Schoorl meeting 2018

Nederlandse **V**ereniging voor **T**heoretische **B**iologie
Thursday 5th and Friday 6th of April, 2018

Nederlandse Vereniging Theoretische Biologie

The Year of Mathematical Biology 2018

NWO
Netherlands Organisation for Scientific Research

Dear participants,

A little earlier than usual, I once again welcome everyone to the Annual NVTB meeting. As every other year, I am looking forward to learn from you all, and I can only assume the same is true for everyone attending! The number of participants (30) is a little low compared to previous years. While we had to sail a pretty tight ship last year, the schedule is much less full this year, meaning there's much more time for informal discussions during coffee!

The invited speaker on Thursday (April 5th) is Bert Theunissen, an expert on the history and philosophy of science, who will talk about scientific integrity and the skills required for future researchers to cope with the everyday dilemas and responsibilities. On Friday, Sander van Doorn will tell us about the bio-molecular network that is the basis of chemotaxis in bacteria as a minimal example of a complex trait.

Like every year, the meeting is subsidised by NWO, allowing us to host an affordable meeting. For this we are of course very grateful. We are also working together with the Year of Mathematical Biology 2018 (YoM), whose main objective is to celebrate the increase and importance of applications of mathematics to biology and life sciences in the last years[1]

Lastly, all young speakers are once again eligible for the NVTB-presentation award of € 100,-! All students (bachelor, masters, and PhD candidates) can win this price, so *zet 'm op*! (give it your best shot!)

We wish you a pleasant stay, and an interesting meeting!

The board of the NVTB,

Jaap Kaandorp (Chair)
Lotte de Vries (Treasurer)
Bram van Dijk (Secretary)

[1]For more information and events organised by YoM, see https://www.esmtb.org/news/141-year-of-mathematical-biology-2018

Practical information:

Address:
Dopersduin
Oorsprongweg 3
1871 HA Schoorl
phone: (072) 509 12 74
website: www.dopersduin.nl.

Nights and Meals:
In "Dopersduin" the overnight accommodation consists of simple 4 person bedrooms with bunk-beds, as well as double and single bedrooms with shower and toilet. All prices include overnight stay and meals. The four-bedded rooms do not included bed linnen, which you can rent for € 8.50, but you can also bring your own sleeping bag and pillow case.

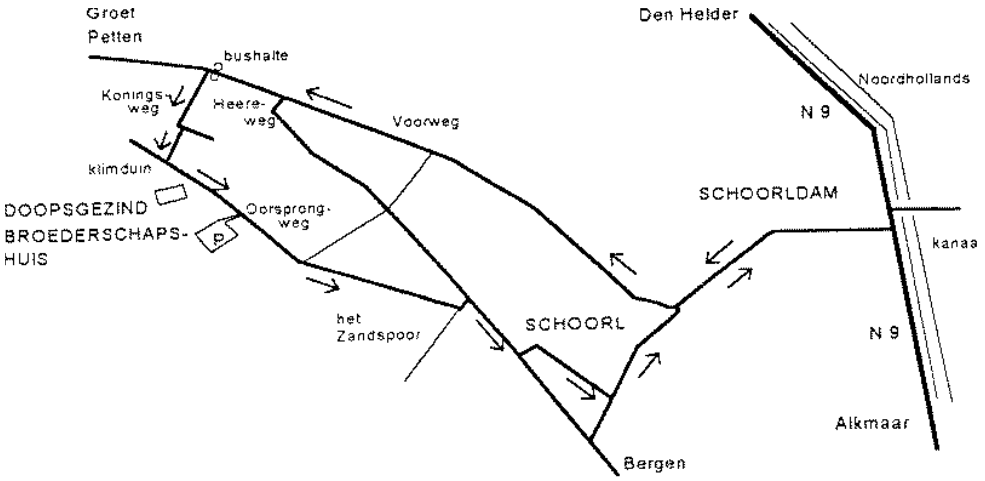
Finances:
Thanks to a subsidy of the NWO–ALW, we are able to keep the costs for participants low. You can either pay at registration thursday morning (make sure to bring some cash to the meeting) or transfer beforehand. Below is an overview of the prices:

Membership fee	€ 12,-
Single day attendance	€ 50,- *(non-members € 65,-)*
Single day students*	€ 30,- *(non-members € 45,-)*
Two days with four-bedded room *(No bed linnen included, bring a sleeping bag or hire bed linnen for € 8,50)*	€ 55,- *(non-members € 70,-)*
Four-bedded room students* *(No bed linnen included, bring a sleeping bag or hire bed linnen for € 8,50)*	€ 35,- *(non-members € 50,-)*
Double room	€ 80,- *(non-members € 95,-)*
Single room	€ 100,- *(non-members € 115,-)*

*Bachelor and master students.

To get to Schoorl by public transport:
• At the trainstation in Alkmaar, you can cross the railway tracks at the end of the platform (in the direction to Den Helder) to arrive at the busstation directly. From Alkmaar take bus 151 (10u03m is recommended) to Sint Maartensvlotbrug and exit at the stop "Schoorl-Koningsweg" (that takes a little over 15 minutes). The bus departs every 30 minutes.
• In Schoorl: cross the road and follow the Koningsweg to the dune. Go left into the Oorsprongweg, and after 100 meters the Doopsgezind Broederschapshuis is on your right. The walk takes about 5 minutes.

To get to Schoorl by car:
Take the N9 (*Alkmaar–Den Helder*) and exit at *Schoorldam* to Schoorl. After about 750 meter keep right at the crosspoint in the direction *Groet/Petten*. This road is called Voorweg and later Heereweg. After about 1 kilometer go straight at the crossing to *Centrum*. After 200 meter turn right onto the Koningsweg (there is a sign Dopersduin there). At the end, turn left onto the Oorsprongweg (this is a one-way road, so be sure to come from this direction). After about 100 meters, Dopersduin is on your right. The driveway leads to the parking lot.



Programme NVTB Meeting 2018

Day 1: Thursday April 5th

10:30	**Registration (Tea/Coffee)**
11:00 - 11.10	Jaap Kaandorp, *Opening statements*
11:10 - 12.00	Bert Theunissen, *What scientific integrity is really about*
12.00 - 12.25	Helen Kruize, *Evolutionary fine-tuning of the bacterial chemotaxis network to different ecologies*
12:30 - 13:30	**Lunch**
13:30 - 13:55	Hilje Doekes, *Evolution of bacteriocin regulation by local cell density cues*
13:55 - 14:20	Inge Wortel, *Learning by example: How T cells learn to discriminate "self" from "foreign" during negative selection*
14:20 - 14.40	**Tea/Coffee**
14:40 - 15.05	Fransje van Weerden, *Quantifying the effects of lateral and frontal visual fields on transmission of information in foraging groups*
15.05 - 15.30	Shabaz Sultan, *How Immune Cells Find Each Other; In Silico Model of Fibroblastic Reticular Network Structure and Morphology*
15:45 - 16.15	**Registration continued**
16:15 - 17:00	**Yearly (general) meeting**
18:00	**Dinner**
19:15	**Getting lost in the dunes of Schoorl**

Day 2: Friday April 6th

08:30	**Breakfast**

9:30 - 10.20	Sander van Doorn, *Unfolding the complexity of a three-part brain*

10:20 - 10.45	Anieke van Leeuwen, *Critical chronicity: acute and chronic infections emerge in a single within-host model due to resource modulation by parasites*
10:45 - 11.05	**Tea/Coffee**
11:05 - 11.30	Bram van Dijk, *Virtual microbes evolve to anticipate the predictable long-term evolution experiment*
11:30 - 11.55	Sjors Stouten, *Mathematical modelling of radiation-induced DNA damage repair and carcinogenesis*
11:55 - 12.25	Glenn Mulder, *Evolution of lysogeny and bacteriophage communication*
12:30 - 13.30	**Lunch**
13:30 - 13.55	Gerard Jagers op Akkerhuis, *Biological organization three decades after Ernst Mayr*
13.55 - 14.20	Remie Janssen, *Combining gene trees into a phylogenetic network*
14:20 - 14.55	Lotte de Vries *Linking life history theory, population genetics and population ecology using evolutionary demography: a matrix population model approach.*
14:55 - 15.15	**Tea/Coffee**
15:15 - 15.30	Jaap Kaandorp, *Presentation price & closing words*

Abstracts

Thursday April 5th

What scientific integrity is really about

Bert Theunissen
Utrecht University

Cases of fraud, the replication crisis, worries about sloppy science in an increasingly competitive academic climate, and public misgivings about the status of scientific knowledge and the independence of scientific researchers, have brought about a feeling of crisis in academic circles. In reaction, most universities and research institutions have drawn up codes of conduct for proper scientific conduct.
As useful as they are, such codes of conduct will not solve the problems at hand. Codes of conduct mostly deal with what is right and wrong, yet they ignore the vast grey area in which it is far from clear what is acceptable behaviour and what is not. Moreover, scientists in different research cultures think differently about such matters. More often than not, integrity issues present themselves to scientists as dilemmas that have no clear-cut solution and that require scientists to reflect more deeply about their role and responsibilities.
At the moment, future researchers are hardly prepared for dealing with such dilemmas. What is needed, therefore, is systematic attention to what research integrity entails in university education. Students must be made streetwise, meaning that they can recognize and effectively deal with the everyday dilemmas of scientific integrity they will encounter as researchers.
My lecture will discuss the nature of such dilemmas by way of a number of examples, and I will venture to suggest some strategies how to deal with them.

Evolutionary fine-tuning of the bacterial chemotaxis network to different ecologies

Helen Kruize
GELIFES and TRÊS

Understanding the evolution of complex traits is a major challenge in post-genomic biology. Here, we study the bacterial chemotaxis network of E. coli as a minimal example of a complex trait, and analyze the optimization of this network to different environments by natural selection. Building on detailed molecular data available for E. coli 's chemotactic network, we developed a computational model to reconstruct the effect of genomic network mutations on cell motility patterns and chemotactic performance. Using in silico evolution experiments, we then searched for genotypes that optimize chemotactic performance in four different environments. Contrary to theoretical predictions, we observed

no strong trade-off between the ability to locate resource peaks and the speed of climbing gradients, two key prerequisites for efficient chemotaxis. Instead, we found both weak positive and weak negative correlations in mutant performance across environments. A molecular analysis suggested single components of the chemotactic network to be the subject of specific positive mutations reoccurring in different ecologies. Our results imply that, although ecology has an effect on systems evolution, the ecological constraints are overshadowed by mechanistic constraints. Potentially, these mechanistic constraints could contribute to the robustness of chemotactic networks to environmental changes.

Evolution of bacteriocin regulation by local cell density cues

Hilje Doekes
Theoretical Biology, Utrecht University

Many bacteria produce toxic molecules targeted at other bacteria, called bacteriocins. This is an example of spiteful behaviour: these cells invest metabolic resources to harm others. The production of most bacteriocins is tightly regulated. In particular, many bacteriocins are produced only if the local bacterial density is high. In these cases, bacteriocin is produced in response to local cell-density cues, which can be signalling molecules or other indicators of the presence of other cells. Although such regulation is frequently observed in nature, it is unclear which selection pressures shaped the evolution of these regulatory systems. We developed a computational model simulating the evolution of bacteriocin production and resistance, and the response to a local density cue. To study how fluctuations in cell density might select for regulation based on the cue, we exposed our simulated bacteria to two different growth regimes: (i) a regime with a fixed habitat in which the population evolves at its dynamic steady state with high population density, resulting in a long-term local competition experiment; and (ii) a serial transfer regime in which a small part of the population is repeatedly selected to colonise a new environment. Under both growth regimes, regulation of bacteriocin production readily evolves. However, the type of regulation found differs. In the local competition experiment cells use the regulation to optimise their competitive strength in response to small fluctuations in local cell density, while under the serial transfer regime they use the regulation to switch between a vulnerable but fast-growing colonisation-phenotype and an aggressive but slower-growing competition-phenotype. Next to providing insights on how bacteriocin regulation by cell-density cues could evolve, our results hence emphasise the important role of growth conditions in the evolution of (social) behaviours in bacteria.

Learning by example: How T cells learn to discriminate "self" from "foreign" during negative selection.

Inge Wortel
Department of Tumor Immunology, Radboud Institute for Molecular Life Sciences

Our immune system's ability to distinguish foreign from self was long thought to require silencing of self-reactive T cells during negative selection in the thymus. Yet recent data have shown that this removal is remarkably incomplete. This raises the question how such incomplete negative selection can nevertheless give rise to T cell repertoires that can discriminate between "self" and "foreign". We therefore devised an Artificial Immune System (AIS) to simulate incomplete negative selection in silico, on repertoires containing billions of T cells. Linking the problem of discriminating between self and foreign peptides to the analogous, more intuitive problem of language classification, we first applied our AIS to text in various languages to investigate under which conditions incomplete negative selection allows self-foreign discrimination. We then used this understanding to examine to what extent negative selection can support self-foreign discrimination by the immune system. We demonstrate that even incomplete negative selection can markedly improve self-foreign discrimination if T cells are moderately cross-reactive. This works especially well when "self" and "foreign" sequences are dissimilar - for example when comparing English text to text from the South-African language Xhosa. Even though similarities between self and foreign peptides hamper self-foreign discrimination in the immune system, we show that this discrimination remains possible - as long as the self peptides presented during negative selection strike a balance between abrogation of self-reactivity and preservation of foreign recognition. Importantly, our model predicts that preferential presentation of peptides with rare amino acids can achieve this balance. Our model explains how negative selection on an incomplete set of self peptides can bias a T cell repertoire towards foreign recognition. Importantly, this does not require the complete removal of self-reactive T cells. This notion reconciles the established negative selection theory with the finding that many self-reactive T cells survive this process.

Quantifying the effects of lateral and frontal visual fields on transmission of information in foraging groups

Fransje van Weerden
GELIFES / ALICE

Foraging individuals often live in groups. This improves their survival through passive risk dilution by sheer numbers and through increasingly more active pro-

cesses, from cue transmission to alarm calling. Already, a fleeing action in itself can give an involuntary visual cue to danger and by contagion this can incite others to flee as well, making these cues functional in anti-predator warning. Visual fields are limited both by morphology, which defines their size, as well as by behaviour. For instance, foraging with the head down can cause an extra blind angle in front, changing an unobstructed frontal visual field to a lateral shape. The questions of the present study are: how do visual fields in terms of their size and frontal versus lateral view influence survival through their effect on transmission of information about predators? We use an agent-based spatial simulation model to investigate the effect of detection and of contagious fleeing on survival rate, for different sizes of the visual field, lateral vs. frontal view, different group sizes and movement rates and styles. We devise a measure for the transmission rates, namely the ratio of the number of others that an individual has actually seen divided by the number of others potentially seen, and we measure the length of transmission chains. We find that in a stationary group, a larger visual field leads to a higher survival rate. Moreover, a lateral view is more effective by increasing indirect reception of visual cues about predators for all but the largest visual fields, which is evident from higher transmission ratios and longer chains of transmission. As long as the visual field is large enough, having a blind angle in front does not detract from optimal transmission. These findings are ecologically relevant to observations of vigilance in groups of foraging animals.

How Immune Cells Find Each Other; In Silico Model of Fibroblastic Reticular Network Structure and Morphology

Shabaz Sultan
Tumor Immunology Lab, Radboud Institute for Molecular Life Sciences

As part of the adaptive immune system, dendritic cells (DCs) take up and process antigens, which they then present to T cells to possibly induce an immune response. For this process to function properly, DCs need to make physical contacts with T-cells. One of the areas where this happens is the paracortical region of lymph nodes. Within such areas, both DCs and T cells have been observed to move on a stromal network structure, the fibroblastic reticular cell (FRC) network. It is tempting to speculate that the structure of this network must somehow increase the efficiency of cell movement and the rate of cell-cell encounters. To test such hypotheses, it would be useful to be able to change the FRC network structure and observe the effect of such changes on T cell/DC crosstalk. However, that is difficult to achieve experimentally. Instead, we aim to build an in silico model of FRC network growth and simulate the impact of targeted adjustments to network structure on cellular behaviour. As current

imaging techniques are unable to capture entire FRC networks in lymph nodes, we are designing a generative model that captures actual morphology and structure of real FRC networks. Similar generative models have been proposed in the literature, but these either do not attempt to match empirical data on real network structure or merely extract abstract topological attributes. So far, no model has tried to capture both the spatial nature of the network and its specific morphological features. We propose a new in silico model of network growth, and use detailed confocal microscopy data of network slices to verify that our model accurately captures these essential aspects of network morphology. We will continue this work by combining our in silico network model with a simulation of T cell migration on the network using the cellular Potts modelling framework.

Friday April 6th

Unfolding the complexity of a three-part brain

Sander van Doorn
University of Groningen

The majority of motile bacteria are capable of using chemical cues to actively direct their motion towards favourable areas in their environment. This adaptive chemotactic behaviour is mediated by a small signal-transduction network that processes information from membrane chemoreceptors and that controls the direction of rotation of the flagellar motor complexes. The bacterial chemotaxis network has been characterised in extraordinary detail, and has emerged as a model system for studying the molecular basis of individual behaviour. I here consider this system as a minimal example of a complex trait, where selection acts on properties that emerge from interactions between molecular components. I will present a computational model that reconstructs the mapping from genotype to phenotype to fitness for this bio-molecular network, and use the model to study how the chemotactic network evolves to compensate for knockout mutations, or to optimise performance in different environments. Analysis of the reconstructed fitness landscapes underscores the pervasiveness of epistasis in complex trait evolution and provides novel clues to understanding the nature of evolutionary innovation.

Critical chronicity: acute and chronic infections emerge in a single within-host model due to resource modulation by parasites

Anieke van Leeuwen
Department of Coastal Systems, NIOZ Royal Netherlands Institute for Sea Research

Over a billion people on earth are infected with helminth parasites and show remarkable variation in parasite burden and chronicity. Although classic statistics such as the negative binomial distribution phenomenologically capture these population level patterns well, our understanding of the within-host processes giving rise to these patterns is limited. Here, to explain variability in infection outcome as an emergent property of within-host processes, we account for energy flows between parasites, immunity, and metabolic processes using Dynamic Energy Budget theory. Manipulation of within-host resources by parasites gives rise to a positive feedback where parasites improve their own resource availability inside the host, which in turn improves parasite growth and subsequent monopolization of resources. This results in bistability between an acute and a chronic infection state. We show that resource-dependence is crucial to explain patterns in natural variation in chronicity that are difficult to explain based on immune phenotype alone.

The evolution of "wild-types" of Virtual Microbes and adaptation to the long-term evolution experiment

Bram van Dijk
Utrecht University

Bacteria and other microbes have coped with harshly fluctuating and unfavorable environments throughout billions of years of evolution. A commonly observed strategy under nutrient starvation is to divert resources away from growth, allowing the cells to survive until conditions improve. Here we investigate the evolution of "wild-type" Virtual Microbes that learn to cope with harsh resource conditions. We show that besides rates of growth, survival strategies are important evolved features of these wild-types. Many of these wild-types evolve regulatory response analogous to the stringent response of Escherichia coli, sensing the quality of the environment and suppressing protein expression when nutrients are limiting. These "wildtypes" are next subjected to the Long-Term Evolutionary Experiment (LTEE), to investigate what selection pressures might be relevant for an evolved, complex microbe, when evolving to a more predictable protocol.

Mathematical modelling of radiation-induced DNA damage repair and carcinogenesis

Sjors Stouten
Centre for safety (RIVM) & Mathematical institute (UU)

We are continuously being exposed to very low doses of radiation coming from a wide variety of sources such as food, soil, buildings, space and medical applications. The linear no-threshold (LNT) model has been developed to quantify possible long-term health effects after ionizing radiation exposure. The model assumes that the risk of developing long-term health effects is proportional to dose. It allows one to sum separate exposure events and determine whether the obtained risk of developing a disease is tolerable within the context of writing health and safety policies concerning occupational hazards. The scientific community is divided regarding the validity of the LNT model when applied to low dose/dose-rate exposure events. Due to technical limitations and ethical considerations it is impossible to directly measure the effects of low dose/dose-rate ionizing radiation exposure on long-term health effects such as cancer induction. In absence of conclusive experimental data, linear extrapolations made from the LNT model remains the default method utilized for estimating low dose cancer risks. In order to assess the validity of the LNT model we are currently developing mathematical models of (i) radiation-induced DNA damage repair and

(ii) murine bone marrow stem cell differentiation in relation to leukaemia onset. We are currently able to quantify radiation-induced DNA damage in terms of dose, dose-rate and radiation quality (photons/particles). This information is the foundation of our bone marrow population model in which a fraction of exposed cells accumulates chromosome aberrations that are critical for the onset of leukaemia. We will apply these models to murine radiation-induced acute myeloid leukaemia datasets. This allows us to compare our model-derived leukaemia risks to values that one might expect from LNT model extrapolations. During my presentation I will show our preliminary results and discuss possible consequences for writing health and safety policies.

Evolution of lysogeny and bacteriophage communication.

Glenn Mulder
Theoretical Biology & Bioinformatics, Utrecht University

Upon infection of a new host cell, bacterial viruses generally enter a lytic life-cycle to kill host cells and produce viral particles. Temperate viruses, alternatively, can integrate their genetic material into the host genome and become lysogenic. Last year, it was found that certain temperate bacteriophages use a communication mechanism that influences this lytic-lysogenic life-style decision (Erez et al., 2017). During the latent phase, these phages induce the secretion of a small peptide and as the concentration of this peptide rises in the medium, so does their propensity for lysogeny. The authors proposed that such a communicative decision-making mechanism provides individual phages with information on the number of infections that are going on in their vicinity. This number of infections gives an indication of the availability of susceptible host cells and hence of the expected viability of a lytic life-cycle. To test the validity of this idea, we develop an ordinary differential equation model that describes growth and infection of communicating and non-communicating bacteriophages, coexisting in a sensitive population. We then examine the competitive capacity of these communicating phages compared to that of non-communicating ones. As the number of infections needs to vary for communication to be effective, we include fluctuations in the number of susceptible cells in our model. We then introduce a communicating phage and examine the evolution of the peptide concentration threshold at which the bacteriophage changes its strategy. In the absence of communication, three distinct strategies can evolve depending on the frequency of transfer to a new sensitive population. A fully lytic strategy is selected for when transfers occur frequently, a fully lysogenic strategy is selected for when transfer events are rare and a bet-hedging strategy is selected for in an intermediary region. In addition, we find that the communication system allows phages to efficiently switch between a fast-spreading lytic strategy during ini-

tial infection of a sensitive population, and a more long-lived lysogenic strategy when the availability of sensitive cells decreases. Indeed, communication allows for an adjusted life-cycle decision, such that communicating phages outcompete non-communicating phages in both the bet-hedging and fully lysogenic regime. From this, we conclude that communication indeed confers a competitive advantage. References: Erez, Zohar et al. "Communication between viruses guides lysislysogeny decisions". In: Nature 541.7638 (January 2017), pp. 488493.

Biological organization three decades after Ernst Mayr

Gerard Jagers op Akkerhuis
Philosophy Wageningen

In 1988 Ernst Mayr wrote: "The complexity of living systems consists at every hierarchical level, from the nucleus, to the cell, to any organ system (kidney, liver, brain), to the individual, to the species, the ecosystem, the society." Mayr added that "The hierarchical structure within an individual organism arises from the fact that the entities at one level are compounded into new entities at the next higher level - cells into tissues, tissues into organs, and organs into functional systems." Even nowadays, many biologists experience no problems when reading Mayr's phrases. After all, isn't this how nature is organized? Of course there is such a hierarchy! Of course individual organisms arise through integration from cells, to tissues, to organs, tot a functional system! But are things really the way Mayr suggests? Has Mayr paid sufficient attention to the logical consistency of his ranking of "hierarchical levels"? Are (multicellular) organisms really constructed the way he sketches? In my talk I will submit Mayr's thoughts to a critical philosophical analysis. To resolve logical inconsistencies in Mayr's perspective, a new approach will be presented that suggests fundamental changes in the way biologists currently analyze systems.

Combining gene trees into a phylogenetic network.

Remie Janssen
TU Delft

This talk is mostly based on the paper 'Polynomial-Time Algorithms for Phylogenetic Inference Problems' available on ArXiv (number 1802.00317). In this paper we study the problem of infering a species phylogeny (network or tree) from a set of gene trees. There are several models restricting the embedding of the gene trees in different ways. We give a polynomial time algorithm for phylogeny inference for two such models that turn out to have the 'same' solution. In this talk I will show some results on the mathematical structure of

these solutions. Furthermore, I will discuss the use of these models in biology.

Linking life history theory, population genetics and population ecology using evolutionary demography: a matrix population model approach.

Charlotte de Vries
University of Amsterdam

Evolutionary change and population dynamics are linked through the birth and death processes that drive them both. Demography is therefore central to understanding evolution and a truly eco-evolutionary framework must provide a map from genotype to phenotype, from phenotype to demographic processes, and from demography to (stage)x(genotype) dynamics (fitness, in a general sense). In this talk, we present a new model framework which incorporates basic Mendelian genetics into the powerful demographic framework of matrix population models. Any kind of ecological process can be included in the demographic component of the model: age- or stage-classified life histories of arbitrary complexity, linear or non-linear (density-dependent) dynamics, constant or time-varying (periodic or stochastic) environments. In addition, the model can incorporate genes that differentially affect males and females, and hence describes the evolution of sexually dimorphic traits. We show that in the presence of sexual dimorphism in demographic rates, the population growth rate can not be used as a proxy for fitness. As a consequence, average fitness in the population does not always increase and populations can go extinct due to evolutionary suicide. We calculate the stability of the homozygote equilibria to invasion by the other allele to derive analytical genotype coexistence conditions. As an example, we present results on the maintenance of a colour polymorphism in the common buzzard (Buteo buteo).

NVTB jaarvergadering 2018

The agenda of the annual meeting on Thursday 5th of April 2018, to commence at 16:15, is as follows:

- Opening
- Annual report secretary
- Annual report treasurer
- Voting for the new chair of the NVTB
- Voting for the new treasurer of the NVTB
- Report treasury committee 2017
  Approved by Jaap Rutten (present)
  Approved by Aline de Koeijer (not present)
- Budget 2018
- Applications for symposia grants for 2018
- Evaluation of the meeting schedule 2018
- Ideas for invited speakers 2019
- Date NVTB meeting 2019
- Survey
- Concluding remarks

Annual report secretary 2017

General information:
Last year, the meeting of the NVTB took place on June 1st and 2nd, 2017. The keynote speakers were Willem Bouten and Frank Bruggeman. In total, we got 16 new members in 2017, whereas 11 members cancelled their membership. 5 other members have been expulsed due to inactivity. This leaves the total number of members at the end of 2017 exactly where it was last year (195).

A new chair and treasurer:
This year, we have 2 people leaving the NVTB board. Jaap Kaandorp (UvA) has been the NVTB chair for 3 years, and now puts forwards Kirsten ten Tusscher (UU) as the next chair. Lotte de Vries (UvA) will also quit. Jeroen Meijer (UU) has volunteered to take on the job of being treasurer of the NVTB. Votes will pass on these appointments during the general meeting.

Kerkzaal:
Last year we had some complaints about the presentations being poorly visible due to the sunlight in the presentation room. The white curtains were not very effective to keep the light out, leaving much to be desired. We raised a complaint at Dopersduin, and they were kind enough to offer us to host the meeting in the "Kerkzaal". They showed us the room, and the light seems much easier to manage (and a lot less light comes in to begin with). I ask the members to pay attention to the pros and cons of using this new room, so I can make an informed decision on improving the meeting next year.

Website use:
Since last year we have our own website. It is now only used to announce the NVTB meeting and some events organised by Theoretical Biology at Utrecht University. If you or any of your colleagues ever feels the need to organise an event and needs an online platform to do so, you can use our website. Also, if you find any other issues with the website, please report them to me via the contact form on the website[2] or email me directly.

[2]http://theoreticalbiology.nl/contact.php

Report of the treasurer of the NVTB on the financial year 2017

2017

In 2017, our society achieved a positive result of €2,635.46. This is due to a couple of factors: no requests for funding of symposia were received in 2017 and we scrapped Bram Kuypers symposium from our financial records (€500, as was discussed during the annual meeting in 2017).

Schoorl 2017
The costs of the Schoorl meeting (€4,153) were partly covered by the participants (€2,500.10) and partly covered by the NWO subsidy (€1,760). We had a net positive result for Schoorl (€107.10) due to the raised Schoorl fees and thanks to high attendance of the Schoorl meeting.

Other costs and benefits
The costs for the bank account and the transfers were €123.64. We received €12.72 interest on the savings account. As was discussed during the 2016 meeting, we bought some serverspace to host our website, in 2017 we paid €96.72 for the serverspace.

2018

Schoorl 2018
Whether Schoorl will give a net negative or net positive result depends mostly on the number of participants. In 2016 we had a small negative result (-€107.25) and in 2017 we had a positive result of the same size (+€107.10). The net result over the whole financial year will mostly depend on whether any money is requested for symposia but a positive result is expected.

Symposia 2018
For 2018, we have again budgeted a total of €1500 for symposia, this amount is to be divided over the number of symposia this year. The maximum amount per symposium is €500. Requests can be submitted at the annual meeting or via email to the treasurer beforehand. No money for symposia has been requested for a few years, maybe we need to advertise this more strongly.

Balans 1-1-2018

Activa (€)		Passiva (€)	
tegoed contributie 2010	10.00	vermogen 1-1-2017	14.372.98
tegoed contributie 2011	50.00		
tegoed contributie 2012	84.00	positief resultaat 2017	2.635.46
tegoed contributie 2013	136.00	vermogen 31-12-2017	17.008.44
tegoed contributie 2014	240.00		
tegoed contributie 2015	384.00		
tegoed contributie 2016	516.00		
tegoed contributie 2017	1.056.00	Vooruit betaalde contributie 2018	60.00
voorschot Schoorl voor 2018	2.821.40		
spaarrekening	5.633.71	reservering Ramiro Magno	500.00
in kas	6.637.84		
Totaal	17.568.95	totaal	17.568.44

Balans 1-1-2017

Activa (€)		Passiva (€)	
tegoed contributie 2010	10.00	vermogen 1-1-2016	13.003.81
tegoed contributie 2011	60.00		
tegoed contributie 2012	96.00	positief resultaat 2016	1.369.17
tegoed contributie 2013	148.00	vermogen 31-12-2016	14.372.98
tegoed contributie 2014	276.00		
tegoed contributie 2015	464.00		
tegoed contributie 2016	708.00		
		vooruit betaalde contributie c. hemelrijk en f. weissing	24.00
voorschot Schoorl voor 2017	0.00	Reservering poster prijs 2017	100.00
spaarrekening	5.620.99	reservering Ramiro Magno	500.00
in kas	8.114.05	reservering workshop Bram Kuijper	500.00
Totaal	15.497.04	Totaal	15.496.98

Resultatenrekening 2017

Kosten (€)		Opbrengsten (€)	
Schoorl 2017	4.153.00	bijdrage NWO Schoorl 2017	1.760.00
Kosten website	96.72	Bijdragen leden Schoorl 2017 *	2.500.10
Poster prijs 2017	100.00		
kosten ING (betaald in 2017)	123.64	rente over 2017 (in 2017 gestort)	12.72
onbetaald 2011 agv opzegging 2017	10.00	Contributie 2009-2017**	102.00
onbetaald 2012 agv opzegging 2017	12.00	verwachte contributie 2017	2.424.00
onbetaald 2013 agv opzegging 2017	12.00	giften	0.00
onbetaald 2014 agv opzegging 2017	12.00	Reservering Bram Kuyper geschrapt	500.00
onbetaald 2015 agv opzegging 2017	36.00		
onbetaald 2016 agv opzegging 2017	24.00		
onbetaald 2017 agv opzegging 2017	84.00		
Totaal	4.663.36	Totaal	7.298.82
		Positief resultaat 2017	2.635.46

* bijdrage leden schoorl is na aftrek van kosten voor wijn voor sprekers en kosten naam stickers
** Een van onze leden betaalde in 2017 alsnog contributie van 2009-2017, nadat we hem al geroyeerd hadden vorige jaar. Hierdoor is er een verschil ontstaan in de contributie te goeden van 2017 en 2016, omdat zijn contributie al was afgeschreven als verloren in 2016.

Begroting 2018

Kosten (€)		Opbrengsten (€)	
Uitgaven Schoorl 2018	4.500.00	bijdrage NWO Schoorl 2017	2.000.00
Posterprijs 2018	100.00	bijdrage leden aan Schoorl 2017	2.500.00
symposia	1.500.00	contributie 2017	2.200.00
geroyeerde leden	200.00	rente	10.00
kosten ING (incl. internetbankieren)	120.00		
kosten website	100.00		
Totaal	6.520.00	Totaal	6.710.00
		Verwacht resultaat 2018	190.00

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