metric and oceanographic conditions swept channel areas clean and, in part, eroded them. We suggest that the ridges flanking valleys and canyons on the mid- and lower slope were probably formed by the overbank deposition of material being transported downslope. Although the ridges do not conform to the classical model of levees, thinning away from primary channels on deep-sea fans (*10*), deposition from closely spaced valleys on the continental slope would not have allowed complete development to occur. Slumping played a greater role among processes extending and eroding canyons in the late Pleistocene than it does now. As the sea reached present levels, deposition slowed, conditions became more stable, and Holocene deposition took place at low rates, mantling the Pleistocene deposits with a thin veneer of fine-grained sediment.

On the basis of our study of closely spaced seismic-reflection profiles, we conclude that the continental slope between Lindenkohl and South Toms canyons has a complex surface formed largely by Pleistocene-aged deposits of variable thickness and complex structure. Tertiary sediments crop out on large areas of the lower slope. Slumping may have been an important process extending submarine canyons and valleys during the Pleistocene and perhaps early Holocene, but it appears to be less active currently. Small slump features are present in the heads and in places along the flanks of canyons or valleys, but the present subaqueous landscape in this area is primarily relict, and present-day landscape modification appears to be active only at low rates. Where identified, slumping occurs within deposits of Quaternary age. Tertiary sediments are flat-lying and undisturbed and show no evidence of slumps or slides. We should emphasize that mapping and geotechnical testing is necessary to determine the potential hazard of any specific area intended for exploitation. Our findings should be applied with prudence, and extrapolation to other areas is open to question.

JAMES M. ROBB
JOHN C. HAMPSON, JR.
DAVID C. TWICHELL
*U.S. Geological Survey,*
*Woods Hole, Massachusetts 02543*

### References and Notes

1. E. Uchupi, *Deep-Sea Res.* **14**, 635 (1967); J. C. McIlvaine and D. A. Ross, *J. Sediment. Petrol.* **49**, 563 (1979); R. W. Embley and R. Jacobi, *Mar. Geotechnol.* **2**, 205 (1977); H. J. Knebel and B. Carson, *Mar. Geol.* **29**, 221 (1979); G. H. Keller *et al.*, in *Geology of Continental Slopes*, L. J. Doyle and O. H. Pilkey, Eds. (Special Publication 27, Society of Economic Paleontologists and Mineralogists, Tulsa, 1979), p. 131.
2. B. A. McGregor and R. H. Bennett, *Mar. Geol.* **33**, 163 (1979).
3. R. A. Slater and D. C. Twichell, dives in the submersible *Diaphus*, August 1978 (personal communication).
4. M. L. Somers, R. M. Carson, J. A. Revie, R. H. Edge, B. J. Barrow, A. G. Andrews, in *Oceanology International 78* (1978), Technical Session J, p. 16.
5. C. W. Poag, *Bull. Am. Assoc. Pet. Geol.* **63**, 1452 (1979).
6. C. W. Poag, personal communication (1979); USGS piston cores taken August and September 1979.
7. J. C. Hathaway, C. W. Poag, P. C. Valentine, R. E. Miller, D. M. Schultz, F. T. Manheim, F. A. Kohout, M. H. Bothner, D. A. Sangrey, *Science* **206**, 515 (1979); L. J. Doyle, O. H. Pilkey, C. C. Woo, in *Geology of Continental Slopes*, L. J. Doyle and O. H. Pilkey, Eds. (Special Publication 27, Society of Economic Paleontologists and Mineralogists, Tulsa, Okla., 1979), pp. 119–129.
8. R. A. Slater, D. C. Twichell, C. V. G. Phipps, J. A. Aaron, *Bull. Am. Assoc. Pet. Geol.* **63**, 529 (1979); W. P. Dillon and H. B. Zimmerman, *J. Sediment. Petrol.* **40**, 524 (1970); W. B. F. Ryan, M. B. Cita, E. L. Miller, D. Hanselman, W. D. Nesteroff, B. Hecker, M. Nibbelink, *Oceanol. Acta* **1**, 233 (1978).
9. C. D. Hollister *et al.*, *Init. Rep. Deep Sea Drill. Proj.* **11**, 351 (1972).
10. H. W. Menard, Jr., *Bull. Am. Assoc. Pet. Geol.* **39**, 236 (1955); W. R. Normark, in *Modern and Ancient Geosynclinal Sedimentation*, R. H. Dott, Jr., and R. H. Shaver, Eds. (Special Publication 19, Society of Economic Paleontologists and Mineralogists, Tulsa, Okla., 1974), pp. 56–68.
11. Funding for this project was provided by the Bureau of Land Management under Memoranda of Understanding AA551-MU8-21 and AA551-MU9-4 between the USGS and the Bureau of Land Management. The seismic profiles were collected aboard R.V. *Iselin* in 1978 and R.V. *Gillis* in 1979. We thank W. P. Dillon, J. S. Booth, H. J. Knebel, C. W. Poag, and B. McGregor-Stubblefield who read the manuscript and made valuable suggestions. Responsibility for the interpretations remains with the authors.

8 May 1980; revised 7 November 1980

## Azidoatrazine: Photoaffinity Label for the Site of Triazine Herbicide Action in Chloroplasts

Abstract. *Binding of the 4-azido analog of the herbicide atrazine to pea chloroplast membranes was compared with that of atrazine. When [$^{14}$C]azidoatrazine was treated with 300-nanometer ultraviolet light in situ, reversibility of binding was lost in proportion to the duration of irradiation. Sodium dodecyl sulfate–polyacrylamide gel electrophoresis of chloroplast membranes irradiated in the presence of [$^{14}$C]-azidoatrazine indicated radioactivity in only one region, corresponding to a protein with a molecular weight of approximately 32,000. Azidoatrazine is a photoaffinity reagent for the triazine binding site in chloroplasts and serves as a label to identify this site, which may be the apoprotein of the secondary electron acceptor in photosystem II.*

Many commercial herbicides act as photosynthetic inhibitors. Some of them, including the chemical groups of ureas, amides, triazines, triazinones, pyridazinones, carbamates, and nitrophenols block photosystem II-dependent Hill reactions. These compounds appear to act at the second electron carrier on the reducing side of photosystem II (*1*). This carrier, called B, is presumed to be a quinone molecule that is bound to a specific polypeptide in the reaction center complex. The carrier, B, is reduced by electrons from the primary electron acceptor, Q, and, in turn, donates electrons to the plastoquinone pool that connects photosystems II and I. In herbicide-inhibited chloroplasts, Q is functional as an electron carrier, but B is no longer active as a functional electron acceptor.

The identity of the apoprotein of B is not known. Binding to chloroplasts of radioactively labeled herbicides, especially atrazine and diuron, has been studied (*2*, *3*). This binding is reversible, and all photosystem II inhibitors appear to compete for the same site. The binding affinity of herbicides for this site is closely correlated with inhibition of photosystem II. These results, together with comparisons of chloroplasts from triazine-resistant and triazine-susceptible biotypes of the same species, indicate that the herbicide binding site is probably the apoprotein of B (*1*).

Photoaffinity labeling is a method by which the herbicide binding site may be specifically identified. This technique has recently become important for identifying complex biological receptors (*4*). A photolabile reagent is anchored to the macromolecule; photolysis of the complex then leads to the generation of a highly reactive species that, by reacting rapidly with its immediate environment, covalently labels the macromolecule or specifically labels the active site.

I now report that a photolabile azido analog of the herbicide atrazine competes with atrazine at the herbicide binding site in pea chloroplasts and serves as a photoaffinity label for that site. Alaska peas (*Pisum sativum* L. cultivar Alaska; Burpee) were grown on vermiculite for 7 to 8 days under Cool White fluorescent lights during an 11:13 light-dark photoperiod. Chloroplasts were prepared and chlorophyll was determined (*3*). Azidoatrazine (4-azido-2-isopropylamino-6-ethylamino-*s*-triazine) was synthesized from atrazine (4-chloro-2-isopropyl-


0036-8075/81/0227-0937$01.00/0  

amino-6-ethylamino-*s*-triazine) by formation of the trimethylamine salt and reaction with sodium azide according to the general procedure for azidotriazines (*5*). [$^{14}$C]Azidoatrazine was synthesized from [$^{14}$C]atrazine (Pathfinder Laboratory, St. Louis, Missouri) by the same procedure. Both starting material and product had specific activities of 8.3 mCi/mmole.

Binding experiments were carried out in 1 m*M* *N*-[tris(hydroxymethyl)methyl] glycine, *p*H 7.8, containing 200 m*M* sorbitol, 10 m*M* $MgCl_2$, and 10 m*M* NaCl, at a chlorophyll concentration of 50 $\mu$g/ml. Duplicate 1.8-ml portions of the chloroplast suspension were adjusted to 0.2 $\mu M$ in either [$^{14}$C]azidoatrazine or [$^{14}$C]atrazine. Nonradioactive compound or solvent (1 percent MeOH) was added, and after 30 minutes at 4°C the suspension was centrifuged for 15 minutes at 20,000 rev/min. The end of each centrifuge tube containing the sedimented chloroplasts was excised; the resultant pellet and tube end were combusted with a sample oxidizer (Packard, model 306), and the radioactivity was measured by liquid scintillation spectrometry. All binding assays were carried out at 0° to 4°C.

Photolysis of the azidoatrazine was carried out in a quartz erlenmeyer flask placed in a Rayonet photochemical reactor (Southern New England Ultraviolet Co., Middletown, Connecticut) equipped with 16 reactor lamps (3000 Å) providing a fluence rate of 47.6 W/m$^2$. The samples were kept on ice during the ultraviolet treatment.

The binding of [$^{14}$C]azidoatrazine to pea chloroplast membranes in the dark can be reversed by higher concentrations of nonradioactive herbicide. The amount of this reversal is referred to as specific binding (*6*). The binding that remains after addition of saturating concentrations of herbicide (10 $\mu M$) is nonspecific, attributable at least in part to radioactive material trapped in the free space in the pellet.

Chloroplasts were mixed with 0.2 $\mu M$ [$^{14}$C]azidoatrazine and irradiated with 300-nm ultraviolet light for various periods (Table 1). As the duration of irradiation increased, the reversibility of the binding by 10 $\mu M$ nonradioactive azidoatrazine decreased. After 30 minutes of irradiation, only 21 percent of the saturable binding of the dark control remained. The effect of ultraviolet irradiation was not on the total amount of binding; the total binding was approximately the same under all conditions. Rather, the ultraviolet light significantly pre-

Table 1. Ultraviolet inhibition of bound azidoatrazine exchangeability. Chloroplasts were mixed with 0.2 $\mu M$ [$^{14}$C]azidoatrazine and irradiated with 300-nm ultraviolet light for the periods indicated. Then solvent or 10 $\mu M$ [$^{12}$C]azidoatrazine was added, the chloroplasts were centrifuged, and radioactivity was determined in each pellet. Specific binding activity is the amount of radioactive ligand displaced by the nonradioactive ligand. All assays were carried out in duplicate at 0° to 4°C, and values are given as means ± standard error.

| Ultraviolet treatment (minutes) | Addition | Pellet (dpm) | Specific binding | |
|---|---|---|---|---|
| | | | Displaced (dpm) | Percent of control |
| 0 (dark control) | | 3452 ± 32 | | |
| | 10 $\mu M$ [$^{12}$C]azidoatrazine | 192 ± 4 | 3260 ± 32 | 100 |
| 10 | | 3455 ± 1 | | |
| | 10 $\mu M$ [$^{12}$C]azidoatrazine | 1814 ± 46 | 1641 ± 46 | 50 |
| 20 | | 3510 ± 11 | | |
| | 10 $\mu M$ [$^{12}$C]azidoatrazine | 2625 ± 32 | 885 ± 34 | 27 |
| 30 | | 3555 ± 38 | | |
| | 10 $\mu M$ [$^{12}$C]azidoatrazine | 2856 ± 29 | 700 ± 48 | 21 |

Table 2. Specificity of azidoatrazine binding and interaction of atrazine at the same site. Chloroplasts were treated in the sequence indicated. Ultraviolet (UV) irradiation was for 20 minutes; concentration of $^{14}$C-labeled ligand was 0.2 $\mu M$; and concentration of nonradioactive ligand was 10 $\mu M$. When indicated, chloroplasts were washed by eightfold dilution in resuspension buffer and centrifuged at 5000*g* for 10 minutes. All other binding conditions were as in Table 1.

| Treatment sequence | Competing ligand | Experiment 1 | | Experiment 2 | | Experiment 3 | |
|---|---|---|---|---|---|---|---|
| | | Pellet (dpm) | Displaced (dpm) | Pellet (dpm) | Displaced (dpm) | Pellet (dpm) | Displaced (dpm) |
| A. [$^{14}$C]azidoatrazine; dark; 4°C | | 3428 ± 16 | | 3557 ± 13 | | 3621 ± 12 | |
| | Azidoatrazine | 300 ± 40 | 3128 ± 43 | 743 ± 20 | 2814 ± 24 | 806 ± 32 | 2815 ± 34 |
| [$^{14}$C]azidoatrazine; 20′ UV; 4°C | | 3892 ± 16 | | 3327 ± 17 | | 3333 ± 52 | |
| | Azidoatrazine | 2836 ± 66 | 1056 ± 68 | 2178 ± 18 | 1149 ± 18 | 2324 ± 12 | 1009 ± 53 |
| | Atrazine | 2271 ± 36 | 1121 ± 39 | | | | |
| B. UV; then [$^{14}$C]azidoatrazine | | 3153 ± 8 | | | | | |
| | Azidoatrazine | 282 ± 20 | 2871 ± 22 | | | | |
| C. [$^{14}$C]atrazine; dark | | 3938 ± 13 | | | | | |
| | Atrazine | 329 ± 15 | 3609 ± 20 | | | | |
| [$^{14}$C]atrazine; UV | | 3380 ± 54 | | | | | |
| | Atrazine | 405 ± 7 | 2975 ± 54 | | | | |
| D. [$^{14}$C]azidoatrazine plus [$^{12}$C]atrazine; dark | | 4 ± 4 | | | | | |
| [$^{14}$C]azidoatrazine plus [$^{12}$C]atrazine; then UV | | 714 ± 72 | | | | | |
| E. [$^{12}$C]atrazine; UV; two washes; [$^{14}$C]atrazine | | | | 1704 ± 0 | | | |
| | Atrazine | | | 134 ± 0.5 | 1570 ± 0 | | |
| [$^{12}$C]azidoatrazine; UV; two washes; [$^{14}$C]atrazine | | | | 523 ± 11 | | | |
| | Atrazine | | | 119 ± 11 | 404 ± 16 | | |
| F. [$^{14}$C]azidoatrazine; dark; 50°C, 15 minutes | | | | | | 178 ± 2 | |
| | Azidoatrazine | | | | | 170 ± 0.5 | 8 ± 2 |
| [$^{14}$C]azidoatrazine; UV; 50°C, 15 minutes | | | | | | 1914 ± 18 | |
| | Azidoatrazine | | | | | 1896 ± 34 | 0 |

vented reversal of the binding by a 50-fold excess of nonradioactive ligand. Prevention of reversibility is the consequence of the formation of a covalent bond between a photoaffinity label and its site of action.

This effect of ultraviolet light is a property of the azido compound, not of atrazine or the chloroplasts. In an experiment similar to that described above, 20 minutes of ultraviolet irradiation prevented reversal of about 66 percent of the specific binding of azidoatrazine (Table 2, section A). In contrast, when the same experiment was done with [$^{14}$C]atrazine, the ultraviolet treatment caused only a small loss of specific binding (~ 18 percent), and this was due primarily to a loss of total binding [from 3938 to 3380 disintegrations per minute (dpm)], probably resulting from ultraviolet damage to the chloroplasts, rather than an increase in irreversible binding (Table 2, section C).

Whereas the exchangeable binding of azidoatrazine in the dark is sensitive to heat treatment, binding after ultraviolet irradiation is quite stable during treatment at 50°C for 15 minutes. After irradiation with ultraviolet, 82 percent (1896/2324) of the nonexchangeable radioactivity survived this heat treatment (Table 2, section F).

The ultraviolet effect is not an effect on the chloroplasts that allows them to react with azidoatrazine (Table 2, section B). Chloroplasts were first irradiated for 20 minutes with ultraviolet, and then a binding assay was carried out with [$^{14}$C]azidoatrazine, with and without cold azidoatrazine. There was a slight (~ 8 percent) loss of specific binding; again, this could be attributed to a loss of total binding rather than to an increase in irreversible binding.

That the photolysis in situ of azidoatrazine occurs at the same site as atrazine binding is indicated by two experiments. The first (Table 2, section D) shows that atrazine "protects" the site from the ultraviolet-induced nitrene. In the dark, 10 $\mu M$ atrazine fully competed against [$^{14}$C]azidoatrazine. However, if the 10 $\mu M$ nonradioactive atrazine was present during the ultraviolet irradiation, the amount of nonexchangeable [$^{14}$C]-azidoatrazine was reduced by 74 percent, from 2771 (section A) to 714 dpm. That the sites were not fully protected, even though the atrazine was in 50-fold excess, can be explained by the length of time necessary for the ultraviolet treatment. Since binding of atrazine is an equilibrium phenomenon, a much greater number of sites would have been occupied by the azido compound at some time during the 20-minute irradiation period than at any one time and, thus, potentially a greater proportion of sites could react covalently. In any case, the observed reduction of nonexchangeable azidoatrazine is evidence that ultraviolet light causes the irreversible binding of azidoatrazine at the noncovalent atrazine binding site. The protection of the azidoatrazine binding sites by atrazine appears to be specific to the active herbicide and not to inactive molecules of the triazine class (results not shown).

Another experiment indicates that the interaction of azidoatrazine after ultraviolet treatment occurs at the atrazine site (Table 2, section E). Photolysis of nonradioactive azidoatrazine in the chloroplast preparation reduced the subsequent ability of the chloroplasts to bind [$^{14}$C]atrazine. After ultraviolet treatment in the presence of 10 $\mu$M nonradioactive azidoatrazine, the chloroplasts were washed twice to remove excess unreacted compound. Compared to a control that was exposed to 10 $\mu$M atrazine during ultraviolet irradiation, the nonradioactive azioatrazine reduced the subsequent specific binding of atrazine by 74 percent, from 1570 to 404 dpm.

Gel electrophoresis of chloroplast membranes labeled with [$^{14}$C]azidoatrazine showed a large number of protein bands on the gel, but radioactivity was located only in one position (Fig. 1). The radioactivity was not associated with a major protein band but was consistently on the high-molecular-weight side of one of the major bands. Although the absolute amount of radioactivity detected in the gel was small, the radioactivity was always found in only one region, and the total amount was proportional to the amount of sample applied to the gel. For example, when 40 $\mu$l of sample was applied to the gel (Fig. 1), a total of 101 dpm above background was found under the peak. This amount corresponds to 25 to 30 percent of the radioactivity applied to that gel. In a parallel gel in the same experiment, 20 $\mu$l was applied and 51 dpm was found in the corresponding peak. When compared with the marker proteins (Fig. 1), the electrophoretic mobility of the radioactivity corresponded to a polypeptide of molecular weight 32,000. Replicate experiments gave similar, or slightly higher, values for molecular weight. Independent experiments with linear gradient gels in the presence of 2-mercaptoethanol also gave a value of 32,000 for the molecular weight of the triazine receptor (*7*).

The results indicate that azidoatrazine is a specific photoaffinity label for the photosystem II binding site, the apoprotein of B, in pea chloroplasts. Even though the binding experiments show

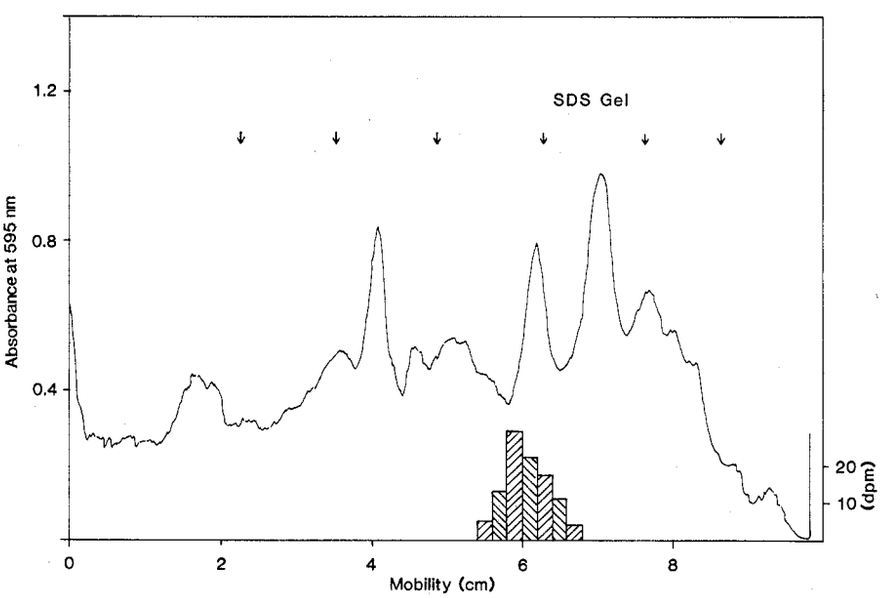


Fig. 1. Sodium dodecyl sulfate–polyacrylamide gel electrophoresis of chloroplast membranes labeled with [$^{14}$C]azidoatrazine. Forty microliters of labeled chloroplast membrane, prepared as for Table 2, containing 500 $\mu$g of chloroplasts per milliliter and 2 percent (weight to volume) SDS was washed in 50-fold excess nonradioactive azidoatrazine and applied to a 7.5 percent gel in 0.1 percent SDS. After electrophoresis, gels were stained with Coomassie blue, destained, and scanned at 595 nm, as shown. Gels were then sliced, and each slice was extracted for determination of radioactivity. Bars show the amount of radioactivity above background in each slice (background was ~ 20 count/min). Arrows indicate position of marker proteins in a parallel gel: phosphorylase b (94,000), bovine serum albumin (68,000), ovalbumin (43,000), carbonic anhydrase (30,000), soybean trypsin inhibitor (21,000), and lysozyme (14,300).

that this is functionally the case, direct proof of a photoaffinity label requires demonstration that the ligand becomes covalently attached to the receptor. Noncovalent interactions would not be expected to survive the denaturing sodium dodecyl sulfate (SDS) gel conditions; thus, localization of radioactivity in a specific region of the gel (shown in Fig. 1) is indicative of a covalent label.

Pfister and Arntzen (*1*) and Pfister *et al.* (*3*) compared chloroplast membrane polypeptides from triazine-resistant and triazine-susceptible biotypes of the same weed species. Triazine resistance was shown to be due to a change in the binding affinity for atrazine. However, since functionality was lost during the analysis, it was not possible to prove which specific peptides were directly related to the phenomenon of herbicide resistance. The photoaffinity approach labels the molecule in a functional situation. Since the label is covalently attached to the receptor, it remains stable under conditions that destroy functional activity. Pfister *et al.* (*7*) showed that only in chloroplast membranes from susceptible plants was there specific covalent attachment of [$^{14}$C]azidoatrazine to a polypeptide of 32,000 daltons.

Mild trypsin treatment of photosystem II particles resulted in loss of sensitivity to the herbicide diuron and concomitant loss of two protein bands of molecular weight 32,000 and 27,000 (*8*). Since diuron and atrazine bind to the same site (*2*, *3*), it is likely that the azidoatrazine receptor identified here is identical to the trypsin-sensitive 32,000-dalton peptide. It is interesting to speculate that this peptide may also be the rapidly turned over 32,000-dalton membrane protein that accumulates during light-dependent chloroplast development (*9*) or the 32,000-dalton peptide implicated in proton translocation in the chloroplast membrane adenosinetriphosphatase, $CF_0$ (*10*).

Little is known about the biochemistry of protein-bound quinones. Azidoatrazine may be useful in the study of the detailed protein chemistry of photosystem II.

GARY GARDNER

*Shell Development Company, Biological Sciences Research Center, Modesto, California 95352*

### References and Notes

1. K. Pfister and C. J. Arntzen, *Z. Naturforsch.* **34c**, 996 (1979).
2. W. Tischer and H. Strotmann, *Biochim. Biophys. Acta* **460**, 113 (1977).
3. K. Pfister, S. R. Radosevich, C. J. Arntzen, *Plant Physiol.* **64**, 995 (1979).
4. V. Chowdhry and F. H. Westheimer, *Annu. Rev. Biochem.* **48**, 293 (1979); W. G. Hanstein, *Methods Enzymol.* **56**, 653 (1979).
5. D. Berrer and C. Vogel (to Geigy Chemical Co.), U.S. Patent 3,583,987 (1971); (to Ciba-Geigy Corp.), U.S. Patent 3,723,088 (1973).
6. P. M. Ray, U. Dohrmann, R. Hertel, *Plant Physiol.* **59**, 357 (1977).
7. K. Pfister, K. D. Steinback, G. Gardner, C. J. Arntzen, *ibid.* **65** (Suppl.), 10 (1980); *Proc. Natl. Acad. Sci. U.S.A.*, in press.
8. E. Croze, M. Kelly, P. Horton, *FEBS Lett.* **103**, 22 (1979).
9. J. R. Bedbrook *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **75**, 3060 (1978); S. A. Weinbaum, J. Gressel, A. Reisfeld, M. Edelman, *Plant Physiol.* **64**, 828 (1979).
10. J. L. Ellenson, D. J. Pheasant, R. P. Levine, *Biochim. Biophys. Acta* **504**, 123 (1978).
11. I thank C. D. Allen for technical assistance and W. B. Burton for synthesis and radiosynthesis of azidoatrazine.

4 August 1980; revised 29 September 1980

## Pelagic Sedimentation of Aragonite: Its Geochemical Significance

Abstract. *The relative importance of the pelagic flux of aragonite, as compared to calcite, to the deep-sea floor has been evaluated by means of a quantitative x-ray diffraction study of samples collected from sediment traps and from an unusually shallow portion of the open Atlantic Ocean (the Rio Grande Rise). The results suggest that the aragonite flux constitutes at least 12 percent of the total flux of calcium carbonate on a worldwide basis. The presence of high-magnesium calcite in several samples suggests that some of the calcareous material falling to the deep-sea floor may be derived from the long-distance transport of debris from shallow-water benthic organisms as well as from the settling of planktonic remains. This observation supports the contention that 12 percent represents a minimum value. Aragonite and high-magnesium calcite transported laterally from shallow-water regions, upon dissolution during settling into deeper water, may contribute to the neutralization of excess anthropogenic carbon dioxide added to the oceans.*

Large amounts of calcium carbonate ($CaCO_3$) are precipitated from seawater by marine organisms, which use the $CaCO_3$ to form shell-like exoskeletons. Upon death, lateral transport, and sedimentation, some of the carbonate undergoes dissolution in the deep sea, and this dissolution, along with the original precipitation, ultimately exerts a major influence on how much $CO_2$, including excess $CO_2$ produced by human activities, can be taken up by the sea (*1*). It is normally assumed that $CaCO_3$ falling to the deep-sea floor is present as calcite in the form of coccoliths and foram tests; however, a large proportion may instead consist of aragonite. This aragonite, which is present in the form of pteropod shells and laterally transported debris from shallow-water benthic organisms, does not accumulate in most places because it is more soluble than calcite and is highly undersaturated in the deep sea and consequently dissolves away before it can be buried. Because of the relative lack of aragonite in deep-sea sediments due to dissolution, it has been assumed that aragonite sedimentation in the pelagic realm is unimportant relative to calcite sedimentation. However, the finding of abundant aragonite in plankton and in rare pelagic sediments which are sufficiently shallow that aragonite dissolution on the bottom has not occurred (*2*) indicates that aragonite sedimentation is probably far more important than previously recognized.

In the earlier work (*2*), the quantitative

Table 1. Aragonite (A) and calcite (C) from sediment traps at the PARFLUX sites.

| Depth (m) | Flux (mg m$^{-2}$ day$^{-1}$) | | | | | | | | A % of total $CaCO_3$ flux |
|---|---|---|---|---|---|---|---|---|---|
| | < 63 μm | | 63 μm to 1 mm | | < 1 mm | | > 1 mm | | |
| | A | C | A | C | A | C | A | C | |
| *Panama Basin (station PB) (5°21′N, 81°53′W)* | | | | | | | | | |
| 667 | (< 1) | 20.4* | 3.0 | 12.0 | | | 2.4 | 4.8 | 12.7 |
| 1268 | (< 2) | 24.4 | 1.0 | 11.6 | | | 1.7 | 0.8 | 6.9 |
| 2869 | (< 1) | 34.3 | 1.7 | 14.9 | | | 1.3 | 1.1 | 5.7 |
| 3769 | (< 2) | 28.8 | 0.4 | 12.9 | | | 1.3 | 0.5 | 3.9 |
| 3791 | (< 2) | 30.8 | 0.8 | 15.4 | | | 1.6 | 1.0 | 4.9 |
| *Equatorial Atlantic (station E) (13°30.2′N, 54°00.1′W)* | | | | | | | | | |
| 389 | | | | | 2.5 | 22.0* | 14.3 | 4.0* | 38.9 |
| 988 | | | | | 2.3 | 23.1* | 1.1 | 0.6 | 12.5 |
| 3755 | | | | | 1.8 | 23.7* | (Total $CaCO_3$ = 0.6) | | 7 to 9 |
| 5068 | | | | | 2.0 | 20.5* | (Total $CaCO_3$ = 0.9) | | 9 to 12 |

*Samples containing high-magnesium calcite.


0036-8075/81/0227-0940$00.50/0  