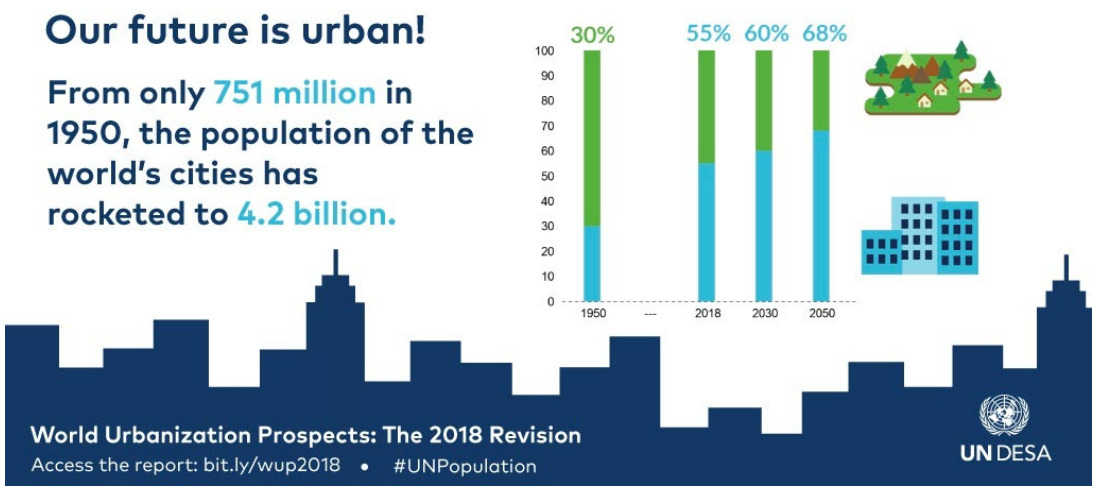
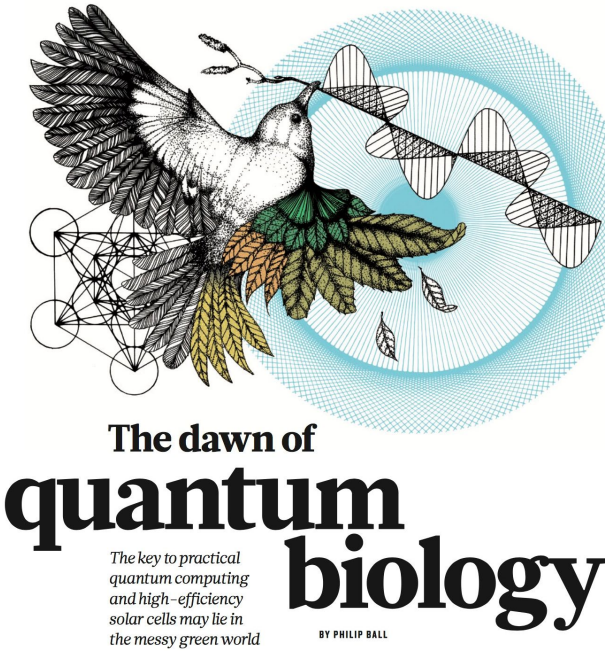
Complex systems from the largest to the lowest scales of organization





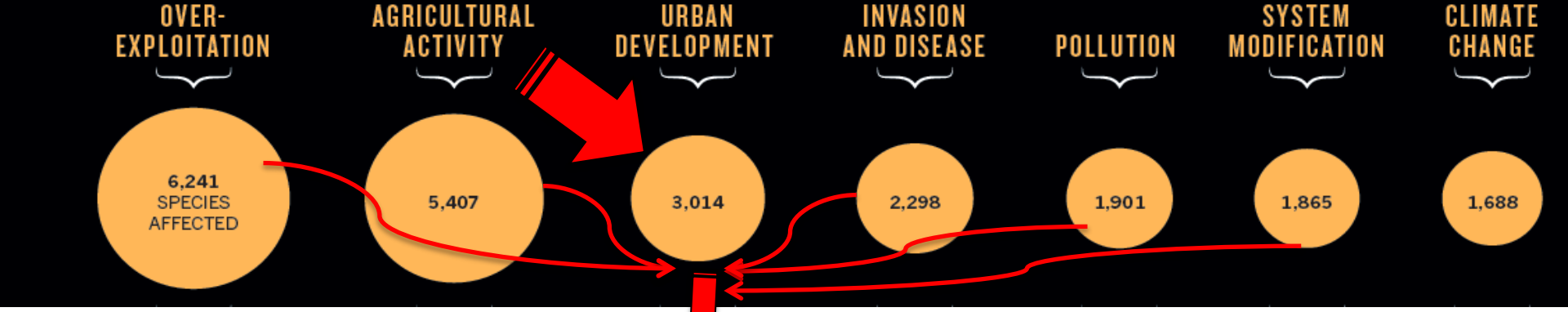
Diego Santiago-Alarcon

Department of Integrative Biology

UNIVERSITY OF SOUTH FLORIDA
COLLEGE OF ARTS & SCIENCES

Outline

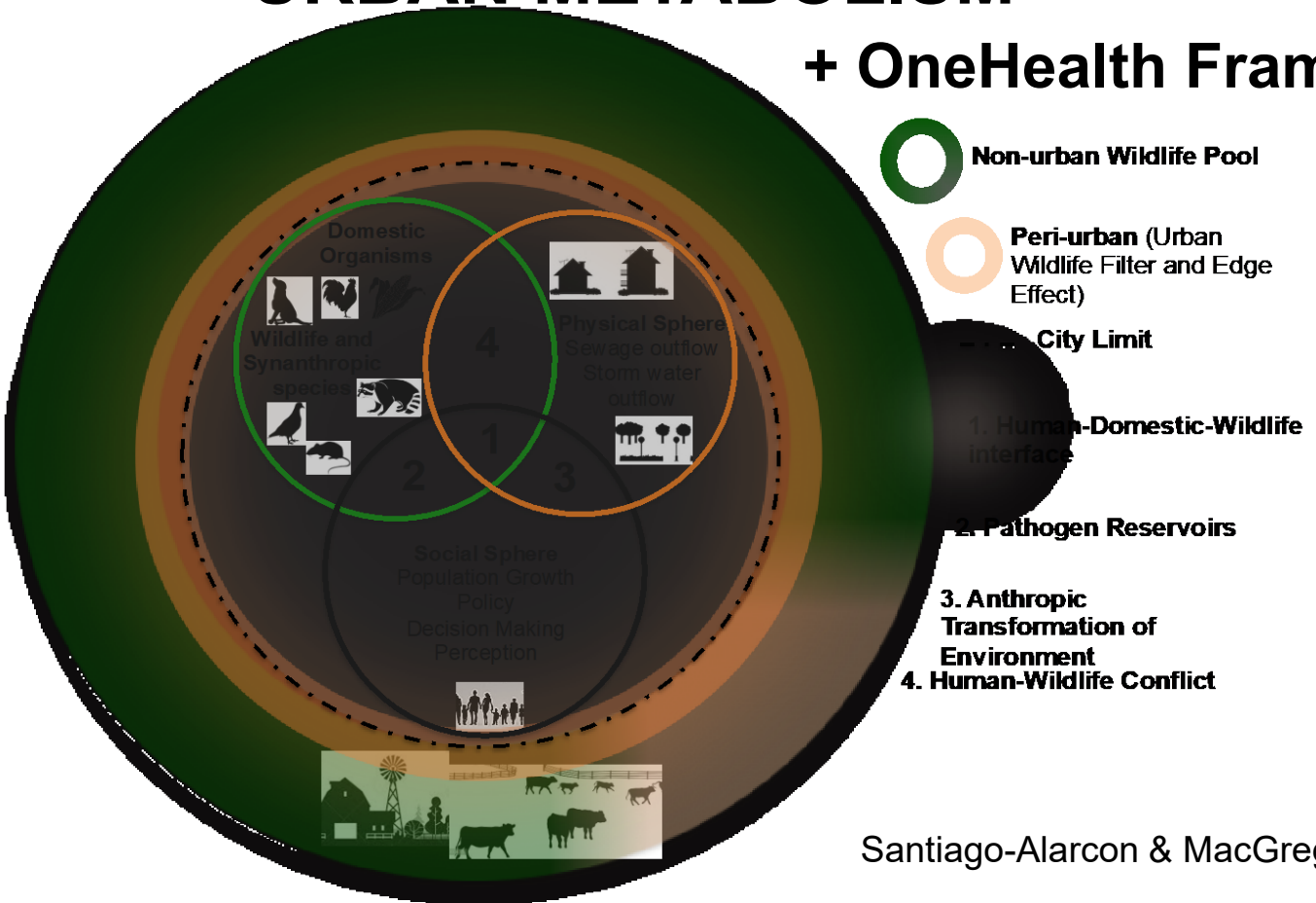
- Disease Ecology and The Urban
- Avian Malaria and Related Parasites Life Cycle
- Global Disease Ecology
- Down to the Quantum
- How we tight up the small to the big



Maxwell et al. (2016) Nature

URBAN METABOLISM

+ OneHealth Framework

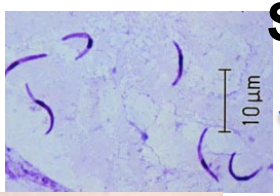


Santiago-Alarcon & MacGregor-Fors (in press)

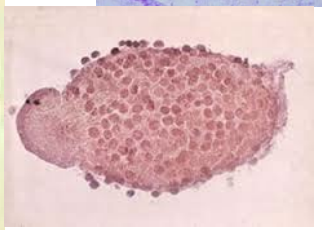
Haemosporidian parasites: life cycle

Exflagelation
Fertilization

Ookynete

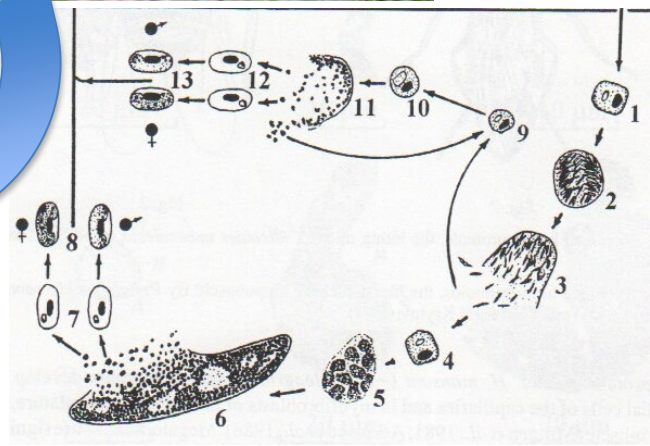


Sporozoites

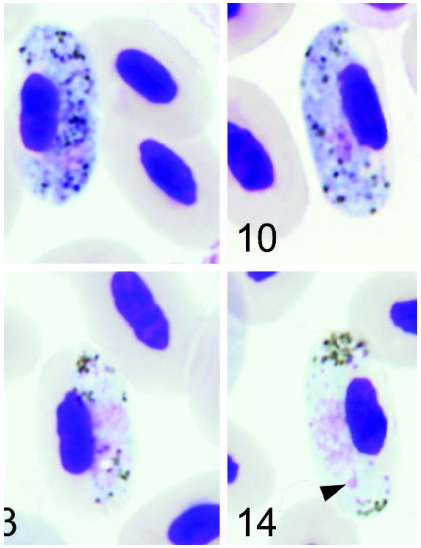


Oocysts

Intermediate
Host



Gametocytes



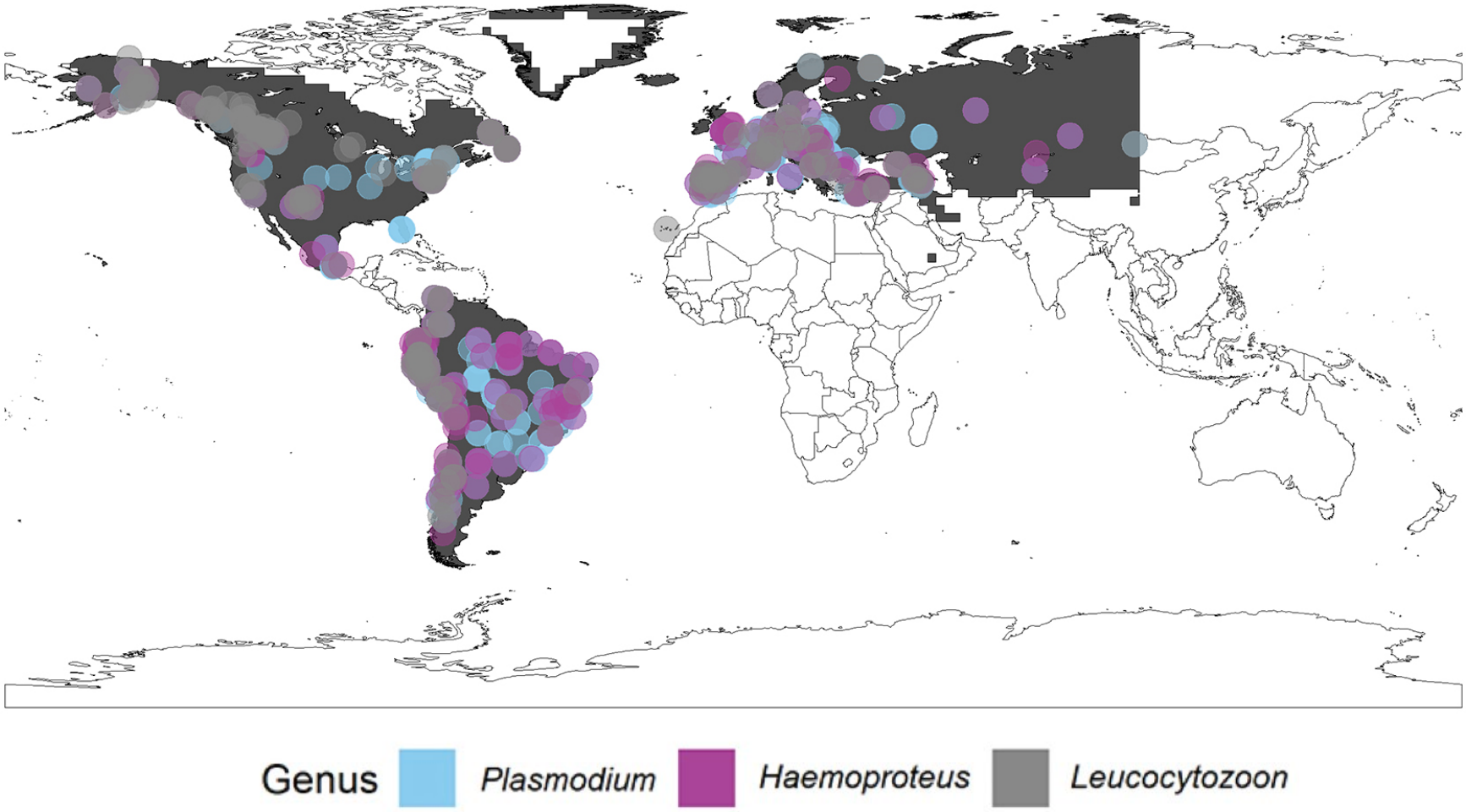
Definitive
Host

GLOBAL SCALE DISEASE ECOLOGY

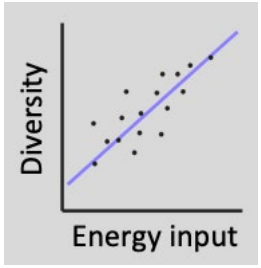
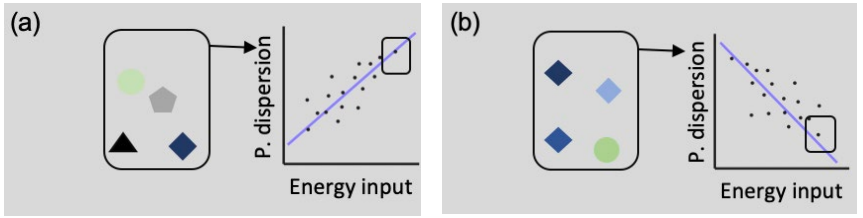
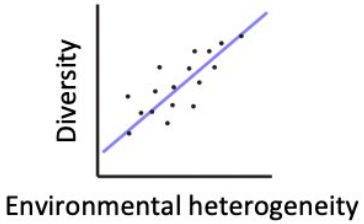
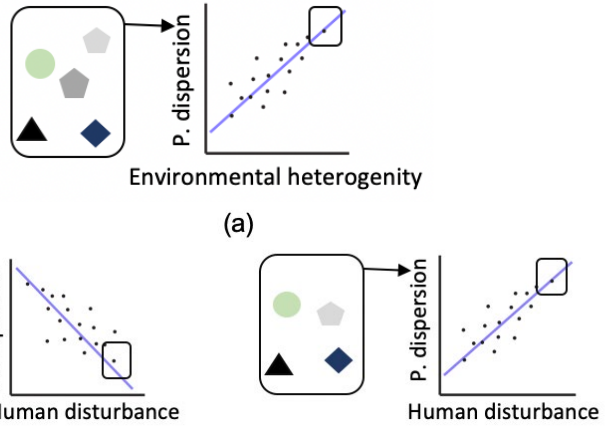
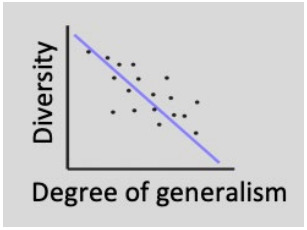
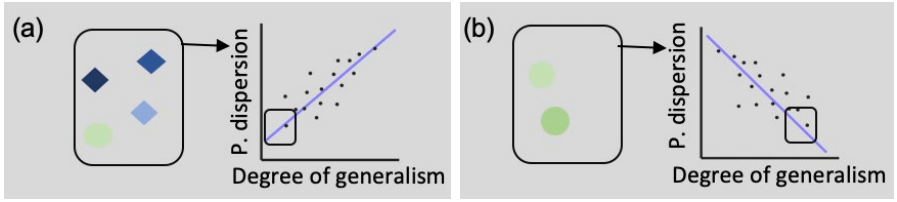
Question: how do energy input, habitat heterogeneity, and host breadth affect the diversity of parasite assemblages?

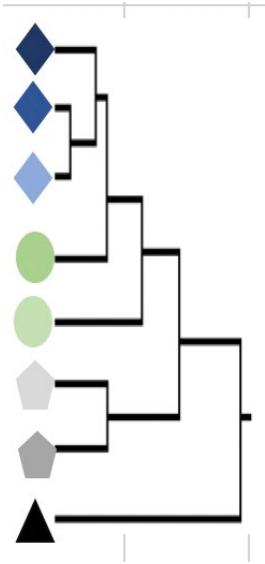
Data for three parasite genera

(a)

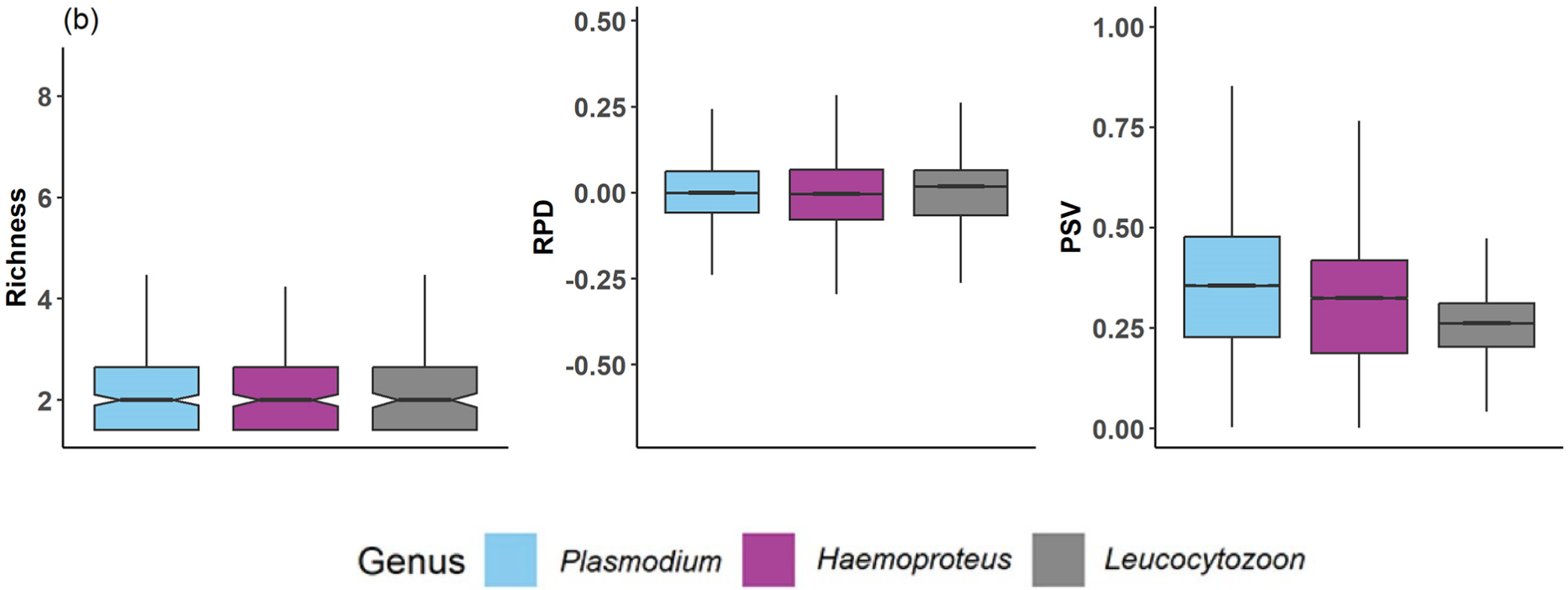


Predictions

Type of predictors	Taxonomic and phylogenetic diversity	Phylogenetic structure
Energy input • Temperature, precipitation, potential evapotranspiration		
Habitat heterogeneity • Climatic heterogeneity (Climatic Seasonality) • Environmental heterogeneity (Vegetation density, Ecosystem heterogeneity) • Internal habitat heterogeneity (Host richness) • Biotic homogenization (Human footprint and population density)		
Ecological interactions • Degree of generalism		

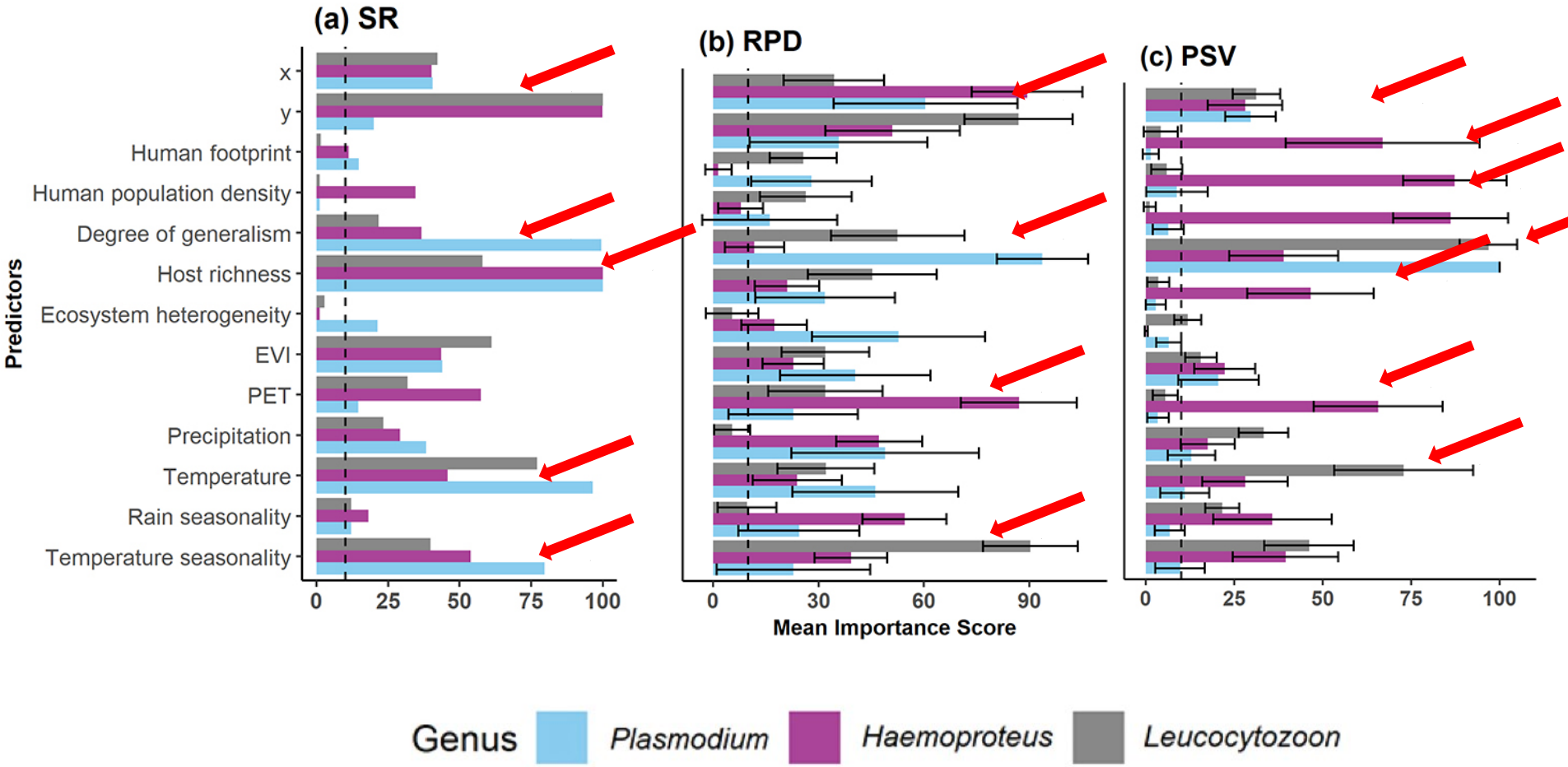


- **Richness = taxonomic richness**
- **RPD = phylogenetic richness**
- **PSV = phylogenetic relatedness of parasite assemblage**

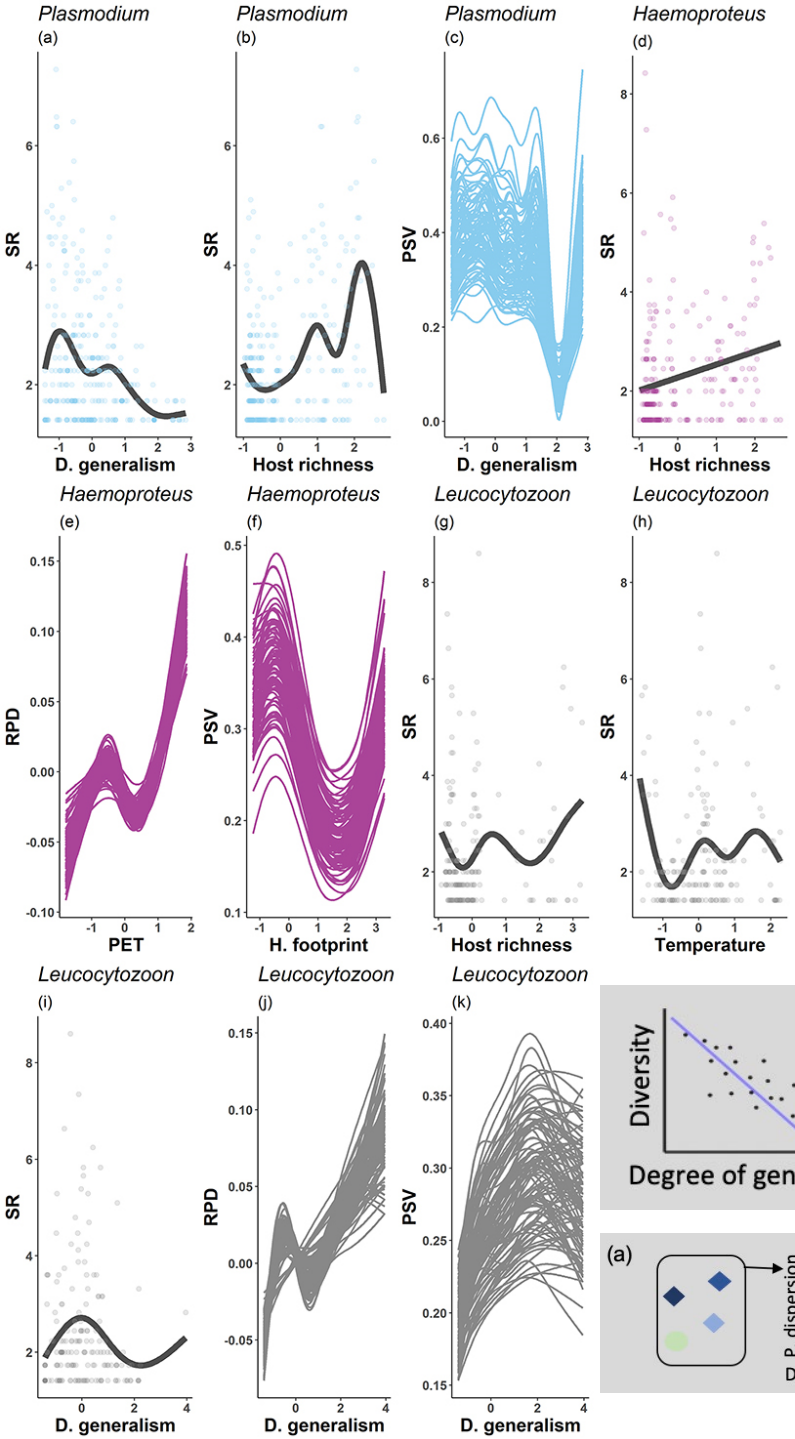


Hernandes Córdoba et al. unpublished

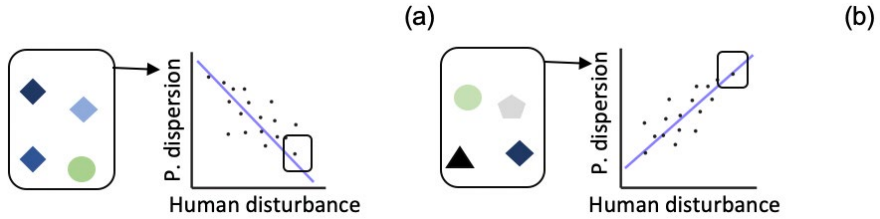
Importance of Variables on Parasite Diversity

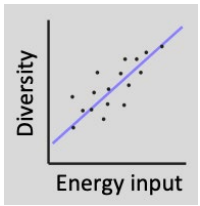


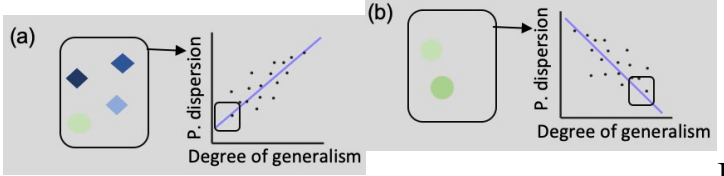
Hernandes Córdoba et al. unpublished



- **SR = taxonomic richness**
- **RPD = phylogenetic richness**
- **PSV = phylogenetic relatedness of parasite assemblage**







Hernandes Córdoba et al. unpublished

QUANTUM BIOLOGY

Question: how can quantum tools help us to understand why the genome works in a non-random way?

The Challenge

Journal of Heredity 2009:100(5):637–647
doi:10.1093/jhered/esp048
Advance Access publication July 22, 2009

Climbing Mount Probable: Mutation as a Cause of Nonrandomness in Evolution

Arlin Stoltzfus and Lev Y. Yampolsky

Mutational Biases Influence Parallel Adaptation

Arlin Stoltzfus*,[1] and David M. McCandlish[2]

Mol. Biol. Evol. 34(9):2163–2172 doi:10.1093/molbev/msx180

Article

Mutation bias reflects natural selection in *Arabidopsis thaliana*

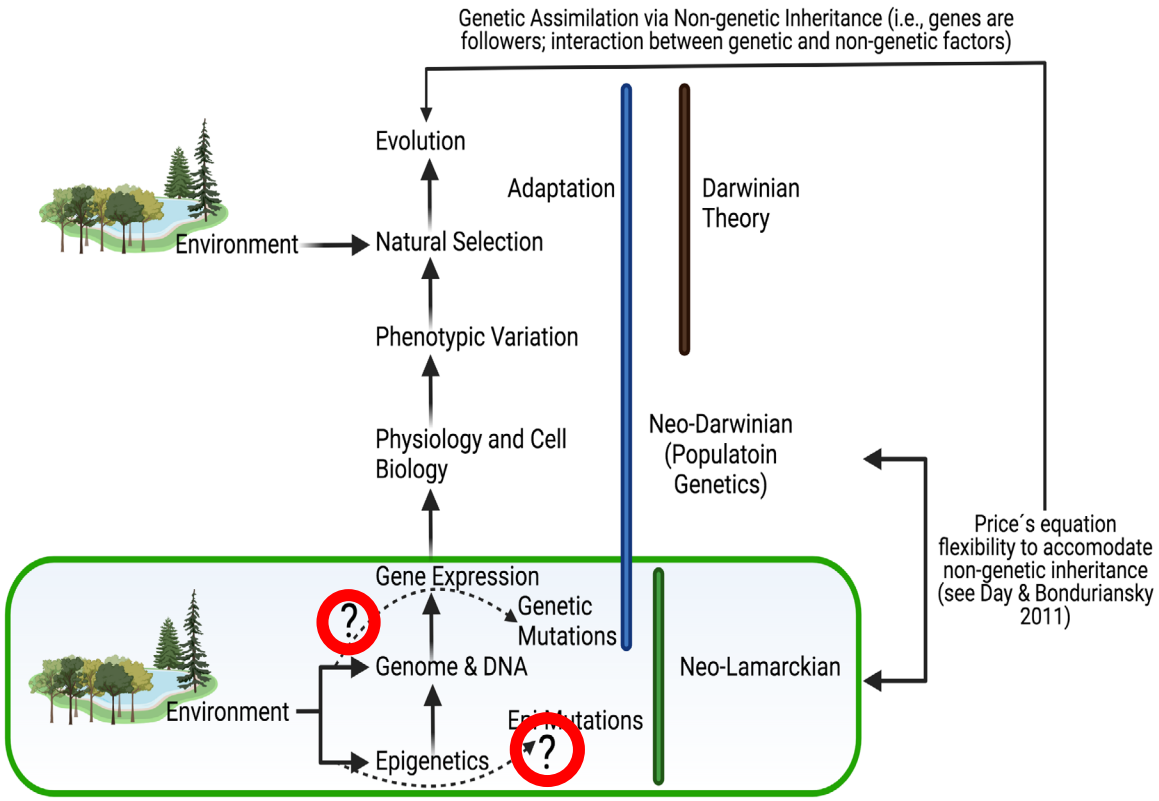
Nature | www.nature.com | 1

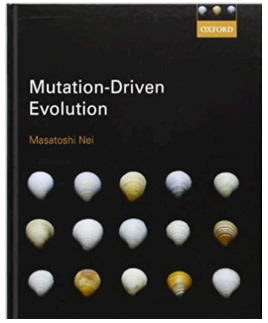
https://doi.org/10.1038/s41586-021-04269-6
Received: 9 November 2020
Accepted: 17 November 2021

J. Grey Monroe[1,2]✉, Thanvi Srikant[1], Pablo Carbonell-Bejerano[1], Claude Becker[1,10], Mariele Lensink[2], Moises Exposito-Alonso[3,4], Marie Klein[1,2], Julia Hildebrandt[1], Manuela Neumann[1], Daniel Kliebenstein[2], Mao-Lun Weng[5], Eric Imbert[6], Jon Ågren[7], Matthew T. Rutter[8], Charles B. Fenster[9] & Detlef Weigel[1]✉

Why this is a challenge?

There is no theory explaining why this nonrandom mutations occur!





"…when it comes to understanding the origins of novelty in evoution…selection cannot select for traits that do not yet exist, accidents can only sort among preexisting variation, and contraints only limit options, but by themselve do not create new ones." (Moczek 2019).

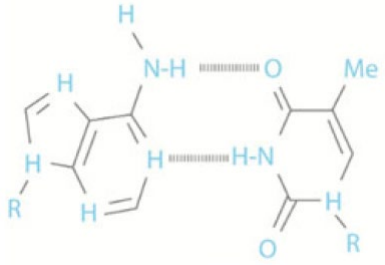
We know the components of the machine and how they interact and what they do, but do we really know why there are nonrandom mutations?

Quantum physics and mutations

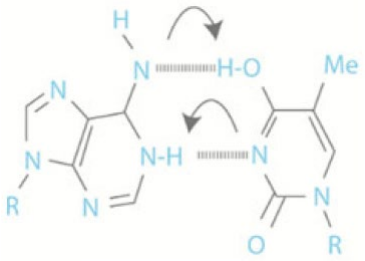
A quantum-theoretical approach to the phenomenon of directed mutations in bacteria (hypothesis)

BioSystems 43 (1997) 83–95

Vasily V. Ogryzko



A–T base pair

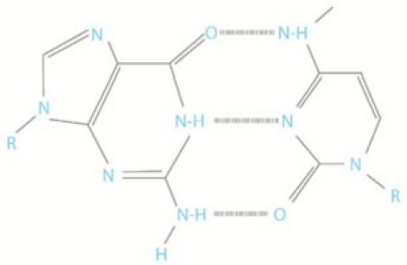


Quantum Genes:
proton superposition

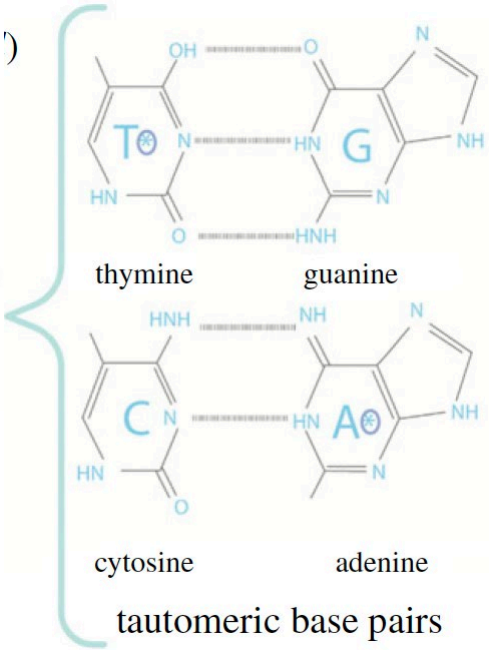
A quantum mechanical model of adaptive mutation

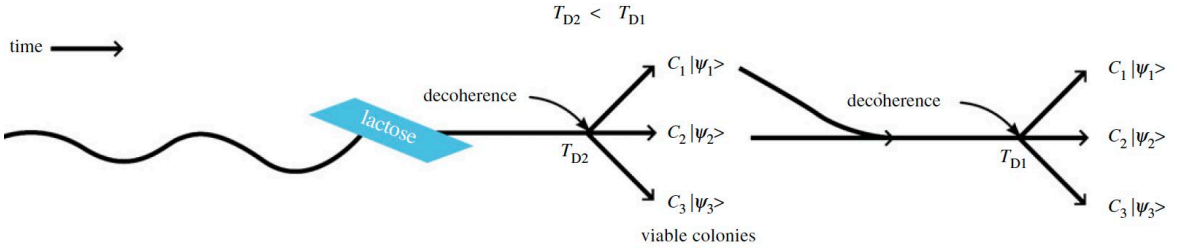
BioSystems 50 (1999) 203–211

Johnjoe McFadden [a,*], Jim Al-Khalili [b]



G–C base pair

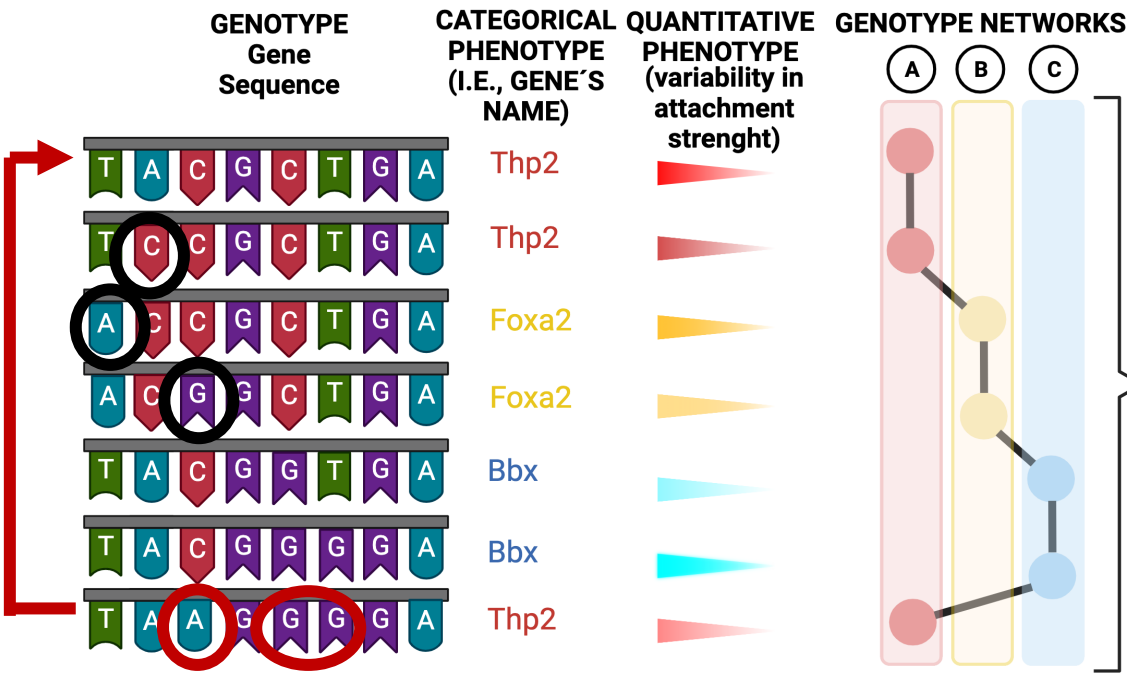




Quantum Superposition, but how does it work in evolution?

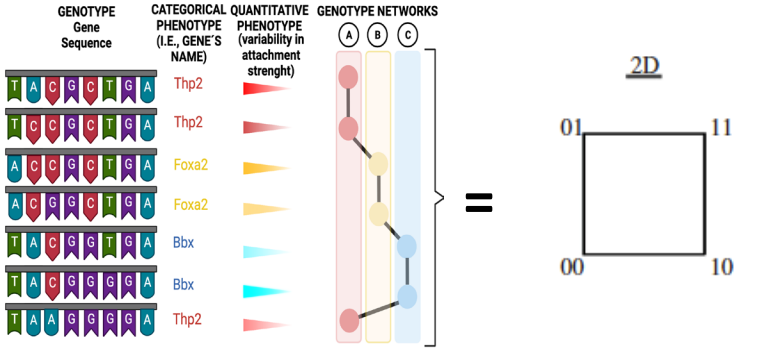
Genotype Networks (*sensu* Wagner 2011)

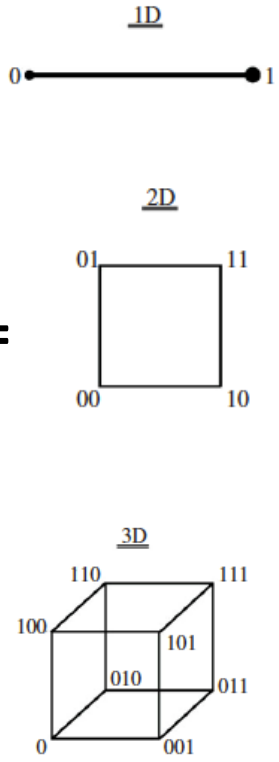
The ADN is digital, **THEN:** How do we codify the digital DNA in evolutionary terms? – Genotype Networks

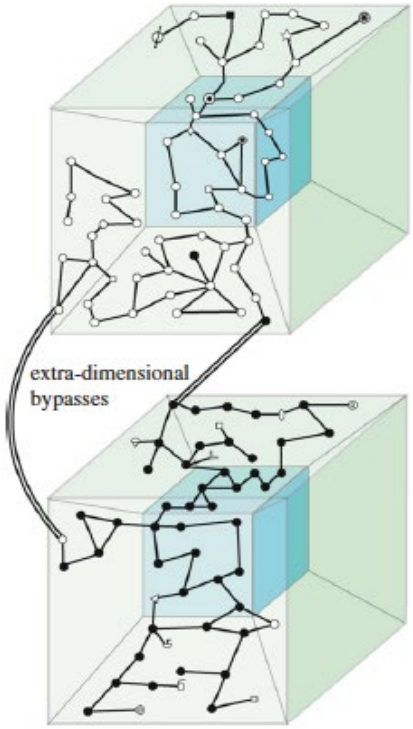


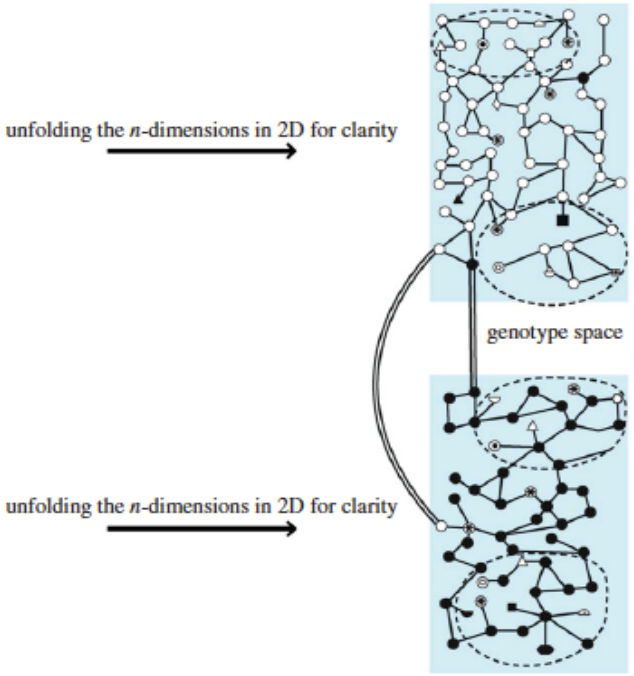
Genotype networks have been explored via classical random walks by Wagner et al. see: https://www.ieu.uzh.ch/wagner/publications.html

Genotype Networks are N-dimensional (*sensu* Wagner 2011)





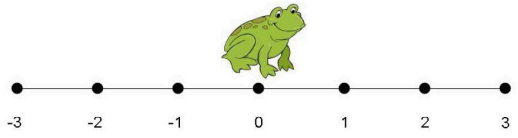




Santiago-Alarcon et al. 2020

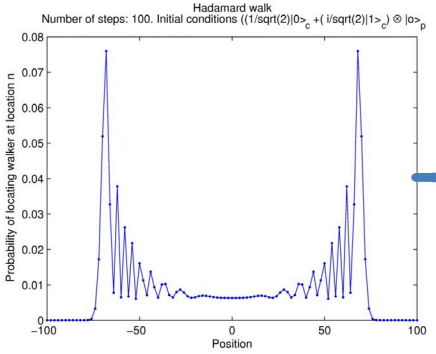
We have the mechanism and the foundation of evolutionary space
Now, how do we implement the superposition?: Random Walks vs. Quantum Walks

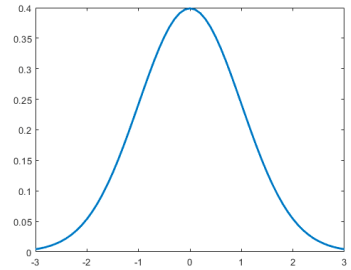
Clasical Random Walks (CRW)



Froggy jumps either forward or backwards, depending on the outcome of corresponding coin toss, heads or tails respectively.

Let us suppose that Mr. Money has a coin with probability p of getting heads and probability q of getting tails.
If Froggy begins its journey in position zero, what is the probability of finding our dear frog at position k after n steps?



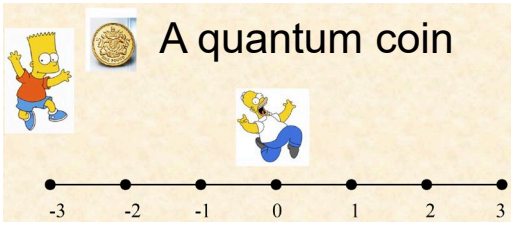


Binomial Distribution with 100 steps

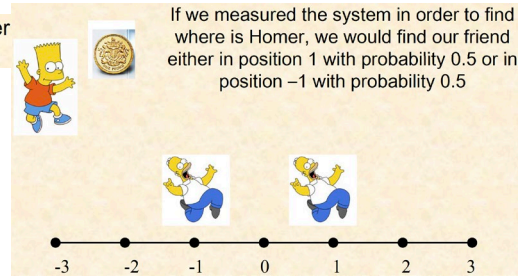
$$P(x)=\binom{n}{x}p^x q^{n-x}=\frac{n!}{(n-x)!x!}p^x q^{n-x}$$

Quantum Walks (QW)

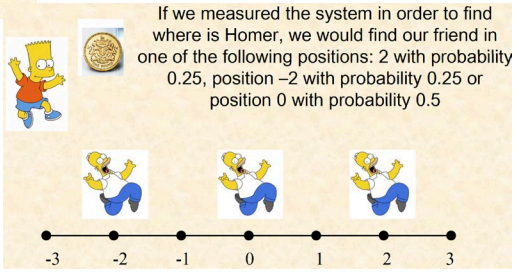
Step 1. Before coin toss, Homer is in position 0



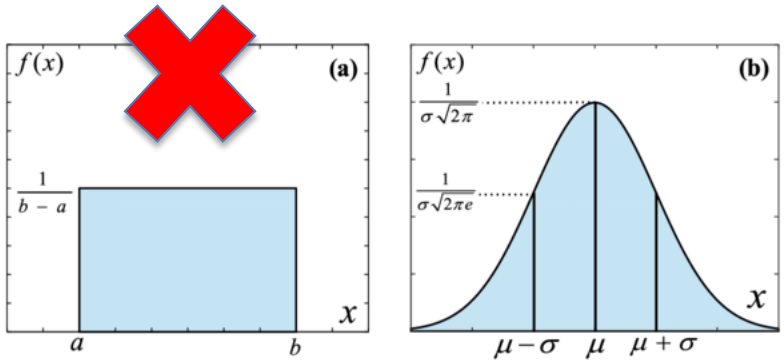
Step 2. After the first coin toss, Homer is in position 1 and -1



Step 3. After the second coin toss, Homer is in positions 2, 0 and -2



TO CLARIFY!



We have the mechanism and the foundation of evolutionary space
Now, how do we implement the superposition?: Random Walks vs. Quantum Walks

Our model for closed systems:

We put together genotype networks + quantum walks

INTERFACE

royalsocietypublishing.org/journal/rsif

Research

Quantum aspects of evolution: a contribution towards evolutionary explorations of genotype networks via quantum walks

Diego Santiago-Alarcon[1], Horacio Tapia-McClung[2], Sergio Lerma-Hernández[3] and Salvador E. Venegas-Andraca[4]

We improved our quantum walk

Quantum Information Processing (2023) 22:224
https://doi.org/10.1007/s11128-023-03972-9

A new definition of hitting time and an embedded Markov chain in continuous-time quantum walks

Miguel A. Ruiz-Ortiz[1] · Ehyter M. Martín-González[1] · Diego Santiago-Alarcon[2] · Salvador E. Venegas-Andraca[3]

Our step approach:

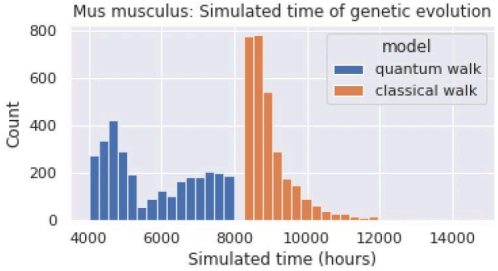
1) **Develop a theory and provide proof of concept in a closed system (Santiago-Alarcon et al. 2020)**
2) **Implement theory to realistic scenarios in closed systems (Ruiz-Ortiz et al. 2023, and in prep. – results below)**
3) **Develop predictive theory for open systems (almost there, first version within the next month)**
4) **Develop lab and field experiments to test the theory (for a grant)**

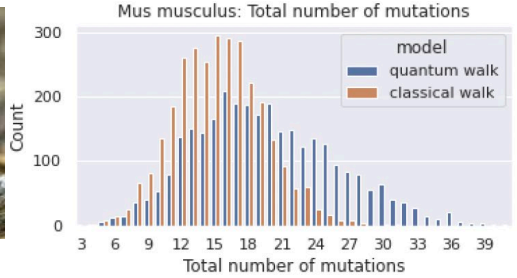
Question: What is more efficient at finding novel phenotypes between CRW vs. QW in a closed system (i.e., no selection or no environmental influence)

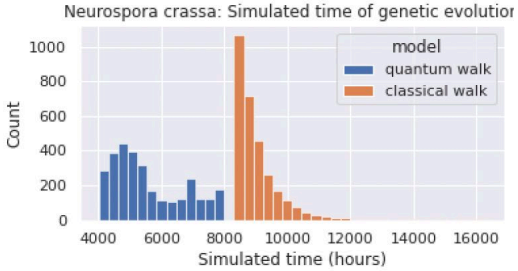
Genotype Networks of Transcription Factors in 3 Model Spp.

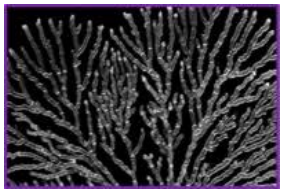
Question: What is more time efficient at exploring evolutionary space via mutations between CRW vs. QW in more realistic closed systems (i.e., no selection or no environmental influence)

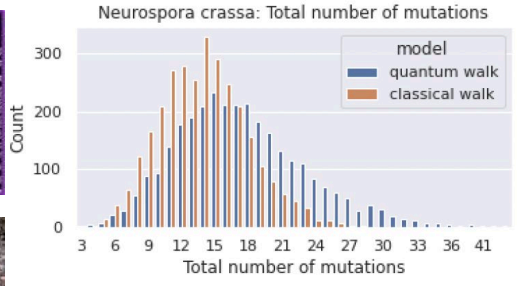
	Arabidopsis thaliana	Mus musculus	Neurospora crassa
# genotypes	16113	19986	17001
# phenotypes	217	190	118

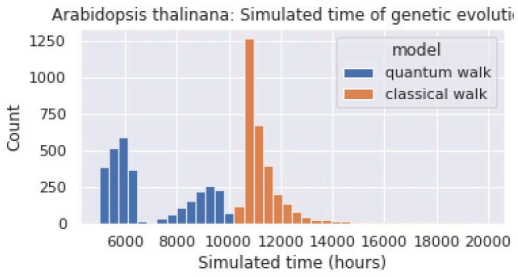


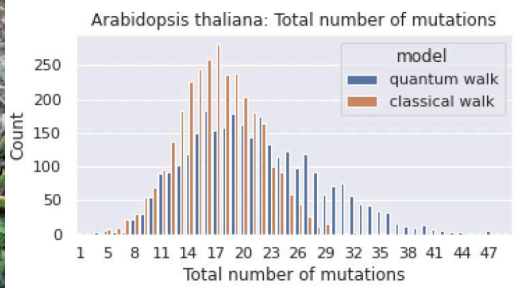






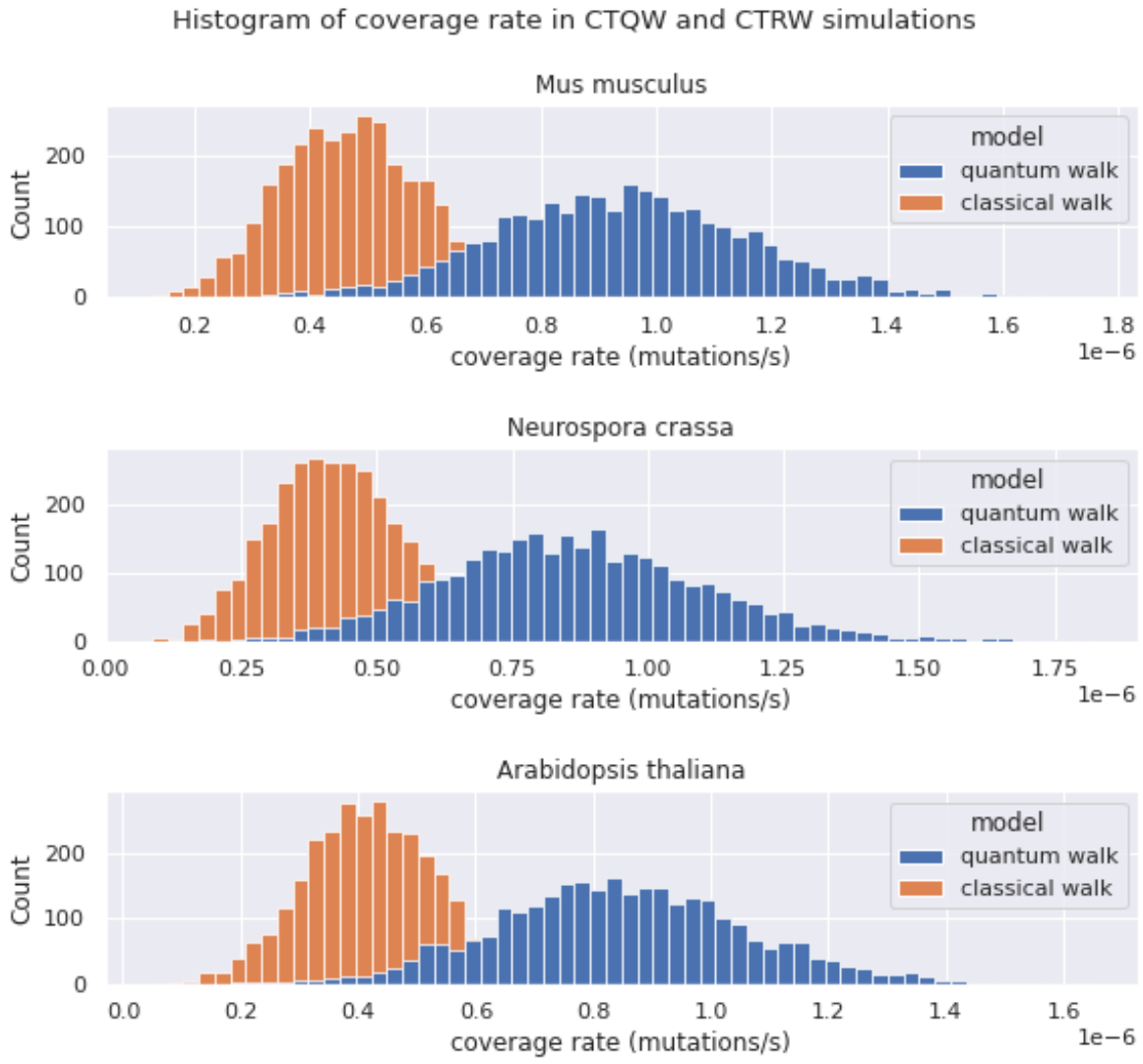


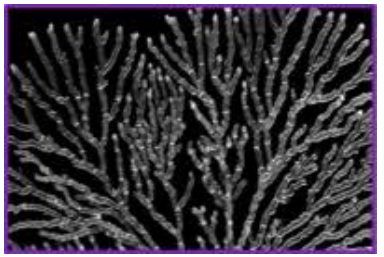




CRW need more genetic evolution time (left-column panels) to produce similar total number of mutations as the QW (i.e., similar distribution of total number of mutations in the right-column panels)

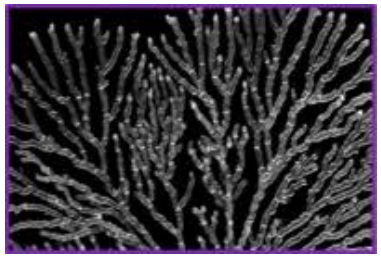
QW efficiency vs CRW: evolutionary neighborhood coverage via mutation

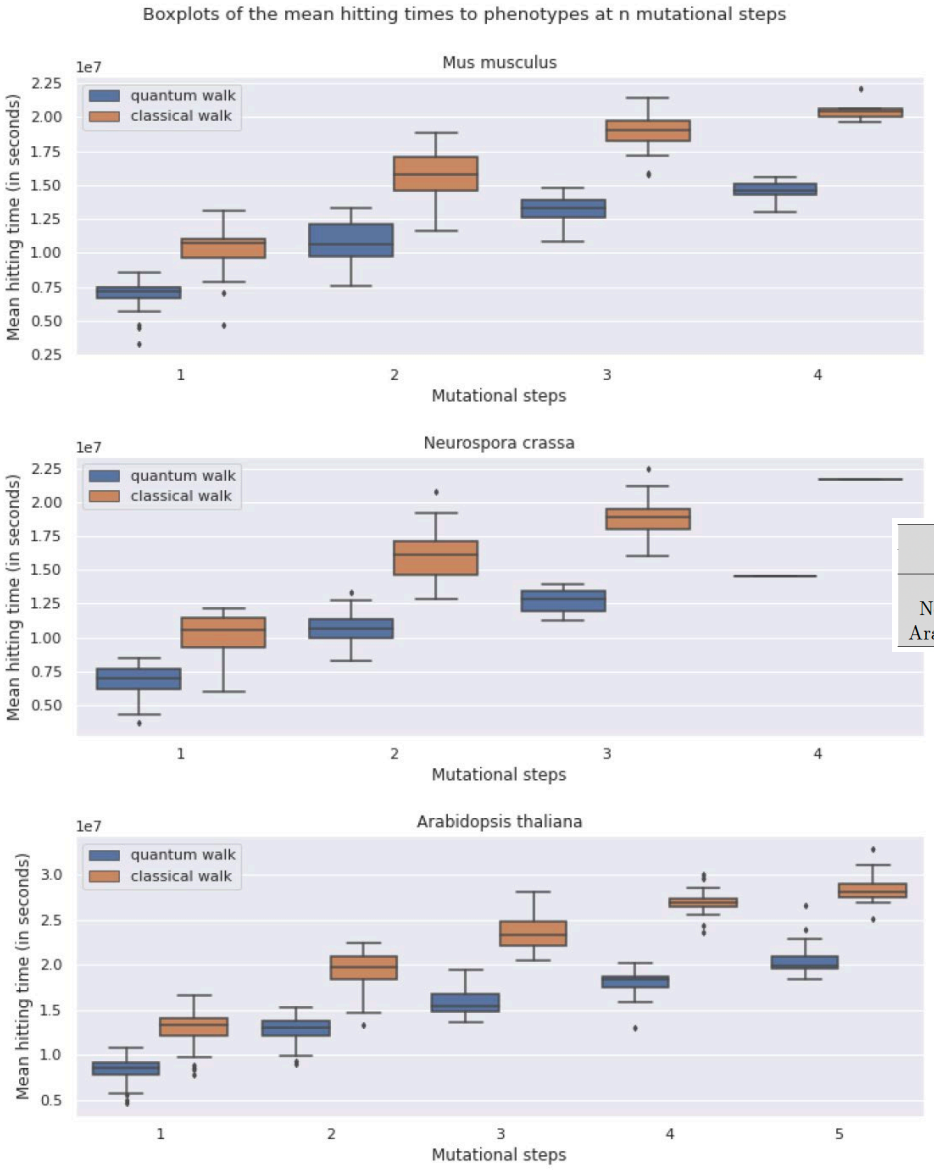




there is a 2-x speed up in the average coverage rate

QW efficiency vs CRW: discovery of evolutionary novelties via mutation





p-values of Mann-Whitney U tests					
n	1	2	3	4	5
Mus musculus	0.0	0.0	0.0	2.82×10^{-28}	
Neurospora crassa	0.0	0.0	0.0	7.98×10^{-10}	
Arabidopsis thaliana	0.0	0.0	0.0	0.0	1.28×10^{-61}

a 1.5x speed up

OUR INITIAL QUESTION:

We know the components of the machine and how they interact and what they do, but do we really know why there are nonrandom mutations?

WHERE WE ARE:

- A conceptual framework to investigate mutation patterns via QW
- QW higher probability of finding a novel phenotype than CRW
- QW efficiency increased in more complex networks
- QW is faster starting at different mutational steps away from focal phenotype
- QW covers a larger genotype space
- **Thus, QW better to investigate nonrandom mutations at the micro scale**

Non-random adaptive mutations are a causal process in evolution with empirical evidence; an effect 4 to 7 times larger than what is expected by the neutral theory of evolution in experiments, and from 2 to 3 times larger in field situations (Stoltzfus & McCandlish 2017)

Following Steps:

- Investigate in open systems (almost there; 3rd step)
- Conduct experimental and empirical field work (proposals in prep.; 4th step)

Conclusions and Following Steps: open vs. closed Hamiltonians

Closed System

$$\frac{\mathbf{d}p}{\mathrm{d}t} = \mathbf{H}p,$$

where $\mathbf{H}$ is the matrix with entries defined as

$$\mathbf{H}_{ij} = \begin{cases} \gamma \mathbf{A}_{ij}, & i \neq j \\ -\gamma \,\mathrm{outDeg}(i), & i = j, \end{cases}$$

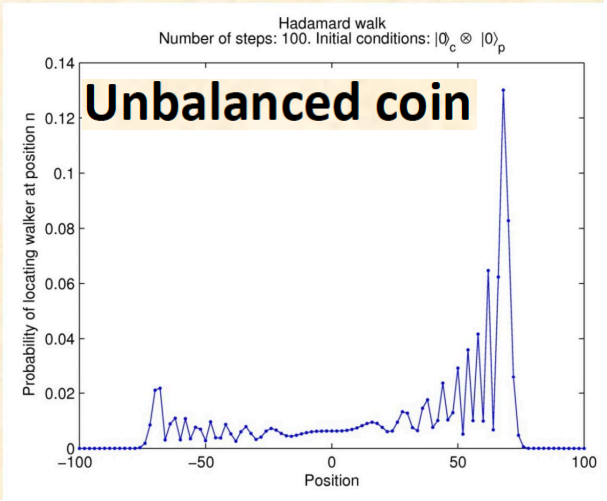
Open System

$$H = H_{DNA} + H_{bath} + H_{int}$$

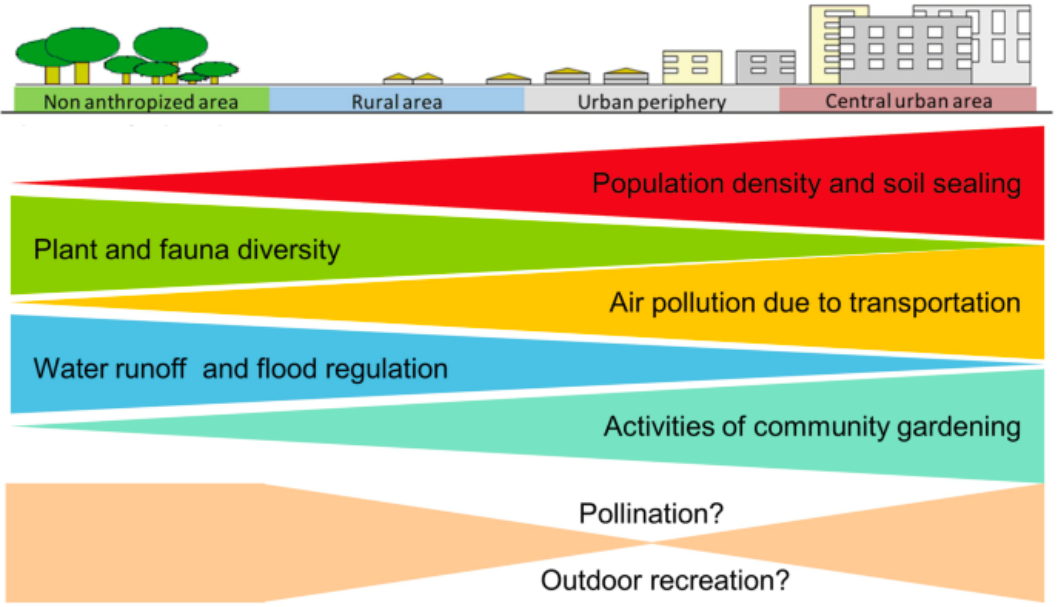
$$H_{DNA} = \sum_{i}^{N} h_i \sigma_i^z + \sum_{\langle ij \rangle} J_{ij} \sigma_i^z \sigma_j^z,$$

$$H_{bath} = \sum_{i}^{N} \sum_{k=0}^{\infty} \hbar \omega_i \left(b_k^\dagger b_k + \frac{1}{2} \right).$$

$$H_{int} = \sum_{i=1}^{N} \sum_{k=0}^{\infty} \left(\alpha_{ik} (b_k^\dagger b_k) \sigma_i^x + \epsilon_{ik} \sigma_i^z (b_k^\dagger + b_k) \right).$$



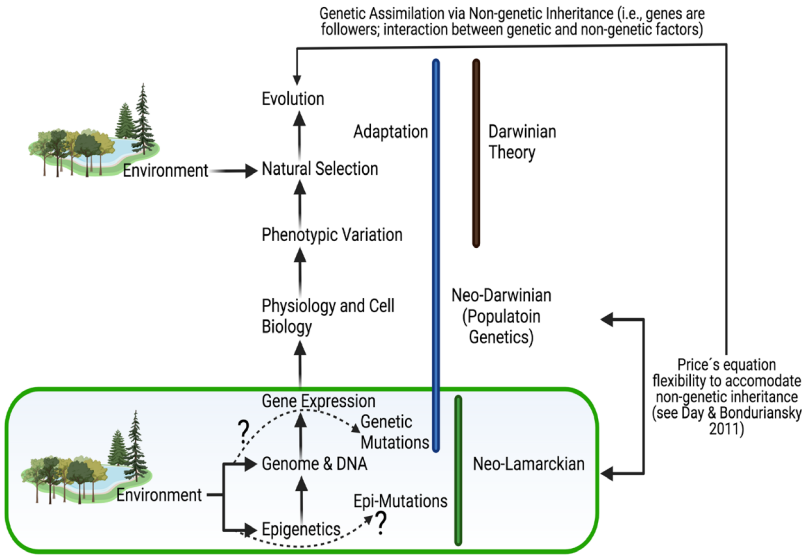
Connecting back to the macro…



- Cities are a drastic and permanent selective pressure
- They are increasing in number and size
- We have plenty of replicates around the world
- Population genetics just summarizes what happens in the genome

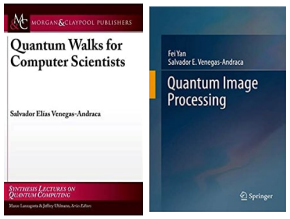
NOW IMAGINE:

- How wonderful would be to have a predictive theory of quantum evolution that can take us a step further in evolution, for example by:
  1) Identifying how many mutations away we are from a faulty protein that increases our risk of cancer
  2) Predicting the effect of the environment (e.g., urban) on the most likely course of genetic modifications



The Quantum Team

Salvador E. Venegas Andraca



Quantum walks: a comprehensive review

Salvador Elías Venegas-Andraca

Quantum Inf Process
DOI 10.1007/s11128-012-0432-5

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Horacio Tapia McClung

IBM

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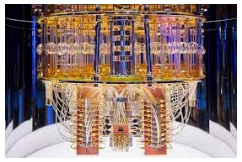


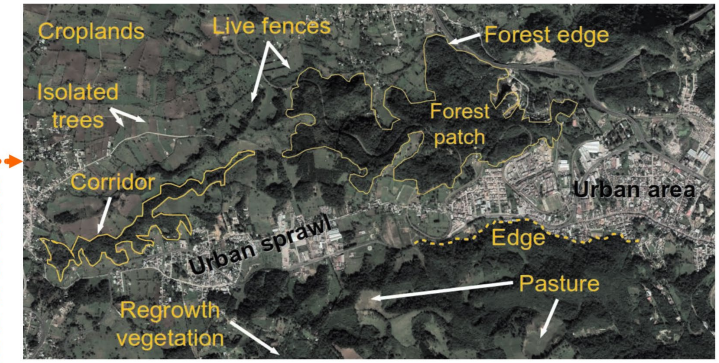
THANK YOU FOR YOUR ATTENTION!

LANDSCAPE SCALE DISEASE ECOLOGY

Question: how does habitat changes affect parasite diversity and infection rate?

How Should the Urban Be Perceived?

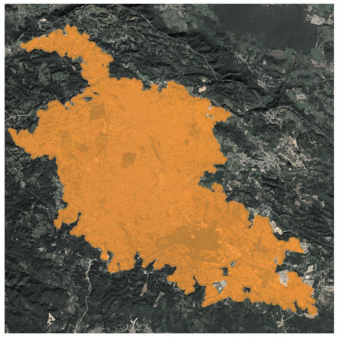
Landscape components

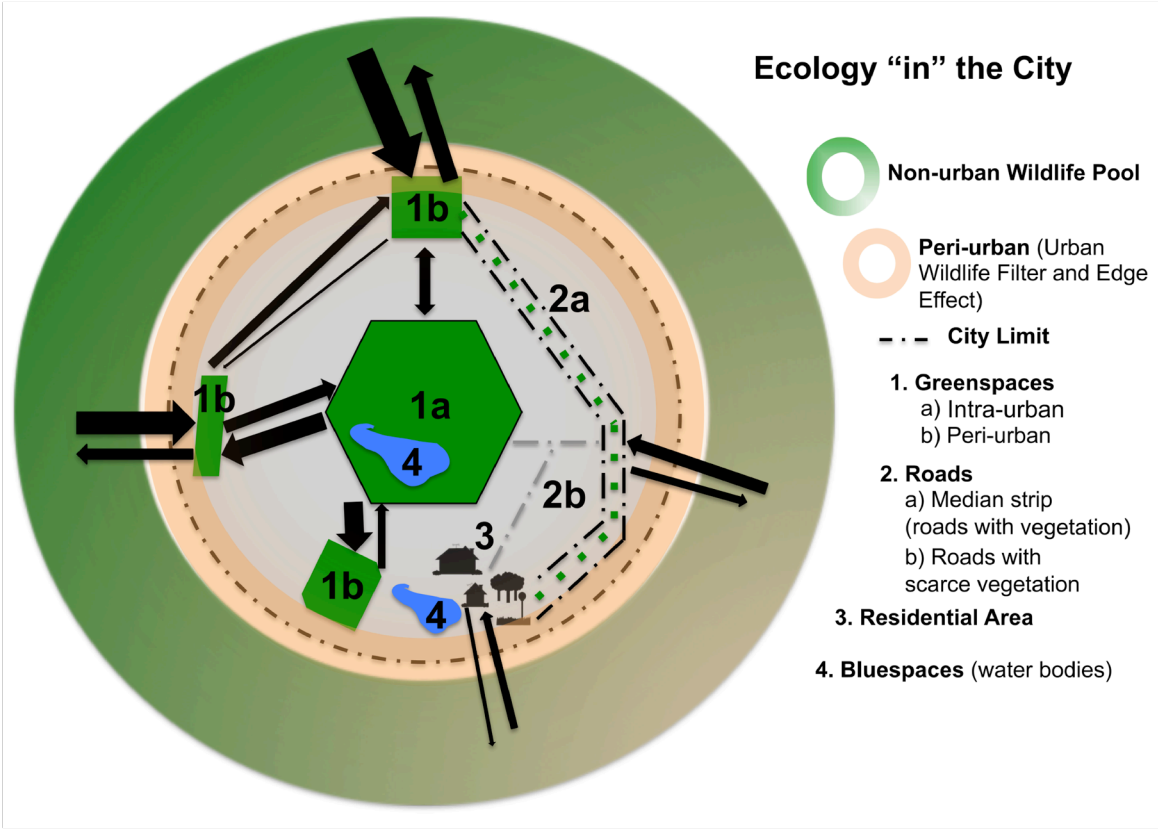


Urban zoning

Region where a citiy is settled

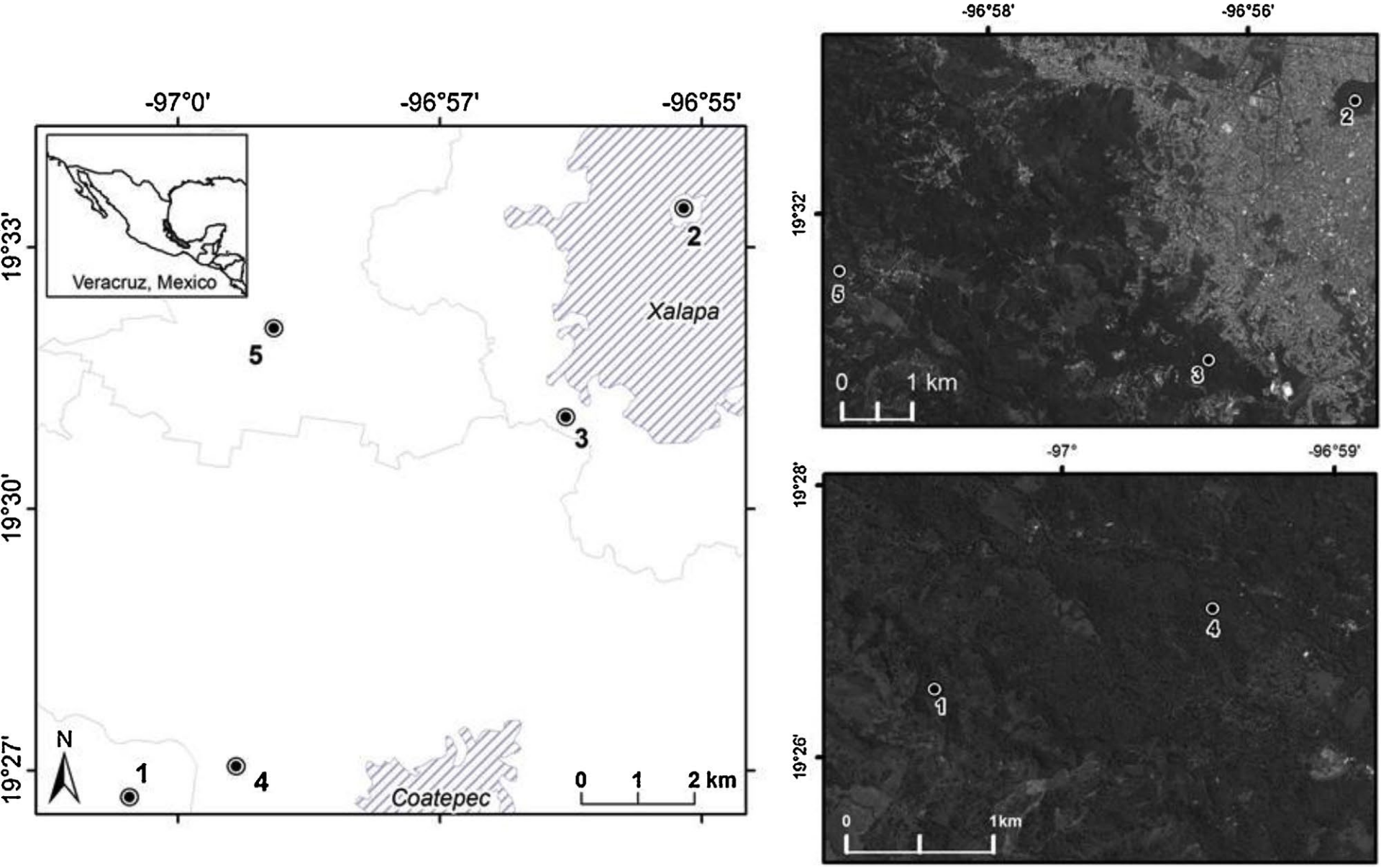
Urban continnuum





Santiago-Alarcon & MacGregor-Fors (in press)

Landscape epidemiology: land use types



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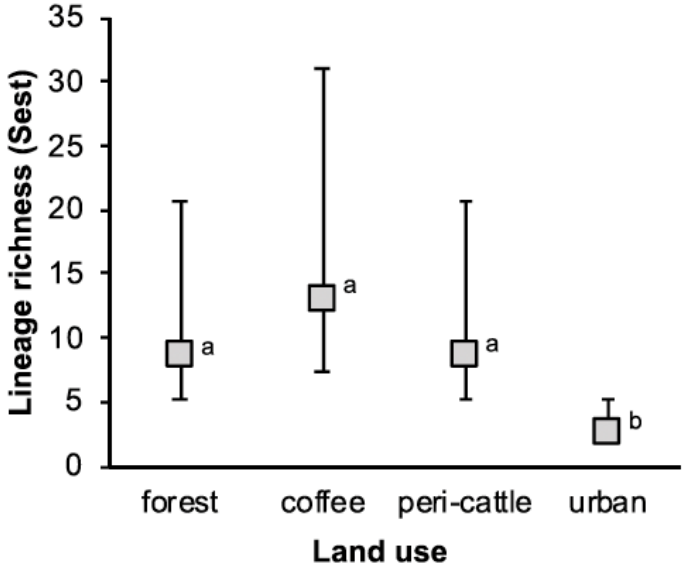
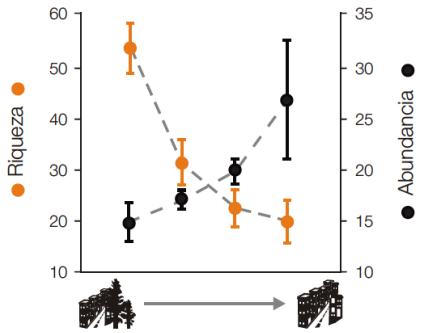
Parasite Richness & Abundance Structure





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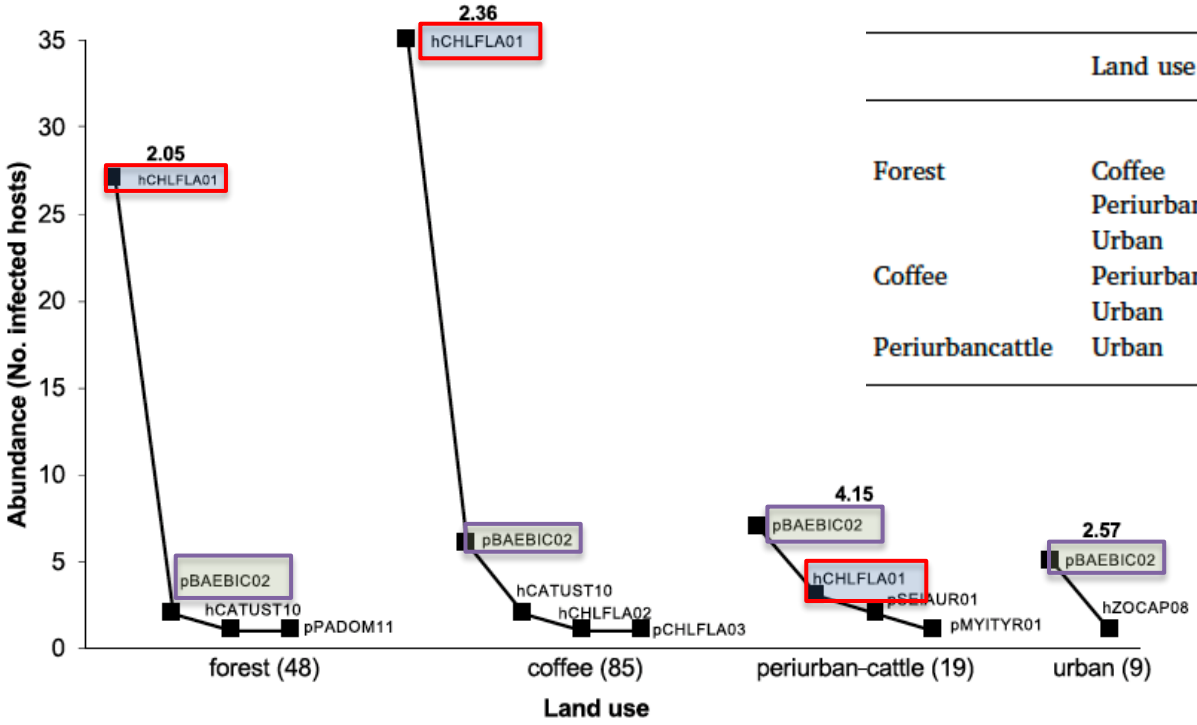
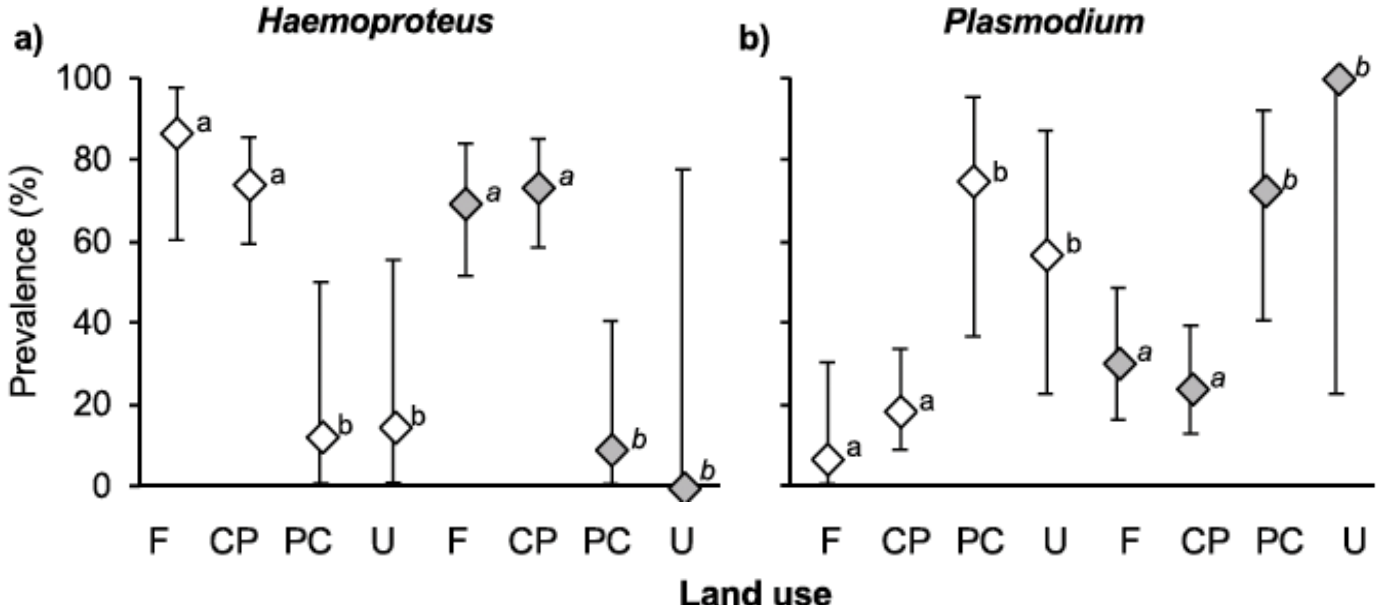
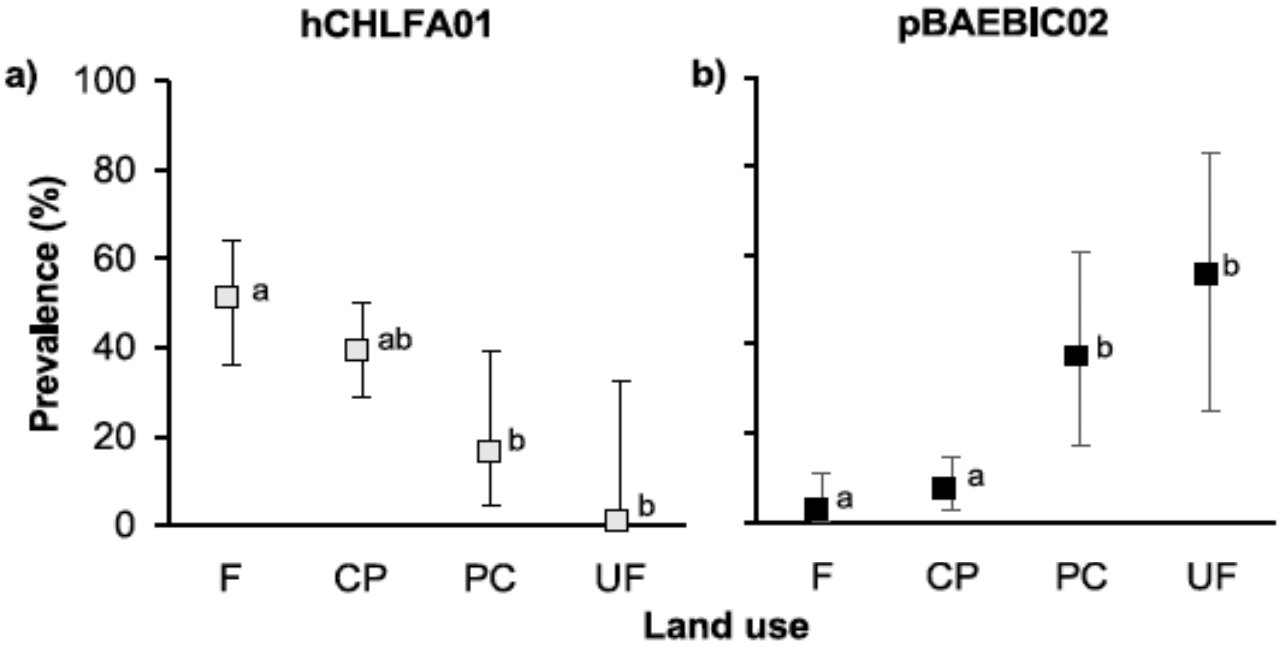


	Land use	β			Phylo β		
		sor	sim	sne	sor	sim	sne
Forest	Coffee	0.33	0.25	0.08	0.04	0.00	0.04
	Periurbancattle	0.50	0.50	0	0.28	0.14	0.14
	Urban	0.67	0.50	0.17	0.10	0.07	0.03
Coffee	Periurbancattle	0.56	0.50	0.06	0.24	0.14	0.10
	Urban	0.71	0.50	0.21	0.07	0.07	0
Periurbancattle	Urban	0.67	0.50	0.17	0.24	0.13	0.11

Parasitological parameters and seasonality





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