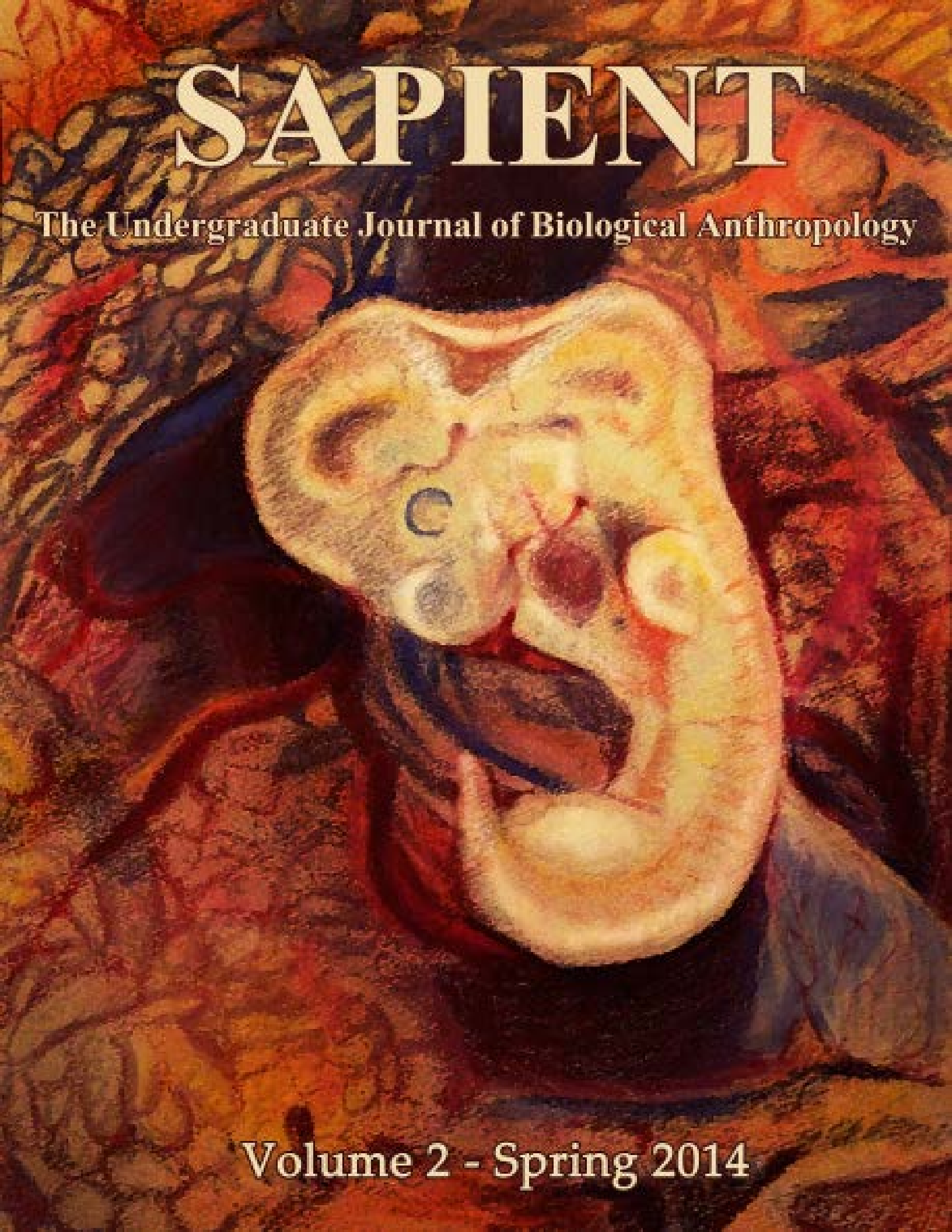
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LETTER FROM THE EDITOR

On behalf on my fellow editors, as well as our faculty liaison Professor Jill Shapiro, it is my honor to bring you the second volume of *Sapient*, the Undergraduate Journal of Biological Anthropology.

This journal was created as an opportunity for students in all academic fields to submit works related to four topics: Human Variation and Genetics; Evolutionary Theory and History; Primate Behavior and Ecology; and Paleoarchaeology and Morphology.

This year, our focus has been on growing the *Sapient* community, soliciting submissions from across the continent and building an online presence across social media platforms. We hope that this community becomes a place for undergraduates to discuss new developments in the field, as well as share their own research with their peers.

— Faith Williams

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Elora López, Columbia University

When British colonists began to arrive and settle in what is now Kenya towards the end of the nineteenth century, it was not a simple matter of one group of people meeting and interacting with a single other group. Kenya had been a site of extensive immigration and intermixing of different peoples, including those of Arabian, Indian, European, and various African tribal descents, since at least the tenth century A.D. (Constantin, 1989). Upon arrival, however, the British settlers brought new ideologies and policies of governing interactions with non-European peoples, thereby altering the dynamics of race relations in the region (Hindlip, 1905). Kenya's premier eugenics organization, the Kenyan Society for the Study of Race Improvement (KSSRI), did not arise until 1933, but policies that discriminated against nonwhite Kenyans were enacted in Kenya as early as 1895 (Campbell 2010; McCormack, 1971). This paper will explore how long-standing sentiments of racial superiority among the white minority in Kenya eventually paved the way for the rise of eugenics in the colony and review the primary literature of the leading Kenyan eugenicists of the 1930's. It will conclude by briefly evaluating the impact that this literature had on racial attitudes and policy in the colony.

One clarification should be made before continuing further into the discussion of race relations in colonial Kenya. From its establishment as a British colony in 1895 until 1920, the region was referred to as the East African Protectorate. The name changed to Kenya Colony in 1920 and remained that way until the country gained independence in 1963. From independence until present the country has been named the Republic of Kenya. For the sake of simplicity, I will refer to the region as simply Kenya or "the colony.

While the eugenics movement did not flourish in Kenya until the 1930s, the underlying pseudo-scientific prejudices on which it was to be based manifested themselves from the outset of British colonization. When settlement began in 1895, Britain swiftly installed a governmental apparatus predicated on the notion of Kenya as a "white man's country into place (Hill, 1944; Hindlip, 1905). Within ten years of the colony's establishment, white settlers had already begun publishing books and papers that described their nonwhite neighbors in terms that clearly indicate notions of biological racial difference. Lord Hindlip, a prominent early settler, referred to venereal disease in the colony as having been imported by Indians and Swahilis (1905). Disease was a primary argument for racial segregation in the early years of the colony (McCormack, 1971). Hindlip's argument for the separation of white and nonwhite settlements in the colony reflects much of the accepted settler mentality of the day:

"…it is to be hoped that no native village or Indian bazaar will be allowed as near the European settlement as at present at Mombasa. Both Germans and Belgians as well as, I believe, our own West Coast authorities have found that the healthiness of a place is greatly increased by not allowing any native habitations within a given distance of the white settlement" (1905, p.37).

Hindlip's assertion that proximity to native and Indian settlements must be the cause of disease in white settlements displays either ignorance of or disregard for the detrimental effects of colonial policy on public health. He neglects to mention that one of the reasons disease affected Indian settlements more severely than white settlements was that the law forbade Indians in Kenya from inhabiting the highland regions where whites chose to settle, and thus forced Indian laborers to settle low-lying regions that were plagued with disease-carrying mosquitoes and less fertile cropland (McCormack, 1971). He instead chooses to attribute disease to racial difference, and thus indicates a perspective that hints at biology rather than geographic or socioeconomic factors. The presence of this perspective in the colony would make it easy for eugenic theory to become accepted by many prominent white settlers in fewer than three decades.

Ethnologists' attempts at racial classification of tribal groups serve as additional indicators of the presence of biological conceptions of race in the early years of the colony. White anthropologists in Kenya, like those in the rest of Africa, devised systems that lumped tribal groups into any number of races. The Masai tribe particularly interested ethnologists because of their European-like noses (Seligman 1930; Shaw 1995). Seligman attributes the Masai's "finer nose" and red-tinted skin to their being in the half-Hamitic racial group, which he explains is a group created by the mixture of the Hamite and Negro races (1930, p.161). The Hamites were supposedly the Arab branch of the Caucasian race, and Seligman asserts that the integration of Hamitic peoples resulted in civilization within black Africa (1930). Shaw argues that the British colonists' declarations of Masai beauty, as opposed to the derogatory descriptions given to the more traditionally "African-looking" Kikuyu tribe, stem from admiration of the Masai's perceived European-like features (1995). This practice of explaining races in relation to British norms, and of claiming innate, Caucasian ancestral superiority, would be critical to the adoption of a eugenic framework in the near future.

The "Black Perils" debates are perhaps the aspect of colonial Kenya that made eugenics theory most amenable to white settlers. White fear of black sexual assault became increasingly more pronounced from 1907 to 1926 (Anderson, 2010). There were few documented cases of Africans sexually assaulting white women and children throughout this period. From 1910 to 1920, Kenya's colonial courts convicted four Africans on the

basis of indecent exposure in front of a white female, three on the basis of attempted rape of a white female, and just one for actually raping a white female (Anderson). These cases generated so much sensation in the Kenyan press, however, that the East Africa Women's League, among other white settler groups, urged that the death penalty be made the punishment for rape and attempted rape (Anderson).

White settlers claimed that their terror had been justified when Mrs. Julia Hepzibah Ulyate, a 69 year-old widow who lived alone, was robbed and raped by an African assailant (Anderson, 2010). The demand for a bill that increased the stringency of punishments for rapists once again rose to the forefront of the colony's politics, but most pertinent to this discussion was the debate over whether or not a standard of racial discrimination should be included in the bill. Capt. Kenealy, a British-employed representative who had spent time in West Africa, argued that

"The issue should not be confused by widening the scope of the legislation and by introducing the element, or rather by introducing a principle of refraining from racial discrimination where racial discrimination in my opinion is essential…It is a cowardly course to ignore the obvious racial differences and it is reasonable to recognize these differences in legislation. We want legislation to protect our womenkind against the native; why obfuscate the issue by pretending there are other factors" (Kenya Hansard, 1926, p.247-248).

Other colonists may have favored Kenealy's blunt argument for a law that would specify the death penalty only in the case of black men assaulting white women. However, they recognized that British Parliament would not approve of such blatant racial discrimination, and so they chose not to include Kenealy's specification (Anderson). The arguments against racial discrimination in the law were not, however, any less racist than Kenealy's argument. Lord Francis Scott, who advocated refraining from specifying race in the bill, explained that although the bill would not blatantly favor racial discrimination, that should not stop people from remembering that Africans and British were descended from savage and enlightened civilizations, respectively, and that rape against a white woman was worse than that against a black woman (Kenya Hansard, 1926). The colony ended up agreeing with Scott, but the fact that even those who argued against legalizing racial discrimination did so while assigning differential moral worth for women of different races indicates that by 1926 Kenyan colonists were in the right frame of mind for a eugenic revelation (Anderson).

By the time of the "Black Perils" debates, white settlers openly acknowledged that their African neighbors no longer considered them demigods (if, indeed, they ever had). The white settlers considered this a frightening challenge to the colony's racial norms (Kenya Hansard, 1926). For years, white settlers had assured themselves that the lines between whites and blacks were obviously apparent, and that white prestige would protect the minority settler group (Shadle, 2010). They reasoned that black men who felt increasingly more emboldened to defy white prestige or demigodliness, were a terrifying prospect for the future of the white settlers in Kenya (Kenya Hansard, Shadle). Thus, as the 1920's came to a close and the colony neared its thirty-fifth year as a British holding, the stage was set for a vehement and vocal eugenics movement (Campbell, 2010).

The leader and primary voice of the Kenyan eugenics movement was Henry Laing Gordon, a psychiatrist who moved to the colony in 1925 (Campbell, 2010). Gordon had no formal training in heredity or evolution, but as no one else in the colony did either, once he assumed the role as expert on the subject, the rest of the colony was willing to regard him as the authority (Campbell). Gordon's primary concern was that European standards of mental capacity, idiocy, lunacy, and the like could not be adequately applied to the natives of East Africa, and he was convinced that it was the responsibility of scientists to delineate the differences between the two racial groups (Gordon, 1934b).

Gordon's first study, which aimed to measure the intelligence of 219 African boys in a Kenyan reformatory, demonstrated his reasoning for advocating separate, race-based standards of mental capacity (1934a). Gordon's finding that eighty-six percent of his subjects were "aments," or mentally deficient individuals, is striking (1934a). Even more striking, however, is Gordon's claim that the fourteen percent who were not labeled aments "could not be fitted into European ideas of normality without creation of two low classes-a low-grade normal and, lower still, a border-line normal. It was clear that a considerable proportion of this alarming result came from the use of European standards on another race" (1934a, p.223). Gordon thus not only emphasized white superiority, but asserted that whites were on a wholly separate metric of intelligence. Scientific eugenic rhetoric had arrived in Kenya.

Another instance in which Gordon argued for the necessity of applying completely different standards between races was in the identification of disease. In an attempt to investigate the claim that African natives more often contracted syphilis in urban environments than in rural ones, Gordon tested 162 healthy adult Africans and 112 Africans suffering from either psychosis or amentia for the presence of spirochaetes, the bacteria responsible for syphilitic infection (1933). He determined that 55.9% of patients suffering from amentia or psychosis tested positive for spirochaete infection (Gordon, 1933). Gordon's concluding remarks, which included that manifestation of disease must be different in every race, and that syphilis reacts differently in different peoples based on their mental and physical condition, were by far the most influential of his entire paper (1933). In essence, he attributed the different manifestations of syphilis in white and in black sufferers (if there really were any at all) to the differences in the sufferers' racially determined mental integration. This was seen as validation of Gordon's argument that mental capacity was based on completely separate, inherited racial standards (1934a, 1934b).

F.W. Vint, a government pathologist, added authority and empirical clout to Gordon's research and assertions of whites' genetic superiority by studying the brains of Kenyan natives (Campbell, 2010). In his study of one hundred African skulls, Vint concluded that the brain weight of the average African male was 10.6% lighter than that of the average male European, and that African pyramidal cells within the brain were smaller than European pyramidal cells (1934). Perhaps more interesting was his conclusion that African brain weight reached its peak before the age of eighteen, and then decreased for the rest of the individ-

ual's life (Vint, 1934). This finding led to the conclusion among many in the colony that the average adult African had about the same intelligence level as the average European eight year-old (Campbell). Vint's highly technical and methodical (but ultimately inaccurate) work lacked Gordon's clearly eugenic rhetoric, but it bolstered the eugenics movement's legitimacy with its clarity and obvious regard for the scientific method (Campbell).

Although Gordon's research focused on the intelligence and brains of Africans, as a staunch eugenicist, he was also concerned with the genetic state and viability of his own race. He warned that Kenya Colony was in danger of developing

"…a poor white group, a submerged Asiatic group, and a huge African group of alarming potentialities. The reason is most evident in the case of the native. TRUSTEESHIP is being interpreted as nurture only. We are trying to create a new civilisation by repeating the old problem of neglecting nature" (Cited in Campbell, 2010, p.294).

In order to inhibit the development of a two-class white group, which would limit the current superiority held by whites in Kenya, Gordon urged that only well-bred, high-quality Britons be allowed to settle in the colony (Campbell). It is interesting that Gordon here mentions the Indian minority in Kenya, for he did not conduct studies of Indian intelligence and brain capacity in relation to those of Africans and Europeans.

The work of Kenyan eugenicists (primarily Gordon) gained considerable attention in Kenya colony, but British reception was not so favorable. When Gordon founded the KSSRI in 1933, its membership constituted a larger percentage of the white Kenyan population than the British Eugenics Society did of the white British population (Campbell, 2010). The society aimed to address both the "backwardness" of the African race and to prevent the degeneration of the white settler race (Campbell; Gordon, 1945). These seemed to be worthwhile aims to white settlers, who were spurred by fear and the need to continue asserting superiority over the Africans, but by the time it became better known in Britain, racial eugenics had begun to be tainted by association with Nazi Germany (Campbell). When Gordon wrote his 1945 article for The Eugenics Review, in which he continued to urge the importance of encouraging young, healthy British men and women to come settle Kenya and explained that further study of African backwardness would shape education, crime, and health policy, the Kenyan eugenics movement had almost completely become a thing of the past (Campbell, Gordon).

The scientific research and support for the Kenyan eugenics movement may have lacked in duration, but it still made an impact on the education and crime policies of the colony, as Gordon said it would (1945). In 1934, primary education became mandatory for white children in Kenya, but no such measure ever passed for the supposedly mentally inferior black children of the colony (Campbell, 2010). Criminality became further identified as a lowly African trait, and Africans were charged with more stringent punishments for crimes than were criminals of other races (Campbell, 2002). It was not uncommon for juvenile Asian law offenders to get by with lesser sentences than juvenile African law offenders, and juvenile European offenders could even manage not to be sent to the reformatory at all for the same crimes, all on the basis of race (Campbell, 2002). Gordon's work may not have received the professional clout that he had hoped for on the international scale, but Kenya's legal system and white culture certainly took heed of his research and conclusions (Campbell, 2010).

Reviewing white settlers' attitudes and policies towards nonwhites in Kenya from the earliest days of settlement to the advent of Kenyan eugenic research elucidates why Kenyan colonists would be so apt to accept eugenic theory is understood. First, Hindlip's characterization of Africans and Indians as vectors of disease demonstrates that settlers made links between race and biology from the outset. Second, Seligman's depiction of the Masais as being more civilized and beautiful due to their supposedly mixed Caucasian blood indicates a tendency to evaluate other peoples in reference to one's own. Third, the fear of a loss of white superiority that stemmed from the "Black Perils" debates shows a tiny population willing to do anything to hold onto their hegemony in a foreign land. All of these factors allowed Gordon, Vint, and others to promote their ideas of genetic African backwardness and the necessity of strengthening the white genetic stock of Kenya without too much opposition from their fellow Kenyans (Campbell, 2010). The implications of the short-lived eugenics movement in Kenya outlived the movement itself, as can be seen in the racist educational and criminal laws that the colonial government established after the publication of Gordon's most seminal works. Gordon cannot hold all of the blame for Kenya's race-based policies, however. As has been explored here, his ideas were only given authority and appreciation because of the decades of semi-science-based racist attitudes that preceded him.

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The Genetic Basis for Taste Perception: A Review

Chris Kendall, McMaster University

There is significant variation in what is considered palatable among different human populations and even among individuals. It is currently believed that humans have five taste sensations: sweet, sour, salty, bitter, and umami (Bakalar, 2012). Nevertheless, it is unclear whether the perception and preference for each of the five tastes are genetically or environmentally determined. Almost 100 years ago, pioneering research projects were undertaken that discovered bitter taste recognition was genetically heritable (Blakeslee, 1932; Drewnowski & Rock, 1995). From then on, there has been a large body of research focused on determining the exact role genetics play in human taste perception, variable taste sensitivity, and personal preference for particular tastes. It is possible that genetic and environmental contributions vary among the five taste sensations. Given that a strong fondness for sweet taste can result in obesity and chronic disease, the study of taste perception is central to human health and diet. At the moment, much of the research in this field is still in its infancy, although new discoveries are continually being announced.

Taste perceptions are recognized by the brain through a chain of receptors. These receptors are housed in the form of taste-receptor cells placed across the tongue on our taste buds (Chandrashekar et al. 2006). Different receptors are responsible for different tastes. Sweet and umami thresholds are created by G-protein-coupled receptors (GPCRs) T1R1 through T1R3 (Chandrashekar et al. 2006). These receptors are created by the family of genes that encode for them, TAS1R, and work together as heterozygotes to form sweet (TAS1R2 and TAS1R3) and umami (TAS1R1 and TAS1R3) receptors (Drayna, 2005). Similarly, bitter taste recognition is also controlled by GPCRs, this time by T2Rs (Chandrashekar et al. 2006), which bond to specific intracellular G proteins, such as G α-gustducin (Drayna, 2005). Sour taste is recognized when two transient receptor potential ion channels are stimulated when acidic items activate the taste buds (Garcia-Bailo et al., 2009). Lastly, salt taste recognition is created when two receptors, the sodium-specific and amiloride-sensitive epithelial sodium channel (ENaC) and the transient receptor potential cation subfamily V member 1 (TRPV1) channel react to a stimulus (Dias et al., 2013). These taste receptors and ion channels then transmit these perceptions to the taste center of the brain, the cortex (Hoon et al., 1999).

Of the five taste sensations, bitterness was the first taste response tested for genetic heritability. It was discovered in the early 1930's that people are either "tasters" or "non-tasters" of bitter compounds like Phenylthiocarbamide (PTC) and Propylthiouracil (Prop) (Blakeslee, 1932; Drewnowski, 1997; Drewnowski & Rock, 1995). In one of the inaugural experiments on bitter taste, Blakeslee (1932) found that when both parents are non-tasters, their child would be a non-taster; but when one parent is a taster, the child has almost a 60 percent chance of being a taster; and when both parents are tasters, this increases to 83 percent, suggesting a partially dominant mode of inheritance for this taste perception. More recent research has discovered that approximately 65 percent of people are tasters of bitter compounds (Birch 1999; Garcia-Bailo et al., 2009; Wooding et al., 2004). The ability to taste bitter compounds has been suggested to be evolutionarily adaptive, as it could have prevented our ancestors from eating toxic substances (Drewnowski & Rock, 1995; Wooding et al., 2006). Glendinning (1994) argues that bitter taste is necessary for carnivores and omnivores (humans, for example), or those furthest trophically removed from ingesting poisonous substances, as this confer protection against harming themselves, while consequently the bitter taste response is maladaptive to herbivores, as it would drastically reduce their range of viable food options. For this reason, it is surprising that the non-taster phenotype exists at such a high frequency in modern populations.

Genetic analysis on the origins of non-taster phenotypes have found that non-synonymous single nucleotide polymorphism (SNP) mutations in the TAS2R38 gene account for 85 percent of the phenotypic variance in bitter taste currently seen in humans (Campbell et al., 2012; Garcia-Bailo et al., 2009; Wooding et al. 2004; Wooding et al., 2006). It is important to note that the TAS2R38 gene is the vehicle that holds the receptors responsible for taste recognition of bitter items (Duffy et al. 2004). Such SNPs alter the amino acid sequence of the encoded protein and appear to have occurred around 2.1 million +/- 455,000 years ago (Campbell et al., 2012). The mutations must have occurred before the divergence of Neanderthals and modern humans, as Neanderthals have been found to be carriers of the non-taster allele (Lalueza-Fox et al., 2009). Interestingly, both chimpanzees and humans exhibit similar mutations in the TAS2R38 gene, but it has been established that these mutations evolved independently of one another in the chimpanzee and hominin lineages (Campbell et al., 2012; Wooding et al., 2004; Wooding et al., 2006).

In order for the diversity of tasters and non-tasters presently seen in modern human populations to have been maintained, balancing selection in favour of heterozygotes must have occurred deep in our evolutionary past (Wooding et al., 2004). This claim is further strengthened by Campbell et al.'s (2012) analysis of TAS2R38 SNPs in African and non-African populations, which indicated that African populations exhibit an abundance of non-synonymous SNPs not seen in non-African populations

that lead to variances in the perceived bitterness. Additionally, Garcia-Bailo et al. (2009) compiled data for a large group of heterogeneous populations and asserted that 97 percent of Africans, 85.5 percent of Chinese, 60 percent of Indians, and 70 percent of Caucasian North Americans sampled could taste bitter compounds . Taken together, there is strong evidence for an ancient mutation on the TAS2R38 gene on chromosome seven, which created a non-taster phenotype seen in human populations. It is likely that the non-taster allele has been maintained through the course of human evolution by the action of balancing selection.

Balancing selection is the act of favouring more than one allele, inhibiting fixation of said allele (Jobling et al. 2004: 499). This evolutionary trajectory of balancing selection creating bitter taste through heterozygosity makes sense if Glendinning's (1994) theory holds true. Humans, being omnivores, even deep in our evolutionary past, relied on a wide variety of food items including fruits, leaves, and C13 enriched foods found in the grasslands, or potentially ate animals who ate these types of food (Sponheimer & Lee-Thorp, 1999; Wrangham, 2009). Thus, it stands that evolution of the bitter taste receptor would have been beneficial to human survival deep in our past, explaining the ancient emergent dates in much of the literature. As time went on, and human groups became more geographically focused, the flora and fauna consumed would have remained relatively constant, creating a limited need for a bitter indicator as cultural practices would warn against ingesting such items. Therefore, more modern selection trends may have favoured non-tasters as it no longer conferred an evolutionary advantage, again promoting balancing selection in favour of non-tasters in some regions of the globe.

All of the information above makes a clear case for a large, or entirely, genetic component for bitter taste perception in humans. However, the additional SNPs on the TAS2R38 gene in African populations only explains no more than 35 percent of the variability seen in bitter taste sensitivity (Campbell et al., 2012). They conclude that there are likely other genetic components, coupled with environmental, and even potential epigenetic contributions, which comprise the entirety of variation seen in human bitter taste (Campbell et al., 2012). Additionally, Birch (1999) discussed a study which found that infants did not immediately reject a bitter substance known as urea, but as time went on, between two and 24 weeks later, the infants began to reject this liquid. Gradual rejection of bitter substances in infants suggests two potential hypotheses. The first is that bitter taste rejection is actually partially shaped by experience or environmental contributions (Birch, 1999). Secondly, if bitter taste is entirely controlled by genetics, then the TAS2R38 gene may not become active until sometime after birth when the infant reaches a maturity in their life . Some evidence exists for this hypothesis. In a study of 980 individuals aged three to 55, it was discovered that children have a stronger revulsion to bitter compounds, and this wanes as time goes on; this was especially significant for heterozygous individuals (Mennella et al., 2010). Therefore, strong genetic control is exhibited on TAS2R38 at an early age, but potential environmental influences reduce the strength to which bitter compounds can be perceived by humans.

Sweet taste perception is the second most researched taste response, only behind bitter taste. As with bitter taste, studies have revealed a strong genetic component for sweet taste. To reiterate, the TAS1R gene family, found on human chromosome one, encodes a family of taste receptors (Garcia-Bailo et al., 2009; Kim et al., 2006) localized on the tongue and palate (Keskitalo et al., 2007). When first discovered, TAS1R1 was believed to work alone, but it is now understood that both TAS1R1 and TAS1R2 work together to detect sweet taste (Chandrashekar et al. 2006; Drayna, 2005; Garcia-Bailo et al., 2009; Kim et al., 2006). Keskitalo et al. (2007) examined 146 subjects, encompassing 26 families, of Finnish descent and studied the intensity and pleasantness of ingesting sucrose liquid, plain water, and PROP impregnated paper. The participants were also genotyped and examined for any linkage between discovered traits (Kekitalo et al., 2007). The report indicates there is a strong link between sweet taste preference and a maker on chromosome 16, known as 16p11.2 (Keskitalo et al., 2007), suggesting that this region may harbour further sweet taste receptors. Young infants exhibit a predisposition to sweetened liquids, and fetuses swallow more often when sweet solutions are added to amniotic fluid (Bakalar, 2012), while similarily newborns continually ingest even the most sweet of liquids (Drewnowski, 1997). All of this data combines to suggest that sweet taste perception is under guided by ingrained, genetic coding.

Sweet taste perception and enjoyment have been found to be partially genetically controlled. Fushan et al. (2010) report that a number of SNPs on the GNAT3 locus, responsible for partial sweet taste perception, located on chromosome seven (7q21, specifically) were found to make up 13 percent of the variability in sweet taste perception across a sample of 160 individuals of varying ancestry. Additionally, Garcia-Bailo et al. (2009) summarize some studies that found sweet taste discrimination to be 33 percent heritable, and the liking of sweet items to be about 50 percent inherited. Kim et al. (2006) assert that the TAS1R family of genes has been under positive natural selection in the past, meaning that these genes were preferentially selected for as they were advantageous, likely as a way to detect many structurally different sweet food items. They further explain that the majority of diversity of these genes will most likely be detected in African populations (Kim et al., 2006), suggested an ancient emergence of these genes.

Addressing similar questions, Keskitalo et al. (2007) report that enjoyment derived from sweet foods was 40 percent inherited, the frequency of sweet food consumption was 50 percent inherited, and the urge to consume sweet items was 31 percent inherited in their study of 26 Finnish families. Evidence exists which connects those who can perceive bitter taste to preference of sweet tasting items as well. Those who find PTC and Prop utterly vile, also known as "super-tasters", have been reported to dislike overly sweet food items (Drewnowski & Rock, 1995). Therefore, it is possible that sweet taste preference is a pleiotropic and polygenic trait, since researchers (e.g. Garcia-Bailo et al., 2009; Kim et al., 2006; Keskitalo et al., 2007) have discovered several genes on different chromosomes which all play a part in the shaping of sweet taste. This suggests that the

TAS2R family of genes may be shaping sweet taste preference as well as bitter taste sensitivity.

Environmental influences also shape how much a person may enjoy sweet items. Newborn infants will continually consume sweet liquids. Beauchamp and Moran (1982) studied sucrose ingestion by 199 infants through the first 6 months of their lives. It was found that all newborns ingest sweet liquids, but when the remaining 140 infants were re-tested again at six months of age, only infants who had been routinely given sweetened water compounds for the past six months showed a preference for this liquid, showing that environment shapes the preference for sweet items (Beauchamp & Moran, 1982). Furthermore, Keskitalo et al. (2007) found sweet food preference was 40 percent heritable, leaving the other 60 percent up to either environmental or epigenetic factors. Therefore, similar to bitter taste thresholds, genetics and environmental factors both influence the urge to consume sweet items.

Sour taste preference has not been researched much to date but it is known that it results from the dual expression of PKD2L1 and PKD1L3, two transient receptor potential ion channels that become activated when acidic items stimulate the taste buds (Garcia-Bailo et al., 2009). Emergence of this taste threshold is believed to have occurred to protect against eating spoiled foods (Garcia-Bailo et al., 2009). Newborns reject sour items, suggesting an innate dislike of these foods (Birch, 1999) and supporting the hypothesis that this taste confers protection for us. The early onset of sour taste aversion also suggests a strong genetic component, given that newborns would not have had the experience required to shape rejection of these items. In order to test the heritability of sour foods, some studies have been undertaken. Kaplan et al. (1967) studied monozygotic and fraternal twins. They discovered that there was no difference between the monozygotic and dizygotic twins in the detection of sour items, suggesting little to no heritability of sour taste (Kaplan et al., 1967). A more recent study by Wise et al. (2007) studying 74 pairs of monozygotic and 35 pairs of dizygotic twins from Ohio found that additive genetic factors made up over 50 percent of the variance in sour taste detection. Lastly, PKD2L1 knockout mice were found to have almost complete loss of sour taste perception, but retain other taste thresholds (Huang et al., 2006). The evidence thus indicates that sour taste detection is under strong genetic control. Unfortunately, there is no current research on how the environment shapes sour taste that has been published at this time.

Similar to sour taste, not much data exists on salt taste preference or detection. However, some genetic contributions have been discovered. Salt taste is thought to be under the control of two receptors, ENaC and the TRPV1 channel (Dias et al., 2013). The genes that encode these two channels, SCNN1B (for ENaC) and TRPV1, have been studied. Dias et al. (2013) found that SCNN1B contained two SNPs which partially controlled salt taste, and one on TRPV1, which probably modifies salt perception for individuals. Keskitalo et al. (2007) discovered that reported pleasantness of a salty solution was about 33 percent heritable, and salt intensity ratings were on average only slightly genetically heritable. Furthermore, Birch (1999) described that newborns favour salty items, just as they do sweet items. As with sweet taste, super-tasters dislike many salty items (Drewnowski & Rock, 1995), suggesting a polygenic and pleiotropic influence of genes that have been or have yet to be discovered.

In addition to the genetic evidence, large amounts of environmental data exist for salt taste thresholds. It has been reported that environmental effects influence salt taste preference more than genetics. For instance, it has been documented that babies under four months of age will drink plain water or moderately salty water without qualms, but by the time the infant reaches two-and-a-half years of age, they greatly prefer saltier water (Bakalar, 2012). In a study by Wise (2007), it was discovered that no significant heritability could be found for salt preference. Birch (1999) describes that as children become older, their preference for salt is mediated by their experience and exposure with it in their food. Moreover, mothers who had moderate to severe morning sickness have children who prefer saltier food, suggesting pre-or-perinatal experience may shape salt taste preference (Crystal et al. 1999; Crystal & Bernstein, 1995; Crystal & Bernstein 1998). Wise (2007) hypothesizes that salt taste may be a by-product of evolutionary forces tailoring our needs for salt based upon the environment in which we live.

Umami, or savoury taste, is the newest discovery in regards to human taste perception. It was discovered in the early 20th century by Ikeda in Japan, and is represented by the taste of glutamate (Kurihara, 2009). Glutamate is an amino acid that is typically found in a variety of foods such as meats, seafood, and some vegetables, and is brought out by monosodium glutamate (MSG) (Garcia-Bailo et al., 2009; Kim et al., 2006, Kurihara, 2009). Since the discovery is so recent, no heritability data for this taste exists. Regardless, there is some evidence for the genetic component to this flavour threshold. The ability to perceive umami results from the TAS1R family of genes, more specifically TAS1R1 and TAS1R3 (Chandrashekar et al. 2006; Drayna, 2005; Fushan, 2010; Garcia-Bailo et al., 2009; Kim et al., 2006). The TAS1R1 gene shows more genetic diversity than TASIR3, which may relate to the influence of the TAS1R on phenotypic variation for both sweet and umami tastes (Kim et al., 2006). Therefore, it is possible that whatever affects sweet taste, such as bitter taste sensitivity, also influences umami taste because as of yet, no environmental influences have been found for this taste perception.

As outlined previously, there is evidence for both genetic and environmental contributions for the five recognized human taste thresholds. Some tastes, such as bitter and sour, exhibit almost exclusively inherited expressions of these traits. Others, such as sweet and salt taste, highlight a balanced mix of genetics and environment contributing to their expression. Lastly, some of the tastes are so new in their testing that not much is really known about how genetics and environment work together to shape these preferences. On average, genetics and environment play an equal role in shaping human taste perception. There is evidence that genetics play a larger role in some taste perceptions. For instance, bitter and sour tastes are at least 50 percent genetically inherited from our parents. Similarly, some genetically measured indexes of sweet taste were found to contribute 13 percent of genetic control over sweet taste perception. In concert, the TAS1R genes and SNPs

on GNAT3 combine to make sweet taste about 55 percent genetically inherited. Some tastes, such as umami, sweet, and salt also exhibit evidence of polygenic and pleiotropic genetic control. Therefore, it is possible that all of the genes which control for these tastes have yet to be discovered, indicating a larger genetic component than is currently presumed. However, to definitively say that genetics controls the majority of human tastes compared to environment would be a major assumption.

Even though it appears that much of human taste preference and enjoyment stems from genetic factors, environment still plays a large part in shaping these distinctions. A phenomenon known as neophobia, or the rejection of novel food items, plays a significant role in what humans consider to be palatable. Birch (1999) summarizes several studies which report neophobia to be nonexistent in newborns, and then it becomes prominent during childhood and adolescence, and finally, almost completely vanishes by adulthood. Bakalar (2012) asserts that food preferences can be shaped very early on, even before birth, when an introduction of garlic to the amniotic fluid caused children to enjoy breast milk with small traces of garlic in it. Thus, it may be possible to mould food choice early on by introducing as many novel food items as possible to newborns to stave off food reluctance, or unhealthy reliance later on in life.

Many of the above tastes have both positive and negative implications on human diet and health. For example, it has already been shown that bitter and sour tastes protect us against the ingestion of toxic and spoiled foods. Nonetheless, bitter tasters, and specifically super-tasters, are likely to be less nutritionally healthy. These groups have been shown to avoid and dislike a wider range of foods (Birch, 1999; Drewnowksi & Rock, 1995). Since they avoid a greater range of foods because they find them unpleasant, they may be at a higher risk for dietary diseases (Drewnowski, 1997). Additionally, they have been found to have a larger number of colon polyps which could lead to colon cancer (Reddy, 2013). Contrary to this, they are probably less likely to be overweight, as they find overly sugary items unpleasant. Conversely though, diets high in sugar and fat consumption can lead to a wide variety of health problems. For instance, in young adults aged 19-29, snacking, including sugary high-fat items, increased from 20 to 23 percent of the total daily energy intake for Americans between 1977 and 1996, possibly contributing to the obesity epidemic seen (Zizza et al., 2001). A second study found that "Western diets" comprising more intake of sugar, and other higher fat foods resulted in significantly higher cases of fatal and non-fatal coronary heart disease compared to those who ate a more traditional, balanced diet in a study group of almost 45000 men aged 40-75 (Hu et al., 2000). Similarly, there have been multiple studies which have found sweet-likers to be overeaters or overweight (Campbell et al., 2012; Drewnowski, 1997). However, much of this has been shown to be a by-product of environment. Birch (1999) discusses that children who were continually fed sweetened items preferentially enjoyed them more than did children who were not exposed to such items. Introducing children to healthy foods when they are young will likely quell neophobia (Birch, 1999), and get them to make healthier decisions as they age (Bakalar, 2012).

In summary, there appears to be dualistic relationships acting on individual taste preferences, coming from both genetics and the environment. While many of the taste thresholds exhibit strong genetic control, environmental factors make up just as much, if not more, of the contribution to taste preferences in some cases. Much more research is necessary to improve our understanding of these taste thresholds. New studies focusing on umami, sour, and salt taste would advance our understanding of the genetic basis of these tastes, as current literature remains unclear on the specifics. Additionally, there is not much available evidence on the change of taste perception over time. For instance, over the course of human growth and development, there are likely epigenetic changes to chromatin that may influence taste discrimination and enjoyment. Furthermore, the addition of preservatives and other chemicals to modern manufactured food may shape taste perception in some way, potentially cross-culturally, or even as we age. Even though data exists for varying regions of the globe, to the knowledge of this author, there has yet to be a comprehensive collection of studies that examines the changing taste preferences of varying cultural and geographic regions as these groups age and diversify. Longitudinal studies of taste perception are also necessary to answer some of these emerging questions.

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Functional Consequences of Evolutionarily Important Genetic Variation in Baboon Major Histocompatibility Complex (MHC)

Meredith Rahman, Duke Univeristy
Collaborators: Jenny Tung, Amanda Lea, Shauna Morrow

Introduction

The major histocompatibility complex (MHC) represents a family of genes that help regulate immune responses by producing peptides that work with T-cells to differentiate self from non-self (pathogens) (Loisel, 2006; Morzycka-Wroblewska et al., 1996). Particular promoter haplotypes of these genes, which may alter their regulation, have been connected with the development of autoimmune diseases such as celiac disease and rheumatoid arthritis (Nepom and Erlich, 1991). Given MHC genes' influence on immunity and fitness of individuals, selection may have determined which alleles were maintained in the gene pool.

A promoter region, which precedes the coding region of a gene, is where polymerase and transcription factors bind to DNA to prepare for RNA production. The promoter area for class II MHC genes contains several conserved regions called the S, X, and Y boxes, which contain sequences essential to transcription factor binding (Benoist and Mathis, 1990). Class II MHC gene HLA-DQA1 has three additional conserved boxes, NF-kB, W, and J, which help construct the peptide binding region (Loisel et al., 2006, Hughes and Yeager, 1998). The polymorphic promoter region of HLA-DQA1 includes at least 47 haplotypes. Within baboons there are 3 promoter haplogroups, groups of associated haplotypes containing SNPs, which include 12 total haplotype s (Loisel et al., 2006).

Variations in transcription regulate MHC class II expression (Morzycka-Wroblewska et al., 1996; Beaty, 1995). Variations in the promoter region may alter the strength of binding transcription and can change the amount of RNA product produced. Single nucleotide polymorphisms (SNPs) have been shown to change promoter strength by four to five times (Morzycka-Wroblewska et al., 1996). By quantifying relative amounts of RNA product produced using each promoter allele we may determine functional effects of sequence variations that may have conserved diversity at this region.

Haplotypes of MHC promoter regions have been preserved over time. Research has found that allelic variants in the cis-regulatory region of HLA-DQA1 are more similar between primate species than within a species. This indicates maintained trans-species polymorphism, perhaps due to balancing selection (Loisel et al., 2006). Genetic variation in the promoter region may impact expression of HLA-DQA1 alleles. Given preservation of allelic variation in the promoter region within species and similarities of alleles between species, we predict there is a functional benefit to heterozygosity at

the promoter region of HLA-DQA1. The idea in evolutionary biology of the "heterozygote advantage" asserts that genetic diversity can make a population more fit because it can combat against a greater variety of pathogens. A study exposed MHC heterozygous and MHC homozygous mice to multiple strains of bacteria and found that the heterozygous mice had higher rates of survival (Penn et al., 2002). Differences in survivability may be due to MHC diversity; changes in the promoter region may have similar benefits. Pyrosequencing has been previously used to measure relative amounts of RNA product of a particular gene with different promoter alleles (Wittkopp, 2011). This method may identify functional effects of differences at the promoter region.

In this study we examine the cis-regulatory region of HLA-DQA1, a class II MHC gene, in Papio cynocephalus (yellow baboon). We focus on the functional importance of variation at this region and what that may demonstrate about the evolution of MHC genetics in primates.

Predictions

We predict that promoter haplotypes of the same haplogroup within an individual will produce similar amounts of HLA-DQA1 transcript. Similarly, we predict that promoter haplotypes of different haplogroups within an individual will produce different amounts of HLA-DQA1 transcript. Differences in abundance of transcript product may affect the gene's ability to identify pathogens. Thus this functional difference in promoter haplotypes may help explain this region's trans-

species polymorphism.

Methods

DNA samples of 21 baboons (Papio cynocephalus) from the Amboseli basin of Kenya were used to analyze the functional consequences of genetic variation in the promoter region of the gene HLA-DQA1. These individuals were selected based on their promoter haplotype: seven have two promoter haplotypes from one haplogroup, seven have two promoter haplotypes from a second haplogroup, and seven were heterozygous and had one haplotype from each haplogroup.

Sanger sequencing was used to determine the promoter genotypes of most of our individuals. Since single nucleotide polymorphisms (SNPs) differentiate promoter haplogroups, we sequenced 200-250bp upstream of HLA-DQA1 to find which SNPs, and therefore haplotypes, were present in each individual. Primers DQApFor (5'-CAGACATGCACACACCAGAGAA-3') and DQApForII (5'-TGCACACACCAGAGAAGATTCC-3') were used with DQApRev (5'-GGATCATCYTCTTCCCAAGG-3') to identify the promoter region for PCR amplification (Loisel et al. 2006). Sizes of fragments produced from the PCR were confirmed using gel electrophoresis. Amplicons were then cleaned using QIAquick kits and cycle sequenced using a "big dye" reaction. The Duke Sequencing Core used Sanger sequencing to provide the DNA sequences of each individual's promoter region. Other individuals were sequenced using capture array followed by Illumina HiSeq. Sequences were then compared to previously published reference sequences of each haplotype; thus each individual's promoter genotype was determined (Loisel et al. 2006).

Downstream in the coding region we began to investigate the sequence of the gene's RNA transcript product. Since RNA is unstable and single stranded, primers would not bind and therefore we converted RNA into cDNA. Primers GH26 (5'-GGTGTAAACTTGTACCAG-3') and GH27 (5'-GGTAGCAGCGGTAGATTG-3') then could amplify our region of interest within the gene (in exon 2; Scharf et al. 1986). Sanger sequencing was used to sequence this exonic region in each individual and identify SNPs that would indicate which haplotype the product belonged.

Results

We successfully determined the sequence of the promoter region of 17 out of 20 samples.[1] Primers DQApFor and DQApForII were used in separate reactions with DQApRev in a previously published Sanger sequencing protocol (Loisel et al., 2006) Repeated attempts to sequence three samples with poor sequencing reads (Vet_F, Nyuki_F, and Nyuki_F2) did not provide clear sequences. Therefore these two individuals, Vet and Nyuki, were not used in further experiments for this project. These sequences allowed us to confirm or determine promoter haplotypes present in each individual.

To prepare for pyrosequencing, we optimized one primer: GH26 and GH27. We found the best reaction conditions for PCR to be two minutes at 98C followed by 30 cycles of 30 seconds at 98C, 45 seconds at 53.2C, and 45 seconds at 72C, followed by 5 minutes at 72C and pausing at 4C. Our optimal annealing temperature, therefore, was 53.2C. The size of the PCR's amplicon, intended to be 242 bp, was confirmed using gel electrophoresis.

Sanger sequencing confirmed or identified the sequence of a segment within exon 2 using cDNA of 21 individuals. SNPs within this region were used to confirm or determine the haplotype(s) present in each individual.

Next steps

SNPs identified within exon 2 using Sanger sequencing will be used to design assays to quantify the relative abundance of each haplotype present in an individual. Quantitative pyrosequencing will be used to find the amount of coding transcript product of the second exon of HLA-DQA1. We can target the second exon and amplify the region using PCR with primer GH26 and GH27. Quantitative pyrosequencing will be used to determine the abundance of cDNA containing each SNP on exon 2, indicative of a particular haplotype. From this we will compare the amounts of RNA transcribed from each promoter haplotype and determine whether promoter alleles have functional differences.

Discussion

Results of this experiment may provide explanations for maintained trans-species polymorphism observed in HLA-DQA1's promoter region in primates. If haplogroups appear to have no effect on the amount of HLA-DQA1 transcript produced, we may interpret these results to suggest that polymorphism has been maintained via neutral selection. This region's high rate of natural mutation (1/2.2bp) may depict genetic variations that are not evolutionarily significant (Loisel et al. 2006). Alternatively, if haplogroups appear to have an effect on the amount of HLA-DQA1 transcript produced, it may suggest that genetic diversity was maintained via balancing selection due to these functional effects. Overdominant selection, in which heterozygotes are actively selected, or frequency dependent selection, in which rare alleles are advantageous and thus increase genetic diversity, are types of balancing selection that may have contributed to this region's maintained diversity. By understanding the cause of this unusual evolutionary find, we may better appreciate the function and evolution of the cis-regulatory region.

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1 Each of the 10 individuals included in this process were sequenced using each of the forward primers separately with reverse primer, which yielded a total of 20 total samples.

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Adam's Baculum: The Loss of the *Os Penis* in *Homo* and Other Primate Genera

Misha Solomon, Columbia University

Introduction

The Judeo-Christian biblical narrative of human origins explains that Eve, the first woman, was created from a bone of Adam, the first man. Although traditional translations refer to this bone as Adam's rib, males and females in fact have an equal number of ribs. Gilbert and Zevit (2001) argue that this narrative is also an explanatory myth for the lack of baculum, or os penis (penis bone), in human males. The authors claim that the Hebrew tzela could be translated to mean any structural support bone and that, since the penis is more associated with generation than the ribcage, the baculum is the best candidate for Eve's generative bone. Whether or not it was required for the genesis of half of the human species, the loss of the baculum in humans is a distinctive trait. Among catarrhines, Homo is the only genus without a baculum; among primates, it is accompanied only by Ateles, Brachyteles, Lagothrix, and Tarsius (Ankel-Simons, 2007; Dixson, 1987; Hobday, 2000). This paper will explore the function of the baculum in mammals, with more specific reference to members of the primate order. After an examination of the baculum, the paper will consider the absence of the baculum in certain primate genera and attempt to draw on behavioral traits to explain this shared anatomical anomaly among phylogenetically distant primates.

Baculum Morphology & Aberrational Ossification

The baculum, also known as os penis, os priapi, or os glandis, is present in a number of mammalian orders (Patterson and Thaeler, 1982). The baculum is a bone within the penis; its specific function remains controversial, but it is clearly a supporting rod that aids in maintaining erection of the penis and possibly in initiating the opening of the vaginal orifice (Long and Frank, 1968). This bone varies in size across and within the mammalian orders in which it is present (Ramm, 2007). The prosimian baculum is relatively large and may be forked at the tip (Ankel-Simons, 2007). The baculum is formed from the ossification of the distal region of the corpora cavernosa followed by an anterior extension into the glans penis (Dixson and Anderson, 2001), and enlarges during puberty as its development is affected by androgens (Dixson and Anderson). The baculum is considered a primitive character in the primate order because it is found in most species; the loss of the baculum in certain primate genera may therefore be a homoplastic trait.

In the absence of a baculum, the penis lacks a supporting bony rod. In species where this is the case, a strong, central distal ligament strengthens the glans (Dixson, 2012). Reports of human penile ossification, specifically of the distal septum of the corpora cavernosa, although rare, exist in the literature (Dixson; Sarma and Weilbaecher, 1990). Although the bone that develops from this ossification resembles the baculum, this aberrational osseous tissue is not a phylogenetic vestige of the ancestral primate baculum (Sarma and Weilbaecher). Unlike the baculum in other primates, this rare ossification interferes with the well-developed erectile tissue present in the human penis, leading to penile curvature and preventing successful copulation (Sarma and Weilbaecher). Furthermore, the resulting bone is larger than the baculum found in other apes and is acquired after puberty, whereas other primates' baculum develops earlier and expands during puberty (Sarma and Weilbaecher). Therefore, human penile ossification is a metaplastic process, often the result of a traumatic injury or a venereal or metabolic disease (Sarma and Weilbaecher).

Bacular Variability

There is considerable variation in baculum length across the primate order. Dixson (2012 p.345) refers to the baculum as "the most diverse of all bones in its morphology." This variation exists in terms of relative baculum length in relation to body size. An adult Gorilla, weighing 136 kg, has a baculum of 11 mm in length, whereas an adult mandrill (Mandrillus sphinx), weighing 27 kg, has a baculum of 23 mm, and a potto (Perodictius potto), which weighs only 1 kg, has a baculum of 21 mm (Ankel-Simons, 2007). These variations are found between species of equivalent size as well; a small marmoset or tamarin and a large galago each weigh 300 g, but callitrichid bacula measure 1.5 to 3.5 mm and prosimian bacula often measure as long as 16.7 mm (Dixson). There is a general trend toward bacular reduction in hominoids as compared to other catarrhines, namely cercopithecoids (Dixson). Within superfamily Hominoidea, smaller bacula are found in African apes (6.0 to 12.5 mm) than in the Asian apes (14.6 to 15.0 mm) (Dixson). Platyrrhines also have reduced bacula-to-body-size ratios compared to other primates (Dixson). Thus, the closest relatives of the majority of primates lacking bacula (the New World genera and Homo) show a reduction in bacular length. The taxonomic classification of tarsiers, which also lack bacula, is notably complex and controversial, making their bacular loss phylogenetically nebulous.

The predominant hypothesis for bacular variation relates to intromission patterns (Dixson, 1987; Dixson, 2012; Dixson and Anderson, 2001; Patterson and Thaeler, 1982; Ramm, 2007). Dixson (2012) organized the copulatory behavior of 34 primate species into three categories of intromission: single prolonged intromission, single brief intromission, and multiple brief intromission. Species with single prolonged intromission copulatory patterns have relatively longer bacula than species

that fall into either of the other two categories (Dixson, 2012). The function of the baculum in prolonged intromission is related to its role as a supporting rod for the glans (Patterson and Thaeler). Furthermore, the bone may help in the act of intromission, by imparting additional stiffness to the glans and aiding in entering the vaginal orifice (Long and Frank, 1968). It should be noted that Larivière and Ferguson (2002), in their study of the mammalian baculum, did not find any correlations between bacular length and sexual behavior, and therefore rejected the notion that elongated bacula aid in prolonged intromission. Their study, however, focused on North American carnivores, not primates, and, as argued by Dixson (2012) in his rejection of their criticism, used a small and inadequate data set.

A number of other hypotheses for bacular variability exist in the literature. Patterson and Thaeler (1982) argue that the baculum functions in reproductive isolation of distinct species, a modification of the lock-and-key hypothesis in which a feature of a species' penile morphology allows males of that species to be the only males capable of successful copulation with females of the species, due to some feature of their vaginal morphology, thus preventing hybridization (Edwards, 1993). This hypothesis relates to facilitating a neuroendocrine response necessary for fertilization (Patterson and Thaeler). A different functional view presents the baculum as a device for facilitating sperm transport within a female's reproductive tract (Dixson, 2012). In primates with an elongated baculum, the tip of the bone emerges from the distal pole of the glans with the urinary meatus on its perineal surface, thereby bringing the os penis into contact with the os cervix during copulation (Dixson, 2012). This intracoital contact may assist in the transfer of semen from the urethral opening into the cervical canal (Dixson, 2012). All of these hypotheses point to facilitated or enabled copulation and fertilization as the source of bacular variability.

Copulatory Patterns in Genera Lacking Bacula

Although prolonged intromission appears to correlate with the possession of an elongated baculum, this copulatory pattern is found in Ateles, Lagothrix, and Homo, three genera altogether lacking in bacula (Dixson, 2012). Assessing contemporary human copulatory behavior in comparison with nonhuman primate copulatory behavior is difficult as underlying human adaptations are heavily obscured by cultural factors (Martin, 2007). There has likely been a reduction in intromission times from ancestral primates to extant apes (Martin). Martin theorizes that a further reduction may have occurred in the course of human evolution. Therefore, although Dixson points to the existence of prolonged intromission in Homo, such broad copulatory patterns cannot be identified in the genus due to the intervention of cultural practices that influence sexual practices.(Martin).

Although patterns of prolonged intromission have been observed in Ateles and Lagothrix, their copulatory behavior appears distinct in comparison with related species (Campbell, 2006; Eisenberg, 1973). The three New World genera lacking in bacula all belong to the subfamily Atelinae. Eisenberg's account of ateline copulation makes reference to their unique copulatory posture. Although Campbell does report prolonged intromission in black-handed spider monkeys (Ateles geoffroyi), the author observed that intromission often appeared difficult to achieve. In light of the baculum's potential function in facilitating prolonged intromission and enabling easier penetration of the vaginal orifice (Dixson, 2012; Long and Frank, 1968), these species' copulatory adaptations and difficulties may be related to their distinctive lack of bacula. Whereas human copulatory behavior is obscured by cultural practice, it is possible that ateline copulatory behavior is obscured by post-adaptive changes in copulatory strategy (Martin, 2007). In other words, the present-day copulatory pattern of prolonged intromission in the atelines likely developed after the baculum was lost (Dixson, 1987). This may result in the alternative copulatory behavior seen in these species, as well the development of vascular mechanisms to maintain sufficient rigidity during erection (Dixson, 1987).

Unlike the atelines, whose copulatory pattern of prolonged intromission, associated with an elongated baculum in other primates, makes their lack of a baculum all the more distinctive, tarsiers' pattern of copulation is more in line with species with reduced bacula (Dixson, 2012). Tarsiers, specifically Tarsius bancanus, show a copulatory pattern of single brief intromission (Dixson). Therefore, the functions of the baculum associated with prolonged intromission are not relevant to tarsier copulatory behavior. Although the primate genera lacking in a baculum do not engage in the same copulatory patterns, they each have features of their copulatory practices that may be correlated with the absence of a baculum.

Hypotheses for Bacular Deficiency

The copulatory patterns found in primates lacking a baculum, although related to bacular absence, are not sufficient to explain why these genera "grew out" of their bacula. Many of the existing works on the absence of a baculum in these genera suggest that this bacular disappearance is the continuation of the reduced bacular length seen in related species (Dixson, 1987; Dixson, 2012; Martin, 2007).

Of the primates lacking in a baculum, there is the least information available on the elusive, unusual, and confounding tarsier, the only nocturnal primate among those discussed (Ankel-Simons, 2007). A connection can be made, however, between the tarsiers' sexual behavior and their bacular deficiency. As Dixson (2012) reports, tarsiers show a pattern of brief intromission with rapid pelvic thrusts. This kind of vigorous copulation has been associated with fractures of the baculum in species such as the European otter (Lutra lutra); although these reports do not come from primates, the species in which this behavior has been reported are similar in that they have bacula (Dixson). Baculum fractures have been known to be fatal due to resulting complications (Bartosiewicz, 2000). It can therefore be argued that the rapid and vigorous thrusting of tarsiers made it disadvantageous and detrimental to possess a baculum; in this model, the trait of lacking a baculum would be positively selected since those individuals without the bone would not succumb to baculum-related injuries and would be more likely to survive and reproduce.

Although the relationship between the general penile morphology of the atelines and their nearly subfamily-wide lack of a baculum is unclear, a potentially related process of baculum

disappearance has occurred in the pithecids, a different family of New World monkeys (Hershkovitz, 1993). Individuals in certain species of Cacajao and Chiropotes have been reported to be lacking in a baculum (Dixson, 2012; Hershkovitz). The bacula of this family are in the process of disappearing in response to differentiation of a unique system of sperm delivery (Hershkovitz). To best deliver sperm directly into the uterus, the penes of these species have seen a reduction or elimination of the baculum, a curvature of the shaft, and a bluntly pointed labile glans (Hershkovitz). Hershkovitz states that a baculum-stiffened glans is less successful than a boneless glans at effecting embrace between the urethral meatus and the cervix. Like pithecine penes, ateline penes also have a blunt distal end and often have penile spines (Dixson). A potential explanation for bacular deficiency is therefore the facilitation of sperm transport to the cervix; successful embrace of the meatus and the cervix allows for semen to be ejaculated directly through the cervix and into the uterus (Hershkovitz).

The human penis is different from the penis of tarsiers or of New World monkeys, and so alternate explanations of its bacular deficiency have been proposed. The human penis is distinctive due not only to its lack of baculum, but also to its size. It is among the largest, in terms of relative proportions, in the primate order; only the chimpanzee penis is of similar or greater length (Hobday, 2000). The human penis may have evolved as a unique semen displacement device; both its size and glans/coronal ridge morphology have been correlated with sperm competition, specifically the displacement of a rival male's semen (Gallup et al., 2003). As ancestral Homo became bipedal, the female reproductive passage moved inwards in the body cavity and became extended, posing a problem for a small-penised, rear-mounting male (Hobday). Due to this extended passage, selection would have favored a longer penis; such an organ would have also had a large supporting baculum, making it increasingly heavy, uncomfortable, and vulnerable, especially exposed as it was in the bipedal position (Hobday). A larger body cavity, unencumbered by a baculum, would have been required for comfortable and protective withdrawal of the penis (Hobday). Furthermore, the loss of the baculum would have allowed for a more vascularized organ in which maximum elongation during erection can be achieved without the baculum taking up space (Hobday). Baculum reduction or deficiency is also associated with a symmetrical glans, as seen in humans, tarsiers, and pithecines; symmetry may provide the necessary rigidity provided by the baculum in an asymmetrical glans, and these baculum-deficient penes are more efficient pistons, better able to remove sperm plugs (Hershkovitz, 1993; Hobday).

Dawkins (2006) presents an alternative, uniquely human hypothesis for bacular deficiency. Without a baculum, a human penile erection is accomplished strictly by blood pressure, by a hydraulic pumping system without a supporting or stiffening rod (Dawkins). Erection failure, or erectile dysfunction, can be an early warning sign of diabetes and certain neurological diseases or can result from psychological factors, including depression, anxiety, and stress (Dawkins). Dawkins suggests that, as females' diagnostic skills were refined, they would have been able to glean clues about a male's health and ability to cope with stress from the firmness and endurance of his erection. A baculum would prevent a useful diagnosis as it provides as additional aid to tumescence, and so pressure from females, who would choose to mate with males who clearly lacked a baculum, selected for males with a boneless penis (Dawkins). Females would have been able to assess whether a male had a baculum based on the difference between his flaccid and erect penis (Dawkins).

Conclusion

Perhaps Gilbert and Zevit's (2001) alternate reading of Genesis presents a situation in which, subsequent to her very creation, Eve was able to assess Adam's health through his ability to achieve or maintain an erection. Regardless of the biblical origin story, the absence of a baculum in the human penis remains an evolutionary conundrum. Although hypotheses for the loss of the baculum in tarsiers, certain New World monkeys, and humans have been proposed, and correlations between these species' copulatory behavior and their boneless penes have been suggested, researchers have not yet agreed on one explanation. It appears that the similarities between the penis of three groups begin and end with the lack of a baculum; diverse evolutionary correlates seem to explain this loss in each group. Further anatomical and behavioral research, both in relation to these species and their ancestors with bacula, is needed to gain a better understanding of this distinctive and potentially disparate deficiency.

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Interobserver Reliability of Entheseal Changes Using the Villotte (2006) Method at Newton Plantation

Rachel Perash and Lisa Harris, University of Indianapolis

Abstract

Entheseal changes occur at attachment sites on bones as a response to muscle, tendon, and ligament loading; they are widely used to explore habitual activities and mechanical stresses caused during life. In this study we examine interobserver reliability of entheses scoring in an osteological collection from Barbados (Newton Plantation) following methods in Villotte (2006). Fifteen markers from the upper and lower skeleton were examined and data were collected from 32 volunteers placed into two groups, experienced and inexperienced. The hypothesis was that experienced and inexperienced scores would differ significantly. A one-way ANOVA was performed to examine overall percent and frequency of agreement between observers based on each location and the scores recorded for these locations (ordinal scores were treated as interval data [e.g., Agresti, 1989]). Our results indicate that scoring is consistent at most markers for both inexperienced and experienced observers, bolstering confidence in the current standard entheses scoring protocol.

Introduction

Muscle, tendon, and ligament attachment sites reflect mechanical stress experienced during life (Hawkey and Merbs 1995) and are used to evaluate probable habitual activities (Lieverse 2009). Several methods of scoring entheseal changes have been proposed, the most common of which is Hawkey and Merbs (1995). Villotte (2006) is a more recent method that focuses on anatomical variation, unlike previous methods, making it more functional and effective. We examine interobserver reliability in scoring entheseal changes by applying the Villotte (2006) method (Figure 1). An important factor, assessing the reliability and consistency of scoring methods, was examined in this study. It is important to have low interobserver error rates in entheseal scoring methods because entheseal changes are employed to differentiate high and low mechanical stress.

Data were collected by thirty individuals varying from low to high experience with osteology and entheseal changes; education levels ranged from undergraduate to PhD. Inexperienced and experienced observers scored fifteen markers that were selected from an osteological collection from Barbados (Newton Plantation). The long bones of the upper and lower body markers were scored, along with markers from the clavicle and os coxae. Observers scored markers according to Villotte (2006); they also self-ranked their confidence in each score on a zero to five scale. We hypothesized that the results would show inexperienced and experienced observer groups to be overall similar, yet the groups would score differently from each other.

Figure 1: Villotte (2006) Scoring Method

0	No irregularity present
1	Presence of slight irregular enthesophyte at outer edge and/or foramina, cystic change, calcification deposition, or bony production present
2	Presence of severe irregular enthesophyte at outer edge and/or foramina, cystic change, calcification deposition, or bony production

Materials and Methods

Data collection was conducted between 2010 and 2012 in two independent sessions. The first entheseal change scoring workshop was held at William Carey University under the direction of the authors by ten observers. The markers (n=15) came from several different, single specimens and were observed on the humeri, radii, ulnae, os coxae, femurs, tibias, and clavicle.

A second scoring session was conducted in 2011, by twenty-two participants, bringing the total number of observers to thirty-two. The same specimens were used to allow for comparison between the groups as well as for the authors to perform further analyses on the data collected in the future.

All observers were given identical forms that requested major, year in school, experience levels of human osteology, and experience levels with entheseal changes. This information was used to assess the differences between the groups and to allow the level of experience of the observers to be determined.

Entheses Literature

For the purpose of this study, we focused on using the Villotte (2006) methodology. The method established by Villotte is different from previous methods in that it utilizes current medical knowledge of the anatomy and pathologies of enthesopathies and claims <10% interobserver error (Villotte 2006 and Villotte et. al 2010). Villotte explains that "the main limitation of existing methods resides in the choice made by the author to consider the evolution of a particular feature of remodeling as an indicator of the evolution of intensity lesion without using existing anatomical descriptions" (Villotte 2006); therefore the 2006 method closely follows current medical knowledge of the health of an entheses. It is broken down into categorizing an insertion site on two different qualifications: fibrocartilaginous and fibrous. Within the two qualifications there are four different groups to categorize into based on similar bone remodeling stages.

This method is particularly advantageous because it focuses on

current medical knowledge of enthesopathies. This is an advantage because it allows the observer to score based on the current knowledge of how the nature of the bone has an effect on an entheseal change. Hawkey and Merbs (1995) propose a method that is based solely on "standardization of the gross morphological expressions" (Hawkey and Merbs 1995). Although useful for inexperienced and experienced observers alike because of the visual reference system, the method established by Hawkey and Merbs does not account for the nature of the bone when scoring an entheseal change, which the authors of this study found to be of importance. Many previous studies (Weiss 2007, Lieverse et al. 2009, Steen et al. 1998) have found Hawkey and Merbs (1995) to be applicable, however the authors felt Villotte (2006) would be better employed for this study of interobserver reliability.

Results

Results showed location thirteen was the only location with any significance between observer scores. A Bonferroni test was then performed to further examine the differences between experience levels. The biggest difference was between observers with no experience (level zero) and those with limited original data collection (level two). The authors have come to the conclusion that this result was spurious and likely due to chance. Results indicate that experience levels do not affect confidence score.

Discussion

Results showed location thirteen was the only location with any significance between observer scores, however the authors came to the conclusion that this result was most likely due to chance. Location thirteen was on the posterior, proximal end of the right femur and represented the gluteus medius insertion. As seen in Figure 2, severe taphonomic damage is present next to the marker. Observers could have easily been confused as to whether or not the missing bone was a part of the entheseal change. While this does not account for the differences between the experience levels zero and two, it could explain why the location had a range of scores.

The authors decided to treat the observers' scores as continuous rather than discrete in order to achieve more accurate results. While the data would normally be seen as ordinal the authors were looking at what score the observers listed, not rank order. Therefore the data were treated as interval-ratio data (see Agresti, 2010).

Previous research (Shuler 2005) has documented poor health among the individuals at Newton. While any infection on the remains used for the interobserver workshops was viewed by the authors as unobtrusive, observers could have found this to be false. The sites were specifically chosen for easy identification of whether or not entheseal changes were present due to different observer experience levels.

While any infection present on the bones did not coincide with the entheseal change sites, observers could have viewed pathology with uncertainty towards scoring or as part of the entheseal change itself. This decrease in confidence levels could have affected how the observers scored.

Several observers (n=10) had participated in a similar workshop in 2010 that utilized the same skeletal remains. These observers had previous experience with the osteological collection, which could have affected their entheseal change scoring.

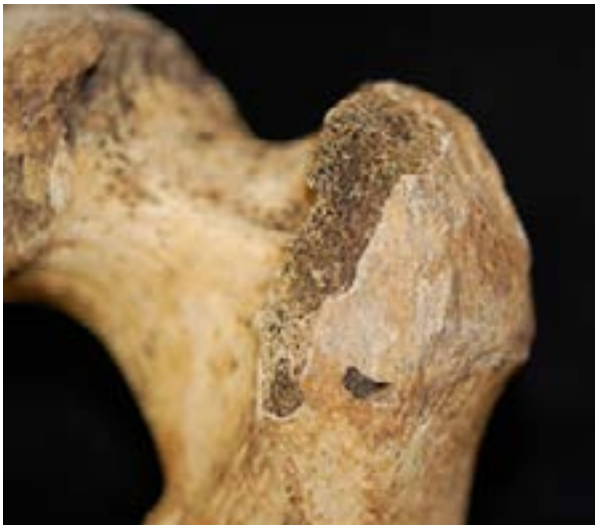

Figure 2: Location 13- Gluteus medius insertion on right femur

Conclusion

Our original hypothesis of experience levels having an effect on scoring patterns was not supported. There was no significant difference between scores of varying experience levels, except between those with no experience (level zero) and those with limited original data collection (level two), however, this difference may be the result of chance. This study shows that experience level does not prove to be a significant factor when using the Villotte (2006) method. This is meaningful for current anthropological studies because it demonstrates that having previous familiarity and understanding of the Villotte (2006) scoring methods is not an essential qualification for documenting entheseal changes. Finally, some people in this study collected data more than once. Because of this a planned follow-up study involves intraobserver reliability.

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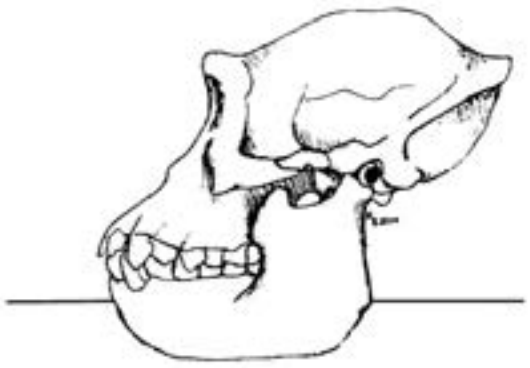
You Are What You Eat (Or Are You?)

An Exploration of the Effects of Dietary Habits on Mandibular and Dental Morphology of the Genus *Gorilla*

Rachel Bell, Columbia University



Abstract

This paper explores, in detail, the various morphological differences in the mandible and dentition between Gorilla gorilla and Gorilla beringei, including their respective subspecies. I explore both landmark and current research on the morphological aspects of the Gorilla mandible and dentition and Gorilla diets to ultimately synthesize conclusions on the relationships between them. By looking at the dietary habits of the various subspecies, I then make connections between their morphology and diet. Due to a lack of seminal and recent research on Gorilla gorilla delhi, I do not include that subspecies in my examination. Ultimately, I realize that there are many other factors besides dietary habits influencing the dental and mandibular morphology of the genus Gorilla. Recent research also shows that fallback foods may affect dietary adaptations more so than those foods normally available in Gorilla environments. Finally, I suggest that altering dietary categories¬—grouping food by their mechanical properties (e.g. toughness and texture), as opposed to type (e.g. fruit, leaves)—may create a more accurate picture of how diet is truly reflected in morphology.

Introduction

As Swindler (1994) succinctly describes, gorillas are virtually vegetarian. Their dietary habits are for the most part folivorous and frugivorous, though diets vary within these categories on the level of species, sub species, and even population (Swindler 1994). Although two species of Gorilla are commonly defined (the eastern gorilla and the western gorilla), it would seem that changes in diet composition are more affected by altitude than by species differences. Gorilla diets also vary greatly with the seasons, and most gorillas exhibit seasonal frugivory, although this occurs more frequently in low altitude populations (Carroll et al. 2001). It was confirmed by Carroll et al. (2001) that the diets of both eastern and western lowland gorillas are generally more varied than those populations at higher altitudes, such as the Virunga mountain gorilla population. Western lowland gorillas ingest the largest proportions of fruit of any other gorilla subspecies and favor softer, fleshy fruits, while also consuming a variety of leaves from trees and other woody plants (Carroll et al. 2001). According to Tutin and Fernandez (1985), 67% of the dietary intake of observed western lowland gorillas consisted of fruits.

By contrast, Robbins and McNeilage (2003) found that the Virunga population of Gorilla beringei beringei feeds primarily on herbaceous vegetation throughout the year, with little fruit. Fewer fruiting tree species grow in these higher altitudes, and the gorilla population is a primarily terrestrial feeder with 96.2% of its feeding done on the ground (Watts 1984). Beyond herbaceous terrestrial plant species of little variety—but even distribution and abundance—they have also been seen eating bark, flowers, roots, and insect cocoons (Watts 1984). The Bwindi population of Gorilla b. beringei varies in that it has frugivorous dietary habits ranging somewhere between that of the western lowland and Grauer's gorilla and the Virunga population of mountain gorilla (Watts 1984). It would be sensible that these variations in diet, if they have been consistently so for a sufficient period of time, would have adaptive implications within the species and subspecies of Gorilla.

As Watts (1984) concludes, "dietary specialization among primates increases as the degree of folivory increases." Pilbrow (2010) suggests that West Africa was the center from which gorilla species and subspecies derived, which further supports the idea that mountain gorillas could be among the most diet-specialized and derived subspecies of Gorilla. With these observations in mind, it seems probable that dietary specialization would be demonstrated in derived dental and mandibular characteristics. This is certainly not an unexplored idea, but one that has been proposed and challenged for many decades. Kay (1975) postulated that primates of the same body size but of different dietary habits will exhibit distinct differences in the crushing and grinding surfaces of their teeth as well as the length of their shearing blades. While in reality such adaptations may not be as clearly formulaic as Kay proposed, there is still a fascinating relationship to examine.

The Hominids can be defined through their dental and

mandibular morphology, though in particular I will focus on the variations of these characteristics relating to the gorilla. Like most other Catarrhines, gorillas exhibit the dental formula 2.1.2.3 on both the upper and lower jaw (Swindler 1994). As Swindler (1994) observes, Hominids generally have broad incisors, the central ones having straight incisal borders and the lateral ones having sloping borders. In particular, gorillas have a tendency towards enlarged central incisors, and canines that are outspread buccally in comparison to the other great apes (Swindler 1984). In examining the upper premolar, which is bicuspid, Swindler (1984) also found that gorillas have wider and deeper mesiodistal developmental grooves between the paracone and protocone cusps than other Hominids. The lower P3 may sometimes have a metaconid and a protoconid, but usually has a single cusp, the protoconid, which is compressed (Swindler 1984). The lower P4, however, most often has the two aforementioned metaconid and protoconid cusps (Swindler 1984). Molars of the upper jaw have four cusps, and although the hypocone and metacone are usually reduced in size compared to the protocone and paracone, the gorillas have the least amount of hypocone reduction of all the Hominids (Swindler 1984). Of the five cusps of the lower molars, those on the lingual side are particularly pointed and high compared to the buccal cusps in gorillas (Swindler 1984). From these data, it can be synthesized that, in general, gorilla teeth trend toward overall enlargement as opposed to reduction, as well as heightening of the cusps.

Just from observation, Gorilla morphology follows and accentuates the anthropoid trends of the mandible. The condyloid and coronoid processes of the mandible sit high above the occlusal plane on the end of the far-ascending ramus. According to Taylor (2002), gorillas have rami and wider mandibular condyles that are relatively higher above the occlusal plane of the mandible even in comparison to the chimpanzee, all trends resulting from the need for a more powerful jaw. As Taylor (2002) states, the mandible of the gorilla has a larger area for the masseter when compared to other Hominids. The need for stronger chewing in gorillas has increased the thickness and size of the masticatory muscles, and since the masseter, temporalis, lateral pterygoid and medial pterygoid all insert at the mandible, their amplification was likely partially responsible for these aforementioned changes. With these general morphological traits in mind, it will now be possible to explore the variations of these traits within the genus Gorilla as potential adaptations to dietary habits.

Dentition

In examining the dental variation within Gorilla, differences can be found between species and subspecies. Uchida (1998) uses slightly different taxonomic categorizations as she places Gorilla beringei beringei and Gorilla beringei grauri into the species of Gorilla gorilla, which has since been challenged. Her examination of variation across these three subspecies reveals a possible correlation between morphological variation and other variable environmental factors, including diet. Uchida (1998) also makes the clarification that these variations should be looked at as population-specific and not species-specific, given that each population is subjected to a different set of environmental factors, which in the long term can shape morphological adaptations.

To begin, the incisors of the gorilla species and subspecies hold variability that may be vital for distinguishing their dietary tendencies. Through measuring the upper central incisor mesiodistal length, Uchida (1998) was able to find that G. g. gorilla, at 14mm, had the widest incisors relative to molar row length when compared to G. b. grauri and G. b. beringei. In anthropoids, primarily frugivorous primates demonstrate a trend towards larger and broader incisors, which correlates to G. g. gorilla's high rate of seasonal frugivory (Uchida 1998). A study by Deane (2009) on the relationship between hominoid incisor curvature and diet supports this conclusion by showing a positive correlation between mesiodistal incisor length and increased frugivory across the Hominoid family. Curved, broader incisors would be less apt for shearing fibrous plant matter, which is consistent with his findings. Additionally, Deane's findings suggest that incisor crown curvature is correlated with frugivory in Hominoids, and that while the incisors of primarily frugivores are mesiodistally wider than those of primarily folivores, the incisor crowns of folivores are labiolingually broader (Deane 2009). Even between the Bwindi and Virunga populations of G. b. beringei, Butynski et al. (1996) finds the significantly more frugivorous Bwindi population to have a wider incisor row and a larger incisor diameter than the mainly folivorous Virunga population relative to body size.

Conversely, canines reflect other attributes of variation, such as sexual dimorphism, that have an uncertain relationship with dietary habits in the genus Gorilla. Uchida (1998) reports that G. b. beringei exhibited the largest amount of sexual dimorphism through their canines, and had larger canines on average when compared to G. b. grauri and G. g. gorilla. Conversely, G. g. gorilla had the least sexual dimorphism in canine size (Uchida 1998). It has been theorized that higher sexual dimorphism in canines could be related to low competition between females for food sources and other needs (Uchida 1998). Thus, terrestrial vegetation feeders would experience less inter-female competition for food, which correlates nicely with Uchida's findings. However, the variation in body size between the species and subspecies of the gorilla, as well as other factors influencing sexual dimorphism, could play a large role in canine size, discrediting Uchida's suggested correlation.

The premolar and molar dentition within Gorilla perhaps provides the most information suggesting dietary adaptations and trends. Taylor (2002) differentiates Gorilla beringei beringei postcanine teeth from G. g. gorilla postcanines as being more defined by sharper cusps and transverse ridges, as well as overall higher crowns. Because of the shearing, cutting, and grinding abilities needed for plant matter, such adaptations would prove advantageous. In all measurements completed by Uchida (1998) on postcanine teeth, G. b. grauri's were the overall largest, followed by G. b. beringei and lastly G. g. gorilla. Buccolingual enlargement of the upper molars was present in G. b. beringei while G. b. grauri shows both buccolingual and mesiodistal enlargement (Uchida 1998). Although one might expect G. b. beringei's postcanines to be the largest overall due to the strain of their diet, other factors such as the altitudinal variance of

the G. b. grauri populations may contribute. When comparing eastern and western gorillas as a whole, eastern gorillas have a tendency towards long, distally splayed molars (Pilbrow 2010). Eastern gorillas also have larger distal molar cusps than western gorillas (Uchida 1984). This inclination in distally oriented and elongated dentition may aid in the continual grinding process of eating particularly fibrous foods. For example, if one were ingesting a stalk of celery it might be more beneficial to have dentition of increasingly efficient processing abilities as the plant matter moves distally through the mouth. For softer foods, such adaptations may be unnecessary.

Additionally, even between the Bwindi and Virunga populations of G. b. beringei, Butynski et al. (1996) notes that the Bwindi population has larger molars but shorter premolar-molar row lengths. The larger molars could, like the broader incisors, be more beneficial for processing meatier foods or for crushing large, hard fruits. Similarly, Butynski et al. (1996) considers the theory that a longer premolar-molar row in the Virunga population could be an adaptation for ingesting bamboo.

Overall, it is vital to note that the variations in dental morphology explored in this section cannot be fully interpreted without additional understanding of mandibular variation, which relates greatly to changes in diet toughness and load.

Mandible

The mandible of the gorilla shows variation between both gorilla species and their subspecies that would suggest changes in jaw loads, and potentially in the mechanical properties of their diet (Taylor 2006). Mandibular morphology reveals the torsion, force, and strain of the jaw, and is thus an excellent reflection of the ingestion process. Understanding the points of stress in the mandible can also illuminate the adaptations in the gorilla jaw to alleviate tension. In Hominids the balancing side of the mandibular corpus undergoes parasagittal bending and dorsoventral shear stress that is caused by increased balancing-side muscle force in the jaw, but a deeper mandibular corpus lessens the stress inflicted by the muscles (Taylor 2002). As previously stated, the genus Gorilla exhibits a wide mandibular symphyses and corpora in comparison to other Hominids, suggesting an overall increase in the need for a more powerful, force resistant jaw that most likely undergoes long periods of masticating tougher foods (Taylor 2006). However, G. b. grauri was found to be proportionately similar to Pan troglodytes verus in one aspect of mandibular dimensions (Taylor 2006). Nonetheless, the general trend seems to increase within the largely folivorous G. b. beringei: the subspecies has the widest mandibular symphysis and corpus compared to jaw length out of all African Apes (Taylor 2006). Taylor (2006) also suggests that these wider mandibular adaptations may help lessen the stress of wishboning, or transverse lateral bending, within the mandible.

Hylander and Johnson's study (1994) examines lateral transverse bending, or wishboning, at the mandibular symphysis to see how mandibular musculature affects its occurrence. They found that the force of the balancing-side deep masseter muscle and the simultaneous relaxation of the superficial masseter muscles on both the balancing and working-sides were causing the tension at the end of the power stroke of mastication (Hylander and Johnson 1994). In such a case, the aforementioned mandibular adaptations of G. b. beringei might actually counteract the stress of the masseter muscles more so than G. g. gorilla or G. b. grauri. (Taylor 2002) suggests that wishboning stress may positively increase with body size, so some mandibular variation in response to wishboning found between G. g. gorilla and G. b. beringei may be largely influenced by body size differences. However, it is still quite feasible that, with such a high-strained fibrous diet of plant vegetation, more resistance to mandibular stress would be necessary to prevent higher rates of mandibular and dental wear.

In terms of the condyloid and coronoid processes of the ascending ramus, there are strong results on their potential link to frugivory or folivory. It has commonly been proposed that a higher mandibular condyle lengthens the moment part of the masseter and medial pterygoid, perhaps allowing these muscles to generate a more powerful moment (Taylor 2005). Additionally, the same proportionately high mandibular condyle length in relation to the occlusal plane of the mandible could allow for comparatively even load distribution in molars and premolars during mastication (Taylor 2005). These results make sense in light of G. b. beringei – as suspected, the mountain gorilla exhibits higher rami and condyles relative to the mandibular occlusal plane as well as relatively wider mandibular condyles when compared to G. b. grauri and G. g. gorilla (Taylor 2005). It would seem that these adaptations would be only beneficial in light of tougher loads and fibrous foods, such as plant matter, that require more muscular power to ingest.

While Taylor (2005) cites studies suggesting that shorter rami relative to the occlusal plane of the mandible would more efficiently allow wide jaw gapes by reducing the amount of muscle stretch required, such results still do not disprove the concept of a higher ascending ramus allowing for more powerful biting force, even if the bite itself is not as wide. A high ascending ramus and mandibular condyle provide more area for masseter muscle attachment, thus allowing for more masseter musculature and a stronger bite (Taylor 2005). Indeed, if one were to more closely examine these results, it would be quite sensible if primates requiring a wider, but not as powerful, jaw gape were those eating larger, fleshy fruits as opposed to strips of vegetation and leaves. Although it would be reasonable to assume that the Bwindi population of G. b. beringei's mandibular morphology is slightly less accentuated than that of the Virunga population, there is too little information on the subject to soundly make such an inference.

Even so, it is apparent that a wider, deeper symphysis and corpus, as well as a higher ramus with consequentially higher condyloid and coronoid processes, are related in some extent to dietary adaptations. The adaptations in the mandible are often clear responses to changes in masticatory stresses and loads. However, a clear problem in relating certain mandibular adaptations to one dietary habit or the other is the fact that texture and toughness can vary within these categories. Although it is difficult to clearly differentiate folivory from frugivory through mechanical properties, the aforementioned trends of mandibular morphology can still be examined through

these categories with some caution.

Conclusion

Diet has certainly affected Gorilla morphology in ways unexamined through this paper: digestive tract, posture, cranial dimensions, and musculature are just a few areas. However, these results can confirm that the dental and mandibular morphology of the gorilla has species and subspecies-specific traits influenced by their particular environmental needs. Because fruits and herbaceous vegetation vary in consistency within their respective categories, it is difficult to say that these adaptations come solely from the type of foods being regularly consumed. A recent study by Vogel et al. (2008), which explores the function of molar enamel thickness in hominoids, suggests instead that dental morphology may be most influenced by fallback foods. Local stressors, geographic variability, competing species, and social behavior also have the potential to influence the mandibular and dental morphology of gorilla species. Additionally, perhaps we should continue to shift our examination of diets to include categorization of the food's overall mechanical properties. Although most gorilla species may show a preference for fruits and other softer foods when they are available, Vogel et al. (2008) find in their comparison of molar thickness and dietary behavior between Pan troglodytes schweinfurthii and Pongo pygmaeus wurmbii that it is the toughness of the fallback foods—and not the qualities of the most preferred or commonly eaten food sources—that place large selective pressures on dental morphology. By further examining the mechanical properties of fallback foods and other commonly eaten foods, we may find that there are qualities within them that shape anatomical adaptations more than the category of food being ingested.

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