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Comprehensive Exam Topics and Theory

November 22-24, 2019

**Primate Energetics & Endocrinology**

1. [Energy allocation and mobilization; Metabolic hormones](#Energetics_allocation)
2. [Evolutionary adaptations to seasonal and prolonged nutritional stress](#Adaptations_to_stress)
3. [Life history, energetics, reproduction, and reproductive fitness](#Life_history_energetics_repro)

**Key words:** total energy expenditure, energy intake, energy balance, energy flux, energetic status, Kleiber’s law, nutrient balance, digestion, gut anatomy, energetic tradeoffs, growth, metabolic and reproductive hormones, reproductive function, Metabolic Load Hypothesis, allostatic load, stress, capital vs. income breeding, Jarman-Bell principle, allometric scaling, anatomical and physiological dietary adaptations, folivory, and insectivory

**Energy allocation and mobilization; Metabolic hormones**

* + What are some of the key demands into which organisms must allocate available energy?
    - **Maintenance**
    - **Growth**
    - **Reproduction**
  + **Lack Hypothesis: a tradeoff**
    - Quantity vs Quality: you can’t have it all
      * Many eggs, little energy per egg
      * Few eggs, lots of energy per egg
  + Finite amount of energy available (and attainable)
    - Tradeoff between repro and survival
    - **R/K selection: R** (short life/many offspring) and **K** (long life/few offspring) strategies
    - Present vs future repro – invest a lot in first baby or save for later babies
    - Quantity vs quality
    - Investment in direct vs indirect reproduction
  + **Kin selection**: investing in the offspring of a relative; still pass on some of your genes
  + **Flight or fight response** and how is it related to 1) stress and 2) changes in energy allocation?
    - A close up of a logo

      Description automatically generatedResponse to stressor- environmental input that we perceive as dangerous/stressful
      * Increase immediate energy availability for necessary organs (muscles, brain, eyes)
        + Glucose production
        + Fatty acid breakdown
        + BP
      * Suppress non-critical systems
        + Repro
        + Immune
        + Digestion
    - CNS informs hypothalamus that you are experiencing a situation that requires a change in homeostasis
      * Can be self-perceived
    - Sympathetic nervous system is mobilized
  + **HPA axis and stress**
    - Hypothalamus 🡪releases CRH (corticotropin releasing hormone)🡪 anterior pituitary via hypophyseal portal system 🡪 releases ACTH (adrenocorticotropic hormone) 🡪 adrenal cortex 🡪 release of cortisol
    - Cortisol them enters bloodstream, impacts target organs
      * Increases liver glucose output
      * It’s a steroid, so can cross BBB, impacting behavior
    - Helps deal w short term stress
      * Hypothalamus wants to go back to homeostasis
      * (-) feedback- decrease in CRH🡪slows down pituitary release of ACTH
  + What are catecholamines and when are they released?
    - Stress response – 2 phases
    - Short term effects:
      * Increases glucocorticoids (cortisol) – 15 min
    - Immediate effects: increases catecholamines including Epinephrine (*adrenaline*) and Nor-Epinephrine
      * Hormones produced in **adrenal medulla**
        + Catecholamines are stimulants used to bring heart back to normal sinus rhythm during **bradycardia**
      * Independent effects on body, but also (+) feedback to speed up cortisol release
        + By signaling hypoth. And AP to speed the release of ACTH
    - Catecholamines increased in the days before stress (before exam) and then decreased after
  + What are energy flux and energy balance, and how are they important for amenorrhea?
    - **Eb** – whether you are losing or gaining weight, ***independent of total weight***
      * Net + or – intake
    - Extreme fluctuation (like training really hard) 🡪 increase in Eflux, causes amenorrhea
    - **Eflux** – rate at which E is moving through body (more intake, more expenditure = higher Eflux)
    - ***Ex. Exercise can suppress hormonal function***
      * Phase 1: woman had cycles suppressed regardless of whether they lost weight
        + All women resume cycling normally after ending training
        + Peak in Estrogen delayed, but not followed with peak in Progesterone production

Follicular suppression – development of follicle delayed

Luteal suppression (no Prog.)

* + - * Phase 2: complete suppression
        + No peak in either hormone while training
      * Rate of weight loss (Eflux) has a big impact, not the actual weight itself
      * Takes a BIG energy flux to stop ovulation
      * Males show changes in repro hormones (LH, T) in as little as a single meal
      * Women can fast for 6 days before showing any impact on ovulation
        + **For fecundity – ovulation is what matter**
      * Ex. Agro people in Poland
        + Those who worked harder in the field had lower progesterone production

Than those who did not work hard

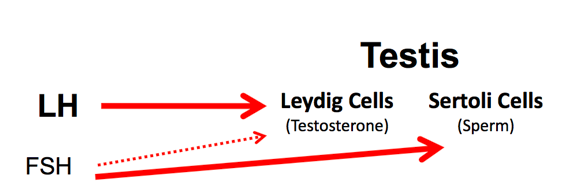
* + - * + **Total energy expenditure** matters more in progesterone production
      * Those who expend more also eat more, but not enough to make up for it
  + How do periods of intense physical activity affect reproductive hormones (estrogen, progesterone, testosterone)?
    - Depends on how intense (Time: 41:00):
      * In general- suppression with intense workout
      * May lead to amenorrhea
  + **Insulin, leptin, and growth hormone**
    - Hormones that convey information on energy intake and flux
      * Insulin- glucose uptake in the muscle and fat, glycogen prdxn in the liver
      * GH- proliferation of cells in the follicle
      * Leptin- signal from body fat, appear to be receptors in endometrium
    - Fat levels in body:
      * Leptin was elevated in pregnancy regardless to body fat
        + Those with more fat were producing more leptin
        + Tells the body = I have energy
  + **Kleiber’s Law:** the observation that, for the vast majority of animals, an animal's [metabolic rate](https://en.wikipedia.org/wiki/Basal_metabolic_rate) scales to the ¾ power of the animal's mass. Symbolically: if *q*0 is the animal's metabolic rate, and *M* the animal's mass, then Kleiber's law states that *q*0 ~ *M*¾. Thus, over the same timespan, a cat having a mass 100 times that of a mouse will consume only about 32 times the energy the mouse uses.
    - A 1932 work by Brody calculated that the scaling was approximately 0.73.[[5]](https://en.wikipedia.org/wiki/Kleiber%27s_law#cite_note-History-5)[[17]](https://en.wikipedia.org/wiki/Kleiber%27s_law#cite_note-17)
    - A 2004 analysis of field metabolic rates for mammals conclude that they appear to scale with exponent 0.749.[[13]](https://en.wikipedia.org/wiki/Kleiber%27s_law#cite_note-Savage_2004-13)
* Metabolic rate (MR) scales to the three-quarters power of body mass (M)across a series of species ranging from mouse to elephant (Hemmingsen, **1960;** Kleiber, **19611)**
* ***Kurland and Pearson 1986:*** *The “Kleiber relationship” describes the interspecific allome- try between body size and metabolism. Like other allometric relationships, the Kleiber relationship not only summarizes scaling effects across species but also provides a standard by which species can be compared. One well-noted deviation from the Kleiber relationship is “hypometabolism”: metabolic rates below that expected for a given size. It has been suggested in the literature that hypometabolism may be* ***1)****a primitive mammalian trait, 2) a thermoreg- ulatory adaptation,* ***3)*** *a n adaptation to arboreal folivory, or* ***4)*** *a n adaptation to a diet that is deviant for body size*…. *Some animals deviate from the Kleiber re- lationship by having MRs much below that expected €or their size, a condition termed hypometabolism (e.g., Goffart, 1977; Muller, 1979)*
* ***4 explanations for Hypometabolism:***
  + *Primitive Trait hypothesis*
    - *Endothermy in mammals – primitive mammals might exhibit metabolic rates (MR) below expected for their size, because their endothermic physiology is under developed (McNab 1978b)*
    - *Prosimians tend to by hypometabolic because of phylogenetic intertia (Eisentraut 1961, Muller 1985)*
  + *Thermoregulatory hypothesis*
    - *Hypometabolism may be a thermoregulatory adaptation to the heat stress of the tropics (Charles-Dominique 1974, Muller and Jaksche 1980).*
    - *Hypometabolism tends to occur in nocturnal animals with a lot of insulation*
      * *Inactivity during the day decreases heat production by the body during heat stress (daytime), and activity at night produces heat during cold-stress*
  + *Arboreal-folivory hypothesis*
  + *Deviant-diet hypothesis*
* **What does testosterone do?**
  + Secondary sexual characteristics (sexually dimorphic traits)
  + Muscle mass
  + Face shape
  + Voice pitch
* ***Gaulin and Sailer 1984:*** The causes of sexual dimorphism have been vigorously debated over the past two decades. Current explanations of primate dimorphism appeal to the effects of both allometry and sexual selection, but there is disagreement over the relative importance of these two factors and the extent and nature of any interaction between them.
  + High degree of correlation between body size and dimorphism suggests that size is a major cause of dimorphism (***Leutenegger and Cheverud 1982****)*
  + *Little obvious dimorphism in monogamous species (Kleiman 1977), suggesting that sexual-selection must be the driver*
    - *Sexual Selection Theory posits that dimorphism is a consequence of sex differences in reproductive potential (RS)*
    - ***Bateman’s Principle (Bateman 1948, Trivers 1972):*** *For example, if males can father more offspring than females can mother-due to differences in the resources that each sex typically contributes to the offspring-males will compete for access to poten- tial mothers. Large size, weaponry, refined courtship, or other traits that increase access to mates would be more strongly favored by selection in the sex that engages in such competition (Bateman, 1948; Trivers, 1972). This theory predicts that, when males and females make roughly equal investments in the production of offspring, thus' differing little in reproductive potential, other sex differences will be small. This situation is approximated most closely among the primates by the pair-bonding, monogamous species.* 
      * ***Counterargument:*** *Leutenegger and Cheverud (1982) did an analysis of 44 primate species and found that sexual selection can no longer be viewed as the major contributor to the degree of sexual dimorphism, since only minor amounts of the variance in weight can be attributed to differences in mating system.*
      * ***The alternative:*** *size isn’t just ONE of the factors affecting dimorphism, it’s the MAJOR one.*
      * ***Clutton-Brock et al.*** *1977 have similar analysis*
        + *Weight dimorphism is strongly correlated with weight (body size)*
  + ***Gaulin and Sailer*** *– resolving the issue:*
    - *Sexual dimorphism in size ~ sexual selection and body size*
    - ***Red-tailed monkeys Haddow 1952***
    - *Sexual-selection theory predicts that, when there are sex differences in reproductive potential, males and females will differ in some way. That is, the sex with higher reproductive potential will evolve mate-maximization traits; thus, compared to the other sex, they might be larger, stronger, more brightly colored, more aggressive, or have superior spatial ability, etc. At present, a weakness of sexual-selection theory is that it does not predict which mate-maximization traits will evolve in any particular species. We now ad- dress this problem by asking, Under what conditions will the dimorphism be expressed as weight dimorphism?*
    - *We think that there are good reasons to predict that sexual dimorphism is more likely to be expressed as weight dimorphism for larger species. Ev- ery trait incurs both costs and benefits, its evolutionary fate being determined by the net balance of the two. The essence of the theory of sexual selection is that traits that decrease survival prospects (costs) may nevertheless spread, if their effects on fertility (benefits) are sufficiently positive*
    - *Increasing male size by an increment is more likely to yield a net benefit in larger species than in smaller ones.*
    - ***Lesser Costs in large species:***
      * *1. Metabolic requirements increase more slowly than body weight (Kleiber 1961)- a strong relationship between body size and diet supports this (Bell 1971, Jarman 1974, Gaulin and Konner 1977, Clutton-Brock and Harvey 1977a, b, Gaulin 1979)*
        + *Larger size leads animals to concentrate on foods that are of lesser quality, but higher abundance. Smaller sizes base their diets on rare, but nutrient rich foods.*
        + *Large-bodied animals who experience selection for larger body-size would have less difficulty harvesting a large amount of food than smaller-bodied ones than smaller animals experiencing similar selection*
        + *The response to sexual selection for increased body size can therefor be more dramatic for large-bodied species (Clutton Brock et al 1977)*
      * *2. When sexual selection increases male weight, it decreases the precision of fit between male boyd weight and diet in promiscuous and polygynous species, whereas in monogamous species both sexes conform equally well to predicted diet-for-weight optima (Gaulin and Sailer 1984).* 
        + *Deviation from the optimal size for a particular feeding niche must entail fitness costs that will be proportional to the extent of interspecific competition for the same resources.*
        + *Because niche space is generally less packed at larger body sizes, such constraints will be less severe for larger species (there are less large-bodied species than there are small-bodied ones)*
    - ***Increased Benefits in large species:***
      * Increased benefits during agonistic interactions between males in large species.
        + Not that larger size is advantageous in battle, but that size increase is advantageous for animals that are already large. In aggressive situations, the most direct predictor of victory is the ability to inflict injury on the opponent. Most conflict is resolved via relatively harmless ritualized displays, but the signaling of the threat is genuine.
        + If display was not a good indicator of chances of victory, weak indivs could display high-intensity signals and gain advantage (Krebs and Davies 1981, Clutton-Brock et al 1982).
        + The potential to inflict damage depends on

The energy that can be delivered

Energy increases with weight

The relative ability of the aggressor and the opponent to absorb the energy without sustaining injury.

Ability to absorb blows increases with area and with length (of, say, bone, or thickness of the skin)

* + - * So for progressively larger sized animals, the differential between the ability to deliver energy and the ability to resist its effects becomes greater and greater. Any increase in size in the aggressor will increase their competitive ability in large-bodied species.
* **How can we think about the function of testosterone in terms of life history theory and energetic trade-offs?**
  + Comes with tradeoffs- energetic costs
  + Consequence of traits of developing-
    - adding muscle mass, costs more to use it
    - T associated with behaviors that are energetically costly- aggression, risks
  + Immune fxn is energetically costly- forces tradeoff
* T: HPG axis: hypo🡪(GnRH) 🡪Ant Pit 🡪 (LH FSH)🡪 Testes
* LH FSH reach teses
* **LH** 🡪 leydig cells (T) [**vital for T prdxn**; cells outside of vescicles]
* FSH 🡪 sertoli cells (Sperm) [FSH minor role in T, but more spermatogenesis; inside vescicles]
* A close up of a logo

  Description automatically generatedHPG axis standard T prdxn pathway
* but also produced in brain, Adrenal glands (and women too in ovaries)
* Neg feedback in T:
* high levels of T circulate; reach hypothalamus 🡪 pituitary
* Body wants homeostasis
* Downregulation occurs at hypoth, T downregulates GnRH
* Neg FB loop
* A close up of a device

  Description automatically generatedAs T decreases, GnRH can rise again
* FSH: interacts w **sertoli cells** to complete spermatogenesis
* FSH go up, testes release Inhibin B (Neg FB loop) 🡪
* **How does testosterone respond to energy intake and expenditure?**
  + T and LH decreased after missing even a meal
    - (responsive in short term to energy intake and expenditure)
  + Males: System that can be turned on/off
  + Females: can’t, because of cycle (can’t skip one meal and not ovulate)
  + Men: fast for >48hrs show castrate levels of T
* **What happens to a man’s testicles if he takes anabolic steroids? What if he takes pregnant horse urine with the steroids?**
  + Synthetic androgens
  + **Hack trade-offs** (forcing the body to put energy in some things and not others; immune suppressed) = downregulates other fxns
  + Mood changes, irritability, anger
  + Affect Neg FB pathway 🡪 reduced LH, FSH prdxn 🡪 smaller testicles, lower sperm count
* **Challenge hypothesis**
  + testosterone promotes aggression when it would be beneficial for reproduction, such as mate guarding, or strategies designed to prevent the encroachment of intrasexual rivals.
  + Sporting events; spike T when needed (redirects E in short term towards a certain behavior)
  + Beneficial when fleeing; beneficial for fighting
* **How does testosterone change with age in industrialized populations?**
  + During energetic stress: T is downregulated
    - Immunocompromising events
    - Reduced energy intake
    - Increase energy expenditure
  + Expect lower T in subsistence populations
  + **Industrialized pops:** 
    - Highest levels in 20s, drop with age
    - Sedentary lifestyle and obesity contribute to age related T change
      * Obesity causes lower T
      * T + fat cells 🡪 convert T to EST (man boobs!)
        + Feedback loop
  + **Subsistence pops**
    - Almost no reduction of T w age!
      * Very active (~4.5 active hrs a day)
      * Woman avg TFR: 9.1
    - We see this in repro hormones in women too, w same amount of repro fxn
    - High disease burden
* **How does testosterone change with age in subsistence populations?**
  + See above
* **How is testosterone linked to prostate enlargement and prostate cancer?**
  + Male exocrine gland that produces seminal gland
  + Prostates enlarge with age
  + Benign prostatic hyperplasia (BPH)
    - With age- by age 70-80 prostates enlarge in almost everyone across many studies
  + Tsimane: don’t enlarge w age
    - Growth is stimulated by T levels
* **Why do we think the Tsimane have much less evidence of enlarged prostates?**
  + Lower T in Tsimane; and T is linked to prostate enlargement – so less enlargement
  + T 🡪 Cell division 🡪 enlarged prostate
  + Tsimane- low baseline T due to environment they live in
  + In industrialized pops: Evolutionary novel levels of T
    - Low t: associated w mortality; cardiovascular morbidity; depression; reduced cognitive ability
    - Evo: trade offs for men in poor condition result in decreased T
    - T as a symptom, not the cause?
    - T supplement: testicular atrophy; Myocardial Infarct risk; Cardiovascular morbidity; thrombosis; immune implications (T decreases during illness)
  + Tradeoff in T: short-term repro benefits; survival consequences
* **How and why does testosterone change during competition?**
* **What is the winner effect that is seen in some studies of testosterone and competition?**
  + It is dependent on the individual- was higher when you think higher of your performance
  + But maybe high performance increases T, so you think higher of yourself
  + Didn’t matter if you won or not
* **Testosterone and Behavior**
  + Young men + T = less generous
    - Men have masculinized brain with high density of T receptors
  + Post-menopausal women + T = no impact on behavior
    - Females have low density of T receptors = less effect of T
  + Congenital Adrenal Hyperplasia- high levels of androgens in utero
    - Females with CAH can have ambiguous genitalia, higher levels of aggression in childhood, higher rates of competitive behavior
    - High levels of androgen based organization (high receptor density), activationally possible from acute increase in T
* **Evolutionary adaptations to seasonal and prolonged nutritional stress**
* ***Beehner and McCann 2008:*** *Some of the most common predictors for hormone profiles in wild animals are seasonal changes in ecology and behavior. For example, we might expect baseline glucocorticoid levels to track changes in food availability or other seasonal stressors such as unusually high or low temperatures.*
  + *In vertebrates, the concentration of glucocorticoids in plasma is widely used as one indicator of physiological stress* *[[1]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib1),* *[[2]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib2),* *[[3]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib3),* *[[4]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib4),* *[[5]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib5). However, much like Heisenberg's uncertainty principle, it can be difficult to obtain accurate glucocorticoid values from serum because the invasive process of drawing blood can itself induce a stress response — thus, changing the variable of interest. This “observer effect” is due to the transient nature of plasma glucocorticoid values, which rise with the onset of a stressor within 2–3 min.*
  + *it is optimal to have non-invasive alternatives for measuring hormones. Consequently, sophisticated methods have been developed for measuring glucocorticoids from “excreta”* *[[6]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib6) — mainly, urine* *[[7]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib7),* *[[8]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib8),* *[[9]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib9),* *[[10]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib10) and feces* [*[8]*](https://www.sciencedirect.com/science/article/pii/S0031938408002412#bib8)*,* *[[11]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib11),* *[[12]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib12),* *[[13]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib13) but increasingly also saliva* *[[14]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib14),* *[[15]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib15),* *[[16]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib16).*
* *In* ***seasonal breeders****, we expect to find an* ***increase in gonadal activity*** *prior to the onset of breeding. However, in both seasonal and non-seasonal breeders, we* ***might also expect baseline glucocorticoid levels to track changes in food availability*** *or other seasonal stressors such as unusually high or low temperatures. Indeed, nutritional stress, heat stress, and cold stress have been associated with elevated glucocorticoids in a variety of species* *[[21]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib21),* *[[22]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib22),* *[[23]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib23),* *[[24]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib24),* *[[25]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib25),* *[[26]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib26),* *[[27]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib27),* *[[28]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib28),* *[[29]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib29),* *[[30]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib30),* *[[31]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib31),* *[[32]](https://www.sciencedirect.com/science/article/pii/S0031938408002412" \l "bib32).*
* ***Shively et al 2009:*** *primates have the capacity to store body fat when opportunities to excess energy arise. During most of primate evolution, such opportunities were rare. More common was food scarcity and high levels of physical activity. A large brain is metabolically demanding. It is thought that the evolution of encephalization is primates was accompanied by the coevolution of elaborate physiological systems to protect against starvation and defend stored body fat to provide the metabolic needs of a large brain -* ***“Thrifty Genotype” (Neel 1962, 1982)***
  + ***Visceral fat*** *is especially well constructed to resist lipolysis*
  + ***Obesity:*** *With these biological mechanisms to maximize fuel efficiency in place,human primates went on to devise technological aids for increasing energy consumption and reducingphysical effort. In the last century, industrialization provided access to great quantities of mass-produced,high-calorie foods and many labor-saving and transportation devices, virtually abolishing starvation andheavy manual work in much of human society. In thismodern obesogenic environment, individuals withthe thriftiest combination of ancestral energy-conserving genes are at greatest risk for obesity andassociated chronic diseases [Bellisari, 2008]. Thus,obesity is the result of the complex interaction ofgenetic and environmental factors. This review willfocus on social environmental factors that promotevisceral obesity* and the metabolic syndrome
  + *chronic, mild, social stress is characteristic of the lives of many, and occurs in a permissive environment of over-nutrition [Adam &Epel, 2007]. The mechanistic pathways through which stress may promote visceral obesity and associated metabolic perturbations have been reviewed in detail [Black, 2006; Kyrou & Tsigos,2007]. Briefly, adipose depot size is a result of the balance between actions which promote lipid accumulation vs. those that promote lipolysis.*
  + ***Cynomolgus monkeys(Macaca fascicularis) are a widely used model of diet-induced atherogenesis, obesity****, and type 2diabetes mellitus. The* ***deleterious effects of social stressors****, including social instability, isolation, challenge, and subordination, on CAA have been welldocumented in this species [Adams et al., 1985;Kaplan et al., 1983, 2009; Shively et al., 1989; Strawnet al., 1991].* ***Social subordination stress is particularly likely to result in pathology in female cynomolgus macaques of reproductive age****. When placed in social groups female cynomolgusmonkeys quickly organize themselves into linearsocial status hierarchies, which are stable forextended periods of time [Shively & Kaplan, 1991].*

***Stini 1969:***

* ***Adrenal cortex exerts adaptive response*** *to starvation- results in* ***muscle resorption****. Muscle resorption results in the actual* ***loss of fibers*** *has been reported in malnourished human cadavers*
* ***Collagen breakdown*** *in malnourished individuals (Dean 1964)*
* *Degree of muscle resorption is* ***under hormonal control***
* ***Malnourished women produce normal size fetuses*** *while gaining little or no weight during pregnancy - extending the likelihood of hormonal control of tissue resorption to human subjects.*
* *The fact that* ***collagen is resorbed*** *to a lesser extent than tissue protein would tend to modify short-term effects of maInutrition on skeletal maturation. However, when a long- term protein deficiency occurs, the appearance of ossification centers would also be delayed. Garn et al. ('63) postulate that the appearance of ossification centers is normally under genetic control, but that when malnutrition is prolonged, genetic factors no longer predominate.*
* *he long-term effects of protein de- privation are more pronounced in males. The role of the sex hormones and the ad- renal cortex in adaptation to severe mal- nutrition is a complex and important one, but more information concerning the mechanisms of hormonal control must be obtained if a real understanding of the process of growth under stress is to be achieved.*

*A close up of a map

Description automatically generated*

***From Chapman et al. 2015 - Competing pressures on populations***

﻿Despite strong links between sociality and fitness that ultimately affect the size of animal populations, the particular social and ecological factors that lead to endangerment are not well understood. Here, we synthesize approxi- mately 25 years of data and present new analyses that highlight dynamics in forest composition, food availability, the nutritional quality of food, disease, physiological stress and population size of endangered folivorous red colo- bus monkeys (Procolobus rufomitratus).

*﻿For example, high stress levels or poor nutrition could lead to a greater suscepti- bility to disease, which together could have a negative influence on population size. Gulland [26] presented an excel- lent experiment in wild sheep (Ovis aries), where she studied the interacting effects of nutrition and parasites on survival. Gulland found that an observed population crash coincided with both malnourishment and high nematode loads. Physiological mechanisms, such as hormonal responses to changing social and environmental conditions, may mediate the impact of nutrition and disease on a population’s abun- dance [27]*

***Figure 1.*** *Theoretical energy mobilization model (adapted from Vogel et al. 2012).*

*An individual is in good physical condition when energetic intake exceeds energetic output* ***(A)****;* ***energy from diet***

*As food availability declines, an individual transitions into a neutral energetic state* ***(B)****;*

*when energetic intake is less than output, an individual begins to show physical condition decline accompanied by breakdown of energy from fat stores* ***(C)****;* ***energy from fat***

*When these reserves are depleted, an individual transitions into an excessive energetic deficit, and energy from body protein (muscles) is consumed for metabolism* ***(D)****.* ***energy from body protein***

*Above is a panel of biomarkers and their relative responses to each energetic state (gain, equilibrium, or loss):*

* *Urinary C-peptide of insulin (****UCP****) increases with increasing energy balance and status (EB and ES, respectively);*
* *Creatinine to specific gravity ratio (****CR:SG****) correlates with muscle to body mass ratio;*
* *triiodothyronine* ***(T3****) increases with thermoregulatory needs as an individual depletes fat stores fat;*
* ***Cortisol*** *increases with energetic and psychological stress;*
* ***Ketones*** *are released with metabolism of fat reserves;*
* *and the ratio of 14N:15N stable isotopes (****δ15N****) decreases with tissue buildup and increases with tissue breakdown for metabolism.*
* *Urinary C-peptide (UCP) of insulin is a by-product of sugar breakdown and offers an* ***accurate measure for positive values of EB*** *(Emery Thompson and Knott 2008) and fat (energy) stores in the body (ES, Pontzer 2015).*
* *The ratio of urinary creatinine to specific gravity (CR:SG) can be* ***used to estimate relative muscle mass*** *(Emery Thompson, Muller, and Wrangham 2012), which is an important factor in calculations of weight loss, gain or equilibrium.*
* *Triiodothyronine (T3) is a thyroid hormone that influences growth, hormonal balance, metabolic activity, and nutritional stress.* ***It can be measured reliably in primate feces and is particularly responsive to nutritional deficits, reducing metabolism and promoting energy conservation*** *(Emery Thompson 2017; Wasser et al. 2010). T3 can be used to tease apart indicators of high energy availability due to large energy stores (high values of UCP, for example) from those of high energy availability resulting from decreased metabolism.*
* *A close up of a map

  Description automatically generatedCortisol indicates adrenal function: when cortisol is high, it directs energy away from the digestive system and towards organ systems involved in sympathetic responses to environmental stress.* ***Chronic activation of cortisol decreases energy allocation towards seeking, acquiring, and digestion of food and uptake of nutrients*** *(Sapolsky 2002). Urinary (Bahr et al. 1998; Crockett et al. 1993; Smith and French 1997) and fecal (Graham and Brown 1996; Monfort et al. 1998; Palme and Möstl 1997)* ***cortisol indicate levels of social and nutritional stress in primates****.*
* *Ketones are* ***produced when the body metabolizes its fat reserves to produce energy*** *and can be used to assess negative energy balance during starvation (Knott 1998; Robinson and Williamson 1980).*
* *The δ15N values from animal tissues largely reflect animal trophic position (Ben-David and Flaherty 2012; Boecklen et al. 2011; Minagawa and Wada 1984) but are also* ***influenced by an organism’s nitrogen balance: anabolic states*** *(tissue and organ buildup)* ***cause a decrease in consumer δ15N values while catabolic states*** *(tissue and organ breakdown) that* ***occur during periods of starvation increase consumer δ15N*** *values (Fuller et al. 2005).*

**Fuller et al 2005: Nutritional Stress – why you’re not what you eat during nutritional stress**

* ﻿*While past experiments on animals, birds, fish, and insects have shown changes in stable isotope ratios due to nutritional stress, there has been little research on this topic in humans. To address this issue, a small pilot study was conducted. Hair samples from eight pregnant women who experienced nutritional stress associated with the nausea and vomiting of morning sickness (hyper- emesis gravidarum) were measured for carbon (d13C) and nitrogen (d15N) stable isotope ratios. The d13C results showed no change during morning sickness or pregnancy when compared with pre-pregnancy values. In contrast, the d15N values generally increased during periods of weight loss and/or restricted weight gain associated with morning sickness. With weight gain and recovery from nutritional stress, the hair d15N values displayed a decreasing trend over the course of gesta- tion towards birth. This study illustrates how d15N values are not only affected by diet, but also by the nitrogen balance of an individual.*

***Armstrong 1990 – Brains, Body, and Metabolism***

* *A close up of text on a white background

  Description automatically generatedDuring episodes of lowered glucose levels, for example during starvation, nonneural tissues stop metabolizing glucose, thereby making it available to the brain.*
* *Small decreases in serum glucose levels are compensated for by increases in its active transport into the brain. Under normal conditions these features main­tain the brain’s activities, but stupor and coma arc associ­ated with decreases of serum glucose and oxygen [Duffy and Blum, 1981]. Because the brain stores little glycogen or oxygen, hypoglycemia or deprivation of oxygen can quickly produce stupor [Duffy and Plum. 1981] and irre­versible neuronal damage [G hajaret al., 1982], Thus, the maintenance of normal brain functioning is dependent upon the availability of an adequate external supply of glucose and oxygen. Minimal levels can be reached rel­atively easily; the maximal levels are much broader and only rarely attained.*
* *If the above features protect an organism’s brain from small perturbations in the levels of 0 2 and glucose, other design features assure that adequate amounts of these substrates reach the brain. An increase in body size pro­vides the brain with a bigger cardiovascular system which can thus deliver more nutrients per unit time [ Wcibcl et al.. 1981; Schmidt-Niclsen, 1984], A higher basal met­abolic level (BM R) produces a faster heart rate and a con­comitantly faster turnover of nutrients [Schmidt-Niclsen. 1979. 1984]. Other characteristics, such as species-spe­cific differences in hemoglobin-carrying capacities of 0 2, glucose carriers, and diffusion distances are not yet known and could play an important role in how the brain sustains its activity and thus influence brain-body rela­tionships in different phylogenetic groups. Such differ­ences have not yet been identified, but researchers have begun to look at the role of metabolism.*
* *An animal’s metabolism is measured both by heat and 02 consumption. The metabolic rate is reported either for the whole animal or, as the specific metabolic rate, for the amount of 02 consumed per unit amount of tissue. Basal metabolism has been determined in a large number of ver­tebrates, and thus give us an opportunity to study the association of the BMR with relative brain weight. When the BMR for the whole organism is analyzed, it is found to scale with body weight in a negatively allometric fashion. If the specific BMR is used, it decreases as body size increases [Schmidt-Niclsen, 1979].If brain-body relationships are influenced by the avail­ability of glucose and 02, then both body size and metabo­lism should have a profound impact on the association [Martin. 1981; Armstrong, 1982, 1983, 1985a, b; Hof- man. 1983].*
* *The basal metabolic rate, measured when the animal is quiet, postabsorptive. unstressed and in a thermoneutral zone, is obviously an artifact of an animal’s real existence, but it allows a standardization of an otherwise protean variable. Careful studies of metabolism, carried out over sufficient time, show that the basal metabolic rate has both circadian and circaannual rhythms. As ait animal becomes active, its metabolism rises from its basal level [Schmidt-Nielsen, 1979. 1984; Taylor, 1987).*
* *An allometric study of brain size as a function of body size and specific metabolism shows several relationships among species that differ from those found when brain weight is standardized solely by body weight.* 
  + *First, the brain weight to available energy slope is close to being iso­metric. That is, differences in the sizes of brains match the differences in the amount of available energy. This find­ing has been documented in both mammals and birds (Marlin. 1981; Armstrong. 1982, 1983. 1985a, b; Arm­strong and Bergeron, 1985; Hofman, 1983). The fact that the brain docs not expand at a rale commensurate with that of the body [White and Gould, 1965] ceases to pre­sent a problem.*
  + *Second, critical relationships among taxonomic and dietary groups shift. Analyses of covariance show that the* ***observed significant differences in relative brain size in prosimians and nonhuman anthropoids (fig. I) can no longer be distinguished if metabolism is considered*** *[ Arm­strong, 1985a], and, similarly, callithricids can no longer be separated from other monkeys [Armstrong, 1990). All groups of nonhuman primates examined to date have the same relative brain size, if metabolism is taken into account, and they have relatively bigger brains for their energy supply than do other mammals [Armstrong, 1982, 1983, 1985b]. With metabolism controlled for, dolphins and other toothed whales have relative brain sizes that are indistinguishable from those of other mammals (Arm­strong, 1983]*
* *Frugivorous bats have brains of the same relative size as insectivorous bats if differences in metabolism are considered [Armstrong, 1983]. These dis­parate data support the idea that* ***the relationship between brains and bodies is mediated by the brain’s requirements for glucose and oxygen.***
* *If one controls for available energy, humans have the highest relative brain size, and the values for nonhuman primates are intermediate between those for humans and those for other mammalian orders.* 
  + *This separation of species matches that derived by neurochemists who have measured the percentage of the body’s energy reserved by the brain. Humans use about 20% of the body’s energy reserves [Kety, 1957], rhesus monkeys about 10% [Schmidt et al., 1945] and rats, dogs and cats about 5% [Geiger and Magues. 1947; Gilboe and Betz. 1973; Nils­son and Siesjo, 1976].*
* ***Thus, the allometric and neuro­chemical data corroborate each other to lend support to the idea that differences in relative brain size arc related to the energy costs of maintaining the brain.***
* *the effects of growth hormone on brain growth occur during later develop­mental stages.*

***Cameron, Helmreich and Schreihofer 1993: Modulation of Reproductive Hormone Secretions by Nutritional Intake: Stress Signals vs Metabolic signals***

* *In humans and many other species changes in the nutritional statusof the body can have profound influences on the activity of thereproductive system. Chronic undernutrition during childhoodand the peripubertal period can significandy delay or prevent thepubertal awakening of the reproductive axis (Kennedy and Mitra,1963; Dreizen et al, 1967; Foster and Olster, 1985; Bronson,1986). In adulthood, chronic undernutrition is associated withan increased incidence of acyclicity or irregular ovarian cyclesin females (Howland, 1971; Vigersky etal., 1977) and withgonadal atrophy and infertility in males (Zubiran and Gomez-Mont, 1953; Howland, 1975). Across species, the suppressionof gonadal activity in undernourished states is associated withdecreased circulating concentrations of luteinizing hormone (LH)and in some, but not all cases, with decreased follicle stimulatinghormone (FSH) concentrations (Zubiran and Gomez-Mont, 1953;Kennedy and Mitra, 1963; Howland, 1971, 1975; Vigerskyetal., 1977; Foster and Olster, 1985; Bronson, 1986; Dubeyetal., 1986).*
* *When monkeys are fasted (i.e. prevented from their normalaccess to food at mealtime) they display a considerable amountof behavioural agitation, marked by a significant increase in activebehaviour such as spinning, pacing, and banging the cage(Schreihofer etal., 1993a,b).*
* *by feeding monkeys about three times their normal allotment of monkey chow on the day prior to fasting we were able to* ***significantly delay several hormonal responses to fasting*** *(i.e. decreases in circulating insulin and triiodothyronine(T3) concentrations).*
* *Results of our recentstudies, taking two approaches towards determining the nature;of the signal which causes suppression of LH secretion duringperiods of fasting, provide no support for the hypothesis that a'stress signal' (independent of metabolic signals provided byfasting) causes fasting-induced suppression of LH secretion.Allowing monkeys to experience the psychological stressassociated with missing a meal, in the absence of the metabolicsignals associated with fasting, does not cause a suppression ofLH secretion. Moreover, pharmacological blockade ofendogenous opioid pathways and the adrenal axis does not preventfasting-induced suppression of LH secretion.*

***Clutton-Brock and Harvey 1979 on Physiological Adaptations and Diet***

***Gaulin 1979 – A Jarman/Bell Model of Primate Feeding Niches***

***Kamau and Muller 1989***:

* KLEIBER'S "mouse to elephant curve" (KLEIBER, 1961) is generally accepted as a reasonable relationship describing the dependence of basal metabolic rate upon body mass in many spe- cies of homeotherms.
  + But not so much in prosimians (Muller 1985) (and marsupials and birds), arboreal folivores (McNab 1978), and inhabitants of dessert or semi-arid environments.
    - In most of these species, basal metabolic rate has been shown to be suppressed by as much as 20-40% of the predicted value using Kleiber’s equation.
  + The main cause of depressed metabolism in animals that aren’t mentioned above is suggested to be due to starvation and/or dehydration stress of different magnitudes.
  + Studies showing depression in resting or basal metabolism have been conducted in a variety of domesticated animals (citations in article)
    - In most cases, the need to decrease metabolic rate has been given as an energy-saving response in order to stretch inadequate food or water stores over a longer period of stress.
    - In rats, this isn’t the case (***Bintz and Roesbery 1978***)
* This study: Would dehydration/starvation stress cause a de- crease in basal metabolic rate of animals whose metabolic rate is known to deviate substantially from what is predicted on the basis of body mass?
  + Primates with varying body masses, varying environmental habitats (dry to rainy, wide temp range)
  + Diet: 50/50 fruit:insects
* *In physiological research, dehydration has usually been assessed by the percentage reduction in body mass. The various levels of mass loss have a practical application in evaluating the severity of dehydration in order to rationalize fluid therapy (HALL, 1967; BLOOD et al., 1979). Additional evidence of dehydration is provided by examination of the elasticity of the skin, urine concentration, plasma osmolality, and fecal water loss all of which provide evidence of concomitant physiological responses driving fluid shifts into various compart- ments as dehydration process progresses.*
* *Weight reduction seems to be the only reliable measure of dehydra- tion. In animals with an efficient renal concentrating ability like the dik-diks, the plasma compartment is protected, thus making hematocrit, plasma osmolality, and total protein concentration unreliable indicators of dehydration (KAMAU & MALOIY, 1983)*
  + Dehydration was shown by a reduction in body mass (~25%). Osmolality of urine doubled (more concentrated)
    - Their *digestive system is important in water conservation*
  + A close up of text on a black background

    Description automatically generated*Respiratory quotient, RQ, is usually taken as an objective assessment of starva- tion. It indicates the bioenergetic substrates in use in the body. During starvation, metabolism shifts to fat utilization with a subsequent shift of RQ to 0.71 (Kleiber, 1961). The results indicate concomitant starvation stress with carbohydrates and proteins being catabolized.*
  + *May not have been held long enough to start metabolizing*
  + *it would seem from this study that bushbabies, animals with a metabolic rate preset inherently low, are unable to decrease their metabolic rate further in the face of starvation and/or dehydration stress.*
  + *Water restriction led fall in metabolic outside of the thermoneutral zone. The decrease is largely due to a reduction in body temperature during this time. Lack of metabolic adjustment implies either that the digestive efficiency is higher in these bushbabies or that the bushbabies live at the lower limit of their ability to acclimatize to starvation/dehydration stress.*

***Ross 1992: Basal metabolic Rate, Body Weight and Diet in Primates: An evaluation of the Evidence***

A close up of a map

Description automatically generated

**Nutritional geometry**

***Raubenheimer et al 2015 (w Jess Rothman)***

﻿Nutritional geometry has shown the benefits of viewing nutrition in a multidimensional context, in which foraging is viewed as a process of balancing the intake and use of multiple nutrients.

Rright-angled mixture triangle (RMT) –

* ﻿We use literature data from field studies of pri- mates to demonstrate how the RMT can provide insight into a variety of important concepts in nutritional ecology.
* **Isometric scaling** happens when proportional relationships are preserved as size changes during growth or over evolutionary time.
* **Isometric scaling** assumes that female fish reproductive output increases proportionately with size increases,
* while **allometric scaling** assumes that female fish's reproductive output increases **allometrically**. Here is an example of **allometric vs isometric scaling** from a paper meant to explain the difference.
* For every unit increase in size, there is a proportionally larger increase in body-part size.
* **Homeostasis:** The tendency of the body to seek and maintain a condition of balance or equilibrium within its internal environment (even when faced w external changes)
  + Implies a “constant state”
    - Being alive is a dynamic process – so misleading
  + Uses + and – feedbacks to perform this
* **Allostasis:** The process of shifting physiology or behavior to obtain a goal or establish a new homeostatic balance point – “readjustment”
* Stress is an imbalance between demands and resources that necessitates reallocation away from the “long term” plan
* **Allostatic load**
  + The chronic burden of repeated stressors
  + The cost of maintaining the state you want to be in
* ***Stress – definition:***
  + A perturbation of homeostasis
  + Stress response – suite of physiological and behavioral responses that attempt to restore homeostasis
* **What are some selective pressures that we expect infants to have evolved responses to solve?**
  + **Predation**- cry when left alone, make sure never alone
  + **Warmth**- body warmth from mom or caretaker
  + **Development of conscious breathing control**- mom helps monitor infant breathing
  + **Need of constant nutrients**- constant breastfeeding
    - Infants evolved to expect
      * Immediate & constant contact
      * Breastfeeding after birth
      * Nursing on demand
      * Contact and nursing throughout night
  + Evolutionary Adaptation: crying is adaptation to solve a problem
  + Behavioral: Babies cry and are reinforced to cry, and so on
* **Life history, energetics, reproduction, and reproductive fitness**

***Chisholm et al 1993: Death, Hope, Sex, Life History and Development of Repro Strategies.*** At the heart of evolutionary theory is the notion that in order to have left descendants all organisms must have solved the problems of survival, growth and development, and reproduction. In biology, the energy, time, and safety consumed in addressing these adaptive problems are are termed somatic effort (for survival or maintenance and growth and development), and reproductive effort (for repro). Reproductive effort itself is further dividing into mating effort (finding a mate, courting them) and parenting effort (gestation, childbirth, lactation and all postnatal care of children) (***Clutton-Brock 1991, Lessells 1991, Low 1978, Williams 1966.***)

* + **Life History Theory**: examining how NS produces adaptations that yield age-related, context-dependent tradeoffs b/w allocation of these resources
    - Diff ages: diff allocation of resources
  + Follow the Calories:
    - Tradeoffs can be quantified by following the movement of calories (proxy for resources)
      * Calorie transfer between individuals
      * Energy allocation within an organism
      * Calories extracted from environment
  + When you stop growing- you switch your allocation from growth to repro
* **Hormones and life history allocations**
  + Hormones are messengers through which organisms can alter their life history allocation
  + Regulate:
    - Growth
    - Menarche
    - Fertility
    - Pregnancy
    - Lactation
    - Immune function
    - Metabolism (also how much you eat, seek food)
  + Testosterone:
    - HPG axis: Testosterone
      * Masculinizing hormone
      * Change energetic costs
      * Decreased fat storage
      * Suppressing of immune system
      * Increases in muscles
      * Increases in mating effort
      * Reduction of pair bonding
      * Aggression, sexual behavior
      * Development of secondary sexual characteristics
  + **Example: Increased Risky Behavior**
    - Prox.:
      * Historical: Ontogeny: growing up in high risk environment causes increased risk taking
      * Single Form: Mechanism: changes in hormone levels (T) cause increase in risk taking
    - Ult.:
      * Historical: Phylogeny: a common primate trait in a high risk environment
    - Single Form: Adaptation: engaging in risky behavior in a high risk environment leads to higher reproductive success (and selected for)
  + **Example: Insulin regulates glucose metabolism** – regulation of available energy in your blood
    - Regulates glucose metabolism – how much glucose is taken up by muscle cells
    - Insulin: tells organs to take up sugar; blood sugar (BS) drops
    - No insulin: high BS
  + Other signals that tell the liver (energy storage) to break down stored energy and release into blood
  + **Homeostasis:** *The tendency of the body to seek and maintain a condition of balance or equilibrium within its internal environment* (even when faced w external changes)
    - Implies a “constant state”
      * Being alive is a dynamic process – so misleading
    - Uses + and – feedbacks to perform this
  + How is **stress related to life history allocations** between growth, reproduction, and maintenance?
    - Purpose of stress is to direct resources appropriately
    - Short term stress – adaptive, long term stress – can become pathological
      * If response is triggered but maintained inappropriate
    - Repeated stressors should promote desensitization
    - Actual experience is less stressful, but also body is getting used to feedback signal (you didn’t actually die from it)
    - **PTSD**
      * Exposure to a single high-stress situation, or repeated high stress situation can result in post-traumatic stress disorder
      * Constant activation and then return to normal can result in **dysregulation**
        + Decreases in cortisol response – reduced cortisol levels
        + Keeping system on all the time
        + Usually C peaks in the morning (***awakening response***), then decreases over the day

In PTSD: less daily change

* + - Stress & cortisol – allocation of energy
      * Cortisol increases energy mobilization
      * Increases attn. to surroundings
  + Energy mobilization affects life history allocations: growth, repro, immune fxn
  + ***Armstrong 1990:*** *Growth hormone, well known to be one of the hor­mones modulating somatic growth, is secreted by cells in the anterior pituitary gland. As a pituitary hormone it has an intermediate role in the cascade of hormones modulat­ing body growth, receiving information from the hypo­thalamus and controlling postnatal somatic growth by regulating insulin-like growth factor I (somatomedin C).*
  + ***Hormonal regulation of menstrual cycle:***
    - **Hypothalamus** releases **GnRH**
      * Causes pituitary to produce **FSH**
      * FSH 🡪 **ovaries**
        + Causes follicles to mature
    - Maturing **follicle** produces **estrogen** & **inhibin**
      * Estrogen 🡪 **brain**, signals to change behavior
      * Slows down **FSH** production (***neg feedback;*** prevents another egg from forming)
      * Starts **LH** production in **Pituitary**
      * Estrogen prepares uterus for ovulation – thickens blood vessels lining the uterus
    - Estrogen peaks !!! & levels off
      * LH, FSH surge (***pos. feedback*** *from estrogen*) and cause **ovulation**
    - **Ovulation**, egg travels via uterine (fallopian) tube 🡪 uterus
    - Empty follicle 🡪 corpus luteum 🡪 produces **progesterone** (pro pregnancy)
      * Progesterone 🡪 brain stop making **LH**
      * Prepares uterus by thickening the **endometrium**
    - Egg waits 24-48 hours
    - If oocyte not fertilized, corpus luteum disintegrates (b/c less FSH&LH) 🡪 **corpus albicans**
      * Stops producing progesterone & estrogen
      * @ day 28, **low levels of P&E** (you lose***neg. feedback***)🡪 **menstruation**
    - Less E&P 🡪 FSH no longer inhibited; Cycle starts over
* How does hormonal birth control work?
  + Interferes w menstrual cycle
  + Most use either E & P (or just P)
    - Suppress ovulation and thicken cervical mucus (reduces sperm motility)
    - P limits ovulation
    - Works via ***neg feedback*** (so that another egg doesn’t ovulate) 🡪 suppress GnRH 🡪 suppresses LH & FSH 🡪 prevents LH surge that causes ovulation
  + The Pill
    - 21 days of P or P&E
    - 7 days placebo
      * (P maintains lining) so removing P triggers menstruation
      * Adding it back in early cycle blocks LH surge
  + Depoprovera
    - Injects a big mass of P 🡪 slowly gets absorbed, keeps P high for months
  + IUD
    - Hormonal
      * Progestin (form of P)
    - Copper
      * Toxic for sperm, recruits immune activity to attack sperm (heavier cycle, cramps)
      * Causing irritation
  + Rhythm method
    - Using tacking of menstrual cycle to try and avoid pregnancy
      * Only ~60-80% effective if done exactly right; why?
        + Irregularity in cycle
        + Seminal fluid contains FSH and LH

To promote ovulation

* + - * + Male sweat contains pheromones that affect LH secretion in women
  + Sex might accelerate follicular development
* **Menstruation and why it occurs?**
  + Shedding of endometrium when implantation does not occur (as a result of decrease in P)
    - common among primates - homologous
  + Why shed/why flush/why overt/covert
    - Protection against STIs (pathogens)
      * endometrium is rich environment
      * good envi for bacteria/others
      * if no pregnancy – good idea to wash out and start over clean
      * especially important in promiscuous spp
    - byproduct of **“spontaneous decidualization”**
      * in many spp. the final differentiation of the endometrium into decidua happens after conception
        + requires trigger from embryo
      * humans and apes it happens automatically, w/ or w/o pregnancy
      * spontaneous decidualization may have evolved as maternal defense against overly invasive embryo
        + sloughing off is developmental change- change to decidua for maternal protection against invasive embryo
        + once you turn it into decidua, you can’t turn it back- have to get rid of it
* What is implantation?
  + Egg is fertilized 🡪 become embryo 🡪 migrates and implants in endometrium
  + **Invasiveness**: degree of placental connection between maternal and fetal nutrients/blood
    - # of layers of tissue separating maternal and fetal tissue
* What are the three major categories of placentas and how do they differ?
  + A close up of a piece of paper

    Description automatically generatedEpitheliochorial 2 (horse, swine)
  + Endotheliochorial 1 (dog, cats)
  + Hemochorial – fetal tissue bathes at the end of the mother’s blood (humans, rodent)
    - more invasive placenta- more transfer of immune factors and nutrients
    - need a lot of nutrients for brain growth
  + Depending on the # of layers- invasiveness; more transfer of nutrients with more direct transfer of blood
* What is preeclampsia and how does it relate to immune function?
  + Preeclampsia
    - 2-8% of pregnancies
    - consequence of shallow implantation
      * doesn’t connect well to mother’s blood flow (spinal arteries)
    - mom gets high BP
    - Protein in the urine
  + Eclampsia
    - All the above, w seizures
  + Poor placentation
    - Oxidatively stressed placenta
    - Inflammatory immune response in mom
    - Fetus might fight back
    - Conflict may cause symptoms
  + Most common during 1st pregnancy
  + More common when women switches partners
    - Resembles symptoms of tissue rejection
    - Prolonged exposure to paternal semen reduces risk
      * Maternal tolerance of paternal genes in semen an infant is important
  + **High BP may be due to maternal-fetal conflict**
* What is **maternal-fetal conflict**?
  + Natural selection acts on fetuses to extract more resources from mom than is optimal for mom
    - Mom and fetus have different interests
    - Especially in mating system in which there is low prob. of same father for multiple offspring
      * Means mom has to deal with multiple lines of invading DNA
    - Acts on mother to limit fetal transfer
      * Arms race between mom/fetus
    - Spontaneous Decidualization may protect against over invasive fetus
  + **CONFLICT:** Mother and fetus do not carry identical genes
    - Their interests do not overlap 100%
    - Especially if fetus has low relatedness to potential siblings (diff father)
    - Conflict of maternal vs paternal genes:
      * Mom:
        + Trade-off against current vs future reproduction
        + Also trading off against own maintenance
      * Dad:
        + Wants offspring to grow as rapidly as possible
        + Paternity is always 100%
        + Partner’s future offspring may not be his own, so wants this one to get it all
  + Study shows that fetus extracts more resources from mom when the odds of high relatedness to siblings is lower
    - Leaves mom with less resources to invest in future offspring
      * **How this happens: Imprinting**
        + Epigenetic marks that signal whether gene came from mom or dad (methylation group on DNA)

Intergenomic conflict

Like genes affecting IGF

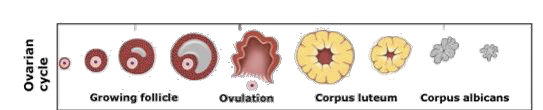
Genes from dad may want to take more resources than mom can provide

* + What are some possible consequences of maternal-fetal conflict? (e.g. defense against invasive embryo, gestational diabetes, preeclampsia)
  + **Other complications during pregnancy:**
    - **Gestational diabetes**
      * Mom’s insulin responds to glucose is exaggerated during pregnancy
        + But insulin less effective
      * **Evo:** Argued that fetal hormones manipulate maternal response to isulin to make more glucose available
* What is fetal selection? Why does it occur? How does fetal selection make sense in light of parental investment theory?
  + Mom will screen for best embryo
    - If embryo isn’t very strong, better to abort and start over than invest in that embryo for a long time
    - First Trial:
      * First thing embryo has to do is signal to mom that it’s there (Don’t menstruate!)
        + Produces Human Chorionic Gonadotropin (hCG) 🡪 binds to same receptors as LH
        + Keeps corpus luteum alive and secreting P
        + P maintain endothelium
        + (at orders of magnitude higher than ovulatory E and P)
  + A close up of a device

    Description automatically generated**Fetal Selection**
    - 2/3 of pregnancies are lost within days
    - from evo standpoint-
      * mother’s best interest to invest only in best embryo
      * embryo’s best interest to commit suicide if not viable, but better to survive if okay, but not the best
        + hCG prodxn is first thing it needs to do to prove it is working
        + spontaneous decidualization may help mom to screen for less fit embryos
    - Follicular maturation
      * Only best one survives
        + Mom doesn’t want to take care of a lesser offspring
        + Fetus doesn’t want to be born if it doesn’t have the best genes

Sibling might have better ones, so better to abort and make room

* + - Production of hCG to prevent decidualization (prove fetus is working);
      * ***what pregnancy tests look for! (diffuses into urine)***
    - Decidualization help mom be selective on how strong embryo is
* What are the hormones that maintain pregnancy?
  + hCG🡪 keeps corpus luteum alive🡪 produces Progesterone
    - maintain endothelium
  + P & E skyrocket during pregnancy
  + hCG as signal of Fetal Quality
    - lower hCG = poor quality embryo and were terminated earlier
* What is a possible explanation for morning sickness? Why might it be better not to treat morning sickness?
  + Most women experience morning sickness
  + Wasted calories
  + Hypotheses:
    - Protection against toxins/parasites
      * MS symptoms peak weeks 6-18, when organs are forming
        + When toxins from envi are most likely to interfere w development
    - Women who vomit are least likely to miscarry
      * Those that vomit more, even less likely
    - Aversion to meat
      * Aversion to meat
        + Parasite avoidance
    - Craving for sugar
    - Aversions:
      * Never see aversions for staple crops
      * Aversion to meat
      * Aversion to a lot of plant products – contain plant toxins
        + Things we can handle as adults wont be tolerated by developing fetus
* What are fertility and fecundity for demographers? What are they for biologists?
  + **Biologists:**
    - Fertility: capacity to have kids
    - Fecundity: rate which she actually has kids
  + **Demographers**
    - **Fertility**: rate at which she has kids (# of kids)
    - **Fecundity**: capacity to have kids
  + Probability of conception (at any age) highest when copulation is days -2, -1 before ovulation
    - But probability itself decreases w/ age (still peaks at -2, but lower rate of conception)
  + ***Fecundability*** – probability of conception with any given cycle
    - Peaks at 22 (highest likelihood to conceive at any cycle)
* A screenshot of a cell phone

  Description automatically generatedUnderstand the different gradations of **reproductive suppression**: follicular suppression, luteal suppression, anovulation, and amenorrhea.
  + **Follicular Suppression** (fecundity somewhat suppressed)
    - Slower follicle growth 🡪 lower estradiol 🡪 lengthen follicular phase (before ovu)
    - Can lead to… Lut. Sup.
  + **Luteal suppression** (more suppression)
    - Limited follicular growth 🡪 limited corpus luteum 🡪 less P 🡪 shorter luteal phase
  + **Anovulation** (no pregnancy)
    - No ovulation 🡪 no increase in P at end of cycle 🡪 anovulatory menstruation may still occur, but timing is off
  + **Amenorrhea** (no pregnancy)
    - No menstruation
* How does menstrual cycle length vary with age?
  + Starts off irregular, becomes fairly consistent (but shortens with age), then variable again
  + **Shorter and more regular as you age, then irregular towards menopause**
  + High levels of within-woman and between-woman variation
  + High levels of age-specific variation and rate of menses
* How is physical activity related to amenorrhea?
  + Amenorrhea: no menstruation from a variety of reasons
    - No follicular, no luteal phases
  + Women who engage in high levels of physical activity experience amenorrhea
  + Not correlated with body fat- but with Eflux
* **Estrogen/estradiol and progesterone and conception risk**
  + Within individual:
  + Progesterone: after ovulation maintains endometrium
    - Important for implantation and maintenance of fertilized egg
    - Lower = lower risk of pregnancy
    - Higher = higher risk of pregnancy
  + When Progesterone is used in follicular phase, can be used to block ovulation
  + Estrogen: increases before ovulation
    - Highest when highest chance of conception
  + Hormones and Conception:
    - Estrogen is higher in conception cycles
    - **Women who have a hard time conceiving seem to have chronically low P**
      * But don’t hold across populations
    - Hormones levels are not connected to fertility in an absolute sense
      * Within-woman variation / between woman variation
    - Symmetrical women have higher fertility
  + Nutrition affects development
    - Asymmetry correlates with infertility
    - Allocation of energy during development
  + **Low progesterone 🡪 infertility?**
    - Having low progesterone is not an indicator of infertility, as women in other countries become pregnant with low levels of P
    - **Ex. Bangladeshi Migrants:**
      * Those who left bangladesh very early in life have higher levels of P
      * Those who left a bit later, have slightly lower
      * Those who left late in life have levels comparable to the pop in Bangladesh
    - **Set point:** 
      * Set point for repro hormones around puberty
      * Good Envi: maintain higher baseline levels of P (and Ovu)
      * Bad Envi: maintain lower baseline levels of P (and Ovu)
    - Implications to hormonal birth control
* How do these hormones vary cross-culturally, and what does this tell us about the absolute meaning of hormone levels (as opposed to relative within person or within culture levels)?
  + Between individuals:
    - Relative to that same person’s other cycles
    - Relative to general group (if they all have much lower levels, but still changes in levels)
  + Set up early in life- migrants moved from high risk environment to low risk
  + Progesterone increased
* Approximately what fraction of pregnancies are lost before the women even knows she is pregnant?
  + 2/3 but difficult to know (some are lost very early without us knowing)
  + lost within the first few days
  + only outward sign is longer period
* How are early pregnancy loss and anovulation related to stressors such as physical work and psychosocial stress?
  + Exercise a lot = suppression of reproductive function
    - Anovulation can be from full suppression of … (34:20)
  + Change in Eb and Eflux both important factors
    - Not overall Eb (thin, heavy), but deltaEb (how fast you’re losing E) (After 35:00)
  + Change in seasonal intake of nutrients (right before harvest season) – increases pregnancy rates
  + Bolivian women in highlands: hard work = increase in pregnancy loss
* What is microchimerism (both maternal and fetal)?
  + Transfer of cells between mother and fetus
  + Fetal cells in mom- fetal microchimerism
    - Fetal cells involved in … (time: 23:00)
  + Mom cells in fetus – maternal microchimerism
    - Mom cells go to where the fetus body is developing (so that fetus immune system does not attack mom!)
      * Help fetus tolerate mom
    - Carry info to fetus about the kind of diseases mom has been exposed to (help regulate fetal immune system in prep for environment)
  + Fetal microchimerism- only detected by Y chromosomes (only sons, not daughters)
* What are some possible consequences of microchimerism?
  + Fetal cells can help w healing
  + Influence hormones related to milk production or mother/offspring bonding
  + Can cause autoimmune damage or cancers
  + If they’re there- provides an opportunity for NS to act on them
    - So if they benefit, they should be selected for
* What are some immune changes with pregnancy, and why do they occur?
  + Why: fetus is only 50% mom; mom’s immune system might recognize fetus as foreign invader
  + Immune system needs to be changed to **tolerate fetus**
  + Ways for cells to identify self/non-self (MHC)
    - Pregnancy modifies these mechanisms
  + Because of these changes- changes is rates of other diseases too- RA, systemic changes (recording time: 21:50)
  + Disease and Pregnancy: **not as reduced as someone who is immunocompromised**
    - Shifting of one kind of immunity for another
    - **Doesn’t suffer from opportunistic infections**
* **Menstrual Cycle**
  + **Steps of the menstrual cycle:**
    - Follicular Phase – Growth of follicle (part that releases egg)
    - Ovulation – egg is released
    - Luteal phase – corpus luteum releases progesterone
    - Menstruation – uterine walls sloughed off
  + Regulated by both (-) and (+) feedback
  + Follicular Phase:
    - Follicle develops in the ovary
      * A major endocrine producer
      * Releases the egg
      * Becomes corpus luteum after ovulation
      * Yellow because collects cholesterol, used to make progesterone
* **Menstruation** 
  + A close up of a map

    Description automatically generatedLining of the uterus (endometrium) receives signal of progsterone and stars preparing in case of pregnancy
  + Thickens
  + Endometrial cells 🡪 decidual cells
    - If an embryo implants – becomes part of placenta
  + Maintenance of decidua requires progesterone
  + When corpus luteum degrades, it stops producing progesterone
    - If nothing implants, decidua sloughs off
    - Spiral arteries behind decidua open up
      * Platelet poor (so no clotting), WBC rich blood flows, washes out endo cells
* A close up of a map

  Description automatically generatedFollicular Development
  + Primordial 🡪 Primary 🡪 Secondary 🡪 Single, selected tertiary follicle (only the best one)
  + **Atresia** – degradation and resorption of follicles that are not selected (discarded)
* Idealized vs. Real Menstrual Cycle
  + Ideal: 28 days (ovul. day 14)
    - Actually rare
  + High levels of within-woman and between-woman variation
  + High levels of age-specific variation and rate of menses
* Primates advertise oestrus with perineal swellings
  + Graded signal- encourages mating with all males
    - Makes mating with dominant male most likely to actually conceive (mate at peak swelling)
    - peaks when ovulating
    - defense against infanticide
      * they all have some probability to sire the offspring, so won’t kill
* Concealed ovulation- an adaptation?
  + Evo of sex swellings at least 3 times in primates
  + Our common ancestor likely didn’t have them-
    - We didn’t lose them, we just didn’t evolve them in our lineage
    - Whereas chimps did
* Advertising Ovulation in Humans
  + A close up of a piece of paper

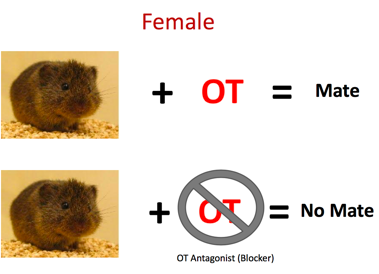
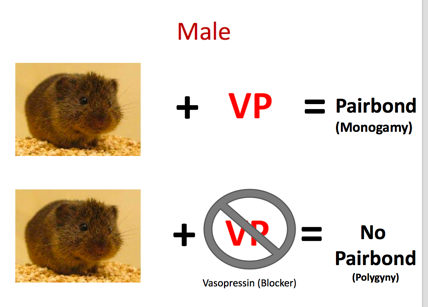
    Description automatically generatedWomen are more interested in mating
  + Wear skimpier clothing
  + More likely to cheat
  + Physical changes – symmetry, skin quality…
* Organizational Effects of Ovulation
  + Male pituitary cannot cause ovulation in implanted ovary
  + Exposure to androgens prenatally destroys the GnRH center in the hypothalamus
    - Permanently eliminates the potential for positive feedback
* Fetal Growth
  + Week 3-5: CNS, heart (then throughout dev.)
  + 4-8: head, extremities
  + 6-9: teeth, external genitalia, ear
  + Growth is pulsatile
    - But more synchronized in females
    - More **canalized**
  + Females grow limbs earlier
  + Males show more **fragility**
    - Fits with higher male spontaneous abortions
* Trans-placental Immunization
  + Mom transfers antibodies and maternal cells across placenta (via vesicles)
  + Transfer related to invasiveness of placenta and gestation length
  + Birds, reptiles, fish, all transfer through eggs
  + Mammals
    - Depends on placentation and length of gestation
  + Humans transfer a lot during pregnancy, less during lactation
    - Primates about half and half
    - Lesser mammals transfer less during pregnancy, more during lactation
* Intergenerational Transmission of Immunity
  + Infants are born with some defense about what they should encounter
    - Ex. infants born in Kenya – infants had levels of antibody specific to those things
  + Infants that are exposed this way
    - Some will gain protection
    - Some will gain tolerance
  + Mom’s body not only giving defenses, but telling them how to respond
    - Sometimes it’s best to tolerate, not fight
* Maternal cell transfer (maternal microchimerism)
  + Affect infant immune response
* Costs of Pregnancy/Lactation
  + Pregnancy extra 90/290/470kcal/day
  + Lactation extra 626kcal/day
* Early Fetal Loss
  + Prob. of fetal loss increases from 45% at age 18 🡪 92% at age 38
  + Detection is key, most go undetected
  + Anovulation in highland Bolivians
    - Risk of anovulation is fairly consistent throughout year with slight dip in winter
      * When environmental conditions are least favorable
    - Turkana Pastoralists:
      * Conception rates increased when there were most resources available (milk)
      * Increased right before time of year when women’s body weight highest
    - US: most births are in summer
    - Means conception is potentially highest in the winter, in prep to have summer babies
      * When conditions were favorable
  + *Maybe body monitors the past 3 months in prediction of what the environment will be like when baby will be born, if conception occurs now*
* Fetal Selection- Not Random!
  + Males are more likely to be aborted
    - Male embryos are larger than females
    - At any time in life- more men are dying
  + Men aged 15-30 are 3x more likely to die than women
  + If times are bad – terminate the male
    - Might be best option
    - More costly to carry males
    - Developing male under poor conditions will be less likely to find mate
      * Bad for everyone
    - Ex. ***Sex ratio after disasters seems to skew- more females***
  + Trivers-Willard
    - Even at max, women produce much fewer offspring than males
    - If you are in good condition, you can pass it off to offspring
      * Then better to have male
    - If you are in poor/moderate conditions, and you have a son, then he will have reduced RS
      * Better to have female – she could still have mates
  + Assumptions
    - Parental condition is associated with offspring condition
    - Differences in offspring condition will persist into adulthood (repro age)
* **Hormones related to labor and birth**
  + **Progesterone** – maintains pregnancy
  + **Cortisol** – signals maternal and fetal energy status, metabolizes glucose (moms can get gestational diabetes b/c of glucose demands…?)
  + **Prostaglandins** – initiate labor (softening of cervix)
  + **Oxytocin** – smooth muscle contractions, orgasm hormones, but orgasm does not necessarily stim birth
    - Bonding/birth hormone- arose for birth, because it was already there- was naturally occurring signal for mom/baby bond; got co-opted for not just mom/baby bonding, but all sorts of bonding (hypothesis, but no evidence)
    - **Pitocin**: synthetic oxytocin stimulates contractions
      * When you induce labor, not all systems are ready to go- so contractions are slower, birth takes longer, may decide to do c-sec
  + **Relaxin** – softens the pelvic cartilage, increase mom’s cardiac input
* **What is the** **metabolic hypothesis for birth timing?**
  + Trigger: readiness of fetus – “all systems are go”
  + When fetal energy demands exceed mother’s ability to meet those demands through placenta
    - Fetus becomes hungry – correlated with when birth happens
      * 38+ weeks: full term
      * 24 wks – viable
* **Why is human birth so difficult?**
  + Constraint of bipedalism: birth is difficult because of pelvic shape
    - Non-human primates – birth canal is mostly straight tube and head is small
    - Humans- because of bipedal pelvis, selection for big brains, head needs to rotate to fit through pelvic inlet/outlet, can cause complications
      * Walking would be less efficient if pelvis was larger to accommodate
  + 3 problems w human birth:
    - 1. Head is big (brain bigger- can’t get any bigger before coming out, so birth earlier, less developed than other primates “altricial”)
    - 2. fixed shoulders for babies
    - 3. Bipedal constraints on mom’s pelvis
      * human head is ovoid, birth canal starts widest side/side, then exit is widest back/front – baby must rotate to align
* **How does the baby rotate during birth?**
  + human head is ovoid, birth canal starts widest side/side, then exit is widest back/front – baby must rotate ¼ turn to align
  + born facing towards mother’s back – Occiput anterior
* **How does the difficulty of birth relate to the need for social support during birth?** 
  + Human infant born less developed (so it can pass through pelvis)
    - Pelvis became broader to support bipedalism first
    - Then brain grew
    - Secondarily altricial (less dev), derived trait due to bipedality
      * ***Exogestates***- can’t live on their own, GI tracts isn’t developed
      * Parental care is more important
  + Primates vs humans:
    - In primates they rotate, but they come out facing front- so mom can pick up and go
      * ***Can do it on her own!*** Can help baby come out
    - Humans- better to be squatting, not lying down
      * Since baby comes out facing posteriorly, mother can’t reach down to help
      * Might create a situation of “***obligate midwifery***” – difficult to give birth on their own
        + Put selective pressure to make humans want others around during childbirth
        + In almost all societies- do not give birth alone
      * Stress or lack of social support delays labor
        + Ex. Guatemala c-sec is 27% w/o doula, 19% with
        + More comfort- less complications
  + **How does this relate to birth outcomes and different cultures/traditions/methods of birth? (Such as in the Born in the USA video)** 
    - Maternal mortality – gone down, but still higher in less developed countries
    - Global causes for M=mortality causes:
      * Hemorrhage 27%
      * Hypertension 14 % (preeclampsia/eclampsia)
      * Sepsis 11%
      * Abortion 8%
      * Preexisting i.e. diabetes, etc. 28%
    - Lifetime risk of maternal death: sub-Saharan Africa highest 1/36
    - U.S. has high maternal mortality compared to other developed countries
      * Depends on racial group (non-Hispanic black at highest risk)
      * Access to HC, and preexisting conditions (diabetes)
  + **How does this relate to postpartum depression?** 
    - Moms that are more likely to be single or have marriage issues are more likely to have PPD; can be influenced by socioeconomic state
  + **How can we use parental investment theory to think about postpartum depression?**
    - Strongly related to social support – 2 purposes:
      * Mechanism to disinvest from baby that mom can’t afford (strategy)
      * Or, mechanism to signal that support is needed
    - ***Postpartum depression (PPD)***
    - PPD- those most likely to exist in women likely to have least social/familial support
    - PPD quite common, increasingly so when people live away from families or other social support
* A screenshot of a cell phone

  Description automatically generated**Hormones involved in lactation**
  + **Prolactin** – redirects energy into the breast for milk production
    - EST + PROG increase during pregnancy
      * Drop off after birth
      * Removes a “block”
    - PRLC increases during pregnancy to trigger milk glands to produce milk
      * Don’t start secreting until dropoff
      * Holds it in check
    - Milk secretion increases after birth
      * Colostrum
      * Trans milk
      * Mature milk
  + Nipple stimulation – releases PRLC and OXYT
    - Nipple stim 🡪 spinal cord 🡪 CNS 🡪 brain stem 🡪 hypothalamus 🡪 keeps PRLC secretion going in Ant. Pit 🡪 to mammary glands
    - Actual nursing needs to occur to keep prdxn going
    - When you stop breastfeeding- prdxn can stop relatively quickly
  + OXYT – nipple stim 🡪 spine 🡪 CNS 🡪 post pit. 🡪 OXYT 🡪 mammary glands
  + Milk insufficiency: most common cause is low intake by baby
    - Decreases prdxn
    - What is cause:
      * Breastfeeding: poor attachment, delayed initiation of breastfeeding, infrequent feeds, short feeds, bottles or pacifiers, nipple shield, giving other food/drink
      * Psychological factors: depression, lack of confidence, worry, stress, dislike
      * Baby – illness, prematurity, congenital abnormality (palate, heart, kidney)
    - Milk
      * True milk insufficiency is rare, caused by
        + PRLC deficiency (pituitary dysfunctions, medications [dopamine], nicotine, alcohol)
        + Bulimia – low E, downregulate many things (incl frertility)

Energy constrains and supplements seem to not have effects- highly buffered

* + - * + Retained placental fragments (responsible for producing EST+PROG- without dropoff, no PRLC prdxn)
        + Breast surgery
        + Obesity
        + Postpartum hemorrhage
* **What are the components of breast milk?** 
  + Colostrum – immunology rich milk, mild laxative to clear first stool
  + Later milk – highly buffered against mom’s nutritional condition
    - Hard to influence with supplements
    - Mobilization of specialized maternal fat stores – important for brain development
    - ~600 cal /day for lactation
  + Immunological
    - Immunoglobulins – protect gut, and by take up in blood stream
    - Fatty acids
  + Hormonal
    - E.g. leptin – signal of maternal fat stores
      * Baby gets indicator of mom’s nutritional status (can have organizational effects)
  + Nutritional
    - Lactose, oligosaccharides, fat, protein, vitamins, nutrients
  + Other
    - Stem cells
    - siRNAs – messenger – protect baby against viruses, or affect gene expression
    - Food components – what mom is eating gets transferred, helps baby learn what is edible (Ex. those exposed to certain foods are more likely to favor that food)
      * Tsimane – omega-3, fatty acids
      * Carrots – babies had increased preference for carrots in cereal
      * **siRNA**: transmission of maternal genes into neonate
        + affect gene expression in infant cells
        + milk contains pluripotent stem cells- maternal stem cells transferred in milk
      * **antibodies**: transferred more during placental in more invasive spp (less in milk/colostrum)
        + transferred least in animals with less invasive (more in milk/colostrum)
        + humans: more transfer during pregnancy, less after
  + **Why might breast feeding be better than formula feeding?**
    - BF: Less likely to develop obesity ~20%
    - Lower mortality, decrease GI illnesses, lower risk of diabetes, some evidence for IQ too
    - Bottle feeding means larger portions per sitting 🡪 obesity
* **How is breast feeding related to fertility?** 
  + **How is energy balance important for lactational amenorrhea?**
* **What is lactational amenorrhea?**
* **How is energy balance related to the length of lactational amenorrhea?**
  + Lactational amenorrhea (LAM) – lack of cycling after pregnancy (varying amounts of months without cycling)
    - No menstruation, no ovulation
  + Disagreement on what causes LAM
    - Bfeeding? Mom is losing weight (ton of calories)? Probably not feeding itself
  + Sensitive to Eb changes – how fast the change is
    - Physiology: PRLC levels? Constant/fluctuating levels?
      * **Proximate**: frequency, duration of nursing
      * **Ultimate**: Life history:
        + high energetic cost of lactation; optimal birth spacing
        + human birth spacing shorter than apes

affecting sensitivity of LAM can be tool to space out births

* + ***Metabolic Load Hypothesis***
    - cost of prdxn is relative to each women (depending on energetic resources and stores)
    - ***so cost of lactation (rather than lactation itself), is what determines length of LAM***
  + **What are some lines of evidence suggesting positive energy balance triggers resumption of menstruation?**
* **Ex. Toba women** (Argentina): started menstruating again when minimum threshold of *positive Eb* was reached
  + Beforehand they were losing weight, then level out, then gain weight again, and when they had Net (+) Eb (i.e. gaining weight), they ovulated again
* **Ex. Chimps**: C-peptide: low c-pep (a measure of Eb) before, and cycling resumes after period of (+) E gain
* So women losing weight will stall ovulation during LAM (similar to ovulation in general)
  + Functional – system would select to work this way – to stall when mom does not have enough E to carry again
* **What are some cultural trends that influenced our understanding of lactational amenorrhea? (Hint, this is from the Ellison reading.)**
  + Studies on how many children women produce- depend on things that influence physiology:
  + Maternal E demands/dietary quality
  + Weaning- when babies introduced foods, stop getting as many calories from mom- mom produces less milk, less E demand on mom, can start ovulating again
    - Cultural decisions
    - Formula feeding
  + Parental investment costs: cost of having kid (money, time)
  + Sex norms and taboos about how long to wait before weaning
  + Duration of LAM – ultimately influences IBI (different across cultures)
    - Kung: 4yrs; Ache: 3-4yrs; Tsimane: 2/5yrs
    - Moms can counter LAM (reduce IBI)
      * Introduce complementary foods
      * Initiate full weaning
      * Alloparenting: other parents supplement food, not mom- positive Eb to mom…
  + End breastfeeding ~29mo
* **What is parental investment?** 
  + **PI:** investment in offspring that increases its chance of survival (and RS), and **reduces** parent’s ability to invest in other offspring
    - **Ex.** protecting young, feeding, producing gametes- eggs or sperm
    - **Conceptually-** PI is **costly**,PI is a **limited resource**
  + **How can we conceptualize as investment more generally, including alloparental investment?** 
    - Subset of Life History
    - Indivs allocate resources between
      * Different juveniles
      * Current/future offspring
      * Quantity/quality of offspring
  + **What might influence who invests in which children in different economies, cultures, or environments?**
  + Parent/fetal conflict: like maternal/fetal conflict
  + Indivs are expected to invest differentially in different juvenile as if in response to these questions:
    - Is he related?
      * Diff degrees of paternal certainty (also for grandparents), in diff cultures
        + Higher degree of promiscuous mating
        + Economics
    - Likely to survive and have kids of its own? (translate my investments into RS)
    - How much can I afford to invest?
      * How much does juvenile need my investment?
      * Can it get investment from someone else, or acquire on its own?
    - How much a kid costs? (college/edu)
      * What are my alternate uses of this investment?
    - All these vary across cultures
  + A lot of alloparental care in humans: and juvenile self-provisioning; food transfer; homosexuality
* **What is couvade? *= pregnancy symptoms in males***
  + Men with pregnant wife work more than those with an nonpregnant wife; pregnant wife works less than nonpregnant wife
  + Men go through changes in preparation for paternity
    - Couvade: males of many spp gain weight or experience symptoms when their partners are pregnant
    - Male calitrichids (marmosets, Tam) gain as much as 10% of body weight
    - Humans: ~20% of men simultaneously experience pregnancy symptoms (couvade)
      * Gain weight, nausea and vomiting, toothaches, appetite loss, abdominal pain, joint swelling
* **What are some of the hormonal changes we see in men that might prepare them for fatherhood?**
  + PRLC: higher in men sampled in last few weeks before birth
  + EST:
  + CORT (stress responsiveness): higher before birth
  + T: decreased immediately after birth
    - Redirects E from immune fxn towards mating effort
    - Decline depends on time spent with children
    - Cosleeping reduces T
  + Prepare men for fatherhood, like women
* **How are oxytocin and vasopressin related to bonding in humans and voles?**
  + Bonding hormone
  + Released at birth, during lactation, by moms holding infant or hearing it cry
  + Increases in babies too
  + Increases in males when exposed to babies
  + Helps expel placenta after birth
  + ***Women with selective c-sec do not experience increase in OXY***
    - Repost less bonding with infant immediately following birth
  + OXY helps increase investment/love in something that would otherwise suck
  + Occurs in most mammals
* **How is oxytocin related to breastfeeding?**
  + OXY: contracts smooth muscle in breasts to squeeze milk into duct system
    - Helps eject milk (“let down reflex”)
* **How is oxytocin related to bonding and other social behaviors?**
  + Hugging, cuddling increases OXY
  + Increase only when shown pic of own partner
  + Relationship quality associated with higher OXY in men/women
* **What are some experimental techniques used in oxytocin studies?**
  + Voles
    - Promiscuous vs monogamous
      * Injected w OXY – how would behavior change
    - Also used OXY blocker (opposite effect)
  + Parents and kids OXY
    - Fathers given OXY touch their kids more, show more social reciprocity
    - Infants given OXY look at faces more and play more with toys given by parents
    - Inhaled (nose)
    - Absorbs and gets into brain
  + OXY increases after orgasm
  + PRLC increases after orgasm
    - (men and women)
  + EPIN, NOREPIN: increases after orgasm
    - CORT decreases
  + OXY beneficial for pair bonding
* **Why do oxytocin researchers study voles? What are the two kinds of voles studied, and why do they vary?**
  + Prairie: not human, good model
    - Engage in monogamous sex, also more than necessary for repro
    - Males help raise young
  + Montane:
    - Closely related
    - Polygynous- males leave immediately after intercourse to seek additional males
  + Same amount of OXY, different amount of receptors for OXY-
    - Monogamous voles (prairie): higher concentration of receptors in reward area of brain
  + **VOLES:**
  + Females:
    - Injected with OXY antagonist (blocker): lose interest in mating/pair bonding
    - Inject with OXY: want to mate immediately
    - Inject with vasopressin: nothing happens
  + Males:
    - injected with VASO blocker: become polygynous
    - injected with VASO: become monogamous
    - Injected with OXY: nothing happens
  + 🡪 Diff hormones have diff effect in M/F
  + ****Human males:
    - exposed to OXY, males in relationships paid less attn. to attractive women
* **What does vasopressin do for voles?**
  + Related to OXY – has similar fxn to bonding, only in male voles (monogamous)
  + Prairie voles have more receptors for VASO than montane
    - Males: Higher concentration of receptors in the reward part of brain
* **Menopause:**
* Age at menarche: earlier- more likely to get menopause earlier
* Contraceptives: later age menopause
* Duration of contraceptive use: 10+ years- later menopause
  + - ~2.5yrs later
* Shuar women, Ecuador
  + # live births: more births 🡪 later age of menopause
* Oral contraceptives:
  + OC use: later age menopause
* (opposite to Shuar): Across countries, higher fertility ~ earlier menopause
* Smoking 🡪 early
* Fertility:
  + Earlier across populations
  + Later within populations
* **Likely:** # menstrual cycles and physical condition the woman is in- what kind of damage is done to those follicles
* **How is the number of children has related to age of menopause?**
  + Women with
  + Across populations
    - Menopause is happening earlier in pops with high fertility
    - But ecological effect- income, pathogen load, that in itself may be causing early menopause
    - Higher rate of loss of developing follicles
  + Within populations
    - More births- later menopause
      * If you’re pregnant, you’re not having cycles/ less disposal of eggs
* **How is oral contraceptive use related to age of menopause?**
  + Delays age of menopause
    - Reduces # of ovulation cycles
    - But what also matters is all the follicles that also develop
    - Suppressing that process delays menopause (when you run out)
* **What triggers menopause?**
  + Reduction in # of eggs left
  + As there are less, more follicles need to develop to produce one good egg- depletion of follicles
  + Rate of follicular loss increases w age
* **Why is there a depletion of follicles throughout the lifespan?**
  + Follicular depletion:
    - # follicles in ovaries:
    - There are less surviving follicles with age
    - Higher rate of depletion with age
      * Because you go through more eggs before you find a good one
  + Once depletion of eggs becomes high enough, menopause initiates
  + Tsimane: are cycling about 50% of their life,
    - Other portions: lactation or Amenorrhea
    - Have less cycles that American women; expected to menopause at later age-
      * But we don’t see it
      * Likely environmental factors (disease, energy)
* **How many cycles do American women typically have in a lifetime? How does this compare to some subsistence populations?**
  + Tsimane- only cycling half of their life (either pregnant of lactating during the rest)
  + # of cycles during life:
    - 350-450 for American women
    - 150-250 for Tsimane
    - 100 for Dogon of Mali
  + More kids = less time for cycling
* **What is follicular atresia?**
  + At most, maybe 400 are lost to ovulation, so ovulation not main cause of atresia
  + During follicular dev. – many start to dev., **but only one can survive**
    - The rest undergo apoptosis
      * So each ovulation results in loss of many follicles
  + EST seems to inhibit atresia, while Gonadotropins increase atresia
  + So parity and birth control are associated with later menopause (less loss)
  + Environment:
    - Smoking, disease, energy constraints
      * Earlier menopause
    - **More atresia- more follicles must grow each time in order for a “good” one to emerge**
* **How is spontaneous abortion related to changes in fertility with age? Why do spontaneous abortion rates change?**
  + **Fecundability**: Women most likely to conceive with any given cycle peaks at ~22
    - Fecundability declines (harder to get pregnant) mostly due to early pregnancy loss
      * Increases w age
      * Rates of spontaneous fetal loss go up because of errors in cell division
      * Trisomy 21
  + **Aneuploidy**: abnormal # of chromosomes: 1 or 3
    - Most of these result in fetal loss (down’s syndrome is an exception)
  + Older age pregnancies tend to have higher rates of early pregnancy abortions, because there is more erosion in eggs remaining
  + Goes up in age
  + Probably explains most of change in fertility rates with age
  + Mechanism for removing fetuses that are abnormal- higher number with mother’s age
    - Chromosomal abnormalities when they finally divide (chromosome stick- so either extra or missing)
  + Strategies to avoid genetic errors:
    - 1: make all gametes early in life before genetic errors can accumulate
    - 2: make a lot of gametes all the time and then weed out the bad ones
  + M/F diff in gamete production
    - Stem from diff in Parental Invest. Leads to diff strategies
      * Females invest more in each offspring, not just DNA
      * Female mammals may want to limit # of simultaneous offspring
    - Males can depend on:
      * competition between sperm
      * Females doing hard work at being choosy
* **How many follicles are lost to ovulation? Why might more than one (in fact many) follicles be used up during each cycle?**
  + 1 is lost in ovulation
  + most those that are lost are those that start to develop and get discarded (**atresia**)
* **How do estrogen, progesterone, FSH, and LH change with menopause?**
  + With menopause- change sin hormonal profile- process becomes less efficient
    - EST
  + See below
* **How does menstrual cycle length change as women approach menopause?**
  + Becomes more variable
  + Becomes longer on average, more missed pieces of the cycle
  + Until ultimately it stops 🡪 causes changes in hormones
  + Hormone changes:
    - EST + PROG decrease with age
    - Cycle length goes up (more variable, but longer overall)
    - FSH and LH go up – really high after menopause
      * To compensate for the failure of follicles, more FSH is needed to produce viable follicle
      * More follicles are being used each round
    - Remains high due to breakdown of negative feedback – no ovulation, so FSH and LH keep increasing trying to make something to happen, but nothing can happen
      * They will be higher overall
  + Tsimane- Same patterns- EST, PROG decrease (but absolute levels are lower too); LH, FSH higher
  + Hormone replacement therapy – associated w negative side effects (reduced hot flashes but gave cardiovascular issues)
* **Why might “hot flashes” be less pronounced in populations with more resource stress?**
  + Women in more industrialized populations have higher hormones levels, so “**fall-off**” during menopause is more distinct- associated with increase in “hot flashes”
    - Hot flashes less pronounced in less industrialized pops-
      * Lower hormonal levels in general before menopause – less absolute change
  + But studies on less industrialized pops done in hot environments, so may be hot anyway and not recognize hot flash
* **What are some hypotheses for why women live so long after reproduction ends?**
  + Options:
    - Human lifespan lengthened by extending post-repro period
    - Humans had the capacity to live long, but recent provisioning made it more pronounced
    - Human lifespan and repro lengthened, but then female repro was shortened to allow for GM hypothesis (benefits)
  + Grandmother hypothesis
  + Likely just a factor of living in a well provisioned environment
* **What is the grandmother hypotheses? Is it well supported?**
  + Length of postrepro life span is due to provisioning by senior women
    - As humans entered a feeding niche w hard-to-obtain foods
    - Juveniles could no longer sufficiently support themselves (more dependent)
    - Mothers faced the choice of foraging where weaned offspring could forage themselves (cost to mom)
    - Or foraging for richer resources while provisioning the weaned offspring
  + Generally, no clear support
  + Most likely a result of available provisioning (zoo conditions vs wild in animals)
  + Allocare evolved to increase fitness w/o producing more of their own
  + Physical constraints on the size of initial follicle pool
  + Follicular atresia may be constrained- less atresia might lead to more defects
  + Constraints on follicle repair/maintenance
* **Do other primates have long post-reproductive lifespans? Under what conditions?**
* In zoo conditions- where resources aren’t limited, have longer life spans, longer post-repro
* **Most sex differences are thought to arise from an initial imbalance in parental investment between two different sizes gametes. What is the logic of this line of thinking?**
  + Sexual dimorphism: humans males are ~15% larger than females
  + Parental investment:
    - **Anisogamy** = difference in gamete size
    - Small difference in initial parental investment leads to bias in commitment to continued investment
    - Leads to adoption of different life strategies, based on commitment
      * F PI is > M PI
      * Mating act that is inexpensive for M trigger a large, costly investment for F
      * **Limits to RS:**
        + F: ability to convert resources to offspring

Evolved to be choosy; mate choice error is very costly

* + - * + M: access to females

Evolved to be competitive; take every mating opportunity

* + - * PI logic: F specialize in parenting effort; M specialize in mating effort
      * So- general differences in sex – in predictable ways
* **Why might some sex differences be smaller in humans than in other animals? Why might some be larger?**
  + High degree of PI
  + Tendency towards monogamy/low levels of polygyny
  + High degree of strategic flexibility
  + Constraints on indiscriminant impregnation by males
  + Culture and learning can counteract or reinforce biological tendencies
  + Apparent division of labor (H vs G)
  + Need for specialization for a large-brained, expensive fetus (fat stores in women)
* **What is the hunter-gatherer theory of spatial sex differences?**
  + Men- spatial ability (wayfinding) essential for hunting; throwing spears/rocks
  + Women- female selection for gathering abilities- capacity to rapidly learn and remember things and relationships of objects to one another
  + Females have larger visual fields than men, can see farther
    - Better at scanning, excelling in perceptions
    - Better memory for high-quality food items
* **If we look at the actual distributions in things like spatial abilities, what do we see about the differences between men and women? How much do they overlap?**
  + Some diff in avg, but there’s a lot of overlap
    - Homosexual F – higher than hetero F
    - Homosexual M – lower than hetero M
* **Why might we say that individual people are mosaics of traits?**
  + Most people are a mix of masculine/feminine traits
  + Shouldn’t be referred to as more masculine/feminine
  + Has to do with the way these traits develop early in life
  + Other behavioral traits- mixed because humans are flexible and need to fill a lot of niches
* **What evidence do we have that some sex differences might be context specific?**
  + Sex difference are different in diff cultures
  + Vary with ecological circumstances
  + Interest in short vs long-term mating
  + Magnitude of differences changes
  + “**Dead reckoning**” – (stand up, close your eyes)
    - in Tsimane- no difference
    - but in industrialized – typically there is a difference
  + Difference in different countries with diff sex ratio
* **What are the general steps of sex determination during development? How are androgens important in sex determination?**
  + **2 models**
    - Early effect of androgens affect brain and continue through life
    - Early effects of hormones affect brain, affect relationship with others, which in turn has effect on brain, feedback
* **Why is digit ratio thought to relate to in utero androgen levels?**
  + Linked to genes that are affected by androgen
  + It’s been observed in other animals
    - Chicken digit ratio association – experimental
    - Extended to humans
  + Studies in humans show some link to cord blood androgen levels
  + Controlled by some of the same genes responsible for sexual differentiation (Hox genes)
    - Sexually dimorphic
    - May be an effect of high T in industrialized pops
* **What traits does digit ratio actually seem to correlate with? Which ones does the evidence seem to be thin on?**
  + Related to athletic performance
  + No clear relation to circulating hormone levels
* **What are some hypotheses for what determines sexual orientation? Is there any good evidence this are due to hormones? (Is there any good evidence supporting any of the current hypotheses?)**
  + Due to large diff in some hormones between males and females
    - Thought that hormones are an obvious cause-
      * Very little evidence
      * No diff in T levels, little evidence of digit ratio
      * Some evidence that “butch” lesbians have lower digit ratio and more T
      * Suggests relationship to physical traits rather than sexual orientation
      * Evidence for diff in behavior (mating system, etc)
* **Why might epigenetics be involved in mosaic expression of sex associated traits?**
  + Downstream effects
  + Different features (esp related to behavioral) might require to flip a bunch of switches
    - Potential for these switches to be passed from 1 gen to another already flipped
    - So won’t be reset early in devo
    - Develop with switches flipped one way or another
  + SRY gene has to flip a bunch of additional switches on all the downstream sexually dimorphic trats
  + Some of these “switches” can get transmitted already flipped between generations
  + Mosaic expression of sexual dimorphic traits
  + Adaptive in humans? Unique behavioral niches?
* Angela’s study:
  + The relationship between perceived lifestyle discrepancy and HPA function offers support for the hypothesis that **individuals use social information to determine appropriate baselines for sufficient resources.**
  + Immigration status likely influences perceptions through differential access to social and economic resource opportunities.
  + Not owing money may reflect a lack of credit, a compromised or resource-poor social network, and greater absolute deprivation and access to resources, including those more readily accessible through borrowing.
  + Regardless of other material welfare, poor access to sanitation could signal a chronic deprivation of basic human needs that relate to individuals’ subjective perceptions of self.

**Health and Disease**

**Gurven and Kaplan 2007**

* In U.S.- Infant Mortality Rate IMR 0.58%
  + E0 = 79
  + E15=65
  + Heart disease, cancer, respiratory, accident, stroke, dementia, diabetes
* In HG- IMR 20% (Same as Sweden in 1751)
  + E0=32 (avg. doesn’t mean everyone is falling dead- just high infant mortality)
  + E15=39
  + Illness (and/or infection), degenerative, violence/accidents, other
* Environmental change >> adaptive change
  + **Mismatch hypothesis**
    - Genetic adaptations and current environmental conditions
* Diet:
  + U.S. more fat, less protein (and saturated fats); less fiber, more sodium, more alcohol
  + HG less fat, more protein
* Diet affects different aspects of health
* HGs: Low-
  + Obesity
  + Hypertension
  + Cholesterol
  + Diabetes (non-existent)
  + CVD cardiovascular disease (rare)
  + Reproductive cancers (rare)
  + *Maximal oxygen intake (excellent)*
* **Mismatch hypothesis**
  + Genetics and lifestyle factors influence likelihood of certain disease manifestation
  + There’s been a decent amount of genetic change since the Pleistocene
    - Diet particularly- changes in diet
    - Exposure to pathogens
    - Disease (immune)
  + But, there are plenty of changes within a few generations with exposure to certain diseases
* Immunological Evidence of past infections with various disease agents (**Brazilian Tribes; Black et al. 1975**)
  + Endemic diseases (high incidence, low mortality)
    - Herpes
    - Hep B
  + Enzootic (low prevalence over long time)
    - Yellow fever
    - Toxoplasmosis
  + Introduced (explosive, transient)
    - Measles
    - Mumps
    - Rubella
    - Influenza
    - Polio
    - TB
    - Malaria
* **Yanomamo**
  + Anemia- common in about all ages
  + Malaria- esp in young kids 1:5
  + Vivax- also in kids
  + Splenomegaly (big spleen)- about everyone
  + Over time- causes of mortality: Precontact 🡪 Postcontact **(Early and Peters 2000)**
    - Postcontact: infectious diseases skyrocketed
    - All the rest pretty low: Unknown, homicide, accident, non-infectious, infanticide
* **Tsimane** 
  + Proximity to markets- linkage to modern society
  + Longevity
  + Men:
    - At old age suffer from skeletal (osteoarthritis), some respiratory (chronic bronchitis and TB) and GI
    - At young age: mostly respiratory
    - More likely to be attacked by snakes than women, and more than they are likely to be attacked by other animal (increases w age, 1:5 by the time they’re old)
  + Women:
    - At old age: equally from skeletal, respiratory, some GI
    - Young age: respiratory and GI
    - More likely to encounter a snake than other animals, but less than men in general
  + Leshmaniasis – שושנת יריחו
    - Protozoan transmitted by sand fly
    - Treatment – heavy metal based
  + Skin infections
* **Parasites – Tsimane**
  + Hookworm, roundworm, whipworm
  + Fairly prevalent, often people have more than one type
  + Hemoglobin- assess anemia
* Infectious diseases – antibodies for these infections (past exposure)
  + Prevalence of Hepatitis A 95%
  + Yellow Fever 87%
  + Toxoplasmosis 84%
  + Rubeola 94%
  + Measles 91%
  + Leptospirosis 56%
  + H-Pylori- Bolivia much higher than U.S. (almost 40%)
* Tsimane have high WBC
  + If you’re fighting multiple infections, not surprising you have high WBC
  + High early in life, lower w age
  + Eosinophils account for 20% of their WBC (US: ~1%)
    - Associated w parasitic infections (intestinal in particular)
  + T-cells: naïve t-cells
    - Matured but haven’t been associated w any particular infections
    - You need these to deal w a new infection
    - Naïve t-cells decrease significantly w age- immunosenescence w age
      * So high exposure to pathogens
      * Over half the deaths in Tsimane are due to infection
* Inbreeding
  + High amount of congenital diseases
    - Cleft palate – breastfeeding issues, can be fatal (respiratory)
    - Extra toes
* Heart disease- mummies CT
  + PAD and Atherosclerosis
  + Heart disease was equally prevalent in many cultures across history
  + 4000yrs of history, across many regions in the world
    - evidence of atherosclerosis (Heart disease)
  + “the common assumption is that heart disease is a product of modern living, and that it could be prevented with adjustment; but our study finds that the cause of CAD is unknown and might be somehow an inherent process of human aging”
  + **Hypothesis**: aging of heart tissue and blood vessels occur in all populations, but active lifestyles and high rates of infections in the past may have prevented significant coronary artery disease capable of producing infarcts and affecting systolic function

Tsimane:

* Almost no evidence of any heart attacks (past or present)
* CAC by age, sex, population: much lower in Tsimane than any other group in the world
* In over 700 adults of age 40+, 85% of them had no Coronary Artery Calcification (CAC)
* Moderate CAD is 2.8% (Coronary Artery Disease)
  + Matched indivs in industrialized pops exhibit 10x as much
* Tsimane- healthiest heart in the world (no one ever studied Hadza hearts, they might be equally healthy)

**Tsimane Diet and Exercise:**

* Diet low in sugar and salt
* No trans fats or preservatives
* Low fat diet (14% of diet is protein, 14% fat, 72% carbs)
* High in omega 3 and fiber
* 4-7hrs of physical activity/day
* <10% of daylight hours sedentary
* Low levels of cardiometabolic risk factors
  + Obesity very low (although about ¼ of adults are overweight, just not obese)
  + Hypertension very low
  + No high cholesterol

**O’Dea 1984, O’Dea and Sinclair 1985, O’Dea 1991, Lee et al 1994**

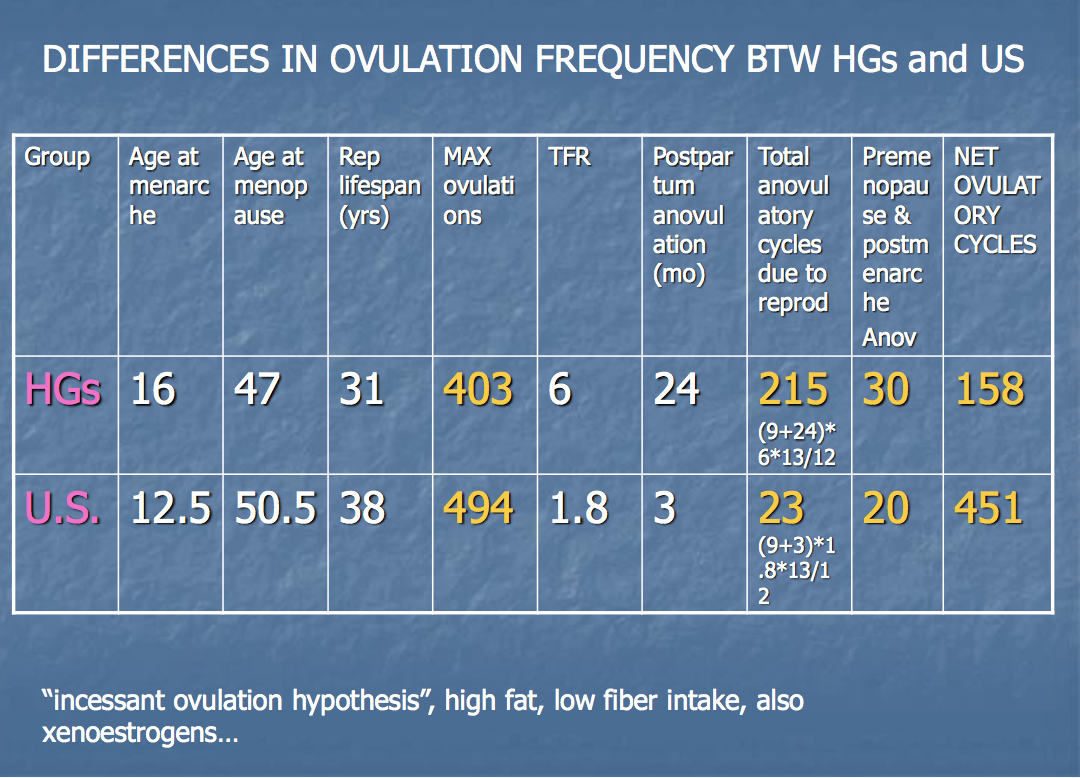
* Some indication that if westerners change their lifestyle to reduce fat, sugars, etc, and increase activity-
  + causes improved metabolism, weight loss, BP, chol, glucose tolerance greatly improved

**Pathogen diversity- “Old Friends” hypothesis**

* Intestinal helminths- host manipulation
* Co-infection, poly-parasitic world
* Immune dysregulation in modern “hygienic” environments
* With infections- in the absence of these parasites that our bodies expect early in life- our immune systems can be dysregulated later on
  + Modern environments are too clean, hygienic
* Helminths as anti-atherogenic
  + Decline in many diseases (Hep A, TB, Mumps, Measles…)
  + At the same time, incline in many auto-immune diseases, diabetes…
    - MS, chrone’s, asthma
* Decreased exposure early in life is throwing our immune system out of whack.
  + Ends up attacking itself

**Helminths and anti-atherogenic effects**

* Helminths are literally consuming some of the fats and cholesterol that we eat, so reducing our uptake
* Lower levels of cholesterol and LDL in people infected with helminths
* Cost of immune function- people infected with helminths had resting metabolic rate 10% higher than those without-
  + So spending more energy if infected
* Worms- anti-inflammatory effect
* If your immune system itself is less likely to react (or overreact) to buildup of junk in arteries
  + then your body isn’t contributing to the buildup
  + immune system itself is actively involved in process of buildup
* immune system is working differently; inflammation and anti-inflammation aren’t working regularly
* modulating immune fxn
* counter-balancing auto-immune effects
  + taking up some of the energy
  + multiple pathways in which helminths can assist in reducing heart disease
  + esp. in interaction with other factors (diets, activity)



Chagas and Valvular heart disease

* Kissing bug
* Transmitting trypanosome
* Buildup of large heart (enlarged)
  + Venezuela and Bolivia

Cancer

* HG: much less ovulations that USA women- net ovulatory cycles 158
* USA: much more ovulations- net ovulatory cycles 451
  + 3x more than HG
* What would this do to cancer reproductive risk?
* **Excessive Ovulation Hypothesis**
  + Rebuilding of endometrium- cell proliferation itself is cause of increased risk of cancer
* Using machinery in ways that would increase risk
* With each cycle- your hormonal exposure will vary
  + Surges in Estrogens and Progesterone
  + Cost of high estrogen is high risk of repro cancer

Hormone replacement treatment- can help with bone density issues upon menopause

* But at a cost of increased risk of repro cancer
  + **Breast cancer**
  + **Endometrial cancer**
  + **Ovarian cancer**

Not cervical cancer- probably unrelated, but higher risk with HPV

Relative risk of cancer: Early use of contraception

* Earlier levels of high hormones levels
* Use of contraceptive help reduce risk
* Higher risk of people who didn’t use contraceptives (U.S. is 240x Paleolithic)

**Machiguenga of Peru**

* Parasite load reduced, BP improved, all look better
* But when you ask people about aspects of well-being- people reported being a lot happier back in the 70s, than in the 90s
* Notions of health are not the same as the actual physical well-being
* Missionaries, oil companies, encroachment by other neighbors- all reduce the mental well-being
  + Subjective aspects of health are diff than objective indicators
  + WHO doesn’t think just life expectancy is a good measure of health
    - Just a single metric of many

**Depression among HGs 11/28/17**

* In the U.S.:
  + Decreased age of onset since WWII- once mid-30s, now mid-teens
  + **Major depression** takes enormous toll on
    - quality of life
    - functional status
    - productivity- affects sleep, eating, habits, jobs, relationships, thoughts, feelings and behaviors
    - associated w suicide and heart disease
    - depression as the “common cold” of mental illness
* **Functional explanation:**
* If emotions evolved by natural selection, they must have been important throughout all of human history, not just modern times
  + Randy Nesse’s (“evolutionary medicine”) theory of depression suggests a period of physiologically induced re-evaluation of strategies (and inactivity) following a failure
    - High modern rates were due to a pathological modern environment
      * But maybe a function of no-diagnoses in the past?
  + “Depression sets in after someone is prepared to suffer a failure or loss. It slows you down and makes you take your bearings” (Eric Klinger)
  + Increased pressure that people feel to ever-larger goals
  + Ancestral hominids may have striven to pick enough berries to last for a week
    - Modern humans want to look like supermodels, make a million dollars and produce flawless children
  + Our HG ancestors should also have experienced conditions eliciting depression often enough for the emotional state to have evolved
    - But HGs lives are much more oriented to sociality than WEIRDs. They are dependent on others for many things that we now purchase as services
  + **Loss of social support** is a universal problem

Ache and Tsimane

* So busy surviving, that they didn’t have the free time or luxury to “dwell” or overly self-reflect in ways required for deep swings in mood
* Generally happy because of limited goals and regularly attained
* Matches Nesse’s statement
* Largely focuses on material concrns

Hiwi

* Spent many hours in camp each day, not foraging, not making tools, not socializing, often sitting in hammocks, drugged
  + Gave them plenty of time to reflect and that appeared to lead to depression
* Their material goals were fairly simple, but the were not meeting them well
* The pop was under severe food stress, there were violent and hostile relations with neighbors, and mortality rates were high
* Hiwi did say they had difficult to achieve social goals- lack of respect by neighbors, looked down-upon
* People were concerned about keeping face in front of peers
* Social interactions led to what appeared to be “depression”

Yuqui (Ache) HGs

* had slaves that did most of subsistence work
* Unlike Hiwi, Ache seem to fit NEsse’s description-
  + They often smile and laugh, and always seem in a good mood
* But after spending much more time w Ache- did not keep up
  + Became moodier, esp women
* Ache had been through near extermination around contact, followed by enforced servitude
  + They still showed a lack of emotional distress
* Individuals who lost most of their family in contact epidemic never seemed to have periods of sadness, deep thought or depression
* “Happy-go-lucky” demeanor

These were all initial impressions…

Hill and Hurtado (KH and MH)

* After 5yrs work with Ache
  + No longer put up appearances
  + Deeper insight into their lives
  + As confidants, serious manifestations of anxiety and depression came to light

Examples (Case studies)

* Krypurangi – married to an older, smarter women of high status
  + She left him, he became distressed; drank ½ gallon of insecticide
  + Survived but serious health issues
  + **First case ever heard of a suicide attempt in Ache**
* One guy:
  + Popular in the group;
  + Heard buzzing in ears, couldn’t fall asleep
  + Headaches, nauseous
  + Suffered from depression and anxiety
  + Pessimistic view that his people were on the road to extinction
    - Responsibility of well-being of the people; couldn’t figure out way out of food stress
    - Avoided social interactions
  + Left the reservation, set up settlement in forest, reverted to traditional HG lifestyle
    - Soon after this, his personality seemed to come back
* Chapanogy
  + Talented guy; mother was Ache, dad was Paraguayan
  + Spent some time in Paraguay, but always came back to Ache
    - Social support
  + Really capable guy- electrical experience, motorcycle
  + Frequently unhappy
  + Tried to become chief, but since no one nominated him, he didn’t
  + Left, got a job at national park
    - Took this seriously- shot each poacher he found
  + Found his way into the army- rode a tank, commanding officer
  + Reality of racism: no Paraguayan employer wanted to hire him
    - “halfbreed”
  + depressed, drinking, gained weight, lost teeth
  + despair, regrouped, returned to Ache, studied bible…
  + didn’t work- went back to city became taxi driver
  + disappeared, killed or got involved with drugs
  + 🡪 goals he set himself that he was unable to achieve
* Kandegi
  + Teenage mom- envied the highest status wife
  + Her husband was passed by for office, and she got into a scandal, and publically shamed
  + Emotional tailspin
  + Still on medication
* Airagi
  + Problems w teenage daughter- daughter went to work in a brothel
  + Ache brought her back
  + Shaved her head
  + Continued to be trouble
  + Married guy, airagi no longer depressed
  + Other daughter ran away with husband of first daughter
  + Became depressed again
* Japegi
  + One of the most skilled hunters on records – “returns rates”
  + Tried to impress village members into giving him a political role
  + Given chance to be named in charge of animals (livestock)
  + He was given an opportunity to speak
  + Rambling, incoherently
    - Others interrupted, told him to sit down
    - He took this personally
  + Humiliated, depressed, never carried out his duty

Is this because more capable indivs develop high expectations, and can more easily fail and lead to realignment of strategies?

***Ache who are not too bright and not too ambitious rarely report depression***

High expectations 🡪 more likely to fail

Loss

* Loss of close kin and social support seems to be major cause of depression in HGs
* Failure in social competition is a very common cause of depression among Hiwi, Ache, and Tsimane
  + This problem is not unique to modern societies

Are HGs happier than modern Americans?

Nesse’s views about unique stresses of modern society suggests that depression should be less common in HGs

* There are two factors that seem likely to lead to higher depression in Americans
* 1. Mass media- causes everyone to believe they are failures on a relative social scale
  + none of us feel we are successful relative to our “TV peers”
  + prediction: those more exposed to this will be more likely to be depressed
  + Facebook: making you more depressed?
  + FOMO- fear of missing out
  + Replacement vs augmentation of networks?
  + Social comparison
  + Depends on personality
    - But breaking it down: Arroyo Bandera: most ethnographers would not observe severe depression without significant time in the field (and good linguistic knowledge)
    - Likewise, little depression would be observed if one spent less than a year watching 10-15 families
    - Prevalence- 11/55 = 20% in 2.5yrs
    - 1 case per 9 person-days (need a long time in the field to be able to observe it)
* 2. **Ache Kuaeme** philosophy – Kuaeme “don’t think about it”, “forget about it”
  + instead of treating depression- this cultural philosophy seems designed to decrease the incidence in the first place
    - what you would do with the death of a loved one
    - Adults who cried too much were ceremoniously beaten with sticks by others, and children were threatened that if they cried too much they would be buried alive with the deceased
    - Older kids often ran away to a distant area after their parents died
  + They were told “kuaeme”
  + It already happened- move on
  + **Tsimane**: version: “Paj mo’ ya”
* Tykuarangi
  + Internet porn- refused to look at it; those women are really attractive, but I can’t have sex with them, Kuaeme.
* Hiwi
  + Dwelled a lot on dead ancestors
  + More depressed than Ache; took drugs constantly

Tsimane depression score increases with deteriorating health (self-reported)

* Their depression score increases with age, and low energetic status
* Can go up to 65% prevalence in adulthood
  + The older you are, the less you are able to be an influential member of society, and the less you can produce and contribute
    - Associated with depression
* **Market proximity** is associated with depression
  + Closer to other influences can increase depression score

If you could change something in your life, what would it be?

* Obtain more modern goods/services
* Easier integration into market
* Return to traditional lifestyle

Conclusions

* HGs get depressed just like modern Americans
* This emotional state evolved in our ancestors and was probably just as common as fever and vomiting
* Most depression in HGs in about social aspirations and social failures
  + Loss of social support
  + Not a modern issue- you don’t need to fail to be a movie star to be depressed
* Unclear if modern life more stressful that that of our ancestors, but if we could identify characteristics of modern world that mimic the problems that were solved by depression in our ancestors, we would have a better set of solutions to the apparent recent increase in depression
* Modern comparison of the indiv to unreal role models (media created) may include feelings of failure more often than would have been the case among our ancestors
* Depression as signal to solicit support among kin, friends, could be greater in modern societies if current social connection are weaker of if people live in more isolated lives
* The use of drugs to alleviate a natural response to social failure should be carefully thought through
  + Analogy about drugs to reduce fever infection may not always be appropriate
  + Do you treat a fever? You’re combating your body’s own defenses
    - Reducing is helpful vs harmful

***Camron 1996: Regulation of reproductive hormone secretion in primates by short-term changes in nutrition REVIEW***

*In primates, as in non-primates, periods of chronic or severe undernutrition often result in a suppression of reproductive hormone secretion with an accompanying decrease in fertility(Zubiran and Gomez-Mont, 1953; Warren and Vande Wiele,1973; Smith et al., 1975; Vigersky et al., 1977; Dubey et al., 1986).This sensitivity of the reproductive axis to severe undernutrition would be advantageous, ensuring that fertility is impaired in times when energy resources are limiting for the high energy-requiring tasks of carrying a pregnancy to term and rearing a growing infant. Such a sensitivity to chronic energy availability would be most advantageous for large animals with relatively large energy stores, who could subsist on internal energy stores in times of famine and then reproduce when food is more readily available.*

**Key words:** [total energy expenditure](#Total_Energy_Expenditure), [energy intake](#Energy_Intake), [energy balance](#Energy_Balance), [energy flux](#Energy_Flux), energetic status, [Kleiber’s law](#Kleibers_Law), nutrient balance, digestion, gut anatomy, [energetic tradeoffs](#Tradeoff), growth, metabolic and reproductive hormones, reproductive function, Metabolic Load Hypothesis, allostatic load, stress, capital vs. income breeding, Jarman-Bell principle, allometric scaling, anatomical and physiological dietary adaptations, folivory, and insectivory

**Primate Behavioral & Community Ecology**

1. Primate socioecology
2. Primate biogeography
3. Trophic interactions (e.g., herbivore-plant and predator-prey), phenology (timing of life events in plants, e.g., fruiting), and seasonality in equatorial rainforests

**Key words:** interspecific competition, limiting resources, limiting nutrients, food availability, resource holding potential, priority of access, phenology, habitat degradation, habitat size reduction and fragmentation, edge effect, biotic and abiotic selective pressures, anthropogenic threats, feeding niche, generalists vs. specialists, ecological niche partitioning, character displacement, competitive exclusion, ecological services, ecological functions, species richness, biodiversity, and distribution

***See folder: ANTH 153T***

**Primate Demography**

1. Carrying capacity and constraints on population growth
2. Viable population structure and size
3. Demographics of populations, metapopulations, and species populations

**Key words:** Death, birth, immigration, emigration, Malthus’ population principle, growth rate, r/K selection, kin selection, overpopulation, Batemen’s principle, sexual selection (intersexual, sexual) and mate choice, density-dependent selection, biomass density, carrying capacity, Life History Theory, survivorship curves, environmental factors, demography, population viability, spatial distribution, genetic diversity, operational sex ratio, Trivers-Willard Hypothesis

**Carrying capacity and constraints on population growth**

Life History Theory (Rec 16)

4 levels of explanations

* Complementary ways of looking at a Q

Hormones

* Coordinating LH tradeoffs
* Body is coordinated system (check/balances – feedbacks to keep systems regulated)
* Measuring hormones- listening in on tradeoffs
* T: regulate tradeoffs between mating behavior/musculature/immunocompetence
* LAM: female fertility regulated by change in energy balance
* Early pregnancy loss/ovulation – related to energy throughput, energy balance
  + - High work/low energy period- more chance of pregnancy loss

Parenting

* Infant/mother/father evolution
* Pair bonding
* Mechanisms that facilitate this
* Medical practices: human birth in diff cultures- what does this mean for birth practices

Menopause

* Humans similar to other spp
* In good conditions- other spp can live into post-repro period
  + - But we don’t see this in the wild
    - May be what humans are doing
    - But also, most spp don’t have an abrupt dropoff of fertility, humans do
      * Perhaps through cultural changes- they were living into PR period

Repro ecology- essential to understand human evolution and adaptations

Make sense of fertility, health, evo by thinking about energy

* Fetal Selection- Not Random!
  + Males are more likely to be aborted
    - Male embryos are larger than females
    - At any time in life- more men are dying
  + Men aged 15-30 are 3x more likely to die than women
  + If times are bad – terminate the male
    - Might be best option
    - More costly to carry males
    - Developing male under poor conditions will be less likely to find mate
      * Bad for everyone
    - Ex. ***Sex ratio after disasters seems to skew- more females***
  + Triver’s-Willard
    - Even at max, women produce much fewer offspring than males
    - If you are in good condition, you can pass it off to offspring
      * Then better to have male
    - If you are in poor/moderate conditions, and you have a son, then he will have reduced RS
      * Better to have female – she could still have mates
  + Assumptions
    - Parental condition is associated with offspring condition
    - Differences in offspring condition will persist into adulthood (repro age)

**Sexual division of labor**

* Mostly universal
* Almost every group- men are more involved in hunting, women in gathering, food processing, child care (generalization)
  + Exceptions: honey gathering- men
  + Shellfish gathering – women (depending how you think of it)
* **Kelly: Male contribution to diet varies**
  + Effective temp:
    - low temp, most hunting (can reach 100% of diet); male contribution to diet high
      * contribution- who is bringing food, killed, extracted, etc
    - high temp, less hunting (but never 0% of diet)- male contribution to diet at 30%
      * no population where male contribution is less than 20%
      * Whereas there are some where men are entire contribution
* **Kaplan et al 2000**: **Production of energy by men and women**
  + Protein based: majority of groups- almost all of it coming from men
    - But overall calories through protein: less than that (60-70-80%)
  + In almost all cases total calories split between men/women
    - Exception: Kung- honey, but Richard Lee drove them out there
  + In population where protein is higher % of diet: men contributing more than women
* Not all work is in providing food: so what is monetary value of labor in the house?
* **Relative energy expenditure**:
  + Energy expenditure is larger for males in general than women
    - Kung, Ache, Inuit
  + In non-human primates- much more even expenditure
  + Modern farmers: some populations men do less
  + HG: men do more
  + 🡪Men and women do different things
* Parental Investment:
  + Females: P effort > mating effort
  + Males: P effort < mating effort
* **Kelly: Showoff hypothesis:**
  + Men’s hunting as a signal of fitness
  + If men are spending so much time hunting, is this a parenting effort (dads) or trying to impress/make allies (other benefits) (cads)
* **Why do men hunt?**
  + Not all calories are equal
  + Less variation when sharing- sharing between sexes
  + Diff nutrients in diff food packages
  + One sex can’t necessarily acquire all resources that are needed
  + Long learning curve for proficiency:
    - Takes a while to be good hunter and/or gatherer
    - Specialization of duties = efficiency; then pool resources
  + Hunting: are there other benefit?
    - Social status, alliances, increased mating access (RS)
* Gurven & Von Ruden 2006: Good hunters have higher fertility, surviving offspring, good mates
* **Why don’t women hunt?**
  + Long days and excursions
  + Incompatible with child care and nursing 🡪 overall reduction of group efficiency
  + Cost of women hunting makes it not worth it
  + Men can’t get pregnant, can’t breastfeed
  + **Increased specialization 🡪 increased efficiency**
* Women who hunt:
  + **Mbuti pygmies & Aka:**
    - Net hunting, husband/wife pairs
  + **Ache, Tsimane, Yora:** 
    - Help spot game (increase encounter rates)
  + **Agta women:**
    - Use bow/arrow
    - Use dogs
    - Close to camp
    - Some women are post-repro or sterile: no repro burden
    - Increased rates of alloparenting
  + Men aren’t only hunting:
    - Hadza: honey
    - Ache: honey, palm larvae
    - Mikea: roots
    - Kung: fruit
* Is sexual div of labor favorable for M/F equally?
  + Possibilities:
  + Men/women marry in all societies
    - People marry to reduce M/M competition: property right (no benefits to women)
  + Families can still gain more meat from good hunter/father over long term
  + Choosing good genes (hunting as honest indicator)
* Men provide support to benefits household, not for advertisement
  + Provide care when mom is gone, occupied; when there is less help available (less older daughters); focus care towards older children
* Good hunters 🡪 higher fitness
  + Provisioning directly benefits spouse/kids 🡪 fertility and child survival
  + Social status promoted 🡪 more alliances, reduces variability in food
  + Extra marital mating
  + Help in child care
  + Trade, insurance
* Prestige for men’s activities:
  + Meat is valued in all known HG groups
  + Meat is widely shared
* When added together- men/women work the same, but on different tasks
  + Away from home:
    - M: hunting a LOT; W: fish, garden
  + At home
    - M: hunt very little, some manufacturing
    - W: a TON of parenting, food processing, household stuff
  + Men’s work gets counted more

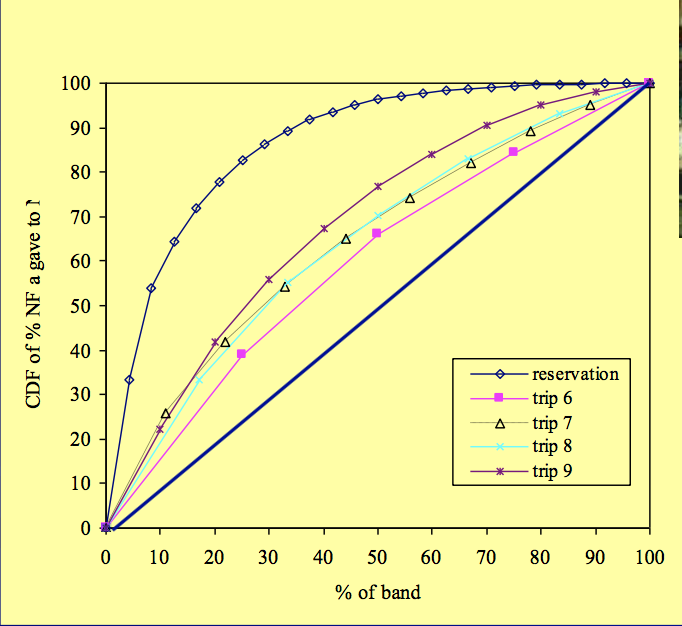
Hormonal indicators for men hunting:

* Provisioning vs signaling:
  + If it’s just provisioning, it doesn’t matter if you killed the animal or not- not gonna be an increase
  + If you’re interested in signaling, doesn’t matter if you killed/not, you’re trying to impress, increase
  + Large kill- show off, increase

**Original affluent society**

**Egalitarianism**

**Egalitarianism**: equality of opportunity, outcomes, health, resources, fitness

* Most primates are dominance hierarchy
* Unique – conditions that favors Eg. populations
* In HG: Big **pressure to redistribute**
  + Tolerated scrounging; demand sharing
  + Benefits of redistrib: in such a difficult foraging niche, it would make sense to redistrib
* Characteristics (**Kelly**):
  + Individual autonomy is central
  + Equality of access to resources
  + Usually no storage
  + Fierce of assertive egalitarianism
* Maintenance of Egalitarianism
  + Demand sharing – redistribution
  + Ethics of fairness
  + Ridicule of upstarts (!Kung); modesty (Ache); ostracism
  + Small, visible communities
* Ultimatum game/egalitarian sanctioning: in some pops- offers that were too high are rejected
  + If I take now, expected to give back
* **Boehn 1993**
  + Groups have different ways of dealing with upstarts – people who disrupt the egalitarian way
  + Execution for deviation from social norm
* Forest vs settlement:
  + Forest: some people net givers, others net receivers (loose sense of property)
  + Reservation: much more people who are net receivers (a lot of personal property, guarding)
    - Esp. for forest food (some people never go to forests); also for fields- some have, some don’t
  + Gov. issues to get people to farm-
    - Some grow all year, big harvest, everything gets taken away by family members/friends
      * Kills motivation- don’t want to do it all over again
      * Others wont do their fair share, you’ll have to give away a lot
  + **Ache**- formal meetings forcing people to farm next year!
* Farming: first place to do so was with complex HGs

Lorenz Curves (Gini index of inequality)

* The middle line is complete equality- 50% of people have 50% of wealth
  + Foraging trips- are pretty close to equality
* The farther away from the regression you get- the less equality you have
  + Reservation- 10% of the people hold 60% of wealth

**Ex. Gana**

* (Elizabeth Cashdan)
  + Unlike Dobe Kung that relied on short- and long-distance trades and social relationships for dealing with risks in a volatile environment…
* Gana had some agro to help with harsh environment
* Also engaged in storage- other ways of dealing with risk
* Notions of private property was allowed-
  + Accumulation of wealth
  + Appealing and praiseworthy
* Few people have many number of wealth items owned
* Much less reliant on sharing to reduce risk- limited to household members
  + Dobe Kung- sharing very common

If you can form a way to rely only on yourself, it shifts your society

* Accumulation of wealth
* Inequality
* Private property

When you need to rely on others

* More difficult to shift away from egalitarianism

**Ex. Tsimane**

* Axes, machetes, shovels, livestock, chickens – subsistence instruments; many have these (more equality)
* Watch, radio- Luxury items- very few have (farther from the gini index curve) most inequality
* Not all resources are the same
* Social Status:
  + Community influence:
  + **Correjidor** – **the corrector**- and imposed leadership position
    - Didn’t really exist formally
    - Doesn’t have real power
    - This person will help run meetings
  + Measure social status: Who has influence
    - Top quintile- social support, bigger (physically), have more skills wealth, better personality, etc.
      * Causality? **What determines status, or what does status give you**
    - **Having allies** is the factors that gives you the most benefits: get your way, win dyadic fights, influence, respect
    - Even in the absence of formal leadership
* Female social status:
  + Women’s attractiveness vs. status
    - Women <25yo – usually high ranking in attractiveness
    - 25-40 – but some of these women are rated higher than women under 25, so physical attractiveness isn’t the only thing that influences social status
    - 40+ - same
  + Status, social support, attractiveness, give them better mates etc.
* Household wealth:
  + Age of household heads- percentiles
  + Wealth increases w age
* Inequality is highest in the poorest Tsimane villages
  + Gini index of Tsimane- varies- can be anywhere between to Sweden to Cuba
  + Only a few people have something, and everyone has nothing… makes for a lot of inequality

**Inequality and Psychosocial distress, independent of wealth**

* With inequality-
  + Depression increases
  + Blood pressure
  + Pulse pressure
* Lower White Blood Cell (WBC) w higher inequality
  + A screenshot of a cell phone

    Description automatically generatedIn Tsimane, WBC high usually
    - Lymphocytes in wealthy indivs and eosinophils usually lower
  + Difficult to make strong inferences

collective action problem

**Complex vs simple HGs** – complex: hierarchical

* Of the first to have a successful system of farming
* Complex usually happen in areas w very predictable environments
  + Less variability in likelihood of catch
  + More coastal
  + Ripe areas
  + Prime real estate
* Diet more reliant on marine, plant foods
* Settlements large
* Mobility- lower, more sedentary
* More food storage
* More hierarchy
* Slavery
* Occupational specialization
* More territorial
* Warfare more common
* Ownership
* Wealth, competition, competitive feasts (exchange)

Simple vs. Complex

**Scalar stress hypothesis**

* Abundant, predictable, localized resources 🡪 large population (life is easier) 🡪 increase conflict, “scalar stress”
* Sedentary
  + 🡪 No: Fission/fusion (diff times of year come together)
  + 🡪 Yes: Hierarchy, status (but for the most part, don’t want to go anywhere- there are good conditions)
    - conflict increases 🡪 need to deal w stress, benefits to high and low status
    - Low status:
      * less stress of anyone stealing from you
      * when you know your status, you aren’t consistently contesting for high status
      * less likely to win a contest- might suffer substantial harm
        + might as well be lower, and do “just fine”
        + most of the contest is within a social status grade, not between
        + lowest never competes with highest
    - knowing where you are- you can avoid some conflict
* Member/joiner conflict- those who first arrived have privilege access- longer history than later joiner who accept lower status
* Is hierarchy something that always follows without regard to the circumstances?

**Pacific North West – Shasta, Pupa**

**Tolowa of California**

* Marine resources and acorns
* Rich, predictable resource base
* Villages had headmen, leadership roles
* Wives brought in from neighboring tribes
* Polygyny sometimes
* Fishers
* Canoes
* Acorn- grind it to flower, meal
* Wealth accumulation
  + Engage in matches as status display
  + Ceremonial gala- more elaborate ceremony associated with these groups
  + Elite had more elaborate dialect than commoners
* NW California- resource abundant
  + Year-round villages
  + Sea lions, shellfish
  + Monetary system- dentalia (mollusk)
    - Strung into necklaces

**Ainu – Northern Japan**

* Autonomous indigenous population
* Lands taken from them
* Didn’t have notions of private property- so said “we don’t own the land”, so it was taken from them by Japanese
* 25,000 people
* Elaborate ceremonies
* “bear ceremony”
* Status symbols
* Storage- “Pu”, gathered vegetables
* Cultivated wild millet
  + Small scale gardening – mostly women
* More closely related to Tibetans than to Japanese

**Human life history**

Human adaptive complex:

* Humans have a long life span
* Long “childhood”, high dependency
* Large, encephalized brain
* Intergenerational provisioning
* Contribution of others to energetics of repro
* Long post-repro lifespan

**Gurven and Kaplan 2007: Ratio of HG to US 2002 hx**

* Early life: much higher mortality in HG than in US ages 0-5, then drops but still higher until age 15
* But still 14x higher mortality in HG
* By age 45: HG 7x than US
* **Most of the differences are early in life.**
* We all still have limitations
* Life expectancy:
  + At age 0: HG: 35
  + At age 15: live another 45 yrs
  + If you survive longer, your expectancy is longer
* Existence of older people isn’t an artifact of modern society, it has existed for a while; similar across populations

**Age of Death**

* Very few people are dying at early ages
* % that are dying past 55 increases, peaks at 72 for HG, 85 for USA

**Chimpanzee life history**

At age 0: expectancy 15, if survive, very few make it past 50yo

Allometric scaling of body with species

* take body size into account: given our body size, humans still have longer life spans than you would expect
* also, humans have bigger brain that you’d expect for our body

**Allometric scaling – *Armstrong 1990 – Brains, Bodies and Metabolism***

* The association between the size of an animal’s brain and the weight of its body has been the basis for many studies in which the interrelationships of major organ sys­tems have been examined in different vertebrates. In most of this work, researchers have used allometric anal­yses based on bivariate regressions in the form of
  + Brain weight = a\*Body weight^b
  + Both the slope, **b**, and the size of the brain at the Y- intercept, **a**, are units for comparison.
* *Allometric analyses have determined how relative brain sizes differ between animals in various taxonomic groups and between animals occupying particular ecologi­cal niches. Additional studies have shown that brain and body weight of individuals within a species scale differ­ently from brain and body weight between species and that these factors at various developmental stages scale differently than they do in adults*
* *Slopes generated by analyzing different fetal stages within a species are higher than those from comparative studies of adults and frequently approach unity. Thus, during the fetal stages of ontogeny, both brain and body experience the same rates of growth, so that the allomel- ric slope is 1 [Count, 1947]. In many mammalian species brain growth ceases shortly after birth, but body weight continues to expand. It is this differential in duration of growth that produces the negative allometric slope in a comparative series of vertebrates [Holt ct al., 1975; Sacher, 1982).*
* *Among primates only the human species continues to have a high brain- body growth exponent for a considerable time after birth.*

**Food:**

* Humans rely much more on hunted and extracted foods; chimps on collected
  + Humans: more difficult to acquire, but more nutrient dense and higher calorie value; package size larger
  + Chimps: easier to obtain, less nutrient dense, small packages
* Life history relies on what they are eating, how they are acquiring that
* **Humans:**
  + don’t directly consume foods with high fiber/toxins
  + process food to remove toxins, fiber and packaging
  + eat bigger, more calorie-dense prey
  + use more tools and tactics to disadvantage prey
  + use more tools to extract resources from packages
  + diets composed of foods acquired differently by indivs of varying age/sex/repro status and characteristics
* Feeding niche based on high-quality, large packaged foods
  + Food sharing
  + Lower mortality
  + Investment in **embodied capital**/lengthened development
    - Large brains
    - High adult productivity: provisioning
* **Lactating**
  + primates work more on feeding than traveling (increased effort)
  + humans work less in general (decreased effort)
* **Historical Hominin:**
  + **Bipedality** 🡪 freeing of hands for extraction, tool use, carrying, efficiency 🡪 new feeding niche
  + **Emergence of savannahs**: higher density of mammals and plant storage (roots) and repro organs (fruit)
  + Investment in **Embodied Capital**

**Embodied capital theory = increased investment in skill**

* **Kaplan et al 2000**: Age-specific production and consumption for human HGs and Chimps
* Age-specific Calories acquired per day vs expenditure
  + Chimps: Shortly after weaning- chimps can feed themselves; but no surplus (amount acquired= amount expended)
  + Humans: humans fall much short of their expenditure until they are ~15, but then can produce a lot more than expenditure until age 60. So there is a LOT of surplus.
    - That surplus allows for an **embodied capital**
    - Skill, knowledge, abilities to obtain income in your environment 🡪 increase fitness
    - Has to do with large brain
      * Return, in adulthood
    - Helps with sharing
  + Delayed maturity: reproductive and functional
* Shift to feeding niche coevolved with the complex (long childhood, long learning)
* Early life- brain growth priority; late life- body, embodied capital
* Humans as “**cooperative breeders**” – sharing, large surplus, etc.

Ache: by 15 make 50% in fruit collection as adult

* When there are no fruit, no one below 10 is getting anything; when there is fruit, even 6 y/o are getting a sig amount
* Skill increases w age; younger and much older people less successful
  + You are at your peak at 25, but some of the pursuit peak (skill) is at 40
* Return rates:
  + 50% of maximum at 20yo
  + only at 35-40 it is highest
    - it takes time to learn to do these things
* Expertise is delayed for most skill areas – music/oral; manufacture; childcare; hunting; fishing; tool use…
  + Experts are usually people older than 40
  + Oral tradition and singing is the most delayed >50
  + All skills begin to be acquired around age 12

The argument for studying HG

* Most on human history we lived as HG
* All major features of life history are present in extant HG groups
* Compared to other primates, human HGs have the most skill-intensive feeding niche
  + Niche requires human children have a long underproductive juvenile training period
    - = Human capital investment
* People begin to repro when they are just able to produce as much as they consume
  + But before they are capable of fully independent repro (~18yo)
* Early repro is supported by non-repro, older relatives
* Lifespans are long
  + b/c more resources are devoted to maintenance during repro period (than among other primates)
  + b/c feeding niche reduces mortality hazards
* Nat Sel favored increase in diversion of resources to maintenance because
  + Increased investment in skill (embodied capital) favors increased investment in longevity
  + The payoffs to post-repro investment increase repro value
    - more than they would if they were directed to own repro

**Infant mortality**

* high among HG 0-5
* not as high among USA 0-5

Total fertility rate (TFR)

**Grandmother hypothesis (Hadza, extracted roots, got berries)**

* humans evolved long post-repro lifespan because investment in offspring and provisioning of offspring increases their RS through descendants
  + does not explain **costly brain**, argues that long childhood is an artifact of long lifespan
  + ignores why **men have long lives**
  + ignores **large male contribution** to diet
  + GM are **not major food providers** in any HGs
  + GMH does not explain **age-trajectories of production**

**GMH vs Embodied Capital**

* Long childhood: GMH says it’s an artifact of lengthening lifespan; EC says that you need long childhood to invest in growing brain and learning
* GMH: childhood is waiting period to adulthood, no functional delay
  + Once you are physically mature, can learn appropriate skills quickly and effectively to become producer
  + Limitations on performance due to small body size
* ECM: human food production niche requires long developmental trajectory
  + Adult-level production limited more by skills: knowledge-based growth rather than physical constraints (strength/body size)

**Post-reproductive lifespan**

* Pool of grandchildren that you can help is much larger when you are 50-65, than when you are 40-45, so makes sense to live that long
  + Past 65 you kind of max out, so the pool of those needing care is declining so no longer needed
    - Costly to keep body alive, maintenance and repair past age 70 is hard
    - Benefits after 65 starting to decline
      * So human life span is about 65 in HG
* **Tsimane:** 
  + You stop investing in your own kids at 55, and increase care of grandkids
  + >70, net flow becomes negative (you are beginning to be a net consumer again; become expensive to maintain)

**Marriage, Mating and Divorce**

* Least known about HG mating behaviors

Fertility

* Low fertility?
  + Nomadic existence, strenuous lifestyle, limited food
  + **Kung** TFR = 4.7, IBI=4yrs (**Howel 1979**)
* All subsistence societies have similar fertility
* L15 (survivorship to age 15) pop growth curves – Pop growth and TFR
  + If survivorship is high, people can still afford lower TFR and maintain population growth
  + Most populations in the graph are above 0: in the (+) range
    - Some up to 3%
  + Fertility isn’t much different, but survivorship is higher- leading to higher growth rate
    - Along evolutionary time- this sums up to a huge amount of growth over time
    - Nn+1 = Nert
  + Some secondary sterility from STDs

**Marriage**

Frequency of marriage systems

* Monogamy 16%
* Polygyny 83% (usually occasional [vs common])
* Polyandry 0.5%
  + But pair bonds are universal

Phylogenetic evolutionary tree- which structure was ancestral?

* mtDNA to infer what marriage system our human ancestor had
  + Out of Africa- arranged marriages more common
  + Within Africa – not as common
  + Bride service- both within and outside of Africa
  + Polygyny- low in HGs
    - Consistent with generalizations we discussed previously
* Most are monogamous (but not for life; serial monogamous)
* Two extremes: Ache (up to 10 partners/life) vs. Hiwi (much fewer)
  + Ache promiscuous - (“Kuaeme”)
  + Hiwi- man can’t talk to woman who isn’t his wife; needs to look the other way and let everyone know their intentions
  + Polygyny relatively rare- why?
    - You need 2 partners to secure welfare of offspring
      * Man: more access to females, the better (if he doesn’t have access to a wife, something is wrong, there’s some limitation)
      * For women: would you rather be a 1st wife of a poor man, or a 2nd to a richer man?
      * Might make sense when there are properties to consider – when wealth is on the table.
      * As an HG, there is some difference in skillsets
      * But for the most part, because of sharing- are you going to be much better off as a second wife to someone vs. a first?
        + Probably not, **stick to monogamy**
        + So relatively little polygyny in HGs
    - From biological perspective, most human monogamy is still effectively polygyny
  + Fertility is different between the sexes –
  + **Reproductive Variance**: the biological explanation
    - If you were completely monogamous, repro variance would be the same for males and females
    - Effective polygyny- serial monogamy, would create differences between male and female fertility
      * That would create a like-polygynous system
    - In the US: After divorce, men are more likely to mare than women, and if they have more children later, then you could end up in a similar situation
* Ache:
  + Women cluster around TFR = 8 (range: 3-12)
  + Very few have no kids
  + Men: more variability
    - More that had none
    - But may go up much higher than 20 if he’s successful
      * No female can come close
  + Variability is typical of polygynous systems
  + But what looks like polygyny can really be serial monogamy
    - B/c of repro variance

**Polygyny Threshold Model**

* Women choose mate based on net available resources (not absolute wealth)
* **Kipsigi pastoralists**: women choose to be second wives of wealthy men
  + Because land became more limited in this area
  + You could predict who will marry who based on simple logic
  + Wealth was the predictor
    - But there is more than just wealth to being married, so some would choose to be first than second

**Cross-cousin marriages – all first cousins**

* Most common pattern among HGs (**Kelly**, table 7.5)
* Norm violation of marriage rules:
  + Flexibility: 17% of Gidjingali marriages fit rule
  + 11% of Gwi marriages fit rule
  + having flexibility is important (small pop density)- if rules were more rigid, you’d have to wait longer to find a partner
* **Yanomamo** men manipulate kinship categories to marry more women

Marriage arrangements between bride and groom’s families

* ***Absence of exchange most common 34%***
* Bridewealth
* Gift exchange
* Brideservice
* Token bridewealth
* Exchange of sisters
* Dowry

**Divorce**

* As a fxn of age- # of spouses as reported by women
* What constitutes as a spouse?
  + No formal ceremonies
  + So maybe “date” is the norm
  + But even with no formal marriages, there’s a recognition that you are paired with someone
  + High divorce rate
* Pair-bond stability:
  + What are the payoffs of staying with a partner vs deserting them
* **Male parental investment:** 
  + Pair-bond stability commonly modeled as a function of trade-offs between Mating Effort vs Parental Investment
  + What’s the impact of male parental investment vs. opportunity costs of staying around
  + Women are more committed to their offspring
  + Monogamy itself is not that common across mammals
    - So desertion is more accepted across societies
    - But divorce is not celebrated
* Reported Causes of Ache Divorce
  + Hill and Hurtado 1996
  + Men: wanted a different spouse
  + Women: spouse was stolen by someone else
  + Fighting, unhappy, sexual infidelity
* The longer a marriage exists, the less likely you will divorce
* **Hadza** – causes of divorce (**Marlowe 2005**)
  + Extra-marital affairs (both for men and women) are highest cause
* Jones and Marlowe (2001)
  + Quantified trade-off of PI vs ME
  + **Cost of desertion: “father effect” on child survivorship**
  + **Potential benefits** **for men**: # of repro-aged females per adult male
  + 1. Hiwi (1)- lowest divorce rate
    - 1.1x more likely to survive if dad was around (little effect)
  + 2. Kung (2)
    - if dad is around 1.3x more likely to survive
  + 3. Hadza (3)
    - if dad was gone, no more likely to survive than not
  + 4. Ache (4)- highest divorce rate:
    - if a father dies, there is a penalty that children pay in survivorship
    - Ache- when people die, often kids are thrown in with them
      * No one wants to take care of them
      * 1.6x more likely to survive if dad was around (high effect)
      * **highest divorce, but father matters the most**
  + Fertility units per male:
    - Highest for Ache
    - Lowest for Hiwi and Kung
* PI is high when fertility units per male are low, and vice versa
  + As a male, it’s smarter to invest in your offspring if there are not many females around for you to fuck around with
  + It pays for men to stick around more than to desert

Hookup culture

* 57:43 F:M nationwide public universities
* Unequal gender ratios at colleges are driving huckup culture
* When there is female bias (more than males): men have the leg-up in the game, it’s better for men to bounce around, less monogamy

**Aka – best fathers in the world**

* While other women hunt, men look after babies
* Let them suck on their nipples
* **Barry Hewlett – anthropologists**

**Other aspects of Sexuality**

* Homosexuality
  + A modern luxury? Product of western culture? Release from ‘constraints of reproduction’?
  + No systematic study of sexuality among HGs
  + Anecdotes of gay men
  + Few to no reports of gay women
* Onset of sexual behavior
  + Early, post-menarche, lots of teen sexual play
  + First marriages and births later (~17-19 in women)

**Sneaky f\*ckers**

* Secondary paternity
* Pregnancy is a result of multiple acts of intercourse often can involve multiple men
  + Sometimes known who “primary father” is, and who “secondary father” is
    - Amazonian groups mostly
    - Survivorship: 2 dads, better than one
      * But 3 is worse
    - Primary fathers have much higher fertility
    - But life-stage strategy
    - you’re more likely to be a sneaky fucker when you’re a young guy
      * when older, almost all of your paternity it from primary paternity
      * very little from secondary paternity

Coke bottle in the Kalahari

Fairness

Historical revisionism

Continuous vs. respecialized foragers

**Malthusian Law of Population**. The theory claims that growing population rates contribute to a rising supply of labour and inevitably lowers wages. In essence, Malthus feared that continued population growth lends itself to poverty. The book was independently cited as a key influence by both [Charles Darwin](https://en.wikipedia.org/wiki/Charles_Darwin) and [Alfred Russel Wallace](https://en.wikipedia.org/wiki/Alfred_Russel_Wallace) in developing the theory of [natural selection](https://en.wikipedia.org/wiki/Natural_selection).

**Bateman's principle**, in evolutionary biology, is that in most species, variability in reproductive success (or reproductive variance) is greater in males than in females.

**Lachester’s law** - calculating the relative strengths of a predator–prey pair, originally devised to analyze relative strengths of military forces.

* The Lanchester equations are differential equations describing the time dependence of two armies' strengths A and B as a function of time, with the function depending only on A and B

**Key words:** Death, birth, immigration, emigration, [Malthus’ population principle](#Population_Theory), growth rate, [r/K selection](#R_K_selection), kin selection, overpopulation, [Bateman’s principle](#Batemans), sexual selection (intersexual, sexual) and mate choice, density-dependent selection, biomass density, carrying capacity, [Life History Theory](#Life_History_theory), survivorship curves, environmental factors, demography, population viability, spatial distribution, genetic diversity, operational sex ratio, [Trivers-Willard Hypothesis](#Trivers_Willard), [metabolic hypothesis for birth timing](#Metabolic_hypothesis_for_birth_timing)