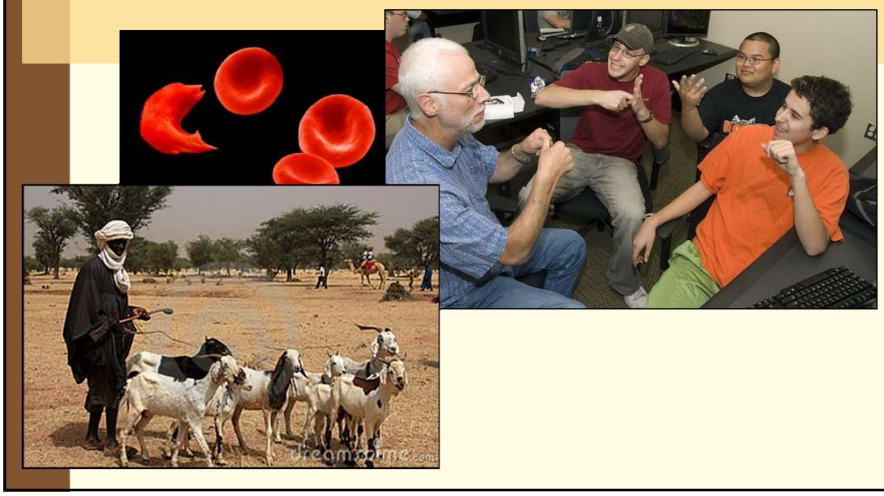
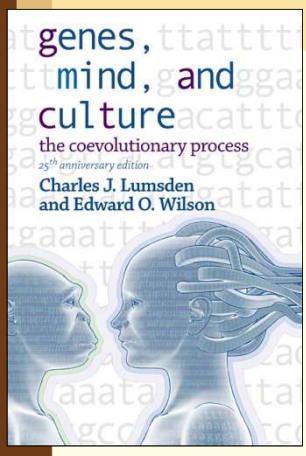
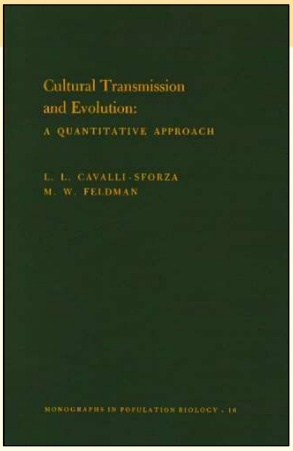
Gene-Culture Coevolution

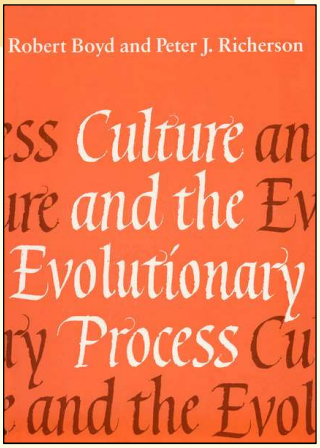


Gene-Culture Coevolution

Lumsden & Wilson 1981
Cavalli-Sforza & Feldman 1981
Boyd & Richerson 1985

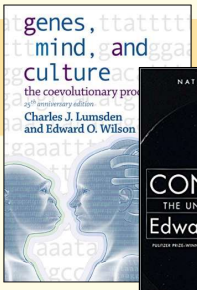




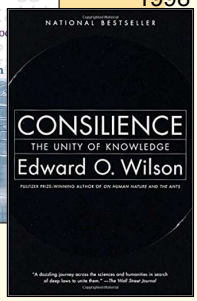


Gene-Culture Coevolution

Lumsden & Wilson 1981



1998



Sociobiology: a Darwinian approach to social behavior

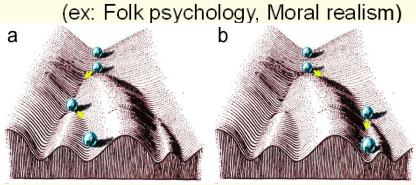
"Culturgens" & transition probabilities

Epigenetic rules:

Gene-directed processes necessary, in the presence of environmental events (especially the opportunity to learn from the culture), for the normal development of the brain and the mind.

Primary rules: brain devel. from sensory reception to early stages of perception;
Secondary rules: later stages of perception to conscious thought and experience;
Reification rules: which act in beliefs about the reality we imagine or conceive
(ex: Folk psychology, Moral realism)

Pure genetic transmission ("genetic robots")
X Pure cultural transmission ("tabula rasa")
X
Gene-cultural transmission:
"the potential field is bent into a landscape of hills and valleys"

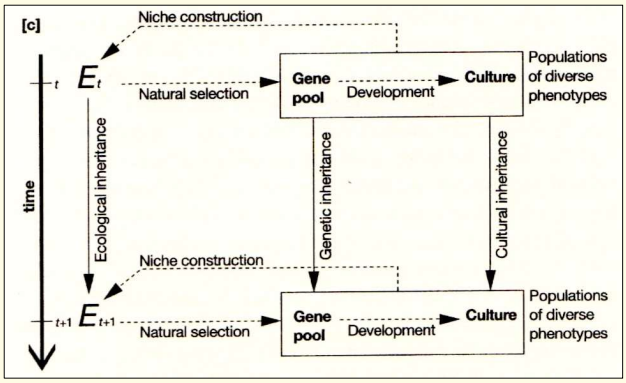


~ "epigenetic landscapes" (Waddington)

Niche Construction and Gene-Culture Coevolution

Cavalli-Sforza & Feldman 1981

Genetic Inheritance
+
Ecological Inheritance
+
Cultural Inheritance



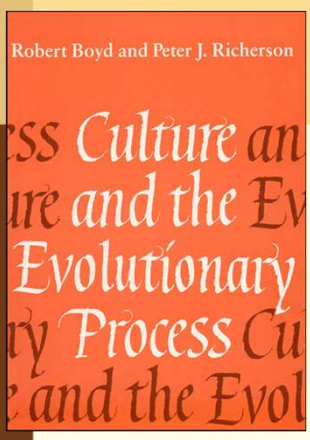
Odling-Smee, Laland & Feldman 2003

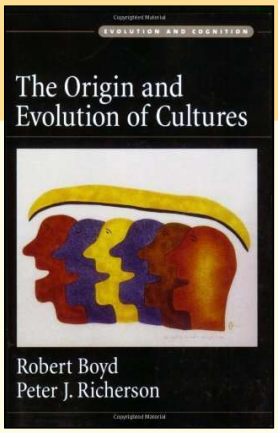
Gene-Culture Coevolution
Feldman & Laland 1996

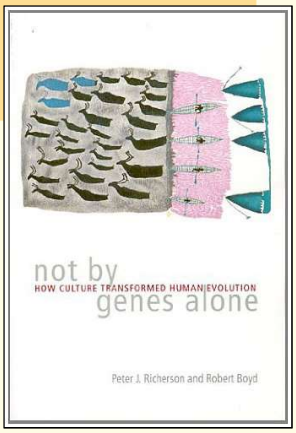
Gene-culture coevolutionary theory is a branch of **theoretical population genetics**, which, in addition to modeling the differential transmission of genes from one generation to the next, incorporates cultural traits into the analysis.

The two transmission systems cannot be treated independently, both because **what an individual learns may depend on its genotype**, and also because **the selection acting on the genetic system may be generated or modified by the spread of a cultural trait**.

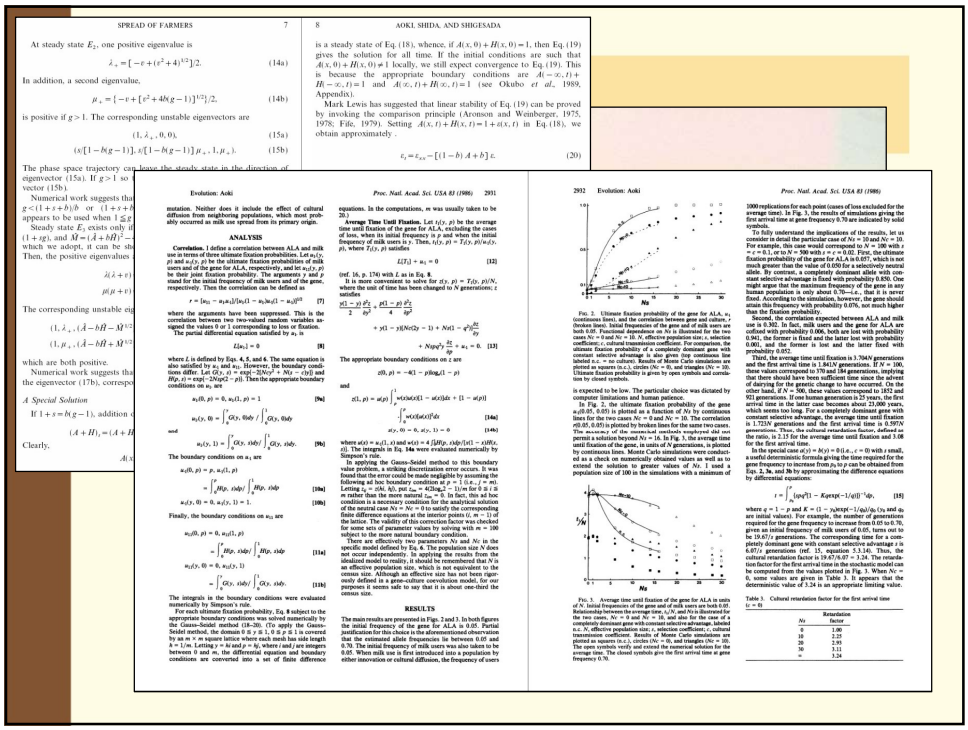
Gene-Culture Coevolution
Boyd & Richerson 1985







Boyd & Richerson (2005) Richerson & Boyd (2005)



Gene-Culture Coevolution

Feldman & Laland 1996

G-C Coevolutionary theory
X
Sociobiology, Human Behavioral Ecology, Evolutionary Psychology:

1. A population's culture is not regarded as largely dependent on either its **genetic constitution** or the prevailing pattern of **ecological resources**: culture as shared ideational phenomena (ideas, beliefs, values, knowledge) that are learned and socially transmitted between individuals.

2. Although the framework of gene-culture coevolutionary theory does not preclude an adaptationist perspective, (...) the gene-culture approach is minimalist in that assumptions about the adaptive importance of traits are not an obligatory step in the modeling exercise.

Gene-Culture Coevolution

Evolution in cultural x acultural species

1. Cultural transmission can modify selection pressures, thereby affecting the course of a population's evolution.

2. Culture can generate new evolutionary mechanisms (ex.: human cooperation and Cultural Group Selection)

3. The interplay between genetic and cultural transmission may produce time lags in the action of any selection operating on a trait.

4. Nonrandom associations between genes and cultural traits can occur that can significantly affect the genetic response to selection.

5. Because of its strong, homogenizing influence on behavior, and capacity for rapid diffusion, culture may some times generate atypically strong selection pressures, leading to very strong selection.

How culture shaped the human genome:
bringing genetics and the human sciences together

Laland, Odling-Smee & Myles 2010

Researchers from diverse backgrounds are converging on the view that human evolution has been shaped by gene–culture interactions.

Theoretical biologists have used population genetic models to demonstrate that cultural processes can have a profound effect on human evolution, and **anthropologists** are investigating cultural practices that modify current selection. These findings are supported by recent analyses of **human genetic variation**, which reveal that hundreds of genes have been subject to recent positive selection, often in response to human activities.

There is a considerable potential for **cross-disciplinary exchange** to provide novel insights into how culture has shaped the human genome.

Mathematical models of gene-culture coevolution
Laland, Odling-Smee & Myles 2010

Table 1 | **Mathematical models of gene–culture co-evolution**

Topic	Assumed gene function	Refs
The evolution of learning, social transmission and culture; the evolution of social learning strategies (unbiased transmission, direct bias, indirect bias, frequency dependent bias, and so on); the analysis of reliance on social learning; the evolution of teaching	Genes that affect learning; genes predisposing individuals to learn from others and to do so in particular ways or under particular circumstances, or to learn from particular individuals	19–21,23,33, 35,36,119–124
The co-evolution of genes for lactase persistence and milk use	Gene for adult human lactase persistence (*LCT*)	13,31,40
The evolution of language; the co-evolution of sign language and hereditary deafness	Language-facilitating genes (for example, forkhead box P2 (*FOXP2*)); genes for hereditary deafness	125–128
The inheritance of intelligence, behavioural and personality traits	Genes that affect personality and intelligence	38,39,54,129,130
The evolution of handedness and lateralized structures	Genes for lateralization of hand preference	37
The evolution of cooperation; the evolution of ethnic markers and conformity	Genes predisposing individuals to cooperate with in-group members, to not cooperate with or be hostile to out-group members, to punish non-cooperators, to express pro-social emotions, to internalize norms and to conform	21,131–138
The evolution of incest taboos and avoidance of sibling mating	Genes predisposing individuals to an aversion to mating with individuals with whom they are reared	139,140
Sexual behaviour; sexual selection with culturally transmitted mating preferences and genetically transmitted traits; culturally transmitted paternity beliefs and the evolution of human mating systems	Genes for skin, hair and eye colour, body and face shape, and facial and body hair; genes that affect degree of character|symmetry, degree of neoteny, level of aggressiveness, emotionality, personality traits, promiscuity, jealousy and faithfulness	42,94,141
The effects of sex-biased infanticide and parental investment; the effects of sex-selective abortion on sex-ratio evolution	Sex-ratio distorter genes	43,142,143
The evolutionary consequences of cultural niche construction	Genes related to metabolism, immunity and pathogen defence and the nervous system	45,46

Actual or hypothetical genes proposed to examine the dynamics of their co-evolution with cultural traits.

Gene-Culture Coevolution: *phenogenotypes*
Feldman & Laland 1996

In a gene-culture model, individuals must be described in terms of both their genotype and their cultural trait, a combination known as a "phenogenotype"

"Typically, it is assumed that the probability of an individual adopting a trait depends on whether its parents have that trait (vertical transmission), but equivalent models have been developed in which learning is from unrelated individuals (horizontal and oblique transmission), key individuals in the social group (indirect transmission), or the majority in the group (frequency-dependent transmission)".

Excess female mortality and Sex Ratios

Direct female infanticide, neglect and abandonment of daughters, differential allocation of resources toward sons, female-biased abortion x genetic variation distorting the human sex ratio (local **Adult Sex Ratios** up to 5mX1f).

Kumm, Laland & Feldman (1994): a gene-culture model to investigate how this **culturally generated excess of males** might influence the selection of distorter genes.

Cultural bias (x no bias) X genes affecting **Primary Sex Ratio**

(1 locus, 2 alleles): A_1A_1, A_1A_2, A_2A_2 X 2 "memes" (Bias/nB)

➡ 6 "fenogenotypes"
Propagation of pro-♂ bias + parental influence

Infanticide and human sex ratio
Kumm et al 1994, Laland et al 1995

In the case of **vertical transmission**, a pro-♂ cultural bias can spread either by cultural selection ("biased transmission" favouring the bias with one "biased" and one "unbiased" parent) or as a consequence of the prejudicial parental behaviour:

if "biased" parents kill more daughters, the bias frequency in the population will be higher among males - then more "biased" males are mated to "unbiased" females than the opposite; so if fathers are also more influential than mothers in the transmission of beliefs, the bias will spread.

PSR: Primary Sex Ratio (rate of births [*+biased abortions!*] m/f)
ASD: Adult Sex Ratio (m/f in the adult population)

[in the following example, SRs are plotted as prop. of ♂ (no distortion = 0,5)]

Infanticide and human sex ratio
Kumm et al 1994, Laland et al 1995

Fixed-adjustment: "biased" parents alter the PSR by a certain fixed amount (independently of the PSR), then compensate the killing by having more children; so, biased and unbiased couples have the same n of children, but biased couples have, on average, more of the preferred sex. BUT **if ASR > 0.5, less males marry than women (so "biased" couples have on average less *grandchildren*)**.

Variable-adjustment: parents use infanticide to bring the sex ratio to a desired value, and **may or not compensate the killings by having more children**. If they don't, **"biased" couples will have fewer children than "unbiased" couples** (= less reproductive fitness).

Infanticide and human sex ratio: A case study
Kumm et al 1994 /Laland et al 1995

TABLE 1
The Transmission of Cultural Phenotypes from Parent to Offspring

Mating	Offspring Phenotype		Adult Sex Ratio	
Father × Mother	Unbiased	Biased	Fixed-Adjustment	Variable-Adjustment
Unbiased × Unbiased	b_3	$\beta_3 = 1 - b_3$	m_{11}	m_{11}
Unbiased × Biased	b_2	$\beta_2 = 1 - b_2$	$m_{11} + d$	$(m_{11} + r)/2$
Biased × Unbiased	b_1	$\beta_1 = 1 - b_1$	$m_{11} + d$	$(m_{11} + r)/2$
Biased × Biased	b_0	$\beta_0 = 1 - b_0$	$m_{11} + 2d$	r

TABLE 2
Phenogenotype Frequencies for Both Sexes

Phenogenotype		Frequency	
Genotype	Phenotype	Male	Female
A_1A_1	Unbiased	x_1	y_1
	Biased	x_2	y_2
A_1A_2	Unbiased	x_3	y_3
	Biased	x_4	y_4
A_2A_2	Unbiased	x_5	y_5
	Biased	x_6	y_6

TABLE 3
Reproductive Fitnesses Associated with Matings in the Variable-Adjustment Model

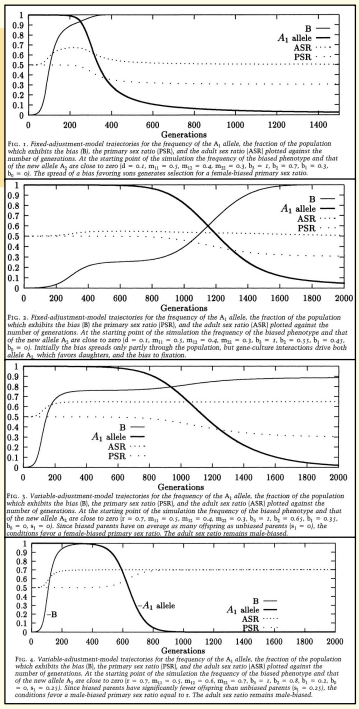
Parents	Genotype		
Male × Female	A_1A_1	A_1A_1	A_1A_1
Unbiased × Unbiased	1	1	1
Unbiased × Biased	f_{11}	f_{12}	f_{22}
Biased × Unbiased	f_{11}	f_{12}	f_{22}
Biased × Biased	F_{11}	F_{12}	F_{22}

Gene-Culture Coevolution

Infanticide and human sex ratio
Kumm et al 1994

When the behavior of a couple alters the mortality ratio favoring one sex, but does not change the number of offspring produced, the Primary Sex Ratio will evolve with a **bias against the favored sex.**

But when the total number of offspring is significantly reduced as a consequence of the prejudice, the PSR will evolve **in favor of the preferred sex.**



Infanticide and human sex ratio
Kumm et al 1994, Laland et al 1995

Effect of cultural bias diffusion on genes:

Four scenarios of bias spread, starting with no cultural bias and 0.5 PSR (2 Fixed-Adj., 2 Variable-Adj.): in all of them, the ASR increases as the bias spread, but in some cases (1&2), it eventually returns to 0.5 – because genetic biases favouring females compensate the cultural bias favouring males.

If the cultural bias towards males favours a genetic bias in the same direction (in the case of Variable-adjustment WITHOUT reposition), PRS and ASR > 0.5 will stabilize (eventually, no need of differential infanticide…)

Infanticide and human sex ratio
Kumm et al 1994, Laland et al 1995

Fixed-adjustment: parents with A_2 alleles (♀-distorting) will have more daughters (= + grandchildren, not all ♂ marry), so PSR < 0.5 compensates "killing"-induced raise of ASR.

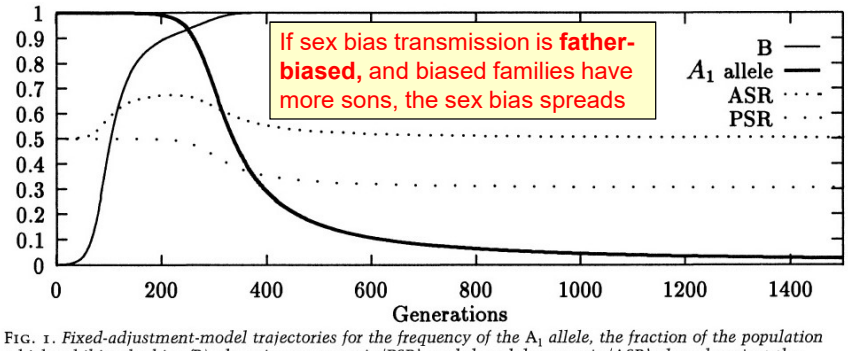


FIG. 1. *Fixed-adjustment-model trajectories for the frequency of the A_1 allele, the fraction of the population which exhibits the bias (B), the primary sex ratio (PSR), and the adult sex ratio (ASR) plotted against the number of generations. At the starting point of the simulation the frequency of the biased phenotype and that of the new allele A_2 are close to zero ($d = 0.1$, $m_{11} = 0.5$, $m_{12} = 0.4$, $m_{22} = 0.3$, $b_3 = 1$, $b_2 = 0.7$, $b_1 = 0.3$, $b_0 = 0$). The spread of a bias favoring sons generates selection for a female-biased primary sex ratio.*

Infanticide and human sex ratio
Kumm et al 1994, Laland et al 1995

Fixed-adjustment, **initial partial transmission** of pro-♂ bias, but **same long-term consequences** on PSR and ASR

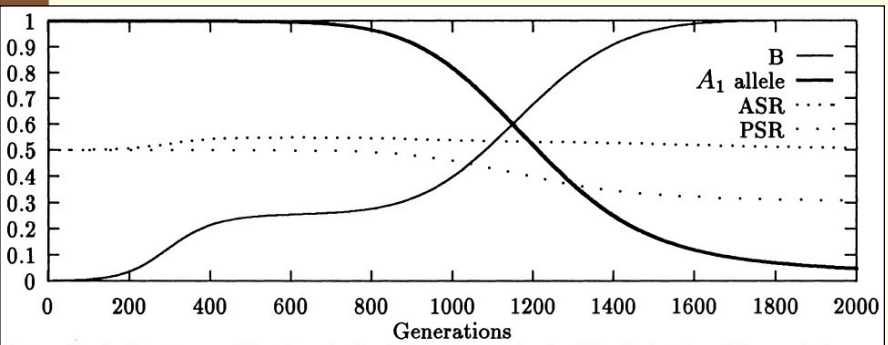


FIG. 2. *Fixed-adjustment-model trajectories for the frequency of the A_1 allele, the fraction of the population which exhibits the bias (B) the primary sex ratio (PSR), and the adult sex ratio (ASR) plotted against the number of generations. At the starting point of the simulation the frequency of the biased phenotype and that of the new allele A_2 are close to zero ($d = 0.1$, $m_{11} = 0.5$, $m_{12} = 0.4$, $m_{22} = 0.3$, $b_3 = 1$, $b_2 = 0.55$, $b_1 = 0.45$, $b_0 = 0$). Initially the bias spreads only partly through the population, but gene-culture interactions drive both allele A_2 which favors daughters, and the bias to fixation.*

Infanticide and human sex ratio
Kumm et al 1994, Laland et al 1995

Variable-adjustment with compensation. "Biased" and "unbiased" couples have the same n of children, so those with (♀-distorting) A_2 alleles will have more fitness due to ASR > 0.5, but as biased parents adjust the SR more and more, ASR remains ♂-biased.

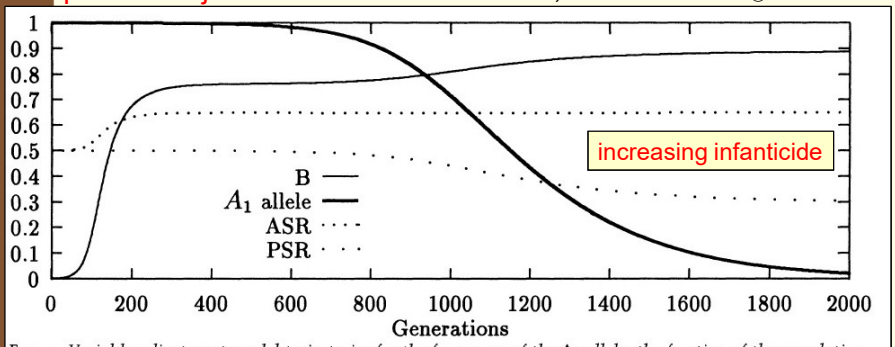


FIG. 3. *Variable-adjustment-model trajectories for the frequency of the A_1 allele, the fraction of the population which exhibits the bias* (B), *the primary sex ratio* (PSR), *and the adult sex ratio* (ASR) *plotted against the number of generations. At the starting point of the simulation the frequency of the biased phenotype and that of the new allele A_2 are close to zero* ($r = 0.7$, $m_{11} = 0.5$, $m_{12} = 0.4$, $m_{22} = 0.3$, $b_3 = 1$, $b_2 = 0.65$, $b_1 = 0.35$, $b_0 = 0$, $s_1 = 0$). *Since biased parents have on average as many offspring as unbiased parents* ($s_1 = 0$), *the conditions favor a female-biased primary sex ratio. The adult sex ratio remains male-biased.*

Infanticide and human sex ratio
Kumm et al 1994, Laland et al 1995

Variable-adjustment WITHOUT reposition. Once the pro-♂ bias spreads, parents whose genes induce a ♀–distorted PSR will kill more daughters and have less offspring (less fitness), so any ♂-PSR-distorting alleles will spread.

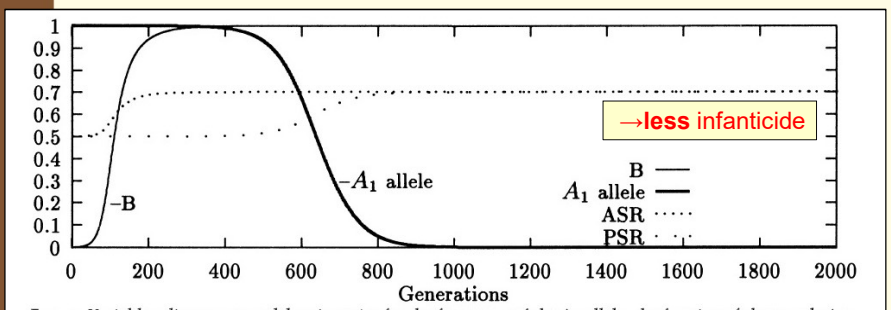


FIG. 4. *Variable-adjustment-model trajectories for the frequency of the A_1 allele, the fraction of the population which exhibits the bias* (B), *the primary sex ratio* (PSR), *and the adult sex ratio* (ASR) *plotted against the number of generations. At the starting point of the simulation the frequency of the biased phenotype and that of the new allele A_2 are close to zero* ($r = 0.7$, $m_{11} = 0.5$, $m_{12} = 0.6$, $m_{22} = 0.7$, $b_3 = 1$, $b_2 = 0.8$, $b_1 = 0.2$, $b_0 = 0$, $s_1 = 0.25$). *Since biased parents have significantly fewer offspring than unbiased parents* ($s_1 = 0.25$), *the conditions favor a male-biased primary sex ratio equal to* r. *The adult sex ratio remains male-biased.*

Cultural Transmission in a Demographic Study of Sex Ratio at Birth in China's Future

Li, Feldman & Li 2000

The sex ratio at birth (SRB[~/x PSR]) is *the ratio of live male to female births*, and its **historical values** for humans have been close to **1.05** in large populations (Chahnazarian, 1988).

In recent years, **values of SRB significantly higher than 1.05 were reported in countries with declining fertility and strong son preference such as China, India, Bangladesh, and South Korea** (Coale, 1992; Das Gupta, 1987; Hull, 1990; Johansson and Nygren, 1991; Park and Cho, 1995), constituting about half of the world's population.

The most obvious consequence of high SRB is an imbalance between the sexes in future marriage opportunities. For a value of SRB of 1.14 in China in 1989, about 8% “excess” males at first marriage was projected in the years around 2015 by Tuljapurkar et al. (1995), and this was confirmed by Zeng et al. (1997).

Oct. 2015

Taxi Tehran takes to the road Plus Peter Bradshaw's verdict

The Farsi & the furious

What's the beef? Drake and the history of rap feuds

Reviews Rod Stewart ★★

Marina Hyde's Lost in Showbiz Robin and Pharrell: Thicke as two short planks

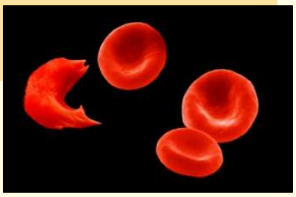
theguardian

China abandons one-child policy

- Couples will be allowed to have two children
- Officials hope move will combat ageing population
- Activists argue the change does not go far enough

Chilcot report into Iraq will finally appear next summer

Gene-Culture Coevolution and Cultural Niche Construction: case studies



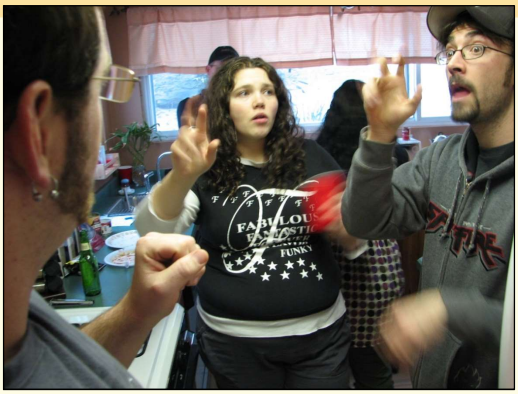
Agriculture, malaria and Sicle-Cell Disease
Jackson 1996

Pastoralism and adults' lactose absorption
Aoki 1986

Hereditary deafness and Sign Language
Aoki & Feldman 1991

Hereditary deafness & sign language

Genetic + Cultural vertical transmission



Aoki & Feldman (1991). Recessive hereditary deafness, assortative mating and persistence of a sign language. *Theor Popul Biol* 39: 358-372.

Feldman & Aoki (1992). Assortative mating and grandparental transmission facilitate the persistence of a sign language. *Theor Popul Biol* 42:107-16.

Hereditary deafness & sign language

Aoki & Feldman (1991) Recessive hereditary deafness, assortative mating and persistence of a sign language. *Theor Popul Biol* 39: 358-372.

Model:
Cultural transmission of sign language
One-locus genetic variation for deafness and hearing

Premises:
(1) deaf are more motivated to learn sign language than the hearing;
(2) a vertically transmitted sign language cannot "jump a generation" (unlike recessive hereditary deafness)

Results:
Conditions for persistence (x loss) of signers more easily satisfied
(1) the greater the fraction of the hearing who also learn sign language;
(2) as the frequency of the recessive gene for deafness increases;
(3) persistence also facilitated by assortative mating for deafness (but not by assortment for signing)
With vertical transmission only, it is necessary that one signer parent be able to transmit sign language with greater than one-half the efficiency of two.

Hereditary deafness & sign language

Aoki & Feldman (1991) Recessive hereditary deafness, assortative mating and persistence of a sign language. *Theor Popul Biol* 39: 358-372.

Under the assumption that the hearing do not learn sign language, the following additional results are obtained:

SL Persistence is more likely with dominant as opposed to recessive inheritance.

When **recessive hereditary** and ***acquired*** **deafness** co-occur, increasing the frequency of the latter has opposite effects depending on the degree of assortment.

Opportunities for the deaf to learn sign language outside the family seem not to affect the conditions for persistence.

Hereditary deafness & sign language

Feldman & Aoki (1992). Assortative mating and grandparental transmission facilitate the persistence of a sign language. *Theor Popul Biol 42*:107-16

Conditions for the persistence (i.e., protection from loss) of a sign language are investigated assuming monogenic recessive inheritance of deafness, assortative mating for deafness or hearing, and cultural transmission of the sign language to deaf individuals from their deaf parents and deaf maternal grandparents.

A new method is introduced to deal with the problem of grandparental transmission in which the basic variables are the frequencies of triplets comprising a mother, a father, and their daughter of permissible phenogenotypes. Usual stability analysis is then done on the system of linear recursions in the frequencies of these triplets, derived on the assumption that signers (users of the sign language) are rare. It is shown that assortative mating is the most important factor contributing to persistence, but that grandparental transmission can also have a significant effect when assortment is as strong as observed in England and the United States.

Hereditary deafness & sign language

Conditions for persistence of sign language:
Fraction of the hearing who also learn sign language
Change in the frequency of the recessive gene for deafness.
Assortative mating for deafness (but not assortment for signing)
Recessive genes: deafness may “jump a generation” (and vertical transmission may not avoid sign language loss): grandmaternal transmission may compensate inefficient vertical transmission.
(model: vertical only, but)
Schools for deaf and assortative mating.

Empiric (genetic) evidence supporting the model

Arnos, Welch, Tekin et al (2008). A comparative analysis of the genetic epidemiology of deafness in the US in two sets of pedigrees collected more than a century apart. *Am J Hum Genet* 83:200–207.
Nance, Liu & Pandya (2000). Relation between choice of partner and high frequency of connexin-26 deafness. *Lancet* 356:500–501.

Hereditary deafness & sign language

Gialluisi et al (2013)

Aoki and Feldman argued that, in the presence of a single monogenic form of recessive deafness in a population, and under the assumption that no hearing individuals learn to sign, the condition for the persistence of a sign language is ensured by the assortative mating for deaf status and the vertical transmission of the sign language, whereas the contribution of oblique and horizontal transmission to the persistence of a sign language is considered negligible.

This model has been recently supported by work on the genetic epidemiology of DFNB1 deafness, highlighting the role of assortative mating in maintaining its high prevalence The DFNB1 locus is made up of two different genes, GJB2 and GJB6, coding for two gap junction proteins highly expressed in the cochlea (Connexin 26 and 30, respectively). The close proximity of the GJB2 and GJB6 genes within the genome, and the co-ordinated regulation of their expression in the inner ear, means that DFNB1 deafness can be considered in many respects as a single, monogenic recessive disorder.

Nance et al compared different US national surveys on deaf-by-deaf marriages to corroborate the hypothesis that the frequency of DFNB1 deafness among the US population has substantially increased over the last two centuries, in parallel with the spread of sign language schools.

Hereditary deafness & sign language

Gialluisi et al (2013)

OTHER CASES, THOUGH, DO NOT FIT THE MODEL:

Al-Sayyid Bedouin Sign Language system (ABSL).
A highly inbred and endogamous community of around 3500 individuals living in the Negev desert in Israel (founded around 200y ago). Consanguineous marriages are the norm (cultural isolation, high endogamy, polygamy, high birthrates). Founder effect: high prevalence of a profound recessive deafness because of mutations in the DFNB1 locus.

The cultural isolation of this community has resulted in the birth of a characteristic sign language. In contrast with other young sign languages, such as Nicaraguan sign language, ABSL is developing in a socially stable community and is widely used in the population, also by hearing people. Given the cultural habits of Bedouins, which include widespread polygamy and extended family households, **many children are exposed to ABSL very early in their childhood, regardless of hearing status**. All of these anthropological factors have surely contributed to the spread of ABSL also to many hearing individuals.

Although deafness and signing coincide in Western communities, this co-segregation is typically not observed in village communities like Al-Sayyid Bedouins.

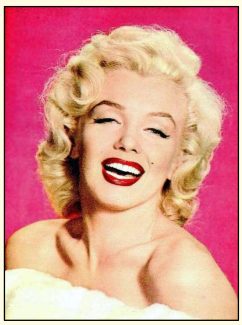
The predictions made by Aoki and Feldman can apply only in part to the ABSL system.

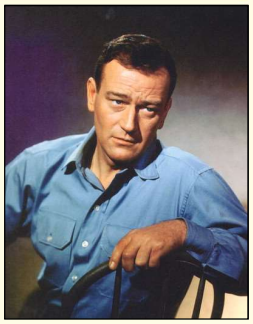
Gene-Culture Coevolution
Feldman & Laland 1996

Other G-C Coevolution studies:

- Laland et al. (1995): gene-culture model of handedness.
- Aoki et al (1996): conditions under which the spread of farming will generate gene frequency clines.
- Soltis, Boyd and Richerson (1995) data on rates of population extinction among New Guinea communities used to test Boyd and Richerson' s group selection hypothesis (see next lecture).
- Laland (1994) model of sexual selection: society-specific correlations for anatomical traits and learned preferences for such traits in the opposite sex.

Sexual selection with a culturally transmitted mating preference (Laland 1994)





Genes that are associated with our externally visible phenotypes show among the strongest signatures of local adaptation.(...) Although some variation can be attributed to natural selection, many of these selective events could potentially be explained through a form of sexual selection in which society-specific culturally learned mating preferences favour biological traits in the opposite sex.
(Laland et al 2010)

Sexual selection with a culturally transmitted mating preference (Laland 1994)

Mathematical model combining sexual selection and gene–culture co-evolutionary theory. **Culturally generated sexual selection** was found to be faster and more potent than its gene-based counterpart. Given the pervasiveness of cultural influences on human mating preferences, social transmission may exert a powerful influence on the selection of secondary sexual characteristics and other physical and personality traits that affect human mate choice.

The hypothesis leads to several predictions:

1. we should expect to see mate-choice copying and the social transmission of mating preferences in humans, predictions that have received recent support.
2. genes that affect such sexually selected traits should show evidence of recent selection, which seems to be the case.
3. there should be population-wide correlations between specific culturally transmitted preferences and gene-based traits in both sexes.

How culture shaped the human genome
Laland, Odling-Smee & Myles 2010

Empirical evidence:
Thicker hair & cultural sexual selection in East Asians?

Bryk, J. *et al.* Positive selection in East Asians for an *EDAR* allele that enhances NF-κB activation. *PLoS ONE* **3**, e2209 (2008):

EDAR allele driven to high frequency in East Asia by positive selection prior to 10,000 years ago. These observations lead us to question why thicker hair may have been advantageous in ancestral East Asian environments. Of course, thicker hair may not have been adaptive at all and may simply be the result of phenotypic hitchhiking: selection on 370A may have targeted a different phenotype and hair thickness may have resulted as a by-product of this selection. Sexual selection also remains a possibility.

How culture shaped the human genome
Laland, Odling-Smee & Myles 2010

Other anthropological evidence of G-C Coevolution

- Spread of genes that confer resistance to crowd diseases;
- Coevolution of diet and genes conferring resistance to disease (animal pathogens etc);
- Coevolution of cooking with genes that are expressed in the brain and digestive tract and involved in the determination of tooth size;
- Coevolution of culturally facilitated dispersal and pigmentation.

How culture shaped the human genome
Laland, Odling-Smee & Myles 2010

Genetic evidence for gene–culture co-evolution

Genome scans have provided the first steps in evaluating without bias the relative contribution of gene–culture co-evolution to human adaptation.

Mathematically minded geneticists have developed methods for detecting statistical signatures in the human genome of recent, strong positive selection — genes that have been favoured by natural selection over the past 100,000 years.

A reasonable reading of the data suggests that somewhere between a few hundred and a couple of thousand regions in the human genome have been shaped by recent selection. BUT in the vast majority of cases it has yet to be proven that the source of selection on the gene is derived from a cultural practice.

How culture shaped the human genome
Laland, Odling-Smee & Myles 2010

Genes subjected to recent rapid selection: cultural pressures?

Table 2 | **Genes identified as having been subject to recent rapid selection and their inferred cultural selection pressures**

Genes	Function or phenotype	Inferred cultural selection pressure	Refs
LCT, MAN2A1, SI, SLC27A4, PPARD, SLC25A20, NCOA1, LEPR, LEPR, ADAMTS19, ADAMTS20, APEH, PLAU, HDAC8, UBR1, USP26, SCP2, NKX2-2, AMY1, ADH, NPY1R, NPY5R	Digestion of milk and dairy products; metabolism of carbohydrates, starch, proteins, lipids and phosphates; alcohol metabolism	Dairy farming and milk usage; dietary preferences; alcohol consumption	6,7,16,41,63, 102,118, 144,145
Cytochrome P450 genes (*CYP3A5, CYP2E1, CYP1A2* and *CYP2D6*)	Detoxification of plant secondary compounds	Domestication of plants	6,63,146,147
CD58, APOBEC3F, CD72, FCRL2, TSLP, RAG1, RAG2, CD226, IGJ, TJP1, VPS37C, CSF2, CCNT2, DEFB118, STAB1, SP1, ZAP70, BIRC6, CUGBP1, DLG3, HMGCR, STS, XRN2, ATRN, G6PD, TNFSF5, HbC, HbE, HbS, Duffy, α-globin	Immunity, pathogen response; resistance to malaria and other crowd diseases	Dispersal, agriculture, aggregation and subsequent exposure to new pathogens; farming	6–8,14,16,50, 63,148,149
LEPR, PON1, RAPTOR, MAPK14, CD36, DSCR1, FABP2, SOD1, CETP, EGFR, NPPA, EPHX2, MAPK1, UCP3, LPA, MMRN1	Energy metabolism, hot or cold tolerance; heat-shock genes	Dispersal and subsequent exposure to novel climates	14,150
SLC24A5, SLC25A2, EDAR, EDA2R, SLC24A4, KITLG, TYR, 6p25.3, OCA2, MC1R, MYO5A, DTNBP1, TYRP1, RAB27A, MATP, MC2R, ATRN, TRPM1, SILV, KRTAPs, DCT	The externally visible phenotype (skin pigmentation, hair thickness, eye and hair colour, and freckles)	Dispersal and local adaptation and/or sexual selection	9,14,63,97, 101,151
CDK5RAP2, CENPJ, GABRA4, PSEN1, SYT1, SLC6A4, SNTG1, GRM3, GRM1, GLRA2, OR4C13, OR2B6, RAPSN, ASPM, RNT1, SV2B, SKP1A, DAB1, APPBP2, APBA2, PCDH15, PHACTR1, ALG10, PREP, GPM6A, DGKI, ASPM, MCPH1, FOXP2	Nervous system, brain function and development; language skills and vocal learning	Complex cognition on which culture is reliant; social intelligence; language use and vocal learning	6,7,14,63, 68–70,78,149
BMP3, BMPR2, BMP5, GDF5	Skeletal development	Dispersal and sexual selection	6,63
MYH16, ENAM	Jaw muscle fibres; tooth-enamel thickness	Invention of cooking; diet	80,113

Function or phenotype	Inferred cultural selection pressure
Digestion of milk and dairy products; metabolism of carbohydrates, starch, proteins, lipids and phosphates; alcohol metabolism	Dairy farming and milk usage; dietary preferences; alcohol consumption
Detoxification of plant secondary compounds	Domestication of plants
Immunity, pathogen response; resistance to malaria and other crowd diseases	Dispersal, agriculture, aggregation and subsequent exposure to new pathogens; farming
Energy metabolism, hot or cold tolerance; heat-shock genes	Dispersal and subsequent exposure to novel climates
The externally visible phenotype (skin pigmentation, hair thickness, eye and hair colour, and freckles)	Dispersal and local adaptation and/or sexual selection
Nervous system, brain function and development; language skills and vocal learning	Complex cognition on which culture is reliant; social intelligence; language use and vocal learning
Skeletal development	Dispersal and sexual selection
Jaw muscle fibres; tooth-enamel thickness	Invention of cooking; diet

How culture shaped the human genome
Laland, Odling-Smee & Myles 2010

Overrepresented categories of genes subject to positive selection

Pathogen response
Immune response
Genetic responses to changes in diet:

Amylase and rates of starch consumption:
agricultural societies + hunter-gatherers in arid environments

Jaw musculature genes and cooking

Selection in the **alcohol dehydrogenase (ADH) cluster in East Asians** (one argument is that hypersensitivity to alcohol has been adaptive through protecting against alcoholism).

How culture shaped the human genome
Laland, Odling-Smee & Myles 2010

Implications for future research

Gene–culture co-evolution may have been widespread, and acted on many human traits, throughout the history of our species. However, this conclusion would be premature. Of the regions of the genome that have been identified as being subject to recent selection, few causal variants have been confirmed or linked to an adaptive phenotype.
It is now an empirical issue to determine the extent of G-C coevolution in humans.
The case for gene–culture co-evolution does not rest exclusively on genetic data. The models, together with the anthropological data, generate a large number of testable hypotheses and predictions, some of which have already been confirmed.
A gene–culture co-evolutionary perspective predicts that the genetic signatures of recent positive selection (for example, since the Out-of-Africa expansion) will more often have been generated by culture than signatures of selection from earlier time periods in human evolution.

Gene-culture coevolution in killer whales

Foote & al (2016). Genome-culture coevolution promotes rapid divergence of killer whale ecotypes. *Nature Communications* 7:1693

Killer whales (*Orcinus orca*)

The largest species in the dolphin family and one of the most cosmopolitan mammals (found in all ocean basins and distributed from the Antarctic to the Arctic).

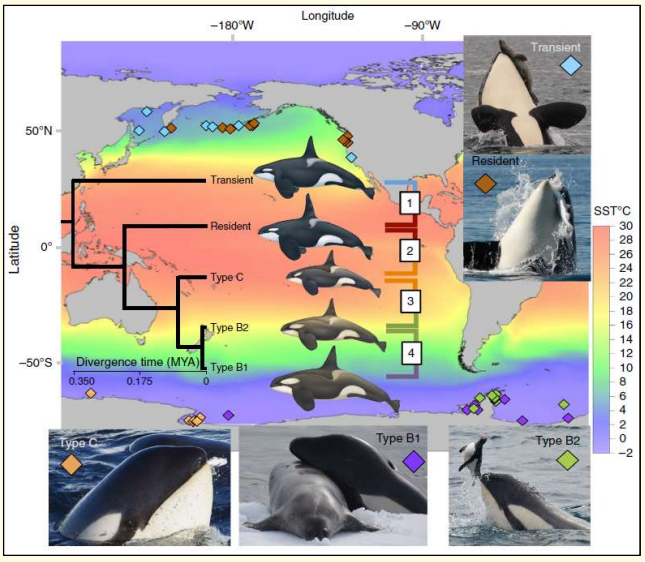
Top marine predator: diverse range of prey species: birds, fish, mammals and reptiles.

Highly stable matrilineal group structure, long postmenopausal lifespan.

Specialized **ecotypes**, with hunting strategies adapted to exploit narrow ecological niches (radiated within less than 250K years).

Gene-culture coevolution in killer whales

Foote & al (2016)



Ecotypes

Gene-culture coevolution in killer whales

Foote & al (2016)

Genetic structuring including the segregation of potentially functional alleles is associated with socially inherited ecological niche. Reconstruction of ancestral demographic history revealed bottlenecks during founder events, likely promoting ecological divergence and genetic drift resulting in a wide range of genome-wide differentiation between pairs of allopatric and sympatric ecotypes.

Evidence for regional **genomic divergence associated with habitat, dietary preferences** and **post-zygotic reproductive isolation**.

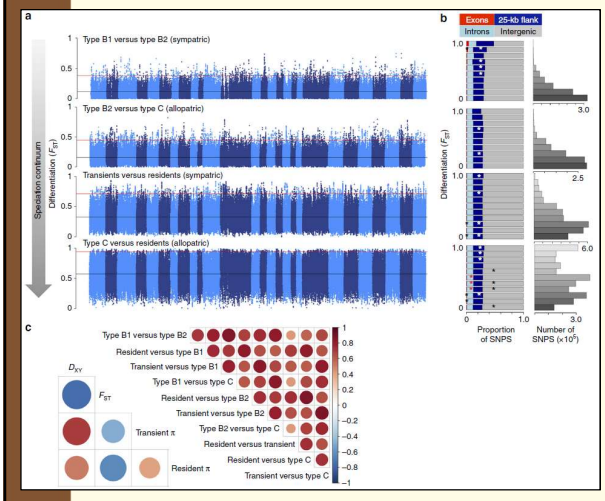
Findings consistent with expansion of small founder groups into novel niches by an initial plastic behavioural response, perpetuated by social learning imposing an altered natural selection regime.

An important step towards an understanding of the complex interaction between demographic history, culture, ecological adaptation and evolution at the genomic level.

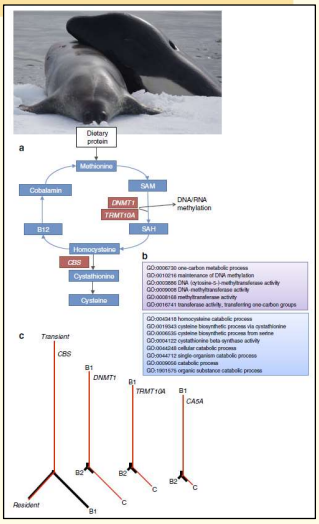
Gene-culture coevolution in killer whales

Foote & al (2016)

Genomic signatures of climate and diet adaptation



Genome-wide distribution of differentiation



Signatures of selection in mammal-eating ecotypes in genes that play a key role in the methionine cycle

Gene-culture coevolution in killer whales

Foote & al (2016)

A highly stable matrilineal group structure and a long postmenopausal lifespan in killer whales is thought to facilitate the transfer of ecological and social knowledge from matriarchs to their kin, and thereby perpetuate the stability of ecotypic variation in killer whales.

Whole-genome resolution confirms that, even in sympatry, contemporary gene flow occurs almost exclusively among individuals of the same ecotype, allowing genomic differentiation to build up between ecotypes so that within an ocean basin ecological variation better predicted genetic structuring than geography.

Gene-culture coevolution in killer whales

Foote & al (2016)

The processes underlying genomic divergence among killer whale ecotypes reflect those described in humans in several respects.

- Behavioural adaptation has facilitated the colonization of novel habitats and ecological niches.
- Founder effects and rapid formation of reproductive isolation, followed by population expansion, have promoted genome-wide shifts in the frequency of alternative alleles in different ecotypes due to genetic drift.
- Demographic changes during founder events and subsequent expansions can also influence cultural diversity, and may have had a role in reducing within-ecotype cultural diversity and promoting cultural differentiation between ecotypes.

Gene-culture coevolution in killer whales

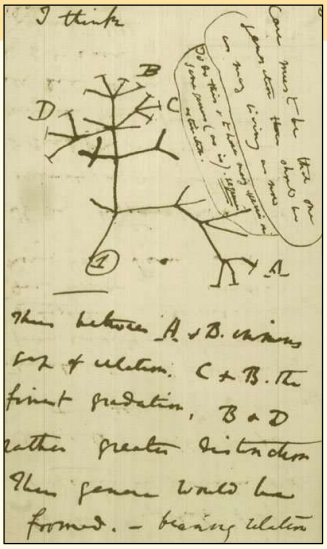
Foote & al (2016)

As with studies on modern humans, it is difficult to demonstrate a causal association between cultural differences and selection on specific genes; however, our findings of divergence in genes with putative functional association with diet, climate and reproductive isolation broadly imply an interaction between genetically and culturally heritable evolutionary changes in killer whale ecotypes. Given these findings, the almost-exclusive focus on humans by studies of the interaction of culture and genes should be expanded, and exploration of culture–genome coevolution models in suitable non-human animal systems encouraged.

Darwinian Models of Cultural Evolution

Cultural Selection:

- Variation
- Inheritability
- Diferential fitness



Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

Debates about the utility of "memes" have revealed some fundamental misunderstandings about the nature of cultural evolution.

Memeticists and their many critics seem to share the view that evolutionary principles can only be applied to cultural evolution if culture can be thought of as arising from the transmission of gene-like replicators. The memeticists believe that such particles (or at least close approximations) exist, and thus Darwinian reasoning -which has proven so useful in biology - can be applied to culture.

Their critics argue that replicating particles do not exist, and therefore, that it is inappropriate to apply Darwinian ideas to culture.

Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

While we think that culture is clearly a Darwinian process, we argue that **both camps have been misguided by an overly enthusiastic analogy between genes and culture.**

Population-dynamic concepts and evolutionary models are extremely useful for understanding how such processes work. But we maintain that constructing appropriate models of cultural evolution demands that close attention be paid to the psychological and social processes involved.

Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

Recurrent misunderstandings about cultural evolution

1. Mental representations are rarely discrete, and therefore models that assume discrete, gene-like particles (i.e., replicators) are useless (Atran 2001).

2. Replicators are necessary for cumulative, adaptive evolution (Dawkins1976, 1982).

3. Content-dependent psychological biases are the only important processes that affect the spread of cultural representations (Sperber 1996).

4. The "cultural fitness" of a mental representation can be inferred from its successful transmission through the population.

5. Selection can only occur if the sources of variation are random (Pinker 1997)

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Five Misunderstandings About Cultural Evolution
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1. Discrete replicator models of cultural inheritance can be useful even if mental representations are never discrete

Memeticists (Blackmore 1999, Aunger 2002): memes must be particulate for cumulative cultural change to occur.

Cultural evolutionary theorists: models of cultural evolution in which cultural traits are assumed to be discrete (BUT also of the evolution of continuous [non-discrete] cultural traits).

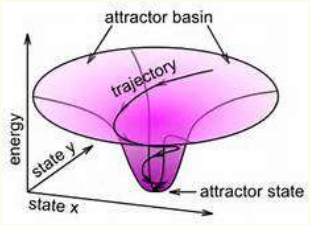
X

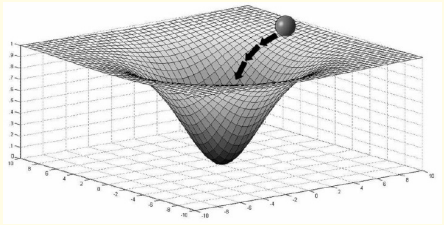
Cognitive anthropologists:
critique of the "replicator approaches"

Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

Atran (2001, 2002): no evidence that the mental representations underpining cultural traits are discrete/gene-like entities: mental representations are continuously graded entities.

Sperber (1996), Atran (2001), and Boyer (1999): ideas are not transmitted intact from one brain to another: mental representations in one brain generate observable behavior, a "public representation". No guarantee that the mental representation in the second brain is the same as in the first. Representations favored by processes of psychological inference (including storage and retrieval) = "cognitive attractors." (Sperber).





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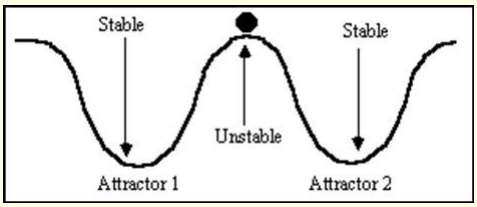
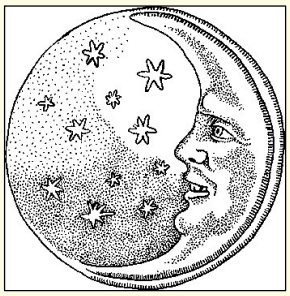


BUT

Models which assume discrete replicators that evolve under the influence of natural-selection-like forces can be useful ***because*** of the action of strong **cognitive attractors** during the social learning. **Instead of a continuum of cultural variants, most people will hold a representation near an attractor.**

Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

Ex (Henrich and Boyd 2002): individuals' beliefs about the moon:



X

x=0: the moon as a self-aware, conscious entity with goals, emotions, and motivations (thus the moon's behavior can be understood using folk psychology).

x=1: the moon as a big rock, lacking goals, consciousness, and emotions; the moon's color, shape, and movement are attributed to the effects of non-agentic interactions with light and the gravity of other mindless bodies, governed by physical laws that operate throughout the universe.

Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

Moon-concepts that mix these poles (0<x<1)?.

One could believe, for example, that the moon's movement and shape are out of its control (governed by physical laws), while its color or hue expresses its mood, which in turn influences the weather. Or, perhaps the moon's color is 23% controlled by its emotions and 77% controlled by the laws of light refraction. One might also believe that on Tuesdays and Thursdays the moon is a goal-oriented agent; on Mondays, Wednesdays, and Fridays the moon is a big rock; and on the weekends these two alternate minute by minute. (…) ***In contrast to intermediate concepts (x values), x=1 or 0 is "easier to think"***

Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

1. Discrete replicator Models of Cultural Inheritance can be Useful Even if Mental Representations are Never Discrete

Individuals acquire their mental representations by observing the behavior of others. Two cognitive mechanisms affect this learning process:

1. Inferential transformation (the manner in which cognitive processes of acquisition, storage, and retrieval alter mental representations in ways to favor some representations over others [cognitive attractors]);
2. Selective attention (the tendency for individuals to pay particular attention to some individuals more than others)

Effects of inferential transformation dominate the early part of the trajectory, rapidly causing almost everyone to have a representation close to one of the two attractors. Once everyone is clustered around one of the two attractors, the rest of the trajectory is dominated by the effects of selective attention.
As long as there are multiple attractors, the resulting population dynamics and the final distribution of mental representations are closely approximated by a discrete-trait replicator dynamics model in which the discrete traits are the strong attractor locations.

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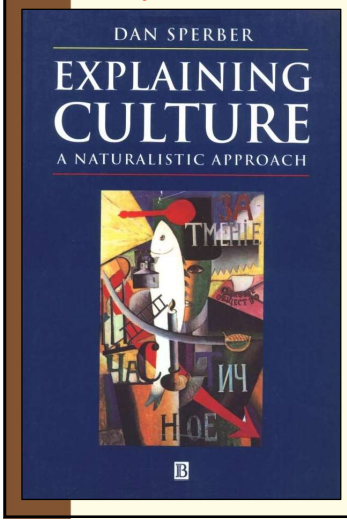
2. Replicators are not Necessary for Cumulative, Adaptive Cultural Evolution



Dawkins: self-replicating entities are a requirement for cumulative evolution and must have the following characteristics:
Fidelity. The copying must be sufficiently accurate that even after a long chain of copies the replicator remains almost unchanged.
Fecundity. At least some varieties of the replicator must be capable of generating more than one copy of themselves.
Longevity. Replicators must survive long enough to affect their own rate of replication.

X

Sperber (1996):
"The Epidemiology of Representations"

Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

Replicators: sufficient but NOT necessary for cumulative adaptive evolution: any process of cultural transmission that leads to accurate replication of the average characteristics of the population will work.

EX.1: Henrich and Boyd (2002):
A discrete trait model with very inaccurate transmission: if nothing else were going on, cumulative adaptive evolution would be extremely unlikely. However, we also assume that individuals have a psychological propensity for conformist transmission.

Conformist transmission effectively corrects even large errors in transmission.

Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

Ex.2: **Blending models of cultural evolution**: No mental representations are replicated, but nonetheless cumulative evolution is possible.

Suppose that in deciding what length to make his arrow, a hunter samples n models from a larger population and adopts as his mental representation (his arrow length) the average of the lengths of the n models. Suppose n=3, and the arrow lengths of the three models are 16 cm, 20 cm, and 21 cm. This means the hunter adopts an arrow length of 19 cm. This 19-cm meme is not represented among the n individuals sampled—there is no replication, fecundity, or longevity.

If we further assume that in selecting their n models, individuals preferentially focus on the best hunters, and that proximity to the optimal arrow length (say 20 cm) contributes to a hunter's success (on average), then blending will generate adaptive evolution on arrow length.

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Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

3. Content-dependent psychological biases are **not** the only processes that affect the spread of cultural traits

Content biases: any situation in which a meme's representational content influences its likelihood of transmission. CBs arise from the **interaction of the representational content of the meme and human psychologies**. Genetically maladaptive "memes" about religion, food taboos, ghosts etc, may readily spread because of their ability to exploit aspects of human psychology in ways that make them more likely to be acquired, stored, and transmitted.

However, humans are quite selective in picking the individuals they will learn from, or be influenced by (**model selectivity**). This means that a meme's mimetic fitness (vs. genetic fitness) will depend **jointly** on **how attractive its content** is to human brains **and** how it affects an individual's likelihood of being selected as a **cultural model** by other individuals.

Context biases: **Conformity, Model-based biases (Prestige etc)**

Five Misunderstandings About Cultural Evolution
Henrich, Boyd & Richerson 2008

3. Content-dependent psychological biases are **not** the only processes that affect the spread of cultural traits

Ex. Fisherman dead at night: a fishing demon?

Two potential stable equilibria:
1. everyone will come to believe the fish-demon story and cease all night fishing;
2. The success costs of not fishing will dominate and the rumor will not be favored in the long run (less success, less chance of becoming a model).

Whether a particular genetic-fitness-reducing meme can spread, and how far it will spread, depends on the details—the dynamics of which are best understood by formally modeling the social and psychological processes involved. The appropriateness of tracking fitness from the perspective of the meme or to individuals is merely a modeling convenience.

Five Misunderstandings About Cultural Evolution
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4. The "cultural fitness" of a mental representation can be inferred from its successful transmission through the population.

5. Selection can only occur if the sources of variation are random (Pinker 1997)

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4. Successful diffusion is **not** a measure of fitness

Authors who adopt the selfish meme concept often give us no causal idea of what actually bestows different "fitnesses" of alternative memes. Fitness = successful spread?

Deleterious or unattractive ideas and practices often spread because they happen to be statistically correlated with attractive individuals or successful groups.

Genetic analogy: acromatopsia in Pingelap.

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4. Successful diffusion is **not** a measure of fitness

Evolutionary biologists escape **circularity** in defining fitness because they have independent means of predicting which genetic variants are more fit.

These principles should also apply to the study of memes – but in many cases it is difficult to predict which representations will spread because we do not understand much about the underlying psychological or ecological processes.

A theory of cultural evolution requires considering all psychological /social/ ecological processes that interact to generate the differential "fitness" of cultural variants.

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Recurrent misunderstandings about cultural evolution

1. Mental representations are rarely discrete, and therefore models that assume discrete, gene-like particles (i.e., replicators) are useless (Atran 2001).

2. Replicators are necessary for cumulative, adaptive evolution (Dawkins1976, 1982).

3. Content-dependent psychological biases are the only important processes that affect the spread of cultural representations (Sperber 1996).

4. The "cultural fitness" of a mental representation can be inferred from its successful transmission through the population.

5. Selection can only occur if the sources of variation are random (Pinker 1997)

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5. Selection does **not** require random variation

Many people have argued that selection cannot affect cultural evolution because cultural variation, unlike genetic mutations, is not based on random copying errors. Instead, cultural changes are systematic, driven by attempts to innovate or by the cognitive machinery by which individuals make inferences about the beliefs of others, and this means selective processes are not important.

As people acquire and modify beliefs, ideas, and values, the variation that is generated can be highly non-random, and these non-selective processes shape cultural variation.
But so what?
Selection occurs anytime there is heritable variation that affects survival or reproduction (transmission). It does not matter whether the variation is random. In cultural evolution, unlike genetic evolution, natural selection may compete with other important directional processes created by human psychology.

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Foundations for a Unified Science of Cultural Phenomena

Some of the essential components for a successful research program in cultural evolution and human behavior:

Rich Psychology

Population Processes

Ecological-Economic Processes

Evolutionary and Culture-Gene Coevolutionary Origins

Methodological Pluralism

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Rich Psychology

Two key components of psychology are of most direct relevance to understanding cultural evolution
1. Understanding how cognition directs social learning toward particular individuals or ideas, beliefs, and so on, and how cognition extracts, or makes use of, the socially available information in a population.

Model-based biases (if relevant social/environmental information is available; else:)
Conformist transmission
Inferentially potent displays: actions that would likely only be performed by those models who actually hold (believe in) the memes they have expressed verbally. Ex: Religion and ritual:
Words in the mouths of prestigious individuals (model-based bias) +
Credibility-enhancing displays: animal sacrifices, donations, painful initiation rites… Large congregations singing in unison and **conformity bias**.

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Rich Psychology

2. Inferential, storage, and recall processes. How do cognitive processes organize and interpret information coming in from the social world? The idea here is to open the black box of imitation.

Ex.: tool making: how do individuals decompose a continuous stream of behavior into steps? How do individuals infer the goals of the individual they attempt to imitate? How do the building blocks of inference (e.g., theory of mind, naïve physics, folk biology) shape the inferences individuals draw from observing these selected cultural models?
Given that public representations of underlying mental representations are nearly always incomplete, how do inferential processes reconstruct mental representations?
How do inference processes deal with the range of different public representations produced by a single individual? How do culturally acquired representations influence subsequent learning processes?

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Population Processes

Knowledge of psychological mechanisms and cognitive structures is insufficient to predict the epidemiology of cultural representations in most cases.

Understanding the population-level consequences of individuals, each possessing learning psychologies and interacting, requires the construction of formal cultural evolutionary models.

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Ecological-Economic Processes

The fitness of cultural variants may be determined entirely by psychological forces, but more commonly different variants have consequences in the environments in which people live. These consequences will often interact with psychological forces (Baum 2005).

The many forms of natural selection are candidates to influence cultural evolution and to produce cultural fitnesses that are close analogs to genetic fitnesses. But these effects are importantly different from those generated by psychological processes.

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Evolutionary and Culture-Gene Coevolutionary Origins

What are the evolutionary origins of the psychological capacities that give rise to cultural evolution?

One of the most important, and least explored, avenues of evolutionary inquiry in human behavior and psychology are the "Baldwinian" processes that arise from the interaction of cultural and genetic transmission. Cultural traditions manifestly change the environments faced by human genes. Human teeth, lack of body hair, digestive processes, malaria resistance, and manual dexterity certainly cannot be understood without realizing that genes responded to the cultural transmission of the use of clothing, fire, agriculture, and tools.

Similarly, culture has likely shaped cognition, both directly and indirectly by changing the selective environment faced by genes.

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Methodological Pluralism

The theoretical and empirical demands of this program exceed those available in any one discipline.

Theoretically, tools have been drawn from population genetics, communication theory, epidemiology, learning theory, statistics, and evolutionary game theory. In the future, insight may come from fields as diverse as information theory and statistical mechanics.

Empirically, this program demands the integration of both observational and experimental data from human biology, psychology, economics and anthropology, as well as studies of processes of long-term change from paleoecology, history, and archaeology.

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Foundations for a Unified Science of Cultural Phenomena

Darwinian approach differs from traditional social sciences approaches in ways that are not yet fully appreciated.

The 5 misunderstandings result from a tendency to think categorically rather than quantitatively.

The proper approach is to recognize that the analogy between genes and culture is quite loose, and to build up a theory of cultural evolution that takes into account the actual properties of the cultural system.

Culture has a much richer array of psychological processes with population level consequences than is the case for genes. But neither particular psychological forces nor the integrated effect of all such forces in any way rules out a role for natural selection, or vice versa.

Can culture be maladaptive? (recap)

Evolutionary Psychology:
adaptations developed in the past (the Environment of Evolutionary Adaptiveness) may not be adaptive in modern environments ("adaptive lag")

Human Behavioral Ecology:
adaptability / human adaptive plasticity
difficulty to explain apparently maladaptive cases.
Ex: "demographic transition" in industrialized societies

Memetics:
optimization of memes' fitness may not favour *genes'* fitness
"The selfish meme"

Gene-Culture Coevolution:
vertical transmission (parallel to genetics) and fitness optimization;
oblique / horizontal transmission... not necessarily.

Darwinian Models of Cultural Evolution

Implications:

Cultural evolution can be Darwinian, though not "Neo-Darwinian"

Variation is not random: Content and context biases, frequency (Conformity) or model-based (Prestige etc).

Inheritance: Vertical + Oblique + Horizontal; no discrete "Replicators".

Differential fitness...? *Cultural* group selection?

Next lecture

Cultural Group Selection and the Evolution of Cooperation