REVIEW ARTICLE

The Fatal Fang: did stress-induced Xerostomia evolve as a strategic offensive/ defensive weapon in hominid combat?

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ABSTRACT

The lack of the traditional natural weapons of combat-horn, claw, extended canine, talon, venom etc. has forced the human species to develop a rather novel and unorthodox biological weapon, namely stress-induced xerostomia, or a reduction in salivary flow into the oropharyngial cavity, during those times of psychological stress that are the usual prelude to combat. Since one of the immunologic functions of the saliva is to keep down the number of micro-organisms in the oral cavity, this reduction of salivation immediately results in a marked increase in the bacterial population of the mouth. Since bites are frequently inflicted in combat between humans, this would result in the inoculation of a substantial bolus of pathogenic micro-organism into the bite wound of the opponent, the subsequent setting up of an infective nidus at the bite site, and thus either in fever/sickness or a severe festering wound necessitating, not infrequently, in a need for frank amputation of the affected digit. In any event the combat ability of the opponent will be substantially reduced as a result. It is therefore likely that there has been a substantial Darwinian selection for this peculiar physiologic trait of a desiccation of the mouth during times of inter-personal stress, when the likelihood of there being a physical conflict between humans rises substantially.

Keywords: Xerostomia, hominid combat, stress, bite, infection, bacteria.

INTRODUCTION

Humans are unfortunate in that unlike most other species that can rely on an array of bristling weaponry be it extended canines

claws, hooves, spines, horns etc, they possess a negligible repertoire of bodily weapons. In addition, they have neither a thick matting of whole body hair or fur to cushion a blow nor a hard cornified exoskeleton or dermal armor that would shield them from a bite, a stab, or a piercing horn. The notorious propensity of humans to engage in combat on the most inane of pretexts only aggravates the situation both for the aggressor and the defendant. Clearly therefore, in the course of human evolution, in the pre-historic era before stone tools/weapons were invented, there was an imperative need to devise some natural weapon that would give both the combating parties an edge over the other. It appears that such a weapon was indeed evolved, but whose existence, let alone remarkable effectiveness - as will soon be demonstrated - has not been appreciated until now. This is the weapon of fear/ anxiety stress-induced oral xerostomia – the "dry mouth" syndrome.

Oral bacteriology:

The oral cavity, under normal conditions, is a seething cauldron of pestilence, housing nearly 190 aerobic and anaerobic bacteria species, some dangerously pathogenic, in its innards (Revis, 2009). The warm environs of the mouth, at a steady 37^{o}C, the availability of trapped food detritus on and in-between teeth, all connive to greatly facilitate bacterial growth. A milliliter of saliva taken from the mouth under normal healthy conditions bears nearly a 100 million bacteria(Revis, 2009). These numbers, while impressive, actually belie the fact that the mouth is in fact substantially under-colonized and can potentially sustain a far greater number of bacteria. That it doesn't owes to the rather remarkable fact that human saliva is actually an unusual liquid with marked antibacterial properties. The sweet breath of the drooling baby owes to the excessive and copious saliva it secretes, which decimates malodor producing bacterial numbers, while the frequent bad breath(halitosis) of the adult human – as most of us are regrettably only too socially aware - is due to a reduction in this flow, which raises the population of odor producing bacteria in the mouth.

The salivary fluid is secreted into the oral cavity on a chronic basis from the three salivary glands, namely the parotid, the sub-mandibular and the sub-lingual, whose combined output amounts to about 1.0 ml per minute(Hightower *et al*., 1973a). Apart from its obvious function of lubricating the mouth, permitting the mastication of food, the enabling of fluid speech, facilitating the digestion of carbohydrate via the ptyalin enzyme that it contains, the saliva performs a less obvious but equally critical function, namely holding down the bacterial populations of the mouth. This it accomplishes through a range of bactericidal and bacteriostatic substances it contains. Firstly, there is the enzyme lactoperoxidase which is capable of generating singlet oxygen, an extremely powerful oxidizing agent that is inimical to bacteria (Brock, 1979). Singlet oxygen is an extremely powerful oxidizing agent generated from ordinary (triplet state) diatomic oxygen when the two electrons present in the two anti-bonding orbitals (π_{2p}*) have opposite spins, as opposed to the identical spins they have in triplet oxygen. This highly reactive configuration gives singlet oxygen an indiscriminate tendency to violently react with, and destroy, any organic substance such as a bacterium in its vicinity (Sharpe, 1992). Secondly, another enzyme called lyzozyme is also present in the saliva and is capable of bringing about lysis of the protective cell walls that surround a bacterium. Among the oral bacteria destroyed by lysozyme are those of the genera *Staphylococcus, Streptococcus, Micrococcus, Proteus, Brucella and Bacillus* (Hightower *et al*., 1973b). Lysozyme is present at a concentration of about 72.9 micrograms per milliliter (Jenzano *et al*., 1986). Finally the saliva contains the antibody Immuno-globulin A, that has a mass between 180-500kd, and which is secretes into the saliva from the blood by the aid of a protein called the Secretory Component. This antibody is capable of binding to

antigens present on the surfaces of bacteria, and the resultant antibody-antigen complex then serves as a molecular attractant signal for the homing in of the macrophages and neutrophils, which are phagocytic cells, into the oral cavity that then proceed to destroy the bacterium (Stryer, 1995). Acting in concert and synergistically with chaotropic ions present in the saliva, this tripartite defense is capable of substantially reducing the bacterial population of the oral cavity, which is why, in the healthy, non-stressed individual, the oral cavity is by and large non-odoriferous.

The primary points of adhesion of bacteria in the oral cavity are the teeth. Even within a few hours of a tooth being thoroughly cleaned and rendered bacteria free, bacterial colonies are evident under microscopic examination of the tooth surface. These colonies then begin to expand exponentially. This is due the formation of dental plague, which is composed of filamentous bacteria such as *Leptotrichia buccalis*, Streptococci, Diphtheroids, Gram negative cocci etc, embedded in an polysaccharide matrix, the entire composite being called a dental plaque (Brock, 1979). The speed of colonization is breathtaking. If the mouth remains un-brushed and there is no evidence that the early hominids practiced any sort of oral hygiene such as the tooth brushing that we practice today the surface area of the plaque rises from 1,436mm^2 to 22,522mm^2 within 240 hours (Lang *et al*., 1972). Yet even this dramatic rise results in the colonization of only about 50 percent of the tooth surface, the bacteria being held at bay in their expansion by the salivary antimicrobial agents afore-mentioned. The friction of the surface of the tooth against the surrounding tissue during a bite event will thus result, via scraping abrasion, in an injection of a substantial bolus of this plaque into the wound, with potentially infective consequences.

Stress-induced Xerostomia

Now it is a well established fact that in the human, any severe psychological stress, be it anxiety, fear or apprehension, can and does immediately bring about a reduction in the flow of the salivary fluid (Bell *et al*., 1963; Bergdahl, 2001). In addition the levels of lysozyme and IgA in the saliva are decreased in individuals under psychological stress (Yu Shan-fa *et al*., 2008; Ng *et al*., 1999; Graham *et al* 1988; Henning *et al*., 1992). This reduction in the flow of saliva is called xerostomia. And its immunologic consequences are as swift and instantaneous as they are serious. Since the typical bacterial division time is only 20 minutes, and since bacterial multiplication proceeds by as an exponential increase, bacterial numbers , which hitherto had been held in check by the antibacterial components of the saliva, now explode into rampant and unrestricted proliferation; and the oral cavity, within a matter of hours, is converted into a cesspit of swarming bacterial colonies. So severe indeed are the deleterious effects of long-term xerostomia, that if the condition is not swiftly corrected it can result in severe dental caries, gingivitis, candidiasis of the mouth, inflammation of the tongue, (glossodynia) and ulceration of the buccal mucosa (ADA, 2000; Astor *et al*., 1999; Greenspan,1996; McDonald *et al*., 1991; CDHA, 2000; Flynn,1993) all as a result of the un-inhibited proliferation of the oral bacteria and fungi, such as *Candida albicans*, that reach such prodigious numbers now that even the squamous epithelium of the oral mucosa is invaded. The reason for this is that normally a opious outflow of saliva washes away and debrides the dead squamous epithelial lining of the oral cavity, thus reducing the food supply to the bacteria in the mouth. The marked reduction, occasionally total shutdown, of salivary flow in xerostomia results in the retention of this desquamated tissue, providing a rich nutrient supply for the bacteria that now not only coat the teeth but the oral mucosal lining as well (Bell *et al*., 1963). The mouth in short is therefore converted into a deadly dangerous biological weapon, whose potential lethality now extends well beyond the mere *physical* damage inflicted by a bite. In the healthy individual this state of affairs fortunately

does not persist indefinitely but lasts only for the period of the psychological stress. The ubiquity of this peculiar phenomenon of a psychogenic stress -induced desiccation of the mouth has, incidentally, been well recognized by writers of thriller novels worldwide. There is hardly a thriller novel in print that does not have the phrase "his mouth turned dry in fear" at some point or other in its narrative, a hackneyed cliché that has been over-used in the fiction book-trade, but which nonetheless accurately reflects physiologic fact; and, as we shall now see, a preparation for battle.

The Toxic tooth as a weapon of combat:

What makes xerostomia pertinent to the central thesis of this paper is that the conversion of the hitherto relatively clean mouth into this "foul mouth" condition, under the conditions of the psychological states of fear and anxiety, that were the prelude to hominid combat in the ancient past, is that the mouth is frequently used in overt aggression to inflict a bite – causing the so-called occlusive injury. And since in a fight the hands are typically extended to either grasp the opponent or strike him, the hand is the site that receives the bulk of bite wounds compared to other parts of the human anatomy. For instance, over a six year period, there were more than 115 visits to the Emergency Department of a Rhode Island hospital for the treatment of bite injuries (Merchant, 2005). And this percentage in all likelihood represents a gross underestimate. The fear of social embarrassment and the even greater risk of legal action keeps a great many of such bite incidents under-reported. The presence of the aforementioned teeming hordes of infective bacteria in the xerostomic mouth, converts the hitherto relatively innocuous mouth and its 32 teeth into a potent weapon of war. It has been found that over 15% of all human bite wounds turn infectious (Revis,2009). Far more ominous is the fact that in the pre-antibiotic days, nearly 20% of all bitten fingers which, as mentioned, is usually the bite site in fighting humans - had to be amputated if the hand was to be saved from the spreading infection(Revis, 2009). Since antibiotics were obviously un-available in the pre-historic past the remarkable effectiveness of this "foul mouth" weapon is dramatically underscored, and provides a resounding endorsement of the hypothesis that the stress-induced xerostomic syndrome arose in hominid evolution exclusively for the purpose of combat, and substantially leveled the playing field between two combatant of unequal size. A hominid that lost his finger, or worse, hand, as a result of such an infection isn't very likely to throw a punch in any haste in the future. It must be borne in mind that even amputation of the affected finger, as was required in the pre-antibiotic era, in order to prevent the proximal spreading of the infection, is a highly skilled surgical procedure that was not therefore available to the earlier hominids. In such a situation it would be predicted that there was a spreading of the infection to the rest of the body potentially resulting in death of the hapless hominid. The power of the bite in producing serious injury is highlighted by the fact that the masseter muscle of the jaw - which, acting in concert with the temporalis muscle, is responsible for the closing of the jaws (Jacob *et al*., 1982a) - *is the strongest muscle in the body* if the shortness of its lever arm is taken into account, capable of exerting a force of 444Kgs, or 4351.2 Newtons ! (McFarlan *et al*.,1992).

What makes this unorthodox biologic weapon so effective is that a bite does not have to be inflicted in order to inimically affect the opponent. Even in the passive situation where the xerostomic human does not have an opportunity to bite but instead receives a clenched fist blow to the face, the cutting action of the frontal incisors or lateral canines on the striking knuckle is capable of causing a severe enough cut (3 – 8 mm in length) to set up an infective locus. Indeed so common is this form of injury that, in the parlance of the hospital ward, it is called a clenched fist injury. And it is just as effective of causing a serious ingress of oral pathogens into the cut knuckle, as the frank, uninhibited bite (Revis, 2009). If the

tooth is partially broken under the force of impact all the better, for the jagged edge of the broken surface will produce an even more severe and deeper laceration and thus a greater deposition of bacteria into the wound. Indeed, in a famous criminal case where a bellhop in a high class hotel had killed a patron when surprised in the act of stealing, it was precisely such a wound that lead to his capture. For the closed fist blow to the woman's mouth while proving lethal to her, also resulted in a tooth grazing injury to the murderer's fist; and when he sought medical attention for the injury, that had turned infectious, he was readily apprehended.

The marked potency of the bite in producing a suppurating infection of the bitten hand is due primarily to the activities of two bacterial species of the many species transferred in the bite event - *Eikenella corrodens* and *Staphylococcus aureus*. The former is an aerobic, Gram-negative bacterium that is capable of causing a persistent infection and abscess formation at the wound site, while the latter, also an aerobic species, intensifies the severity of the infection and enhances the post-trauma complications developed at the injured zone (Revis, 2009). Among the other bacteria found in the injured tissue are the anaerobic Bacteroides species, Enterobacter, Klebsiella, E. coli, Proteus and Pseudomonas (Mann *et al*., 1977; Goldstein *et al*., 1978; Bilos *et al*., 1978; Shields *et al*., 1975). What makes the hand particularly vulnerable to a bite injury is its peculiar anatomy. The dorsal (upper) surface of the hand that is the surface opposite to the palmar surface is a very exiguous structure, with a but a very thin layer of protective fat. Thus during a clenched fist injury to the hand, several possible destructive scenarios can unfold. First the Metacarpophalageal (MCP) joints of the five fingers, what in layman language are called "knuckles", which are the "hinges" between the metacarpals (the five cylindrical bones radiating out from the wrist region to the five knuckles) and the phalangeal bones of the fingers, are completely exposed, with but layer of skin covering them, and the impacting tooth can thus either easily shatter the joint or tear through its protective capsule and deposit the bacteria directly in the joint cavity itself. Since the middle finger MCP joint is the largest it tends to bear the brunt of the impact and is the one most often damaged. The resultant infection, is on occasion so extreme that the joint can no longer be flexed. Secondly, if the fist smashes into the tooth on the phalangeal surface the phalanges can be broken, and the broken ends of the bone exposed to the exterior.

In the case of the occlusive bite, the injury can, of course, occur on either the dorsal(upper) or ventral (i.e. the palmar) surface of the hand and the deposition of the bacteria can occur via the multiple tooth puncture wounds on these two surfaces. What makes the hand so particularly vulnerable to infection is that it is not only poorly vascularized, thus preventing the immune part of the blood circulation from accessing it, but in addition, the bacteria inoculums, as they multiply, can spread along the tendon sheaths to other parts of the hand and set up further infective loci. The hand in the vicinity of the fingers has very little musculature and the action of the proximal flexor muscles in the forearm such as the Flexor digitorum superficialis is transmitted to the phalanges of the fingers via the flexor tendons, or in the case of extender muscles like the Extensor indicis muscle via the extender tendon(Jacob *et al*., 1982b). Thus once the skin is penetrated by the tooth and the bacteria are deposited in the cut, the bacteria multiply and expand to the flexor and extender tendons. In addition the flattened anatomy of the hand results in its muscles being present in several layers (Jacob *et al*., 1982c), and thus the infection can spread proximally (i.e in the direction from finger to wrist) via the gaps present between these tissue planes. In addition there are two sets of muscles present in the hand itself. These are the lumbrical muscles present on the palmar side of the hand and the Inteosseous muscle present between the forefinger and the thumb. These muscles are surrounded by sheaths the so-called interossei and lumbrical sheaths

which regrettably can also serve as conduits by which the infection can spread from the bite site to the rest of the hand (Mann *et al*., 1977). As the wound festers the hand turns painful and tender and may discharge a malodorous pus (Welch, 1936). The patient frequently develops chills and a fever as well (McMaster, 1939). The tendons of the hand are enclosed in sheaths called tendon sheaths *within which* the tendon slides, and one of the complications of the infected hand develops when these sheaths become so inflamed that the tendon can no longer slide freely within its annular jacket. This pathological phenomenon is called Tenosynovitis, and when it affects the tendon sheaths of the flexor tendons it can progress to the point where it is no longer possible to extend the finger, the finger remaining locked in the flexed curled position in what is aptly and colorfully called "the trigger finger". (Jacob *et al*., 1982d; Cartoto, 1986).

This form of clenched fist injury to the striking assailant may also help clear up a long standing evolutionary mystery. A peculiar physiognomic feature of *Australpithecus afarensis* is the prognathous jawline, with the frontal incisors and canines projecting outward at so sharp an angle from the maxilla and the mandible as to give this extinct hominid a most unsightly "buck-tooth" configuration (Boaz *et al*., 1997a). While aesthetically unpleasant, this configuration of the frontal tooth-line would be expected to have considerable defensive value. In the modern human the orthognathous configuration of the face results in the frontal teeth having virtually a near vertical disposition, such that the striking fist will slam into the tooth surface on its *flat broadside* rather than on its sharp edge. This regrettable disposition, while causing negligible injury to the clenched fingers of the aggressor, will impose such a severe mechanical stress what, in the terminology of engineering, is called a "bending moment" on the tooth surface as to essentially knock it out of its socket, as more than one schoolboy, who has been forced to get a set of false replacement teeth after a fight, has discovered to his chagrin. The unique defensive power of the Australopithecine frontal teeth on the other hand derives from the fact that they project so sharply outward, at an angle of nearly forty five degrees to the horizontal (Boaz *et al*., 1997a), that the *likelihood of the fist in its swinging arc striking the sharp edge of the tooth rather than its flat broadside increases dramatically.* This has two consequences. Firstly, since the tooth experiences a force, a substantial cosine component of which is directed *inwards* into the gum cavity, along the longitudinal axis of the tooth, the probability of the tooth being smashed out of its anchorage is diminished. And this is an important consideration, for the loss of one or more teeth in combat, in a species that , not having access to fire (Boaz *et al*., 1997b), was forced to eat very hard and tough food material, would have disastrous consequences for its survival. Equally importantly, since the outward knuckle surface now will come in contact with the sharp edge of the tooth, the depth and severity of the injury to the hand of the offender will be far more serious, the resultant infection the more intense. It would be tantamount to punching into the sharp edge of a held knife - not a very pleasant thought and an even less pleasant sensation and outcome. The pugilistic wisdom of the Australopithecene "buck-tooth" is thus readily evident. This might have been particularly relevant in the case of *Australopithecene gracilis*, rather than *Australopithecine robustus* since the more delicate structure of the cranial vault and associated facial bone structure would render the former much more vulnerable to a blow to the facial region (Boaz *et al*. 1997c).

The bite as a offensive weapon was probably even more effective in the more ancient ape-like hominids such as *Ardipithecus ramidus* (circa 4.4Mya) that had more extended upper and lower canines(Boaz *et al*., 1997d) which would give a greater depth of teeth penetration and thus the deep-seated deposition of a greater number of bacteria into the wound. Indeed, so large and prominent are the canines of this hominid compared to the post-canine teeth that they resemble more fangs than teeth, converting

the canine tooth literally into a potentially fatal fang.

CONCLUSION

The feral bite is a mode of combat with an ancient and time-honored pedigree. Other species closely related to humans such as the chimpanzee, the gorilla, and the Old and New World monkeys frequently bite each other - and regrettably also humans when raised as pets – during conflict situations. For instance, nearly 80% of Macaques secrete the *Cercopithecine herpes virus* – 1(B-Virus) into their saliva (Ostrowski, 1996). This shedding of virus, which increases when the animal is under stress, (Weigler,1992) can cause a meningoencephalitis in bitten humans, sometimes so severe in manifestation as to result in either death (Ostrowski, 1996) or marked neurologic impairment and the permanent institutionalization of the patient (Palmer, 1987) . Hence there is the real possibility that the xerostomic mouth as a mode of offense/ defense arose very early in the evolution of the primate family; indeed its antecedents might well lie much earlier to the Hominidae. How much earlier, if such is the case, is unclear for the incidence of xerostomia in non-human primates has not been rigidly quantified. The fact that nearly 20% of all male chimpanzees die in combat only serves to underscore the gravity of the situation.

With the advent of the Bronze Age and the invention of the classic weapons of war, it is likely that this uncouth form of defense and offense rapidly became obsolete. The fact that in the modern day setting subjective feelings of anxiety and fear easily and routinely evoked in a multitude of social situations such as preparing for exams, appearing for job interviews, delivering a public speech, going out on a blind date - still evoke dryness of the mouth clearly bespeaks the activation of an atavistic mode of defense; a regrettable and counter-productive activation that no longer serves any useful purpose. Furthermore, the rise in the incidence of HIV AIDS has brought this issue to the fore. Since HIV in its latter stages affects brain function, at which point primary social inhibitions are lost, and since a great many of these patients come from the impoverished, marginalized section of society, frequently needing to supplement their income and medical bills by crime, their apprehension by law enforcement personnel poses a grave risk, for in the tussle of being apprehended it is not unknown for the convict to threaten to bite the law officer, full aware of the disastrous sequelae that will follow. And it is a threat not to be taken lightly coming as it does from a person who does not have much longer to live anyway, and thus has little to lose by taking liberties with the law.

The novelty of the highly infectious xerostomic mouth, as a combat weapon, should not obscure the fact that it, in fact, represents a re-discovery by the primate(?)/human species of a mechanism that is far more ancient. On the islands of Komodo, Gili Dasami, Flores and Rinca in Indonesia exists a reptile, the ill-famed Komodo Dragon, that uses precisely this form of attack in bringing down its prey (Ciofi, 2004). The foul, saliva dripping, mouth of this beast, whose lineage extends back to the Jurassic, teems with bacteria of such lethal potency, such as *Pasteurella multocida*, that a single bite of the dragon on the body of the ambushed prey animal is enough to ensure its doom. The injected inoculum works on the hapless beast over a period of days, while the komodo bides its time. And as the bacterial population in the wound skyrocket and invade the blood stream in prodigious septicemic numbers, the animal progressively becomes weak, falls immobile, and, within a few days after sustaining the bite, is dead (Auffenberg, 1981; Montgomery *et al*., 2002). It is a simple matter for the dragon to then waddle up to the now putrid carcass and feed off it at leisure, the highly toxic bacteria laden innards of the dead animal having no effect on the dragon itself. This remarkable impunity to these bacteria is critical for the survival of the dragon, for the "foul mouth"

condition is chronic and ever-ready for deployment; the festering putrefying condition of the oral cavity sustained by the rotten fragments of meat trapped in the tooth crevices which provide an effective culture medium for the bacteria. So powerful and potentially lethal are these bacteria that though the beast has no natural predators on the island that it needs to fear, its entire exterior is covered with a thick and impervious keratinized armor, so hard that a hand run over it returns the tactile impression of chain-mail. The function of this remarkable armor is simple to shield the komodo dragon from its own kith and kin, a single bite of which can well turn the beast into a meal for its compatriots.

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