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Hypothesis

Coevolution of Hominin Social Abilities and Infant Vision have Raised the Specter of Autism and Prevented Delay of Presbyopia

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Abstract

It is proposed that early, intense, visual communication between human infants and their primary caretakers, particularly mother, has been a crucial first step in the development of the complex, sophisticated social abilities that increasingly distinguished evolving hominins from other hominoids. This suggests in turn that aberrations in the development of infant vision might negatively impact the development of social abilities, potentially landing at risk children on the autism spectrum. It is further proposed that genes accumulated by selection because of their positive contribution to the visual abilities of infants often will have downstream negative pleiotropic effects that potentially contribute to presbyopia's relatively early manifestation, well ahead of senescent decline of most other adaptations.

Available evidence bearing on these twin hypotheses is reviewed, and additional tests are proposed. If they prove to be supportive, new approaches to prevent both autism and presbyopia should follow.

Keywords: Antagonistic pleiotropy; Asynchronous aging; Autism; Infant vision; Presbyopia

Introduction

Two linked hypotheses will be presented and discussed.

1. Complex human social abilities and behaviors begin with, and require for optimal development, infants being able to focus clearly on their primary caretaker's (usually mother) eyes and other facial expressions soon after birth. Deficits in infant near-vision therefore might alter or otherwise compromise the development of social abilities and propensities, leading in some instances to social impairments that fall along the autism spectrum.
2. Strong selection over the course of hominin evolution for the ability to see and interpret mother's eyes and facial expressions as a neonate, as well as later in infancy, is expected to have accumulated numerous antagonistic pleiotropic genes (AP genes) with effects that enhance these early abilities, accompanied by later-occurring negative effects that impair vision, perhaps accounting for early manifestation of the panhuman condition known as presbyopia-the blurring of near-vision that progresses rapidly during the 5th decade of life, well out of sync with other age-related declines in somatic function.

Six topics that provide a foundation for these hypotheses will be presented in turn, and then synthesized and discussed. They are the AP theory of the evolution of aging, asynchronous aging mediated by AP genes, the timing and characteristics of hominin divergence from other hominoids, presbyopia, infant vision and its relationship to unique human eye and facial characteristics, and autism spectrum disorder.

The antagonistic pleiotropic theory of aging

The AP theory of the evolution of aging (or senescence) has broad acceptance among evolutionary biologists due to its compelling logic, solid mathematical foundation, and increasingly strong empirical support [1-5]. The gist of the theory is that the force of natural selection eventually declines with chronological age in nearly all organisms, which results in early-appearing genetic effects being overweighted by selection relative to later effects. This in turn allows the accumulation and fixation of genes with effects that are beneficial early but detrimental (even highly so) later in the lifespan-so called AP genes.

Importantly, a growing number of genes with the above characteristics have been identified [4,5], and theoretical arguments gird claims for their near ubiquity as well [6-9]. Thus, a key point to keep in mind is that factors that strengthen or

weaken the force of selection at older ages influence the accumulation and expression of AP genes, and in turn the rate of senescent decline.

Asyncrhonous aging

The AP theory of the evolution of aging briefly reviewed immediately above predicts that adaptations generally will senesce in close synchrony with one another. This follows because developing and maintaining adaptations requires effort, and effort is fueled by resources that are limited (e.g., time and energy), implying that an important adaptation that routinely fails sooner than others should be allocated more effort, whereas one that routinely outlasts others should be allocated less effort.

However, the argument for synchrony, despite its underlying logic, should be considered a trend, not a rule without exceptions. Several examples of asynchronous senescent decline have been identified, and a formal model for the evolution of asynchronous aging recently has been presented; it finds that selection will favor the evolution of the most rapid rates of aging in those traits that are under the strongest selection at early ages because selection for these traits erodes the fastest [10]. This in turn can accommodate the accumulation of AP genes with outsized, relatively early appearing, downstream negative effects acting on the trait in question.

A brief history of the hominin divergence

Estimates of the timing of hominin divergence from other Hominidae are complicated by the occurrence of hybridization events, but it is clear that australopithecine hominins in our direct line of descent had evolved by at least 3 million years ago. They were the progenitors of the first members of the genus Homo, which evolved approximately 2 million years ago, and their descendants, anatomically modern *H. sapiens*, evolved about 300,000 years ago [11].

What drove our divergence from other apes? Although many hypotheses have been proposed, it now generally is conceded that social competition was the primary driver of the evolution of our most distinguishing features, including physical altriciality, cognitive precociality, increased reliance on learning, the use of sophisticated language, and vast changes in social organization, including involvement of fathers in childcare, and formation of large, extended kinship networks that cooperate to provide alloparental care and communal defense [12-16].

Of the foregoing 'distinguishing traits,' changes in social organization-particularly increasing infant/child altriciality, and increasing participation in the care of children by fathers and other genetic relatives such as grandparents-are expected to have worked in combination over the course of hominin evolution to allow individuals on the cusp of old age to be a bit more reproductively viable than their ancestors had been at similar ages. This would have delayed the decline in the force of natural, which in turn would have delayed the onset of age-related deterioration, thus leading eventually to a maximum potential lifespan double that of other apes, both living and extinct [17-19].

However, in light of the asynchronous model of aging described in the previous section, some of the traits comprising our phenotype are expected to have experienced less delay in senescent decline than others. The progressive inability to focus clearly on up-close objects, which begins in middle age, is a proposed example.

Presbyopia

Presbyopia is a panhuman trait that begins at around age 40, and results in blurry near-vision. It is attributable to the lens of the eye becoming increasingly rigid, which limits its ability to correctly focus light from near objects onto the retina [20]. This raises the question, why hasn't selection slowed the rate of decline in near-vision as much as it has for most other traits? To my knowledge, a compelling explanation has not yet been proposed, but drawing a comparison between chimpanzees and bonobos, our two closest nonhuman primate relatives, seems to be a propitious place to begin, because like us they also develop presbyopia at around age 40 [21,22]. This is an important fact because it suggests that presbyopia predates the divergence of hominins from the other hominoids. However, unlike us, chimpanzees, bonobos, and our most recent common ancestor rarely would have survived long enough to experience this particular decline in vision (i.e., most wild-living chimpanzees and bonobos die before reaching age 40, as did, presumably, our common ancestor), and those individuals that did survive to age 40 would have experienced presbyopia for no more than a few years, owing to having much shorter maximum potential lifespans. In stark contrast, a large proportion of humans, including extant hunter-gatherers, and thus presumably Pleistocene hunter-gatherers, have survived long enough to experience presbyopia for two or more decades [19].

The asynchronous aging model discussed earlier gives a potential explanation for selection's relative impotence in slowing this particular age-related decline. Namely, strong selection for infants to quickly and clearly focus on mother's eyes and facial expressions (see the next section for details) is expected to have resulted in especially rapid and significant decay in the force of selection acting on the visual system, thus potentially accounting for why hominins have continued to manifest presbyopia at about the same age that chimpanzees and bonobos do so.

Infant vision

Visual acuity in human neonates is best at a distance of about 8-12 inches, which matches the distance between a suckling infant's eyes and mother's face, particularly her eyes. Human neonates are highly attracted to faces, and to sharp contrasts, such as that at the intersection of the colored iris and white sclera of mother's eyes [23]. Humans are the only primate with white sclera [24]. Furthermore, humans of all ages are capable of a greater variety of facial expressions than other primates, and have relatively hairless faces (especially women), which gives neonates and older infants an especially clear view of these various expressions; and our distinct vermillion border that separates the lips from the face, which is absent in other primates, also lends clarity to facial ex-

pressions, particularly smiles and frowns [25]. Additional evidence of attunement to mother, independent of vision, is that newborns almost immediately are able recognize mother's voice and odor, and they exhibit a preference for them [26].

All of these distinctly human traits exhibit the hallmark of being adaptations, in so far as they exhibit functional design. Namely, they appear to facilitate social interaction between infant and mother, as well as other caretakers, such as father and grandparents, which in turn presumably facilitates social learning. Infants learn who to trust, and who to be fearful of, and they learn the subtleties of what mother is thinking and feeling, and how to respond to her, and even manipulate her (and eventually others), which are skills needed to thrive at older ages in a hyper-social species such as ours.

Autism spectrum disorder

Autism occurs along a spectrum that exhibits considerable variation in its presentation, but difficulty with social interaction is the core characteristic-particularly a decreased ability to perceive and understand the feelings and thoughts of others. Early indicators are lack of eye contact, and failure to develop social smiling in the first two months of life; by 18 months of age, not pointing at objects, absence of pretend play, and failure to communicate by showing, are additional risk factors suggestive of autism spectrum disorder, or ASD [27].

ASD's diagnosis has been increasing world-wide, and is now estimated to afflict as many as 1 in 44 children in the United States. The question is, why? Although much of this increase can be attributed to broadening of diagnostic criteria, and increased awareness of ASD, causative environmental factors also have been proposed and remain under consideration.

Synthesis and Discussion

An association between visual impairment and risk of ASD is well established [28-30].

The foregoing is intended to advance an understanding of this association by framing it in an evolutionary context that links changes in hominin visual and cognitive abilities to underlying genetic pleiotropies that improve early close vision but harm it later in life. The two hypotheses outlined in the introduction can now be restated with some additional details.

- The abilities needed to thrive in the increasingly complex social world of evolving hominins have been-and remain-contingent on infants quickly gaining sophisticated social capacities and propensities, which in turn depend initially and crucially on observing the emotional state of caretakers, particularly mother, but also an increased number of closely related individuals such as father and grandparents. This need would have powered selection for the visual and facial characteristics described above, predicting specifically that when neonatal vision develops aberrantly, or appropriate social cues are absent for other reasons (see below), the risk of landing on the autism spectrum is increased.
- The genes accumulated by especially powerful selection that confer the visual abilities required to jump-start social perspicacity are expected often to have associated downstream negative effects, and delaying, dampening, or eliminating these effects is expected to have been hampered by the especially rapid decay in the force of selection acting on the visual system predicted by the asynchronous aging model. This would potentially explain why humans, alone, live with presbyopia for decades, despite it being maladaptive.

A suggested experimental test of the prediction that aberrant development of vision in infants will increase ASD risk will be outlined below, but existing evidence consistent with the prediction is that congenitally blind children have a much greater than normal risk of developing autism [30]. The fact that most congenitally blind children do not fall on the autism spectrum could be construed as going against the hypothesis developed here, but more likely is an indication of the robustness of the systems in place to assure the development of social sophistication.

Additional evidence for the importance of early vision in the development of social skills is that children with extended stays in neonatal intensive care units (NICUs) due to prematurity or illness also are at higher risk of autism [31]. This is expected because prematurity, illness, and the NICU environment, which limit parental interaction with neonates, presumably interfere with infants receiving the visual cues proposed to be critical to social and cognitive precociality. It is important to emphasize, however, that these findings are not definitive in their support of the hypothesis because blindness, prematurity, and neonatal illness can come with significant comorbidities and barriers to optimal cognitive and social development, independent of those suggested here.

Another potential weakness of the hypothesis is that it does not directly explain ASD's increased prevalence among males. However, there is a cogent hypothesis that does so: The 'extreme male hypothesis,' which attributes increased male prevalence primarily to effects on emotional and social development due to excess early exposure to testosterone [32]. Complementarily, rather than alternatively, the hypothesis here agrees that maleness puts children closer to the cliff-edge leading to ASD [33], but it also maintains that it is early visual impairment that often pushes an excess number of males over the edge.

An experiment that would definitively support or reject the proposed causal link between early vision impairment and autism risk would require screening a large number of neonates for defects in close vision, and correcting them when found. Fitting them with corrective lenses is presumably the most feasible approach to correction. The predicted outcome would be that ASD will develop significantly less frequently in the screened, corrected population than in the population at large.

Evidence connecting infant vision and midlife presbyopia *via* AP gene-effects is lacking, perhaps because no one has thought to look for it. But it could be identified, at least in principle, by looking at selection hotspots in the human ge-

nome. Targets in this search would be genes with effects on cognition, social precociality, and development of the visual system, particularly features of human lens growth absent in other primates [34], as well as genes implicated in autism, of which there are many [35,36].

Conclusion

The hypotheses presented above are speculative, but the arguments underlying them are well established and seem to fit together without need for contrivances. And because they reveal a straightforward approach to preventing at least a portion of ASD, as well as a potential pathway to better understand and prevent or correct presbyopia, it seems reasonable to pursue additional evidence, including by conducting the studies outlined in the discussion section. Interest by pediatric ophthalmologists would be especially welcome.

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Conflict of Interest

None

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