

Lawrence Berkeley National Laboratory

Recent Work

Title

REGULATION OF PHOTOSYNTHETIC CARBON METABOLISM

Permalink

<https://escholarship.org/uc/item/1s68x9gf>

Author

Bassham, James A.

Publication Date

1971-08-01

Published in Proceedings of the
II International Congress on
Photosynthesis Research,
Stresa, Italy, 1971, Dr. W. Junk,
Publishers, The Hague, Netherlands

LBL-1014
c.1

REGULATION OF PHOTOSYNTHETIC
CARBON METABOLISM

James A. Bassham

August 1971

RECEIVED
LAWRENCE
RADIATION LABORATORY

NOV 1 1972

LIBRARY AND
DOCUMENTS SECTION

AEC Contract No. W-7405-eng-48

For Reference

Not to be taken from this room



LBL-1014
c.1

DISCLAIMER

This document was prepared as an account of work sponsored by the United States Government. While this document is believed to contain correct information, neither the United States Government nor any agency thereof, nor the Regents of the University of California, nor any of their employees, makes any warranty, express or implied, or assumes any legal responsibility for the accuracy, completeness, or usefulness of any information, apparatus, product, or process disclosed, or represents that its use would not infringe privately owned rights. Reference herein to any specific commercial product, process, or service by its trade name, trademark, manufacturer, or otherwise, does not necessarily constitute or imply its endorsement, recommendation, or favoring by the United States Government or any agency thereof, or the Regents of the University of California. The views and opinions of authors expressed herein do not necessarily state or reflect those of the United States Government or any agency thereof or the Regents of the University of California.

REGULATION OF PHOTOSYNTHETIC CARBON METABOLISM

James A. Bassham

The Reductive Pentose Phosphate Cycle

The elucidation of metabolic paths and of their in vivo regulation by means of substrates labeled with radioisotopes has been particularly useful for photosynthesizing cells. The reductive pentose phosphate cycle (RPP cycle, Figure 1) was completely mapped by such studies by Calvin and coworkers.^{1,2} The mechanisms of in vivo metabolic regulation of this cycle and of paths leading from it to end products are being revealed through the use of similar techniques.

This approach to investigation of pathways and their regulation contrasts with perhaps more conventional studies which depend primarily on the isolation of enzymes and investigation of their properties. Of course, the isolation and characterization of enzymes which catalyze steps of the photosynthetic paths have been required to confirm the paths and the mechanisms of their metabolic regulation. In fact, certain enzymes of the RPP cycle, ribulose-1,6-diphosphate carboxylase (RuDPCase), fructose-1,6-diphosphatase (FDPase), and sedoheptulose-1,7-diphosphatase were found to be so low in activity when isolated from green leaves that it was doubted that they could catalyze the proposed in vivo reactions.³ These enzymes catalyze regulated (rate-limiting) steps of the RPP cycle,⁴⁻⁶ and must be provided with optimal conditions (including high Mg⁺⁺ ion concentration)^{7,8} in order to provide just the minimal activity required for rate-limited steps.

In a complete turn of the RPP cycle, carboxylation of three RuDP molecules gives 6 molecules of PGA, which can be reduced to 6 molecules of

triose phosphate by reactions using 6 ATP molecules and 6 NADPH molecules. Only 5 triose phosphate molecules are needed to regenerate 3 Ru5P molecules which are then converted to 3 RuDP molecules with 3 more ATP molecules.

The triose phosphate molecule gained from the reduction of 3 CO₂ molecules may be condensed with triose phosphate to give sugar monophosphates and eventually carbohydrates, including starch, sugar, and cellulose.

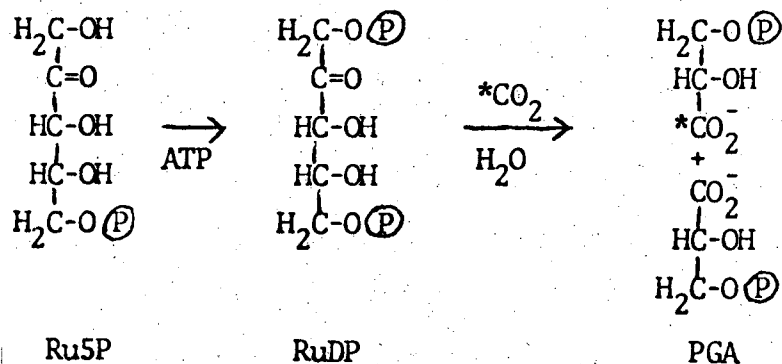
Alternatively, the triose phosphate molecules can be converted to glycerophosphate for fat synthesis or transformed via PGA and phosphoenolpyruvate along many well known biochemical pathways to give various amino acids, fatty acids, and other molecules needed by the cell.

For the discussion that follows, it is helpful to review briefly some techniques used to map the RPP cycle and still used in studying regulation. When plants were exposed for a few seconds to ¹⁴CO₂ while photosynthesizing, killed, and the products of photosynthesis analyzed by two-dimensional paper chromatography and radioautography, the first product was 3-phosphoglyceric acid (PGA) followed immediately by simultaneous appearance of sugar phosphates with 3 to 7 carbon atoms. Chemical degradation of these sugars to reveal the location of ¹⁴C in the carbon chains, and knowledge of previously determined reactions of glycolysis permitted formulation of most of the cycle, from PGA to pentose phosphate.

A different method was required to discover the carboxylation substrate. The algae photosynthesized for several minutes with ¹⁴CO₂ under steady-state conditions. After about 5 min, the intermediate compounds of the primary photosynthetic pathway were later found to be completely labeled with ¹⁴C; that is, the radioactivity in a pool of a given compound no

longer increased. The ¹⁴C content of each pool of compounds then represented the actual concentration of that compound within the cells.

An environmental condition then was changed; for example, the light was turned off. Samples taken rapidly after this perturbation revealed on subsequent analysis the changes in concentration accompanying the perturbation. In the case of the light to dark transient, it was found that the level of radioactive PGA rose very rapidly, as expected upon interruption of the supply of photochemically produced cofactors required for its reduction to triose phosphate (Figure 2).⁹ The level of ribulose-1,5-diphosphate (RuDP) dropped rapidly, suggesting that RuDP is formed from ribulose-5-phosphate (Ru5P) by a reaction utilizing ATP from the light. The carboxylation of RuDP to give 2 molecules of 3-PGA clearly proceeded in the dark. When the light was left on, but the level of CO₂ was reduced, RuDP increased and PGA decreased as expected.¹⁰



RuDP, with an open chain and two phosphate groups, is so reactive that carboxylation takes place without added energetic cofactors. In fact, the free energy change accompanying the reaction at steady-state conditions in photosynthesizing algae is -10 Kcal, by far the most negative ΔG^S for any step in photosynthetic carbon metabolism in such organisms.¹¹

Plants containing the oxygen-evolving mechanism and the RPP cycle in the same chloroplasts pay a high price for the chemical reactivity of RuDP. Molecular O_2 (or intermediate oxidants leading to the oxidation of water to O_2 ¹²) can attack RuDP, perhaps bound to the carboxylation enzyme,¹³ giving PGA plus phosphoglycolic acid, instead of 2 PGA molecules. When algae are deprived of CO_2 , after photosynthesis with $^{14}CO_2$, the rapid rise in ^{14}C -RuDP level, mentioned earlier, is quickly followed by a large increase in ^{14}C -glycolic acid.¹⁰

When Chlorella pyrenoidosa are exposed to $^{14}CO_2$ in the presence of either oxygen, CO_2 -free air, or nitrogen, the inhibition of photosynthesis, the production of glycolic acid and of phosphoglycolic acid, and the decrease in the carboxylation substrate, ribulose diphosphate, are well correlated with the presence of oxygen (Table I).¹⁴ Once formed, phosphoglycolate is hydrolyzed by glycolate phosphatase, an active enzyme found in chloroplasts.¹⁵ The glycolate is then metabolized outside the chloroplasts,¹⁶ giving CO_2 and organic compounds that can be reincorporated into photosynthetic pathways. The oxidation of newly formed photosynthetic intermediates to CO_2 by this route is probably the principal component of photorespiration, though other mechanisms may contribute.

Photorespiration thus is a function of O_2 tension, temperature, and light intensity, and an inverse function of CO_2 pressure. Thus, it is especially deleterious to plants whose native habitat is warm, sunny, and dry (because closing the stomata lowers CO_2 pressure in the leaf). It is not surprising that some species of such plants have evolved carbon fixation pathways to overcome the effect of RuDP oxidation by O_2 . This apparently has been accomplished by separating the sites of the RPP cycle

from the water oxidation- O_2 evolving mechanism, and by refixing any CO_2 that does form by photorespiration.

In certain tropical grasses, such as maize and sugar cane, and assorted other plant species, there are found two kinds of cells, in which the chloroplasts differ between cell types.¹⁷⁻¹⁹ In the vascular bundle (parenchyma) cells the chloroplasts contain only single lamella (they are without grana) and reduce carbon via the RPP cycle (or something like it). In the mesophyll cells, which are closer to the leaf surfaces, chloroplasts contain grana and fix CO_2 via part of a cycle which does not involve RuDP.²⁰ Since the agranal chloroplasts apparently do not have a photosystem 2 and do not evolve O_2 ,²¹ but do contain the RPP cycle, the oxidation of RuDP and other sugar phosphates is minimized. Some such oxidation does occur, and the resulting photorespiratory CO_2 is to a large extent refixed by the mesophyll chloroplasts and does not escape the leaf.

This pathway of CO_2 fixation (Figure 3) was mostly elucidated by Hatch and Slack,^{22,23} after Kortchack, Hartt, and Burr²⁴ had found that primary CO_2 incorporation in such plants occurs via a carboxylation of phosphoenolpyruvate to give malic and aspartic acids. The pathway is known as the C-4 pathway. Presumably, the C-4 acids, once formed in the mesophyll cells, are translocated, via clearly visible plasmadesmata,²⁵ to the parenchyma cells, where they are oxidatively decarboxylated to give CO_2 and NADPH to the agranal chloroplasts. These chloroplasts, which produce their own ATP, then may carry out the RPP cycle. However, additional reduced cofactors (a total of 2 NADPH molecules per CO_2 molecule are required) would have to come from the mesophyll cells, according to this hypothesis.

Metabolic Regulation of Carbon Metabolism: Within the RPP Cycle

Since several intermediate compounds of the Calvin cycle are used as starting points for biosynthesis, some points of metabolic regulation of reactions of the cycle and of paths leading from it are required. The techniques of suddenly interrupting the steady-state photosynthesis and observing the transient effects on metabolite concentrations, mentioned earlier, have been very valuable in locating sites of metabolic regulation.⁶ A few examples from many studies will illustrate the method.

When the light-dark steady-state experiment with algae is followed by turning the light on again, additional interesting transient changes are observed.^{5,26} The level of fructose-1,6-diphosphate (FDP) very suddenly increases for about 30 sec when the light comes on, and then, just as suddenly, decreases (Figure 4). After several oscillations the level settles down to the light steady-state level. The initial rise indicates that the enzyme (FDPase) which converts FDP to F6P was inactive in the dark and remained inactive during the first 30 sec of light at a time when the rapid reduction of PGA to triose phosphates (which condense to make FDP) took place. Then the FDPase was activated and the FDP was more rapidly hydrolyzed than formed. Finally as the cycle reached its full steady-state rate due to activation of the various control points, the steady-state levels of FDP and other intermediates were approached.

Similarly, light activation of the carboxylation enzyme (which converts RuDP and CO₂ to PGA) was observed, both with algae and with isolated spinach chloroplasts. The activations of these enzymes and of phosphoribulokinase (which converts Ru5P with ATP to RuDP) are not direct photochemical activations, since reversible inactivations can

be seen even with the light on when chemical inhibitors are added which reversibly inactivate photosynthesis.^{4,27} Rather, it appears that the mechanism of activation of these regulated enzymes may involve the levels of Mg^{++} ion, pH, and the ratio of reduced to oxidized cofactors (such as NADPH/NADP⁺).⁶ A light-activated factor for RuDPCase has been reported.²⁸ Also, studies of the transport properties of the outer chloroplast membrane carried out with isolated chloroplasts^{29,30} suggest a correlation between such transport and CO₂ fixation activity.

One clue to the purpose of these regulated enzymes was found in the appearance of 6-phosphogluconic acid in the dark and its disappearance in the light. This metabolite, which is a marker for the oxidative pentose phosphate cycle, can be made to appear in Chlorella even with the light on when vitamin K₅ is added to the photosynthesizing algae suspension.²⁷ Vitamin K₅ is readily oxidized by air and light. In its oxidized form, it apparently accepts electrons from the photochemical electron transport system of the chloroplasts, thereby diverting the electrons from the reductive steps of the reductive pentose phosphate cycle. Since 6-phosphogluconic acid appears upon the addition of vitamin K₅, even with isolated chloroplasts,²⁷ it is clear that the oxidative pentose phosphate cycle can operate inside the chloroplasts. It is significant that addition of vitamin K₅ in the light also causes a blocking of reactions catalyzed by FDPase and SDPase, RuDP carboxylase, and phosphoribulokinase (Figure 1). For example, in Figure 5 we see that the level of SDP rises abruptly, while the level of RuDP does not decline to below detectable levels, as it would if the carboxylation enzyme remained active. Figure 6 shows clearly the sudden rise in pentose

monophosphates resulting from inactivation of phosphoribulokinase (level of ATP did not drop). Thus, the sites of inactivation in the dark are all revealed in the light by addition of vitamin K₅. The oxidized form of vitamin K₅ is in time destroyed in light and air, forming an inactive colored compound, and the effects on photosynthesis are partly reversed provided only a low level of vitamin K₅ was added at the start.

One purpose of the light activation of carboxylase, FDPase, and phosphoribulokinase and their inactivation in the dark would appear to be to permit the blocking in the dark of reactions unique to the reductive pentose phosphate cycle so that the oxidative pentose phosphate cycle can operate in the chloroplasts (Figure 1). This oxidative cycle would produce NADPH, which together with ATP from the mitochondria could then be used for biosynthesis in the dark inside the chloroplasts. As a corollary to this postulate, the oxidative pentose phosphate cycle must be prevented from operating in the light, presumably through the inactivation of glucose-6-phosphate dehydrogenase.

Studies with unicellular algae in which growth has been synchronized so that all cells in a given culture are growing or dividing at the same time, have indicated that these same regulatory sites may be involved in the allocation of carbon from the reductive pentose phosphate cycle to biosynthetic pathways leading to synthesis of carbohydrate, fat and protein.^{31,32} As these algae pass from a stage of rapid growth to one of cell division, their needs for the synthesis of protein and fat as compared with the synthesis of carbohydrates apparently change.

Looking at the photosynthetic carbon reduction cycle (Figure 1), we can see that the compounds required for protein and fat synthesis

are derived from PGA and triose phosphate, which lie after the carboxylation reaction and before the FDPase and SDPase reactions. On the other hand, the compound required for carbohydrate synthesis, fructose-6-phosphate, lies after the FDPase reaction and before the carboxylase reaction. Since the carboxylase reaction and the FDPase and SDPase reactions are rate-limiting steps in the Calvin cycle, a small change in the ratios of activities of these enzymes can cause carbon to accumulate either in the half of the cycle between carboxylation and FDPase or in the other half of the cycle after FDPase and before carboxylation. Thus the regulatory mechanisms can accumulate reduced carbon at the point where it is needed for subsequent biosynthesis.

Regulation of Carbon Metabolism: Outside the RPP Cycle

Additional regulatory steps are required on the pathways leading from the cycle to control more accurately the amount of carbon withdrawn for biosynthesis. The sites of two such regulatory points in Chlorella pyrenoidosa have been revealed by studies of the effects of adding ammonium ion.³³ As might be expected, the addition of ammonium ion in 1 mM concentrations to algae cells results in an increased synthesis of amino acids and proteins. When ammonium ion was added during photosynthesis by algae, several dramatic changes in the rates of flow of carbon compounds through the metabolic pathways were observed. First of all, sucrose synthesis almost completely stopped, and starch synthesis declined somewhat. At the same time, there was an increased rate of synthesis of fats and a greatly increased rate of synthesis of amino acids.

The main reason for the increased amino acid formation became apparent when we examined the changes in steady-state levels of phosphoenopyruvic

acid and of pyruvic acid. The conversion of PEPA to pyruvate is a key reaction along the path leading from PGA to the synthesis of amino acids and fatty acids. Immediately after the addition of ammonium ion the level of PEPA dropped, while the level of pyruvate rose (Figure 5). From these changes in level, and from the known free energy change accompanying the conversion of PEPA to pyruvate, it is possible to calculate that the ratio of the forward over the back reaction changed by a factor of four.

The light to dark transition also causes a change in activities of enzymes catalyzing key reactions on pathways leading from the RPP cycle to end products. A recent experiment by Kanazawa et al.³⁴ demonstrates dramatically the cessation of starch synthesis in the dark. Preiss and coworkers³⁵⁻³⁸ had found that isolated ADP-glucose pyrophosphorylase is subject to inhibition by phosphate and activation by PGA. They proposed that this enzyme, which mediates the reaction of glucose-1-phosphate with ATP to give ADP-glucose and pyrophosphate, is the key enzyme in shutting down starch synthesis in the dark, probably due to increased levels of phosphate in the chloroplasts³⁹ We have now examined the level of ADP-glucose and other intermediates in Chlorella pyrenoidosa during steady-state photosynthesis and afterwards in the dark.³⁴ The level of ADP-glucose declines very rapidly within a few seconds after the light is turned off. It is clear that ADP-glucose formation, and consequently starch synthesis, stops suddenly in the dark due to inactivation of the ADP-glucose pyrophosphorylase. From this and past experiments, we know that the level of glucose-6-phosphate remains high when the light is turned off. In this experiment, ATP level, after a transient downward fluctuation when the light was turned off, was the same in the dark as in the light. Similarly, the level of PGA

was about the same in the dark as in the light. Presumably the activation of ADP-glucose pyrophosphorylase in the light was due to a lower level of phosphate in the chloroplasts or to other as yet unidentified factors.

Such findings are providing us with a clearer picture of the dynamic control of in vivo metabolism in photosynthetic cells. We are hopeful that besides aiding in the general understanding of metabolic regulation, this knowledge may form a basis for a better evaluation of how plants adapt to environmental and physiological change at the level of metabolism.

References

1. J. A. Bassham, A. A. Benson, L. D. Kay, A. Z. Harris, A. T. Wilson, and M. Calvin. J. Amer. Chem. Soc. 76, 1760 (1954).
2. James A. Bassham and Melvin Calvin, "The Path of Carbon in Photosynthesis," Prentice-Hall, Inc., Englewood Cliffs, N. J., 1957, pp. 1-107.
3. A. Peterkofsky and E. Racker, Plant Physiol. 36, 409 (1961).
4. T. A. Pedersen, Martha Kirk, and J. A. Bassham, Biochim. Biophys. Acta 112, 189 (1966).
5. T. A. Pedersen, Martha Kirk, and J. A. Bassham, Physiol. Plantarum 19, 219 (1966).
6. J. A. Bassham, Science 172, 526 (1971).
7. J. A. Bassham, Pamela Sharp, and Ian Morris, Biochim. Biophys. Acta 153, 901 (1968).
8. J. Preiss, M. L. Biggs, and E. Greenberg, J. Biol. Chem. 242, 2292 (1967).
9. M. Calvin and P. Massini, Experientia 8, 445 (1952).
10. A. T. Wilson and M. Calvin, J. Amer. Chem. Soc. 77, 5948 (1955).

11. J. A. Bassham and G. H. Krause, *Biochim. Biophys. Acta* 189, 207 (1969).
12. A. Plaut and M. Gibbs, *Plant Physiol.* 45, 470 (1970).
13. W. L. Ogren and G. Bowes, *Nature New Biology* 230, 159 (1971).
14. J. A. Bassham and M. Kirk, *Biochem. Biophys. Res. Commun.* 9, 376 (1962).
15. K. E. Richardson and N. E. Tolbert, *J. Biol. Chem.* 236, 1285 (1961).
16. R. Rabson, N. E. Tolbert, and P. C. Kearney, *Arch. Biochem. Biophys.* 98, 154 (1962).
17. W. M. Laetsch and I. Price, *Amer. J. Bot.* 56, 77 (1969).
18. A. J. Hodge, J. D. McLean, and F. V. Mercer, *Biochem. Biophys. Cytol.* 1, 605 (1955).
19. M. Johnson, "An Electron Microscope Study of the Photosynthetic Apparatus of Plants," Ph.D. Thesis, University of Texas, Austin, 1964.
20. T. J. Andrews and M. D. Hatch, *Biochem. J.* 114, 117 (1969).
21. K. C. Woo, J. M. Anderson, N. K. Boardman, W.J.S. Downton, C. B. Osmond, and S. W. Thorne, *Proc. Nat. Acad. Sci. U.S.* 67, 18 (1970).
22. M. D. Hatch and C. R. Slack, *Ann. Rev. Plant Physiol.* 21, 141 (1970).
23. M. D. Hatch and C. R. Slack, *Biochem. J.* 101, 103 (1966).
24. H. P. Kortschack, C. E. Hartt, and G. O. Burr, *Plant Physiol.* 40, 209 (1965).
25. W. M. Laetsch, *Sci. Prog. Oxf.* 57, 323 (1969).
26. J. A. Bassham and Martha Kirk, in "Comparative Biochemistry and Biophysics of Photosynthesis," K. Shibata, A. Takamiya, A. T. Jagendorf, and R. C. Fuller, eds., University of Tokyo Press, 1968, pp. 365-378.
27. G. H. Krause and J. A. Bassham, *Biochim. Biophys. Acta* 172, 553 (1969).

28. G. F. Wildner and R. S. Criddle, *Biochem. Biophys. Res. Commun.* 37, 952 (1969).
29. R. E. Moore, H. Springer-Lederer, H.C.J. Ottenheim, and J. A. Bassham, *Biochim. Biophys. Acta* 180, 368 (1969).
30. J. A. Bassham, A. M. El-Badry, M. R. Kirk, H.C.J. Ottenheim, and H. Springer-Lederer, *Biochim. Biophys. Acta* 223, 261 (1970).
31. T. Kanazawa, K. Kanazawa, M. R. Kirk, and J. A. Bassham, *Plant & Cell Physiol.* 11, 149 (1970).
32. T. Kanazawa, K. Kanazawa, M. R. Kirk, and J. A. Bassham, *Plant & Cell Physiol.* 11, 445 (1970).
33. T. Kanazawa, M. R. Kirk, and J. A. Bassham, *Biochim. Biophys. Acta* 205, 401 (1970).
34. T. Kanazawa, K. Kanazawa, M. Kirk, and J. A. Bassham (in preparation).
35. J. Preiss, H. P. Ghosh, and J. Wittkop, in "Biochemistry of Chloroplasts," Vol. II, T. W. Goodwin, ed., Academic, New York and London, 1967, pp. 131-153.
36. H. P. Ghosh and J. Preiss, *J. Biol. Chem.* 241, 4491 (1966).
37. G. G. Sanwal, E. Greenberg, J. Hardie, E. C. Cameron, and J. Preiss, *Plant Physiol.* 43, 417 (1968).
38. G. G. Sanwal and J. Preiss, *Arch. Biochem. Biophys.* 119, 454 (1967).
39. J. Preiss and T. Kosuge, *Ann. Rev. Plant Physiol.* 21, 433 (1970).

Table I. Effect of Oxygen on Photosynthesis with $^{14}\text{CO}_2$

	(μc ^{14}C /gm algae)		
	O_2	CO_2 -free air	N_2
Total ^{14}C fixed	508.0	642.7	732.9
Glycolic acid	48.2	9.2	2.0
Phosphoglycolic acid	3.4	2.1	1.7
Ribulose diphosphate	58.4	91.0	87.6
Other sugar diphosphates	0.3	0.5	1.3
Phosphoglyceric acid	20.1	25.3	40.6

FIGURE CAPTIONS

Figure 1. The Reductive Pentose Phosphate Cycle (Calvin Cycle) of Photosynthesis.

Solid lines indicate reactions of reductive cycle, dashed lines reactions of oxidative cycle. Open arrows indicate positions of enzymes activated in light; dark blunt arrow indicates position of enzyme activated in dark.

GAld3P, glyceraldehyde-3-phosphate; FDP, fructose-1,6-diphosphate; F6P, fructose-6-phosphate; DHAP, dihydroxyacetone phosphate; E4P, erythrose-4-phosphate; SDP, sedoheptulose-1,7-diphosphate; S7P, sedoheptulose-7-phosphate; R5P, ribose-5-phosphate; Xu5P, xylulose-5-phosphate; Ru5P, ribulose-5-phosphate; RuDP, ribulose-1,5-diphosphate; G6P, glucose-6-phosphate; 6-PGluA, 6-phosphogluconate; *C₂ is actually enzyme-bound thiamine pyrophosphate-glycoaldehyde, an intermediate in the two reactions mediated by transketolase.

Figure 2. Light-dark and High CO₂-low CO₂ Transient Changes in Levels of ¹⁴C-Labeled Photosynthetic Intermediates.

Idealized curves based on studies by Calvin and Massini⁹ and Wilson and Calvin.¹¹

RuDP, ribulose-1,5-diphosphate; PGA, 3-phosphoglyceric acid.

Figure 3. The C-4 Acid Cycle of Photosynthetic Carbon Fixation.

Figure 4. Transient Changes in Levels of FDP and DHAP during Light-dark and Dark-light Transitions.

Figure 5. Effects of Addition of Vitamin K₅ to Photosynthesizing Chlorella pyrenoidosa on Levels of SDP and RuDP.

Figure 6. Effects of Addition of Vitamin K₅ to Photosynthesizing Chlorella pyrenoidosa on Levels of Pentose Monophosphates.

Figure 7. Changes in Levels of Pyruvate and Phosphoenolpyruvate upon Addition of NH₄⁺ to Photosynthesizing Chlorella pyrenoidosa.

Figure 8. Transient Changes in Level of ADP-glucose during Light-dark Transition in Chlorella pyrenoidosa.

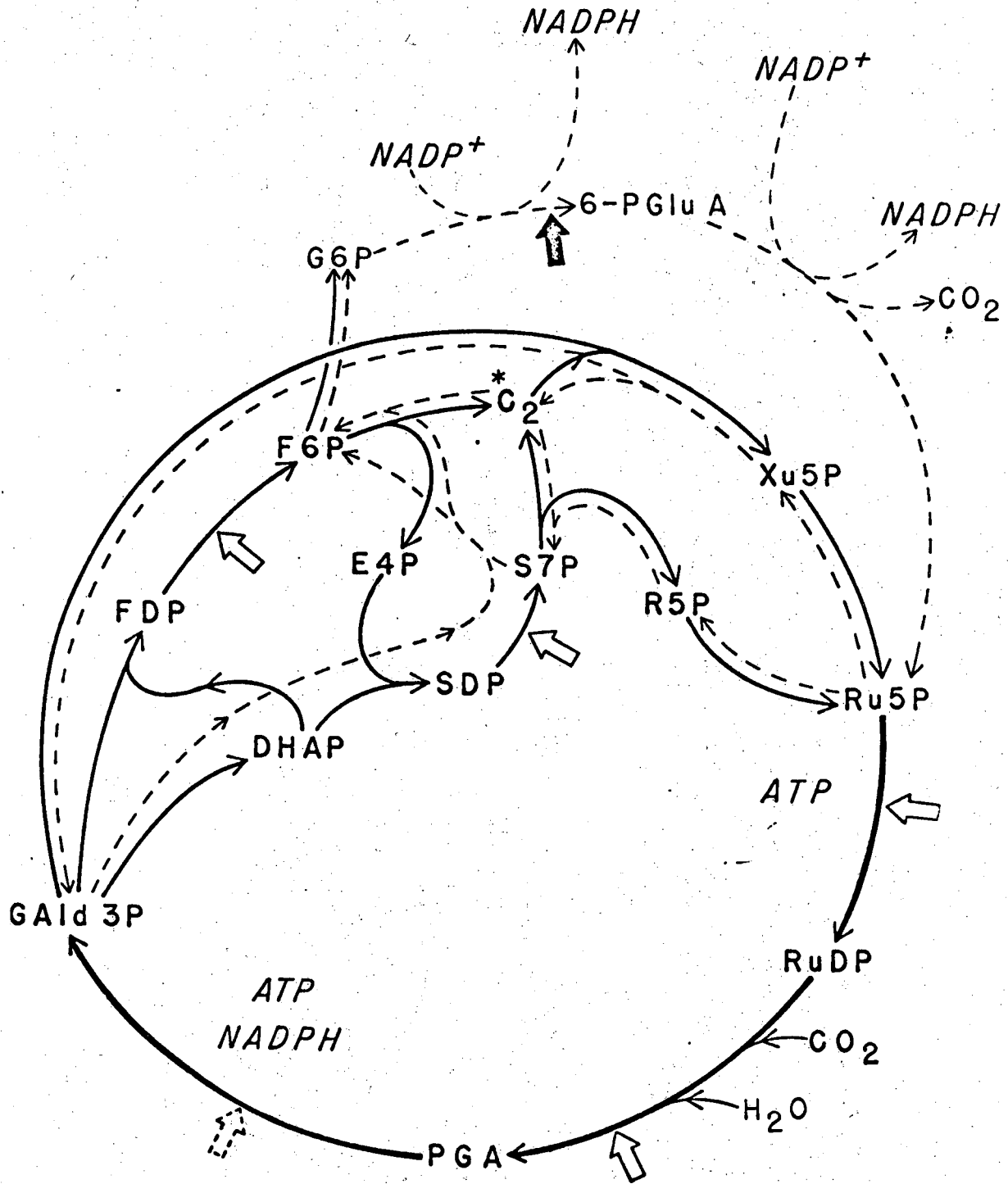


Fig. 1

XBL 706-5260 A

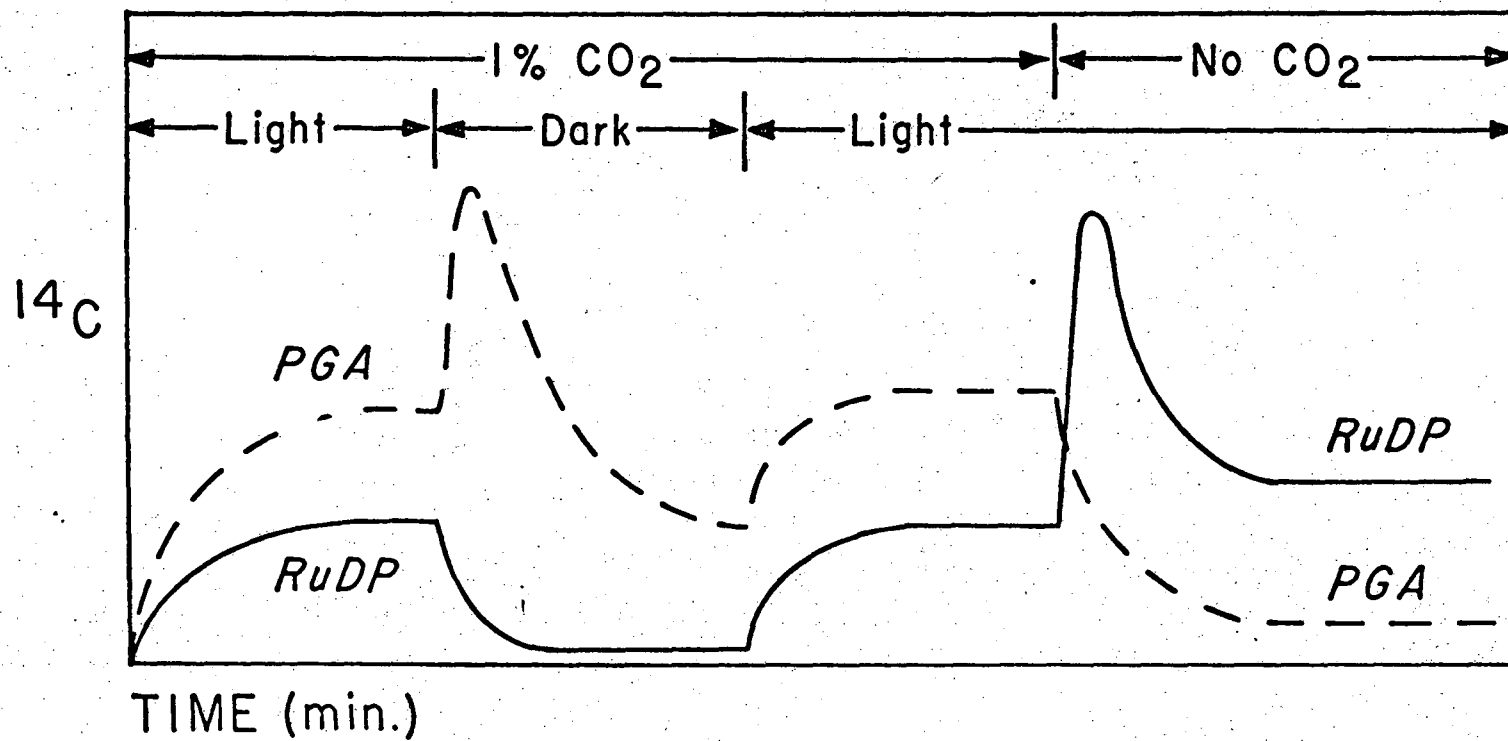


Fig. 2

XBL 714-5102-A

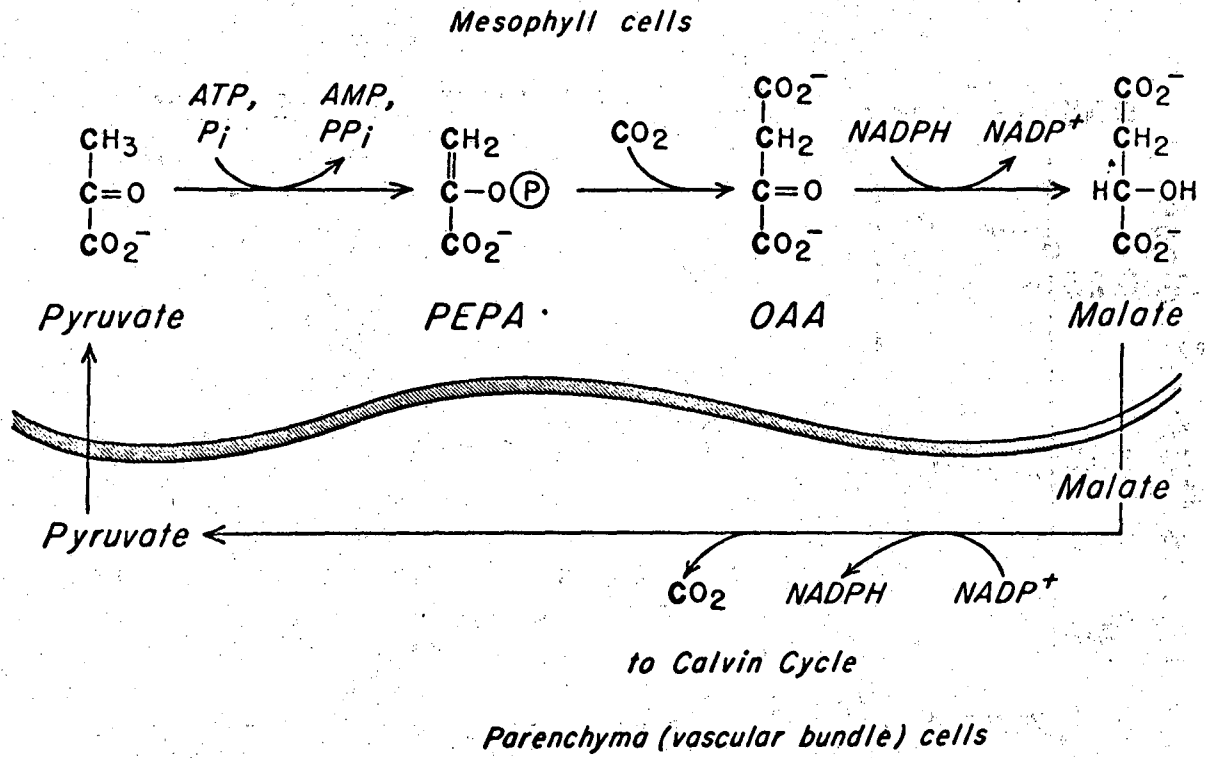


Fig. 3

XBL714-5113

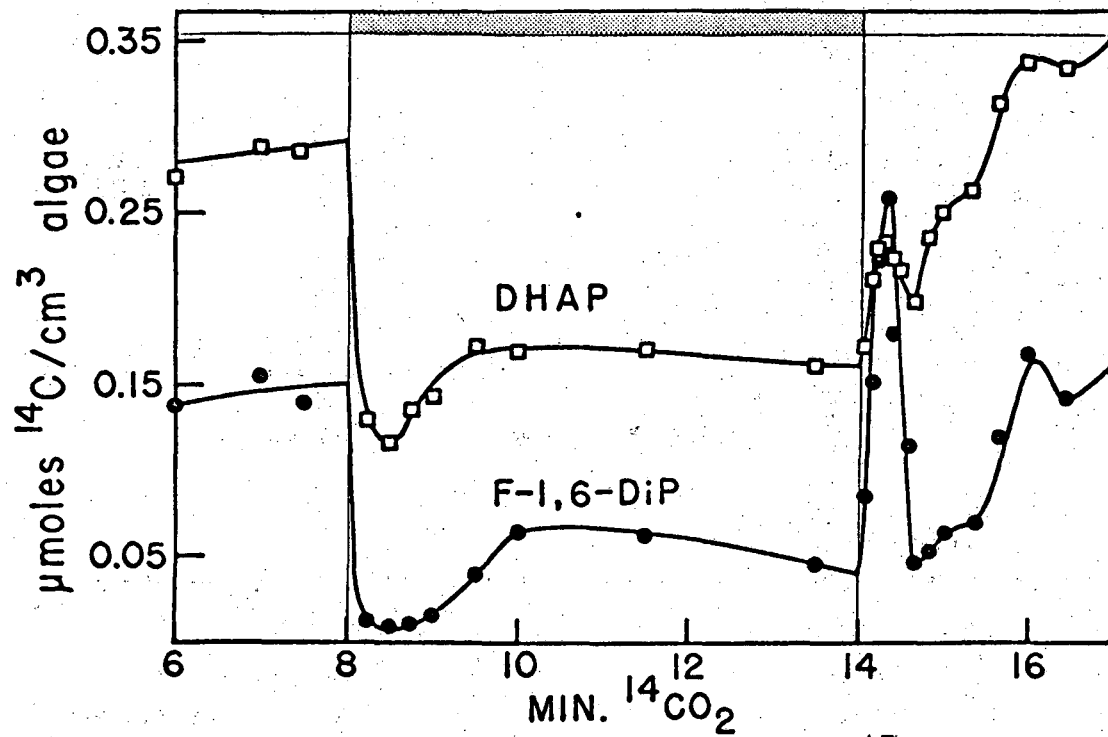


Fig. 4

XBL 674-1041

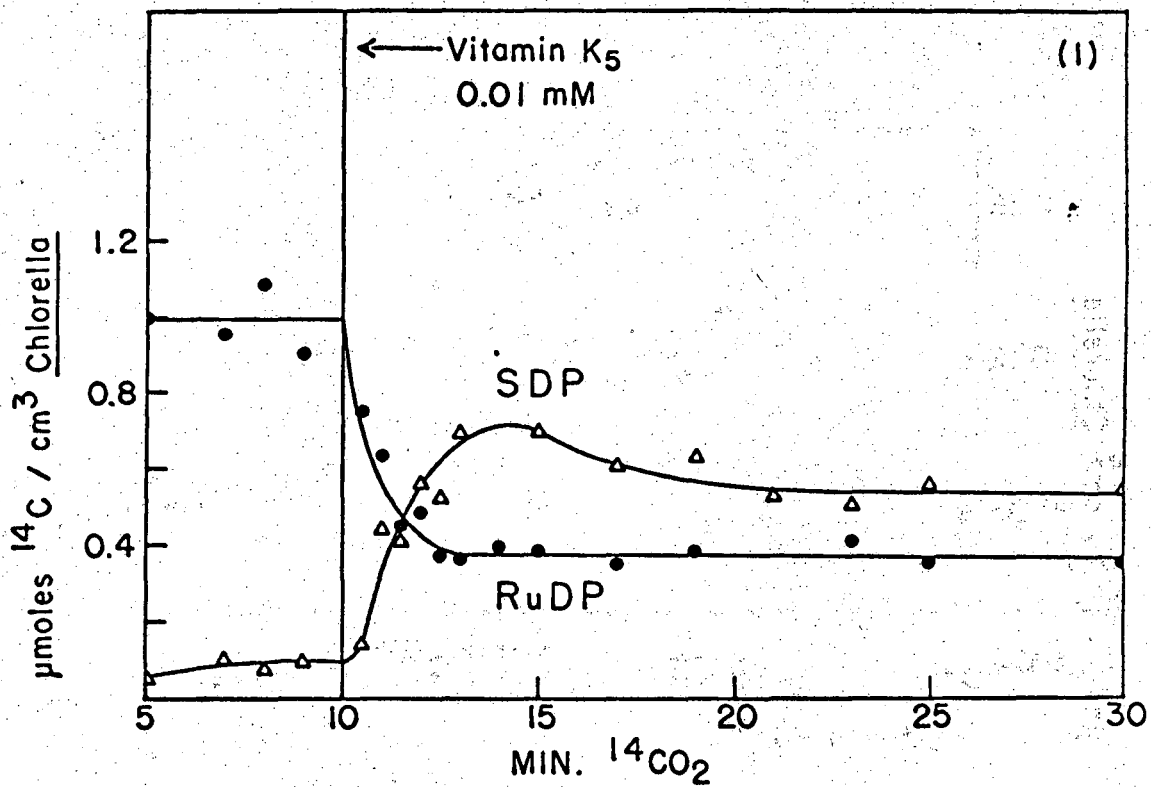


Fig. 5

XBL 687-4293

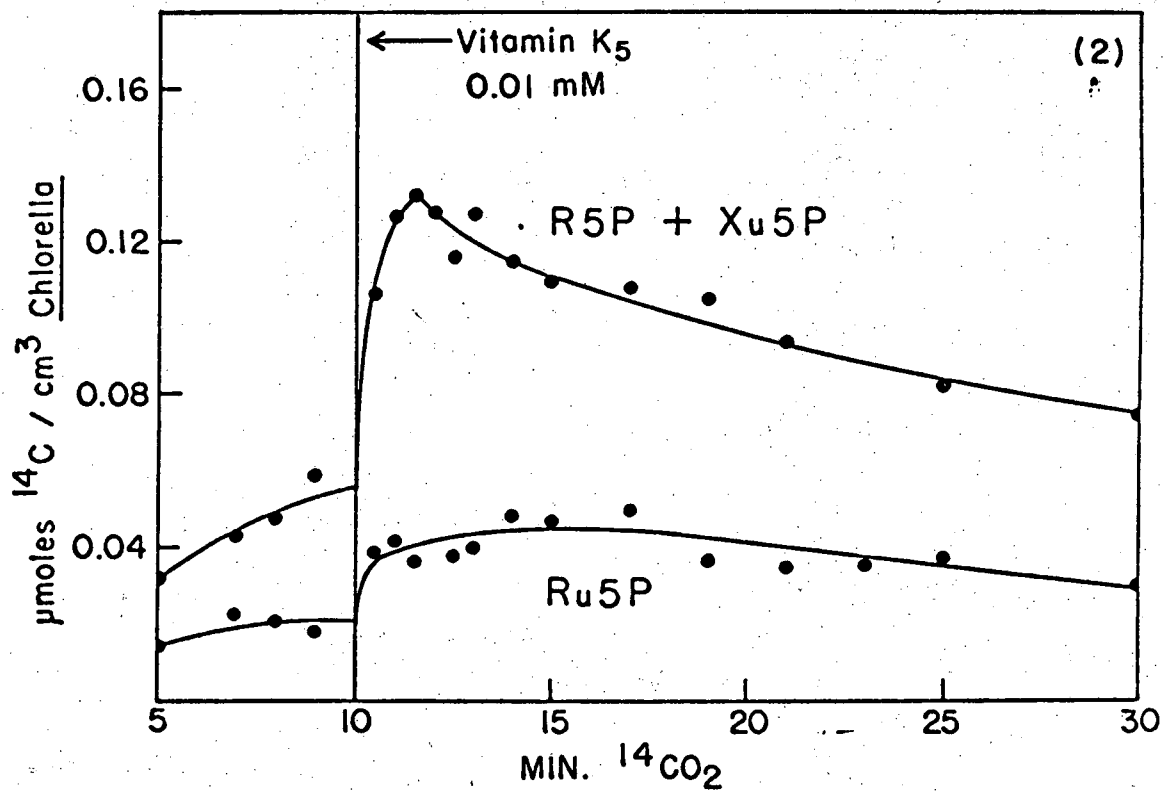


Fig. 6

XBL 687-4292

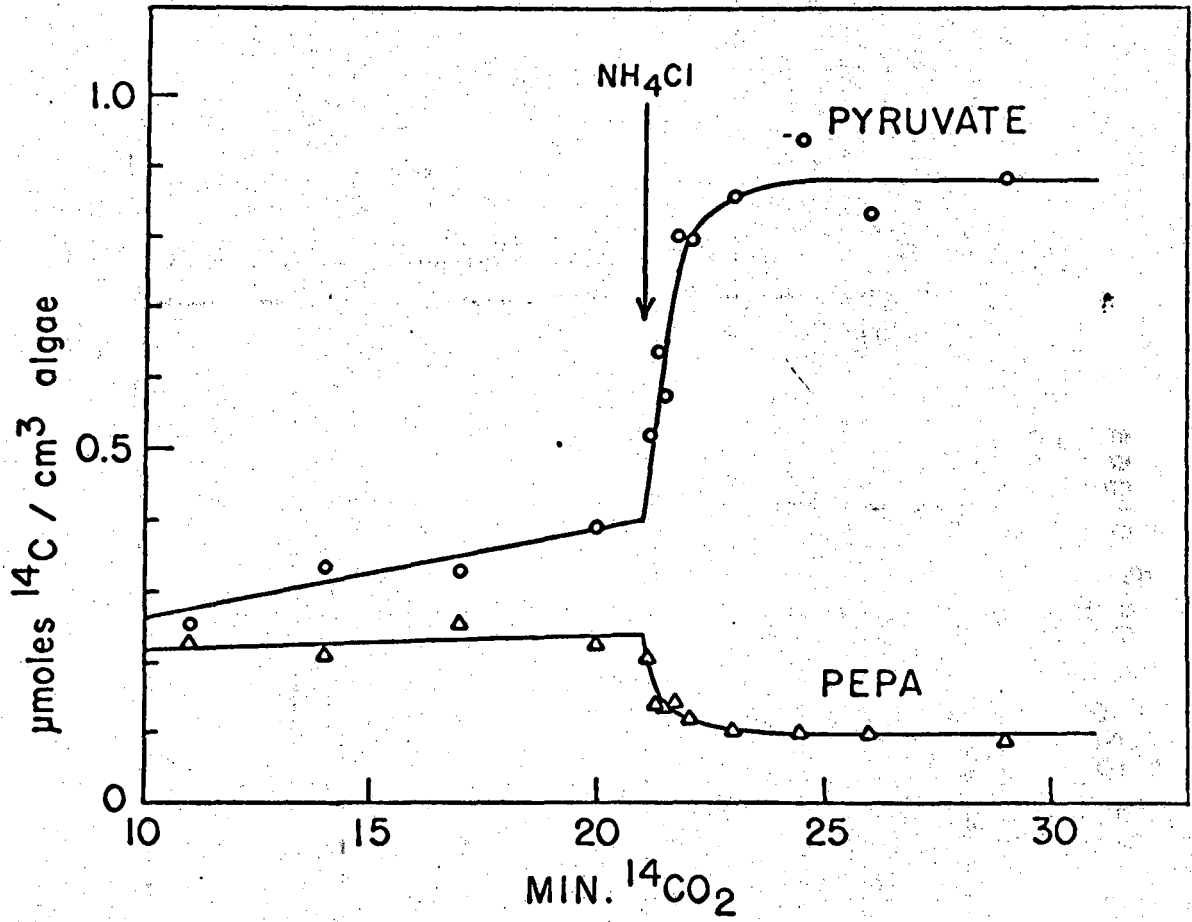


Fig. 7

XBL716-5194

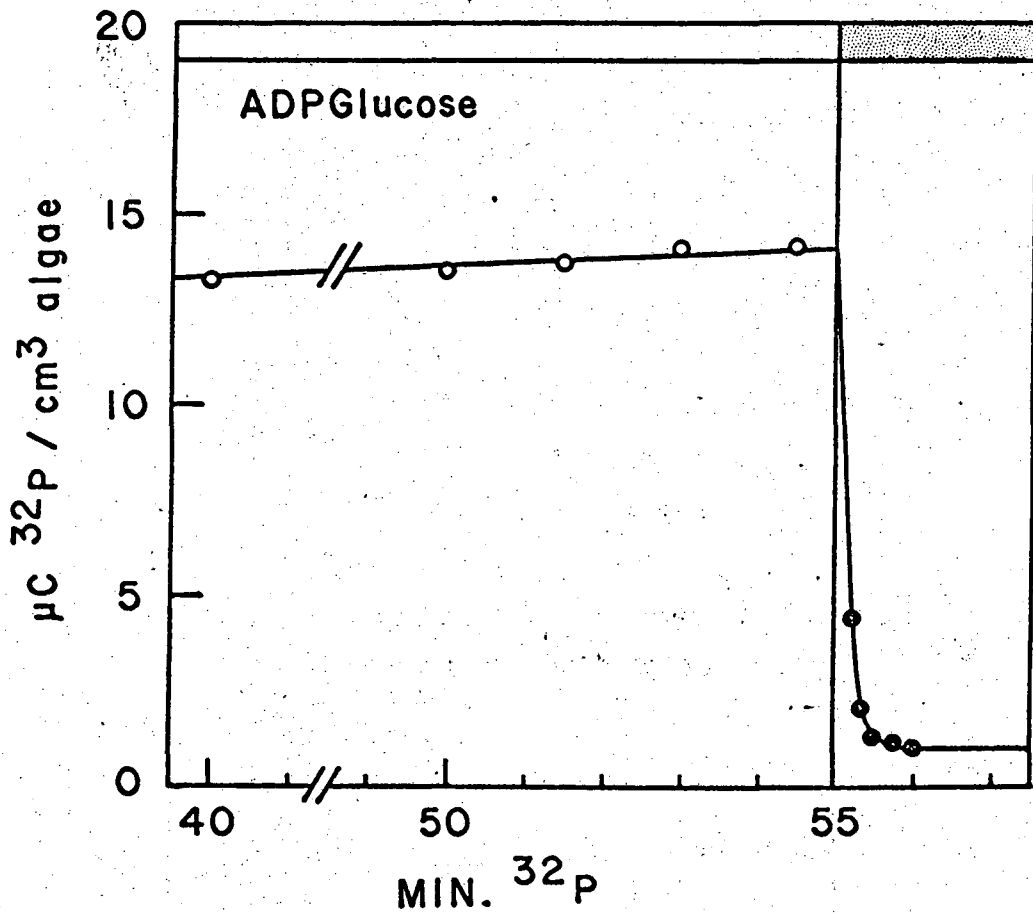


Fig. 8

XBL 716-5210

LEGAL NOTICE

This report was prepared as an account of work sponsored by the United States Government. Neither the United States nor the United States Atomic Energy Commission, nor any of their employees, nor any of their contractors, subcontractors, or their employees, makes any warranty, express or implied, or assumes any legal liability or responsibility for the accuracy, completeness or usefulness of any information, apparatus, product or process disclosed, or represents that its use would not infringe privately owned rights.

TECHNICAL INFORMATION DIVISION
LAWRENCE BERKELEY LABORATORY
UNIVERSITY OF CALIFORNIA
BERKELEY, CALIFORNIA 94720