rockets is technical—the need to carry on board both fuel and oxygen. That imposes an inescapable weight burden on rockets of any kind and a minimum cost of at least $5000 per pound to put something in space. The National Aerospace Plane (NASP), a third generation launcher, will nearly eliminate that oxygen penalty."

Keyworth and Abell are quite right. NASP will use the oxygen in the atmosphere to accelerate to near orbital speeds. While there will be savings with regard to the cost of carrying oxygen, however, other costs may offset these savings.

If one achieves high speeds in the atmosphere, one suffers a drag proportional to the density times the square of the speed. In order to overcome the enormous atmospheric drag of high speeds, enormous thrust is needed. The oxygen in the atmosphere needed to provide the thrust that overcomes the drag is also proportional to the density. Thus, higher altitudes mean not only less drag but less thrust. This thrust, of course, requires a large engine and additional fuel. Indeed, I don't believe anyone has demonstrated a high Mach–number (greater than 10) scramjet with a thrust greater than its drag. The larger engine and additional fuel required may offset the savings in oxygen weight, but one must also consider the weight penalties of thermal protection and structure required by the high heating rates and dynamic loads.

It is, therefore, not clear, as Keyworth and Abell state, that the NASP concept is an important one for launch vehicles. Nor does it seem likely that the NASP will provide efficient and effective high-speed transport.

Funds for the NASP program could be better used to provide the research for and the development of a Twenty-First Century supersonic transport. An aggressive program by NASA, and by aircraft and engine manufacturers, could provide a supersonic transport that would be economic in operation on long-distance ocean routes. While it is not likely that such a transport would repay any of its research and development costs, it would provide valuable improvements to civilian transport.

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Response: The design of the scramjet should enable it to provide nearly constant power over a wide range of altitudes. This is done by changes in the engine configuration in different speed regimes so that oxygen can be scooped up with optimal efficiency. The object of the current scramjet development program is to optimize the thrust of the engine at high altitudes. How to achieve Mach numbers greater than 10 is one of the program's central objectives. The simulations and measurements over the past year have so far strongly supported the premise that such high Mach numbers will be achieved. The central premise of our argument is that the National Aerospace Plane, unlike a rocket, can dramatically offset the oxidizer burden that contributes to the high cost of space access. The precise extent of that offset is the objective of the X-30 research program.

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ADA Deficiency Treatment

The article "ADA deficiency: A prime candidate" by Barbara J. Culliton (News & Comment, 10 Nov., p. 751) refers to treatment of immunodeficiency resulting from deficiency of adenosine deaminase (ADA) with polyethylene glycol–modified ADA (PEG-ADA). The article states that "after a few doses its effectiveness is often lost, for reasons that are not understood." This assessment is inaccurate.

Trials of PEG-ADA have been conducted in 12 patients for from 5 to 44 months (weekly dosing; 20 to 190 weekly injections per patient). Treatment with PEG-ADA has corrected the metabolic abnormalities caused by ADA deficiency in every patient, as judged by maintenance of plasma ADA at a level sufficient to eliminate deoxyadenosine triphosphate (dATP) in red cells, the biochemical goal of therapy. Objective improvement in tests of immune function has occurred in 11 patients, and clinical improvement has occurred in all 12. A misunderstanding seems to have arisen concerning information about one patient, in whom enhanced clearance of PEG-ADA occurred after 4 months due to development of antibody to ADA. The patient's treatment was interrupted for 8 weeks, but then was resumed. Immune tolerance to PEG-ADA was induced and this patient has now been receiving PEG-ADA for 7.5 months, with restoration of immune function and clinical improvement.

ADA deficiency is a very rare disorder, and the degree of immune dysfunction it causes is variable. As with other therapy for the type of immunodeficiency caused by ADA deficiency, for example, bone marrow transplantation, it may take years to assess accurately the degree of long-term clinical benefit from PEG-ADA. However, we are encouraged by results to date and feel that PEG-ADA has already been shown to be safe and effective. It is at this time the only example of effective enzyme replacement therapy for an inherited metabolic disease.

Enzyme replacement is a treatment, not a cure. We are encouraged by the efforts of Michael Blaese, his colleagues, and others to develop "gene replacement," which can, ideally, cure genetic diseases. Nevertheless, until safe and effective gene therapy is a reality, we plan to continue our efforts to develop enzyme replacement therapy for other metabolic diseases.

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Erratum: In the report "X-ray diffraction to 302 gigapascals: High-pressure crystal structure of cesium iodide" by H. K. Mao *et al.* (3 Nov., p. 649), reference 10, to a paper by R. Reichlin *et al.* [*Phys. Rev. Lett.* **56**, 2858 (1986)], was incorrectly numbered (*9*) in the text (p. 649, column 3, line 1; p. 650, column 1, line 49).