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Publication Date
1967-08-01

UCRL-17748

Cy. 2

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The necessity for metabolic regulation of carbon reduction pathways in photosynthesis became apparent as the result of two discoveries which have altered earlier concepts of photosynthesis. The first of these was the recognition that direct products of photosynthesis include not only carbohydrates, but also amino acids and other products. The other discovery was / exchange of molecules of some intermediate compounds of
that there is an
photosynthesis with molecules of the same compounds involved in glycolysis.

Some of the earliest studies of photosynthesis with $^{14}CO_2$ revealed labeling of amino acids and fats at times comparable to those required for the labeling of sugars.[1] Later quantitative tracer studies with $^{14}CO_2$ and photosynthesizing Chlorella pyrenoidosa showed that alanine and other amino acids are formed directly from intermediates of the basic carbon reduction cycle,[2] and that their rate of labeling with ^{14}C can account for as much as 30% of the total $^{14}CO_2$ incorporation during the first minutes of photosynthesis.[3] During that same period only about one-third as much ^{14}C was found in sucrose, a major carbohydrate product of photosynthesis in Chlorella. Benson and coworkers[4] have shown that galactolipids are rapidly labeled by Chlorella, with 40% of the ^{14}C label of the lipids appearing in the fatty acid moieties after 5 min photosynthesis in Chlorella with $^{14}CO_2$.

This direct formation of amino acids and fatty acids from intermediates of the photosynthetic carbon reduction cycle makes these compounds equivalent in some respects to free sugars as immediate products

of carbon reduction in photosynthesis. It seems likely that some regulation of the flow of carbon from the basic cycle to the production of these compounds must be required in order to maintain a balance between their syntheses and their utilization in the further syntheses of macromolecules.

Green plant cells pass through various stages of development requiring synthesis of changing proportions of fats, proteins, carbohydrates and other macromolecules such as nucleic acids. Cells in the mature leaves of such plants as sugar beet may produce almost exclusively carbohydrate for translocation to other parts of the plant. Yet in young, rapidly growing leaves of these same plants, a considerable portion of the photosynthetically reduced carbon must be allocated to the production of proteins and lipids and other materials required for the building of the chloroplasts themselves and of the new cells.

To a large extent, of course, the regulation of these syntheses of macromolecules will be accomplished through the control of enzyme synthesis, according to mechanisms that are rapidly being discovered. Nevertheless, there would seem to be a need for a dynamic metabolic regulation of the pool sizes of these compounds. This regulation should be capable of rapid response to changing environmental conditions and should not be dependent upon synthesis of new enzymes. In short, the type of regulation needed to maintain reasonable pool sizes for small molecules produced directly from the photosynthetic carbon reduction cycle probably is dynamic regulation of key enzymes at or near the branch points in the cycle itself.

The second development which focused attention on the need for metabolic regulation was the finding that the intermediate compounds of photosynthesis and glycolysis in green cells are not completely isolated from one another. Earlier studies with Chlorella and $^{14}CO_2$, as well as ^{14}C-labeled glucose, had suggested that there is some separation between certain pools of intermediates of the photosynthetic carbon reduction cycle and of glycolysis and respiration.[5] However, it is now clear that there is an important exchange between some pools of photosynthesis and of glycolysis. Heber and Willenbrinck[6] reported that some labeled intermediates of the photosynthetic carbon cycle are transported from the chloroplast to the cytoplasm in Elodea. Based on his experiments with Elodea and Spinacia, Heber[7] concluded that the phosphoglyceric acid (PGA)*, dihydroxyacetone phosphate (DHAP), and fructose-1,6-diphosphate (FDP) "function as transport metabolites in photosynthesis." While those results were obtained by a post mortem "non-aqueous" isolation of chloroplasts following photosynthesis with tracers by the intact leaf, the findings have in general been confirmed by other techniques described below.

In the meantime, studies of ^{14}C-and ^{32}P-labeling of metabolites by Chlorella photosynthesizing in light and respiring in the dark had demonstrated a clear interaction between the intermediate compounds of photosynthesis and glycolysis.[8] The results of a similar study are shown in Fig. 1. After some 14 min of photosynthesis with $^{14}CO_2$ and 34 min with ^{32}P-labeled phosphate, PGA was saturated with respect to labeling by each of these isotopes. When the light was turned off and the flow of electrons and ATP from the photochemical apparatus stopped, the reduction

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*A list of abbreviations used in this paper is given with Fig. 2.

of PGA abruptly ceased, leading to a rapid rise in both ^{32}P and ^{14}C label. After about 30 sec the level of PGA declined, since the photosynthetic carboxylation reaction had by then stopped and PGA was no longer being formed. The decline in the level of labeled PGA is due to its conversion to other products such as alanine and its consumption by respiration via the tricarboxylic cycle (see Fig. 2).

After about 2 min the level of ^{32}P-labeled PGA rises to a steady-state level in the dark which is higher than the steady-state level in the light, while the ^{14}C-labeling of PGA continues to decline. This shows that the doubly-labeled PGA is being replaced by newly formed, singly-labeled PGA made from the glycolysis of unlabeled carbohydrate stores. Glycolysis of course introduces the same ^{32}P label as photosynthesis, since in each case the phosphate group is derived from a rapidly turning-over pool of ATP, whether the latter is formed by photosynthetic phosphorylation or oxidative phosphorylation.

Up to this point the results could be interpreted as indicating isolated pools of PGA for photosynthesis and glycolysis. However, if this were the case, when the light is again turned on the glycolytic pool of PGA should be unaffected and the ^{32}P-labeling should not drop significantly more than the ^{14}C label. The ^{32}P label of the PGA drops very much more than the ^{14}C label. This drop indicates that all of the ^{32}P-labeled PGA is immediately affected by the light. Thus, photosynthesis acts on the entire labeled pool of PGA. This data clearly established the fact that PGA, formed in the dark by glycolysis, can be immediately used in the photosynthetic carbon reduction cycle when the light is turned on.

With the interchangeability of PGA between photosynthesis and glycolysis complicating the kinetic studies, it became increasingly urgent that we perform parallel studies of photosynthesis in isolated chloroplasts . Chloroplasts capable of photosynthetic reduction of carbon dioxide at only 20% (usually much less)[9] of the rates to be expected _in vivo_ did not appear to be suitable for quantitative studies, particularly of regulatory mechanisms. By modifying a number of conditions in isolation and incubation, it was possible to obtain rates of carbon dioxide assimilation by isolated spinach chloroplasts approaching _in vivo_ rates for healthy leaves, at least for 10 or 15 min.[10] However, when the labeling of various products of this photosynthesis by isolated spinach chloroplasts were measured,[11] it was apparent that the levels of certain intermediates of the photosynthetic carbon reduction cycle, particularly DHAP, were abnormally high, as compared with levels seen with intact plants. The reason for this difference became clear when we studied the distribution of intermediate products between the chloroplasts themselves and the supernatant solution in which the chloroplasts had been suspended.[11,12] Over 80% of the carbon fixed appeared in the suspending medium. Of this, by far the greater portion was in PGA and DHAP, with substantial amounts appearing also in FDP, sedoheptulose-1,7-diphosphate (SDP), and glycolic acid. Ribulose-1,5-diphosphate (RuDP), fructose-6-phosphate (F6P), glucose-6-phosphate (G6P), and sedoheptulose-7-phosphate (S7P) were well retained in the chloroplasts.

Thus, there is a striking differential behavior in the migration of photosynthetic intermediates from chloroplast to suspending medium. In general, those compounds of the photosynthetic carbon reduction cycle

following the carboxylation reaction and preceding the diphosphatase

reaction (see Fig. 2) tend to diffuse from the chloroplast to the

medium, while those compounds coming after the diphosphatase reaction

and preceding the carboxylation reaction (except for pentose monophos-

phates) are well retained inside the chloroplasts.

Given the interaction between the intermediates of photosynthesis

and glycolysis and the demonstrated movement of intermediates between

chloroplasts and cytoplasm, it seemed clear that some form of light-dark

dynamic metabolic regulation of key enzymes of photosynthesis and gly-

colysis is required.

Direct kinetic evidence for light-dark changes in the activities

of enzymes of the carbon reduction cycle came from the same light-dark

studies with Chlorella in the presence of ^{32}P and ^{14}C that were reported

above.[8] The level of FDP plus SDP in light and dark is shown in Fig. 3.

When the light is first turned off, these sugar diphosphates drop in

concentration to nearly zero, as would be expected from the fact that

light is no longer supplying cofactors for the reduction of PGA to

triose phosphate, from which these sugar diphosphates are formed.

However, the level of FDP plus SDP then rises, passing through a maxi-

mum to a new steady-state dark level. This result clearly suggests

that the diphosphatase has become inactive and that a phosphofructokinase

has been activated in the dark, and is utilizing ATP formed by oxidative

phosphorylation. Note also that the sugar diphosphates formed in the

light are made partly from endogenous sugars that are not fully labeled

with ^{14}C.

The levels of ATP, as well as UTP and ADP, in light and dark are

shown in Fig. 4. It can be seen that while the ATP initially drops

rapidly when the light is turned off, it soon rises to a high dark
steady-state level due to oxidative photophosphorylation. Thus, there
is ample ATP for the PFkinase reaction.

The time required for activation of the diphosphatase reaction
in light was revealed by a detailed kinetic experiment with Chlorella
in which, following a period of steady-state photosynthesis with $^{14}CO_2$
and a period of darkness, the light was turned on again and samples
were taken at 10-sec intervals for the next 2 min. In Fig. 5 it can be
seen that the level of FDP rises very sharply for 30 sec. We attribute
this rapid rise to the onset of reduction of PGA due to cofactors from
the light reactions, together with the inactivity of the diphosphatase
following a period of darkness. After 30 sec the diphosphatase has
been activated and the level of fructose diphosphate then drops rapidly,
since the photosynthetic carbon reduction cycle is not yet fully operative
and the rate of reduction of PGA to triose phosphate has not reached the
steady-state level. Later, the labeled fructose diphosphate level rises
more slowly towards the steady-state level.

Fig. 5 also shows that the transient increase in DHAP is much
smaller than that of FDP. This means that the transient peaks are not
due merely to a wave of carbon coming from the sudden reduction of a
large pool of PGA. If that were the case, the transient peak in DHAP,
which precedes FDP in the cycle, would be higher than the peak in FDP.
Instead, the smaller DHAP peak is a reflection of the higher FDP peak,
and indicates an active, reversible aldolase reaction in light and dark.

Also, if the transient peaks were merely wave phenomena induced by
the sudden reduction of PGA, one would expect the wave to pass on to

the next intermediate in the cycle, F6P, but at a diminished magnitude. What actually happens can be seen in Fig. 6, which compares the behavior of labeled F6P with that of FDP. The level of F6P initially drops while the diphosphatase is inactive and only begins to rise at 30 sec or precisely the time when the diphosphatase has suddenly become activated. Once the diphosphatase is activated there is a transient peak in the levels of F6P before steady state is achieved. This is due to the sudden removal of the diphosphatase bottleneck, allowing carbon from the accumulated FDP to flow into the sugar monophosphate pool. Precisely the same situation is seen with SDP and S7P (Fig. 7). The only difference is in the steady-state pool sizes of the compounds involved. This similarity in the transient peaks of FDP and SDP strongly suggests an identical mechanism of regulation and quite possibly that the same diphosphatase operates for both reactions.

The possibility that the transient peak in FDP and SDP could be caused by residual dark PFkinase activity seems unlikely. PFkinase activity during the first seconds of light should not be greater than the dark activity. The level of ATP does not rise more than 15% when the light is turned on (Fig. 4). The level of F6P, as just mentioned, initially drops.

Evidence for the light-dark regulation of the carboxylation reaction also came first from the light-dark studies of <u>Chlorella pyrenoidosa</u> with $^{14}CO_2$ and $H^{32}PO_4^{-2}$, described earlier.[8] When the light was turned off, the level of the carboxylation substrate, RuDP, fell rapidly at first. After 2 min darkness, the rate of fall in RuDP level was no longer proportional to the RuDP level, indicating that the activity of the enzyme for this reaction had diminished.

Further evidence for the light activation of the carboxylation reaction came from studies of isolated spinach chloroplasts.[11] Fig. 8 shows that when the light was turned off following a period of photosynthesis with spinach chloroplasts the level of ribulose diphosphate at first dropped rapidly but then leveled off at virtually a constant value in the dark, indicating that the carboxylation reaction was no longer active. When the light was turned on again the level of RuDP rose very rapidly due to the inactivity of the carboxylation reaction and to the production of ATP from the light which is required by the ribulose phosphate kinase to convert Ru5P to RuDP. Subsequently the level of RuDP declined towards a steady-state level.

An even more impressive proof of the inactivity of the carboxylation reaction in the dark is shown in Table I.[13] In this light-dark-light experiment with spinach chloroplasts photosynthesizing with $^{14}CO_2$, additions of ATP and of ATP + R5P were made to separate flasks of chloroplasts just after the light was turned off. As can be seen, the effect of the additions was to maintain the level of RuDP in the dark at its previous light level. In spite of this there was no fixation in the dark following a period of very active fixation of $^{14}CO_2$ in the light. When the light was again turned on, a smaller but substantial rate of $^{14}CO_2$ fixation was seen in all three flasks. Thus, in this experiment both substrates, RuDP and $^{14}CO_2$, were present in the chloroplasts in the dark but no carboxylation occurred. Nevertheless, the system was not permanently inactivated, for subsequent light was able to stimulate high fixation rates.

The reason for light-dark diphosphatase regulation seems obvious. Given the diffusion of ATP and of some sugar phosphates between chloroplasts

Table I

	1st Light Period			Dark Period		2nd Light Period	
	Rate*	RuDP*		Rate	RuDP	Rate	RuDP
Control	143	0.08		-3	0.02	82	0.23
+ ATP	154	0.10		-10	0.07	104	0.37
+ ATP, R5P	145	0.08		-3	0.08	86	0.27

*Rates are given in μmoles $^{14}CO_2(mg\ Chl\ hr)^{-1}$; RuDP concentration is given in μmoles $^{14}C(mg\ Chl)^{-1}$.

and cytoplasm, phosphofructokinase and fructose diphosphatase should not be active at the same time, lest they together operate as an ATPase.

The reasons for regulation of the carboxylation reaction were not so apparent. Green cells contain a highly active oxidative pentose phosphate cycle. This cycle is immediately activated when the light is turned off, as indiciated by the data shown in Fig. 9, obtained during the previously described steady-state experiment with photosynthesizing <u>Chlorella</u> in the presence of $^{14}CO_2$ and ^{32}P-labeled phosphate. As soon as the light is turned off both the ^{32}P and ^{14}C label of 6-phosphogluconic acid rise rapidly and are maintained at an appreciable level during the entire dark period. When the light is again turned on the formation of 6-phosphogluconic acid ceases.

The site of the operation of this oxidative pentose phosphate cycle may well be within the chloroplasts. It was found earlier[14] that the addition of vitamin K to photosynthesizing <u>Chlorella</u> causes the immediate appearance of 6-phosphogluconic acid even while the light is on. Vitamin K is supposed to cause the short circuiting of electrons being transported through the photoelectron transport system, thereby leading to a cyclic photophosphorylation and preventing the reduction of $NADP^+$.

There is evidence[15] that $NADP^+$ and NADPH are not transported between chloroplasts and cytoplasm. Since the effects caused by vitamin K or by interruption of the light seem to be likely results of interruption of photoelectron transport, the sudden appearance of 6-phosphogluconic acid looks like an indication of operation of the oxidative pentose phosphate cycle within the chloroplasts.

While there is probably no oxidative electron transport system within the chloroplasts, NADPH produced by the operation of the oxidative pentose phosphate cycle could be used for biosyntheses within the chloroplasts—for example, the conversion of sugars to fatty acids. Apparently ATP diffuses readily from the cytoplasm into the chloroplasts,[12] and ATP, as already mentioned, is rapidly produced by oxidative phosphorylation in the cytoplasm. Thus, biosynthesis can proceed inside the chloroplasts in the dark with ATP from the cytoplasm and NADPH from the operation of the oxidative pentose phosphate cycle in the chloroplasts.

If this hypothesis is correct, then there should be a regulatory mechanism which in the dark would block the transformation of pentose monophosphates to RuDP to PGA. Only one of these two steps need be inactive in the dark, and it appears that it may be the carboxylation step from ribulose diphosphate to PGA. We have already demonstrated (Table I) that with added ATP, pentose monophosphates can be converted to ribulose diphosphate in the dark.

What is the mechanism of the light-dark regulation of the diphosphatase and ribulose diphosphate reactions? Isolated enzymes which catalyze these reactions are: D-fructose-1,6-diphosphate, 1-phosphohydrolase, 3·1·3·11 (hexose diphosphatase), and 3-phospho-D-glycerate carboxy-lyase (dimerizing), 4·1·1·39 (ribulosediphosphate carboxylase). Both enzymes are characterized by a pH optimum which is higher than normal physiological pH for chloroplasts and a requirement for high level of magnesium ion.[16,17] Activation of these enzymes by light could

be the result of movements of magnesium and hydrogen ions through membranes. Dilley and Vernon[18] reported a light-dependent uptake of H^+ and an efflux of K^+ and Mg^{++} ion by isolated spinach chloroplasts. This reported ion flow might appear at first to be contrary to that required for enzyme activation. However, the light-driven flow of ions is across the thylakoid membranes within the chloroplasts, and probably leads to a flow of hydrogen ions from the stroma region into the membrane-enclosed thylakoids, and a flow of magnesium ions into the stroma. Thus measurements of pH and of metal ions in the suspending medium may well reflect changes occurring within the stroma, particularly if the chloroplasts are broken or "leaky".

Another indication that light-induced ion flow may be responsible for light-dark regulation of the enzymes is to be found in the fact that certain fatty acids and fatty acid esters simultaneously inhibit photophosphorylation, the carboxylation reaction, and the diphosphatase reaction.[19] These inhibitions, which are reversible, are thought to be accomplished by some indirect effect of alteration in the properties of the chloroplast membranes. A likely alteration in membrane properties would be in the light-induced ion pumping capacity. Photophosphorylation is thought to require a pH gradient across the thylakoid membranes,[20] and, like the other inhibited enzymes, photophosphorylation requires magnesium ion.[21]

While light-induced ion pumping provides a plausible mechanism for the light-dark regulation of the diphosphatase and carboxylation reactions, we visualize a different, but as yet unknown, mechanism as providing a

fine regulation of the diphosphatase reaction when plants are photo-synthesizing with the lights on. At all times while photosynthesis is proceeding there would be sufficient diphosphatase activity to permit the cycle to regenerate pentose phosphates at a steady-state rate suffi-cient to provide for the operation of the basic photosynthetic carbon reduction cycle. However, additional diphosphatase activity would be so adjusted as to either pile up the excess fixed carbon at the level of PGA, DHAP, and FDP, which would be used for biosynthesis of proteins, fats, and other materials; or, with greater diphosphatase activity, the excess carbon would be accumulated in fructose-6-phosphate which could then be converted to carbohydrates (Fig. 2). This type of regulation would thus satisfy the changing requirements for biosynthesis in response to physiological change, such as growth and development.

We are inclined to make two predictions about this mechanism. First, it should be related to hormonal or genetic control and may well involve the adjustment of a regulatory factor by the activity of an inducible enzyme. Second, this regulatory factor may exert its effect through some quantitative change in the light activation. In this way it could pro-vide the fine, or vernier, control needed to insure the precise diphos-phatase activity to keep the basic carbon cycle running while at the same time adjusting the supply of carbon to biosynthetic pathways.

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FIGURE CAPTIONS

Fig. 1. Levels of ^{14}C and ^{32}P-labeled PGA in <u>Chlorella</u> <u>pyrenoidosa</u>
during photosynthesis and respiration.

Fig. 2. Relations between photosynthesis, glycolysis, and synthesis
of macromolecules, and regulatory control points.

Compounds of the photosynthetic carbon reduction cycle above
the dotted line are those which tend to be retained in the
chloroplasts, those below the dotted line have been found to
be readily transported from chloroplasts. Intermediate com-
pounds in paths other than the photosynthetic carbon reduction
cycle are not shown.

Abbreviations used in this figure and in the text are: PGA,
3-phosphoglyceric acid; DHAP, dihydroxyacetone phosphate;
Ga3P, glyceraldehyde-3-phosphate; FDP, fructose-1,6-diphosphate;
F6P, fructose-6-phosphate; SDP, sedoheptulose-1,7-diphosphate;
S7P, sedoheptulose-7-phosphate; E4P, erythrose-4-phosphate;
Xu5P, xylulose-5-phosphate; R5P, ribose-5-phosphate; Ru5P,
ribulose-5-phosphate; RuDP, ribulose-1,5-diphosphate; G6P,
glucose-6-phosphate.

Fig. 3. Levels of ^{14}C-labeled FDP plus SDP in <u>Chlorella</u> <u>pyrenoidosa</u>
during photosynthesis and respiration.

Fig. 4. Levels of ^{32}P-labeled ATP, UTP, and ADP, in <u>Chlorella</u> <u>pyrenoidosa</u>
during photosynthesis and respiration.

Fig. 5. Levels of ^{14}C-labeled FDP and DHAP, in <u>Chlorella</u> <u>pyrenoidosa</u>,
during photosynthesis and respiration.

FIGURE CAPTIONS (Cont.)

Fig. 6. Levels of ^{14}C-labeled FDP and F6P, in <u>Chlorella pyrenoidosa</u>, during photosynthesis and respiration.

Fig. 7. Levels of ^{14}C-labeled SDP and S7P, in <u>Chlorella pyrenoidosa</u>, during photosynthesis and respiration.

Fig. 8. Levels of RuDP labeled with ^{14}C and ^{32}P in isolated spinach chloroplasts, photosynthesizing, and in the dark.

Fig. 9. Levels of 6-phosphogluconic acid labeled with ^{14}C and ^{32}P, in Chlorella pyrenoidosa, during photosynthesis and respiration.
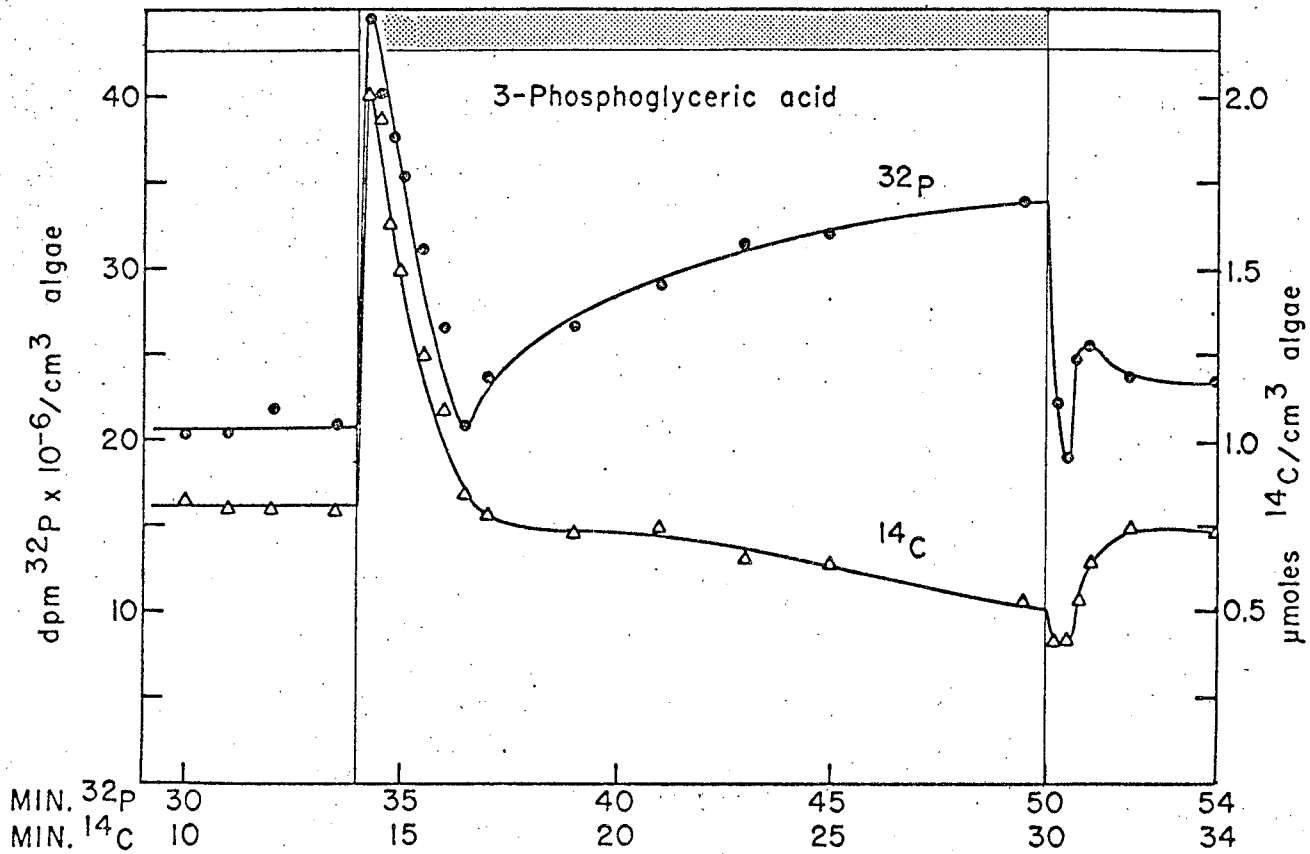
Fig. 1
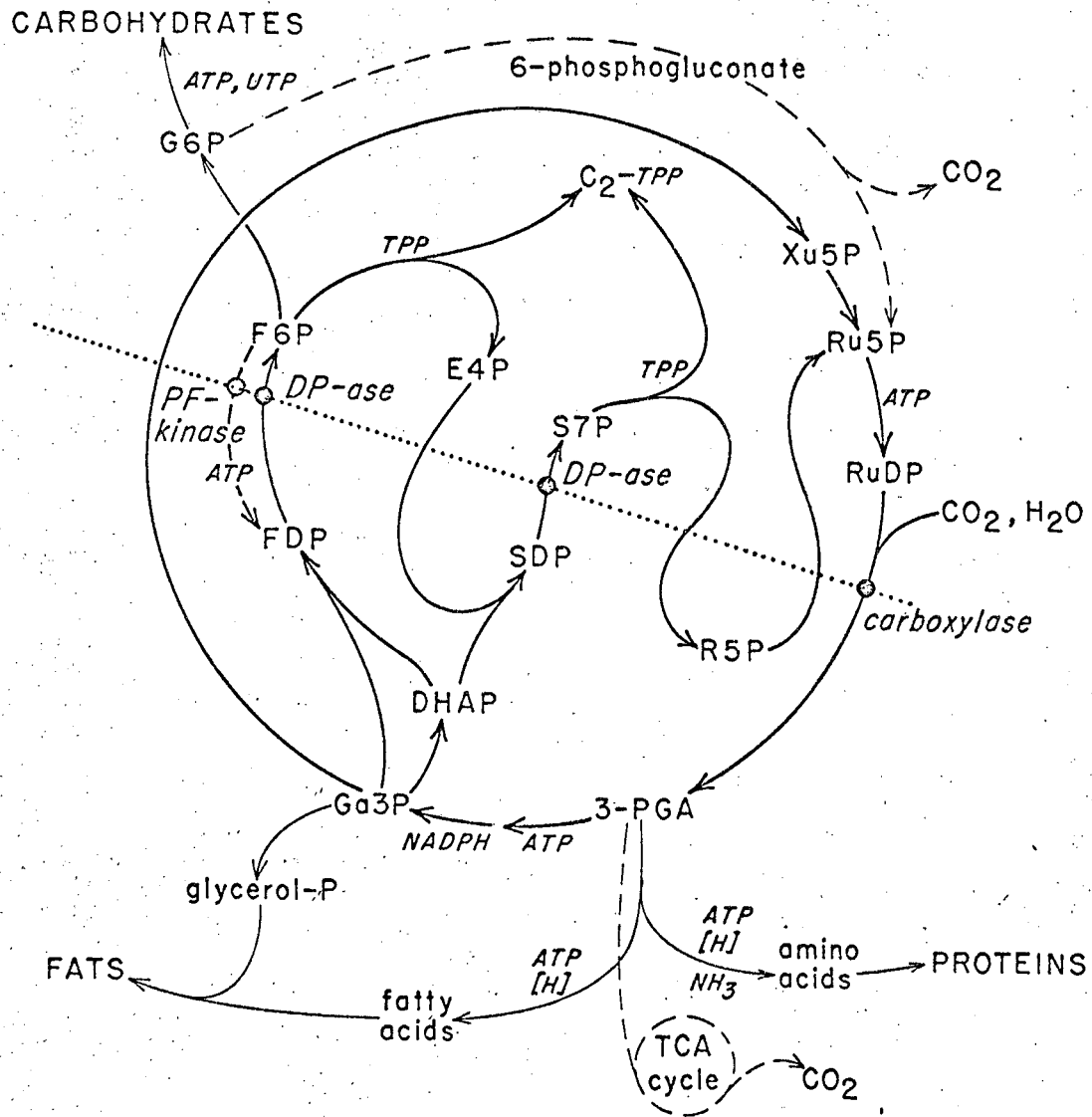
Fig. 2

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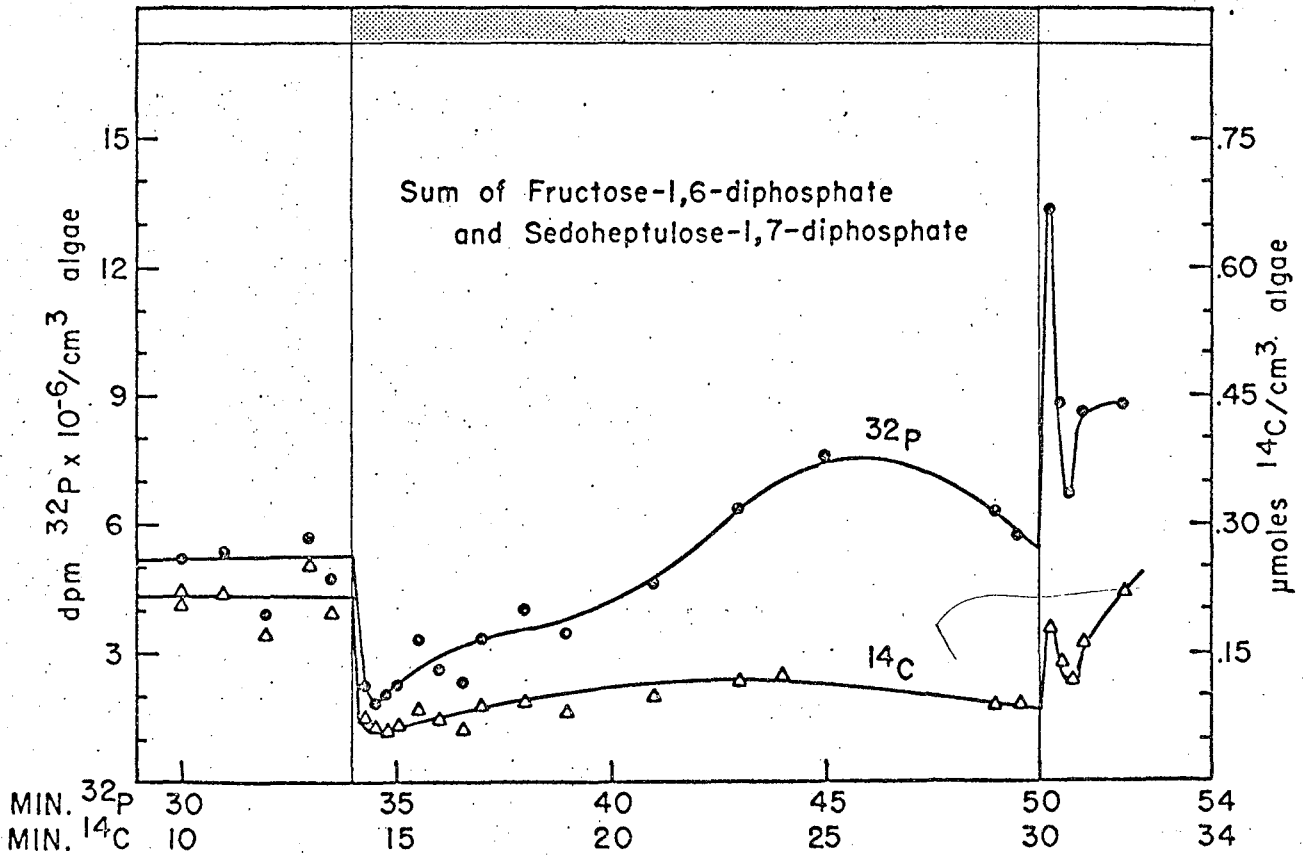
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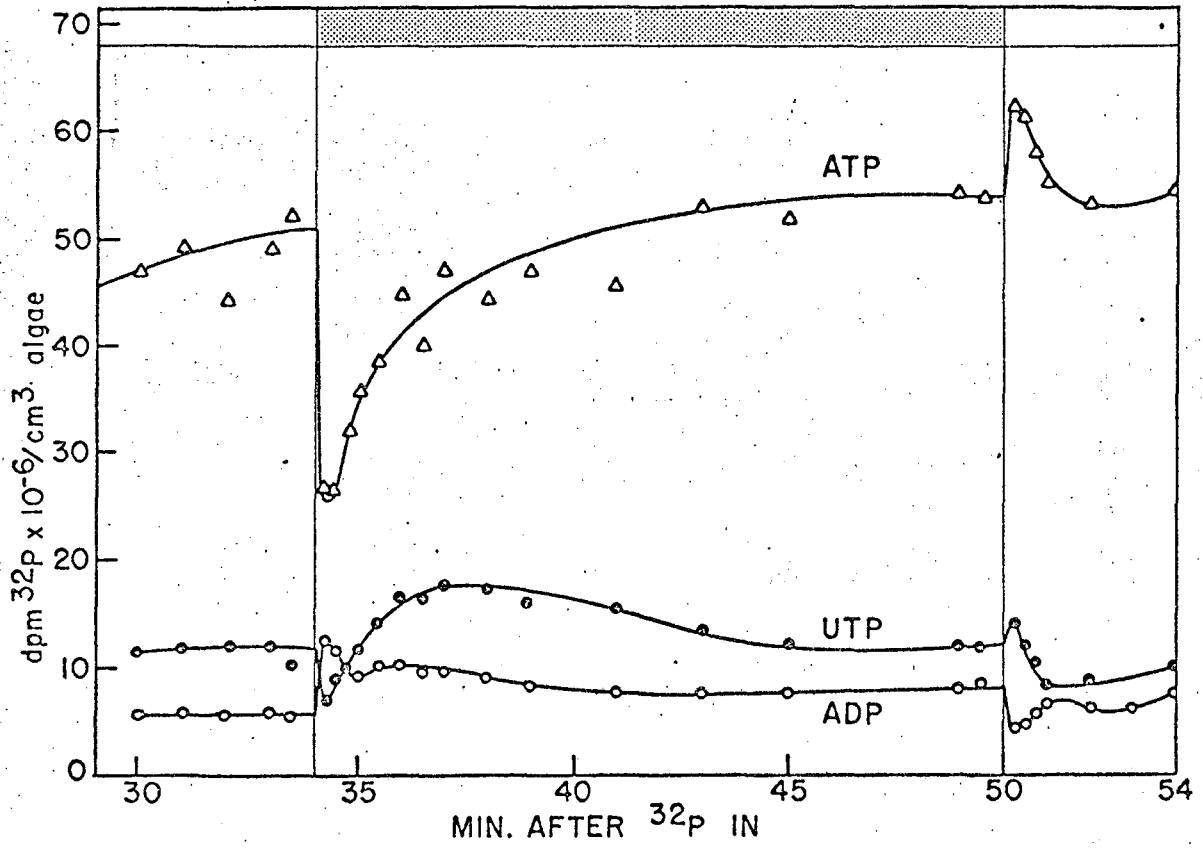
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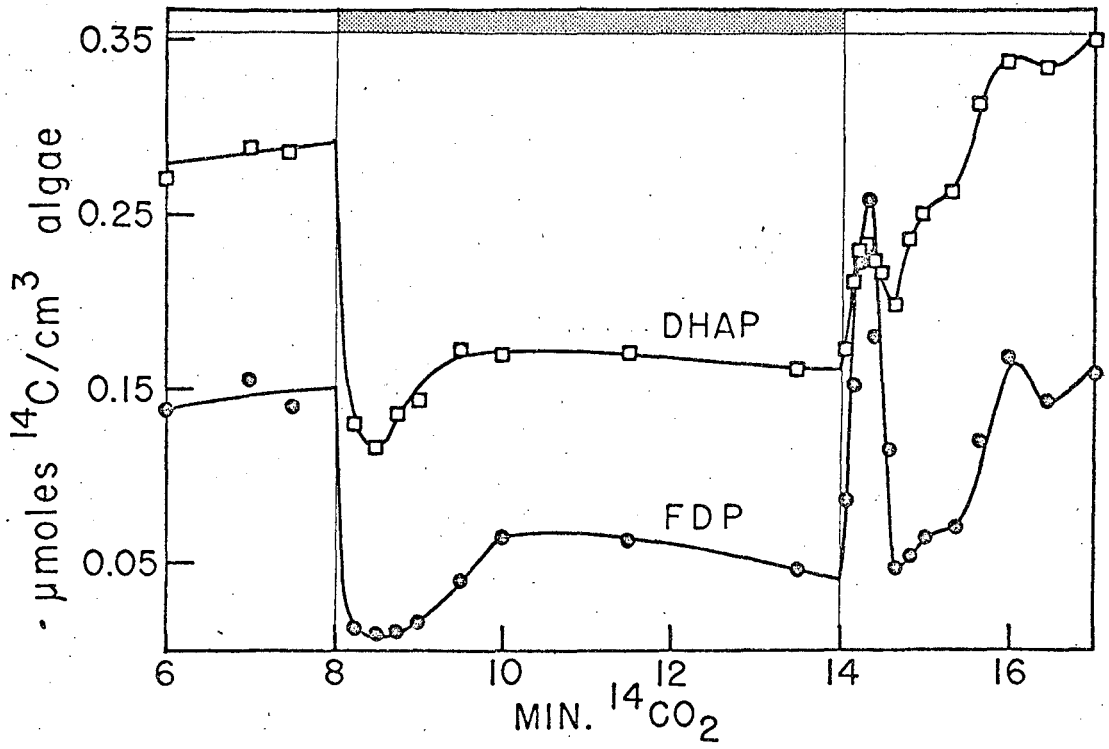
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Fig. 5

XBL 674-1035

Fig. 6

Fig. 7

Fig. 8

Fig. 9

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