Evolutionary Origins of ORL Disorders
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ABSTRACT
It is understood that our ancestors gained a strong evolutionary selective advantage in shaping the vocal tract that allowing them speak more intelligibly. However, humans pay heavy prices for the uniqueness of vocal tracts configuration. We precariously swallow our mouthful in exchange for speaking clearly, and the second heavy price humans pay is sleep snoring. Their evolutionary cause is that the respiratory system deriving on the wrong side and too late from the alimentary system. The fact that our nasal septum is usually curved contributes to these disorders. The reason of this is that a few million years ago humans had to compromise from the width of the birth canal with their pelvis in order to stand upright. As for our children’s ears, their development resembles the evolution of hominoids and they are far from smooth.

Keywords
Evolution, Aspiration, Sleeping apnea, Septum deviation, Sinusitis, Otitis media.

Introduction
Evolution is a process by which a living organism transmits its genome through mutations to succeeding generations by making compromises and trade-offs in exchange [1]. A living being does not need to be in perfect condition to meet these conditions; it only needs to be fit enough to transmit its genome [2,3]. The most distinctive trait of humans is probably bipedalism [4]. Bipedalism has been selectively maintained throughout human evolution probably because of the fitness advantages it offers [5]. However, our ancestors had to make a series of compromises in exchange for these advantages; this is how evolution works [2]. Every living being’s body is adorned with countless inventions that were selected because they maximize reproduction and occasionally even work unfavourably of health, they were acquired in exchange for trade-offs and not necessarily to help maintain health [6]. As a result, our bodies are a bundle of compromises where various adaptations compete with each other, which can bring disadvantages as well as advantages [7]. On the other hand, evolution cannot selectively destroy defective traits of an organism and rebuild it in a way that natural selection will work on [8]. That is to say, many structures of our bodies are created with temporary and improvised solutions [5]. These improvised solutions are permanent when they allow us to find a mate and transmit our genome, but the compromises in return can only be noticed when a disturbance occurs [6].

Man has recently stood upright and, as a reward for standing upright, has gained the ability to speak much more recently; however, the price he has paid for this is asphyxia while swallowing mouthful and sleeping, sinusitis and otitis media [9,10]. Sinusitis and otitis media are the compromises of bipedalism [11]. Other important changes in the human respiratory apparatus and function, such as postnasal laryngeal slope, early contact of the palate and epiglottis, nasal aperture shape and prolonged exhalation period, are all by-products of bipedal locomotion [10]. Briefly, no part of our face is perfect [12]. This manuscript will examine the price the human face pays for its restructuring after standing upright from an evolutionary perspective.

Speech and asphyxia
It remains to be understood whether chatter is more than a wasted energy, but has a role in developing and firing our neural networks [13]. We owe the cultural heritage we have created over tens of thousands of years to the methods of communication we have diversified from the cave wall to the Internet. But our ten-thousand-
Most mammals make sounds, but the human vocal tract is special for two reasons. First, our brain has the exceptional ability quickly and accurately moves our tongue and other structures that modify the shape of the tongue [17]. Second, our distinctive short face has given our vocal tract a configuration seen only in modern humans, along with beneficial acoustic properties [18]. The horizontal and vertical parts of the uniquely shaped human vocal tract are of equal length, allowing us to speak. This vocal tract has two equal-length tubes that require less effort to produce vowels with more open frequencies [19]. The structure of human mouth allows producing abrupt vowels that allow the listener to understand correctly [20]. It is conceivable that there was a strong selective advantage that shaped the vocal tract, facilitating our ancestors to speak more intelligibly [21].

Nonetheless, human beings pay a heavy price for the uniqueness of vocal tract configuration. When we eat, we are performing two independent tasks at once: speaking intelligibly and swallowing our mouthful somewhat precariously [22]. This conundrum is peculiar to modern humans, and our unusually small and retracting face makes our unique ability to speak risky [23]. Presumably other Homo’s may have spoken with their mouths full at dinner as well, but their speech was not as clear as ours, thus they were less likely to aspirate their mouthful [24]. Neanderthal jawbones, for example, were wider and stronger than ours, meaning that Neanderthal wisdom teeth would not have suffered from tooth pain [25]. Homo sapiens distinguish itself from Neanderthal with a flatter face, a smaller jaw, and a tongue that goes deeper into the larynx than any other mammal. Humans were able to make sounds other than grunting by virtue of this new equipment [26]. Conversely, the position of the Homo sapiens tongue during feeding makes breathing difficult. The mouthful can easily go down the wrong pipe, a biological problem seen only in modern humans [27].

The respiratory and alimentary tracts would have to be separate places in order for us not to choke. Essentially, our pharyngeal configuration in infancy is such that babies can breathe through the nose while breastfeeding [28]. For us, all the air we breathe passes through our mouth and what we gain in return is the privilege of being able to chatter [19]. Choking as swallowing down a food is caused by dysfunction of the epiglottis. The epiglottis is a cartilaginous valve that reflexively closes the trachea during swallowing. Also at rest, the epiglottis is located at the posterior edge of the soft palate in other mammals. Unlike all other mammals, the epiglottis in humans is several centimetres lower [29]. With a lower larynx, humans have developed a large shared volume behind the tongue where air and food travel together to enter either the esophagus or the trachea. As a result, food sometimes is stuck in the back of the throat, obstructing the airway [30]. Humans are the only species at risk of asphyxiation when carelessly swallowing large mouthfuls, and this is a more frequent cause of death than previously thought [31]. Deaths due to food aspiration may be under classified due to lack of diagnosis [32]. We pay a heavy price for speaking more intelligibly.

The pickwick

Snoring, the second trade-off of human throat anatomy, can be a sign of a serious disorder called “sleep apnea” [33]. The elongated soft palate tissue in humans allows the airway to close during routine breathing, triggering the sleep apnea cycle [34]. In sleep apnea, the larynx closes randomly throughout the night, preventing body tissues from getting enough air. The inability to take air causes blood oxygen levels to drop and blood pressure to spike. Depending on the severity of the condition, people with sleep apnea are more likely to develop health problems such as hypertension, kidney damage, visual disturbances and cardiovascular diseases [35]. Widespread acceptance of idea is that the disorder can be a side effect of obesity [36], whereas sleep apnea is a breathing disorder and is caused by the position of the tongue and tissues of the larynx [37]. The risk of developing sleep apnea increases with weight because the tissues of the larynx thicken in obese people, blocking the airway during sleep. The Western-style and fast-food diet has played a leading role in spreading sleep apnea along with obesity [38].

As for the evolutionary explanation, the anatomical trade-off that humans made in order to use a complex language may be the cause of sleep apnea [13]. The airway lies within the cranio-facial skeleton in all mammals [39]. The body of a four-legged animal changes as it evolves and stands upright, the upper extremities shorten, the spine stiffens and deforms, the brain grows, but the face also changes [40]. The anteroposterior pole of the face shortens and lengthens vertically, the forehead widens, the neck lengthens, and the cranio-facial skeleton no longer protects the airway [41]. Changes in human facial morphology have enlarged the cranium and shortened the cranio-facial part of the airway within the skull, while the elongation of the neck has left the neck part of the airway unprotected [42]. Only soft tissues such as skin and muscle protect the largely unprotected airway, leaving it vulnerable to collapse and trauma [43]. This explains why snoring and obstructive sleep apnea are much more common in humans than in other animals. Sleep apnea is a defect of the human body plan [10].

Nasal septum deviation

Nasal septum deviation occurs when the septum is displaced laterally, either to one or both sides [44]. This displacement indicates that the nasal septum and surrounding structures are growing abnormally [45]. The nasal septum is deviated in most adult humans; in facial trauma, the nose often takes the brunt of the impact [46]. Nevertheless, the majority of deviations occur due to the pressure to which the face is subjected during birth [47]. The pelvic outlet of the human female is narrower than that of non-human primates because of adaptation to bipedalism, and parturition is difficult only in humans, despite the fact that human
The Neanderthal skull was more primitive than that of modern humans and more similar to that of Java man [62]. The evolutionary transition from Neanderthal to modern humans involved the brachicephalization of the cerebral part of the skull, the formation of the forehead, and the reduction of the brow ridges and maxillae [62]. Therefore, it can be assumed that from Neanderthal to modern humans, there began to be transitions in the shape of the cranium that had not occurred before, and that there was an active influence on the formation of the maxillo-facial cranium in Neanderthal. Thus, it can be argued that septum deviation began to form in the Neanderthal stage [63].

**Sinusitis**

We don’t know anyone who does not have retro-nasal discharge, tonsillitis-pharyngitis or sinusitis [64]. The evolutionary adaptive purposes of sinuses are to stabilize and strengthen the skull [65]. Sinuses moisturize the air inside by producing mucus. When a pathogen or allergen reaches our airways, our nose begins to produce mucus, thus preventing the foreign substance from travelling downwards, and in response our nose starts to run. When this overproduction of mucus blocks the pathways connecting the nose to the sinuses and the sinuses to the cranial cavities, these air pockets, whose drainage is impaired, are filled with mucus and cannot ventilate and internal pressure increases. The sinus lining also swells, contributing to the increase in pressure. This is the cause of headache [66].

Man has four sinuses that cause sinusitis pain: Frontal, maxillary, ethmoid and sphenoid sinuses [67]. In H. neanderthalensis, on the other hand, the lateral wall of the nasal cavity is unique [68]. Especially our sinuses may be evidence for ape and human evolution. Gorillas and chimps have all four sinuses, but orangutans and gibbons lack the frontal and ethmoid sinuses, but have preserved the maxillary and sphenoid sinuses, which is enough for them to suffer the same headaches as us [69]. The reasons why Asian apes have lost their ethmoid and frontal sinuses are unknown. The distance between the orangutan’s eyes is very narrow and its forehead is more flattened and concave than its African cousins. Therefore, there may not be enough volume for air pockets to form [70]. Chimpanzees, gorillas and humans inherited the ethmoid and frontal sinuses from the oldest apes at least 30 million years ago. The gibbon and orangutan lost these sinuses independently after diverging from the rest of the apes; the gibbon evolved about 18 million years ago, while the orangutan diverged from the other great apes 15 million years ago [71]. If humans had diverged from Asian apes and not from African apes, perhaps they would not produce as much mucus and the pain of sinusitis would be more bearable [72].

**Otitis Media**

All reptiles and birds have only one middle ear ossicle: the stapes/columella [73]. The placement and functioning of two extra ossicles in the mammalian middle ear is a classic example of how some structures can change over the course of evolution to work in a different way for a new purpose [74,75]. The evolution of the middle ear and jaw joint are critical steps in mammalian evolution. The evolution of the middle ear with three ossicles is linked to the evolution of the middle ear and jaw joint are critical steps in mammalian evolution. The evolution of the middle ear with three ossicles is linked to
the evolution of a different jaw joint; these two structures evolved together to form the typical mammalian skull [76]. The first jaw joint fused with the middle ear to form the mammalian middle ear with three ossicles in a remarkable transition in mammalian evolution [77]. This middle ear with three ossicles has now become one of the defining characteristics of mammals [78]. The incorporation of a jaw joint into the mammalian middle ear was only possible after the evolution of a new articulation of the upper and lower jaws, which required the formation of the temporomandibular joint. The two new ossicles of the mammalian middle ear, the malleus and the incus, are homologs of the articular and quadrate bones in the upper and lower jaw joint of non-mammalian jawed vertebrates. Consequently, malleus, incus and stapes are homologous to articular, quadrate and columella, respectively [79].

Primate auditory morphology shows great variability and this variety has long piqued the interest of systematists [80]. Among extant strepsirrhines (wet-nosed primates), the tympanic ring of most lemurs is suspended in a single-chambered middle ear, while the tympanic ring of extant anthropoids like lorises is fused to the lateral tympanic wall in a multi-chambered middle ear. Lorises have a laterally fused tympanic ring and additional pneumatic fields, such as the diverticulum in the Eustachian tubes [81].

As far as is known, humans are the only species to have otitis media (OM) and evolution has had a significant impact on the development of OM in humans [79]. Otitis media is an evolutionary compromise for the ability to speak [82]. The mastoid process and Eustachian tube are two fundamental structures that are closely related to OM. The mastoid process, like bipedalism, is unique to humans and owes its big size to bipedalism [83]. OM is essentially a childhood “disease” and children who had OM frequently have smaller mastoid foramina. There is also an association between having small mastoids and Eustachian tube hypofunction; a less effective Eustachian tube creates more pressure differential over time, increasing the risk of infection [84]. The fact that the coexistence of chronic OM with effusion with Eustachian tube constriction may indicate that OM is a result of adaptation to speech [82]. OM can also be seen in modern human adults due to other human adaptations: the development of speech means that palatal morphology has diverged from that of other primates, with a decrease in prognathism [85]. There are important anatomical and physiological differences between humans and apes, particularly in the Eustachian tubal muscles [83]. The absence of an OM-like problem in extant great apes suggests that negative selection is at work in those [86].

The discipline of developmental plasticity (Evo-devo) seeks to draw a correspondence between our ancestral phenotype and the developmental stages we go through from zygote to adulthood [87]. The evolutionary process observed in facial structure, from the quadruped primate to the biped Homo continues in a similar manner from infant human to adulthood [88]. Remembering this principle, we would expect to see middle ear structures in pre-sapiens Homo similar to those of children. Indeed, mastoid process dimensions are significantly smaller in Neanderthal specimens than in H. sapiens specimens. The mastoid process must represent the ancestral condition in both Neanderthal and sapiens evolutionary lineages [89]. Neanderthals must be retained the juvenile condition from their precursors and this must have evolved into a derived trait in Neanderthals [90]. The structure of Neanderthal Eustachian tubes should also be understood by remembering the evo-devo principle: Eustachian tube morphology must have left Neanderthals vulnerable to middle ear diseases [91]. In fact, the basicranium appears to have undergone significant morphological change in the entire hominin clade [92]. Thus, cranial base morphology in the hominin clade must have continued to evolve before and after the genus Homo [93].

Eustachian tube malfunction is definitely associated with OM. In the human infant, the Eustachian tube is shorter, wider and more horizontal than in the adult, and cannot ventilate and drain the middle ear cavity as well as the adult tube [94]. The risk of regurgitation from nasopharynx in childhood is also greater than in adults. Children who are accustomed to breathing through the mouth are more prone to OM due to the immature respiratory structure and function [94]. However, significant developments occur after the child stand upright; the verticality of splanchocranium and basicranial kyphosis increase, so that the Eustachian tube acquires its physiological effect by dilating and ultimately reduces the risk of OM [95]. The tiny and undeveloped mastoids of the human infant begin to expand with cranio-facial changes when they begin to walk. As mastoids begin to expand, the cavity of the middle ear also begins to ventilate better and becomes less prone to infections [96].

Adult human susceptibility to OM is reduced by cranial and postcranial changes caused by upright walking; the skull, mastoid process, Eustachian tube, and palatal musculature are structures that reduce the likelihood of OM in adulthood [97]. The adult modern human mastoid should have grown larger as bipedalism evolved; the increased pulling power of the sterno-cleido-mastoideus muscle with bipedalism leads to enlargement of the mastoids into adulthood [98,99]. The human Eustachian tube becomes more vertical as it grows. This verticalization of the palatal musculature alters the structure and function of breathing and swallowing significantly [82].

The tympanum ventilates a normal middle ear, regulates air pressure and restores its microflora [100]. The tympanum is horizontal in the human infant, as it grows the tympanic ring becomes vertical laterally to form the bony external auditory canal. It is almost entirely vertical in adults, and when it ruptures it drains effectively. The ventilation potential of the middle ear increases into adulthood due to the progressive verticalization of the tympanum [101]. The human external auditory canal has also undergone significant ontogenetic changes [102]. Susceptibility to diseases is high in childhood before these ontogenetic changes occur. While the role of ontogenetic predisposing factors has been demonstrated in humans, it does not necessarily translate into increased risk in other species [103,97]. Basicranial flexion increases with mastoid, tympanic, and splanchocranal...
development from childhood to adulthood. The auricular of the ear continues to grow visibly until the age of 9 years and continues to grow slowly into adulthood. As mandibular and facial development progresses, the verticality of the external auditory canal increases, as does the Eustachian tube [104]. The adult auricle and external auditory canal should be differentially selected to facilitate the drainage of middle ear effusions. This design ensures that the removal of secretion and foreign bodies from the ear canal and middle ear drainage in tympanic tearing [105]. There is significant variation in auricular shape among other primates, with the auricula height/width ratio being highest in Lorises (the most pointed ears) and lowest in Apes (the most rounded or humanoid ear). The auricula shape in Strepsirrhines is longer and narrower than in anthropoids [81].

For these anatomical, physiological and evolutionary reasons, as well as developmental and epigenetic processes, human OM is essentially a childhood disease.

Conclusion
Comprehending human evolutionary adaptations is important in determining disease risks. Our faces became flattened to be able to stand up, thus we gained the ability to speak, but it came with the risk of choking while eating, which is probably why some cultures don’t speak at the table. Our brain was able to enlarge as our faces flattened when we were able to stand, thus we evolved into intelligent beings, but on the other hand, being born and giving birth is so difficult only in humans, and our nasal septum, which is deviated when passing through the birth canal, is not completely corrected in most of us and the pain of sinusitis haunts us. Our middle ear and annexial structures show so much variation that the “diseases” of this region can be considered as remedies invented by evolution [106]. Middle ear effusion, on the other hand, is almost a physiological condition and does not even require treatment in many folkloric medicines [107]. When we understand the “diseases” we observe, from sinusitis to asphyxia to ear discharge, as medical as well as evolutionary consequences, we can stop being surprised at how common they are and reconsider our treatment options.

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