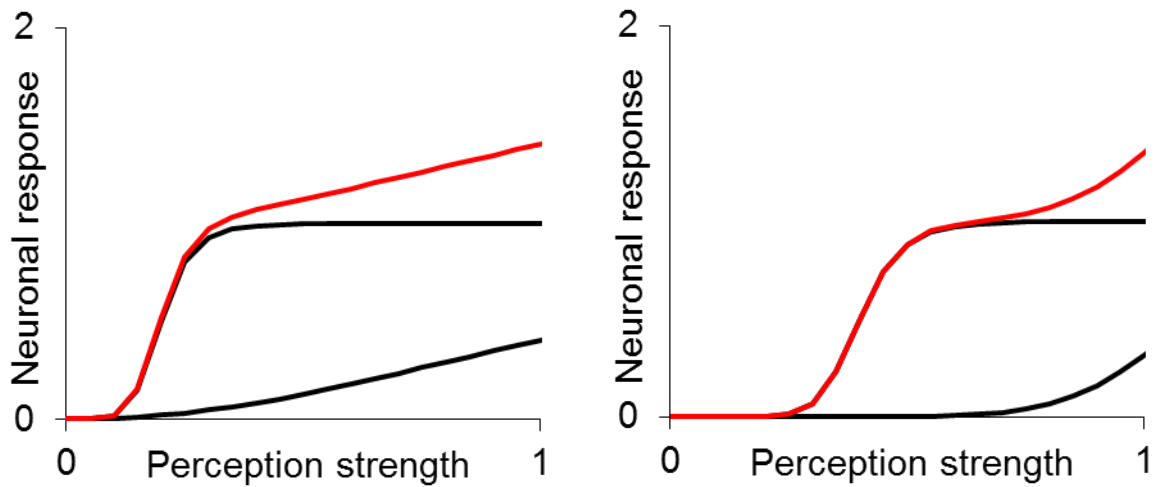


Figure 27: Complex neuronal responses that have evolved in the new version of the model. These complex neuronal responses are used to keep a sigmoid shape and still have increasing response at the highest perceptions. Complex neuronal responses to light (left, gene values: $x_1(0.62)$, $y_1(0.02)$, $x_2(0.22)$, $y_2(0.12)$, $x_3(0.31)$, $y_3(0.69)$) and predators (right, gene values: $x_1(0.90)$, $y_1(0.04)$, $x_2(0.82)$, $y_2(0.11)$, $x_3(0.97)$, $y_3(0.14)$) that contribute to fear when choosing GOS. The black lines indicate the single neuronal response components and the red line is the complex neuronal response. In both panels, the third component has evolved to very low values and therefore its graph does not show.

For the neuronal response to stomach capacity, the same complex neuronal response has evolved by use of both one and two components (Figure 28). There are here two very similar complex functions in the same population, but where only about half have utilized the second component to slightly alter the response to stomach capacity. The parameter values ('alleles') of the second components in these two complex functions are not very similar. This means that it is not just a trait that happens to have some variation in the population, but two distinct genotypes. The strongest components have very similar values which can change the response a lot by just small mutations, so in practice, they have the same phenotype.

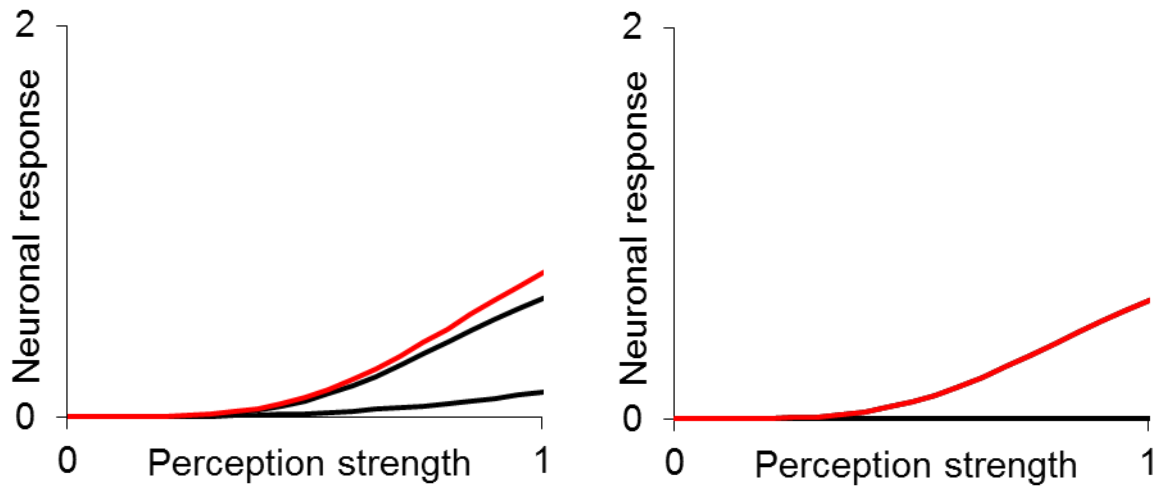


Figure 28: Complex neuronal responses that have evolved in the new version of the model. Neuronal responses to stomach capacity that contributes to hunger. Two solutions that evolved in the population where one (left, gene values: $x_1(0.41)$, $y_1(0.09)$, $x_2(0.30)$, $y_2(0.19)$, $x_3(0.72)$, $y_3(0.45)$) utilized two simple responses and the other utilized only one (right, gene values: $x_1(0.40)$, $y_1(0.09)$, $x_2(0.47)$, $y_2(0.70)$, $x_3(0.73)$, $y_3(0.55)$).

3.3.4.2 Choosing depth

Another way to utilize the increased complexity of the neuronal responses is to have some response even at low perceptions by use of the second (and possibly third) component. This is seen for a response to light in the GOS ‘Afraid’ (Figure 29, left plot). If the perception of light becomes high, this complex function will ensure a strong response and the organism will move according to light. When perception of light is weak, this function will still be able to discriminate between two depths because of the effect from the second component. This would be valuable when the concentration of conspecifics is the same in the evaluated depths since choosing the darkest depth will give lower predation risk.

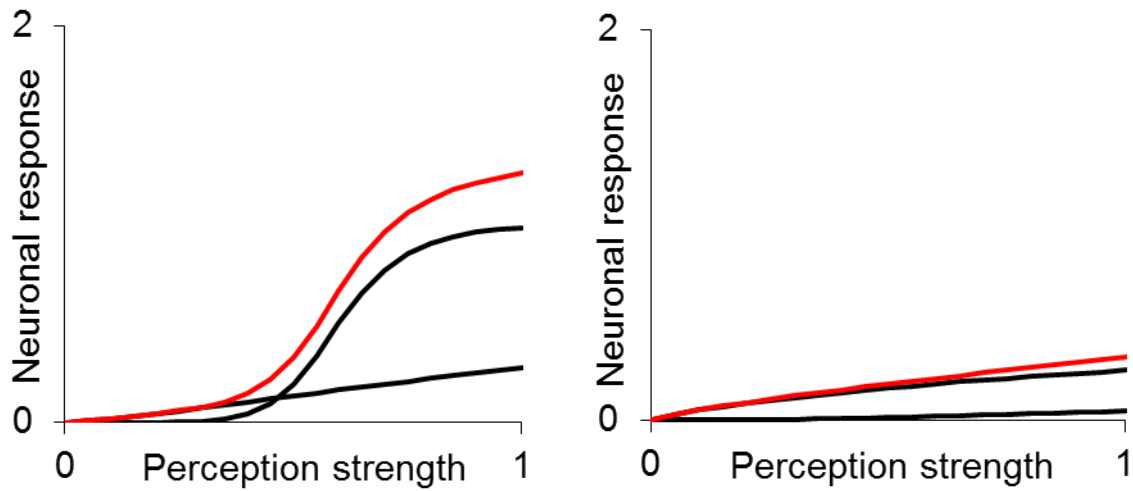


Figure 29: Complex neuronal responses that have evolved in the new version of the model. Neuronal responses to light when in the GOS 'Afraid'. The black lines indicate the three simple neuronal responses (here one is unused in both cases) and the red line is the complex neuronal response. The figure shows how combination of multiple responses can give a more complex response function that gives responses even at low levels of perceptions without losing the sigmoid shape (left, gene values: $x_1(0.78)$, $y_1(0.06)$, $x_2(0.13)$, $y_2(0.21)$, $x_3(1.00)$, $y_3(0.61)$). It also presents how a population can evolve more than one solution (left and right), in this case as the response to light when in the GOS 'Afraid'. (Right panel gene values: gene values: $x_1(0.08)$, $y_1(0.38)$, $x_2(0.18)$, $y_2(0.53)$, $x_3(0.17)$, $y_3(0.96)$.)

It is worth mentioning that the perception of light intensity rarely exceeds 0.1. The right half of the functions (Figure 29) are therefore only used by individuals that stay high in the water column at the highest surface light intensities.

As for previous versions of the model, evolution yields more than one solution for the neuronal response functions (Figure 29, both plots). Even though multiple components were used in the response to light when afraid, this was not the case for the responses to conspecifics when hungry, which also exhibited two very different complex neuronal response functions (Figure 30). This was also the neuronal response that showed very little variation between simulations (Figure 22 B).

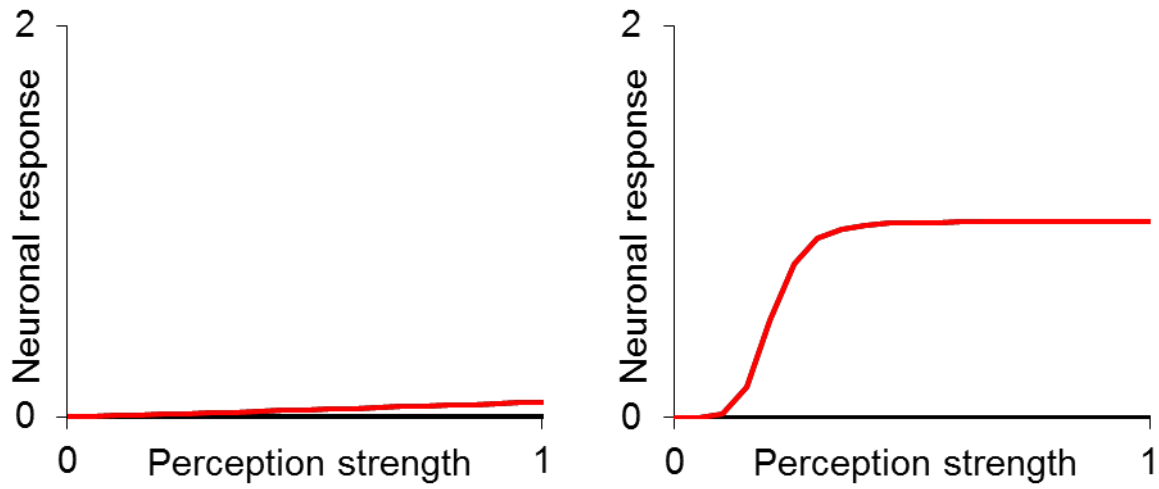


Figure 30: Complex neuronal responses that have evolved in the new version of the model. Two different neuronal responses to conspecifics when in the GOS 'Hungry' (left panel gene values: $x_1(0.11)$, $y_1(0.97)$, $x_2(0.46)$, $y_2(0.76)$, $x_3(0.70)$, $y_3(0.85)$, right panel gene values: $x_1(0.58)$, $y_1(0.02)$, $x_2(0.54)$, $y_2(0.41)$, $x_3(0.90)$, $y_3(0.84)$). The red line is the complex neuronal response. Only one neuronal response component was utilized in both cases and this therefore gives the complex function.

4 Discussion

4.1 Optimization, adaptation and emotion systems

Evolutionary optimization theory was founded by Lotka early in the 20th century. He only considered age to predict survival and fecundity, by use of the Euler–Lotka equation (Euler, 1760, Lotka, 1925), which allowed changing priorities through life. This method works well if age is the major explanatory variable. By the development of optimization theory into Optimal Foraging Theory (Emlen, 1966, Macarthur and Pianka, 1966), Life History Theory (Murdoch, 1966, Williams, 1966) and Game Theory (Maynard Smith and Price, 1973), other aspects of the individuals were taken into consideration. Exclusively for Game Theory is that the strategies of other individuals are considered. All these theories are based on optimality, but differ in what is to be maximized (Giske et al., 1998).

Combining Life History Theory and state dependency, Stochastic Dynamic Programming was developed (Mangel and Clark, 1986, Houston et al., 1988), still based on the simplicity assumption of the optimal solution. This approach allowed studies of trade-offs between the different components of fitness (Mangel and Clark, 1986, McNamara and Houston, 1986) and multiple physiological states could be evaluated in the same model together with age. All of these methods based on optimization have the advantage that they will arrive at the best solution, based on what that is to be maximized. However, if the number of solutions to be compared, i.e. the solution space, gets too large because of many variables, the computational complexity soon becomes enormous (Houston et al., 1988).

A way to solve complex problems in ecological research, including density- time- and state-dependency simultaneously is by use of adaptive models (Huse and Giske, 2004), utilizing techniques such as the Genetic Algorithm (Holland, 1975) and Individual-Based Modeling (Deangelis et al., 1980, Huse et al., 2002, Railsback and Grimm, 2011). This approach produces increasingly better solutions after each cycle in the algorithm. The Genetic Algorithm can be linked with a system for processing stimuli, e.g. Artificial Neural Networks (ANN) (Huse and Giske, 1998, Huse et al., 1999, Strand et al., 2002), specific equation sets (Giske et al., 2003) or

emotion systems (Giske et al., 2013), which yields insight into how individuals make their decisions. The need to include proximate elements in ecological research to explain the ultimate motivation has been called for by the scientific community (Ricklefs and Wikelski, 2002, McNamara and Houston, 2009, Fawcett et al., 2013). The proximate mechanisms and their constraints are of great interest of research on animal personalities (Dingemanse and Reale, 2005, Bell, 2007, Dingemanse and Wolf, 2013) and this have now been investigated by modeling an emotion system (Giske et al., 2013). To use an Individual-Based Model gives the advantage that each individual is represented by its own characteristics and behavior (Railsback and Grimm, 2011).

However, a problem with adaptive models is that the final solution can be at a local optimum, stuck on a small peak in the large fitness landscape (Mitchell, 1998). It is also a method that requires a lot of computational power. Whether to use optimization or adaptation as an approach is therefore dependent on the complexity needed to answer the question asked.

Diel vertical migration (DVM) has been investigated in many groups of animals by use of optimization modeling for more than 50 years (McLaren, 1963, McLaren, 1974, Clark and Levy, 1988, Hugie and Dill, 1994). A group of organisms that are thoroughly studied by use of such models is mesopelagic fish (Giske and Aksnes, 1992, Rosland and Giske, 1994; 1997, Rosland, 1997, Staby et al., 2013) and these models gave results that were consistent with field observations (e.g. Giske and Aksnes, 1992, Rasmussen and Giske, 1994, Staby et al., 2011). These models, which calculate behavior through rational equations, therefore seem to describe these organisms relatively well. However, more recent results (Giske et al., 2013, this thesis) show the same consistency with field data when fish behavior is modeled according to the theory of emotion systems. These results show that whether to base modeling on rational optimization or emotion theory is still an unanswered question. The theory of emotion systems is recent and therefore few field and lab studies have investigated such behavior. As a consequence, work by others has to be interpreted according to emotion theory since the authors did not consider it. However, it is likely that there will be produced more and better field and lab studies that investigate emotion theory in the years to come.

4.2 Stabilization of GOS levels

The still increasing level of fear (especially for females) after 4000 generations (Figure 12) in experiments with all tested levels of temporal resolution may be interpreted as the number of generations was not sufficient. However, when investigating much longer simulations, the levels of fear remain unstable even after 120,000 generations. Longer simulations are therefore not the answer to the unstable levels of fear. Neither did the increase in number of genes and hence the possibility to evolve more complex neuronal response functions yield stable fear levels or convergence of fear levels between simulations. There are two possibilities to why this does not happen. First, the unpredictability may be a consequence of the behavioral architecture and actually reflects the evolutionary dynamics of natural populations. Alternatively, to achieve stability, a change in complexity in the architecture of the model is needed. Architecture here refers to how the different genes and responses affect each other and are essential in generating phenotypic variation (Kirschner and Gerhart, 1998, Doyle and Csete, 2011).

4.2.1 Natural instability

The chance of becoming afraid is influenced by all five neuronal responses, those relating to fear and those relating to hunger (Equations 2 and 3). The chance of becoming afraid is also modulated by the four developmental genes. This results in freedom of usage of one of the many possible behavioral pathways. This freedom may explain the unpredictability at architectural levels between the genotype and the phenotype that is seen in the model. Similar explanations have previously been suggested by Wagner (2011) for biochemical systems. Other adaptive models using other brain configurations have also produced such pathway-dependent unpredictability, e.g. ANNs (Enquist and Arak, 1994, Huse and Giske, 1998, Strand et al., 2002, Duarte et al., 2011).

In addition to the freedom generated by the unpredictability in the behavioral pathways, the evolutionary history of a population is also a source of path dependency. Based on whether some strategies persist or go extinct, a population will end up with a certain configuration, leading to an evolutionary historic path dependency (Mangel, 1991). The numerous ways that the genes of an individual interact gives multiple sources of variation. Since the genes of an individual are

dependent on the other genes of that individual and all individuals are dependent on what the others do, the mechanisms above result in a historic contingency. As for this model, diversity in behavior has also been demonstrated between and within natural fish populations (Brown et al., 2005a, Wark et al., 2011, Ariyomo and Watt, 2012). If the unpredictability produced by the behavioral architecture and the historic contingency represents natural populations, these studies indicate that diversity in genetics and neurobiological states can emerge without long term differences in environment.

4.2.2 Model-dependent instability

In the complexity of the architecture lays many opportunities for change that can contribute to more stable levels of fear. The developmental modulation system is currently linked to the sex-gene in the model and this yields clear differences between the sexes. However, it is possible to couple other genes together to achieve more stability. This could be to group all genes for a specific GOS or all genes for responses to a perception (e.g. all genes for the three neuronal responses for conspecifics) on the same chromosome and hence decrease the complexity. A way to increase the architectural complexity of this model is the addition of new modulation systems (in addition to the current developmental modulation system). These modulation systems (inherited as gene values, operating as hormones) can relate to personality traits like aggression, boldness, exploration etc. and can modulate many of the existing neuronal responses. This may lead to the evolution of distinct personalities in the model, as speculated by Giske et al. (2013). It is possible that this can result in convergence and stability of fear, but this remains to be seen. I have tried to achieve this by increasing the temporal resolution and the genetic complexity, with only negative results.

4.3 Increased genetic complexity

When the second or third set of components of the complex neuronal response was needed to pick the right GOS, this was mainly to become afraid during a predator attack (Figure 18). Contributing to the GOS 'Afraid' is light, conspecifics and predation. In most simulations, either light or predation contributed with > 90 % of the strength from the second and third set of

components (Figure 19). This illustrates that it is possible to achieve the same GOS, but by utilizing the neuronal responses in different ways.

A predator attack can be seen as one of the more complex features of the environment in this model since these events occur at random intervals and at all stages of life. Thus, when dealing with a more complex and unpredictable environment, a more complex neuronal response may seem to be beneficial. The level of complexity that the model is at today appears not to be in need for more complex neuronal responses. However, if the complexity of the environment is increased, an increase in complexity similar to what I have introduced can be beneficial.

With respect to the neuronal responses, different simulations emphasize the different factors in their environment in totally different ways (Figure 24-26). This has also been found in earlier versions of the model, without the increased complexity in the neuronal responses (Giske et al., 2013). In Equations 2-5 there are very many parameters that can evolve in relation to each other. This means that several distinctly different genetic parameter sets can evolve, all resulting in the same behavior, as also seen for ANNs (Strand et al., 2002). However, this can be an artefact because natural fish in the natural world live in much more complex environments. Their “equations” have an even larger amount of parameters and a model with even more complex equations might give more consistency between simulations.

The complex neuronal responses that evolved in the new version of the model use the second and third components to make relatively small alterations of the neuronal response function. One of these changes is to have still increasing response at high perceptions while keeping the sigmoid shape (Figure 27). The other change is to have graded response at weak perceptions also keeping the sigmoid shape (Figure 29). These two forms of the complex neuronal response function were not possible with only one component. It is therefore reasonable to think that these new functions are “better” in some way than the ones appearing in the version of the model by Giske et al. (2013). However, higher fitness was not found (Figure 20).

The number of generations the genetic algorithm used to find the best solution (highest level of fecundity) did not increase with the increase in genetic complexity (Figure 20). Therefore,

increasing the genetic complexity is not a problematic alteration to the model. Another reason why more generations were not needed could be that the additional genes were minimally utilized. Each neuronal response component can take the shape of a sigmoidal function, chosen for this and earlier versions of the model to make responses at very low perceptions possible. (For discussion see Brown and Holmes (2001), Brown et al. (2005b) and Bogacz et al. (2007).) Even though much more complex functions were allowed to evolve, they did not differ much from those allowed by a single sigmoid function. Since more genes for possibly more complex functions requires more hours of computation, more memory and more storage and did not yield faster or better solutions, it is not advantageous to include increased genetic complexity before the complexity of the architecture and environment in the model is increased.

4.4 Emotion systems in fish

The similarities between teleosts and tetrapods that were outlined in section 3.1 regarding anatomy, neurochemistry and behavior indicate that teleosts experience both pain and fear, a view supported by many (Sneddon et al., 2003, Braithwaite and Huntingford, 2004, Chandroo et al., 2004). The sufficient cognitive capacities, which are essential (at least according to some) for considering welfare (Duncan and Petherick, 1991), may therefore be present in fish. This is of major importance to problems regarding fish welfare: if fish do not suffer physically (pain) or mentally (fear), it may not matter if they are treated in a way that would inflict such discomfort in other species, e.g. mammals (Bermond, 1997).

Over the last decades there has been a substantial increase in the interest for fish welfare (Needham and Lehman, 1991, Chandroo et al., 2004, Branson, 2008, Braithwaite, 2010, Turnbull and Huntingford, 2012, Kittilsen, 2013). Research conducted with the purpose of increasing the welfare of fish will to some degree include harming living individuals. If models are developed that are able to replace research with living animals, this could be of great benefit for fish welfare research.

The most obvious benefit with using models in welfare research is that no real animals suffer. The number and length of experiments are only limited by computation time capacity. When

studying welfare in fish, both mental states and behavior are used as indicators (Martins et al., 2012), e.g. fear as an indicator of negative welfare and feed intake as an indicator of positive welfare (Huntingford et al., 2006). To measure a mental state like fear or even behaviors as proxies for these states in fish, are very hard. Working with a model, all data are available, e.g. the mental states of the modeled individuals.

In the model currently used, only the 'Hungry' and 'Afraid' states of the fish are included. A natural fish has many more states and to include some of these in the model can possibly improve its predictive value. However, my results imply that in order to arrive at a stable solution, a lot of computation time is required and even then there can be large variation between populations. This variation indicates that there are several equally good solutions to the use of the two states. More fundamental research as suggested above is needed before GOS can be modulated for natural populations, and it is then likely that more precise and complex modeling of both environment and physiology is required.

4.5 Sensitivity analysis of temporal resolution

As the initial simulations indicated, the experiment with the lowest resolution (four diel cycles and 67 time steps per cycle) gave simulations that were not stable and hence were not adapted. It is therefore not meaningful to run the model with so low resolution. For all other experiments, all traits (except fear) had reached a stable level after just about 1000 generations. In the next 3000 generations, there was little change. As such, if anything except the tendency to be hungry or afraid is to be studied with this model, the number of generations needed could be further adjusted down.

It was found that time steps per diel cycle and not number of diel cycle repetitions during a lifetime was most important for the number of eggs that can be produced in a generation (fecundity). This is not very surprising since the addition of more diel cycles only makes the individuals repeat the same behavior (migrate vertically). The increase of time steps per diel cycle gives the opportunity to make more choices during one cycle, both of GOS and of vertical

movement. It would have been interesting to see if further increase in resolution will result in even higher fecundities.

Changing the resolution from 200 to 600 time steps in the experiments gives a higher population fecundity that is due to an increase in body mass and not a decrease in mortality. The possibility to switch faster between the two GOS can be the reason for the increase in final body mass without increase in mortality and hence the increased population fecundity. In addition, the higher temporal resolution also gives the opportunity to change depth more frequently during a diel cycle.

There is a large effect of GOS when it comes to feeding and predation risk. When afraid, feeding is reduced to 10 % of the potential due to attention restriction (Giske et al., 2013). Therefore, feeding is mainly initiated by entering the GOS 'Hungry'. With low temporal resolution, the amount of food available in a time step when the fish is hungry is very large, maybe much larger than the individual really needs. By increasing this resolution an individual can choose the appropriate size of its meals by adjusting the number of time steps that it stays hungry. This is one possible mechanism that could explain the increase in body mass without increasing the mortality. However, the details behind such mechanisms can be very complicated and hard to extract.

Based on the current results, the model can be run with fewer diel cycles, but will benefit from an increased number of time steps per diel cycle. Four diel cycles and 600 time steps per cycle gives simulation of 2400 time steps per generation and this increases calculation time with 70 % compared to the previous standard. Dependent on what traits that is to be investigated this can be compensated for in fewer generations. Also, there may not be need to increase the number of time steps to 600 as 400 might be enough. However, to increase the number of time steps per diel cycle even more (e.g. 1200 per cycle) can possibly yield even better results.

All my experiments were based on 50 simulations with identical parameters. This required a large amount of computation time and also storage space for output data. In all experiments, there was

found little variation between simulations for all traits except level of fear. Based on this, the number of simulations can be heavily reduced in experiments where GOS is not the main focus.

A pattern in the overall level of fear between the experiments were not found, but the continual periods of fear (scaled to the maximum length of a life) become shorter when increasing the number of time steps in each diel cycle (Figure 17). This of course means that the relative length of the continual hunger periods also becomes shorter. More frequent changes of GOS gave increased fecundity, here found to be mainly due to an increase in body mass and not a decrease in mortality. How individuals achieved higher body mass without higher mortality rates is not known. Possible explanations can be a better fine tuning of the DVM or more precise meal sizes, but to get these answers, more research is needed.

An interesting experiment that could be done with this model in the future is to run it with a full annual cycle with seasonal changes. To run simulations with 365 diel cycles per year, and maybe several years per generation, would take too many hours of computation. Based on the current sensitivity analysis on temporal resolution, the resolution in each diel cycle is more important than the repetitions of cycles. Therefore, to run the model for one or more entire annual cycles, 12 diel cycles can be run with high temporal resolution (e.g. 600 time steps) and different seasonal environment and the individual result from each diel cycle (mortality risk and weight gain) can be multiplied with 30. As such, the model can be run for a full annual cycle with good temporal resolution without using an enormous amount of calculation time. Modeling seasonal cycles of mesopelagic fish have been done before, but then by use of Stochastic Dynamic Programming that did not account for density dependence for competition and predation risk (Rosland and Giske, 1997). This model also had a much coarser temporal resolution (time steps of five days). 17 years later, the computational power is available to increase the temporal resolution, as I have suggested.

5 Recommendations

The increased complexity of the neuronal responses was only utilized at a very small degree by the populations in these experiments and this did not result in increased population fecundity. This may be due to the lack of complexity of the environment and the behavioral architecture in the model. Increasing the genetic complexity as I did is therefore not needed before other changes to this model have been done.

To improve the quality of the output from the model, I recommend that it should be run with higher temporal resolution in each diel cycle (>200 time steps). However, the number of diel cycles can be reduced to save computation time. Also, the number of simulations per experiment and the length of these can be reduced. I suggest experiments with a number of generations between 2000 and 4000, reducing computation time more than tenfold compared to Giske et al. (2013) where simulations were 50,000 generations long. It is possible that even more than 600 time steps per diel cycles will give better results and to determine this there has to be done more research.

To further investigate levels of fear within and among populations the effects of complexity in the architecture of the model can be explored. This can be done by increasing the number of GOS, implementing and connecting new modulating systems or changing the genetic complexity. These changes can be accompanied by a more complex model environment.

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7 Appendices

Contents

A	Model description	83
A.1	Environment	83
A.2	Reproduction	84
A.1	References in Appendix A.....	85
B	Major changes to the Fortran code HED23.f90.....	85
B.1	Adding multiple neuronal responses.....	85
B.2	New calculation of the length of a predator attack	86
C	Data processing with a Fortran program	87
C.1	SR19.....	87
C.2	SR13.....	105
D	Figures for neuronal responses and their strength: Simulations 6-10.....	113
E	Figures for all 9 experiments: Two figures for each experiment	117
F	Comparison of length of fear periods	133
G	Matlab script for statistical comparison of experiments and making figures.....	134
G.1	Explanation.....	134
G.2	The script.....	134
H	Matlab script for efficient uploading processes to FIMM.....	138
H.1	Explanation for the script “Simulations on FIMM”	138
H.2	The script.....	138

A Model description

A.1 Environment

The model consists of a population of mesopelagic planktivorous fish, performing diel vertical migration and starts off with 10.000 individuals which is also the highest possible number. Diel cycles are represented by cycles of surface light intensity (Equation A1) and the light is attenuated down in the water column according to Beer's law (Aksnes and Giske, 1993, Aksnes and Utne, 1997).

$$L(t) = L_{max} 0.5 \sin\left(\frac{\pi dt}{\Omega}\right) \quad [A1]$$

assumes 50 % scattering in the surface layer and gives the light intensity just beneath this layer, $L(t)$. Also, d is the number of diel cycles in a simulation, t the current time step, Ω the total number of time steps and L_{max} the maximum surface irradiance. Prey also moves vertically, following the light with a vertical bell-shaped distribution. The amount of food an individual can consume in a time step $f_c(z, t)$ is the dependent of the attention a , visual range $r(z, t)$, local prey density $C(z, t)$ and number of conspecifics in the same depth $N(z, t)$ where z is the depth and t is the time step:

$$f_c(z, t) = a\pi r(z, t)^2 C(z, t) N(z, t)^{-1} \quad [A2]$$

However, constraining the food consumption is the stomach capacity of the individual. Calculation of visual range is based on the improved model for visual range in fish by Aksnes and Utne (1997). Every time step, the prey population is renewed, bringing prey densities back to initial concentrations. The environment also varies between generations. Each generation can exhibit one of nine fluctuation patterns which again contain shorter random fluctuations (full description of the environments in the online supplementary in Giske et al. (2013)). In the generation long fluctuations the rate of predation, prey density and light density are different. One of these patterns (Normal) is used when comparing simulations and are those presented in the results. The 'Normal' generations have intermediate levels of predation risk and food

concentration and the last generation of a simulation is always a ‘Normal’ one. It is also the most common of the nine generation long fluctuation pattern (occurring in 20 % of the generations) and have both intermediate food concentration and predation risk in addition to a fixed number (four) of predator attacks starting when a fixed fraction of the time steps have passed. The standardization of the predator attacks in time is done make accurate comparison of simulations possible.

During the whole life cycle of an individual, there is always a relatively low predation risk. The predators that are eating the planktivorous fish are visual feeders and hence the risk of being eating diminishes with light intensity down in the water column (Aksnes and Utne, 1997). Larger body mass also increases the chance of being detected. Predation risk $M(z,t)$ is determined by

$$M(z,t) = am(t)r(z,t)^2BN(z,t)^{-0.5} \quad [A3]$$

Where a is the fraction of attention towards predators derived from the developmental modulation genes, $m(t)$ the background mortality, $r(z,t)$ the visual range of the fish, B is body mass and $N(z,t)$ is the number of fish at a specific depth. A few times in every generation, there is an attack from a school of predators. This increases the predation risk to 10 times the background risk.

A.2 Reproduction

Generations are non-overlapping and reproduction happens only at the end by those who survive. Females produce eggs dependent of own body mass and search for males in the ambient depth. The largest among three randomly encountered males, are chosen. All six genes for a neuronal response are inherited together on one separate chromosome. The sex gene along with the four D-genes are located on a chromosome together and hence allow differences between the sexes. This chromosome with its five genes makes up one of the in total 10 chromosomes with 59 genes altogether.

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B Major changes to the Fortran code HED23.f90

B.1 Adding multiple neuronal responses

To test the effects of increased genetic complexity in the model, the number of genes was increased. In the code, this was done by extending the genes with one dimension. The result were more genes, I run the model with three for each trait. This gives three simple neuronal response functions to make up one complex function. An example of code that is extended with this extra dimension is given under. Such alterations were done in the entire code.

Extending all genes with an extra dimension (n) that is the number of simple neuronal responses:

```
do n = 1, NRcomp !Number of neuronal responses
  do gn = 1, 3 !Components of each gene, only 2 (x and y) used. 'egg' identifies a
newbord individual
    geneAflight(egg, n, gn) = min(max(EGGAflight(egg, n, gn), allmin), allmax)
    geneAFother(egg, n, gn) = min(max(EGGAfother(egg, n, gn), allmin), allmax)
    geneAFmort(egg, n, gn) = min(max(EGGAFmort(egg, n, gn), allmin), allmax)
    geneAHstom(egg, n, gn) = min(max(EGGAHstom(egg, n, gn), allmin), allmax)
    geneAHfood(egg, n, gn) = min(max(EGGAHfood(egg, n, gn), allmin), allmax)
    geneHflight(egg, n, gn) = min(max(EGGHflight(egg, n, gn), allmin), allmax)
    geneHfother(egg, n, gn) = min(max(EGGHfother(egg, n, gn), allmin), allmax)
    geneHHfood(egg, n, gn) = min(max(EGGHHfood(egg, n, gn), allmin), allmax)
    geneHHother(egg, n, gn) = min(max(EGGHHother(egg, n, gn), allmin), allmax)

    mutAflight(egg, n, gn) = mEGGAflight(egg, n, gn)
    mutAFother(egg, n, gn) = mEGGAfother(egg, n, gn)
    mutAFmort(egg, n, gn) = mEGGAFmort(egg, n, gn)
    mutAHstom(egg, n, gn) = mEGGAHstom(egg, n, gn)
    mutAHfood(egg, n, gn) = mEGGAHfood(egg, n, gn)
    mutHflight(egg, n, gn) = mEGGHflight(egg, n, gn)
    mutHfother(egg, n, gn) = mEGGHfother(egg, n, gn)
    mutHHfood(egg, n, gn) = mEGGHHfood(egg, n, gn)
    mutHHother(egg, n, gn) = mEGGHHother(egg, n, gn)
  end do
end do !n
```

B.2 New calculation of the length of a predator attack

The number of attacks is the same in each of the nine experiments. To make the accumulated risk the same in all experiments, the number of time steps had to be adjusted according to the number of time steps in the experiment. For this purpose, a new variable was introduced, 'attacklength'. In the old code, predation risk was increased over a few time steps before reaching the maximum risk. This is not done in this new version.

Code for calculating length of predator attack:

```
!BSA 16.12.13
attacklength = NINT((1.*flifespan) / (67. * 4.))
!67*4 is set because length of maximum risk (attack) when flifespan=1400 is 5.
```

(following if-statement done for all ages)

```
if (dev > 1) then ! predator schools may attack at any random time step
  if (attack == 0) then !no current predator attack
    call random_number (rand)
    if (rand < attrisk) then ! attack initiated just now
      if (screenplot == 2) write(6,*)"attack now"
      oldrisk = autorisk !memory of risk before attack (and after)
      attack = 1 !current status
      attacks = attacks + 1 !sum of attacks in generation
      duration = 1 !counter for duration of attack
      danger = 10 !present elevation BSA 16.12.13 set to 10
      autorisk = oldrisk * danger
    end if
  else !ongoing attack
    duration = duration + 1
    if (duration .le. attacklength) then
      ! danger = 4 !BSA 16.12.13 removed
      ! autorisk = oldrisk * danger
      !else if (duration < 8) then
        danger = 10
        autorisk = oldrisk * danger
      else !attack terminated at time step 8
        attack = 0
        duration = 0
        danger = 1
        autorisk = oldrisk !background risk reestablished
      end if !duration
    end if ! attack = 0
  else !dev = 1, i.e. "Normal"
    !5 fixed time steps of initiation of attack
    if (age == flifespan/7 .or. age == flifespan/3 .or. age == int(0.6*flifespan) .or.
age == int(0.85*flifespan)) then
      oldrisk = autorisk !memory of risk before attack (and after)
      attack = 1 !current status
      attacks = attacks + 1 !sum of attacks in generation
      duration = 1 !counter for duration of attack
```

```

    danger = 2    !present elevation
    autorisk = oldrisk * danger
    else if (attack == 1) then !attack is already going on
        duration = duration + 1
    if (duration .le. attacklength) then
        ! danger = 4 !BSA 16.12.13 removed
        ! autorisk = oldrisk * danger
        !else if (duration < 8) then
            danger = 10
            autorisk = oldrisk * danger
        else    !attack terminated at time step 8
            attack = 0
            duration = 0
            danger = 1
            autorisk = oldrisk    !background risk reestablished
        end if !duration
    end if !age
end if !dev

```

C Data processing with a Fortran program

Data from the simulations are run through another Fortran program for further processing after the simulations are done. The program consists of many subroutines. The two subroutines (SR19 and SR13) that I worked the most with, and done most changes to, are presented here.

C.1 SR19

This subroutine had to be written from scratch to process the new data that the model produces due to the adding of multiple components of the neuronal responses. This subroutine ranks the three components due to their strength and then finds the fraction of the total strength of the complex neuronal response that is due to the strongest (NR1) of the three components. It also calculates how many sets of neuronal responses that are needed to find the GOS that the individual choose in the actual simulation.

```

!-----
subroutine
SR19NRComponents(runtag,first,files,milestone,flifespan,datetag,dumpinterval,normal) !BSA
28.01.14
!-----
!Find the effects of each component in complex neuronal response functions
!in each age in last generation in all simulations
! - find the fraction of strength of each complex NR from parts 1, 2 and 3
! - find the probability of change in GOS from 1 to 2 components, and from 2 to 3

```

```

implicit none
!Variables used in this Subroutine
integer g,j,e,z,b,c,d,n,first,files,milestone,nextdir,flifespan
integer dummy,dummy1,fpop,forget(12),new,newz
integer MS,a,i,s,IND,nr,test,test2,jump,comps,comp,choice,zrange
integer numfish(30,9600),autocop(30,9600),alive(2,9600)
integer comphabval(4,9600),comp1GOS(9600),comp2GOS(9600),comp3GOS(9600),comp4GOS(9600)
!BSA 30.01.14
integer idepth(9600),iweight(9600),dumpinterval,za,neu !BSA 28.01.14
integer thirdH,thirdA !BSA 28.04.14
real streHs, streHF, streFl, streFo, streFm !BSA 28.04.14
  real stocap(9600), normal !BSA 28.01.14
real hunger(3,10000),fear(3,10000) !BSA 31.01.14
real a1(3),a2(3),a4(3),a5(3),a6(3),a7(3),a8(3),a9(3),BM,rdummy1,rdummy2,rdummy3,rdummy4
real
p1,p2,p3,p4,p5,p7,p8,FL(5,3),FO(5,3),HF(5,3),HO(5,3),HdomF(5,3),HdomO(5,3),FdomL(5,3),Fdom
O(5,3)
real sumlight,sumother,sumotherA,sumotherH,sumfood,summort,sumstom,living,Hsize
real errorP,skip(7),MaxPercept(7),finfo(19)
real autorisk(9600),visran(30,9600),zlight(30,9600)
real geneAHsize(4),geneAFlight(3,2),geneAFother(3,2),geneAFmort(3,2),geneAHstom(3,2) !BSA
29.01.14, changed from (2,3) to (3,2)
real geneAHfood(3,2),geneHFlight(3,2),geneHFother(3,2),geneHHfood(3,2),geneHHother(3,2)
real
gamma2gene,gamma3gene,linearABM,linearABM2,ABMgenes(4),frac(9,3,9600),strength(9,3,9600)
!BSA 10.02.14
real
Flight(3),Fother(3),Fmort(3),Hstom(3),Hfood(3),lightA(3,2),otherA(3,2),foodH(3,2),otherH(3
,2)
real sumfrac(9,3) !BSA 30.01.14
real avgstrength(9,3), avgfrac(9,3) !BSA 11.02.14
integer rank(9,3) !BSA 30.01.14
real interval(9,3) !BSA 03.02.14
real Hsize1,Hsize2
real hu1,hu2,hu3,fe1,fe2,fe3,e1,e2
real light32(2),food32(2),cons32(2) ! BSA 03.02.14
real sumHO,sumHF,sumFL,sumFO,r ! BSA 03.02.14
real habval(3,5),value !BSA 04.02.14
character (1) GOS(9600)
character(12) T12
character(7) T7 !BSA 28.01.14
character(8) T8 !BSA 28.01.14
character(6) runtag
character(20) string1 !BSA 28.01.14
character(11) string8 !BSA 28.01.14
character(10) string9 !BSA 28.01.14
character(7) string10 !BSA 28.01.14
character(45) stringMAX
character(51) stringENV
character(45) stringHAB
character(45) stringHED
character(56) stringZ !BSA 28.01.14
character(56) stringBM !BSA 28.01.14
character(56) stringMS
character(56) stringSTO
character(49) stringNRs
character(48) stringNRstrength !BSA 10.02.14
character(32) stringAVGF !BSA 11.02.14

```

```

character(32) stringAVGS !BSA 11.02.14
character(4) datetag !BSA 28.01.14

```

```

write(6,*) "starting SR19NRComponents"
!write(6,*) "milestone ", milestone
write(6,*) "runtag ", runtag
test = 0 !1 if searching for errors
test2 = 0

```

```

!set level of individual error-making
errorP = 0.1 !BSA 28.01.14

```

```

!for each simulation SIM:
  !open files for each simulation (BSA: First part of the names to the right are old, but
  that does not matter)
  ! read max perception in final generation for each simulation from HED18-JerP10-Exx-
o014-max-perception.txt
  ! read environment each depth & time from HED18-JerP10-Exx-
o029-environment-each-age.txt
  ! read habitat each depth & time from HED18-JerP10-Exx-
o011-habitat-part10.txt
  !read data for one and one individual from these files
  ! read genes of each individual from HED18-JerP10-Exx-
o030-HEDgene-part10.txt
  ! read depth for each ind each age from HED18-JerP10-Exx-
o060-depth-all-ages-genr-50000.txt
  ! read body mass for each ind each age from HED18-JerP10-Exx-
o061-bmass-all-ages-genr-50000.txt
  ! read stomach for each ind each age from HED18-JerP10-Exx-
o063-stcap_rst-all-ages-genr-50000.txt
  ! read motivational state for each ind each age from HED18-JerP10-Exx-
o062-affect-all-ages-genr-50000.txt
  ! calculate the contribution to the statistics for this individual throughtout all ages
it was alive
  !next ind
  !close all input files
  !
  !write output file for each SIM of ..
  !for each age
  !      NR      fraction of NR strength      fraction decided by component
  !      (#      1      2      3      1      2      3) x 9
!read for next simulation

```

```

nextdir = milestone
if (nextdir > 9999999) then
  write(string8,1008) "g-",nextdir
  else if (nextdir > 9999999) then
  write(string8,1007) "ge-",nextdir
  else if (nextdir > 9999999) then
  write(string8,1006) "gen-",nextdir
  else if (nextdir > 999999) then
  write(string8,1005) "gene-",nextdir
  else if (nextdir > 99999) then
  write(string8,1004) "gener-",nextdir
  else if (nextdir > 999) then
  write(string8,1003) "genera-",nextdir

```



```

else if (nextdir > 99) then
  write(string8,1002) "generat-",nextdir
else if (nextdir > 9) then
  write(string8,1002) "generati-",nextdir
else
  write(string8,1001) "generatio-",nextdir
end if
1001 format(A9,I2)
1002 format(A8,I3)
1003 format(A7,I4)
1004 format(A6,I5)
1005 format(A5,I6)
1006 format(A4,I7)
1007 format(A3,I8)
1008 format(A2,I9)

if (nextdir > 9999999) then ! BSA 28.01.14 different string for o062
  write(string9,1608) "g-",nextdir
  else if (nextdir > 999999) then
    write(string9,1607) "ge-",nextdir
  else if (nextdir > 99999) then
    write(string9,1606) "gen-",nextdir
  else if (nextdir > 9999) then
    write(string9,1605) "gene-",nextdir
  else if (nextdir > 999) then
    write(string9,1604) "gener-",nextdir
  else if (nextdir > 99) then
    write(string9,1603) "genera-",nextdir
  else if (nextdir > 9) then
    write(string9,1602) "generat-",nextdir
  else
    write(string9,1601) "generati-",nextdir
end if
1601 format(A9,I1)
1602 format(A8,I2)
1603 format(A7,I3)
1604 format(A6,I4)
1605 format(A5,I5)
1606 format(A4,I6)
1607 format(A3,I7)
1608 format(A2,I8)

if (nextdir > 9999999) then ! BSA 28.01.14 different string for o063
  write(string10,1506) "g-",nextdir
  else if (nextdir > 999999) then
    write(string10,1506) "ge-",nextdir
  else if (nextdir > 99999) then
    write(string10,1506) "-",nextdir !BSA This does not work for 100k or more. (!)
  else if (nextdir > 9999) then
    write(string10,1505) "g-",nextdir
  else if (nextdir > 999) then
    write(string10,1504) "gn-",nextdir
  else if (nextdir > 99) then
    write(string10,1503) "gnr-",nextdir
  else if (nextdir > 9) then
    write(string10,1502) "genr-",nextdir
  else
    write(string10,1501) "gener-",nextdir

```

```

end if
1501 format(A6,I1)
1502 format(A5,I2)
1503 format(A4,I3)
1504 format(A3,I4)
1505 format(A2,I5)
1506 format(A1,I6)
!1507 format(A3,I7)
!1508 format(A2,I8)

!BSA: Opening AVG-files
write(stringAVGF,7010)"HED23-",datetag,runtag,"o048-NR-AVGF.txt"
write(stringAVGS,7011)"HED23-",datetag,runtag,"o049-NR-AVGS.txt"
7010 format(A6,A4,A6,A16) !BSA 11.02.14
7011 format(A6,A4,A6,A16) !BSA 11.02.14
open(48, file = stringAVGF) !BSA 11.02.14
open(49, file = stringAVGS) !BSA 11.02.14

write(48,4802)"sim", "f1cFLIG", "f2cFLIG", "f3cFLIG", "f1cFOTH", "f2cFOTH", "f3cFOTH", "f1cFPRD",
"f2cFPRD", "f3cFPRD", &

"f1cHSTO", "f2cHSTO", "f3cFSTO", "f1cHFOD", "f2cHFOD", "f3cHFOD", "f1cALIG", "f2cALIG", "f3cALIG",
&

"f1cAOTH", "f2cAOTH", "f3cAOTH", "f1cHFOD", "f2cHFOD", "f3cHFOD", "f1cHOTH", "f2cHOTH", "f3cHOTH"
write(49,4802)"sim", "f1sFLIG", "f2sFLIG", "f3sFLIG", "f1sFOTH", "f2sFOTH", "f3sFOTH", "f1sFPRD",
"f2sFPRD", "f3sFPRD", &

"f1sHSTO", "f2sHSTO", "f3sFSTO", "f1sHFOD", "f2sHFOD", "f3sHFOD", "f1sALIG", "f2sALIG", "f3sALIG",
&

"f1sAOTH", "f2sAOTH", "f3sAOTH", "f1sHFOD", "f2sHFOD", "f3sHFOD", "f1sHOTH", "f2sHOTH", "f3sHOTH"
4802 format(28A14)

do nr = first,files
  write(6,*)"starting simulation nr",nr

  !Changing datetags and skipping crash-simulations
  if (nr == 20) GOTO 4343 ! D8 200
  if (nr == 1) datetag = '1219'
  if (nr == 2) datetag = '1219'
  if (nr == 3) datetag = '1220'
  if (nr == 4) datetag = '1219'
  if (nr == 5) datetag = '1220'

  !reset arrays for this SIM
  do a = 1,flifespan
    comp1gos(a) = 0
    comp2gos(a) = 0
    comp3gos(a) = 0
    comp4gos(a) = 0 !BSA 30.01.14
    do comp = 1,4
      comphabval(comp,a) = 0
    end do
    do MS = 1,2
      alive(MS,a) = 0
    end do !MS
  do n = 1,9

```

```

do comp = 1,3
  frac(n,comp,a) = 0.
  strength(n,comp,a) = 0.
end do
end do !n
end do
!BSA 28.01.14

!!! Opening files !!!

if (nr < 10) then
  write(string1, 5001)"HED23-",datetag,runtag,"-E0",nr !BSA 28.01.14
else
  write(string1, 5002)"HED23-",datetag,runtag,"-E",nr !BSA 28.01.14
end if
write(6,*)" "
write(6,*)"now starting simulation ", string1
5001 format(A6,A4,A6,A3,I1) !BSA 28.01.14
5002 format(A6,A4,A6,A2,I2) !BSA 28.01.14

!files for input data
write(stringMAX,7001)string1,"-o014-max-perception.txt"
write(stringENV,7002)string1,"-o029-environment-each-age.txt"
write(stringHAB,7001)string1,"-o011-habitat-part10.txt"
write(stringHED,7001)string1,"-o030-HEDgene-part10.txt"
write(stringZ,7003) string1,"-o060-depth-all-ages-",string8,".txt" !BSA 28.01.14
write(stringBM,7003) string1,"-o061-bmass-all-ages-",string8,".txt"
write(stringMS,7007) string1,"-o062-affect-all-ages-",string9,".txt"
write(stringST0,7008)string1,"-o063-stcap_rst-all-ages-",string10,".txt"
!files for output of results
write(stringNRs,7005)string1,"-o042-NR-components.txt"
write(stringNRstrength,7009)string1,"-o043-NR-strengths.txt"!BSA 20.02.14
7001 format(A20,A24) !BSA 28.01.14
7002 format(A20,A30) !BSA 28.01.14
7003 format(A20,A21,A11,A4) !BSA 28.01.14
7007 format(A20,A22,A10,A4) !BSA 28.01.14
7008 format(A20,A25,A7,A4) !BSA 28.01.14
7004 format(A16,A25,F5.2,A4)
!7005 format(A16,A23,F5.2,A4)
7005 format(A20,A23) !BSA 28.01.14
7009 format(A20,A22) !BSA 10.02.14
7006 format(A16,A22,F5.2,A4)

open(11, file = stringHAB)
open(14, file = stringMAX)
open(29, file = stringENV)
open(30, file = stringHED)
open(60, file = stringZ)
open(61, file = stringBM)
open(62, file = stringMS)
open(63, file = stringST0)
open(42, file = stringNRs)
open(43, file = stringNRstrength) !BSA 10.02.14

write(6,*)"reading file 29"
!jump = (milestone-1)*flifespan + 1 !(milestone-1) generations, flifespan ages and 1
heading line

```

```

jump = (((dumpinterval*normal) - 1)*flifespan) + 1 !dumped generations (200) -1,
flifespan and 1 heading line, !BSA 28.01.14
do g = 1,jump
  read (29,*) !skip all but the last generation
end do

!read environment information for each age
do a = 1,flifespan
  read(29,2929) dummy,T12,dummy1,rdummy1,rdummy2,rdummy3,rdummy4,autorisk(a) !, &
  !           Rage,foodmax,Rage,autocop(foodmax,age),Rage,autotemp(foodmax), &
  !           alive,avgdepth,neighbors,mortnow,feednow,growthnow,fearlevel
end do
2929 format(I12,A12,I10,F10.3,F10.2,F10.3,2F10.5)
!,F10.3,I10,F10.3,I12,F10.3,F12.2,I10,F12.2,F12.0,2F12.4,2F12.3)

write(6,*) "reading file 11"
jump = 10*flifespan*33 !generations, ages, 30 z + 3 heading lines
do g = 1,jump
  read (11,*) !skip all but the last generation
end do
do a = 1,flifespan
  read (11,*) !skip 3 heading lines
  read (11,*) !skip 3 heading lines
  read (11,*) !skip 3 heading lines
  do z = 1,30
    read(11,1102) dummy,dummy,dummy,zlight(z,a),visran(z,a),T7,autocop(z,a),numfish(z,a)
!BSA 28.01.14, added T7
!           ,Hnum(dep),Anum(dep),HavgBM(dep),AavgBM(dep),Havgcost, &
!           Aavgcost,HavgPstAvail(dep),AavgPstAvail(dep),didgain(dep), &
!           fgain(dep),restattention*fgain(dep),didrisk(dep),frisk(dep), &
!           Hrisk,Arisk,100.*ffeeling(dep,age)
  end do !z
end do !a
1102 format (I5,2I5,F10.4,F9.4,A7,I8,I7) !I7,2F8.1,4F7.1,F10.1,3F10.3,3F13.7,F10.1)

close(11)
close(29)

!read maximum perception data
write(6,*) "reading file 14"
write(6,1404)"Ex", "Stomach", "Light", "Agents", "Bodymass", "Prey", "Age", "Risk"
do g = 1,milestone + 1
  read (14,*) !skip all but the last line
end do
read (14,1402)dummy,(skip(i),i=1,7),(MaxPercept(i),i=1,7)
write(6,1403)nr,(MaxPercept(i),i=1,7)

close(14)
1402 format(I8,16F12.2)
1403 format(I12,7F12.2)
1404 format(8A12)

!read individual-data for genetics
write(6,*)"now reading ", stringHED
!read genetic data for each individual
!This file contains the data for the 10 last generations

```

```

!The procedure is to skip the 9 first and read the tenth
do g = 1,9 !the 9 first and unused generations in the file
  read(30,*) !skip first line with simulation number
  read(30,*) !skip second line with generation number
  read(30,*) !skip third line with survivors
  read(30,3001)fpop !read number of inds to be skipped
  do i = 1,fpop+1
    read(30,*) !skip heading line line and all inds in generation
  end do
end do

read(30,*) !skip first line with simulation number
read(30,3001) dummy !read second line with generation number
read(30,*) !skip third line with survivors
read(30,3001)fpop !read number of inds to be read
write(6,*) "generation",dummy,"has",fpop,"individuals. Reading file."
read(30,*) !skip heading line for last generation
!read individual-data for each age
read(60,*) !heading
read(60,*) !heading
read(61,*) !heading
read(61,*) !heading
read(62,*) !heading
read(62,*) !heading
read(63,*) !heading
read(63,*) !heading

!----- LONG individual-loop starting here -----
!data are read individual-by-individual to save memory (individuals are not in arrays),
!but at the cost of slower reading of files 30 and 60-63.

do while (.not. EOF(30)) !read all inds in this sim
  read(30,3010) ind,(forget(j), j = 1,7), & ! BSA 29.01.14 modiefied
    (forget(j), j = 1,3),rdummy1, forget(j),rdummy2, &
    rdummy3,forget(j),T8, (forget(j), j = 1,10),(forget(j), j = 1,5), &
    rdummy1,rdummy2, &
    (geneAHsize(g),g = 1,4),((geneAFlight(n,g),g = 1,2),n=1,3), &
!gABM = 4
    ((geneAFother(n,g),g = 1,2),n=1,3),((geneAFmort(n,g),g = 1,2),n=1,3),
&
    ((geneAHstom(n,g),g = 1,2),n=1,3), &
    ((geneAHfood(n,g),g = 1,2),n=1,3),((geneHFlight(n,g),g = 1,2),n=1,3),
&
    ((geneHFother(n,g),g = 1,2),n=1,3),((geneHHfood(n,g),g = 1,2),n=1,3),
&
    ((geneHHother(n,g),g = 1,2),n=1,3)
  read(60,6102)i,(idepth(a), a=1,flifespan)
  read(61,6103)i,(iweight(a), a=1,flifespan)
  read(62,6104)i,(GOS(a), a=1,flifespan)
  read(63,6106)i,(stocap(a), a=1,flifespan)

  3001 format(I6)
  3010 format(8I8, &
!i,fgender(i),fbd,frd,fmove,fdeathdepth(i),fdeathage(i),fdeathaffect(ind),
  3I8,F8.0,I8,F8.0, &
!fdeathBM(ind),fstatus(i),fnumerafraid(i),fbodymax(ind),strBM,fbodymass(i),

```

```

        F8.3,I8,A8,15I8, &
!fgonad(i),foffspring(i),"x",motherpop(i),mcreation(i),gcreation(i),acreation(i),icreation
(i),
        2F8.4,          & !(motherstring(i,e1),e1 = 1,motherlength),(geneMemo(i,g),g
= 1,2),
        200F8.2)          !(geneAHsize(i,g),g = 1,gABM),(geneAFlight(i,ng),g = 1,1-
3), n = 1,NRcomp), etc

6102 format(I6,14000I4)
6103 format(I6,14000I6)
6104 format(I6,14000A4)
6106 format(I6,14000F6.2)

!write(6,*)"find NRs of hungry and afraid"
!Perceptions, Neuronal responses and size-dependent developmental modulation

do a = 1,flifespan
  do comp = 1,3
    do neu = 1,5
      strength2(neu,comp,a) = 0.
    end do
  end do
end do

!!!! Take the organism through all ages !!!!
do a = 1,flifespan

  if (a > 1) then !test for alive
    if(iweight(a-1) < 100.) goto 990
  end if

  !Global Organismal State
  if (GOS(a) == "H") then
    MS = 1
  else
    MS = 2
  end if

  !count inds alive in each MS
  alive(MS,a) = alive(MS,a) + 1
  !reset arrays and variables
  !do comp = 1,3
  ! fear(comp,a) = 0.
  ! hunger(comp,a) = 0.
  !end do !comp

  do n = 1,9
    do c = 1,3 !reset
      sumfrac(n,c) = 0.
    end do
  end do !n

  !Perception of predators is the same for all inds
  finfo(19) = autorisk(a)/MaxPercept(7)

  !Developmental modulation
  do g = 1,4
    ABMgenes(g) = geneAHsize(g)
  end do
end do

```

```

end do

!find body mass (finfo(12))
!Bodymass in this routine is BEFORE behaviour in time step, so it is the stored BM
from the time before.
if (a == 1) then
    BM = 250. !the birth weight of all inds (cop2egg)
else
    BM = iweight(a-1)
end if

!rescale BM to fit finfo(12) in the 0-1 range:
BM = BM/MaxPercept(4)
!interpolate between nearest two values to find F&Hsize at current BM
e1 = 0.1
e2 = -.1
Hsize1 = linearABM2(ABMgenes,BM,4,e1)
Hsize2 = linearABM2(ABMgenes,BM,4,e2)
if (MS == 1) then
    Hsize = max(Hsize1,Hsize2)
else
    Hsize = min(Hsize1,Hsize2)
end if
b = max(a-1,1) !last age, where 0 --> 1
z = idepth(b) !the position of the fish BEFORE its move
!Fear of light
finfo(4) = zlight(z,a)/MaxPercept(2)

!Fear reduction by others
!if (a == 1) then
    finfo(9) = numfish(z,a)/MaxPercept(3)
! else
!     ! finfo(9) = 0.5*(numfish(z,a-1)+numfish(z,a))/MaxPercept(3)
!     finfo(9) = numfish(z,a-1)/MaxPercept(3)
! end if

!Hunger from stomach
! full capacity is MAXSTOMCAP * body mass
!comp1 = 1.- fstomach(ind,1)/(maxstomcap*fbodymass(ind)) !fraction of allowed capacity
remaining
!error coorection from stored values:
!correct C: comp1 = 1-stom/(0.15BM)
!stored L: Lastgenstocap = 1-stom/(0.25BM)
! 5/3 (L-1) = stom/0.15BM = K
! K = 5/3 - 5/3L
! C = 1 - K = 1 - 1.66 + 5/3L = 5/3L - 2/3
finfo(1) = 1.666*stocap(a) - 0.666 !BSA 05.02.14 changed from b to a
finfo(1) = finfo(1)/MaxPercept(1)
!Hunger from food
!finfo(15) = visran(z,a)*visran(z,a)*autocop(z,b)/MaxPercept(5)
finfo(15) = visran(z,b)*visran(z,b)*autocop(z,b)/MaxPercept(5)

!Neuronal responses
p1 = finfo(1)
p2 = finfo(19)
p3 = finfo(9)
p4 = finfo(4)
p5 = finfo(15)

```

```

do comp = 1,3
  ! gamma2gene is a function that calculates the response based on the perception
  and the error
  Flight(comp) = gamma2gene(10*geneAFlight(comp,1),10*geneAFlight(comp,2),p4,errorP)
  Fother(comp) = gamma2gene(10*geneAFother(comp,1),10*geneAFother(comp,2),p3,errorP)
  Fmort(comp) = gamma2gene(10*geneAFmort(comp,1),10*geneAFmort(comp,2),p2,errorP)
  Hstom(comp) = gamma2gene(10*geneAHstom(comp,1),10*geneAHstom(comp,2),p1,errorP)
  Hfood(comp) = gamma2gene(10*geneAHfood(comp,1),10*geneAHfood(comp,2),p5,errorP)
  sumfrac(1,comp) = sumfrac(1,comp) + Flight(comp)
  sumfrac(2,comp) = sumfrac(2,comp) + Fother(comp)
  sumfrac(3,comp) = sumfrac(3,comp) + Fmort(comp)
  sumfrac(4,comp) = sumfrac(4,comp) + Hstom(comp)
  sumfrac(5,comp) = sumfrac(5,comp) + Hfood(comp)
  ! fear(comp,a) = (1.-Hsize) * (Flight(comp) - Fother(comp) + Fmort(comp))
  ! hunger(comp,a) = Hsize * (Hstom(comp) + Hfood(comp))
end do !comp

!!!What fraction of total NR did each component contain?
!!sumlight = Flight(1) + Flight(2) + Flight(3)
!!sumother = Fother(1) + Fother(2) + Fother(3)
!!summort = Fmort(1) + Fmort(2) + Fmort(3)
!!sumstom = Hstom(1) + Hstom(2) + Hstom(3)
!!sumfood = Hfood(1) + Hfood(2) + Hfood(3)
!!do comp = 1,3
!! if (sumlight > 0.) frac(1,comp,a) = frac(1,comp,a) + Flight(comp)/sumlight
!! if (sumother > 0.) frac(2,comp,a) = frac(2,comp,a) + Fother(comp)/sumother
!! if (summort > 0.) frac(3,comp,a) = frac(3,comp,a) + Fmort(comp)/summort
!! if (sumstom > 0.) frac(4,comp,a) = frac(4,comp,a) + Hstom(comp)/sumstom
!! if (sumfood > 0.) frac(5,comp,a) = frac(5,comp,a) + Hfood(comp)/sumfood
!!end do
end do !a
990 continue !with next ind, after skipping dead fish

!rank the 3 components of each NR by numerical significance in falling order
do n = 1,5
  do c = 1,3
    rank(n,c) = c !initiate assumed rank order: 1,2,3
  end do
  do comp = 2,3
    do c = 1,comp-1 !compare with better ranked components and change rank order
if wrong
      if (sumfrac(n,comp) > sumfrac(n,c)) then
        rank(n,comp) = rank(n,comp) - 1
        rank(n,c) = rank(n,c) + 1
      end if
    end do !c
  end do !comp
end do !n

if (ind == 100 .or. ind == 200 .or. ind == 300) then
  write(6,*)ind
  write(6,166) (Flight(c),c= 1,3), (sumfrac(1,c),c= 1,3), (rank(1,c),c= 1,3)
  write(6,166) (Fother(c),c= 1,3), (sumfrac(2,c),c= 1,3), (rank(2,c),c= 1,3)
  write(6,166) (Fmort(c),c= 1,3), (sumfrac(3,c),c= 1,3), (rank(3,c),c= 1,3)
  write(6,166) (Hstom(c),c= 1,3), (sumfrac(4,c),c= 1,3), (rank(4,c),c= 1,3)

```



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    write(6,166) (Hfood(c),c= 1,3), (sumfrac(5,c),c= 1,3), (rank(5,c),c= 1,3)
end if
166 format (6E12.4,3I3)

    !reset arrays and variables
do n = 1,9
    do c = 1,3 !reset
        sumfrac(n,c) = 0.
    end do
end do !n

!Recalculate rank-sorted NR-compoents through all ages
do a = 1,flifespan

    if (a > 1) then !test for alive
        if(iweight(a-1) < 100.) goto 994
    end if

!Global Organismal State, BSA uncommented
if (GOS(a) == "H") then
    MS = 1
else
    MS = 2
end if
!count inds alive in each MS
! alive(MS,a) = alive(MS,a) + 1

!reset arrays and variables
do comp = 1,3
    fear(comp,a) = 0.
    hunger(comp,a) = 0.
end do !comp

!Perception of predators is the same for all inds
finfo(19) = autorisk(a)/MaxPercept(7)

!Developmental modulation
!do g = 1,4
! ABMgenes(g) = geneAHsize(g)
!end do
!find body mass (finfo(12))
!Bodymass in this routine is BEFORE behaviour in time step, so it is the stored BM
from the time before.

if (a == 1) then
    BM = 250. !the birth weight of all inds (cop2egg)
else
    BM = iweight(a-1)
end if

!rescale BM to fit finfo(12) in the 0-1 range:
BM = BM/MaxPercept(4)
!interpolate between nearest two values to find F&Hsize at current BM

e1 = 0.1 ! BSA inserted if-statement
e2 = -.1
Hsize1 = linearABM2(ABMgenes,BM,4,e1)
Hsize2 = linearABM2(ABMgenes,BM,4,e2)

```

```

if (MS == 1) then
  Hsize = max(Hsize1,Hsize2)
else
  Hsize = min(Hsize1,Hsize2)
end if

!Hsize = linearABM(ABMgenes,BM,4,errorP)
! Hsize2 = linearABM(ABMgenes,BM,4,errorP)

b = max(a-1,1) !last age, where 0 --> 1
z = idepth(b) !the position of the fish BEFORE its move
!Fear of light
do d = -2,2
  s = max(z+d,1)
  s = min(30,s)
  finfo(4+d) = zlight(s,a)/MaxPercept(2)
end do

!Fear reduction by others
! if (a == 1) then
do d = -2,2
  s = max(z+d,1)
  s = min(30,s)
  finfo(9+d) = numfish(s,a)/MaxPercept(3)
end do
!else
!! finfo(9) = 0.5*(numfish(z,a-1)+numfish(z,a))/MaxPercept(3)
! do d = -2,2
!   s = max(z,1)
!   s = min(30,s)
!   finfo(9+d) = numfish(s,a-1)/MaxPercept(3)
! end do
!end if

!Hunger from stomach
! full capacity is MAXSTOMCAP * body mass
!comp1 = 1.- fstomach(ind,1)/(maxstomcap*fbodymass(ind)) !fraction of allowed capacity
remaining
!error correction from stored values:
!correct C: comp1 = 1-stom/(0.15BM)
!stored L: Lastgenstocap = 1-stom/(0.25BM)
! 5/3 (L-1) = stom/0.15BM = K
! K = 5/3 - 5/3L
! C = 1 - K = 1 - 1.66 + 5/3L = 5/3L - 2/3
finfo(1) = 1.666*stocap(a) - 0.666 !BSA 05.02.14 b to a
finfo(1) = finfo(1)/MaxPercept(1)

!Hunger from food
do d = -2,2
  s = max(z+d,1)
  s = min(30,s)
  ! finfo(15+d) = visran(s,a)*visran(s,a)*autocop(s,b)/MaxPercept(5)
  finfo(15+d) = visran(s,b)*visran(s,b)*autocop(s,b)/MaxPercept(5)
end do

!Neuronal responses
p1 = finfo(1)

```

```

p2 = finfo(19)
p3 = finfo(9)
p4 = finfo(4)
p5 = finfo(15)

IF (MS == 1) then !hungry
    e1 = -.1
    e2 = .1
else          !afraid
    e1 = .1
    e2 = -.1
ENDIF

do comp = 1,3 ! this time we do not follow the gene order, but the rank order!
    c = rank(1,comp) !the genes in comp shall be used in Flight for rank c
    Flight(c) = gamma3gene(10*geneAFlight(comp,1),10*geneAFlight(comp,2),p4,e1)
    c = rank(2,comp)
    Fother(c) = gamma3gene(10*geneAFother(comp,1),10*geneAFother(comp,2),p3,e2)
    c = rank(3,comp)
    Fmort(c) = gamma3gene(10*geneAFmort(comp,1),10*geneAFmort(comp,2),p2,e1)
    c = rank(4,comp)
    Hstom(c) = gamma3gene(10*geneAHstom(comp,1),10*geneAHstom(comp,2),p1,e2)
    c = rank(5,comp)
    Hfood(c) = gamma3gene(10*geneAHfood(comp,1),10*geneAHfood(comp,2),p5,e2)
end do

do c = 1,3 !now find fear and hunger for COMP in falling rank order
    fear(c,a) = (1.-Hsize) * (Flight(c) - Fother(c) + Fmort(c))
    hunger(c,a) = Hsize * (Hstom(c) + Hfood(c))
end do !c

!What fraction of total NR did each component contain?
sumlight = Flight(1) + Flight(2) + Flight(3)
sumother = Fother(1) + Fother(2) + Fother(3)
summort = Fmort(1) + Fmort(2) + Fmort(3)
sumstom = Hstom(1) + Hstom(2) + Hstom(3)
sumfood = Hfood(1) + Hfood(2) + Hfood(3)

do comp = 1,3 ! BSA 10.02.14 extended to strength
    !Calculation of strength of NR1 as fraction of total strength
    if (sumlight > 0.) frac(1,comp,a) = frac(1,comp,a) + Flight(comp)/sumlight
    if (sumlight > 0.) strength(1,comp,a) = strength(1,comp,a) + Flight(comp)
    if (sumlight > 0.) strength2(1,comp,a) = strength2(1,comp,a) + Flight(comp)
    if (sumother > 0.) frac(2,comp,a) = frac(2,comp,a) + Fother(comp)/sumother
    if (sumother > 0.) strength(2,comp,a) = strength(2,comp,a) + Fother(comp)
    if (sumother > 0.) strength2(2,comp,a) = strength2(2,comp,a) + Fother(comp)
    if (summort > 0.) frac(3,comp,a) = frac(3,comp,a) + Fmort(comp)/summort
    if (summort > 0.) strength(3,comp,a) = strength(3,comp,a) + Fmort(comp)
    if (summort > 0.) strength2(3,comp,a) = strength2(3,comp,a) + Fmort(comp)
    if (sumstom > 0.) frac(4,comp,a) = frac(4,comp,a) + Hstom(comp)/sumstom
    if (sumstom > 0.) strength(4,comp,a) = strength(4,comp,a) + Hstom(comp)
    if (sumstom > 0.) strength2(4,comp,a) = strength2(4,comp,a) + Hstom(comp)
    if (sumfood > 0.) frac(5,comp,a) = frac(5,comp,a) + Hfood(comp)/sumfood
    if (sumfood > 0.) strength(5,comp,a) = strength(5,comp,a) + Hfood(comp)
    if (sumfood > 0.) strength2(5,comp,a) = strength2(5,comp,a) + Hfood(comp)
end do

```

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end do !a
994 continue !with next ind, after skipping dead fish , BSA 31.01.14

!!! New individual loop !!!!
!--- Calculations for GOS ---!

do a = 1,flifespan
  !How many components were needed to find Global Organismal State?
  ! BSA 30.01.14: Introduced comp4gos
  if (a > 1) then !test for alive
    if(iweight(a-1) < 100.) goto 995
  end if

  if (GOS(a) == "H") then !hungry
    if(hunger(1,a) > fear(1,a)) then !case solved after 1 component
      comp1gos(a) = comp1gos(a) + 1
    else if (hunger(1,a) + hunger(2,a) > fear(1,a) + fear(2,a)) then ! 2 needed
      comp2gos(a) = comp2gos(a) + 1
    else if (hunger(1,a) + hunger(2,a) + hunger(3,a) > fear(1,a) + fear(2,a)
+feared(3,a)) then ! 3 needed
      comp3gos(a) = comp3gos(a) + 1
      ! Found the right state, HUNGRY, with all three: This means that hunger(3,a)
was bigger than feared(3,a)
      thirdH = thirdH + 1
      streHs = streHs + strength2(4,3,a)
      streHf = streHf + strength2(5,3,a)
    else
      comp4gos(a) = comp4gos(a) + 1 ! Depth was not found with all 3 components -
error
    end if
  else !fish afraid
    if(hunger(1,a) < fear(1,a)) then
      comp1gos(a) = comp1gos(a) + 1
    else if (hunger(1,a) + hunger(2,a) < fear(1,a) + fear(2,a)) then
      comp2gos(a) = comp2gos(a) + 1
      thirdA = thirdA + 1
      streF1 = streF1 + strength2(1,2,a)
      streFo = streFo + strength2(2,2,a)
      streFm = streFm + strength2(3,2,a)
    else if (hunger(1,a) + hunger(2,a) + hunger(3,a) < max(0.001, fear(1,a) +
fear(2,a) +feared(3,a))) then ! 3 needed
      if (fear(1,a) + fear(2,a) +feared(3,a) .ge. 0.001) then !BSA 03.05.14
        comp3gos(a) = comp3gos(a) + 1
        thirdA = thirdA + 1
        streF1 = streF1 + strength2(1,3,a)
        streFo = streFo + strength2(2,3,a)
        streFm = streFm + strength2(3,3,a)
      else
        comp5gos(a) = comp5gos(a) + 1 ! afraid due to lack of hunger
      end if
    else
      comp4gos(a) = comp4gos(a) + 1 ! Depth was not found with all 3 components -
error
    end if
  end if
end do !a

```

```

995 continue !with next ind, after skipping dead fish , BSA 31.01.14

997 continue !with next ind, after skipping dead fish , BSA 31.01.14

end do !while file 30...
!----- LONG individual-loops ending here -----
-----

!!!! PRINTING RESULTS !!!!

!Results for use of GOS and what factors that contribute
write(9191,9199) thirdH, thirdA, ((1.*thirdH)/(1.*(thirdA+thirdH))), streHs, streHf,
streFl, streFo, streFm
9199 format(2I16,F16.4,5F16.4)

!after all individuals are calculated for all ages, print results for each age
write(6,*) "make prints"
!make headings for prints from each simulation
write(42,4242)"age", "hungry", "afraid", "age", "f1cGOS", "f2cGOS", "f3cGOS", "f4cGOS", "age", "f1c
DEP", "f2cDEP", "f3cDEP", "f4cDEP", & ! BSA 29.01.14 removed " ",

"age", "f1cFLIG", "f2cFLIG", "f3cFLIG", "age", "f1cFOTH", "f2cFOTH", "f3cFOTH", "age", "f1cFPRD", "f
2cFPRD", "f3cFPRD", &

"age", "f1cHSTO", "f2cHSTO", "f3cHSTO", "age", "f1cHFOD", "f2cHFOD", "f3cHFOD", "age", "f1cALIG", "f
2cALIG", "f3cALIG", &

"age", "f1cAOTH", "f2cAOTH", "f3cAOTH", "age", "f1cHFOD", "f2cHFOD", "f3cHFOD", "age", "f1cHOTH", "f
2cHOTH", "f3cHOTH"

do a = 1,flifespan
!print results
living = 1.*(alive(1,a) + alive(2,a))
do N = 1,5 !find GOS
do c = 1,3
if (living > 0.) frac(N,c,a) = frac(N,c,a)/living
end do
end do
do N = 6,7 !when GOS = afraid
do c = 1,3
if (alive(2,a) > 0) frac(N,c,a) = frac(N,c,a)/(1.*alive(2,a)) !divide by the
frightened
end do
end do
do N = 8,9 !when GOS = hungry
do c = 1,3
if (alive(1,a) > 0) frac(N,c,a) = frac(N,c,a)/(1.*alive(1,a)) !divide by the
hungry
end do
end do

!BSA 04.02.14: If-statement to avoid writing 0 when no ind in GOS at TS
if (living > 0.) then
if (alive(2,a) == 0) then

```

```

        write(42,4243) a,alive(1,a),alive(2,a),a,comp1gos(a)/living, &
comp2gos(a)/living,comp3gos(a)/living,comp4gos(a)/living,a,((comphabval(c,a)/living),c=1,4
), &
a,(frac(1,c,a),c=1,3),a,(frac(2,c,a),c=1,3),a,(frac(3,c,a),c=1,3),a,(frac(4,c,a),c=1,3),a,
(frac(5,c,a),c=1,3), &
a,"None","None","None",a,"None","None","None",a,(frac(8,c,a),c=1,3),a,(frac(9,c,a),c=1,3)
    else if (alive(1,a) == 0) then
        write(42,4244) a,alive(1,a),alive(2,a),a,comp1gos(a)/living, &
comp2gos(a)/living,comp3gos(a)/living,comp4gos(a)/living,a,((comphabval(c,a)/living),c=1,4
), &
a,(frac(1,c,a),c=1,3),a,(frac(2,c,a),c=1,3),a,(frac(3,c,a),c=1,3),a,(frac(4,c,a),c=1,3),a,
(frac(5,c,a),c=1,3), &
a,(frac(6,c,a),c=1,3),a,(frac(7,c,a),c=1,3),a,"None","None","None",a,"None","None","None"
    else
        write(42,4240) a,alive(1,a),alive(2,a),a,comp1gos(a)/living, &
comp2gos(a)/living,comp3gos(a)/living,comp4gos(a)/living,a,((comphabval(c,a)/living),c=1,4
), &
a,(frac(1,c,a),c=1,3),a,(frac(2,c,a),c=1,3),a,(frac(3,c,a),c=1,3),a,(frac(4,c,a),c=1,3),a,
(frac(5,c,a),c=1,3), &
a,(frac(6,c,a),c=1,3),a,(frac(7,c,a),c=1,3),a,(frac(8,c,a),c=1,3),a,(frac(9,c,a),c=1,3)
    end if
    end if
end do !a

4240 format(4I12,4E12.5,I12,4E12.5,9(I12,3E12.5))
4243 format(4I12,4E12.5,I12,4E12.5,5(I12,3E12.5),2(I12,3A12),2(I12,3E12.5))
4244 format(4I12,4E12.5,I12,4E12.5,5(I12,3E12.5),2(I12,3E12.5),2(I12,3A12))
4242 format(49A12)
!9922 continue !avoid SIM 22
close(42)

!BSA 10.02.14 - Making print for strengths:
!after all individuals are calculated for all ages, print results for each age
write(6,*) "make prints for strength"
!make headings for prints from each simulation
write(43,4242)"age","f1sFLIG","f2sFLIG","f3sFLIG","age","f1sFOTH","f2sFOTH","f3sFOTH","age
","f1sFPRD","f2sFPRD","f3sFPRD", &
"age","f1sHSTO","f2sHSTO","f3sFSTO","age","f1sHFOD","f2sHFOD","f3sHFOD","age","f1sALIG","f
2sALIG","f3sALIG", &
"age","f1sAOTH","f2sAOTH","f3sAOTH","age","f1sHFOD","f2sHFOD","f3sHFOD","age","f1sHOTH","f
2sHOTH","f3sHOTH"

do a = 1,flifespan
    living = 1.*(alive(1,a) + alive(2,a))
    do N = 1,5 !find GOS
        do c = 1,3

```

```

        if (living > 0.) strength(N,c,a) = strength(N,c,a)/living
    end do
end do
do N = 6,7 !when GOS = afraid
    do c = 1,3
        if (alive(2,a) > 0) strength(N,c,a) = strength(N,c,a)/(1.*alive(2,a)) !divide
by the frightened
    end do
end do
do N = 8,9 !when GOS = hungry
    do c = 1,3
        if (alive(1,a) > 0) strength(N,c,a) = strength(N,c,a)/(1.*alive(1,a)) !divide
by the hungry
    end do
end do

```

```

!BSA 10.02.14: If-statement to avoid writing 0 when no ind in GOS at TS
if (living > 0.) then
    if (alive(2,a) == 0) then
        write(43,4303) a,(strength(1,c,a), c=1,3),a,(strength(2,c,a),
c=1,3),a,(strength(3,c,a), c=1,3),&
            a,(strength(4,c,a), c=1,3),a,(strength(5,c,a), c=1,3),&
            a,"None","None","None",a,"None","None","None",&
            a,(strength(8,c,a), c=1,3),a,(strength(9,c,a), c=1,3)
        else if (alive(1,a) == 0) then
            write(43,4304) a,(strength(1,c,a), c=1,3),a,(strength(2,c,a),
c=1,3),a,(strength(3,c,a), c=1,3),&
            a,(strength(4,c,a), c=1,3),a,(strength(5,c,a), c=1,3),&
            a,(strength(6,c,a), c=1,3),a,(strength(7,c,a), c=1,3),&
            a,"None","None","None",a,"None","None","None"
        else
            write(43,4301) a,(strength(1,c,a), c=1,3),a,(strength(2,c,a),
c=1,3),a,(strength(3,c,a), c=1,3),&
            a,(strength(4,c,a), c=1,3),a,(strength(5,c,a), c=1,3),&
            a,(strength(6,c,a), c=1,3),a,(strength(7,c,a), c=1,3),&
            a,(strength(8,c,a), c=1,3),a,(strength(9,c,a), c=1,3)
        end if
    end if
end if

```

```
end do ! a
```

```

4301 format(9(I12,3E12.5))
4303 format(5(I12,3E12.5),2(I12,3A12),2(I12,3E12.5))
4304 format(7(I12,3E12.5),2(I12,3A12))
4302 format(36A12)

```

```
close(43)
```

```
!BSA 12.02.14 Making prints of AVG values
```

```

do i = 1,9
    do c = 1,3
        avgstrength(i,c) = 0.
        avgfrac(i,c) = 0.
    end do
end do

```

```

do a = 1,flifespan
  do i = 1,9
    do c = 1,3
      avgstrength(i,c) = avgstrength(i,c) + strength(i,c,a)
      avgfrac(i,c) = avgfrac(i,c) + frac(i,c,a)
    end do
  end do !i
end do

do i = 1,9
  do c = 1,3
    avgstrength(i,c) = avgstrength(i,c)/(1.*flifespan)
    avgfrac(i,c) = avgfrac(i,c)/(1.*flifespan)
  end do
end do

write(48,4801) nr,((avgfrac(i,c), c=1,3),i=1,9)
write(49,4801) nr,((avgstrength(i,c), c=1,3),i=1,9)

4801 format(I12,27E14.5)

4343 continue
end do !nr (simulations)
close(48)
close(49)

end !SR19NRComponents

```

C.2 SR13

This subroutine is used to calculate the mean values and 95 % confidence intervals of the mean for all 50 simulations at each 1000th generation for all experiments and print them in different formats dependent on which figures the data is to be used in.

```

!-----
subroutine SR13Normalo12(runtag,first,files,datetag,alpha)
!-----
!Denne SR skriver ut verdier for
!generation, %mortality, offspring, %fear females, % fear males
!for hver NORMAL generasjon i hver simulering
!i en felles fil der hver nye simulering følger under den forrige men med data i egne
kolonner
!SR altered by BSA 21.03.14 to write CI to file 113
! variables used in last part of routine, where only one gen from each pop is used, are
have a S in the name (e.g. SUMSmort..)

implicit none
character(8) deviation
character(7) help
character(6) runtag

```



```

character(20) string1 ! BSA 13.01.14
character(36) string12 ! BSA 13.01.14
character(55) string13! BSA 13.01.14
character(55) string15! BSA 13.01.14
character(55) string16
character(55) string14! BSA 13.01.14
character(4) datetag !BSA 13.01.14
integer expmt,files,first,line,l,e,g,k,p,Nnr,genera,ngen,Npoint,divgen
real m,ff,mf,alpha,bfM,bfF
real fmultrisk,reprocap,fsurvR0,AvMut
integer attacks,preymax,fpop,alive,fnewpop,neverA(2)
integer kids(20000,0:100),Ckids(20000,0:100) ! BSA 13.01.14 Extended 2nd dimension to
0:100
real mort(20000,0:100),femF(20000,0:100),maleF(20000,0:100)

!BSA 21.03.14 Added variables for CI
real Cmort(20000,0:100),CfemF(20000,0:100),CmaleF(20000,0:100)
real
SUMCmort(8,0:100),SUMCkids(8,0:100),SUMCfemF(8,0:100),SUMCmaleF(8,0:100)!,SUMCbmF(8,0:100)
,SUMCbmM(8,0:100)
real AVGCmort(8,0:100),AVGckids(8,0:100),AVGcfemF(8,0:100),AVGcmaleF(8,0:100)
real
VARCmort(8,0:100),VARCkids(8,0:100),VARCfemF(8,0:100),VARCmaleF(8,0:100)!,VARCbmF(8,0:100)
,VARCbmM(8,0:100)
real CIMort(8,0:100),CIkids(8,0:100),CIFemF(8,0:100),CIMaleF(8,0:100)
!BSA added variables for second part
real
Smort(8,0:100),Skids(8,0:100),SfemF(8,0:100),SmaleF(8,0:100),SbmF(8,0:100),SbmM(8,0:100)
real SUMSmort(8),SUMSkids(8),SUMSfemF(8),SUMSmaleF(8),SUMSbmF(8),SUMSbmM(8)
real AVGSmort(8),AVGSkids(8),AVGSfemF(8),AVGSmaleF(8),AVGSbmF(8),AVGSbmM(8)
real VARSmort(8),VARSkids(8),VARSfemF(8),VARSmaleF(8),VARSbmF(8),VARSbmM(8)
real SCImort(8),SCIkids(8),SCIfemF(8),SCImaleF(8),SCIbmF(8),SCIbmM(8)

!dummy variables in reading 12
real b1,b2,b3,b4,b5,z1,z2,z3,z4,z5,z6,z7,b11,b12,b13,b14,b15
integer n1,n2,n3,n4,n5,n6

write(6,*) "starting SR13Normalo12"

! read(12,1201) g,deviation,help,fmultrisk,reprocap,attacks,preymax,fpop,alive, &
! m,fsurvR0,k,fnewpop,AvMut,neverA(1),XneverA(2),ff,mf
! 1201 format(I6,2A9,2F8.2,I4,3I8,2F8.2,2I8,F8.2,2I8,2F9.4)

!open output file
write(string13, 5003)"HED23-",datetag,runtag,"-SR13-ALL-Ngener-mort-kids-fear.txt"
write(string14, 5003)"HED23-",datetag,runtag,"-SR13-ALL-Ngener-ConfidInterval.txt"
open(13, file = string13)
open(113, file = string14)
5003 format(1A6,A4,1A6,A35) ! BSA 13.01.14

!Creating output file second part
write(string15, 5003)"HED23-",datetag,runtag,"-SR13-CI-Comparing-simul-series.txt"
open(20, file = string15)
write(string16, 5003)"HED23-",datetag,runtag,"-SR13-Obs-for1-4kgen-MultCompar.txt"
open(21, file = string16)
2021 format(1A6,A4,1A6,A35)

!Resetting variables used in CI-calculations, BSA 21.03.14

```

```

do expmt = first,files
  do p = 1,4 !Number of compared data points
    SUMCmort(p,expmt) = 0.
    SUMCkids(p,expmt) = 0.
    SUMCfemF(p,expmt) = 0.
    SUMCmaleF(p,expmt) = 0.
    AVGCmort(p,expmt) = 0.
    AVGckids(p,expmt) = 0.
    AVGcfemF(p,expmt) = 0.
    AVGcmaleF(p,expmt) = 0.
    VARCmort(p,expmt) = 0.
    VARCkids(p,expmt) = 0.
    VARCfemF(p,expmt) = 0.
    VARCmaleF(p,expmt) = 0.
    VARCmort(p,expmt) = 0.
    VARCkids(p,expmt) = 0.
    VARCfemF(p,expmt) = 0.
    VARCmaleF(p,expmt) = 0.
    CImort(p,expmt) = 0.
    CIkids(p,expmt) = 0.
    CIfemF(p,expmt) = 0.
    CImaleF(p,expmt) = 0.
    Smort(p,expmt) = 0.
    Skids(p,expmt) = 0.
    SfemF(p,expmt) = 0.
    SmaleF(p,expmt) = 0.
    SbmF(p,expmt) = 0.
    SbmM(p,expmt) = 0.
  end do
end do
do p = 1,4
  SUMSmort(p) = 0.
  SUMSkids(p) = 0.
  SUMSfemF(p) = 0.
  SUMSmaleF(p) = 0.
  SUMSbmF(p) = 0.
  SUMSbmM(p) = 0.
  AVGSmort(p) = 0.
  AVGSkids(p) = 0.
  AVGSfemF(p) = 0.
  AVGSmaleF(p) = 0.
  AVGSbmF(p) = 0.
  AVGSbmM(p) = 0.
  VARSmort(p) = 0.
  VARSkids(p) = 0.
  VARSfemF(p) = 0.
  VARSmaleF(p) = 0.
  VARSfemF(p) = 0.
  VARSmaleF(p) = 0.
  SCImort(p) = 0.
  SCIkids(p) = 0.
  SCIfemF(p) = 0.
  SCImaleF(p) = 0.
  SCIbmF(p) = 0.
  SCIbmM(p) = 0.
end do

```

!!!FIRST PART

!Used to calculate CI and mean values for each 1000th gen for each experiment
 !!!! Loop over number of simulations to analyse !!!!

```

do expmt = first,files

!reset arrays (set all data in all lines in output file to zero)
do e = first,files
  genera = 0
  Nnr = 0
  Npoint = 0
  do line = 1,20000 !never more than 20.000 generations, 30 simulations =600'
    mort(line,e) = 0.
    kids(line,e) = 0
    femF(line,e) = 0.
    maleF(line,e) = 0.
    CMort(line,e) = 0.
    Ckids(line,e) = 0.
    CfemF(line,e) = 0.
    CmaleF(line,e) = 0.
  end do
end do
if (expmt < 10) then
  write(string1, 5001)"HED23-",datetag,runtag,"-E0",expmt! BSA 13.01.14
else
  write(string1, 5002)"HED23-",datetag,runtag,"-E",expmt! BSA 13.01.14
end if
write(string12,5013)string1,"-o012-popdyn.txt"
open(12, file = string12)
read(12,*)
line = 0 !no data lines are used, so far
write(6,*)"now reading ",string12
do while (.not. EOF(12))
  read(12,1201) g,deviation,help,fmultrisk,reprocap,attacks,preymax,fpop,alive, &
m, fsurvR0,k, fnewpop,AvMut,neverA(1),neverA(2), ff,mf,b1,b2,b3,b4,b5,bfF,z1,z2,z3,z4,z5,z6,z
7,n1,n2,n3,n4,n5,n6,b11,b12,b13,b14,b15,bfM
  1201 format(I6,2A9,2F8.2,I4,3I8,2F8.2,2I8,F8.2,2I8,2F9.4,6F9.4,7F9.4,6F9.4,6F9.4)
  if(deviation == "Normal") then
    line = line + 1 !make new line in output file
    mort(line,expmt) = m
    kids(line,expmt) = k
    femF(line,expmt) = ff
    maleF(line,expmt) = mf
    l = line
    write(13,1202)
g,(mort(l,e),e=first,4),g,(mort(l,e),e=first,files),g,(kids(l,e),e=first,files),g,(femF(l,
e),e=first,files),g,(maleF(l,e),e=first,files) ! BSA 13.01.14 1,30 to first,files

  end if

  if(deviation == "Normal") then
    if ((g .gt. 800 .and. g .le. 1000) .or. (g .gt. 1800 .and. g .le. 2000) .or. (g
.gt. 2800 .and. g .le. 3000) .or. (g .gt. 3800 .and. g .le. 4000)) then
      genera = genera + 1 !number of normal generations passed in this block
      Nnr = Nnr +1 !number passed in blocks in total
      CMort(Nnr,expmt) = m
      Ckids(Nnr,expmt) = k !Fecundity
      CfemF(Nnr,expmt) = ff

```

```

        CmaleF(Nnr,expmt) = m
    end if
end if

!Calculations of CI
if (g == 1000 .or. g == 2000 .or. g == 3000 .or. g == 4000) then
    ! The number of normal generations to use is now genera
    p = NINT(g/1000.) ! Gives p of 1,2,3,4
    do ngen = (Nnr-genera+1),Nnr
        SUMCmort(p,expmt) = SUMCmort(p,expmt) + Cmort(ngen,expmt)
        SUMCkids(p,expmt) = SUMCkids(p,expmt) + Ckids(ngen,expmt)
        SUMCfemF(p,expmt) = SUMCfemF(p,expmt) + CfemF(ngen,expmt)
        SUMCmaleF(p,expmt) = SUMCmaleF(p,expmt) + CmaleF(ngen,expmt)
    end do
    AVGCmort(p,expmt) = SUMCmort(p,expmt)/(1.*genera)
    AVGCkids(p,expmt) = SUMCkids(p,expmt)/(1.*genera)
    AVGCfemF(p,expmt) = SUMCfemF(p,expmt)/(1.*genera)
    AVGCmaleF(p,expmt) = SUMCmaleF(p,expmt)/(1.*genera)
    do ngen = (Nnr-genera+1),Nnr
        VARCmort(p,expmt) = VARCmort(p,expmt) + (Cmort(ngen,expmt) -
AVGCmort(p,expmt))**2.
        VARCkids(p,expmt) = VARCkids(p,expmt) + (Ckids(ngen,expmt) -
AVGCkids(p,expmt))**2.
        VARCfemF(p,expmt) = VARCfemF(p,expmt) + (CfemF(ngen,expmt) -
AVGCfemF(p,expmt))**2.
        VARCmaleF(p,expmt) = VARCmaleF(p,expmt) + (CmaleF(ngen,expmt) -
AVGCmaleF(p,expmt))**2.
    end do
    VARCmort(p,expmt) = VARCmort(p,expmt)/(1.*genera) !This is the variance
    VARCkids(p,expmt) = VARCkids(p,expmt)/(1.*genera) !This is the variance
    VARCfemF(p,expmt) = VARCfemF(p,expmt)/(1.*genera) !This is the variance
    VARCmaleF(p,expmt) = VARCmaleF(p,expmt)/(1.*genera) !This is the variance
    !find confidence interval (= alpha*SD/sqrt(N)) = alpha*sqrt(VAR/N))
    CImort(p,expmt) = alpha * sqrt(VARCmort(p,expmt)/(1.*genera))
    CIkids(p,expmt) = alpha * sqrt(VARCkids(p,expmt)/(1.*genera))
    CIFemF(p,expmt) = alpha * sqrt(VARCfemF(p,expmt)/(1.*genera))
    CImaleF(p,expmt) = alpha * sqrt(VARCmaleF(p,expmt)/(1.*genera))
end if

if (g == 800 .or. g == 1800 .or. g == 2800 .or. g == 3800) then
    genera = 0 ! Start counting normal generations for new point
end if

if (g == 1000 .or. g == 2000 .or. g == 3000 .or. g == 4000 .or. Npoint == 1) then
    if (deviation == "Normal") then
        p = NINT(g/1000.) ! Gives p of 1,2,3,4
        Smort(p,expmt) = m
        Skids(p,expmt) = k !Fecundity
        SfemF(p,expmt) = ff
        SmaleF(p,expmt) = mf
        SbmF(p,expmt) = bfF
        SbmM(p,expmt) = bfM

        Npoint = 0
        GOTO 1314 !Jump out of if-statement if normal is found
    end if
    Npoint = 1 !Entering IF next gen and hoping for a normal gen
end if

```

```

        end if
        1314 CONTINUE

1202 format(I8,100F8.2,I8,100F8.2,I8,100F9.4,I8,100F9.4) ! BSA 13.01.14 extended to 100
sim
!1202 format(t1, I6,t1, I6,t1, I6,t1, I6)
        end do !while
        close(12)
        1313 CONTINUE
end do !expmt
close(13)

!Writing CI and avg
write(113,5015)
"Generation", "CIMort", "HighMort", "LowMort", "AVGMort", "CIKids", "Highkids", "Lowkids", "AVGkids",
"s", "CIfemF", "HighfemF", "LowfemF", "AVGfemF", "CImaleF", "HighmaleF", "LowmaleF", "AVGmaleF"
do p = 1,4
        do expmt = first,files
                write(113,5014) (p*1000),CIMort(p,expmt),(AVGCmort(p,expmt) +
CIMort(p,expmt)),(AVGCmort(p,expmt) - CIMort(p,expmt)),AVGCmort(p,expmt), &
                CIkids(p,expmt),(AVGCkids(p,expmt) +
CIkids(p,expmt)),(AVGCkids(p,expmt) - CIkids(p,expmt)),AVGCkids(p,expmt), &
                CIfemF(p,expmt),(AVGCfemF(p,expmt) +
CIfemF(p,expmt)),(AVGCfemF(p,expmt) - CIfemF(p,expmt)),AVGCfemF(p,expmt), &
                CIMaleF(p,expmt),(AVGCmaleF(p,expmt) +
CIMaleF(p,expmt)),(AVGCmaleF(p,expmt) - CIMaleF(p,expmt)),AVGCmaleF(p,expmt)
                end do
                write(113,*) "" !Blank line
        end do

5001 format(1A6,1A4,1A6,A3,I1)! BSA 13.01.14
5002 format(1A6,1A4,1A6,A2,I2)! BSA 13.01.14
5013 format(A20,A16)! BSA 13.01.14
5014 format(I12,16F12.4)
5015 format(17A12)

!SECOND PART
!Calculations and output for CI - between series

divgen = 0
do expmt = first,files
        if (expmt == 11) GOTO 1315
        do p = 1,4
                SUMSmort(p) = SUMSmort(p) + Smort(p,expmt)
                SUMSkids(p) = SUMSkids(p) + Skids(p,expmt)
                SUMSfemF(p) = SUMSfemF(p) + SfemF(p,expmt)
                SUMSmaleF(p) = SUMSmaleF(p) + SmaleF(p,expmt)
                SUMSbmF(p) = SUMSbmF(p) + SbmF(p,expmt)
                SUMSbmM(p) = SUMSbmM(p) + SbmM(p,expmt)
        end do
        divgen = divgen + 1
        1315 CONTINUE
end do

do p = 1,4
        AVGSmort(p) = SUMSmort(p)/(1.*divgen)

```

```

    AVGSkids(p) = SUMSkids(p)/(1.*divgen)
    AVGSfemF(p) = SUMSfemF(p)/(1.*divgen)
    AVGSmaleF(p) = SUMSmaleF(p)/(1.*divgen)
    AVGSbmF(p) = SUMSbmF(p)/(1.*divgen)
    AVGSbmM(p) = SUMSbmM(p)/(1.*divgen)
end do

do expmt = first,files
    do p = 1,4
        VARSmort(p) = VARSmort(p) + (Smort(p,expmt) - AVGSmort(p))**2.
        VARSkids(p) = VARSkids(p) + (Skids(p,expmt) - AVGSkids(p))**2.
        VARSfemF(p) = VARSfemF(p) + (SfemF(p,expmt) - AVGSfemF(p))**2.
        VARSmaleF(p) = VARSmaleF(p) + (SmaleF(p,expmt) - AVGSmaleF(p))**2.
        VARSbmF(p) = VARSbmF(p) + (SbmF(p,expmt) - AVGSbmF(p))**2.
        VARSbmM(p) = VARSbmM(p) + (SbmM(p,expmt) - AVGSbmM(p))**2.
    end do
end do

do p = 1,4
    VARSmort(p) = VARSmort(p)/(1.*divgen)
    VARSkids(p) = VARSkids(p)/(1.*divgen)
    VARSfemF(p) = VARSfemF(p)/(1.*divgen)
    VARSmaleF(p) = VARSmaleF(p)/(1.*divgen)
    VARSbmF(p) = VARSbmF(p)/(1.*divgen)
    VARSbmM(p) = VARSbmM(p)/(1.*divgen)

    !find confidence interval (= alpha*SD/sqrt(N)) = alpha*sqrt(VAR/N))
    SCImort(p) = alpha * sqrt(VARSmort(p)/(1.*divgen))
    SCIkids(p) = alpha * sqrt(VARSkids(p)/(1.*divgen))
    SCIfemF(p) = alpha * sqrt(VARSfemF(p)/(1.*divgen))
    SCImaleF(p) = alpha * sqrt(VARSmaleF(p)/(1.*divgen))
    SCIbmF(p) = alpha * sqrt(VARSbmF(p)/(1.*divgen))
    SCIbmM(p) = alpha * sqrt(VARSbmM(p)/(1.*divgen))
end do

write(20,5016)
"CI Mort", "HighMort", "LowMort", "AVGMort", "CI Kids", "Highkids", "Lowkids", "AVGkids", "CI femF", "
HighfemF", "LowfemF", "AVGfemF", "CI maleF", "HighmaleF", "LowmaleF", "AVGmaleF", &
    "CI bmF", "HighbmF", "LowbmF", "AVGbmF", "CI bmM", "HighbmM", "LowbmM", "AVGbmM"
do p = 1,4
    write(20,5017) SCImort(p),AVGSmort(p) + SCImort(p),AVGSmort(p) -
    SCImort(p),AVGSmort(p), &
        SCIkids(p),AVGSkids(p) + SCIkids(p),AVGSkids(p) -
    SCIkids(p),AVGSkids(p), &
        SCIfemF(p),AVGSfemF(p) + SCIfemF(p),AVGSfemF(p) -
    SCIfemF(p),AVGSfemF(p), &
        SCImaleF(p),AVGSmaleF(p) + SCImaleF(p),AVGSmaleF(p) -
    SCImaleF(p),AVGSmaleF(p), &
        SCIbmF(p),AVGSbmF(p) + SCIbmF(p),AVGSbmF(p) - SCIbmF(p),AVGSbmF(p), &
        SCIbmM(p),AVGSbmM(p) + SCIbmM(p),AVGSbmM(p) - SCIbmM(p),AVGSbmM(p)
end do
close(29)

!Output used in Matlab procedure to compare experiments
do p = 1,4
do expmt = first,files
    !if (expmt == 20) GOTO 1316

```

```

        write(21,5018) Smort(p,expmt)
        !1316 CONTINUE
end do
end do
do p = 1,4
do expmt = first,files
    !if (expmt == 20) GOTO 1316
    write(21,5018) Skids(p,expmt)
    !1316 CONTINUE
end do
end do
do p = 1,4
do expmt = first,files
    !if (expmt == 20) GOTO 1316
    write(21,5018) SfemF(p,expmt)
    !1316 CONTINUE
end do
end do
do p = 1,4
do expmt = first,files
    !if (expmt == 20) GOTO 1316
    write(21,5018) SmaleF(p,expmt)
    !1316 CONTINUE
end do
end do
do p = 1,4
do expmt = first,files
    !if (expmt == 20) GOTO 1316
    write(21,5018) SbmF(p,expmt)
    !1316 CONTINUE
end do
end do
do p = 1,4
do expmt = first,files
    !if (expmt == 20) GOTO 1316
    write(21,5018) SbmM(p,expmt)
    !1316 CONTINUE
end do
end do

5016 format(24A12)
5017 format(24F12.4)
5018 format(F12.4)
close(21)

end !SR SRNormalo12

```

D Figures for neuronal responses and their strength: Simulations 6-10

The following four figures are equivalents to Figures 23-26 in the main text, but these represents simulations 6-10.

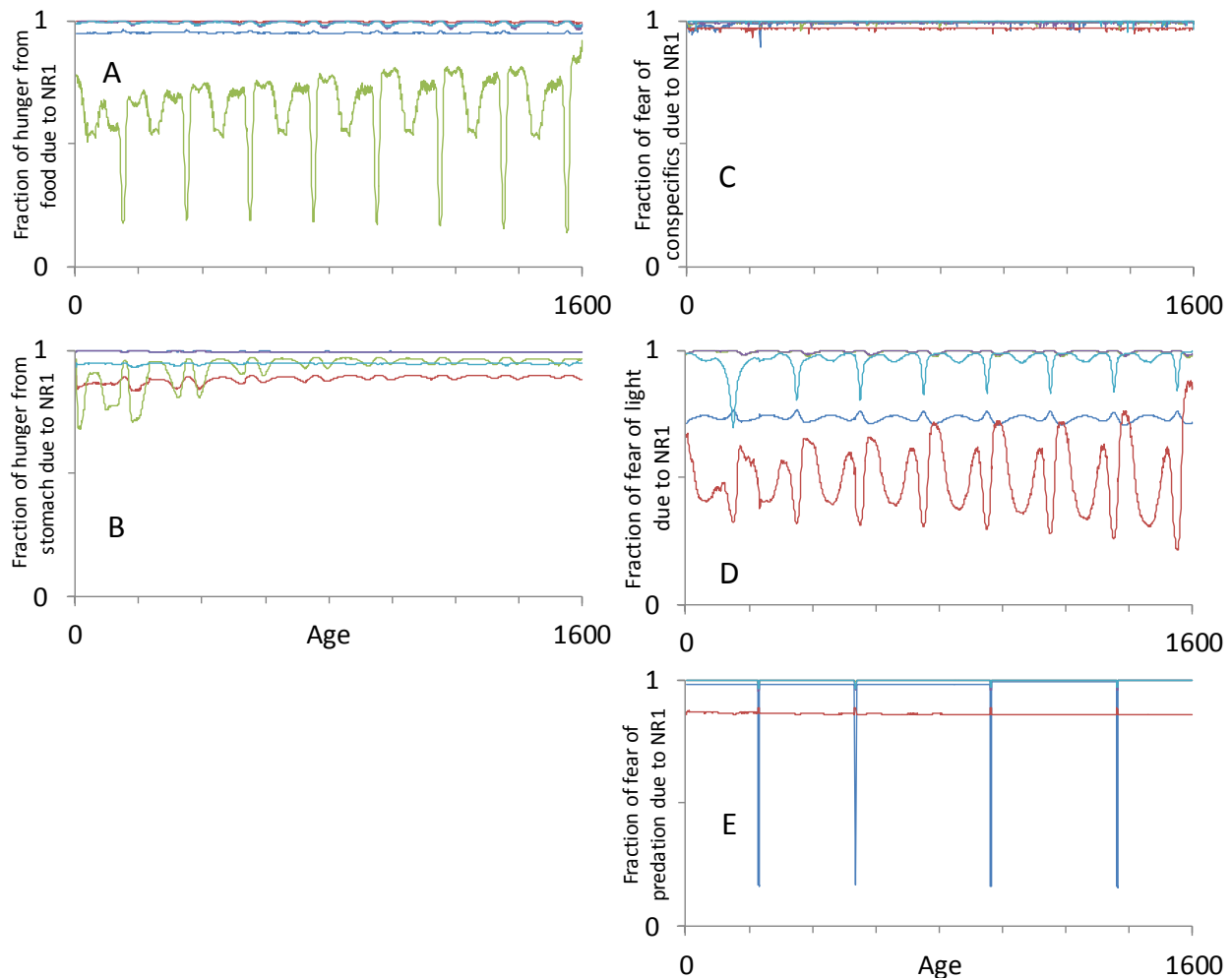


Figure A1: Relative contribution from components in complex neuronal response functions: Comparison of the last generation of five simulations, shown with same color in all panels. A-B) The fraction of the total neuronal response contributed by the stronger (NR1) of the two contributors to hunger: food (A) and available stomach capacity (B) through life. C-E) The fraction of the total neuronal response contributed by the stronger (NR1) of the three contributors to fear: reduction by conspecifics (C) and increase of fear by light (D) and predation (E) through life.

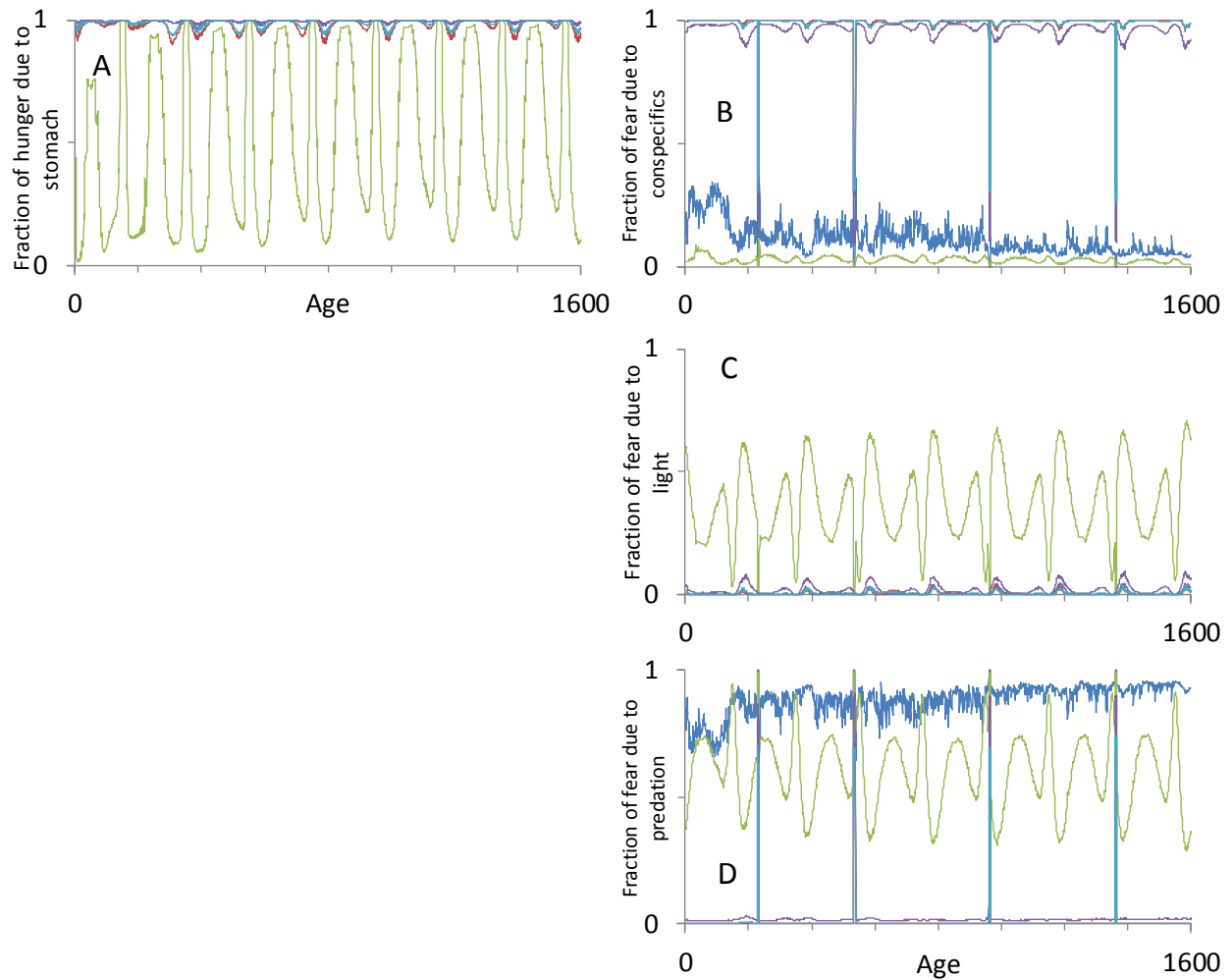


Figure A2: Relative contribution from the complex neuronal response functions when selecting GOS: Comparison of the last generation of five simulations, shown with same color in all panels. A) The relative strength of the neuronal response from available stomach capacity impacting hunger through life. B-D) The relative strength of the three neuronal responses impacting fear: conspecifics (B), light (C) and predation (D) through life. Data are from all individuals in the last generation of the simulations.

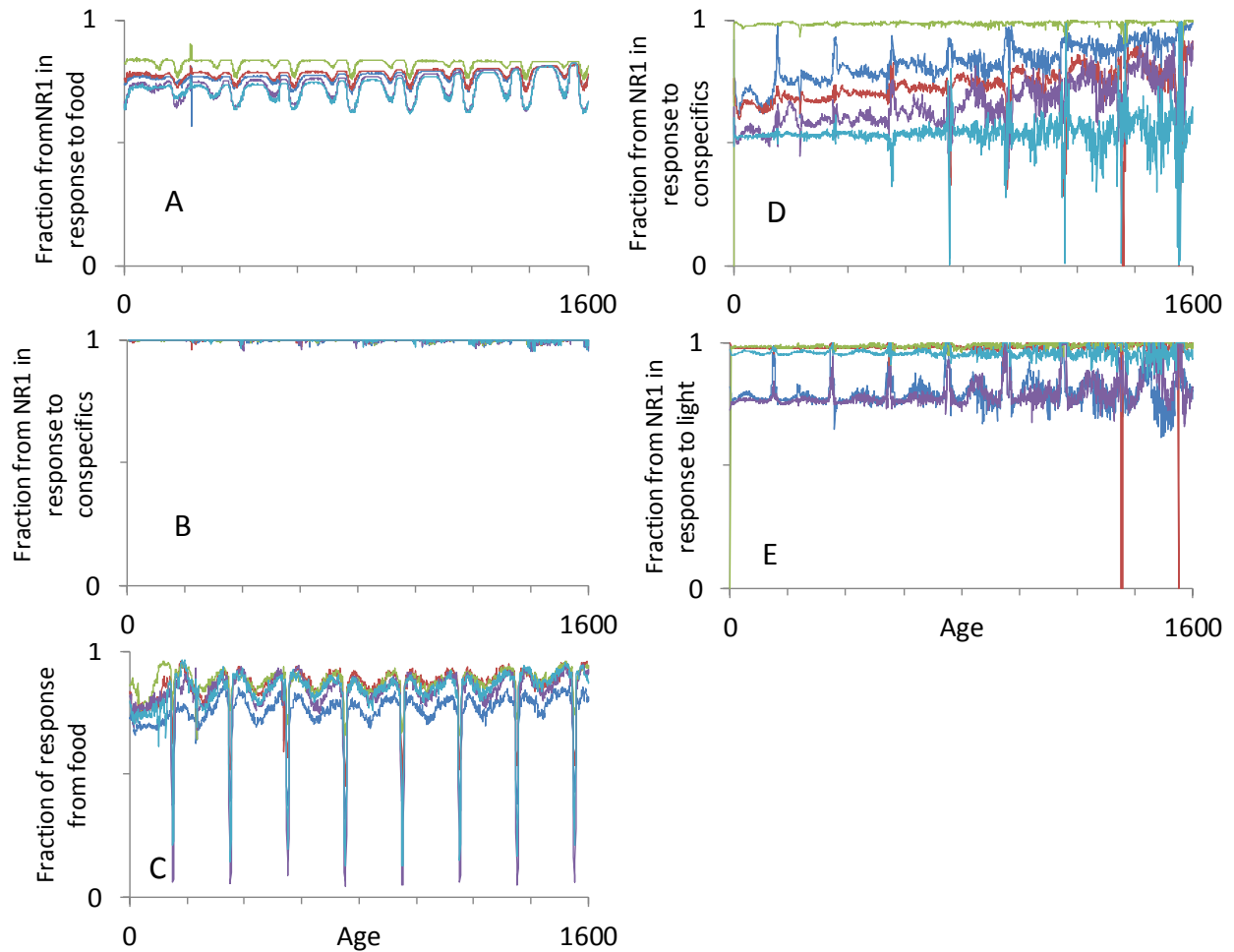


Figure A3: Relative contribution from components in complex neuronal response functions and contribution from the complex neuronal response functions when selecting depth: Comparison of the last generation of five simulations, shown with the same color in all panels. A-B) The fraction of the total neuronal response contributed by the stronger (NR1) of the three components through life when hungry for attraction from food (A) and repulsion from conspecifics (B). C) The fraction of the attraction towards a depth coming from food when in the GOS hungry through life. D-E) The fraction of the total neuronal response contributed by the stronger (NR1) of the three components through life when afraid for attraction from conspecifics (D) and repulsion from light (E).

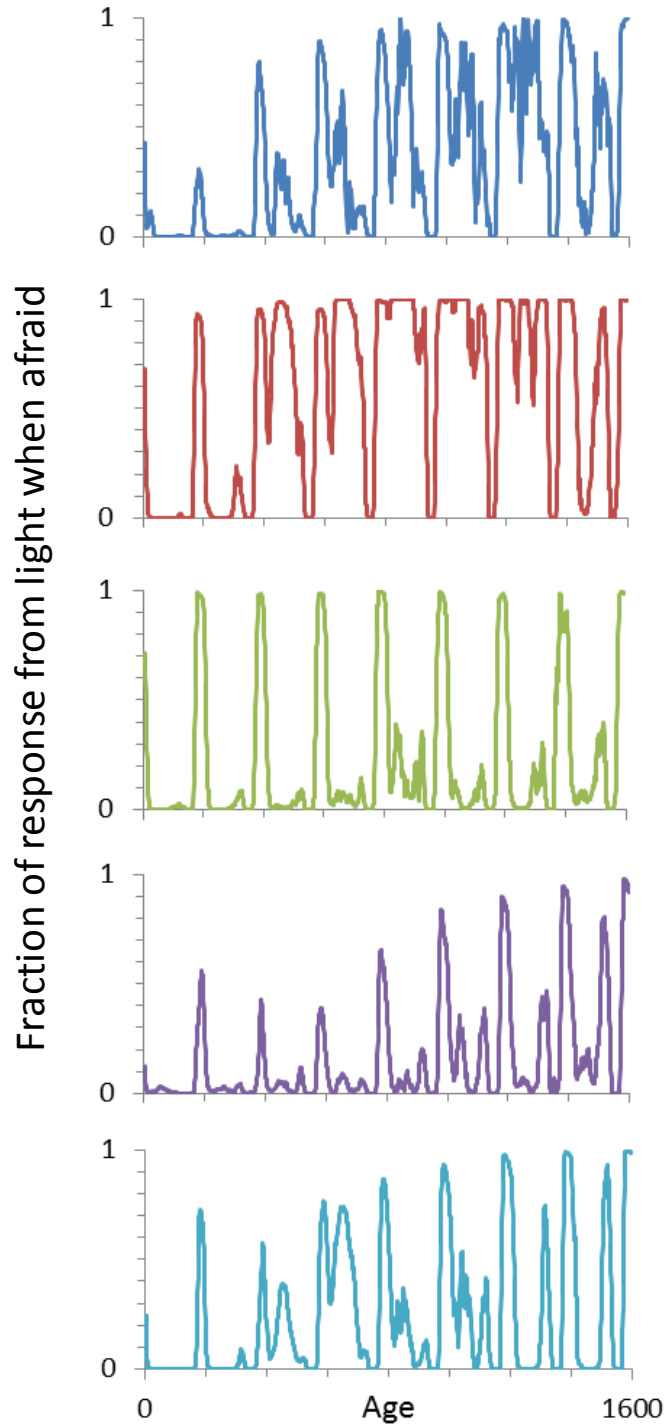


Figure A4: Relative contribution from light when selecting depth: The fraction of the repulsion from a depth coming from light when in the GOS afraid through life. Data are from all individuals in the last generation of simulations 6 (top) to 10 (bottom). The fraction at all 1600 time steps in each panel are the average of four time steps before and after that time step. This is to make graphs easier to interpret.

E Figures for all 9 experiments: Two figures for each experiment

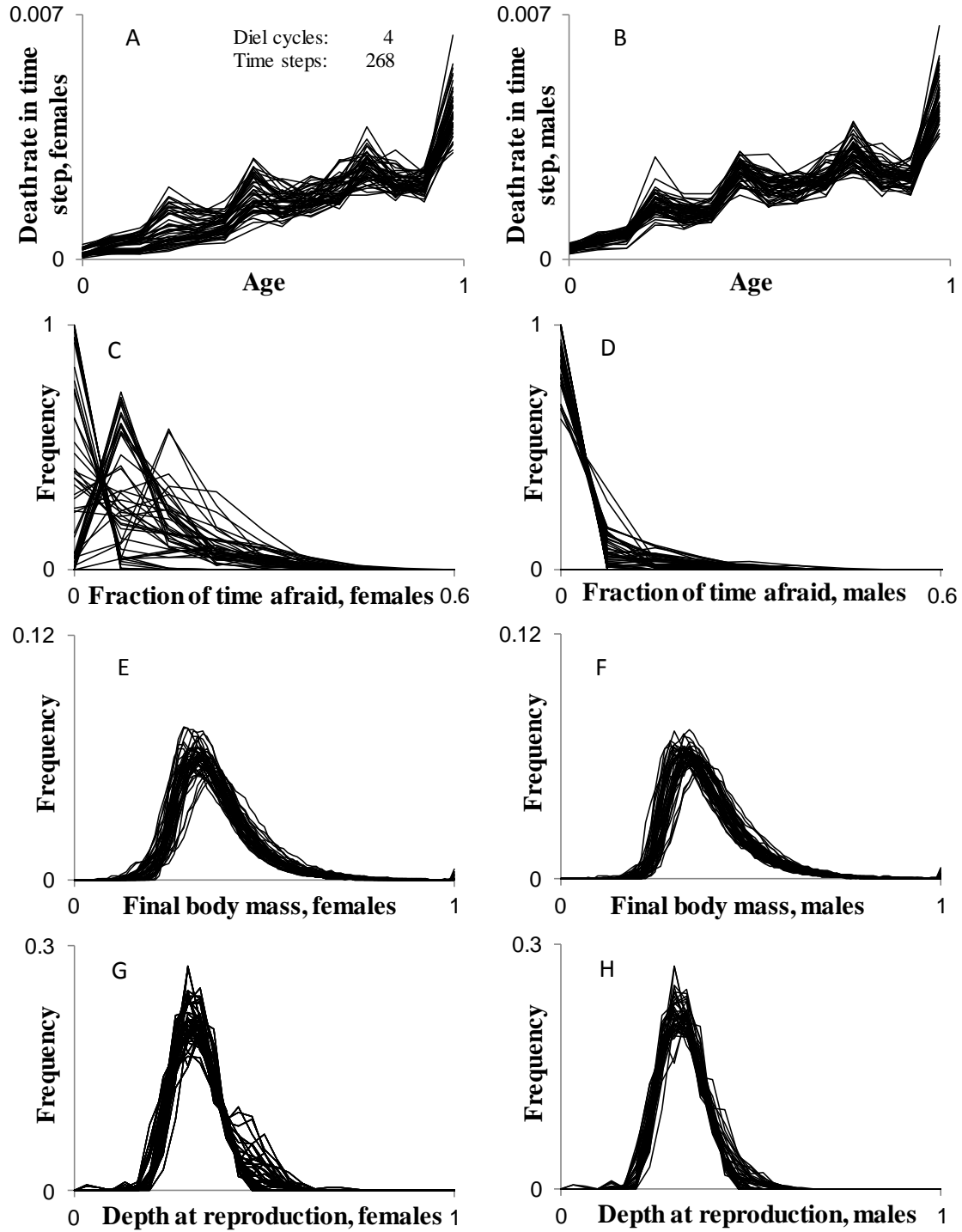


Figure A5, previous page: Comparison of the last 20 generations in the 50 simulations in the series with 4 diel cycles and 67 time steps per cycle. A- B) Death rates through life for females and males, respectively. C- D) The frequency of individuals and what fraction of the time they spent in the GOS afraid. E-F) Body mass at the end of a generation for females and males, respectively. G-H) The depth at which individuals reproduce (i.e. are located in the last time step) for females and males, respectively. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively. 'Normal' generations are explained in Appendix A.1. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively.

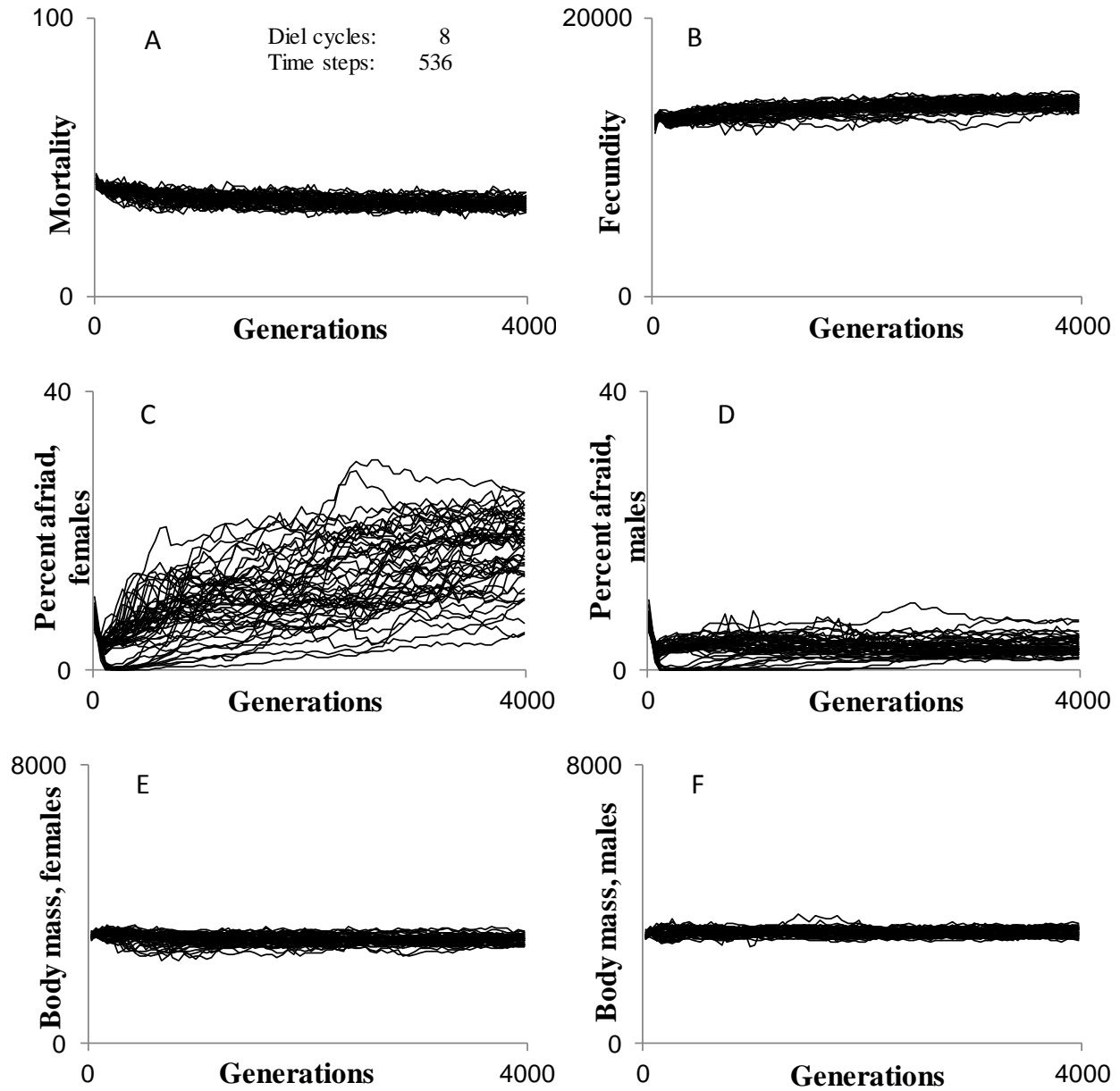


Figure A6: The average in each generation of mortality (A), fecundity (B), percent of time steps in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F) for all 50 simulations with 8 diel cycles and 67 time steps per cycle. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

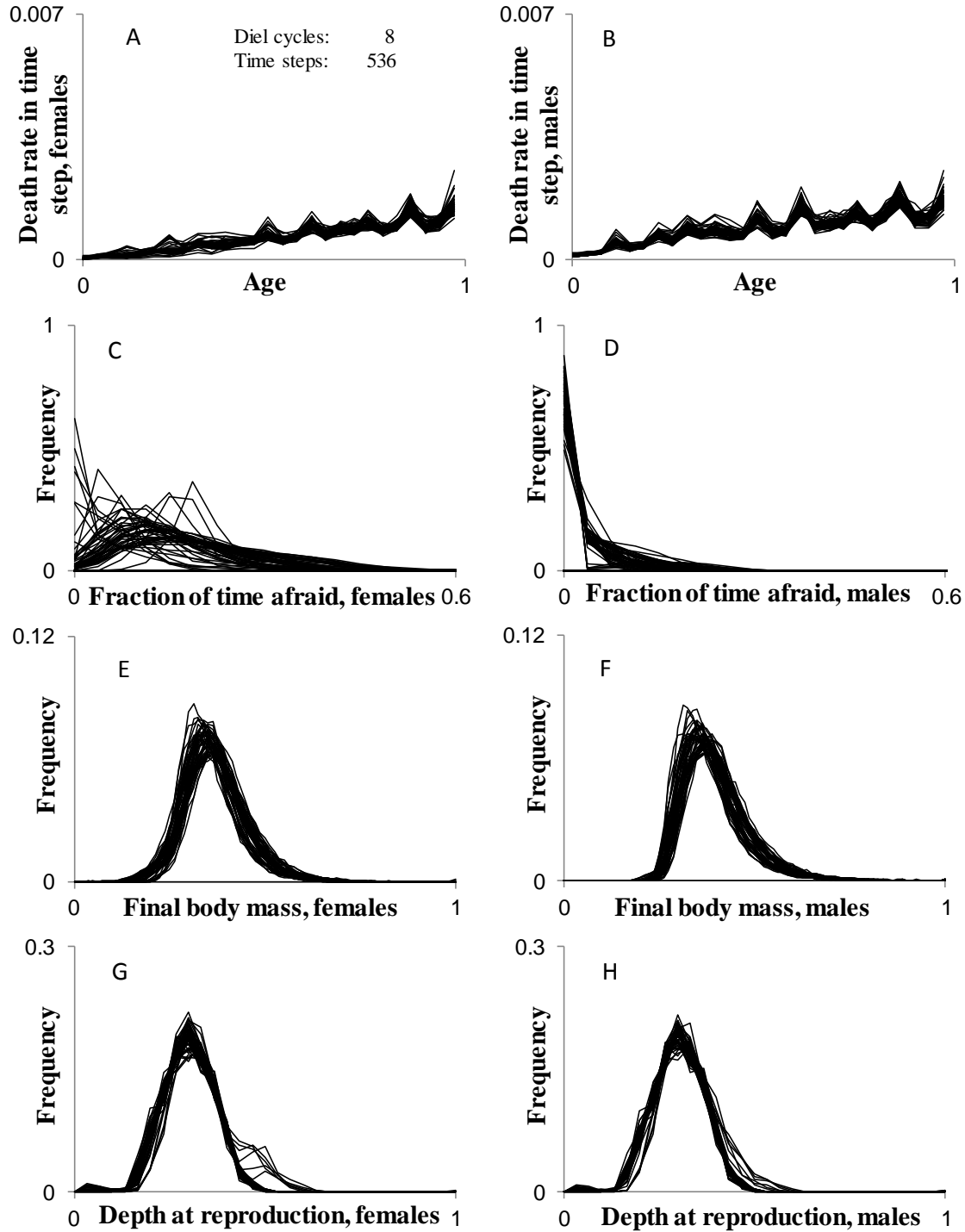


Figure A7: Comparison of the last 20 generations in the 50 simulations in the series with 8 diel cycles and 67 time steps per cycle. A- B) Death rates through life for females and males, respectively. C- D) The frequency of individuals and what fraction of the time they spent in the GOS afraid. E-F) Body mass at the end of a generation for females and males, respectively. G-H) The depth at which individuals reproduce (i.e. are located in the last time step) for females and males, respectively. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth,

respectively. 'Normal' generations are explained in Appendix A.1. X-axes in the panels A and B, E and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively.

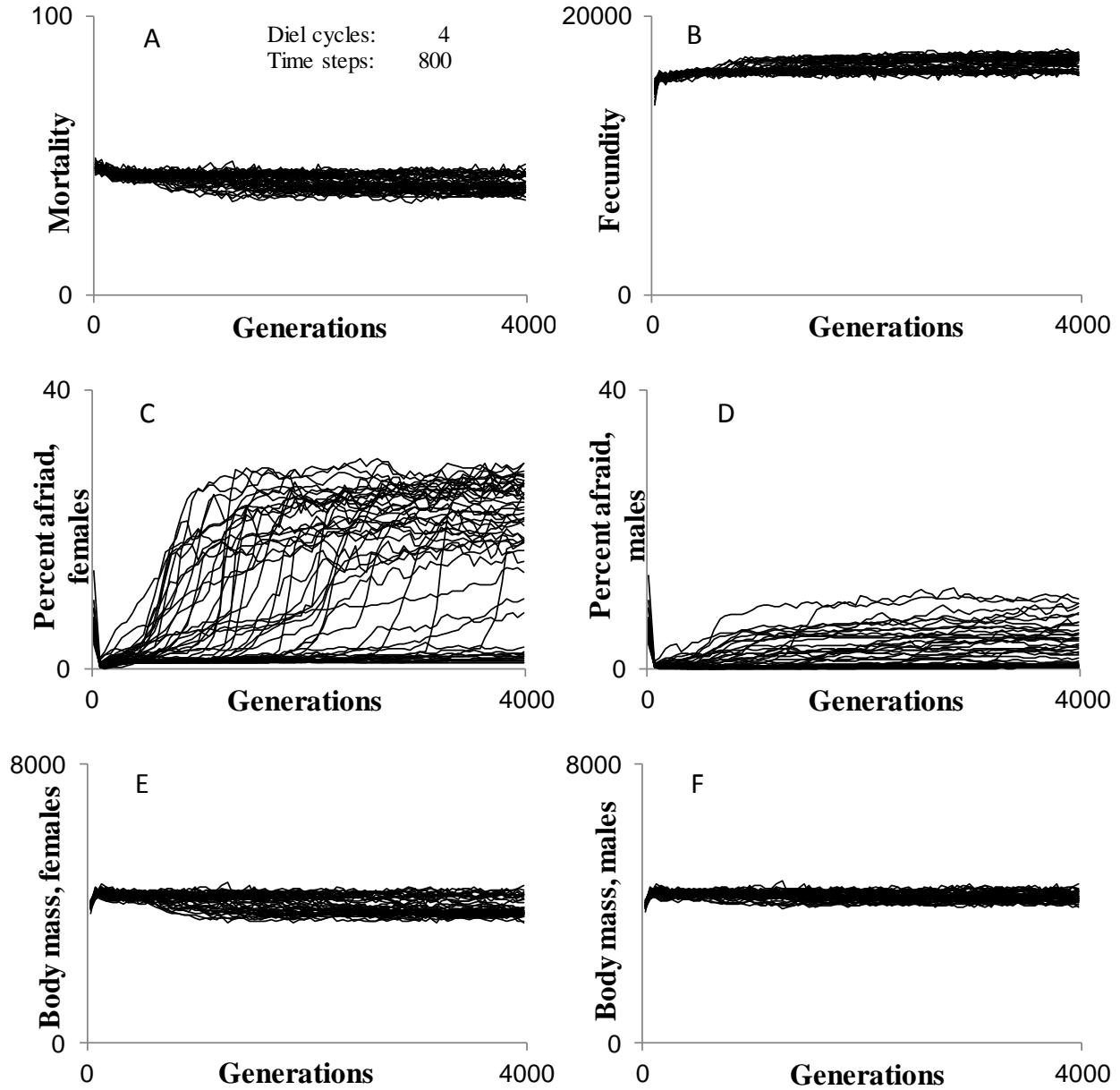


Figure A8: The average in each generation of mortality (A), fecundity (B), percent of time steps in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F) for all 50 simulations with 4 diel cycles and 200 time steps per cycle. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

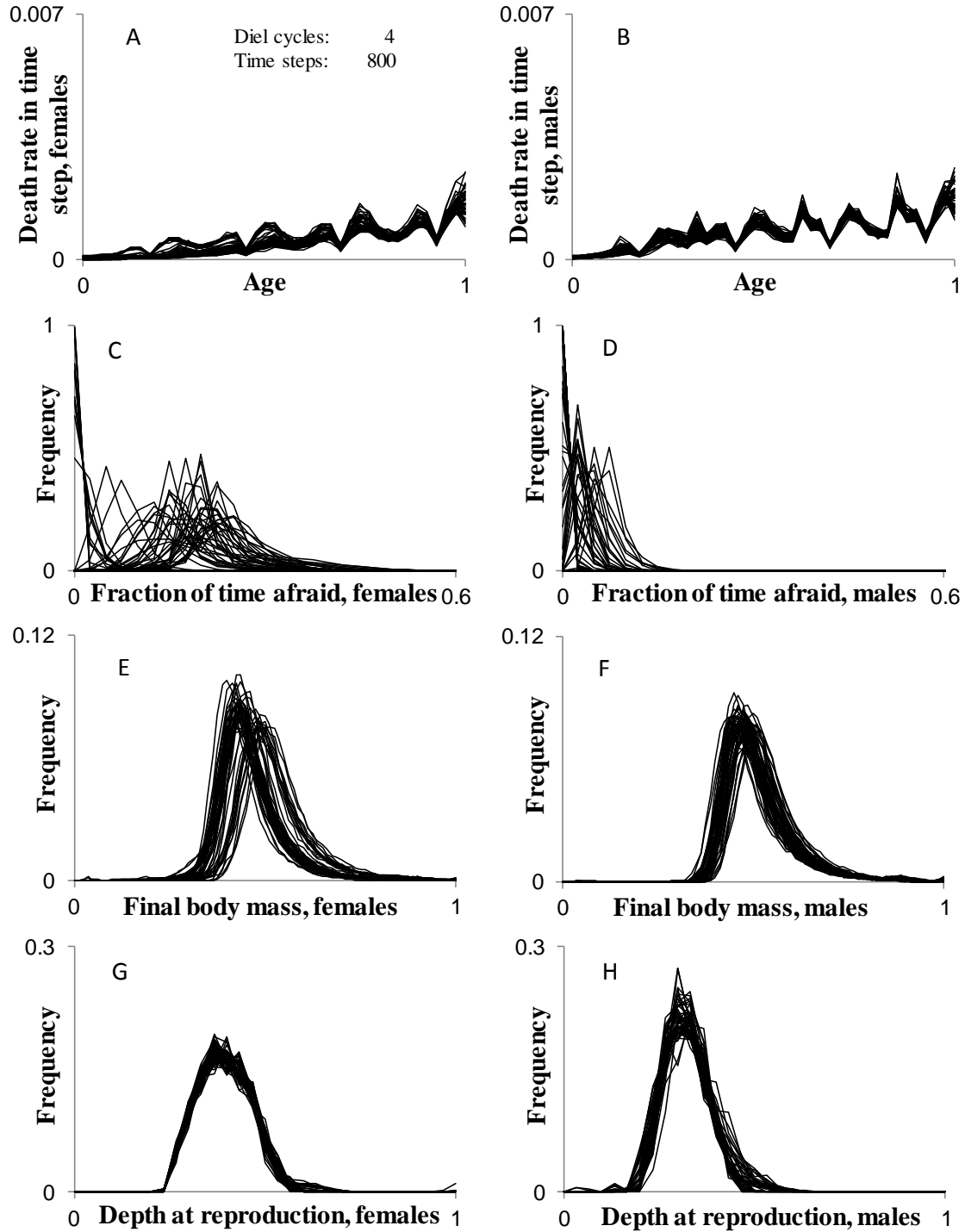


Figure A9: Comparison of the last 20 generations in the 50 simulations in the series with 4 diel cycles and 200 time steps per cycle. A- B) Death rates through life for females and males, respectively. C- D) The frequency of individuals and what fraction of the time they spent in the GOS afraid. E-F) Body mass at the end of a generation for females and males, respectively. G-H) The depth at which individuals reproduce (i.e. are located in the last time step) for females and males, respectively. 'Normal' generations are explained in Appendix A.1. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of

maximum age, maximum body mass and maximum depth, respectively. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively.

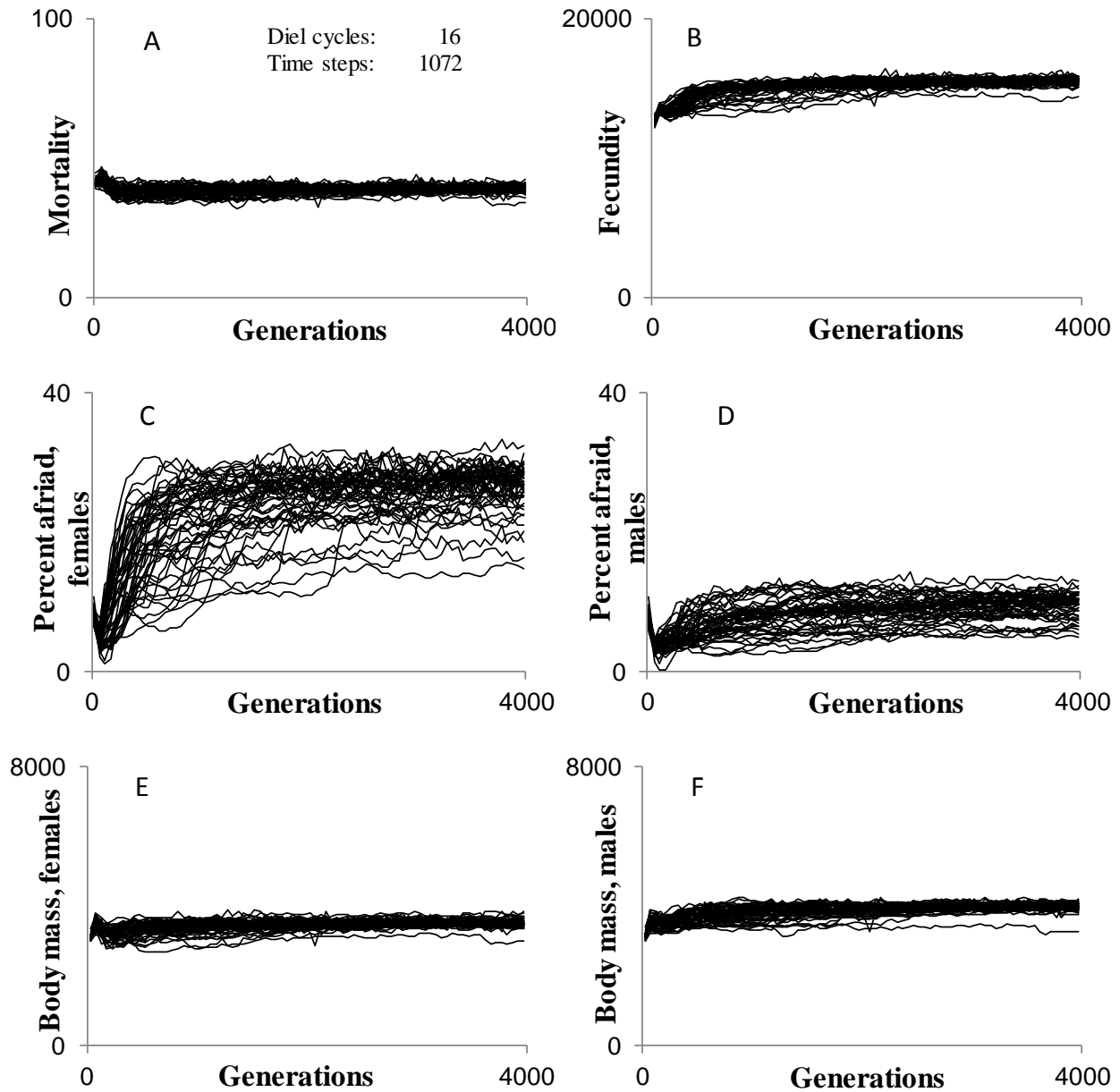


Figure A10: The average in each generation of mortality (A), fecundity (B), percent of time steps in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F) for all 50 simulations with 16 diel cycles and 67 time steps per cycle. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

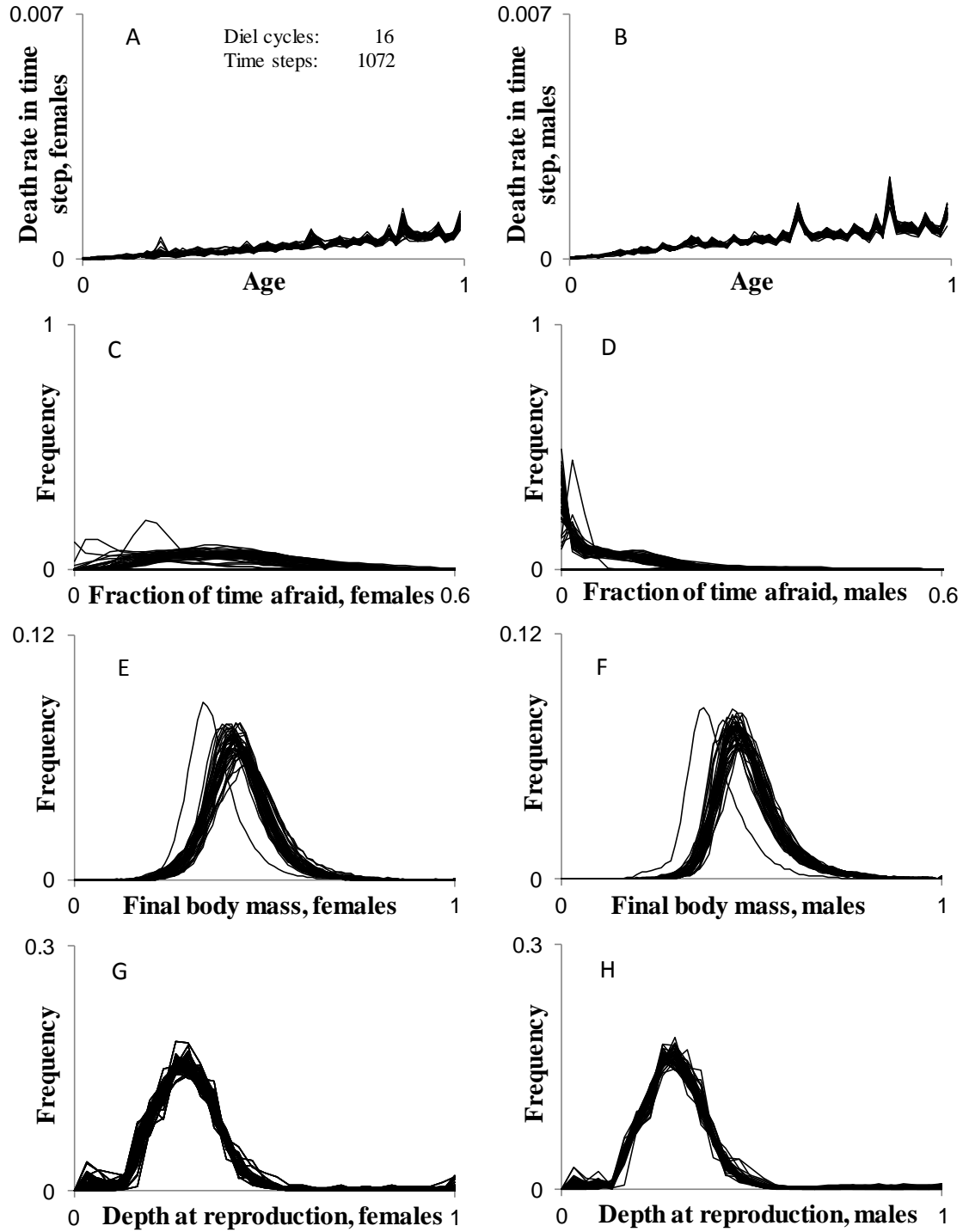


Figure A11: Comparison of the last 20 generations in the 50 simulations in the series with 16 diel cycles and 67 time steps per cycle. A- B) Death rates through life for females and males, respectively. C- D) The frequency of individuals and what fraction of the time they spent in the GOS afraid. E-F) Body mass at the end of a generation for females and males, respectively. G-H) The depth at which individuals reproduce (i.e. are located in the last time step) for females and males, respectively. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth,

respectively. 'Normal' generations are explained in Appendix A.1. X-axes in the panels A and B, E and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively.

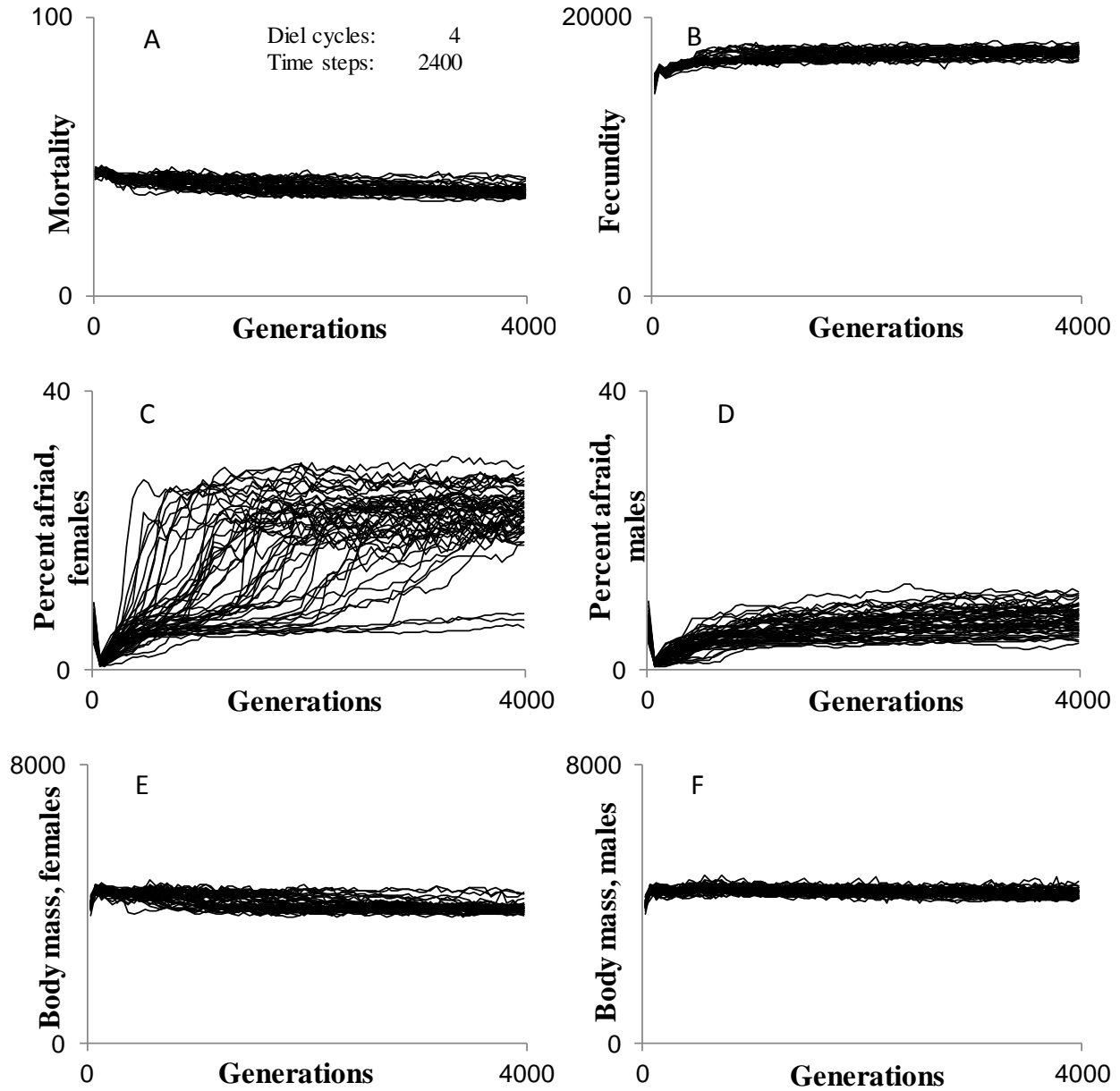


Figure A12: The average in each generation of mortality (A), fecundity (B), percent of time steps in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F) for all 50 simulations with 4 diel cycles and 600 time steps per cycle. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

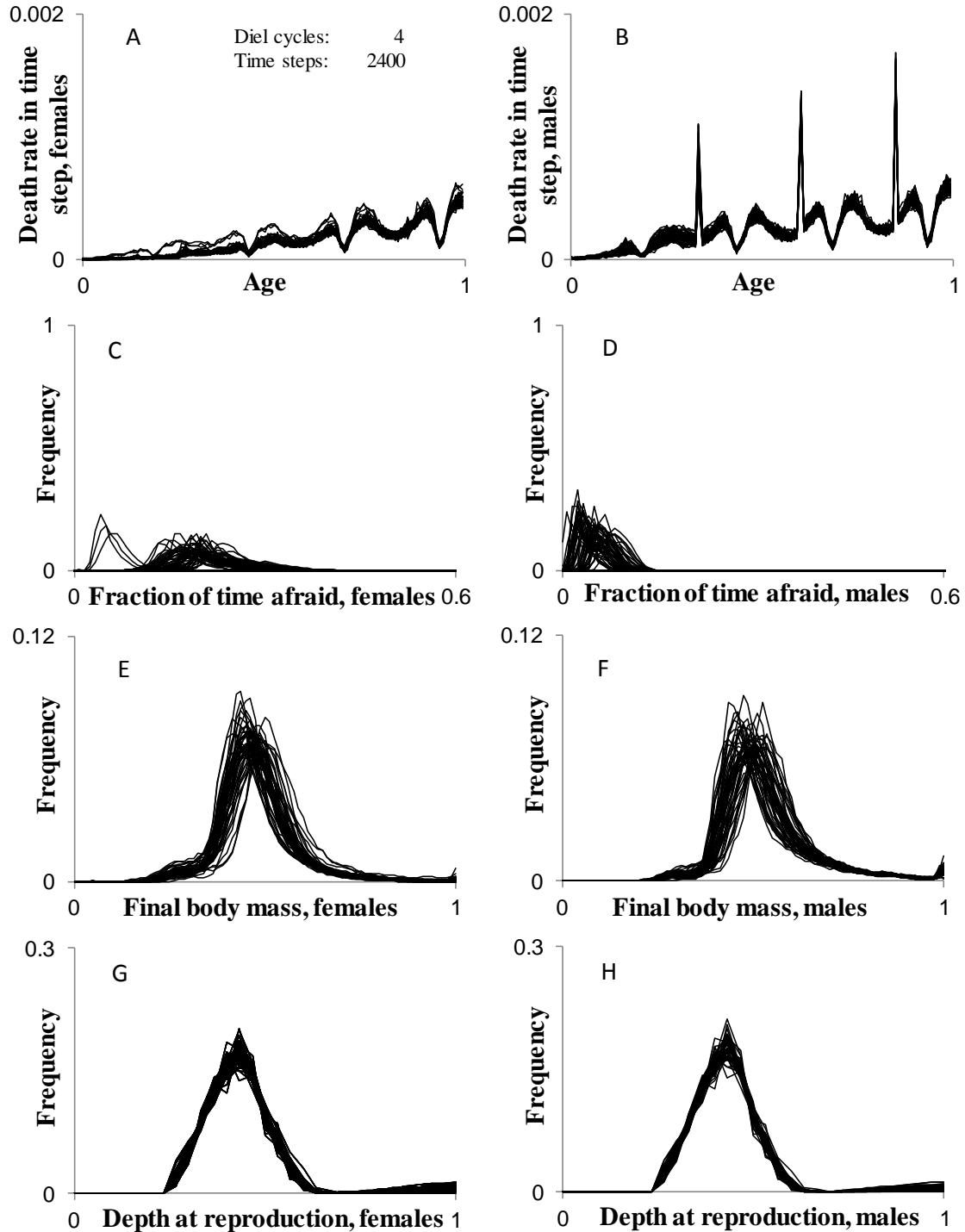


Figure A13: Comparison of the last 20 generations in the 50 simulations in the series with 4 diel cycles and 600 time steps per cycle. A- B) Death rates through life for females and males, respectively. C- D) The frequency of individuals and what fraction of the time they spent in the GOS afraid. E-F) Body mass at the end of a generation for females and males, respectively. G-H) The depth at which individuals reproduce (i.e. are located in the last time step) for females and males, respectively. 'Normal' generations are explained in Appendix A.1. X-axes in the panels A and B, E and F and F and G are scaled as a

fraction of maximum age, maximum body mass and maximum depth, respectively. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively.

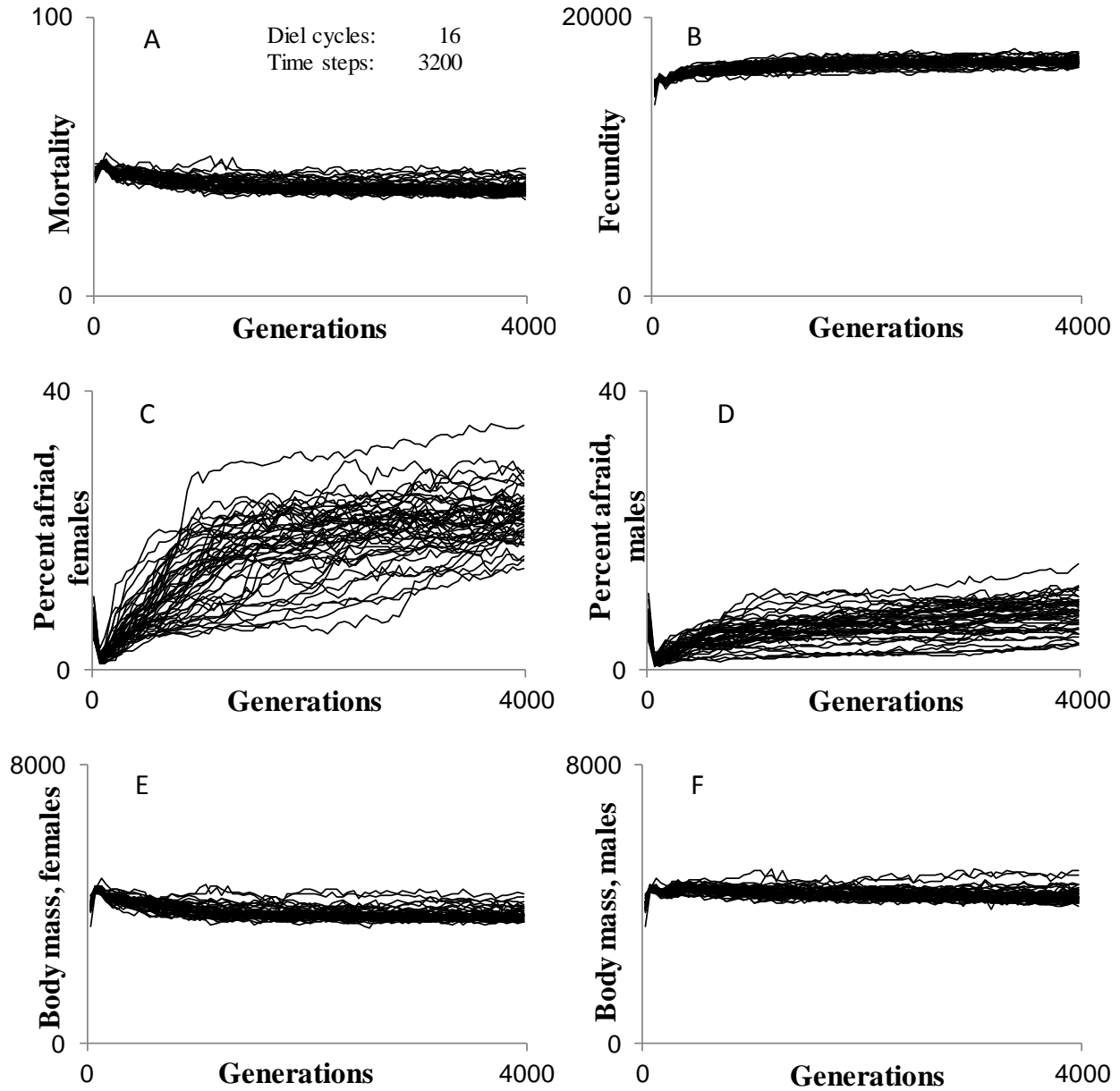


Figure A14: The average in each generation of mortality (A), fecundity (B), percent of time steps in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F) for all 50 simulations with 16 diel cycles and 200 time steps per cycle. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

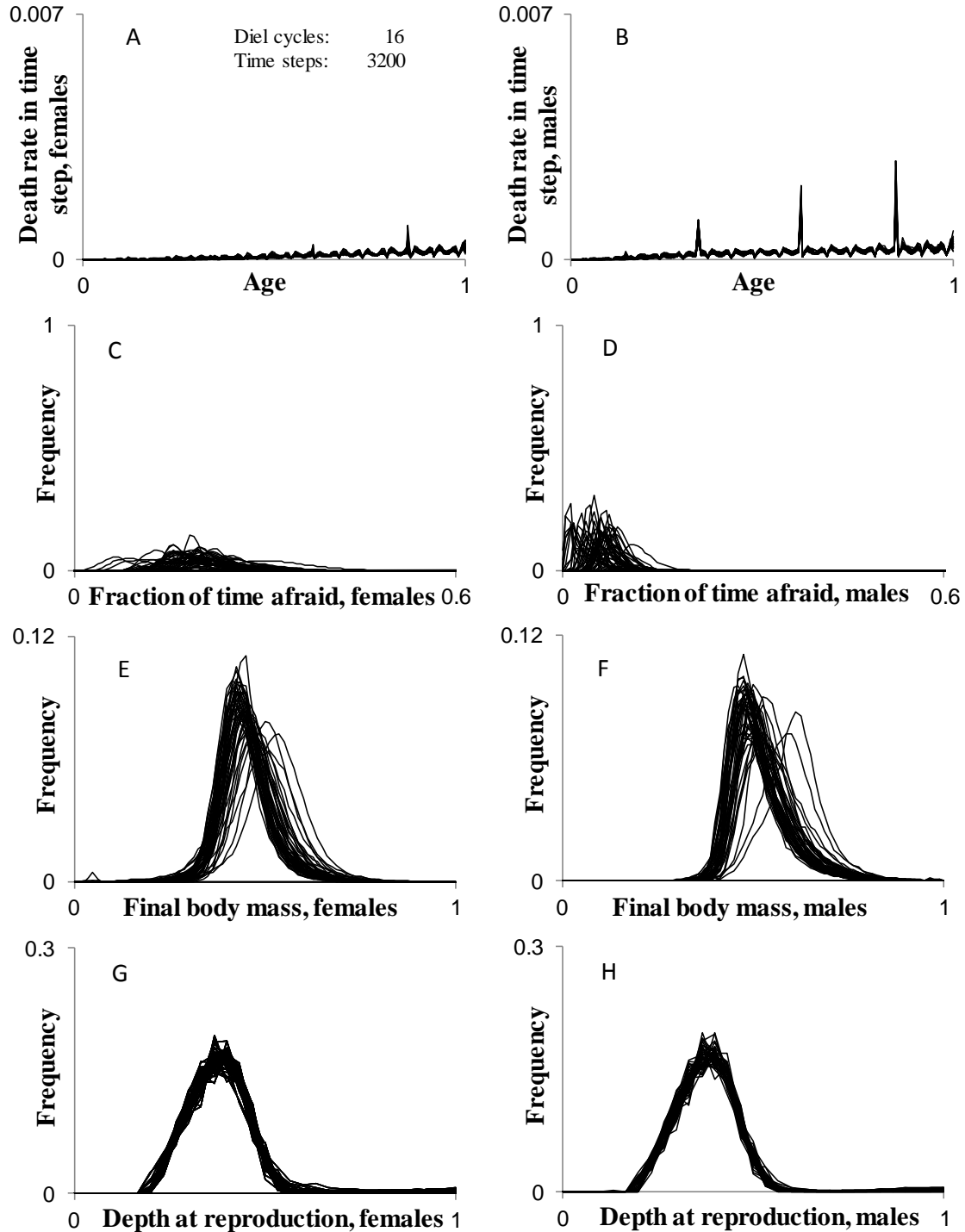


Figure A15: Comparison of the last 20 generations in the 50 simulations in the series with 16 diel cycles and 200 time steps per cycle. A- B) Death rates through life for females and males, respectively. C- D) The frequency of individuals and what fraction of the time they spent in the GOS afraid. E-F) Body mass at the end of a generation for females and males, respectively. G-H) The depth at which individuals reproduce (i.e. are located in the last time step) for females and males, respectively. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and

maximum depth, respectively. 'Normal' generations are explained in Appendix A.1. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively.

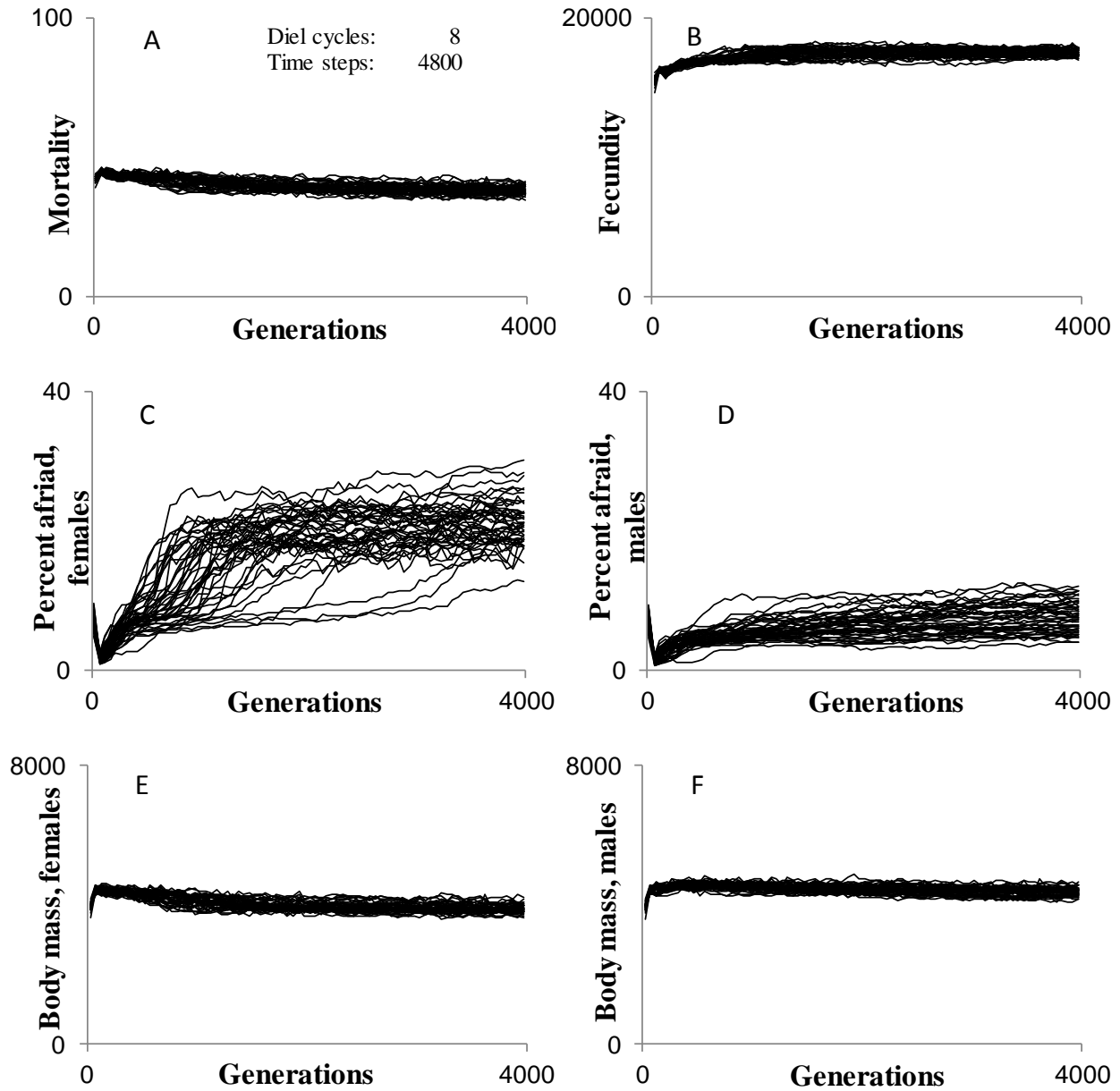


Figure A16: The average in each generation of mortality (A), fecundity (B), percent of time steps in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F) for all 50 simulations with 8 diel cycles and 600 time steps per cycle. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

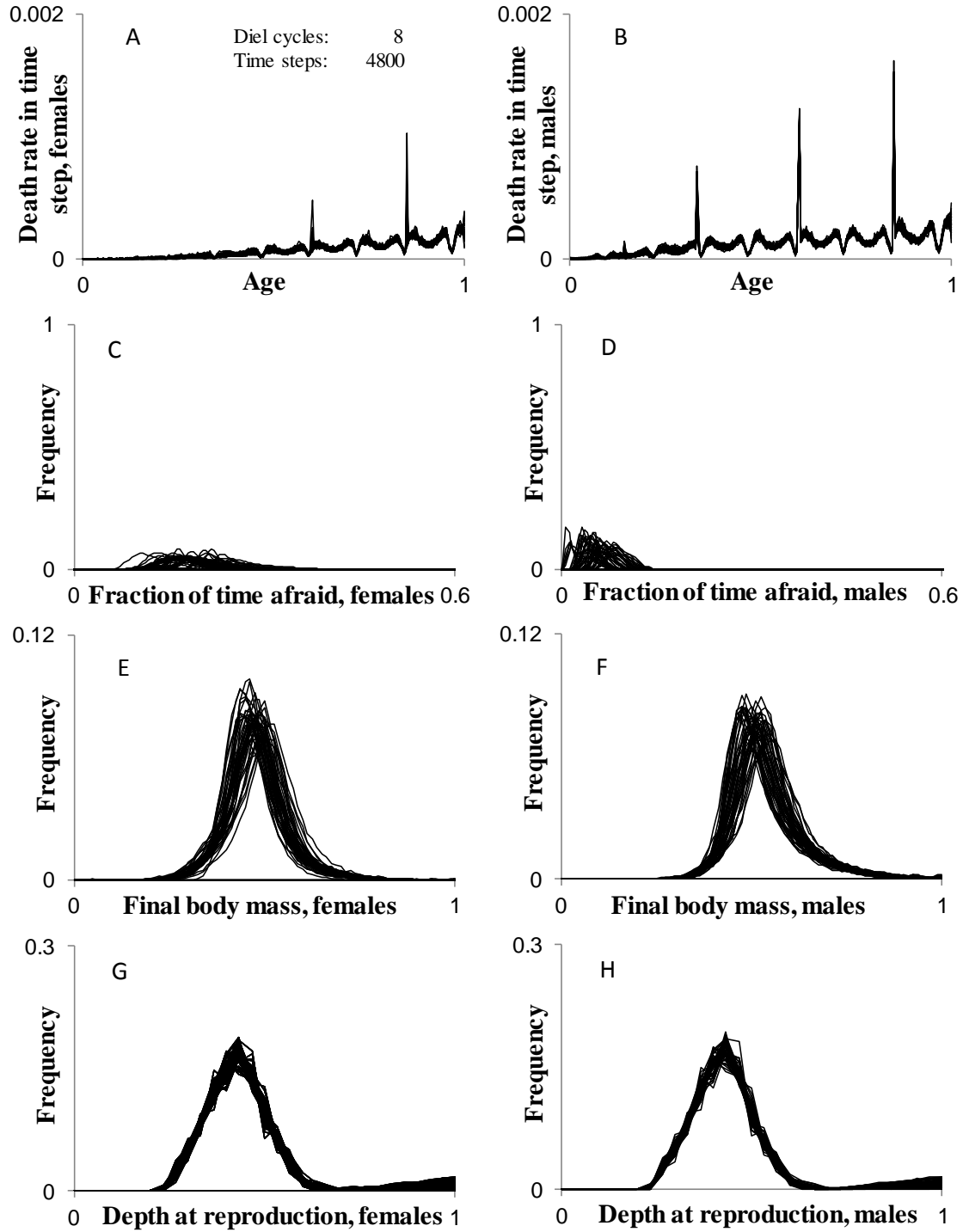


Figure A17: Comparison of the last 20 generations in the 50 simulations in the series with 8 diel cycles and 600 time steps per cycle. A- B) Death rates through life for females and males, respectively. C- D) The frequency of individuals and what fraction of the time they spent in the GOS afraid. E-F) Body mass at the end of a generation for females and males, respectively. G-H) The depth at which individuals reproduce (i.e. are located in the last time step) for females and males, respectively. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and

maximum depth, respectively. 'Normal' generations are explained in Appendix A.1. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively.

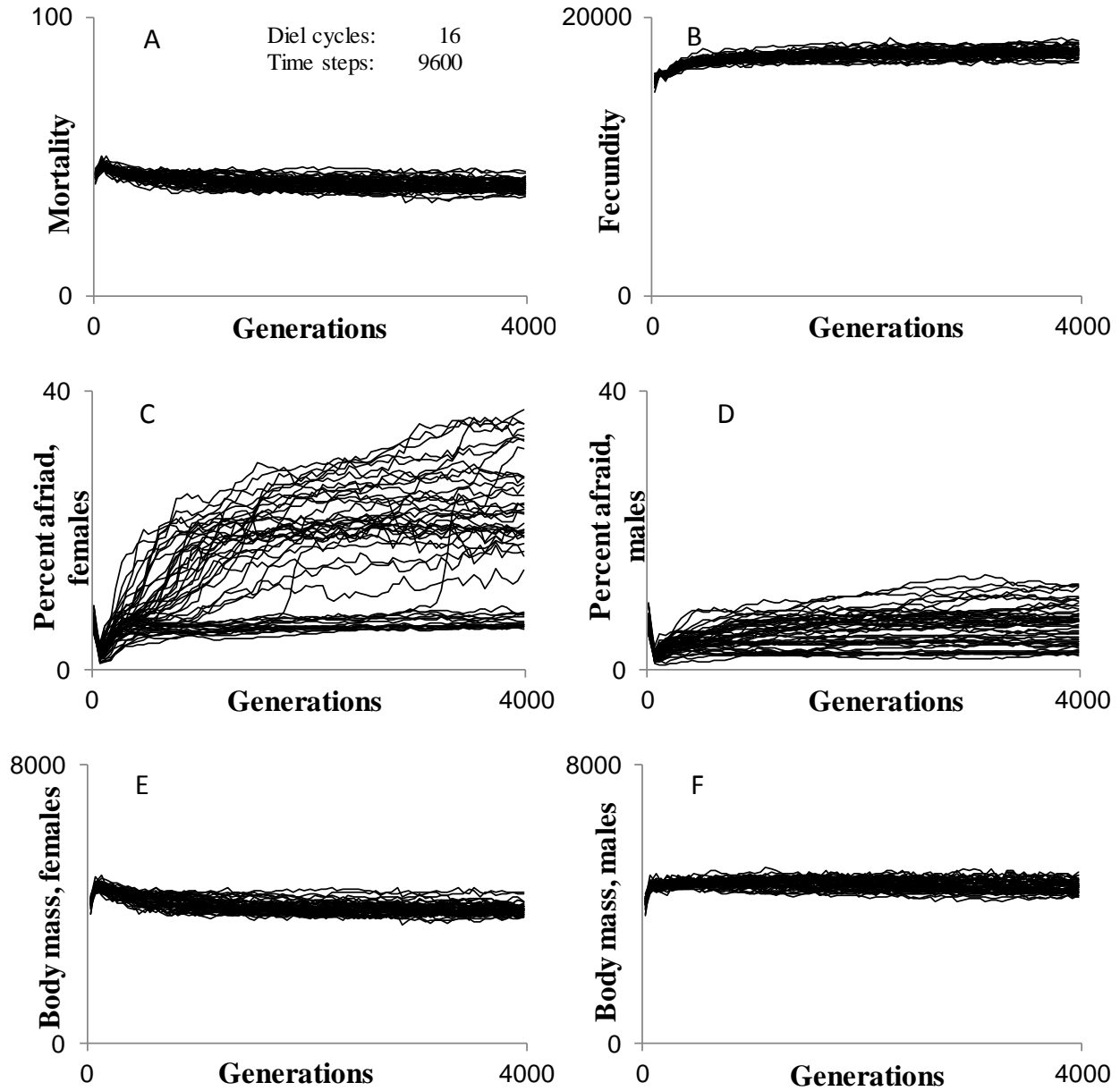


Figure A18: The average in each generation of mortality (A), fecundity (B), percent of time steps in the GOS afraid for females (C) and males (D) and final body mass for females (E) and males (F) for all 50 simulations with 16 diel cycles and 600 time steps per cycle. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

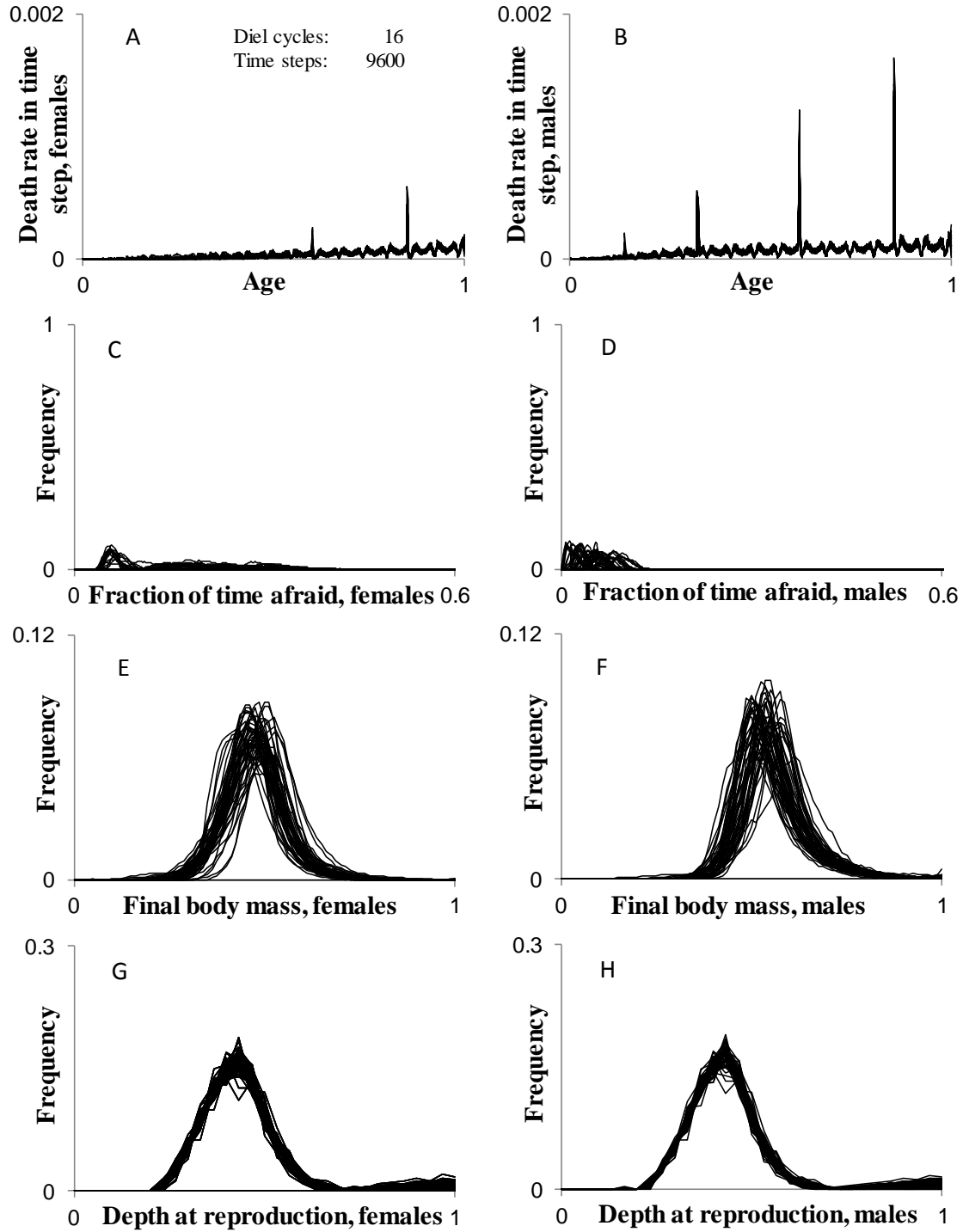


Figure A19: Comparison of the last 20 generations in the 50 simulations in the series with 16 diel cycles and 600 time steps per cycle. A- B) Death rates through life for females and males, respectively. C- D) The frequency of individuals and what fraction of the time they spent in the GOS afraid. E-F) Body mass at the end of a generation for females and males, respectively. G-H) The depth at which individuals reproduce (i.e. are located in the last time step) for females and males, respectively. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and

maximum depth, respectively. 'Normal' generations are explained in Appendix A.1. X-axes in the panels A and B, E and F and F and G are scaled as a fraction of maximum age, maximum body mass and maximum depth, respectively.

F Comparison of length of fear periods

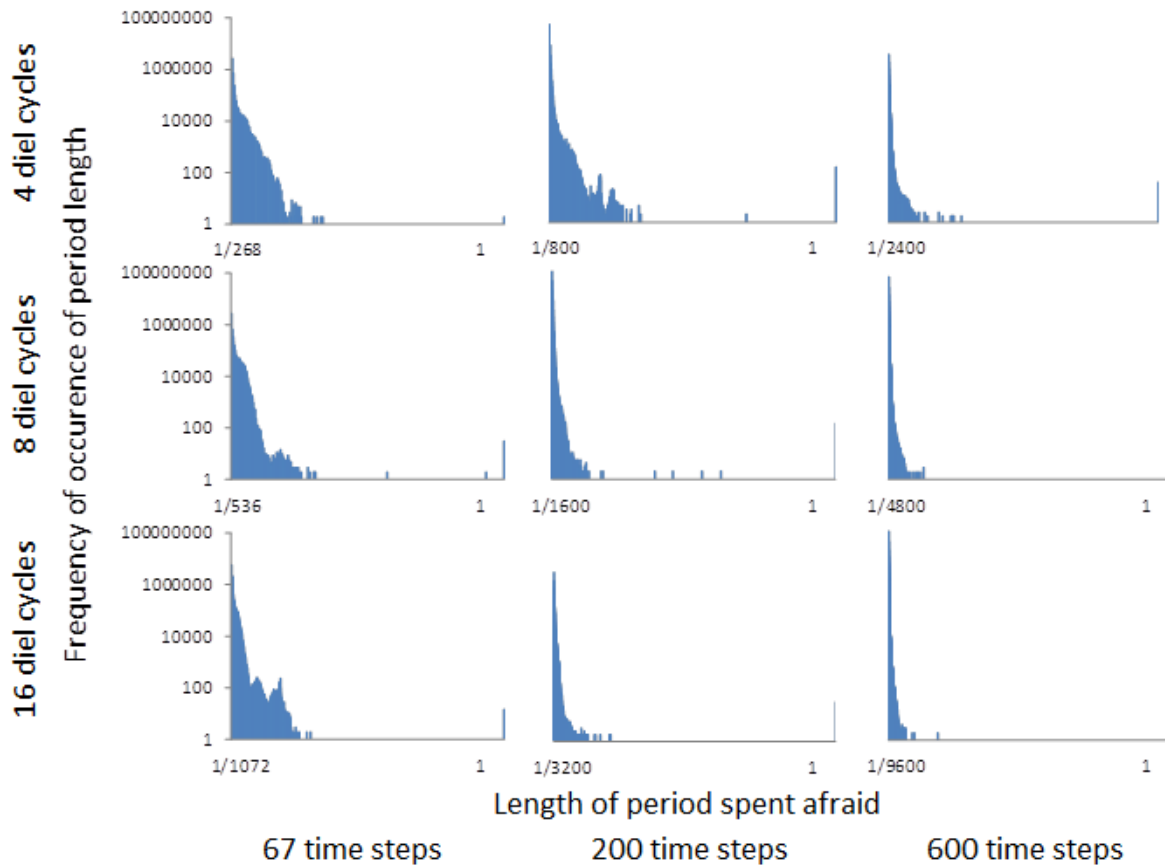


Figure A20: Comparison of length of the periods spent in the state afraid for both sexes in the last generation for the nine experiments. The X-axes display the length of the period in time steps divided by the total number of time steps in that experiment. The Y-axes are on a logarithmic scale. To the right in all panels, there are little peaks. These peaks represent individuals that were afraid in all time steps in the last generation. Only 'Normal' generations are used for comparison. 'Normal' generations are explained in Appendix A.1.

G Matlab script for statistical comparison of experiments and making figures

G.1 Explanation

This Matlab script contains two procedures. All data this script reads is produced by the program presented in App. 3.2. The first procedure uses the ‘anova1’ and ‘multcompare’ functions to test for differences between the nine experiments and to do a multiple comparison analysis. It also produces the plots in figure 14, 15 and 16 in the main text. The second procedure calculates linear least square lines for multiple traits between each 1000th generation that is needed for Table 1 and producing Figures 4 and 5 in the main text.

G.2 The script

```
%Comparing experiments and making figures
%Containing two prodecures
%BSA 26.03.14

clear all;
close all;
cd 'W:\Snorre\Fungerende maler til Matlab';

file = 'W:\HED23NR\FIMM\HED23 3NR DAYSandTS - series compasrion.xlsx';
sheet = 'Sheet5';
range = 'A1:I1200';

C = xlsread(file,sheet,range);

%Parameters for plot
l = 4;      %Line width CI
w = 2;      %Line width Marker
s = 15;     %Size marker
x = 'x';    %Marker

%Making tag to label all subplots. OBS start with 9!
tag =
{'16D,600TS','8D,600TS','16D,200TS','4D,600TS','8D,200TS','16D,67TS','4D,200TS',
', '8D,67TS','4D,67TS'};

% %Procedure 1
% %This precedure below was done for mortality, fecindity, fear (females and
males)
% %and final body mass (females and males) at generation 4000. (Only
% %mortality is shown.)
```

```

%Mortality
Mort4 = C(151:200,1:9);
[p,t,stats] = anova(Mort4); %ANOVA to test for differences between
experiments
[c,m,h,nms] = multcompare(stats); %Multiple comparison test to test which one
that are different
[nms(c(:,1)), nms(c(:,2)), num2cell(c(:,3:5))];
xlabel('Mortality','FontSize',24);
% Create ylabel
ylabel('Simulation series','FontSize',24);
set(gca, 'Parent',figure,...

'YTickLabel',{ '16D,600TS', '8D,600TS', '16D,200TS', '4D,600TS', '8D,200TS', '16D,67
TS', '4D,200TS', '8D,67TS', '4D,67TS'},...
'YTick',[1 2 3 4 5 6 7 8 9],...
'XLim',[30 50],...
'XTick',[30 50],...
'Position',[0.0701047542304593 0.141414141414141 0.901692183722804
0.783585858585859],...
'FontSize',20);
set(gca, 'XTickLabel', {'30','50'})
box(gca,'off');
set(gcf, 'Tag','boxplot','Name','Multiple comparison of means',...
'Color',[1 1 1]);
set(gcf,'NumberTitle','off');
graphnr = get(gca, 'Children');

%Changing colors on the plot that was made by MULTCOMPARE
set(graphnr(1), 'MarkerEdgeColor','none','LineStyle','-','Color',[0 1
0], 'LineWidth',1);
set(graphnr(3), 'MarkerEdgeColor','none','LineStyle','-.','Color',[0 1
0], 'LineWidth',1);
set(graphnr(5), 'MarkerEdgeColor','none','LineStyle','-','Color',[1 0
0], 'LineWidth',1);
set(graphnr(7), 'MarkerEdgeColor','none','LineStyle':','Color',[0 1
0], 'LineWidth',1);
set(graphnr(9), 'MarkerEdgeColor','none','LineStyle','-.','Color',[1 0
0], 'LineWidth',1);
set(graphnr(11), 'MarkerEdgeColor','none','LineStyle','-','Color',[0 0
1], 'LineWidth',1);
set(graphnr(13), 'MarkerEdgeColor','none','LineStyle':','Color',[1 0
0], 'LineWidth',1);
set(graphnr(15), 'MarkerEdgeColor','none','LineStyle','-.','Color',[0 0
1], 'LineWidth',1);
set(graphnr(17), 'MarkerEdgeColor','none','LineStyle':','Color',[0 0
1], 'LineWidth',1);
set(graphnr(2), 'MarkerEdgeColor',[0 1 0], 'Marker',x, 'Color',[0 1
0], 'MarkerSize',s, 'LineWidth',w);
set(graphnr(4), 'MarkerEdgeColor',[0 1 0], 'Marker',x, 'Color',[0 1
0], 'MarkerSize',s, 'LineWidth',w);
set(graphnr(6), 'MarkerEdgeColor',[1 0 0], 'Marker',x, 'Color',[1 0
0], 'MarkerSize',s, 'LineWidth',w);
set(graphnr(8), 'MarkerEdgeColor',[0 1 0], 'Marker',x, 'Color',[0 1
0], 'MarkerSize',s, 'LineWidth',w);
set(graphnr(10), 'MarkerEdgeColor',[1 0 0], 'Marker',x, 'Color',[1 0
0], 'MarkerSize',s, 'LineWidth',w);

```

```

set(graphnr(12), 'MarkerEdgeColor', [0 0 1], 'Marker', x, 'Color', [0 0
1], 'MarkerSize', s, 'LineWidth', w);
set(graphnr(14), 'MarkerEdgeColor', [1 0 0], 'Marker', x, 'Color', [1 0
0], 'MarkerSize', s, 'LineWidth', w);
set(graphnr(16), 'MarkerEdgeColor', [0 0 1], 'Marker', x, 'Color', [0 0
1], 'MarkerSize', s, 'LineWidth', w);
set(graphnr(18), 'MarkerEdgeColor', [0 0 1], 'Marker', x, 'Color', [0 0
1], 'MarkerSize', s, 'LineWidth', w);

% % Procedure 2
% % Comparing four points in evolution: 1000, 2000, 3000 and 4000 generations
% % Calculating a linear least squares line to see the amount of change after
% % generation 1000.
% % Done for Mortality, fecindity, fear (females and males)and final body
% % mass (females and males), but only shown for mortality

% MORTALITY

for j = 1:9;
Mort{j} = zeros(50,4);
end;

for j = 1:9;
    for i = 1:4;
        %Fecundity
        Mort{j}(1:50,i) = C((1+((i-1)*50)):(50+((i-1)*50)),j);
    end;
end;

B = zeros(9,4);
jj = 10; %Ensuring right tag on plots
for j = 1:9;
    jj = jj - 1;
    [p,t,stats] = anova1(Mort{j});
    [c,m,h,nms] = multcompare(stats);
    xlabel(string, 'FontSize', 24);
    % Create ylabel
    set(gca, 'Parent', figure, ...
        'YTickLabel', {'4000', '3000', '2000', '1000'}, ...
        'YTick', [1 2 3 4], ...
        'YLim', [0.5 4.1], ...
        'XLim', [0 100], ...
        'XTick', [0 100], ...
        'FontSize', 20);
    box(gca, 'off');
    set(gcf, 'Tag', 'boxplot', 'Name', 'Multiple comparison of means', ...
        'Color', [1 1 1]);
    set(gcf, 'NumberTitle', 'off', 'PaperSize', [1 1]);
    set(gca, 'XTickLabel', {'0', '100'});
    graphnr = get(gca, 'Children');

    set(graphnr(1), 'MarkerEdgeColor', 'none', 'LineStyle', '-', 'Color', [1 0
0], 'LineWidth', 1);
    set(graphnr(3), 'MarkerEdgeColor', 'none', 'LineStyle', '-', 'Color', [1 0
0], 'LineWidth', 1);

```

```

    set(graphnr(5), 'MarkerEdgeColor', 'none', 'LineStyle', '-', 'Color', [1 0
0], 'LineWidth', 1);
    set(graphnr(7), 'MarkerEdgeColor', 'none', 'LineStyle', '-', 'Color', [1 0
0], 'LineWidth', 1);

    set(graphnr(2), 'MarkerEdgeColor', [1 0 0], 'Marker', x, 'Color', [1 0
0], 'MarkerSize', s, 'LineWidth', w);
    set(graphnr(4), 'MarkerEdgeColor', [1 0 0], 'Marker', x, 'Color', [1 0
0], 'MarkerSize', s, 'LineWidth', w);
    set(graphnr(6), 'MarkerEdgeColor', [1 0 0], 'Marker', x, 'Color', [1 0
0], 'MarkerSize', s, 'LineWidth', w);
    set(graphnr(8), 'MarkerEdgeColor', [1 0 0], 'Marker', x, 'Color', [1 0
0], 'MarkerSize', s, 'LineWidth', w);

    set(graphnr(9), 'Color', [1 1 1]); %Making vertical marker lines white
title([]); %Making empty title
legend(tag(jj), 'EdgeColor', [1 1 1], 'Location', 'SouthWest');

%Finding slope for alle nine series, linear regression
F = Mort{j};

for h = 1:4
    for i = 1:50
        F2(i+(h*50)-50,1) = h;
        F2(i+(h*50)-50,2) = F(i,h);
    end;
end;

%Making scatterplot with linear least squares line
scatter(F2(:,1),F2(:,2));
lsline; %linear least squares line
stop = 200;
F3 = zeros(200,1);
F3 = F2(:,2);

N = (F2(:,2));
rem = isnan(N);
F4 = F2(~rem); %Removing rows that was unfinished simulations
F5 = F3(~rem);

p = polyfit(F4,F5,1); %Finner stigningstall og skjæring med Y-akse.

B(j,1:2) = p;
B(j,3) = nanmean(F2(1:50,2)); %Mean at gen 1000
B(j,4) = B(j,1)/(nanmean(F2(1:50,2))); % Prosentvis økning for hver 1000 gen
etter gen 1000 ifølge lineær regresjon
for h = 1:4
    for i = 1:50
        F2(i+(h*50)-50,1) = 0;
        F2(i+(h*50)-50,2) = 0;
    end;
end;
end;
end;

```


H Matlab script for efficient uploading processes to FIMM

The main experiment for this master thesis consists of 600 simulations. These simulations were executed at Parallab, the UiB center for high performance computing, operated by Uni computing. The computer used is called FIMM. Every simulation needs a parameter file where the simulation number and the conditions the particular simulation are specified. In addition, a batch file is needed. To avoid having to manually changing the specifications in all these files, I wrote a script in Matlab that did this for me.

H.1 Explanation for the script “Simulations on FIMM”

Before starting the program, the folder that is to be copied to FIMM needs to contain the preferred version of the program that will be run. In this case HED23.f90. Also the folder needs to contain the parameter file Commonfish.txt and i101-parameters.txt. The latter one has to be renamed “Ni101-parameters.txt” before starting.

The Matlab script runs through a loop of the length of the wanted number of simulations. This loop starts of by creating a folder for the simulation and copies a version of Commonfish.txt and the HED23.f90 into it. After this it writes the DOS-code needed for uploading on FIMM to a pbs file, here called snorre2.pbs. This file is located in the folder that contains all the folders that each contains one simulation. Then a batch file is written and put in the simulation folder. This file is called snorre.pbs. The same is done with the parameter file i101-parameters.txt.

All the simulation folders can now be uploaded to FIMM and copying all the DOS-code from the snorre2.pbs file to the transfer window, all simulations will be executed.

H.2 The script

```
%      SIMULATIONS on FIMM      %  
  
%Making folders, snorre2.pbs, pbs-files and parameter files  
clear all;  
sep = filesep; % = \  
slash = '/';
```

```

%%%%%%%%%% PARAMETERS %%%%%%%%%%%

number = 54; % Number of duplicates
that will be made
fdir = ['W:' sep 'HED23NR' sep 'FIMM' sep '24Feb']; % Directory where all
folders are created
f90 = 'HED23.f90'; % Name of HEDxx.f90 file
para = 'il01-parameters.txt'; % Name of parameter file
common = 'Commonfish.txt'; % Name of Commonfish file
letter = 'X'; % Letter before expt nr
(X or S)
pbs = ['snorre.pbs']; % Name of pbs.files
dest = ['cd /work/snorre/24Feb']; % Destination for sim series
(on fimm)
trans = ['sed -i -e 's/^M$/'' -e 's/\\x0D$/''']; % Two backlashed before
x0D,becomes one when printed to txt-file.
jobn = ['NR-24Feb-']; % Name used on sim set on
fimm
runtag = ['20T07D']; %! runtag 6 alphanumeric
characters, must be allowed in file name

%%%%%%%%%%

%% PROGRAM STARTS

cd (fdir);
fdirf90 = strcat(fdir,sep,f90);
fdirP = strcat(fdir,sep,para);
fdirC = strcat(fdir,sep,common);
letter2 = strcat(letter,'0');

T = strcat(dest,slash,letter); %Directory
V = strcat(dest,slash,letter2);%Directory
U = ['qsub ' pbs];
S = [trans ' ' pbs];
pbs2 = ['2' pbs];
zz = '';

file = fopen(pbs2,'w'); %Opening pbs file

% il01-parameters.txt:
dd = ' ! experiment simulation number in current experiment';
rn = ' ! runtag 6 alphanumeric characters, must be allowed in file
name';
run = strcat(runtag,rn);

% pbs file:
a = '#! /bin/sh -';
b = '#';
c = '# Give the job a name (optional)';
d = strcat('#PBS -N ', jobn);
f = '#';
g = '# Specify the project the job should be accounted on (obligatory)';

```

```

h = '#PBS -A bio';
k = '#';
l = '# The time this job needs on 1 CPU (obligatory)';
m = '#PBS -l ncpus=1,walltime=300:00:00'; % WALLTIME
n = '#';
o = '# Memory requirements (obligatory)';
p = '#PBS -l mem=1000mb';
q = '#';
r = '# Write the standard output and errors of the job to file (optional)';
s = '#PBS -j oe -o HED23.out.txt'; %HED23
t = '# Make sure I am in the correct directory';
u = '# set unlimited stack size';
v = 'ulimit -s unlimited';
w = 'module unload pgi';
y = 'module load intel';
aa = 'ifort -O1 HED23.f90 -o HED23'; %HED23
bb = '# Invoke the (sequential!) executable';
cc = './HED23'; %HED23

%Starting loop
for i = 1:number
    %%% Creating folders and Copy Commonfish and .f90 into all folders
    z = num2str(i);
    if (i > 9)
        dir = letter;
        dz = strcat(d,letter,z,zz);
    else;
        dir = letter2;
        dz = strcat(d,letter2,z,zz);
    end;
    dirz = strcat(dir,z);
    mkdir(fdir,dirz)
    fdirz = strcat(fdir,sep,dirz);
    copyfile(fdirf90,fdirz);
    copyfile(fdirC,fdirz);

%% 2snorre.pbs
TT = strcat(T,z);
VV = strcat(V,z);
if i < 10 ;
    fprintf(file,VV);
    fprintf(file,'\r\n'); %New line
else;
    fprintf(file,TT);
    fprintf(file,'\r\n');
end;
fprintf(file,S);
fprintf(file,'\r\n');
fprintf(file,U);
fprintf(file,'\r\n');

%%%%%%%%%% pbs files

    cd (fdirz);
    filen = fopen(pbs,'w');

```

```

%Start printing
fprintf(filen,a);
fprintf(filen,'\r\n');
fprintf(filen,b);
fprintf(filen,'\r\n');
fprintf(filen,c);
fprintf(filen,'\r\n');
fprintf(filen,dz);
fprintf(filen,'\r\n');
fprintf(filen,f);
fprintf(filen,'\r\n');
fprintf(filen,g);
fprintf(filen,'\r\n');
fprintf(filen,h);
fprintf(filen,'\r\n');
fprintf(filen,k);
fprintf(filen,'\r\n');
fprintf(filen,l);
fprintf(filen,'\r\n');
fprintf(filen,m);
fprintf(filen,'\r\n');
fprintf(filen,n);
fprintf(filen,'\r\n');
fprintf(filen,o);
fprintf(filen,'\r\n');
fprintf(filen,p);
fprintf(filen,'\r\n');
fprintf(filen,q);
fprintf(filen,'\r\n');
fprintf(filen,r);
fprintf(filen,'\r\n');
fprintf(filen,s);
fprintf(filen,'\r\n');
fprintf(filen,'\r\n'); %Extra line
fprintf(filen,t);
fprintf(filen,'\r\n');

if i < 10 ;
fprintf(filen,VV);
fprintf(filen,'\r\n'); %New line
else;
fprintf(filen,TT);
fprintf(filen,'\r\n');
end;

fprintf(filen,'\r\n');
fprintf(filen,u);
fprintf(filen,'\r\n');
fprintf(filen,v);
fprintf(filen,'\r\n');
fprintf(filen,'\r\n');
fprintf(filen,w);
fprintf(filen,'\r\n');
fprintf(filen,y);
fprintf(filen,'\r\n');
fprintf(filen,'\r\n');
fprintf(filen,aa);

```

```

fprintf(filen, '\r\n');
fprintf(filen, '\r\n');
fprintf(filen, bb);
fprintf(filen, '\r\n');
fprintf(filen, cc);

%%%%%%%%%%%%% Creates a parameter file in every folder with the right
%%%%%%%%%%%%% experiment nr
cd (fdir);

fid = fopen('Ni101-parameters.txt', 'r');
j = 1;

tline = fgetl(fid);
A{j} = tline;
while ischar(tline)
    j = j+1;
    tline = fgetl(fid);
    A{j} = tline;
end
fclose(fid);

cd (fdirz);

par = fopen(para, 'w');
fprintf(par, '%s', A{1});
fprintf(par, '\r\n');
zdd = strcat(z, dd);
fprintf(par, zdd);
fprintf(par, '\r\n');
fprintf(par, run);
for ii=3:length(A)
    fprintf(par, '%s', A{ii});
    fprintf(par, '\r\n');
end;

end; %End loop for nr sim

```