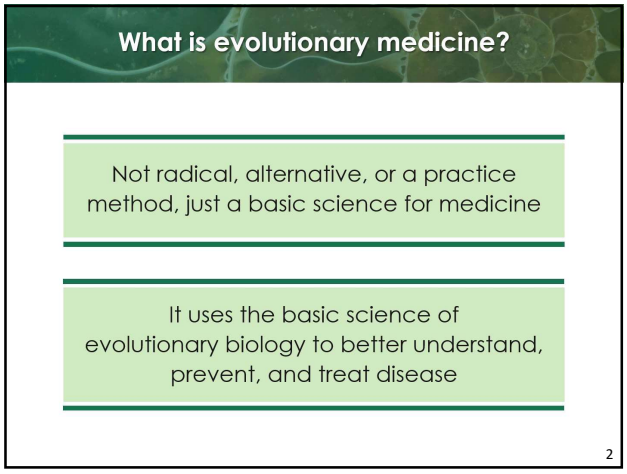
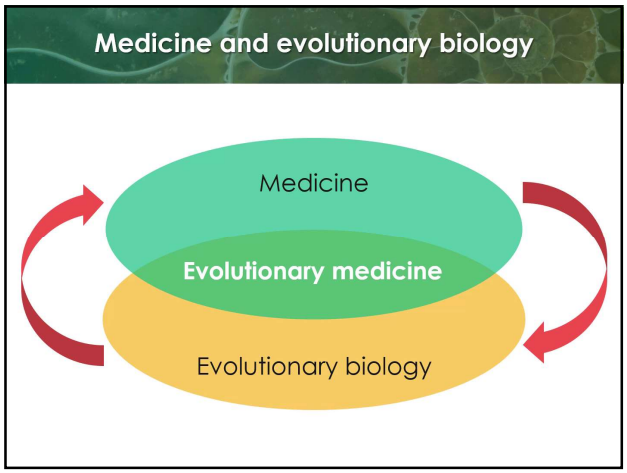
Prof. Randolph Nesse– University of Michigan, USA

HSTalks

1



2



3

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

The growth of evolutionary medicine

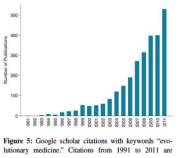


Figure 5: Google scholar citations with keywords "evolutionary medicine." Citations from 1991 to 2011 are

EVOLUTION, MEDICINE, & PUBLIC HEALTH

INTERNATIONAL SOCIETY FOR EVOLUTION, MEDICINE & PUBLIC HEALTH

EvMedEd

HSTalks

4

4

Traits need two kinds of explanation

E. Mayr, 1982

"No biological problem is solved until both the proximate and the evolutionary causation has been elucidated"

Furthermore, the study of evolutionary causes is as legitimate a part of biology as is the study of the usually physico-chemical proximate causes

Medicine has been using only one half of biology

5

5

Natural selection

When inherited variations in a trait influence the number of offspring, the trait will change over the generations

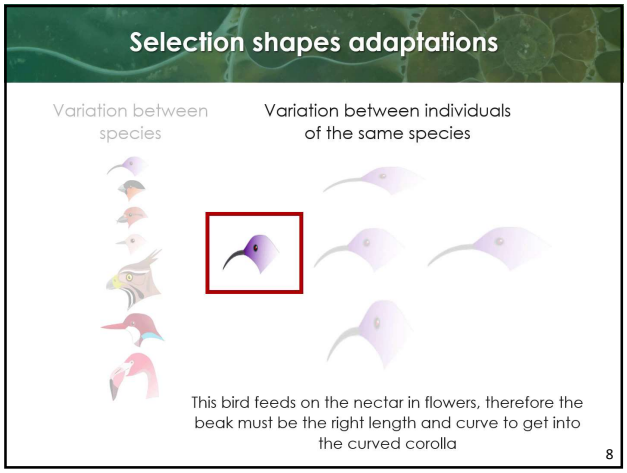
6

6

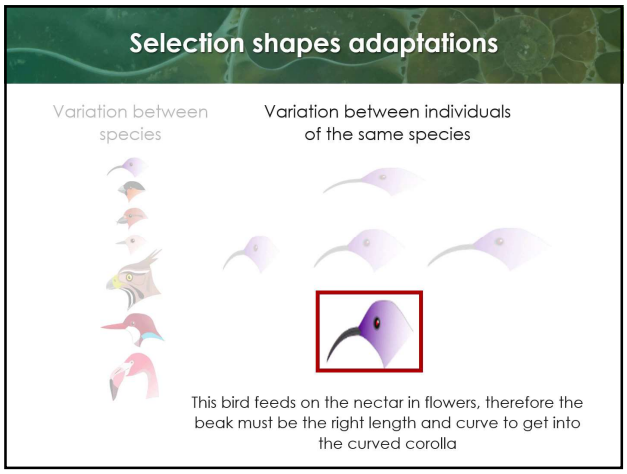
Prof. Randolph Nesse– University of Michigan, USA

HSTalks

7



8



9

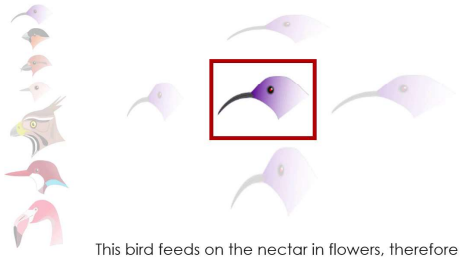
Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Selection shapes adaptations

Variation between species

Variation between individuals of the same species



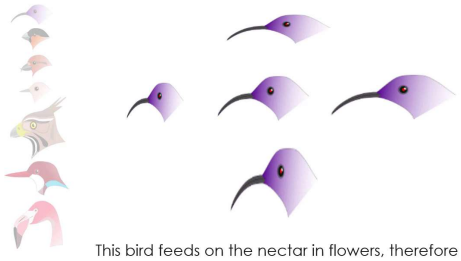
This bird feeds on the nectar in flowers, therefore the beak must be the right length and curve to get into the curved corolla

10

Selection shapes adaptations

Variation between species

Variation between individuals of the same species



This bird feeds on the nectar in flowers, therefore the beak must be the right length and curve to get into the curved corolla

11

Apparent maladaptation

9

12

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Apparent maladaptation

13

Tinbergen's 4 questions

Nico Tinbergen, 1963

01. How does it work?

02. How did it develop?

03. How is it useful?

04. What is its phylogeny?

10

14

Tinbergen's 4 questions, organized

Tinbergen's four questions		Two objects of explanation: Sequence	Two objects of explanation: Single form
Two kinds of explanation	Proximate: Mechanisms and their ontogeny	Ontogeny: Q: How does the trait develop in individuals?	Mechanism: Q: What is the structure of the trait; how does it work?
	Evolutionary: Functions and phylogeny	Phylogeny: Q: What is the history of the trait?	Adaptive significance: Q: How have variations in the trait influenced fitness?

Nesse, R. M. *Trends in Ecology & Evolution*, 2013 **28**(12), 681–682

11

15

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Tinbergen's 4 questions, organized

Tinbergen's four questions		Two objects of explanation: Sequence	Two objects of explanation: Single form
Two kinds of explanation	Proximate: Mechanisms and their ontogeny	Ontogeny: Q: How does the trait develop in individuals?	Mechanism: Q: What is the structure of the trait; how does it work?
Two kinds of explanation	Evolutionary: Functions and phylogeny	Phylogeny: Q: What is the history of the trait?	Adaptive significance: Q: How have variations in the trait influenced fitness?

Nesse, R. M. *Trends in Ecology & Evolution*, 2013 **28**(12), 681–682

16

Tinbergen's 4 questions, organized

Tinbergen's four questions		Two objects of explanation: Sequence	Two objects of explanation: Single form
Two kinds of explanation	Proximate: Mechanisms and their ontogeny	Ontogeny: Q: How does the trait develop in individuals?	Mechanism: Q: What is the structure of the trait; how does it work?
Two kinds of explanation	Evolutionary: Functions and phylogeny	Phylogeny: Q: What is the history of the trait?	Adaptive significance: Q: How have variations in the trait influenced fitness?

Nesse, R. M. *Trends in Ecology & Evolution*, 2013 **28**(12), 681–682

17

Tinbergen's 4 questions, organized

Tinbergen's four questions		Two objects of explanation: Sequence	Two objects of explanation: Single form
Two kinds of explanation	Proximate: Mechanisms and their ontogeny	Ontogeny: Q: How does the trait develop in individuals?	Mechanism: Q: What is the structure of the trait; how does it work?
Two kinds of explanation	Evolutionary: Functions and phylogeny	Phylogeny: Q: What is the history of the trait?	Adaptive significance: Q: How have variations in the trait influenced fitness?

Nesse, R. M. *Trends in Ecology & Evolution*, 2013 **28**(12), 681–682

18

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

The paradox

Near perfection for some traits

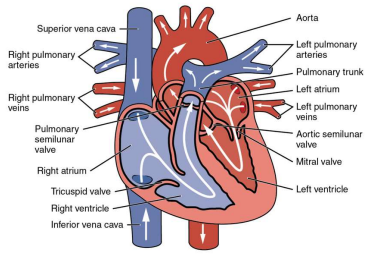
Grossly poor design for others

12

19

Amazing perfection

The heart and valves

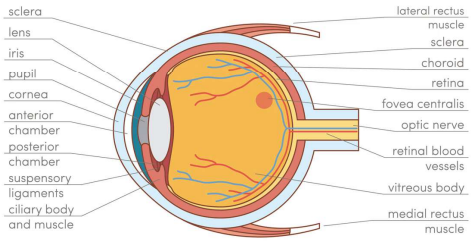


13

20

Amazing perfection

The eye



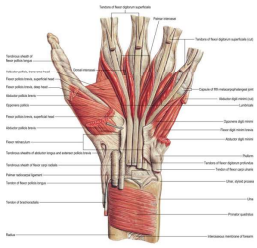
21

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Amazing perfection

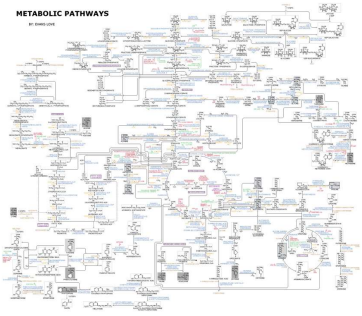
The human hand



22

Amazing perfection

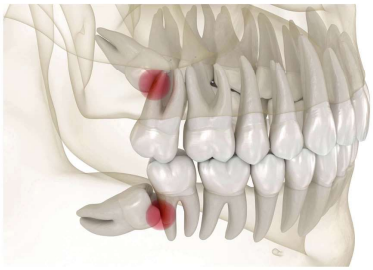
Metabolic pathway



23

Crude imperfection

Wisdom teeth



14

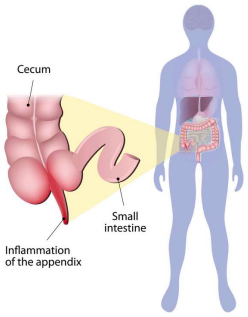
24

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Crude imperfection

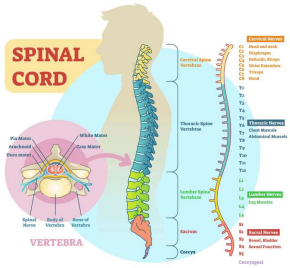
Appendix



25

Crude imperfection

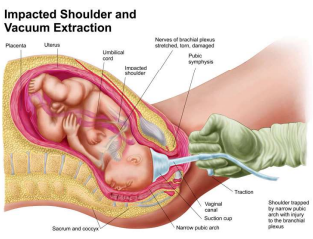
Spine



26

Crude imperfection

Birth canal



27

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

A new question

Why did natural selection leave the body vulnerable to disease?

Not why some individuals get sick

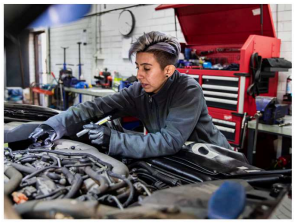
Why all individuals in a species have traits that make them vulnerable?

15

28

The usual question

A mechanic/medic asks



How does it work?

What is broken?

How can we fix it?

16

29

The evolutionary question

An engineer/evolutionist asks

What is the history of this part?

Why wasn't it designed better?

17

30

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Darwin made two discoveries

Phylogeny

The unity of all life

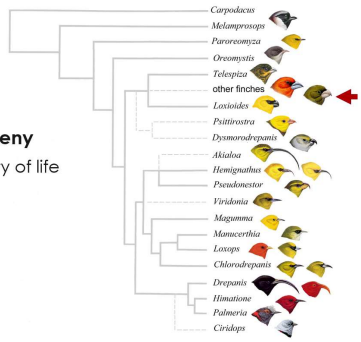
Adaptation

How traits are shaped to be suited to their functions

18

31

Phylogeny and adaptation



Phylogeny

The unity of life

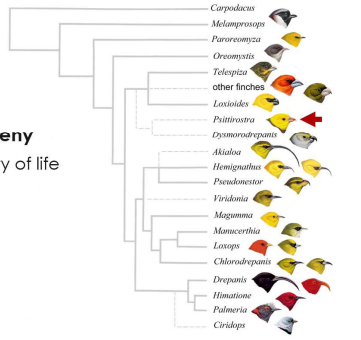
Adaptation

Why traits work so well

19

32

Phylogeny and adaptation



Phylogeny

The unity of life

Adaptation

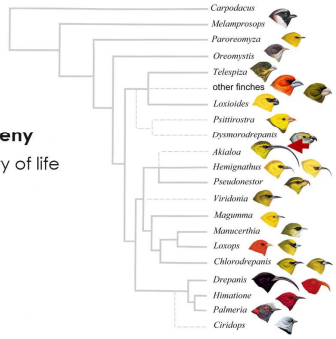
Why traits work so well

33

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Phylogeny and adaptation

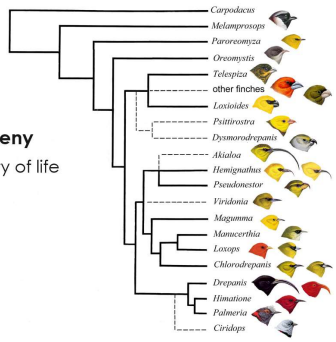


Phylogeny
The unity of life

Adaptation
Why traits work so well

34

Phylogeny and adaptation



Phylogeny
The unity of life

Adaptation
Why traits work so well

35

Two distinct areas of evolutionary medicine

20

36

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

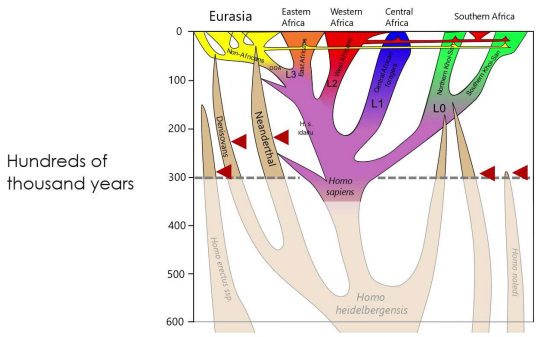
The phylogenomic half of evolutionary medicine

- Human ancestry
- Pathogen origins and evolution
- Selection influences on human genes

21

37

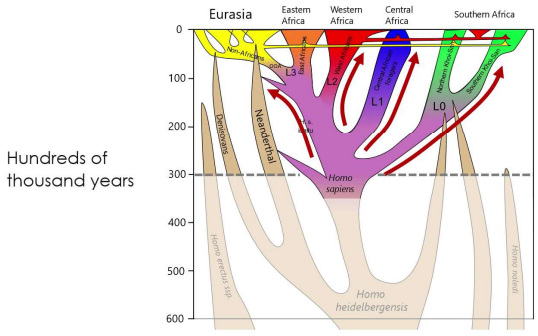
Homo sapiens phylogeny



22

38

Homo sapiens phylogeny

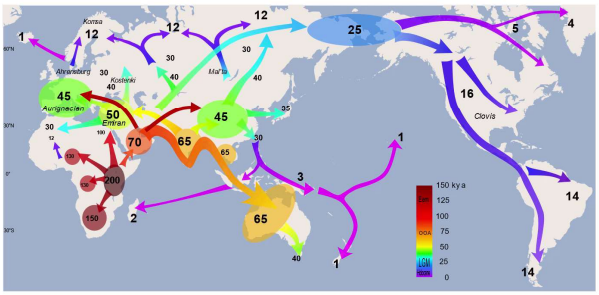


39

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

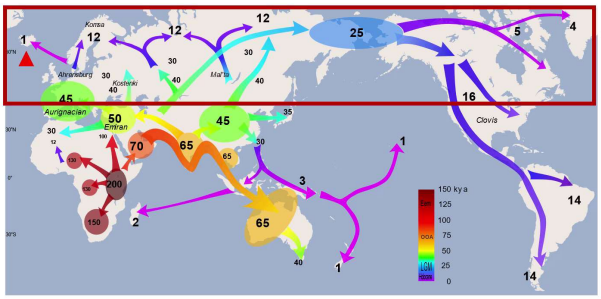
Tracing human origins



23

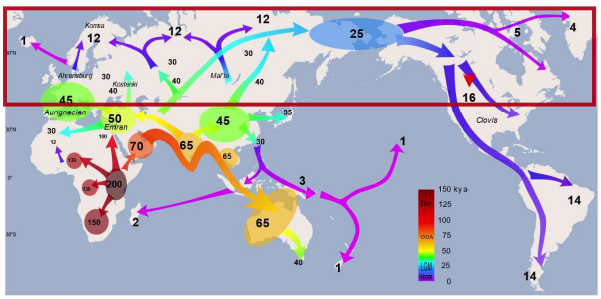
40

Tracing human origins



41

Tracing human origins



42

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

TB in the New World from seals

Abnormalities in skeletons showing individuals had suffered from TB were discovered in the New World *before* people had come from Europe

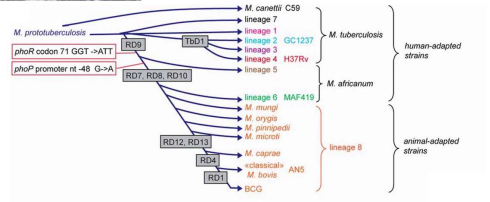
How did it get there?

Bos, K. I. *et al., Nature*. 2014; **514**(7523):494-7

24

43

TB in the New World from seals

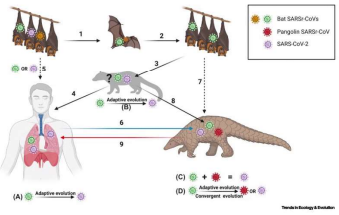


Bos, K. I. *et al., Nature*. 2014; **514**(7523):494-7

44

COVID-19 origins

25



1. Jinyintan Hospital; 2. Wuhan Central Hospital, Houhu Branch (no. 2); 3. Hubei Provincial Hospital of Integrated Chinese and Western Medicine; 4. Wuhan Central Hospital, Nanjing Road Branch; 5. Tongji Hospital; 6. Union Hospital; 7. Zhongnan Hospital; 8. Wuhan Jiangxia First People's Hospital

Worobey, M. *et al., Science*. 2022; **377**(6609):951-959
Banerjee, A. *et al., Trends Ecol Evol*. 2021; **36**(3):180-184

45

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Selection for TB protection in cities

TB became worse in urban environments

ORIGINAL ARTICLE

doi:10.1111/j.1558-5646.2010.01132.x

ANCIENT URBANIZATION PREDICTS GENETIC RESISTANCE TO TUBERCULOSIS

Ian Barnes,[1] Anna Duda,[2] Oliver G. Pybus,[3] and Mark G. Thomas[2,4,5]

Conclusion

SLC11A1 protection factor has become more common in areas that were urbanized earlier rather than later

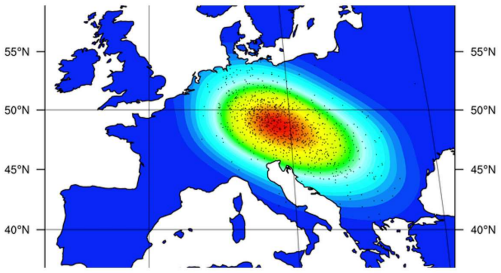
Barnes, I. *et al., Evolution*. 2011; **65**(3):842-8

26

46

The inability to digest milk as an adult

The inability to digest milk is a variation that happens in some individuals



Itan, Y. *et al., PLoS Comput Biol*. 2009; **5**(8):e1000491

27

47

Strep mutans + carbohydrates --> cavities

"*S. mutans* populations started expanding exponentially approximately 10,000 years ago, coincidental with the onset of human agriculture"

Cornejo, O. E. *et al., Mol Biol Evol*. 2013; **30**(4):881-93

28

48

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Why natural selection left us vulnerable to disease

The newer half
of evolutionary medicine

29

49

Explaining disease vulnerability

How did natural selection shape this disease?

30

50

Explaining disease vulnerability

How did natural selection shape this disease?

A bad question

Why has natural selection left the body vulnerable to this disease?

A ***very*** good question

51

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Why did natural selection leave us vulnerable?

- It can't prevent all mutations and variation
- It can't start fresh or avoid genetic drift
- It is slow
- It maximizes reproduction at a cost to health
- It optimizes trade-offs to maximize inclusive fitness at a cost to health

31

52

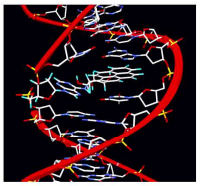
Explanations for vulnerability

01. Individual variations, genetic and developmental
02. Species-wide suboptimal designs
03. Arms races with fast-evolving pathogens
04. Mismatch with novel environments
05. Trade-offs that reduce robustness
06. Gene transmission at the expense of health
07. Defenses are adaptations, not diseases

32

53

Mutations



- About 100 new mutations per individual
- Deleterious variants only slowly purged
- Mutation selection balance
  - Explains individual differences
  - But not why a trait is vulnerable

33

54

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Apo E4

- 25% heterozygous
- Increases inflammation and risk for Alzheimer's and atherosclerosis
- Ancestral, being replaced by E2 and E3

34

55

Apo E4

THE FASEB JOURNAL • RESEARCH • www.fasebj.org
Trunble, B. C. *et al., FASEB J.* 2017; **31**(4):1508-1515

Apolipoprotein E4 is associated with improved cognitive function in Amazonian forager-horticulturalists with a high parasite burden

Benjamin C. Trumble,*,†,‡ Jonathan Stieglitz,‖,§ Aaron D. Blackwell,¶ Hooman Allayee,‖,# Bret Beheim,**

- Slows cognitive decline in the Ache
- Apo E4 improves cognition!
- A gene is never beneficial or harmful unless except in a particular environment

56

Developmental variation

- Ontogeny is astoundingly canalized
- But stochastic variation nonetheless leaves some individuals impaired

The brain is especially vulnerable

- It's important to recognize that there are going to be multiple explanations for a single trait

35

57

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Species-wide vulnerabilities

Path dependence

- Can't start fresh
- Useful changes disrupt other systems

Genetic drift

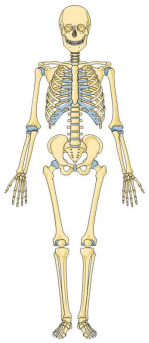
- Fixation of deleterious alleles
- Loss of useful alleles

36

58

Wrenching transitions: to bipedality

- Neck pain
- Back Pain
- Dystocia
- Hernia
- Arthritis
- Varicose veins
- Ankle problems

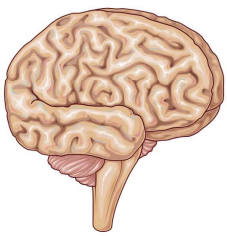


- Rotator cuff injuries
- Broken hip
- Pelvic prolapse
- Knee injuries
- Shin splints
- Plantar fasciitis

37

59

Wrenching transitions: cognitive/social niche



What needs to change in the brain to…

- Speak
- Remember
- Form complex social relations

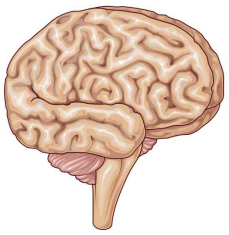
38

60

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Wrenching transitions: cognitive/social niche

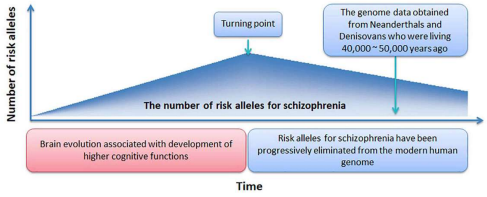


- Dyslexia
- Social anxiety
- Autism
- Schizophrenia
- Biopolar disorder

61

Schizophrenia from the transition to the cognitive-social niche

Did the wrenching transition to the social/cognitive niche make some neutral alleles pathological?



Older genetic variations that might increase the risk of schizophrenia are being selected against

Subsequently, there has been selection for new alleles that reduce the risk of schizophrenia

Liu, C. *et al., Front Genet.* 2019;**10**:389

39

62

Older alleles increase risk, newer alleles decrease risk

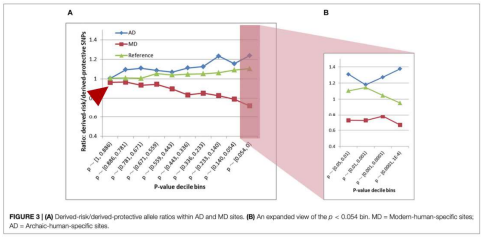


FIGURE 3 | **(A)** Derived-risk/derived-protective allele ratios within AD and MD sites. **(B)** An expanded view of the $p < 0.054$ bin. MD = Modern-human-specific sites; AD = Archaic-human-specific sites.

Liu, C. *et al., Front Genet.* 2019;**10**:389

40

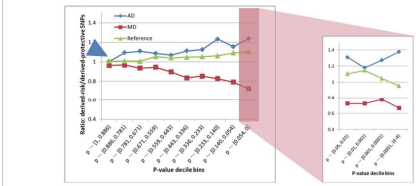
63

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Older alleles increase risk, newer alleles decrease risk

Selection against ancient deleterious alleles



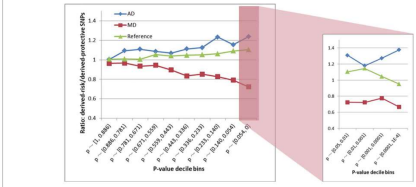
Selection for modern protective alleles

Liu, C. *et al., Front Genet.* 2019;**10**:389

64

Older alleles increase risk, newer alleles decrease risk

Selection against ancient deleterious alleles



Selection for modern protective alleles

No similar pattern for SNPs for bipolar, autism and major depressive disorder

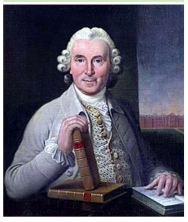
Liu, C. *et al., Front Genet.* 2019;**10**:389

65

Scurvy

- Vitamin C was ubiquitous 60 million years ago, so there was no selection pressure to maintain the L-gulono-lactone oxidase gene

James Lind



41

66

Prof. Randolph Nesse– University of Michigan, USA

Scurvy

- Vitamin C was ubiquitous 60 million years ago, so there was no selection pressure to maintain the L-gulono-lactone oxidase gene
- It has recently been suggested that it wasn't just lack of selection, but that loss of that gene may have increased the ability to store fat

67

Why billions of years for complex organisms to emerge?

Infection

Small organisms evolve faster than the larger ones that they eat

Cancer

Cells that replicate faster are selected for even if they harm the individual

Enforcing cooperation is a huge challenge

42

68

Infection: we are vulnerable because…

- Pathogens evolve 10,000x faster
- Astounding that bodies control them
- They are shaped to maximize spread
- Mode of transmission matters

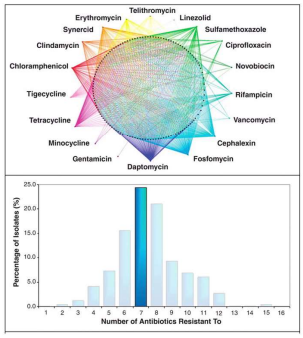
43

69

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Antibiotic resistance



Antibiotic resistance profiling of 480 soil-derived bacterial isolates

Why is this?

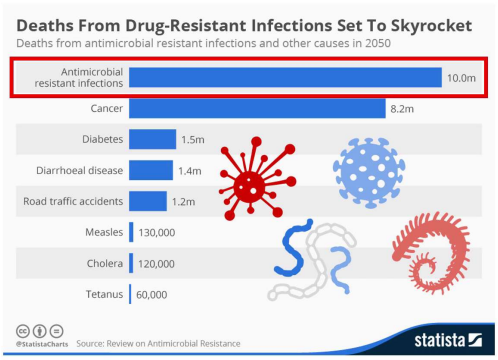
Biochemical warfare between bacteria and between bacteria and other organisms has shaped molecules that kill competing bacteria

D'Costa, V. M. *et al., Science*. 2006; **311**(5759):374-377

44

70

Death from drug-resistant infections



45

71

Take every pill in the bottle?

Andrew Read

May cause faster development of antibiotic resistance

46

72

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Revised recommendation

BMJ 2017;358:j3418 doi: 10.1136/bmj.j3418 (Published 2017 July 26) Page 1 of 5

ANALYSIS

The antibiotic course has had its day

With little evidence that failing to complete a prescribed antibiotic course contributes to antibiotic resistance, it's time for policy makers, educators, and doctors to drop this message, argue **Martin Llewelyn and colleagues**

No mention of prior work

No mention of 'evolution'

Llewelyn, M. J. *et al.*, *BMJ*. 2017; **358**:j3418

47

73

Cancer: why aren't we protected?

We **are**!

Astoundingly well

But trade-offs limit protection

48

74

Peto's Paradox proves strong selection against cancer

Josh Schiffman

Why do elephants with 10,000x more cells than a mouse have less cancer?

He predicted that the p53 genes that cause cells to self-destruct may play a role in this

49

75

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Reproductive patterns are different now

50

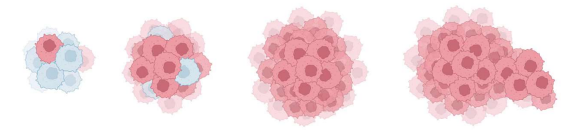
Rapidly increasing rates of breast cancer are associated with incessant ovulation, excess nutrition and decreased breast feeding

	USA	Dogon (Mali)
Age at menarche	12	17
Age at first pregnancy	26	19
Lifetime menses	400+	100
Never pregnant	17%	1%

Strassmann, B. I. *J Womens Health*. 1999; **8**(2):193-202

76

Cancer and somatic evolution



Variation in cells in a malignancy

Those that divide faster take over

A tumor is an ecosystem

HUGE implications for chemotherapy

51

77

Adaptive cancer therapy

Less chemo → Longer survival

The most malignant cells are inhibited from further growth by the cells that surround them

If these healthy cells are killed by chemotherapy, the ecosystem is opened up, and the faster dividing cells can expand

Gatenby, R. A. *et al.*, *Cancer Res.* 2009;**69**(11):4894-4903

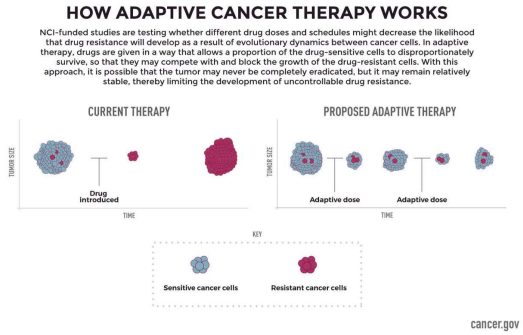
52

78

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

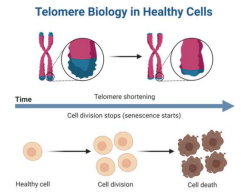
Adaptive cancer therapy



Gatenby, R . A. *et al., Cancer Res.* 2009;**69**(11):4894-4903

79

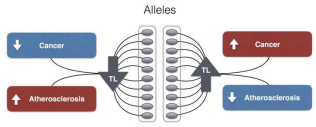
Cancer *vs.* aging: the telomere trade-off



- Each time the cell replicates, the telomere shortens a bit

A safety mechanism

- When a telomere becomes too short, the cell self-destructs



- Alleles associated with shorter telomeres diminish the risk of cancer
- Alleles associated with longer telomeres increase the risk cancers

Stone, R. C. *et al., PLoS Genet.* 2016; **12**(7):e1006144

53

80

Atherosclerosis

Studies of mechanisms are important

Why some are more vulnerable

Evolutionary approach are also essential

Why we are all vulnerable

Inflammation is useful

But excessive in modern societies

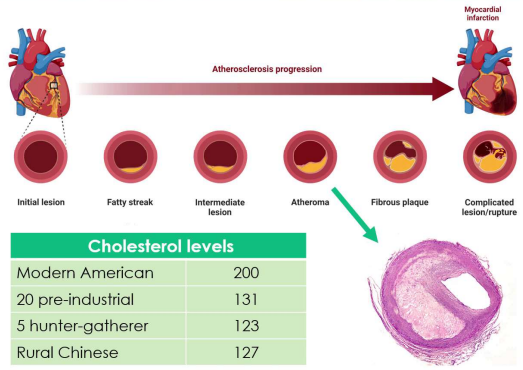
54

81

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Atheroma

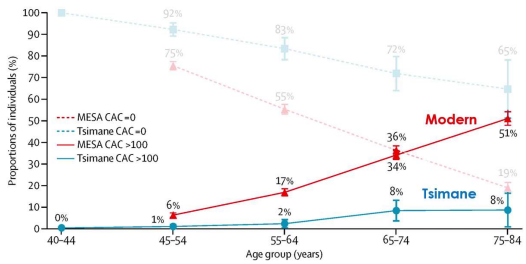


Cholesterol levels	
Modern American	200
20 pre-industrial	131
5 hunter-gatherer	123
Rural Chinese	127

55

82

Coronary disease in the Tsimane

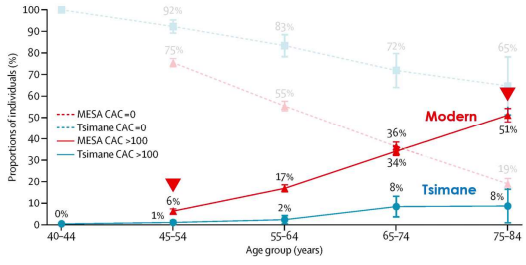


Kaplan H. *et al., Lancet.* 2017; **389**(10080):1730-1739

56

83

Coronary disease in the Tsimane



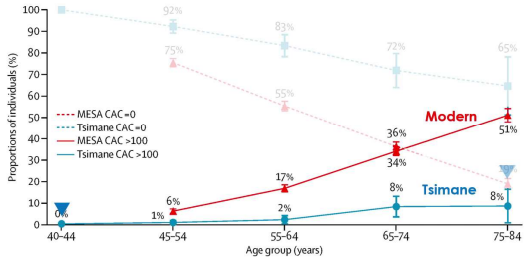
Kaplan H. *et al., Lancet.* 2017; **389**(10080):1730-1739

84

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

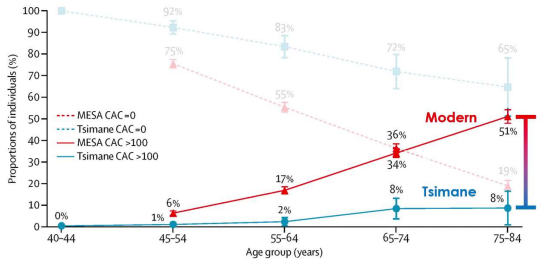
Coronary disease in the Tsimane



Kaplan H. *et al., Lancet.* 2017; **389**(10080):1730-1739

85

Coronary disease in the Tsimane



Exercise? | Diet? | Immune system?

Kaplan H. *et al., Lancet.* 2017; **389**(10080):1730-1739

86

Obesity and diet

There's no such thing as 'the normal diet'

But most of us eat very abnormal diets!

57

87

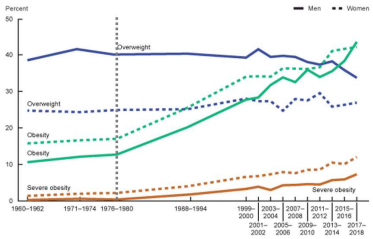
Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Obesity and diet

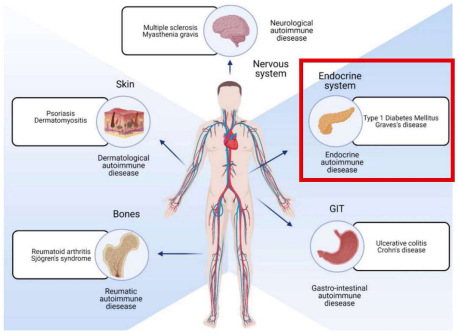
Huge epidemic of chronic disease

Age-adjusted trends in overweight, obesity, and severe obesity among men and women ages 20-74 in the United States



88

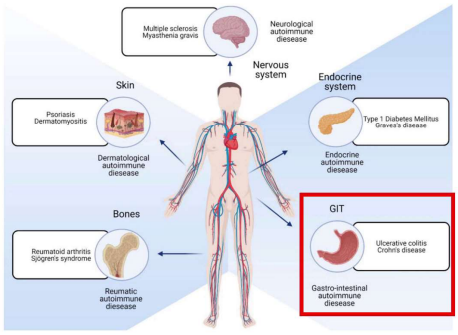
Autoimmune diseases increasing



58

89

Autoimmune diseases increasing

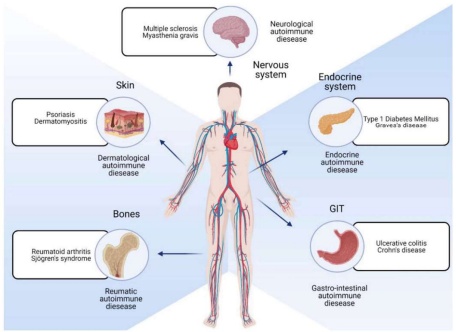


90

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

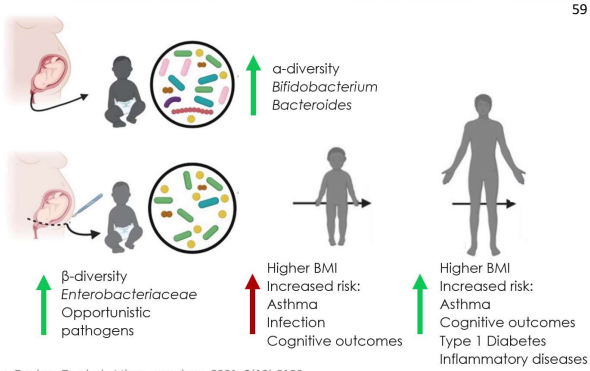
Autoimmune diseases increasing



91

C-section increases risks

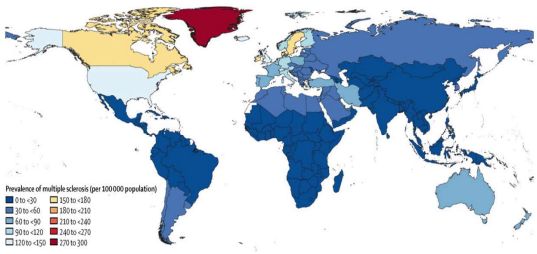
59



Rios-Covian, D. *et al., Microorganisms*, 2021; **9**(10):2122

92

Multiple sclerosis



Evolution: mismatch with missing worms

Mechanism: autoimmune damage

GBD 2016 Multiple Sclerosis Collaborators. *Lancet Neurol.* 2019; **18**(3):269-285

60

93

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Helminths protect against multiple sclerosis

ORIGINAL ARTICLES

Association Between Parasite Infection and Immune Responses in Multiple Sclerosis

Jorge Correale, MD, and Mauricio Farez, MD

Individuals uninfected by worms were much more likely to have multiple sclerosis, while those infected by worms were unlikely to suffer from exacerbations

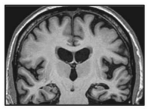
Correale, J. and Farez, M. *Ann Neurol.* 2007; **61**(2):97-108

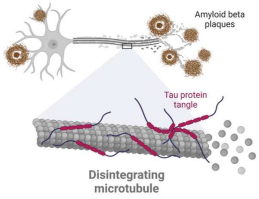
61

94

Why amyloid beta?

Alzheimer's brain





Amyloid beta interacts with Tau to cause plaques/ tangles that interact with the compliment system and immune system to kill off neurons, eventually causing Alzheimer's disease

62

95

Why amyloid beta?

It's a fine antimicrobial

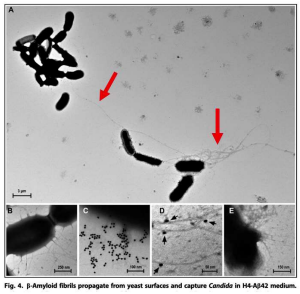


Fig. 4. β-Amyloid fibrils propagate from yeast surfaces and capture *Candida* in H4-Aβ42 medium.

Kumar, D. K. V. *et al., Sci Transl Med.* 2016; **8**(340):340ra72

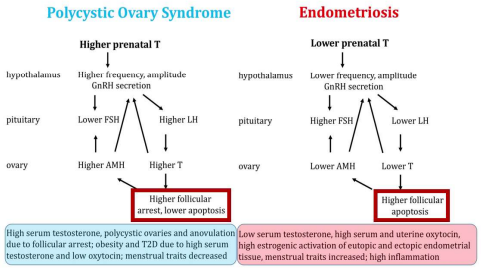
96

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Diametric diseases

Sets of disease that show opposite patterns in their nature, meaning if you suffer from one you are less likely to suffer from another



Dinsdale, N. *et al., Evol Med Public Health*. 2021; **9**(1):174-191

63

97

Painful symptoms

Why such excess?

They are useful

Optimal systems have false alarms

64

98

Defenses vs. defects

Defects	Defenses
Seizures	Fever
Cancer	Cough
Paralysis	Pain
Jaundice	Anxiety
Injury	Depression

65

99

Prof. Randolph Nesse– University of Michigan, USA

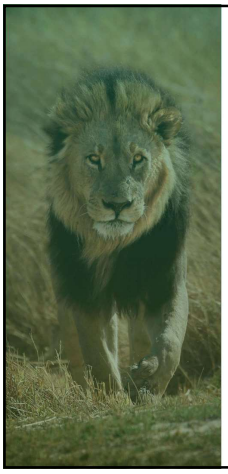
HSTalks

Panic disorder

A false alarm in the fight-flight response

66

100



How loud a noise before you flee?

Cost of fleeing
100 calories

Cost of not fleeing if there is a lion
100,000 calories

Optimum
Flee whenever p (lion) > 1/1000

999/1000 panic attacks will be unnecessary, **but normal**

67

101

The smoke detector principle

False alarms are essential

This is why we can block pain, anxiety, cough and fever safely

Except when the defense is needed

68

102

Prof. Randolph Nesse– University of Michigan, USA

Complex genetic diseases

Why didn't selection eliminate the alleles?

- Mutation selection balance
- Balancing selection
- Antagonistic pleiotropy

Genetic 'quirks' harmful only in modern environments

69

103

Highly heritable diseases are mostly caused by environments

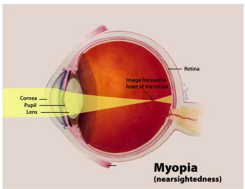
"Their widespread dispersion indicates that [common risk] alleles are benign… so if they are associated with disease the causal finger actually points to recent environmental change rather than primarily to genetic etiology"

Weiss, 2008, p1750

70

104

Myopia



h^2 = 0.8 but rapid increase

So why didn't natural selection eliminate these alleles?

Myopia was rare in hunter-gatherers so there was no selection against it

71

105

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Myopia

1950 2010

10% 80%

Light? Sugar? Reading?

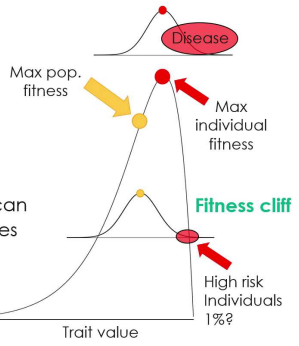
Jonas, J. B. *et al., Lancet.* 2016; **388**(suppl_1):S20

106

Cliff edge fitness functions

72

Cliff edged fitness functions can cause highly heritable diseases influenced by thousands of alleles with tiny effects

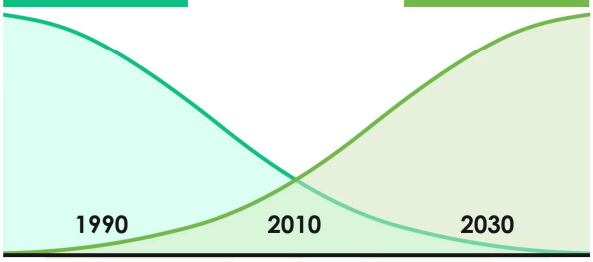


107

Evolutionary medicine progress

What is evolutionary medicine?

How can we teach it?



73

108

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Evolution: a basic science for medicine

Nesse, R. M. *et al., PNAS.* 2010; **107**(suppl_1):1800-1807 74

109

Evolution: a basic science for medicine

PNAS

Making evolutionary biology a basic science for medicine

Randolph M. Nesse[a,1], Carl T. Bergstrom[b], Peter T. Ellison[c], Jeffrey S. Flier[d], Peter Gluckman[e], Diddahally R. Govindaraju[f], Dietrich Niethammer[g], Gilbert S. Omenn[h], Robert L. Perlman[i], Mark D. Schwartz[j], Mark G. Thomas[k], Stephen C. Stearns[l], and David Valle[m]

[a]Departments of Psychiatry and Psychology, University of Michigan, Room 3018, East Hall, 530 Church Street, Ann Arbor, MI 48104; [b]Department of Biology, University of Washington, Seattle, WA 98195-1800; [c]Department of Human Evolutionary Biology, Harvard University, 11 Divinity Avenue, Cambridge, MA 02138; [d]Office of the Dean, Harvard Medical School, 25 Shattuck Street, Boston, MA 02115; [e]Centre for Human Evolution, Adaptation, and Disease Liggins Institute, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand; [f]Department of Neurology, Boston University School of Medicine, 72 East Concord Street, Boston, MA 02118; [g]Department of Hematology, Children's University Hospital, 72076 Tübingen, Germany; [h]Center for Computational

Nesse, R. M. *et al., PNAS.* 2010; **107**(suppl_1):1800-1807

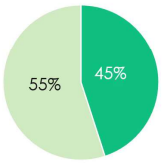
110

EvMed teaching

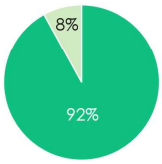
75

Grunspan, *et al.*, 2019

R1 schools with courses on evolution and medicine



R1 schools teaching evolution and medicine



Currently no medical school teaches evolutionary medicine as a basic science

It's going to take time but there is hope for the future

111

Prof. Randolph Nesse– University of Michigan, USA

HSTalks

Evolutionary medicine programs

76

112

Conclusion

01. Evolutionary biology is finally becoming a basic science for medicine

02. The over 50 Henry Stuart talks in this series offer a superb introduction

03. The International Society for Evolution, Medicine and Public Health offers more

For more, visit RandolphNesse.com and ISEMPH.org

For further information, please see the Links tab

77

113

HSTalks

By leading world experts

114