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Bryozoans are a major component of rocky bottom subtidal marine communities. Pictured here is an orange and brown cyclostome bryozoan colony of *Heteropora pacifica* along with articulated, more delicate crissid bryozoans, the pink branched hydrocoral *Allopora californica*, coralline and fleshy red algae, sponges, and compound ascidian tunicates. The phylogeny of the lophophorates (bryozoans, brachiopods, and phoronid worms) is discussed on page 1641. [Photo: James G. Morin at the "Pinnacles" off Seventeen Mile Drive, near Monterey, California, at a depth of 25 meters]

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The Crystal Ball and the Trumpet Call

In the 1860s Abraham Lincoln's commissioner of patents recommended that he plan to close the commission in a few years. The reason: the rate of discovery had become so great that everything that needed to be discovered would have been discovered by then. The patent commission would have no business.

In this issue of Science a number of scientists who are leaders in their disciplines were asked to cast off inhibitions and predict the future. They have done so in exemplary fashion. They clearly do not agree with that commissioner of patents of yesteryear. Great discoveries with great import for the future of science and the well-being of the citizen are in the offing. That we have come so far so fast is not an indication that we have saturated the discovery market, but rather that new discoveries will come even faster.

These scientists indicate areas, from room-temperature superconductors and rational drug design to whole new approaches to economics and social science, that are ripe for development in the future. They are not asked to be, and should not be judged on being, practical or conservative and safe, but rather asked to be adventurous but sound—no perpetual motion machines, but plenty of extrapolations beyond the state of the art. This editor was personally disappointed that none of them predicted an "abolish sleep" pill. No one knows how much sleep we really need—some people sleep very little and are perfectly healthy, and a pill to get sleep over quickly and release many more hours per day for productive activity would seem a boon to harassed people. Despite this disappointment, the list of new ideas and vast new fields is impressive and reading it is enjoyable. It is a trumpet call to get on with the new challenges and surmount the new obstacles.

It could be asked, "Is this all pie in the sky—the unlikely guesses of some hallucinating scientists?" On the basis of past history, these guesses are very likely to be prophetic. On several occasions the National Academy of Sciences (NAS) has asked leading scientists of various disciplines to outline the futures of their professions. The Westheimer report of 1965, *Chemistry: Opportunities and Needs*, and the Pimentel report of 1970, *Opportunities in Chemistry*, predicted the progress of chemistry. Leading physicists speculated about advances in their discipline in the Fake report, *Physics: Survey and Outlook*, in 1966, and later in the Brinkman report of 1986, *Physics Through the 1990s*. In 1970, Philip Handler, chairman of the NAS Survey Committee on the Life Sciences, edited opinions of prominent biologists on future directions of research in *Biology and the Future of Man*. The predictions made in these excellent volumes have, for the most part, been fulfilled—except that the most revolutionary and unexpected findings were not predicted. Physicists did not suggest the transistor and the laser, chemists missed buckyballs, biologists did not foresee recombinant DNA. Thus, history would suggest that scientists tend to underestimate the future.

The list in this issue was not designed and does not pretend to be comprehensive. It was an experiment to see if leaders in the field would be willing to go out on a limb of crystal ball gazing. Future issues can deal with other areas and other authors. For this moment, however, the responses show the enormous reach of modern science and its potential for improving the lot of humanity. Budget-cutters in the world's governments should look at this list and ask themselves whether they wish to be the person who prevented the development of a defense against drug-resistant bacteria or the building of superconducting medical machines, or closed the exploration of the interior of the Earth.

For the generation now carrying out research, it is worth acknowledging the altruism of their seniors who have, with great restraint, not solved all the problems, but have left a generous share for those who follow. We can expect that the new generation will act with the enthusiasm and resourcefulness of their predecessors and that those holding the power of the purse will allow them to do so.

Daniel E. Koshland Jr.
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Malaria Vaccine

As one who, in 1976 with J. B. Jensen, provided methods for the continuous culture of Plasmodium falciparum (1), methods which have led to and greatly facilitated not only the development of SPf66 but also other work on an erythrocytic-stage vaccine for malaria, I would like to comment on the “dilemma” raised by the malaria vaccine (John Maurice, News & Comment, 20 Jan., p. 320). I see no serious dilemma. It is clear that SPf66 is sufficiently effective to warrant additional and more extensive trials. It is good to note that such a large trial is already in the planning stage for 1995 in Tanzania. Meanwhile, results from several trials now in progress are expected to be in during this year. If they are disappointing, the clamor for a million doses will cease. If they are good, there will be little reason to restrict development as long as the funds for it can be provided. Of course, there is a lot we do not know, but we will be learning much in the next few years, with careful follow-up of those already vaccinated. Of special interest might be trials combining SPf66 with the use of insecticide-impregnated bed nets for the protection of young children, as suggested in a recent review by M. Tanner et al. (2).

If Manuel Patarroyo’s work had been received with less criticism and more cooperation, perhaps we would be 5 years ahead of where we are now. It was, it seemed to me, an ideal example of how basic science should be applied to problems in developing countries. The basic work—culture of the parasites and use of the methods of Bruce Merrifield for peptide synthesis—was done at the Rockefeller University. A creative young scientist then applied these methods to a major medical problem in his country. He had support from his government, excellent laboratory facilities, and lots of drive and motivation. He furthermore had the special advantage of the availability of large numbers of Aotus monkeys in which to do his preliminary testing of some of the many antigens of Pl. falciparum. He came up with a combination of three peptides that together gave protection in monkeys. He then did something both clever and original—he took small fragments of these peptides and polymerized them into a synthetic polypeptide with a molecular weight of about 20,000. There was accordingly no need for him to use a carrier protein (the method others were using). In his first trials in humans, he showed great courage. He chose a cutoff point for treatment that turned out to be safe, yet provided for a significant result.

Very likely there will be a better malaria vaccine than SPf66. Meanwhile, we ought to be using what we have. I am reminded of a conversation with Tom Rivers many years ago. He was relating how he had to decide whether to go ahead with large-scale use of the Salk-killed vaccine for poliomyelitis or wait for the live vaccine, which might be better. He did not wait.

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References


Detecting Alzheimer’s Disease

The report “A potential noninvasive neurobiological test for Alzheimer’s disease” by Leonard F. M. Scinto et al. (11 Nov., p. 1051) describes patients with Alzheimer’s disease who exhibited marked hypersensitivity in pupil dilation to a dilute solution of tropicamide. As ophthalmologists who routinely dilate patients’ eyes with tropicamide, we have observed extremely variable pupil response to dilating agents. We repeated the protocol described in the report with 13 healthy subjects with a mean age of 32 years, and with no family history of Alzheimer’s disease. The pupils of these subjects diluted (paired t test, P < 0.0005, data not shown) in a fashion similar to that of patients with Alzheimer’s disease, as reported by Scinto et al.

While dilute tropicamide solution may be investigated as a screening test for Alzheimer’s disease in elderly patients, we urge caution if it is used for this purpose in young, healthy adults.

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LETTERS
We recently studied 20 elderly persons from a community sample of psychiatric patients in London. Ten were diagnosed as having probable Alzheimer’s disease and 10 as having multi-infarct dementia (MID) by clinical criteria and by computerized axial tomography. Patients who were using any medication (such as anticholinergic or opioid drugs) that might interfere with pupillary response were excluded. We used a methodology (1) similar to that used by Scinto et al. There were no significant differences between the groups for age, sex, or cognitive score. While the group with Alzheimer’s disease reacted almost identically to that in the study by Scinto et al., the reaction of the group with MID was indistinguishable from that of the Alzheimer’s disease group \((P = 0.72, \text{ data not shown})\). Therefore, it seems that anticholinergic drops may not enable one to distinguish between different forms of dementia. Although the posterior part of the eye is a central nervous system organ, pupillary control is a function of the peripheral nervous system. It seems possible that concurrent administration of systemic drugs to some of the patients in the study by Scinto et al. affected those results.

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References  

We studied 20 patients with Alzheimer’s disease for pupillary response to topically administered dilute pilocarpine (0.125%), a cholinergic agonist (1). We analyzed changes in pupillary size using three methods. The average median and mean pupillary size differences in the group with Alzheimer’s disease were compared to those of corresponding eyes in a control group matched for age and iris pigmentation (a relevant variable, as lightly pigmented irides often respond more to topically administered agents). To take into account the initial pupil size, we also calculated the ratio of the postdrug pupil size to predrug pupil size; mean ratios in the group with Alzheimer’s disease were compared to those of corresponding eyes in the control group. Last, a 13-point scoring system was developed to combine analyses of changes in pupillary response and take into account the inherent imprecision in pupillary eye measurements.

Our patients with Alzheimer’s disease demonstrated hypersensitivity in pupillary miosis induced by a cholinergic agonist, dilute pilocarpine. Induced miosis was more than twofold greater in the group with Alzheimer’s disease than in the control group. Our pretesting evaluation of patients and normal subjects excluded Adie’s pupils as a possible source of confounding error, and demonstrated normal tear lakes, normal tear break-up time, and normal corneal sensitivity in both groups, minimizing (although not eliminating) the likelihood that underlying corneal pathology might account for increased permeability of topically administered medication in patients with Alzheimer’s disease.

Our results complement those of Scinto et al., and yet we see two possible interpretations of both studies. One is that the two studies seem to support each other and offer evidence for upregulation of cholinergic receptors within the iris of the patient with Alzheimer’s disease; there was exaggerated mydriasis to a cholinergic antagonist (tropicamide) and exaggerated miosis to a cholinergic agonist (pilocarpine). Although the pharmacology of the agents was different, the relevant receptor involved would be the same. Another interpretation, however, is that both
studies might merely illustrate increased corneal penetration of any topical agent in the presumed Alzheimer’s patient.

There may be a range of induced pupillary mydriasis, or miosis, or both, that could be used as a marker for individuals with Alzheimer’s disease. Such pharmacologic investigations need to be pursued, but they should take into account matching control populations for iris pigmentation, scoring the change in anisocoria after topical administration of an agent to one eye as the true drug-induced effect, and using the second eye as a control to take into account elements such as fatigue before concluding that upregulation of cholinergic receptors in the iris allows for recognition of the effects of plaques and tangles.

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In a 1981 pupillometric study, we found a supersensitive miotic response to ocular administration of the muscarinic agonist pilocarpine (0.125%) in patients with Alzheimer’s dementia as compared with normal or mildly impaired subjects matched for age, sex, race, and iris pigmentation (1). The instrument used was identical and the methodology comparable to those in the report by Scinto et al. We chose not to follow up on the pilocarpine results because of difficulties associated with controlling for potentially variable corneal penetration, aqueous fluid turnover, and iris pigmentation across different subject groups. Furthermore, we did find a statistically significant difference (Student’s t test, P < 0.05) in pilocarpine miosis between brown-eyed and nonbrown-eyed (hazel, blue, or green) subjects independent of the above patient-control difference.

Data from a study by Sacks and Smith (2) suggest that eye color may also modulate the mydriatic response to tropicamide, the muscarinic antagonist studied by Scinto et al. Sacks and Smith found that the mydriatic responses to tropicamide appeared to be greater in Down syndrome subjects with blue and hazel eyes than in those with brown eyes. In the study of Scinto et al. the sizes of some of the subgroups tested were relatively small (for example, four patients with non-Alzheimer’s type dementia were studied), so suggestions about the specificity of their findings to Alzheimer’s disease seem premature in the absence of data from subject groups matched for eye color.

We would also like to point to an apparent pharmacological anomaly, namely that Alzheimer’s disease is associated with iris cholinergic receptor supersensitivity to both an agonist (pilocarpine) and an antagonist (tropicamide). One would expect to find agonist supersensitivity with antagonist subsensitivity or vice versa. At this point, we suggest that the most parsimonious explanation of our data as well as the findings of Scinto et al. is that of a nonspecific corneal epithelial tissue degeneration that permits abnormal penetration of drugs across the cornea in Alzheimer’s disease. Even if the reported difference between patients with Alzheimer’s disease and normal subjects proves to be a result of nonpharmacological factors, it may still represent a potentially valuable diagnostic tool.

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95 (suppl.), 134 (September 1988).

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Response: We thank our colleagues for their comments about our report of an enhanced pupillary response to dilute tropicamide on the part of patients with probable Alzheimer's disease. Several letters suggest that our finding may have been a result of an increased permeability of the cornea in patients with probable Alzheimer's disease. We agree that a change in permeability is one possible mechanism.

Marx et al. discuss the potential variability in dilation response. They calculated the change in pupil diameter by subtracting the values for the untreated eyes from the treated eyes. In contrast, our measurements were calculated as the increase in diameter over the pretreatment baseline of the same eye. When our method of data analysis was applied to the data of Marx et al., the response of their middle-aged, healthy control group was more similar to that of our elderly, healthy control group, allowing age differences, than to that of our patients with probable Alzheimer's disease. Apparent differences in findings might be a result of the type and resolution of the measurement system used, the preparation of the agent used to dilate, or the conditions under which measurements are made. Our experiments suggest that young subjects (20 to 40 years of age) show a minimal response (less than or equal to 5% over baseline measurement of the treated eye) to tropicamide in concentrations of 0.25% and 0.01% (data not shown).

Treloar et al. report that, using methods similar to ours, they were unable to distinguish patients with a clinical diagnosis of MID from patients with a diagnosis of probable Alzheimer's disease. Patients clinically diagnosed with vascular dementia, when autopsied, are often (40 to 50%) found to have sufficient plaques and tangles to meet the pathological criteria for Alzheimer's disease (1). Katz and Pomara and Sitaram report finding a hypersensitivity to cholinergic agents in the pupil response of patients with Alzheimer's disease. Although some of these studies used pilocarpine, a cholinergic agonist, they underscore the potential benefits of the pupil dilation response as an assay for degenerative dementia.

Some investigators have suggested that screening for drugs that affect pupillary response could explain the differences between their results and ours. We screened our subjects for use of medications that have known pupillary effects, and other medication use in all of our study groups was similar.

In light of comments about the methodology in our report, we offer the following clarifications. (i) We measured baseline pupil diameter in both eyes before any intervention with a control or drug agent. (ii) Fatigue, anxiety, or hyper-arousal were carefully monitored during the time of testing and no differences were noted between patients and control subjects. (iii) Both patients and control subjects were tested at different times of the day with no systematic bias in time of test for any group. (iv) No group had a systematic bias to a particular iris color. (v) Over the course of measurement intervals, there was no significant difference in the pupil diameter of the untreated eye for patients with Alzheimer's disease as opposed to normal control subjects (Kruskal-Wallis pairwise multisample test, P = 0.15). (vi) A random examination of the raw data revealed no difference in blink rate for patients or controls during measurement periods. (vii) Although our results were reported in terms of percentage change over baseline, the absolute differences over baseline yield the same curves as appear in the figures in our report.

We did not report a mechanism to ex-
plain the finding. The intent of our study was to determine if patients with a clinical diagnosis of probable Alzheimer's disease could be distinguished from healthy controls on the basis of changes in pupil diameter to the topical administration of a dilute solution of tropicamide.

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References

Corrections and Clarifications
A correction (25 Nov., p. 1308) for the caption that accompanied photographs in the article (Research News, 11 Nov., p. 973) about the report by Leonard F. M. Scinto et al. "A potential noninvasive neurobiological test for Alzheimer's disease" (11 Nov., p. 1051), was itself incorrect. The photos showed the undilated and dilated eye of a normal 36-year-old member of the Scinto group, not the undilated and dilated eye of an Alzheimer's patient, as the correction stated. The error was the result of a misunderstanding between Science and the Brigham and Women's Hospital new office, which took the photos. A patient photo was not supplied, Scinto says, because subjects were not then being studied and hospital policy does not permit treatment of a patient, even with dilute eyedrops, for the purpose of taking a photo. As the photo subject was a normal individual, Scinto also notes that the dilation was achieved by a 1% solution of tropicamide (a standard dose), rather than the 0.01% used in the trial. Science regrets the error.

The News & Comment article "Brookhaven prepares for boron trials" (17 Feb., p. 956) by Andrew Lawler omitted the information that terminally ill patients who have undergone conventional treatments, including radiation and chemotherapy, are not eligible for the boron trials expected to begin this month at Brookhaven National Laboratory.

In a correction that appeared in the Book Reviews section of 13 January (p. 267), Solomon W. Golomb's name was misspelled.

In figure 5 (p. 1372) of the report "Activation and regeneration of rhodopsin in the insect visual cycle" by A. Kiselev and S. Subramaniam (25 Nov. 1994, p. 1369), the labels "thermally unstable" and "thermally stable" were inadvertently interchanged.

In the report "A three-dimensional model for the hammerhead ribozyme based on fluorescence measurements" by T. Tuschl et al. (4 Nov. 1994, p. 785), the text of lines 28 through 30 in column 3 on page 785 should have read, "... we located 5-carboxyfluorescein at (d, -29.5°, L, -3.75 Å) and 5-carboxytetramethyl-
rhodamine at (a, -29.5° - Δ, -3.75 Å)." In the same report, the second line of equation 1 in the legend to figure 3 was incorrectly printed. The correct equation appears below.

\[ E = \left\{ 1 + \left[ \frac{(2.81 Å(N - 1)+L)^2 + a^2 + d^2}{2 \cdot a \cdot d \cdot \cos(32.7° \cdot (N - 1) + \Delta)} \right]^{1/2} \right\}^{1/2} \]

Equation 1 in note 17 of the same report was also incorrectly printed. The correct equation appears below.

\[ E = 1 + \left[ \frac{(d^2 + a^2 + d^2) \cdot \cos(32.7° \cdot (N - 1) + \Delta)}{2 \cdot a \cdot d \cdot \cos(32.7° \cdot (N - 1) + \Delta)} \right]^{1/2} \]
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Katherine Livingston

Books Received

Abell’s Exploration of the Universe. David Morrison, Sidney Wolff, and Andrew Fraknoi. 7th ed. Saunders (Harcourt Brace), Fort Worth, TX, 1994. xiv, 622 pp., illus. + plates. $52.


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