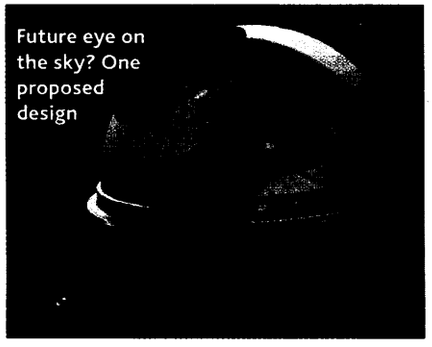
The economics of "overwhelmingly large" telescopes are discussed in a letter from the director of the "European Southern Observatory." A large number of letters were received about a feature article, "The (political) science of salt." The director of the National Heart, Lung, and Blood Institute found that the article "misrepresents both the process of public health policy-making and the data." A majority of the letters, however, were more positive. One reader wrote that the article "is the finest example of scientific journalism I have read."

Telescope Costs I read with interest the article by Robert Irion (News of the Week, 4 Sept., p. 1428) reporting on the "maximum-aperture telescope" (MAXAT) workshop, which was held on 28 and 29 August in Madison, Wisconsin.

Certainly as we go to more and more expensive facilities, such as the OWL ("overwhelmingly large") telescope or (as it is named in the United States) the MAXAT, they will become possible only through intercontinental cooperation and years of technological developments. I was glad, therefore, to see the beginning of an interest in such a facility in the United States.

In the same article, there are complimentary references to the European Southern Observatory (ESO) Very Large Telescope (VLT) program in Chile. I would like to clarify, however, that the ESO is not "pouring $800 million" into the VLT. The total cost of the program (including the capital investment and labor costs, as well



Future eye on the sky? One proposed design

as the costs for the first 3 years of operation) is currently estimated at $540 million. We are proud that this cost was reduced from the 1993 estimates by 7% (in response to financial restraints requested by member states) without a substantial reduction in scope.

Irion quotes as his source the article by Govert Shilling (News & Comment, 1 May, p. 670). That article, however, does not reflect ESO's budgetary numbers. Europe is not producing the VLT by a lavish expenditure of funds. In effect, the cost per telescope of VLT is competitive with that of most U.S. projects when one takes into account the actual content of the programs. This will be a good basis for future cooperation.

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Cleaning CJD-Contaminated Instruments Nigel Williams (News of the Week, 4 Sept., p. 1422) notes several critical concerns about the spread of epidemic Creutzfeldt-Jakob disease (CJD) involving surgery and infected peripheral tissues. In concluding, he cites potential transmissions from CJD-contaminated instruments, with the mandate that decontamination procedures for surgical instruments should be further assessed. Because of similar concerns, several years ago we identified a commercially available solution (with GdnSCN) that does not corrode fine stainless steel instruments. When instruments were exposed to this solution, CJD infectivity was reduced more than 100,000-fold in crude tissue (*1*), and none of the intracerebrally inoculated animals developed symptoms or lesions during 2 years of observation. This established procedure may be useful as a general and inexpensive method to reduce the inadvertent risk of CJD (or bovine spongiform encephalopathy abbatoire) transmission before test results from biopsy specimens can be analyzed.

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Salt Wars Gary Taubes's article about dietary sodium and hypertension (News Focus, 14 Aug., p. 898) depicts the National Heart, Lung, and Blood Institute (NHLBI) as sticking to a public policy position in favor of "universal salt reduction," in spite of published data questioning salt's effect on blood pressure.

Not so. NHLBI's recommendation for Americans to consume a moderate salt intake is based on a thorough, impartial, and continual review of the published science.

Far from setting the record straight, Taubes's article distorts an already confused issue. It misrepresents both the process of public health policy-making and the data. Instead of helping to pave the way toward good policy, it carves dangerous potholes.

The totality of information about sodium so far suggests that consuming a moderately reduced intake causes no harm and contributes to lowering blood pressure. The data also indicate that some individuals have a greater blood pressure response to sodium than others, but as yet science cannot identify them.

That is why NHLBI, along with many other health agencies, recommends a moderate intake of 2400 milligrams of sodium (or 6 grams of salt) per day for all Americans.

NHLBI funds a range of research to find out about factors that may affect the development of hypertension. As new findings from these and other studies appear, they are carefully reviewed. NHLBI has already held workshops on dietary sodium and will convene another in the coming months to examine the latest findings.

NHLBI sets its policies based on the science—all the science. And it will continue to ensure that good public health policy is based only on sound science, no matter how controversial the topic.

Claude Lenfant
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Response

Lenfant simply repeats what he and NHLBI have been saying since at least 1983, as my article pointed out. Nonetheless, the case then, as now, is that what constitutes "the totality of information" about sodium is a matter of much contention, and Lenfant's belief that "consuming a moderately reduced intake causes no harm and contributes to lowering blood pressure" may indeed be a minority opinion.

Gary Taubes

Taubes's article "The (political) science of salt" is the finest example of scientific journalism I have read. *Science* has done

us a service in publishing it, first, because a popular, thorough, and objective synopsis of this dauntingly complex literature was sorely needed, and second, because it has been written in such a way as to serve as a model for regarding all evidence in the field of dietary control of health. I wish that those responsible for shaping public health policy would read and understand what Taubes has taken the trouble to clarify for them.

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I was pleased to read Taubes's article on the salt-versus-hypertension controversy. This is one of several cases in which shaky epidemiology has ignored established physiology. The human kidney has a prodigious capacity to excrete sodium, just as it does water. I have not (yet) heard cries to limit water intake as a palliative for hypertension. The very fact that sodium levels in the blood are so tightly regulated should generate a certain skepticism about simple osmotic hypotheses for explaining hypertension. Gottschalk and Lassiter summarized it in 1980: "the rate of salt excretion is appropriate for the rate of salt ingestion even in advanced renal disease" (*1*). Conclusion: sodium intake is not now, nor has it ever been, a significant contributor to prevalent forms of chronic hypertension. Pass the pretzels, please.

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In his excellent article, Taubes notes that most of the large-scale epidemiologic studies and meta-analyses were conducted in general populations and did not show a beneficial effect of salt-lowering on blood pressure. However, high blood pressure is a polygenic disease and is therefore unlikely to respond to one specific treatment in a large population. It must be noted that salt reduction does lower blood pressure under conditions such as salt-sensitive hypertension, which is particularly common among African-Americans and which is an independent predictor of cardiovascular death (*1*). Salt-sensitive hypertensive patients are normotensive on a low sodium-diet and become hypertensive only when dietary salt is increased. Salt-sensitive hypertension in humans and animals is associated with endothelial dysfunction (*2*, *3*) resulting from reduced bioavailability of nitric oxide, a condition predisposing for coronary heart disease and stroke, which can be corrected by salt-lowering or pharmacotherapy (*2*, *3*). In line with L. K. Dahl *et al*'s suggestion that salt-sensitivity may be genetically linked (*4*), a cosegregation for the calcium-independent nitric oxide synthase (*NOS II*) gene with salt-sensitive hypertension has been reported (*5*), and recently a restriction length polymorphism of the gene has been identified (*6*). Recent *NOS II* data from our laboratory indicate that salt-sensitive hypertension is associated with a complete defect to increase *NOS II* activity and generation of the endogenous vasodilator nitric oxide in response to salt loading, further corroborating a pathobiological link between blood pressure and salt intake. (*7*). Further work will help to determine whether this abnormality also plays a role in humans and whether genetic screening will allow identifying patients at risk. Lowering of dietary salt remains an important factor of antihypertensive therapy in these patients.

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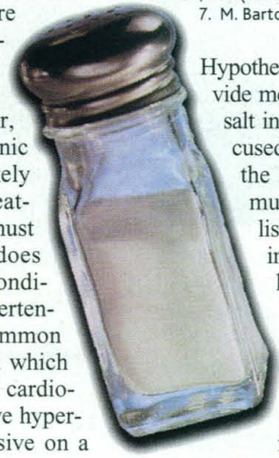

Hypotheses that have attempted to provide mechanistic links between a high salt intake and hypertension have focused on the relationships between the kidneys and vascular smooth muscles. The hallmark of established essential hypertension is an increase in the peripheral vascular resistance that to a large extent is determined by the tone of vascular smooth muscles. It was therefore logical to propose that in susceptible persons "salt sensitivity" arises from renal dysfunction in excreting a high salt load and that this is somehow relayed to vascular smooth muscles that

then increase in tone and consequently raise the blood pressure. The increase in the peripheral vascular tone must result from an increase in calcium load in vascular smooth muscle cells, because cytosolic calcium is the penultimate signal for vascular smooth muscle contraction. Hence, the effective use of calcium channel blockers as antihypertensive drugs. However, renal dysfunction in salt excretion might also be expressed by a rise in the calcium load in cells other than vascular smooth muscle cells. Because the cytosolic calcium is a second messenger for multiple cellular functions, it is possible that "salt sensitivity" is in fact a reflection of renal dysfunction in excreting a high salt load, expressed bv cellular abnormalities that exert deleterious effects on the cardiovascular system regardless of whether or not there is a rise in blood pressure.

The important question is not whether a high salt intake causes hypertension in some humans, but whether it increases (or for that matter diminishes) morbidity and mortality from any cause.

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Permit a physicist to make a few comments. Chemical potentials are typically proportional to the logarithm of concentrations. Most of the comparisons of sodium ingestions have been over a narrow range (between 6 and 9 grams daily, for example), which may be too small to show an effect, even if one is present. In order to demonstrate the existence of an effect, genuinely low sodium diets (less than 100 milligrams daily) should be used. But this was done, successfully, at least for severe hypertensives, by Kempner half a century ago. The open, and perhaps unanswerable, question is whether these results can be extrapolated to small reductions of sodium intake in the general population. This question resembles the similarly unanswered question of the validity of linear extrapolation of the effects of ionizing radiation to small doses. In the case of sodium ingestion, because of the chemical potential argument, a plausible hypothesis is that effects are proportional to the logarithm of the ingestion rather than to the ingestion itself.

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Quantum Computing

In April 1996, 5 milliliters of di-bromothyophene, a molecule containing two coupled hydrogen atoms, were placed inside a 500-megahertz magnet. The sample was then subjected to a sequence of radio frequencies (rf) and gradient pulses, after which the collective behavior of the molecules of the sample was that of a quantum system in a pure state. Another sequence of rf pulses implemented the first quantum logic gate produced by nuclear magnetic resonance (NMR). The nuclei of the hydrogen atoms acted as two quantum bits, and thus the first implementation of a two-qubit NMR quantum computer came to be. This experiment was performed at the Francis Bitter Magnet Laboratory at Massachusetts Institute of Technology by David Cory, Tim Havel, and me. We wrote up our exciting results and submitted them to Tom Toffoli of Boston University, chair of Physcomp '96, where the paper appeared on the conference Web site in July and in the proceedings in November 1996 (*1*).

In a Research Commentary, "Fast searches with nuclear magnetic resonance computers" (*Science*'s Compass, 10 Apr., p. 229), Jonathan A. Jones states that "two different two-qubit NMR computers have been built: one by Chuang and co-workers [he cites a paper then in press (*2*)] . . . and one by my research group in Oxford . . . [he cites work then in preparation (*3*)]." Although the *Physcomp '96* paper was mentioned (but not cited) by Gary Taubes in a Research News article ("Putting a quantum computer to work in a cup of coffee," 17 Jan. 1997, p. 307), this first implementation was not cited in a *Science* report ("Bulk spin-resonance quantum computation" by N. A. Gershenfeld and I. L. Chuang, 17 Jan. 1997, p. 350) or technical comments ("The usefulness of NMR quantum computing" by W. S. Warren, 12 Sept. 1997, p. 1688; response by N. Gershenfeld and I. Chuang, p. 1689) concerning quantum computation by NMR.

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Response

Fahmy is quite right to draw attention to his seminal work in NMR quantum computing. His pioneering studies of 2,3-dibromothiophene, conducted with Cory and Havel, included the first experimental demonstrations of effective pure states and quantum logic gates in NMR. This work was originally described in the proceedings of *Physcomp '96* (*1*) and was swiftly followed by a detailed theoretical paper