

An evolutionary explanation of the plasticity of salt preferences: Prophylaxis against sudden dehydration

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Summary Salt preferences, which vary widely across individuals, are a function of past exposure to both levels of dietary salt and dehydration. From an evolutionary perspective, such plasticity is puzzling, as the health costs of high salt intake, combined with the increased time and energy needed to obtain large quantities of salt under ancestral conditions, suggest that natural selection should have eliminated the plasticity in preferences that can produce such behavior. This puzzle is resolved once it is recognized that high salt intake may provide protection against sudden dehydration, a benefit that outweighs the costs associated with this pattern. It is proposed that an adaptive mechanism calibrates salt preferences as a function of the risk of dehydration as indexed by past dehydration events and maternal salt intake.

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INTRODUCTION

Because of its possible role in hypertension and other disorders, considerable attention has been devoted to the factors that influence salt consumption. Although preferences for the taste of salt exhibit substantial interindividual variation, this heterogeneity is not due to genetic factors (1). Rather, it appears that past experience is a key determinant of salt preferences and consumption rates. To date, investigators have focused primarily on the proximate mechanisms that mediate the effect of experience on salt preferences (cf. 2). While published results thus hint at complex neurophysiological machinery underlying the establishment of set points for salt intake and the maintenance of homeostasis there-

after, viewed from an ultimate perspective, the plasticity of salt preferences is a puzzling phenomenon. Although debate continues over the health consequences of high salt intake, the consensus is that, at least in regard to some conditions, a high-salt diet is harmful (3). This alone is reason to ponder why natural selection would have produced mechanisms exhibiting plasticity such that individuals are capable of developing preferences that result in high intake. Moreover, because salt is not easy to come by, this plasticity is intriguing even in the absence of any negative health consequences that may accompany a high-salt diet.

Optimal foraging theory holds that natural selection should have crafted organisms in such a fashion that they adjust their foraging behavior so as to optimize the benefits obtained and minimize the costs incurred. The behaviors of both human (4) and nonhuman (5) foragers frequently conform to this general prediction. Investigators typically evaluate 'benefit' in terms of calories consumed, and measure 'cost' in terms of calories and time expended. However, just as calories alone are not the sole determinant of nutritional adequacy, so too caloric intake is not the only value that must be maxi-

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mized: the logic of optimal foraging theory entails the prediction that organisms' pursuit of micronutrients should likewise be such as to maximize the benefit/cost-of-procurement ratio. Under the environmental conditions in which most terrestrial mammals live, including the conditions likely characteristic of our own species for most of its history, salt must often be actively sought out if it is to be obtained. It is therefore reasonable to expect that natural selection should have disfavored any mechanism that leads to a greater intake of salt than is needed. Even holding aside any negative health consequences of high salt intake, because a heightened preference for salt results in a greater expenditure of time and energy in the pursuit of salt relative to the expenditures characteristic of individuals possessing the average phenotype, the existence of developmental plasticity in the preference system suggests that there must be some hidden benefit attending increased salt intake – if only costs and not benefits accompanied heightened salt intake, natural selection would long ago have eliminated the plasticity in the preference system that produces such behavior.

In the rat, episodes of salt depletion produce enduring increases in salt intake (6,7). Epstein (8) has suggested that such changes may be adaptive, as experiencing salt scarcity indicates that such events are likely to recur – if one lives in a world in which salt is rare, it pays to possess an elevated preference for salt, as this will result in greater time and energy spent pursuing it, thus ensuring an adequate intake despite the possibility of episodic scarcity. The core insight, that experience is a source of information about probable future events in the local environment, is an important one. However, while Epstein's hypothesis accounts for increased preferences in response to past scarcity, it does not clearly explain several other experiential determinants of salt intake. When salt intake is artificially depressed in pregnant rats, the resulting offspring exhibit lifelong increases in salt preference and consumption (2,9; but see 20). This result is consistent with Epstein's hypothesis, as it suggests that facultative mechanisms begin collecting information prenatally about the risk of salt scarcity in the local environment. Oddly, however, the opposite effect also occurs, since, when salt intake is artificially elevated in pregnant rats, the resulting offspring also exhibit lifelong increases in salt preference and consumption (9,11). Similar effects occur when pregnant females are fed high-salt diets (12,13), i.e., offspring intake is elevated even though salt is superabundant in the local environment. More puzzling still, it is not only maternal salt intake that affects offspring preferences, as exposure to dehydration also leads to lasting increases in the preference for salt (14,15). This effect is particularly striking when exposure occurs

prenatally or during the initial postnatal period, as the resulting increased preference for salt both manifests early and appear to be robust over the lifespan, patterns which have been demonstrated in both rodents (16) and humans (17–19; but see 20). Following the reasoning that (a) the value of the resources sought should exceed the costs both of the search and of consumption, and (b) experience is a source of information about probable future circumstances, heightened salt intake may thus reflect both the possibility of salt scarcity and the possibility of sudden dehydration. While the value of increased preferences is clear in the former condition, the link between dehydration and salt intake begs further scrutiny.

SALT PROTECTS AGAINST SUDDEN FLUID LOSS

A wide variety of pathogens cause rapid fluid loss in hosts, often as a means of enhancing transmission (21). Mammals subjected to rapid fluid loss risk impairment and death as a consequence of dehydration. Importantly, high salt intake promotes water retention via fluid sequestration in extracellular spaces (22). By increasing bodily fluid reserves high salt intake may thus provide protection against catastrophic fluid loss. Such protection constitutes a benefit that can outweigh the costs of increased risk of salt-related disorders and increased expenditures of calories and time in the procurement of supranormal levels of dietary salt.

The risk of sudden fluid loss due to infection is likely to vary over time as a function of seasonal and climatic variation, host population density, and evolutionary arms races between the offensive capabilities of pathogens and the defensive capabilities of hosts. In general, the magnitude of the variation in risk of fluid loss will be a function of the magnitude of the variation in these factors. In relatively invariant environments (particularly those not conducive to pathogen proliferation, i.e., arid and very hot or very cold locales) in which host population levels are relatively stable, the risk of catastrophic fluid loss will not vary much from generation to generation. As a consequence, holding aside the need to calibrate preferences in light of salt availability, natural selection can be expected to fix baseline salt preferences, and hence default salt consumption, at a level that maximizes the ratio between the benefits of dehydration protection given local pathogen prevalence and the costs of salt intake and procurement. In contrast, in environments in which the risk of pathogen-induced fluid loss varies substantially over multi-generational time spans, no such stable optimum can be achieved. Under such conditions natural selection is likely to favor not a fixed baseline salt preference, but rather a facultatively adjustable system whereby salt

preferences can be calibrated as a function of the probable future risk of sudden dehydration due to disease.

Because the environmental factors favoring pathogen proliferation and transfer are similar across multiple species of pathogens, infection by one species is often predictive of the risk of infection by other species as well. Likewise, due to the short generational span of pathogens relative to that of their mammalian hosts, a given strain may present in multiple guises during the course of a single host's lifetime. Both of these features of pathogen–host interaction lead to the same conclusion, namely that exposure to dehydration-causing illness is crudely predictive of the risk of equivalent events in the future. Natural selection has thus been able to effectively link salt preference to the likelihood of dehydration by using disease events as an index of future risk of infection, i.e., a simple heuristic that can be instantiated in the mechanism is 'increase salt preference in a ratchet-like fashion with each successive dehydrating illness.'

THE BENEFITS OF EARLY ACTIVATION AND SOCIAL REFERENCING

A mechanism that calibrates salt intake as a function of expectable disease risk can be expected to be particularly responsive early in development due to the benefits of equipping the organism with preferences appropriate to the local environment as soon as possible. Similarly, establishing such preferences early in life ensures that the appropriate level of prophylaxis is in place before increased individual foraging, enhanced mobility, and heightened social interaction all exacerbate exposure to pathogens. Exactly this kind of early calibration system exists in the form of sensitivity to both maternal dehydration during gestation and own dehydration during the early postnatal period. Early dehydration events provide information that is used to adjust the salt-preference system so as to appropriately balance the prophylactic benefits of salt consumption against the costs thereof.

The behavior of older conspecifics can provide a valuable source of information about prevailing disease risk, as older individuals have had greater opportunity to optimize their salt intake as a function of exposure to pathogens. Because many of the determinants of immunovulnerability are heritable (23,24), the ideal target for such socially mediated sampling of the environment is a close relative, as the two individuals will face similar disease risks. This explains the results described earlier wherein high maternal salt consumption increases offspring salt preferences – the mechanism that calibrates salt intake as a function of dehydration risk exploits not

only maternal disease events, but also maternal behavior as a source of information about that risk.

CONCLUSION

In sum, it is proposed that an adaptive mechanism underlies the increase in preference for salt that results from early exposure to dehydration and/or high maternal salt intake. This mechanism was produced by natural selection due to the protection that heightened salt intake provides against catastrophic sudden fluid loss, a benefit that outweighs the multiple costs attending this behavior. This hypothesis is imminently testable via two distinct avenues of investigation, as follows:

- In species evincing the patterned early establishment of salt preference discussed herein, compared to normal controls, individuals who either (a) have a history of early exposure to dehydration, or (b) are born to or reared by dams possessing a history of early exposure to dehydration should suffer reduced mortality and morbidity when challenged with sudden fluid loss due to disease or an artificial model thereof.
- Across species, the behavioral impact of early exposure to dehydration should vary as a function of the magnitude of variation in the prevalence of pathogens causing catastrophic fluid loss in the species' environment of evolutionary adaptedness. Because the magnitude of variation may be difficult to measure, this prediction can be crudely reformulated as stating that the salt preference calibration mechanism should be relatively sensitive in species whose typical circumstances are conducive to pathogen growth and transmission (i.e., species inhabiting tropical environments and/or having high population densities) and relatively insensitive, or even absent, in species whose typical circumstances are not conducive to pathogen growth and transmission (i.e., species inhabiting arid and cold or hot environments and/or having low population densities).

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