forced by the experimental observation that protection against salt-evoked alterations may be achieved by blockade of endothelin overexpression, for instance with the calcium channel blocker lacidipine[5].

Further studies are obviously required in order to better characterize the molecular mechanisms involved and our hope is that Denton's report will prompt several laboratories to analyse the various modifications evoked by high salt diet.

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. . . and salt resistance

To the editor — Denton and colleagues provide unequivocal evidence, that blood pressure in man's closest living relatives is affected by dietary salt intake[1]. To those familiar with the idea that salt sensitivity may indeed be the result of evolution in a low salt environment, this comes as no surprise. The real surprise, rather, comes from the fact that despite the high salt intake, blood pressure was not affected in five of the thirteen animals (38%). This study thus incidentally provides the first unequivocal evidence that salt resistance, often described in humans[6], also exists in primates. Advocates favoring a low salt intake for society in general, have often challenged the existence of this phenomenon considering it an artifact attributable to the relatively short duration of dietary-intervention studies in humans[7]. However, as pointed out by Denton *et al.*, even in societies on an excessively high-salt diet, the majority of individuals remain normotensive. If, as the authors suggest, the genetic basis for salt sensitivity may be the same in humans and chimpanzees, then by the same argument, the genetic basis for salt resistance should be similar in the two species. However, while the authors go to considerable lengths to provide plausible arguments for the evolution of salt sensitivity in a salt-poor environment, they fail to explain why salt resistance should have evolved under the same conditions. It certainly appears more than a coincidence, that salt-resistance should have evolved independently in the two species. If, as is more likely, the evolution of salt resistance does indeed date back to the common ancestors of humans and chimpanzee, the question remains how these "poor salt conservers" should have survived the salt-poor environment and maintained their salt resistance trait through the millennia of evolution.

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Denton replies — Godfraind and colleagues cite evidence that salt intake, at least in spontaneously hypertensive stroke-prone rats, may have pathological cardiovascular effects independent of those contingent on blood pressure rise. In relation to the 80 percent of the population who are seemingly not affected with high blood pressure, in their News & Views article,