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INHIBITOR STUDIES ON THE PHOTOSYNTHETIC CARBON REDUCTION CYCLE IN *CHLORELLA PYRENOIDOSA*

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SUMMARY

Transient changes in the levels of intermediates of the photosynthetic carbon reduction cycle induced by the addition of various chemical compounds to *Chlorella pyrenoidosa* photosynthesizing under steady-state conditions have been studied. Vitamin K_5, hexylresorcinol, DCMU, and CCCP were found to give rapid inhibition of photosynthesis, accompanied by rapid changes in the levels of intermediate compounds of the photosynthetic carbon reduction cycle. DCMU and CCCP produced effects similar to those seen during earlier light-dark transient studies. Vitamin K_5 produced effects which, for the most part, could be explained by assuming a diversion of electrons from the photoelectron transport system to cyclic photophosphorylation. Some of the observed results are best interpreted in terms of a separation of the site of the photosynthetic carbon reduction cycle from a site of other metabolic pathways. Hexylresorcinol reproduced some of the effects of each of the other inhibitors studied. The inhibition of the conversion of fructose and sedoheptulose diphosphates to their corresponding monophosphates was noted with both hexylresorcinol and Vitamin K_5.

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Much of the evidence leading to the formulation of the photosynthetic carbon reduction cycle (PSCR cycle) was obtained from kinetic studies of the in vivo incorporation of $^{14}CO_2$ during photosynthesis by unicellular algae[1]. Transient changes in the levels of labeled intermediates in the cycle caused by the change from light to dark[2] led to the formulation of the reduction of 3-phosphoglyceric acid (PGA) to triose phosphate and the conversion of ribulose-5-phosphate to ribulose-1,5-diphosphate (RuDP) as reactions requiring cofactors formed in the light. The formation of RuDP was presumed to require ATP, while the reduction of PGA was presumed to require both ATP and $NADPH_2$. The transient changes accompanying the reduction of CO_2 level from 1% to .003%[3] led to the formulation of the carboxylation reaction as a reaction which converts CO_2 + RuDP + H_2O $\longrightarrow$ 2 PGA.

Supporting evidence for the correctness of the PSCR cycle has come from studies of enzymic activities obtained by disruption of photosynthetic cells[4]. However, the inadequacy of enzymic activity for the catalysis of certain steps in the postulated cycle and the absence of activity for some steps in certain organisms[5-7] have been used as a basis for questioning the formulation of the PSCR cycle[8]. In a different interpretation, these reported enzymic inadequacies, together with certain peculiarities in the reported kinetics of carbon labeling in vivo, have been cited as evidence for the catalysis of the PSCR cycle by an organized or multifunctional enzyme system, possibly utilizing photochemically-produced cofactors other than reduced $NADPH_2$ and ATP[9]. The existence of an in vivo reductive carboxylation reaction has been suggested. An intimate structural relationship between the photoelectron transport system located in the chloroplast

lamellae and hypothetical multifunctional enzyme system for the carbon reduction cycle has been proposed.

In an attempt to obtain additional kinetic evidence from the in vivo system which might bear on these proposals, we have undertaken a series of studies to investigate the transient effect of the addition of various chemicals to the algae photosynthesizing with $^{14}CO_2$ under steady-state conditions. With the substances which penetrate the cell rapidly and produce immediate effects, rapid transient changes in the levels of intermediates of the carbon cycle can be seen. This type of chemically-induced change in the metabolite concentration provides a more direct kind of information about the mode of action of the chemical than does the preincubation of the whole cells with inhibitor followed by the application of tracer to the already damaged system. Observation of the immediate effects of a chemical on a steady-state system seems more likely to provide information about the primary site of action.

We have administered chemical compounds known to produce large effects on the photoelectron transport system or photophosphorylation system in isolated, or broken, chloroplasts. Not all such substances produced effects with whole cells, perhaps due to their failure to penetrate the cell wall. However, we have been able to find several substances which cause complete inhibition of photosynthesis within 1-2 minutes after their addition to the algae which were photosynthesizing under steady-state conditions. We have studied the resulting transient changes in the levels of radioactive intermediate compounds of the photosynthetic carbon reduction cycle and related substances.

It has been found that methyl lipoic and lipoic acids produce rapid reversible inhibition of photosynthesis accompanied by very rapid transient changes in the levels of intermedate compounds of the photosynthetic carbon reduction cycle[10,11]. Therefore we tested several other lipid-soluble compounds. Of these substances, hexylresorcinol caused rapid inhibition, and the transient changes produced by this substance were studied. We tested a number of other substances which, for one reason or another, might have been expected to affect photosynthesis, but these did not cause immediate effects.

METHODS

The unicellular algae, Chlorella pyrenoidosa, was grown in continuous culture tubes[12], harvested by centrifugation, washed once, and resuspended in the medium used for the photosynthesis experiment. The medium for the experiments with DCMU and CCCP was the nutrient solution described previously[13], whereas that for experiments with Vitamin K and hexylresorcinol (HexRes) was 10^{-3} M KH_2PO_4 without ammonia or trace metal ions added. The pH values for all experiments were close to 5.0.

The algae suspension (1% wet-packed volume/suspension volume) was placed in the steady-state photosynthesis apparatus which has been described previously[14]. This apparatus has provision for monitoring the levels of CO_2, O_2 and $^{14}CO_2$ in the gas phase of a closed system, in which the gas is made to recirculate through the algae suspension. The signals from these instruments are continuously recorded, and from their rate of change with time and the known volumes of the system, rates of photosynthesis can be calculated. Because of the time required for equilibration between gas and liquid phases, volume of the gas phase of the system and

in the instruments, and instrument time constants, approximately 30-60 seconds is required to see sudden changes induced in the rates of photosynthetic gas exchange.

With each chemical tested, preliminary experiments were carried out to determine the concentration required to obtain essentially complete inhibition. Where such inhibition was obtained, experiments with $^{14}CO_2$ as a tracer were then performed. Following a preliminary period of photosynthesis in 1-2% $^{12}CO_2$, $^{14}CO_2$ was introduced and its level and specific radioactivity maintained so that the photosynthetic steady state was not interrupted. Several 1 ml samples of algae suspension were taken into weighed test tubes containing 4 ml of methanol for killing, in order to establish the steady-state level of intermediate compounds. After 10 minutes or more of photosynthesis with $^{14}CO_2$, the inhibitor was added and samples were then taken periodically, as indicated in the results. Aliquot samples of the killed algal material were analyzed by two-dimensional paper chromatography and radioautography, as previously described[12,14]. The radioactivity of each compound was determined by means of the automatic spot counter[15].

RESULTS

The concentrations of various inhibitors used in this study and the time period required for visibly complete inhibition of photosynthesis by illuminated Chlorella pyrenoidosa are listed in Table I.

Partial recovery was observed with CCCP (10% after 10 minutes), with Vitamin K (20% after 25 minutes), and with hexylresorcinol (20% after 15 minutes), but not with DCMU. Since Vitamin K_5 solutions deteriorate upon standing at room temperature, solutions of this inhibitor were prepared immediately before use.

TABLE I

Concentrations and Inhibition Periods

Inhibitor	Concentrations $10^5 \times M$	Inhibition Times Uptake of CO_2 (seconds)	Inhibition Times Evolution of O_2 (seconds)
DCMU	1 [a]	70	70
CCCP	1	80	80
Vitamin K_5	10 [b]	80	30
HexRes	20	100	50

[a] With a DCMU concentration of 10^{-6} M, photosynthesis was about 65% inhibited within 6 minutes and 85% inhibited in 18 minutes. At 10^{-7} M inhibitor concentration, there was no significant effect. DCMU did not block respiration.

[b] The same concentration of phenazine methosulfate, a second known catalyst for cyclic photophosphorylation, was without effect on the rate of photosynthesis. Its addition led to the gradual development of a rich blue color in the mixture.

In addition to the inhibitors listed in Table I, the following compounds were tested and found to have negligible effect on the rate of photosynthesis by illuminated Chlorella at concentrations up to 3×10^{-4} M: ethanol, n-octyl alcohol, t-heptyl mercaptan, thioglycolic acid, trimethylene disulfide, magnesium perchlorate, eugenol, phenol, pyrocatechol, hydroquinone monomethyl ether, 1-hydroxy-2-amino-4-naphthalene sulfonic acid (added as its potassium salt), the herbicide "paraquat"[16], the electron-transfer reagent pyridine-4-carboxylato-pentamminecobalt(III) perchlorate and its N-methyl derivative[17]. Slight inhibition was observed with pyrocatechol (10% at 2×10^{-4} M), with resorcinol (35% at 2×10^{-4} M), and with hydroquinone (25% at 2×10^{-4} M), but these diols were much less effective inhibitors than hexylresorcinol.

Figures 1 and 2 show the levels of the various labeled compounds obtained from samples taken during the DCMU experiment; Figures 3 and 4 indicate the corresponding data for the CCCP experiment. "Micromoles of ^{14}C" designates the total quantity of carbon (as measured by the ^{14}C level) incorporated into the various species after introduction of $^{14}CO_2$ [14].

The changes in ^{14}C labeling patterns resulting from additions of these two inhibitors are very similar, and both follow closely the changes observed when the light is turned off during the light-dark transient study[13]. As when the light is turned off, the sudden rise in the PGA level is significantly greater than the drop in the level of ribulose diphosphate, and there is an equally sudden drop in fructose diphosphate. The UDPG level (not shown) remains very nearly constant. Among the monophosphates (Figs. 2 and 4), the addition of DCMU or CCCP

(or turning off the light) causes the level of glucose phosphate to increase while, at the same time, the levels of fructose and sedoheptulose monophosphates decrease.

The levels of the various labeled compounds derived from the inhibition experiment with hexylresorcinol are shown in Figures 5 and 6. In common with the previously mentioned inhibitors and with the light-dark transients, addition of hexylresorcinol causes a sharp rise in PGA level and a sharp drop in the level of ribulose diphosphate. Similarities are also found in the patterns of changes of sugar monophosphates. However, the levels of fructose and sedoheptulose diphosphate, after a very slight drop immediately following the addition of the inhibitor, rise steadily to values very much larger than their steady-state levels before inhibition. This feature is similar to that observed in experiments with Vitamin K, described below. However, with inhibition by hexylresorcinol, the levels of sedoheptulose diphosphate become comparable to those of fructose diphosphate, whereas with Vitamin K the fructose diphosphate level is by far the larger.

The pattern of changes accompanying inhibition by Vitamin K was quite different from that observed with inhibitors of the other classes. Figure 7 shows the radioautograph of a two-dimensional chromatogram of a sample taken from the reaction mixture and killed 11 minutes after addition of Vitamin K_5. Note the heavy streak of labeled polysaccharide lying along the lower edge of the chromatogram and coalescing with the origin. This feature is generally absent from the previously described inhibition experiments, unless an extended period of time was allowed to elapse between the addition of radiocarbon and inhibitor. At the

same time, activity in the sugar monophosphate area is unusually light, in contrast to experiments with other inhibitors in which a major fraction of the added radiocarbon is found in various sugar monophosphates. Finally, there is an additional spot, lying near the sugar monophosphate area but closer to the origin, which has not been reported in inhibitor studies of this type. This was identified as 6-phosphogluconic acid by cochromatography with an authentic sample of this acid. The unlabeled phosphogluconic acid was located by spraying with the Hanes-Isherwood solution[18] and exposing to sunlight. This acid did not appear in samples taken before addition of Vitamin K.

Figures 8 and 9 show the levels of labeled sugar monophosphates and diphosphates from the Vitamin K experiment, and Figure 10 indicates the levels of diphosphates and PGA. The most striking feature of the monophosphate curves is the very sudden drop in the levels of glucose, sedoheptulose, and and fructose monophosphates during the first minute after addition of inhibitor. The sugar diphosphates show a rapid drop in the same period, but it is followed by a rise which is very large for fructose diphosphate, smaller for sedoheptulose diphosphate, and only temporary (and not always observed) for ribulose diphosphate. PGA increases rapidly while all the sugar phosphates are falling; then PGA falls while fructose diphosphate (in particular) rises. About 30 seconds after addition of the inhibitor, the combined levels of fructose mono- and diphosphates are only about 50% of the fructose diphosphate level late in the experiment. Phosphogluconic acid appears a few seconds after addition of the inhibitor, and ribose phosphate graudally builds up until, 14 minutes after inhibition, it has become the major component in the monophosphate mixture. Measurable

levels of ribulose monophosphate were found in samples taken six or more minutes after addition of Vitamin K. These are not shown in Figures 8 and 9, but were, in all cases, about one-third of the levels of ribose monophosphate.

DISCUSSION

The transient changes in concentrations of metabolic intermediates induced by the addition of CCCP and DCMU, which are very similar to light-dark transient changes, provide a useful standard against which other types of chemically induced transient changes may be compared. The light-dark type of transients are characterized by a rapid drop to zero concentration of ribulose diphosphate. This drop may reasonably be presumed to result from the depletion of ATP or an equivalent form of high-energy phosphate compound derived from the photophosphorylation reaction of photosynthesis. The same depletion of ATP or its equivalent can account for the rapid transitory rise in PGA, since ATP is required in the initial step leading to the reduction of PGA. The assumed uncoupling of photophosphorylation by CCCP thus could be expected to give the light-dark transient affect. In the case of DCMU, interruption of the flow of electrons from water to NADP must be accompanied by cessation of noncyclic photophosphorylation[19], with a consequent light-dark type of transient. Apparently, the interruption of this flow of electrons from water to NADP *in vivo* does not result in a greatly accelerated cyclic photophosphorylation. Such cyclic photophosphorylation, seen in the experiments with Vitamin K, results in a greatly stimulated synthesis of oligosaccharides, polysaccharides, and this synthesis is not seen in the experiments with DCMU.

The effects on the concentration of photosynthetic intermediates produced by hexylresorcinol resemble in some respects the light-dark type transients induced by CCCP and the DCMU, but are markedly different in other respects. The transient rise and fall in PGA characteristic of the light-dark transient is again seen. Ribulose diphosphate level falls rapidly, as in the case of the light-dark transient. A more pronounced difference is seen in the behavior of sedoheptulose and fructose diphosphates. The levels of these sugar diphosphates rise very markedly during several minutes following the addition of the inhibitor, while the levels of the corresponding monophosphates drop appreciably. This apparent inhibition of the conversion of fructose diphosphate and sedoheptulose diphosphate to the corresponding monophosphates of these sugars has now been seen with several seemingly unrelated inhibitors. For example, lipoic acid and other fatty acids[10,11] inhibited the conversion of fructose and sedoheptulose diphosphates to their monophosphates. In the case of Vitamin K, described in this report, the levels of fructose and sedoheptulose diphosphates dipped momentarily and then rose markedly at the same time that the levels of the monophosphates are decreasing. There is no obvious reason why such diverse chemicals should effect the removal of the phosphate group on carbon atom number one of these sugars. It may be that the enzyme system responsible for the removal of this phosphate group is in some way closely associated with a lipoprotein or lipid membrane surface. In this case, the introduction of such lipophyllic substances as fatty acid and hexylresorcinol might alter this enzymic activity. On the other hand, it can be seen

that the principal effect of Vitamin K *in vivo* seems to be a diversion of electrons from the photoelectron transport system and a stimulation of cyclic photophosphorylation. Perhaps one or both of these actions is related in some way to an inhibition of the removal of the phosphate group on carbon atom number one of ketose sugars.

Vitamin K has long been known to be an effective inducer of cyclic photophosphorylation in whole and broken chloroplasts[20]. The transient changes and complete inhibition of photosynthesis produced in this study by the addition of Vitamin K_5 suggest that it is an effective inducer of cyclic photophosphorylation *in vivo*. It appears to have diverted electrons from noncyclic flow since there is evidence of complete disruption of the operation of the carbon reduction cycle in spite of high levels of ATP.

The first indication of this diversion is the initial rise in level of PGA. We interpret this rise as a partial diversion of electrons away from the reactions responsible for reduction of PGA. However, this reduction of PGA to triose phosphate, with subsequent formation of fructose-1,6-diphosphate, does not stop until two minutes or more have elapsed after addition of inhibitor. This delay in reaching complete inhibition of the reductive reaction is evidenced by the rise in level of fructose-1,6-diphosphate for two minutes.

The fall in PGA subsequent to its rise in the case of Vitamin K_5 addition, as well as with CCCP, DCMU, hexylresorcinol, and the light-dark transients, probably is due mainly to the conversion of PGA to alanine and other amino acids by normal photosynthetic reactions[21].

The greatly accelerated formation of oligosaccharides and polysaccharides indicated by the appearance of these compounds from the

radiochromatograms shown in Figure 7 indicates a high level of formation of ATP or other high energy phosphate compounds. It is probable that this synthesis of carbohydrates accounts for most of the very rapid drop in glucose-6-phosphate produced by the addition of inhibitor.

The oxidation of glucose-6-phosphate to 6-phosphogluconic acid and to ribose-5-phosphate in the light provides strong evidence for an abnormal conversion of $NADPH_2$ to its oxidized form. This oxidation occurs more slowly than the initial transients. The large rise in the level of ribose-5-phosphate at a time when photosynthesis is completely inhibited and when there is presumably a high level of ATP requires that this formation of ribose-5-phosphate be at a site separated from the carbon reduction cycle. If the ribose-5-phosphate were formed at the site of the carbon reduction cycle, there would seem to be no reason why it would not be converted to ribulose-5-phosphate, phosphorylated to give ribulose-1,5-diphosphate and then carboxylated to give PGA. Thus, its accumulation would not occur.

The location of these different sites, and the mechanism for transfer of material and energy from one to another, are interesting subjects for speculation. For our present purposes it is sufficient to state that these kinetic effects suggest the separation of the enzymic system responsible for the operation of the primary carbon reduction cycle of photosynthesis from other biosynthetic pathways. Whether this separation is between chloroplast and cytoplasm, between interlamellar stroma and extralamellar stroma within the chloroplast, or between some other as yet unknown compartments remains to be seen.

The *in vivo* inhibition by Vitamin K_5 is seen to be the result of upsetting the balance of cofactors supplied to the carbon reduction cycle by the photoelectron transport mechanism due to the stimulation of excessive cyclic photophosphorylation, at the expense of noncyclic electron transport.

If Vitamin K_5 functions at lower concentrations as a natural cofactor for cyclic photophosphorylation, its level could very well be a factor in the regulatory mechanism in the chloroplast for the control of the ATP/$NADPH_2$ ratio.

ACKNOWLEDGEMENTS

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FIGURES

Fig. 1. Transient Changes in Concentrations of Intermediates of the Photosynthetic Carbon Reduction Cycle Caused by Addition of DCMU.

Fig. 2. Transient Changes in Concentrations of Intermediates of the Photosynthetic Carbon Reduction Cycle Caused by Addition of DCMU.

Fig. 3. Transient Changes in Concentrations of Intermediates of the Photosynthetic Carbon Reduction Cycle Caused by Addition of CCCP.

Fig. 4. Transient Changes in Concentrations of Intermediates of the Photosynthetic Carbon Reduction Cycle Caused by Addition of CCCP.

Figs. 1-4. Levels of ^{14}C-labeled compounds in inhibition experiments with DCMU and CCCP: o, PGA; ●, glucose-6-phosphate; ■, fructose-6-phosphate; ▲, fructose-1,6-diphosphate; Δ, ribulose-1,5-diphosphate; □, sedoheptulose-7-phosphate.

(It is suggested that Figs. 1-4 be placed on one page.)

Fig. 5. Transient Changes in Concentrations of Intermediates of the Photosynthetic Carbon Reduction Cycle Caused by Addition of Hexylresorcinol.

Fig. 6. Transient Changes in Concentrations of Intermediates of the Photosynthetic Carbon Reduction Cycle Caused by Addition of Hexylresorcinol.

Figs. 5-6. Levels of ^{14}C-labeled compounds in inhibition experiments with hexylresorcinol: o, PGA; ●, glucose-6-phosphate; ■, fructose-6-phosphate; ▲, fructose-1,6-diphosphate; Δ, ribulose-1,5-diphosphate; □, sedoheptulose-7-phosphate; ◇, 6-phosphogluconic acid; ◆, ribose-5-phosphate; x, sedoheptulose-1,7-diphosphate.

FIGURES (Continued)

Fig. 7. Radiochromatogram of Photosynthetic Intermediate Compounds from Chlorella Following Inhibition by Vitamin K_5.

Radioautograph of a two-dimensional paper chromatogram made from Chlorella pyrenoidosa which had photosynthesized for 15 minutes in $^{14}CO_2$ prior to addition of inhibitor and had remained in $^{14}CO_2$ for 11 minutes after the addition of Vitamin K_5.

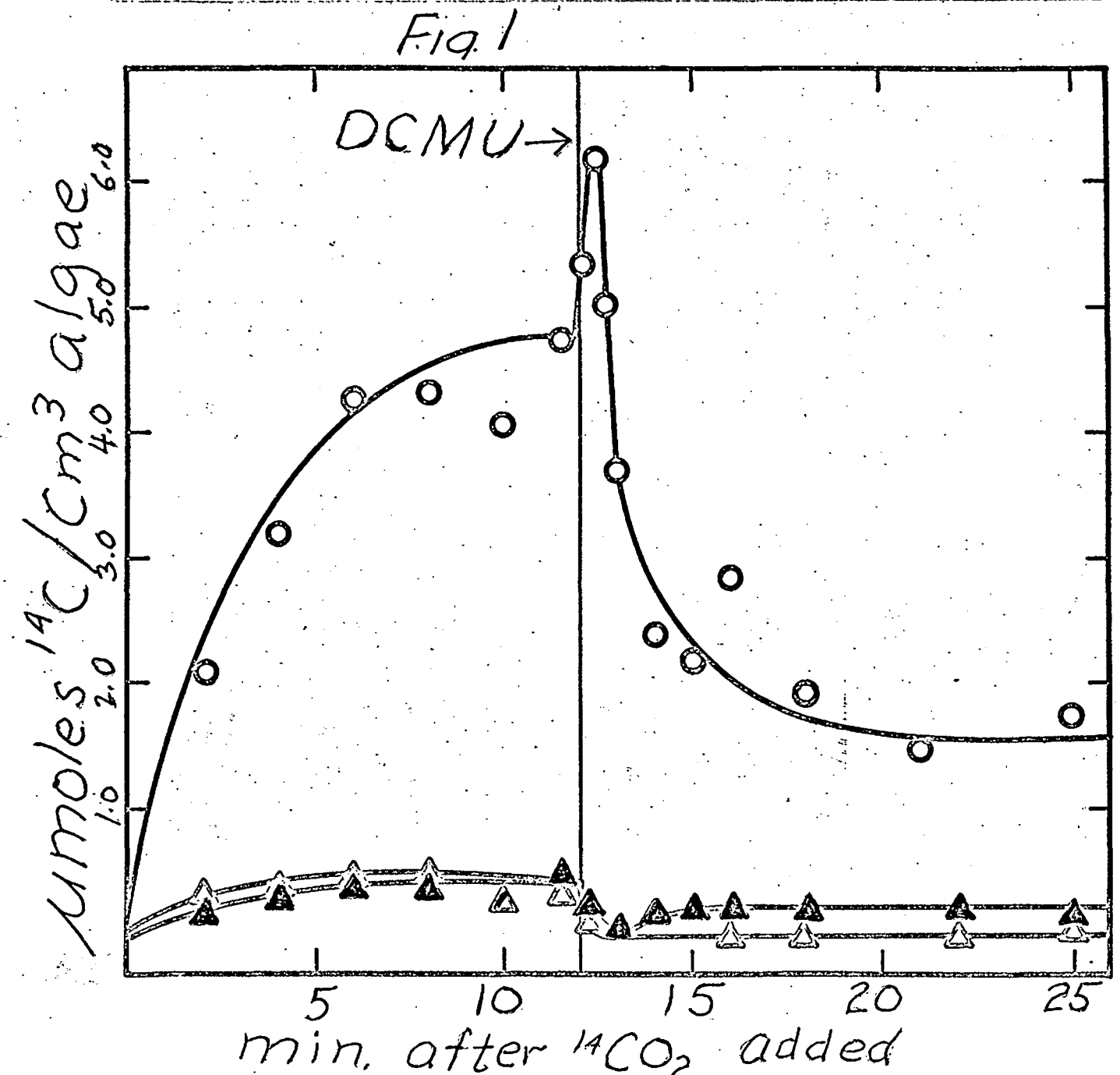
Fig. 8. Transient Changes in Concentrations of Intermediates of the Photosynthetic Carbon Reduction Cycle Caused by Addition of Vitamin K_5.

Fig. 9. Transient Changes in Concentrations of Intermediates of the Photosynthetic Carbon Reduction Cycle Caused by Addition of Vitamin K_5.

Fig. 10. Transient Changes in Concentrations of Intermediates of the Photosynthetic Carbon Reduction Cycle Caused by Addition of Vitamin K_5.

Figs. 8-10. Levels of ^{14}C-labeled compounds in inhibition experiments with Vitamin K_5: o, PGA; ●, glucose-6-phosphate; □, sedoheptulose-7-phosphate; ■, fructose-6-phosphate; △, ribulose-1,5-diphosphate; ▲, fructose-1,6-diphosphate; ◇, 6-phosphogluconic acid; ◆, ribose-5-phosphate; x, sedoheptulose-1,7-diphosphate.

Fig. 1. E. S. Gould & J. A. Bassham. Inhibitor Studies on the Photosynthetic Carbon Reduction Cycle in Chlorella pyrenoidosa.



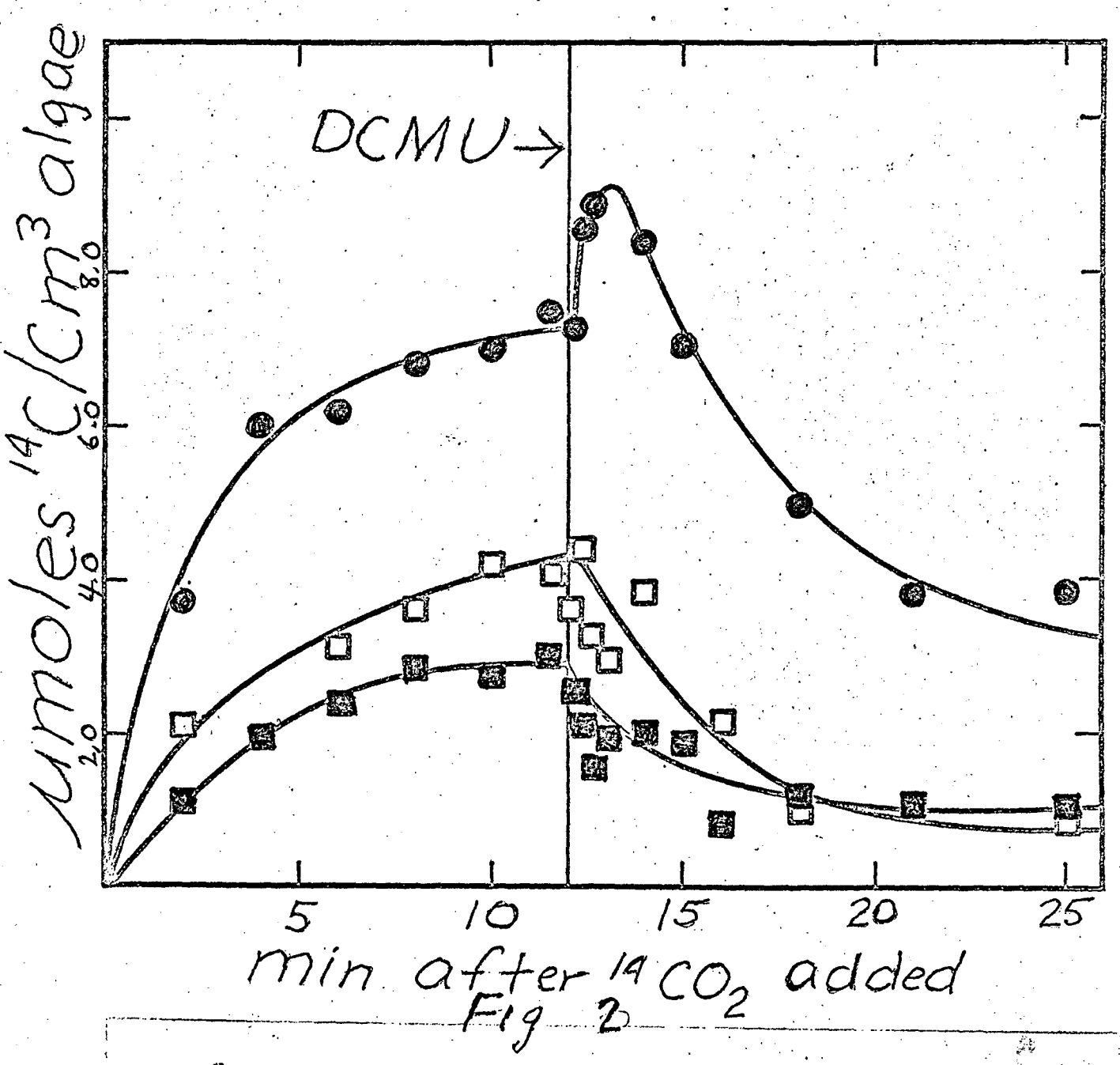
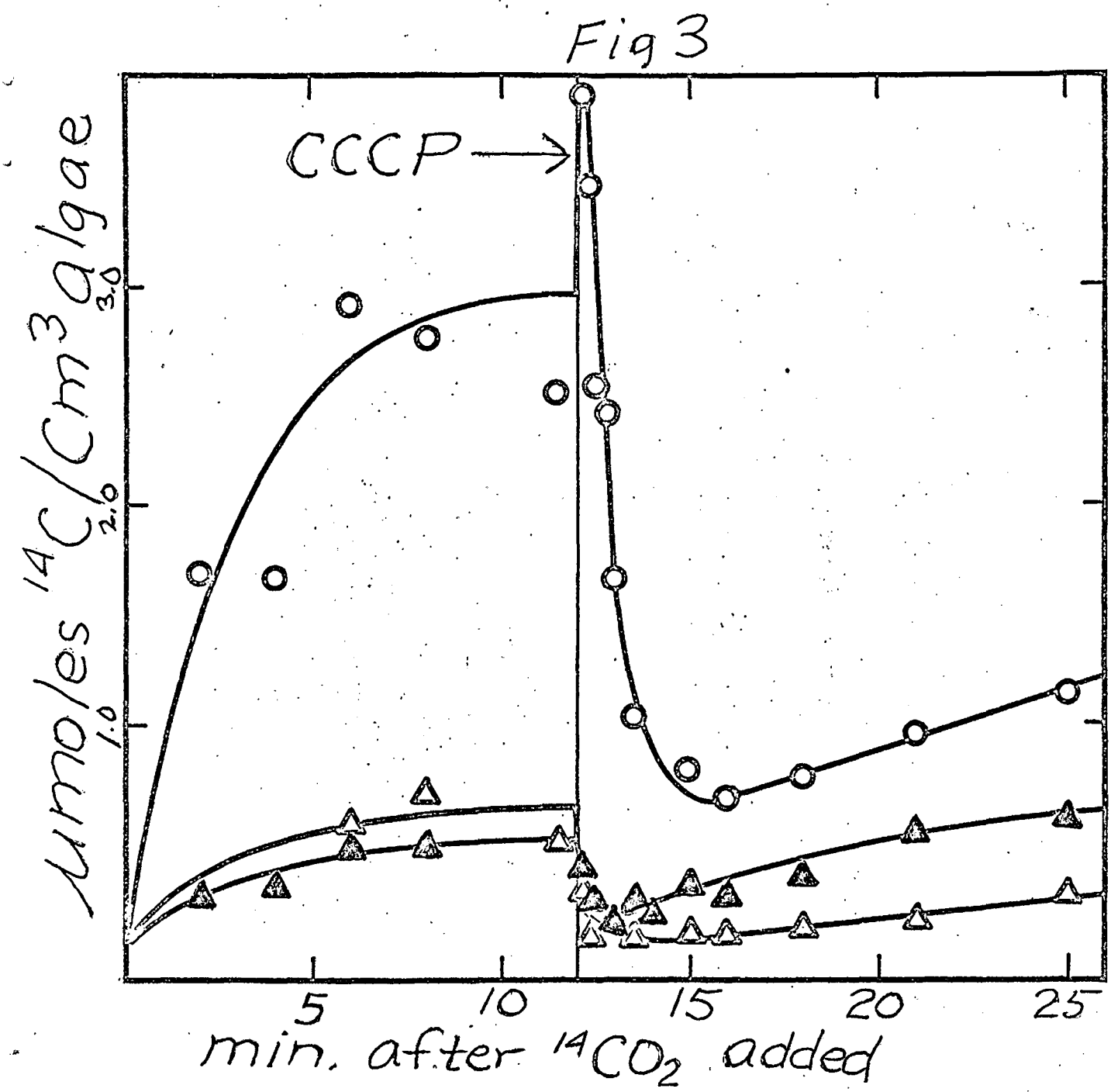


Fig. 2. E. S. Gould & J. A. Bassham. Inhibitor Studies on the Photosynthetic Carbon Reduction Cycle in Chlorella pyrenoidosa.

Fig. 3. E. S. Gould & J. A. Bassham. Inhibitor Studies on the Photosynthetic Carbon Reduction Cycle in Chlorella pyrenoidosa.



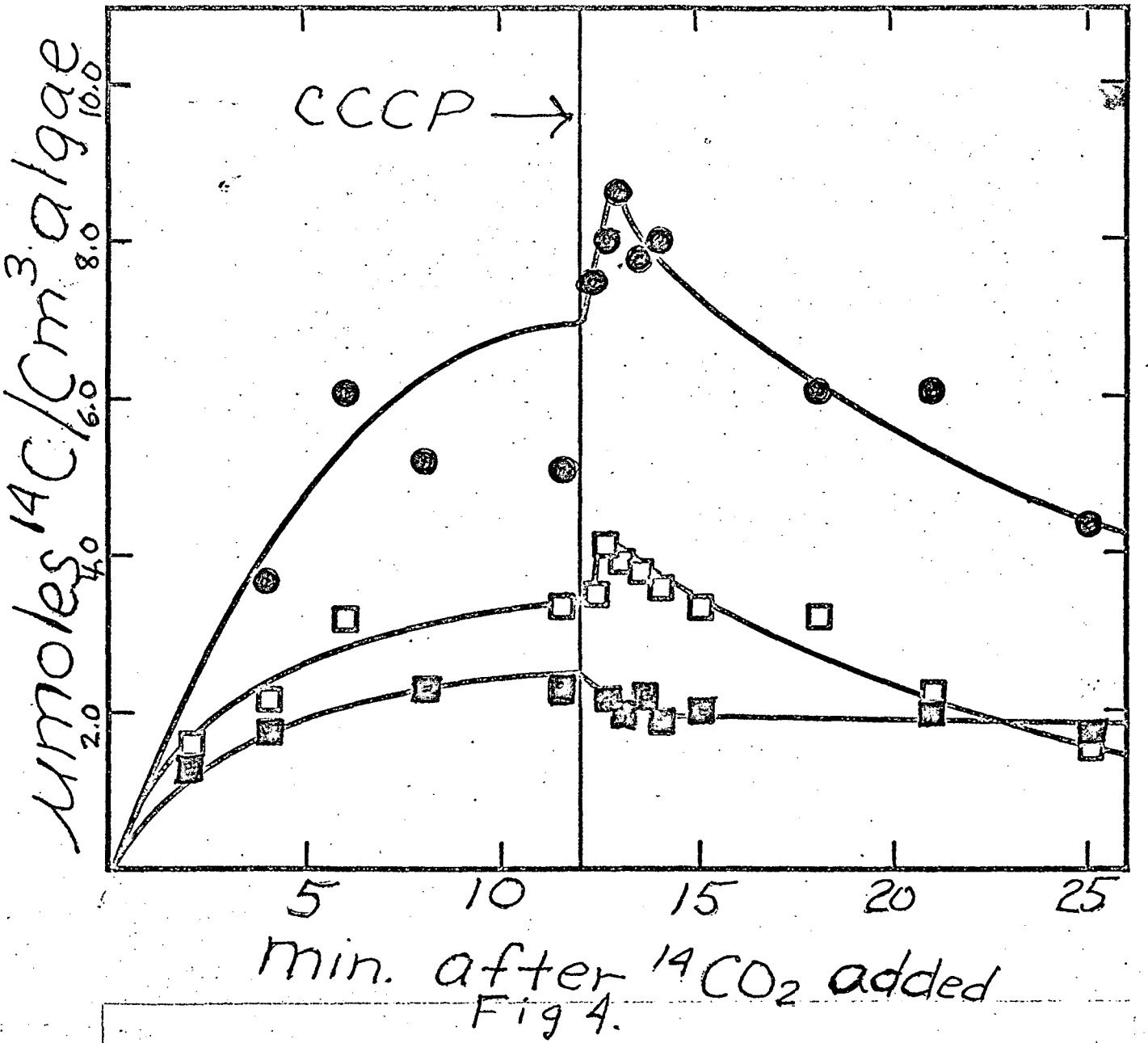


Fig. 4. E. S. Gould & J. A. Bassham. Inhibitor Studies on the Photosynthetic Carbon Reduction Cycle in Chlorella pyrenoidosa.

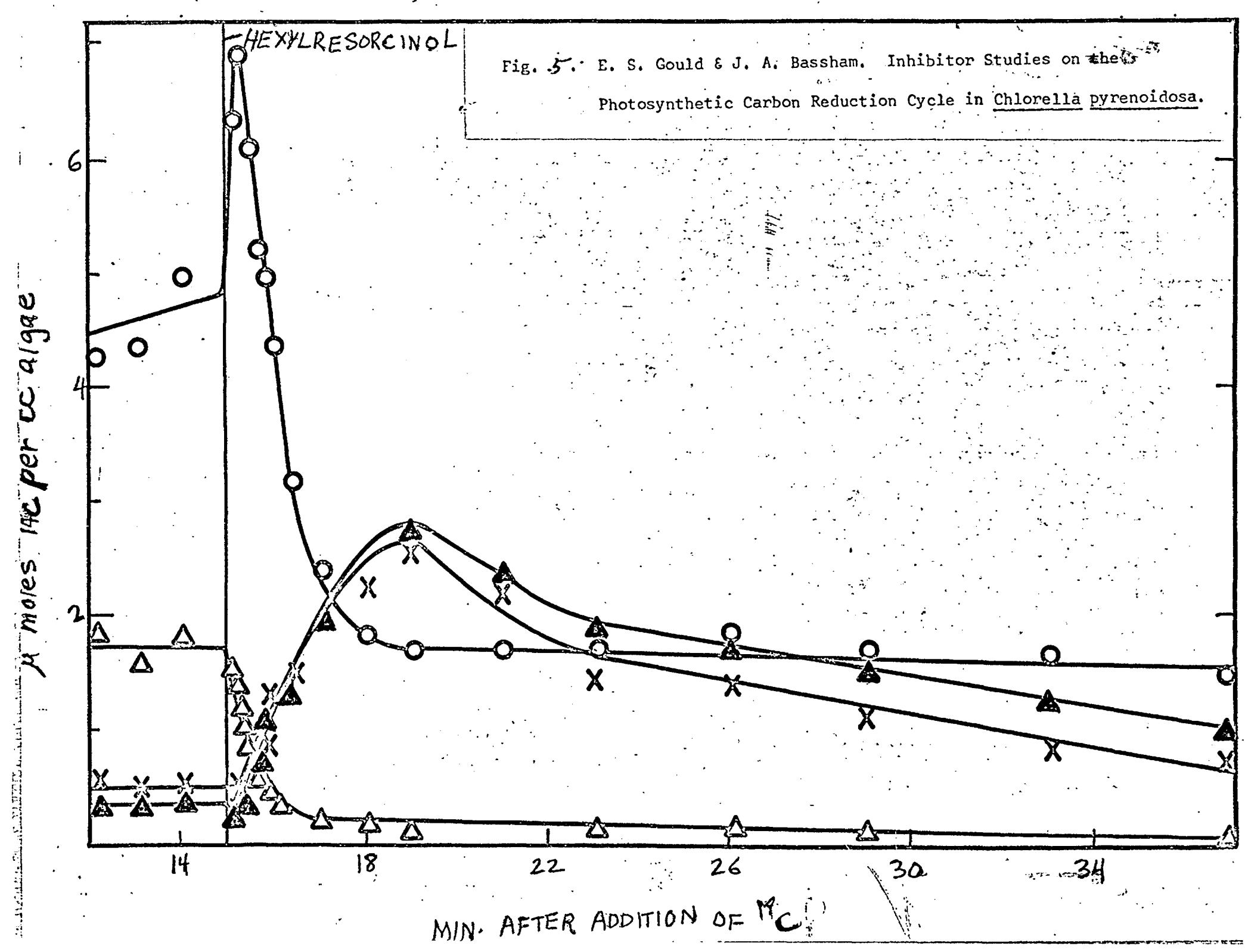


Fig. 5. E. S. Gould & J. A. Bassham. Inhibitor Studies on the Photosynthetic Carbon Reduction Cycle in Chlorella pyrenoidosa.

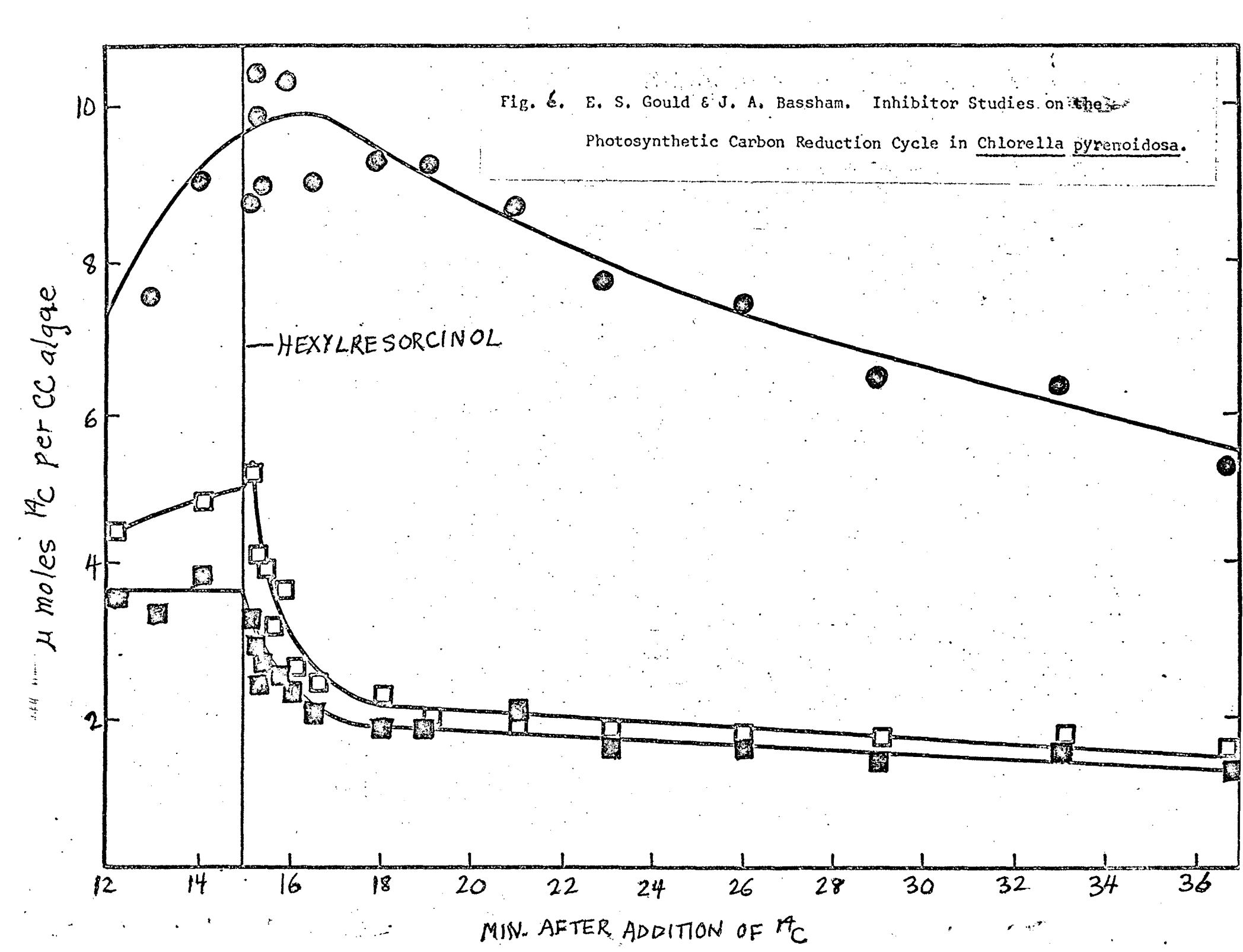


Fig. 6. E. S. Gould & J. A. Bassham. Inhibitor Studies on the Photosynthetic Carbon Reduction Cycle in Chlorella pyrenoidosa.
HEXYLRESORCINOL
μ moles ^{14}C per cc algae
MIN. AFTER ADDITION OF ^{14}C
10
8
6
4
2
12
14
16
18
20
22
24
26
28
30
32
34
36

Biochem 872

Fig. 7

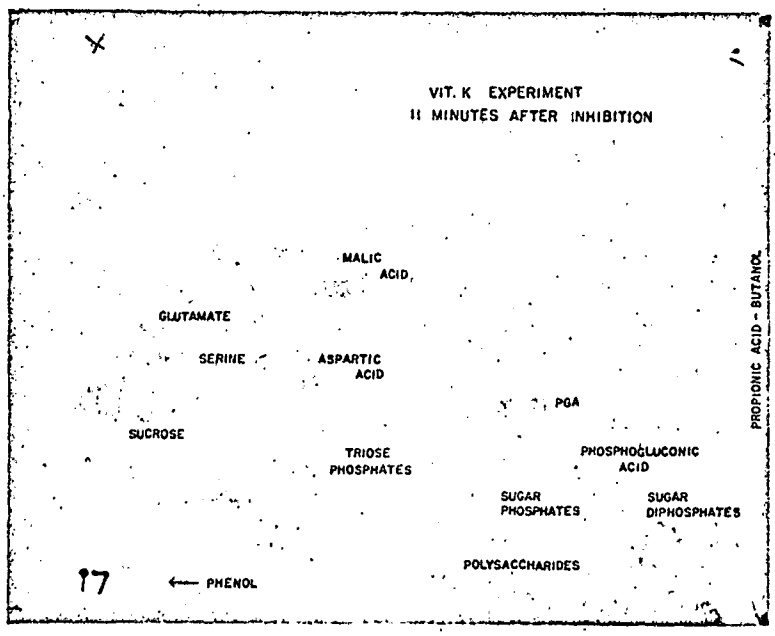


Fig. 7. E. S. Gould & J. A. Bassham. Inhibitor Studies on the Photosynthetic Carbon Reduction Cycle in Chlorella pyrenoidosa.

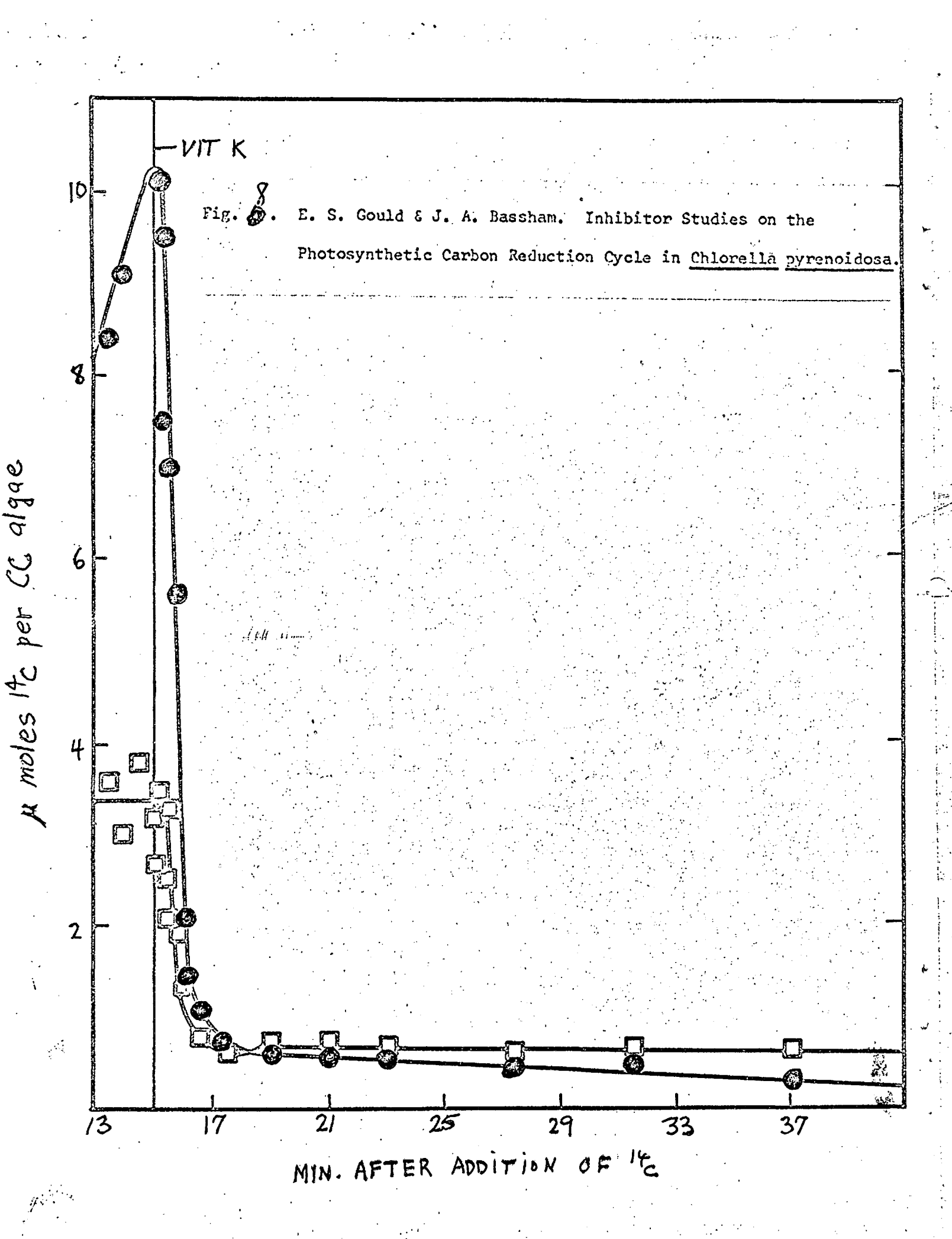
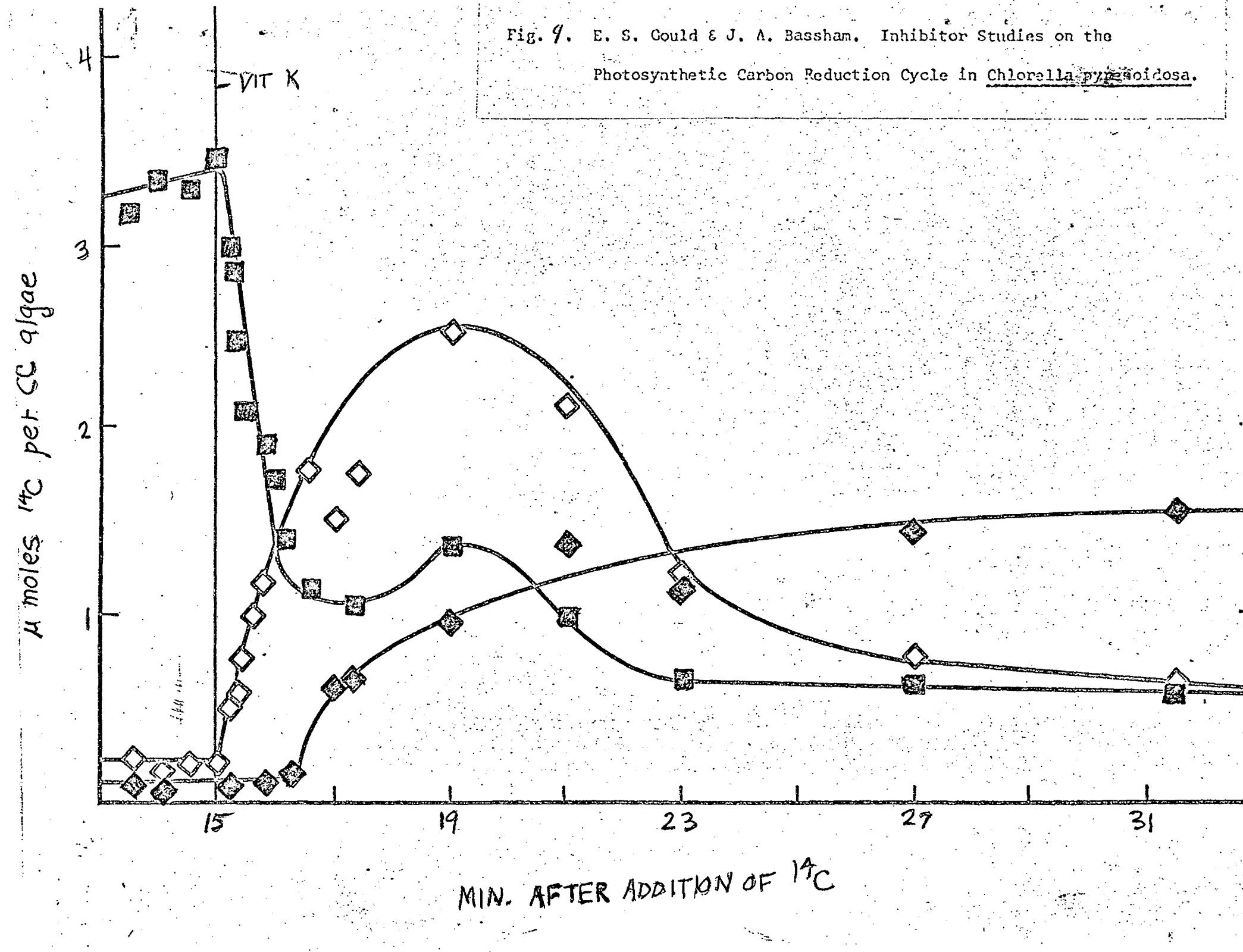


Fig. 8. E. S. Gould & J. A. Bassham. Inhibitor Studies on the Photosynthetic Carbon Reduction Cycle in Chlorella pyrenoidosa.

Fig. 9. E. S. Could & J. A. Bassham. Inhibitor Studies on the Photosynthetic Carbon Reduction Cycle in Chlorella pyrenoidosa.



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