Influence of Adenine Nucleotides on the Inhibition of Photophosphorylation in Spinach Chloroplasts by N-Ethylmaleimide*

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SUMMARY

The incubation of spinach chloroplasts with 1 mM N-ethylmaleimide in light for 60 to 90 s results in a partial, irreversible inhibition of photophosphorylation. The inhibition was not overcome at infinite light intensity or at infinite concentrations of the phosphorylation substrates. Although the inhibition diminished with decreasing concentrations of adenosine diphosphate in the assay of phosphorylation, the inhibition of guanosine diphosphate phosphorylation was independent of the concentration of this nucleotide.

Although adenosine di- or triphosphate (10 to 30 μM) alone partially prevented the development of the N-ethylmaleimide inhibition of phosphorylation, these nucleotides were more effective when either 1 mM inorganic phosphate or arsenate was also present. The light-dependent incorporation of N-ethylmaleimide into chloroplast-bound coupling factor 1 was affected by adenosine triphosphate and inorganic phosphate in a manner similar to the onset of N-ethylmaleimide inhibition.

Since guanosine diphosphate did not protect phosphorylation from N-ethylmaleimide inhibition but is phosphorylated at rapid rates, it is apparent that coupling factor 1 in chloroplasts has multiple nucleotide recognition sites.

Low concentrations of ADP or ATP enhance the extent of light-dependent H⁺ uptake (4, 5) resulting in an increased pH differential across the thylakoid membranes (6), an inhibition of electron flow (7), and an increase in delayed light emission (8). The binding of adenine nucleotides to membrane-bound CF₁ may modify the structure of CF₁ in such a way that the permeability of the membranes to H⁺ is reduced (4). This possibility is strengthened by the finding (1) that ATP also protects phosphorylation from inhibition by NEM.

The adenine nucleotide protection of phosphorylation from inhibition by NEM provides an indirect approach to the study of some of the interactions of membrane-bound CF₁ with nucleotides. We report that P₁ or HAsO₄⁻ enhances the effectiveness of ATP or ADP in preventing the inhibition of phosphorylation by NEM. In addition, evidence is presented which suggests that CF₁ has at least two sites which can recognize nucleotides.

MATERIALS AND METHODS

Class II chloroplasts were prepared from market spinach (9). Chloroplasts (0.1 mg of chlorophyll per ml) were treated with 1 mM NEM at room temperature in a 0.5- or 1.0-ml reaction mixture which contained, unless otherwise indicated: 50 mM Tricine-NaOH (pH 8.0), 50 mM NaCl, 5 mM MgCl₂, and 0.05 mM pyocyanine. Controls without NEM were also run. After 90 s in white light (2.0 to 2.5 x 10⁶ erg/cm² s) dithiothreitol was added in a small volume to a concentration of 1 mM to incubations containing NEM and to 0.5 mM to the controls. Aliquots of these mixtures were assayed for phosphorylation with pyocyanine at a light intensity of about 2 x 10⁶ ergs/cm² s. Unless otherwise indicated, the reaction mixtures contained in 1.0 ml: 50 mM Tricine-NaOH (pH 8.0), 50 mM NaCl, 5 mM MgCl₂, 0.05 mM pyocyanine, 3 mM ADP, and 2 mM potassium phosphate buffer (pH 8.0) containing between 10⁶ and 10⁶ cpm of 3²P. Chlorophyll concentrations were determined spectrophotometrically (10).

Light-dependent H⁺ uptake was assayed as described previously (4) except that a Heath model EU-200 electrometer was used. Incorporation of [²H]NEM into the 7 subunit of chloroplast-bound CF₁ was determined as described previously (2), except that the chloroplasts were treated with EDTA to remove the CF₁ and that the CF₁ in the EDTA extracts was partially purified by chromatography on columns (1 x 7 cm) of DEAE-Sephadex A-50 (11) prior to electrophoresis in the presence of sodium dodecyl sulfate (12). Protein was determined by a colorimetric method (13).

Adenine nucleotides and NEM were purchased from the Sigma Chemical Co. The purity of the nucleotides was ascertained by thin layer chromatography on PEI (polyethyleneimine)-cellulose using LiCl as the solvent (14). Impurities detected in one lot of AMP were removed by repeated recrystallization from dimethylformamide (15).

For N-ethylmaleimide to inhibit partially photophosphorylation, chloroplasts must be illuminated with this reagent prior to the assay of phosphorylation (1). Light also markedly enhances the incorporation (2) of a small amount of [²H]NEM into chloroplasts. In view of the similarities in the inhibition of phosphorylation by NEM and its incorporation into CF₁, it is probable that the reaction of CF₁ with NEM is the cause of the inhibition of phosphorylation.

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The abbreviations used are: NEM, N-ethylmaleimide; Tricine, N-Tris(hydroxymethyl)methylglycine; CF₁, coupling factor 1.
were removed by chromatography on Dowex 1-Cl. Ethyl-2-[3H]-
NEM was purchased from New England Nuclear. 4'-Deoxyphlor-
izin was a gift of Dr. S. Izawa of Michigan State University.

RESULTS

Requirements for Maximal Inhibition of Phosphorylation by
NEM—Even after prolonged periods of illumination and at
high concentrations of NEM, the inhibition of phosphorylation
is not complete (1). Before the effects of adenine nucleotides on
the extent of phosphorylation inhibition by NEM were investi-
gated, it was necessary to establish conditions under which
NEM inhibits phosphorylation to its maximal extent. High light
intensities are required to elicit maximal inhibition when pyo-
cyanine is used as the mediator of electron flow. Half-maximal
inhibition was achieved at about $2 \times 10^5$ ergs/cm$^2$-s under
the standard incubation conditions with NEM, and saturation
was approached at $2 \times 10^6$ ergs/cm$^2$-s. Pyocyanine-dependent cyclic
phosphorylation, assayed under similar conditions, but in the
absence of NEM, responds to light intensity in a manner quite
parallel to that of the development of the NEM inhibition.

Maximal inhibition was reached when the chloroplasts were
illuminated with NEM over the pH range of 8 to 9 and the
inhibition fell dramatically with decreasing pH. For example,
the inhibition at pH 8.5 was 75%, but was only 9% at pH 7.0.
Pyocyanine-dependent cyclic phosphorylation showed a similar
dependence on pH.

Mg$^{2+}$ was also required to elicit maximal inhibition by NEM
in the light. In the absence of Mg$^{2+}$ and the presence of 1 mM
EDTA, the inhibition of phosphorylation by 1 mM NEM within
90 s of illumination ranged from 40 to 50%, whereas at a satu-
rating Mg$^{2+}$ concentration (5 mM) the inhibition varied from
60 to 70%. Other divalent cations including Ca$^{2+}$, Co$^{2+}$, and
Mn$^{2+}$ did not replace Mg$^{2+}$ and tended to decrease the extent of
inhibition by NEM. Under the standard conditions, the inhibi-
tion of phosphorylation elicited by 1 mM NEM in 90 s of illumi-
nation can be routinely observed to be greater than 50%.

Characteristics of Phosphorylation in NEM-inhibited Chloro-
plasts—The effects of light intensity on pyocyanine-dependent
phosphorylation in NEM-inhibited and control chloroplasts are
shown in Fig. 1. At low light intensities, the inhibition tended
to be more severe than at high intensities. However, double
reciprocal plots of these data revealed that the inhibition of
phosphorylation extrapolated to 50% at infinite light intensity.

The extent of inhibition of phosphorylation by the NEM and
light treatment was not sensitive to the concentration of P$_i$
present during the assay of phosphorylation (Fig. 2). Although
the maximal rate of phosphorylation in the inhibited chloroplasts
was decreased by 58%, the apparent $K_m$ for P$_i$ was little affected.
In contrast, both $V_{max}$ and apparent $K_m$ of phosphorylation for
ADP were changed by the prior exposure of chloroplasts to NEM
in the light (Fig. 3A). $V_{max}$ is about 64% inhibited whereas the
apparent $K_m$ decreased from 55 μM to 25 μM.

Surprisingly, GDP phosphorylation was affected by the NEM
and light treatment in a manner quite different from that of
ADP phosphorylation (Fig. 3B), even though the $V_{max}$ for the
phosphorylation of the two nucleotides was similar. For the
phosphorylation of GDP, $V_{max}$ was inhibited, but $K_m$ was little
affected by the NEM and light treatment. This difference be-
tween GDP and ADP was not limited to NEM-inhibited chloro-
plasts. The energy transfer inhibitor, 4'-deoxyphlorizin (15),
affected the dependence of phosphorylation on GDP concentra-
tion in a different manner from that of ADP (Fig. 4). In contrast,
limiting the rate of phosphorylation by lowering the light inten-
sity resulted in a decrease in $V_{max}$ as well as apparent $K_m$ for both
GDP and ADP.

Effects of Nucleotides on Onset of NEM Inhibition—Although
ADP alone partially prevented the inhibition of phosphorylation
when it was present with NEM in the light, it was much more
effective in the presence of 1 mM HASO$_4^-$(Fig. 5A). Arsenate
alone had little effect on the extent of inhibition and could be
replaced with 1 mM P$_i$. Arsenate also enhanced the protection
of phosphorylation from inhibition by NEM afforded by ATP (Fig.
5B). The concentrations of ATP required for protection were very
similar to those of ADP. The effect of P$_i$, concentration on the
extent of inhibition of phosphorylation by NEM in the presence
of 10 μM ATP was examined. In the absence of P$_i$, phosphoryla-
tion was inhibited 58%, whereas in the presence of 2 mM P$_i$, the

![Fig. 1](http://www.jbc.org/)

**Fig. 1.** Effect of light intensity on pyocyanine-dependent phosphor-
ylation in NEM-inhibited chloroplasts. Chloroplasts were
incubated 120 s in the light ($2 \times 10^5$ ergs/cm$^2$-s), or dark with 1
mm NEM. After addition of an amount of dithiothreitol slightly
in excess of the NEM, aliquots of the incubation mixtures which
contained 50 μg of chlorophyll were assayed for pyocyanine-de-
pendent phosphorylation. Light intensity was varied through the
use of calibrated wire screens and was estimated through the use
of a YSI model 65 radiometer. The illumination time in the assay
of phosphorylation was 1 min. Phosphorylation rates are expressed
as micromoles of P$_i$ esterified per hour per mg of chlorophyll.

![Fig. 2](http://www.jbc.org/)

**Fig. 2.** Effect of P$_i$ concentration on phosphorylation in NEM-
inhibited chloroplasts. Chloroplasts were treated with or without
1 mm NEM for 90 s in the light and aliquots containing 10 μg of
chlorophyll were then assayed for phosphorylation with pyocy-
anine at the concentrations of P$_i$, shown. The illumination time
was 15 s and phosphorylation rates are expressed as micromoles of P$_i$
esterified per hour per mg of chlorophyll.
inhibition was only 29%. The half-maximal effect was elicited at 0.4 mM Pi, a value quite close to the apparent $K_m$ of phosphorylation for Pi.

Arsenate enhanced the protection afforded by ADP (10 or 30 μM) even in the presence of 0.125 mM 4'-deoxyphlorizin. This result might be taken to indicate that rapid turnover of CF₁ is not involved in the protection of phosphorylation from NEM inhibition by ADP and HAsO₄²⁻. However, the inhibition of phosphorylation by 4'-deoxyphlorizin decreases with decreasing ADP concentration. Although phosphorylation in the presence of 400 μM ADP was 60% inhibited by 0.1 mM 4'-deoxyphlorizin, the inhibition was 99% at 90 μM ADP.

The rate of development as well as the extent of inhibition of phosphorylation by NEM was affected by ADP or ATP. With low concentrations of ATP (10 μM or less) the extent of inhibition was not markedly affected after prolonged incubation, but the inhibition developed more slowly (Fig. 6). With higher concentrations of ATP, or even 10 μM ATP in the presence of Pi, the rate of development of the inhibition was markedly slowed and the extent of the inhibition was quite significantly decreased even after 5 min of illumination.

**Influence of Mg²⁺ on Protective Effect of ADP and ATP**—Although ADP and ATP exert similar effects on the inhibition of

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**Fig. 3.** Effect of ADP or GDP concentration on phosphorylation in NEM-inhibited chloroplasts. Chloroplasts were incubated for 90 s in the light in the presence or absence of 1 mM NEM. A, for ADP phosphorylation, aliquots of these mixtures equivalent to 10 μg of chlorophyll and an illumination time of 10 s were used in the subsequent assay of pyocyanine-dependent phosphorylation. B, for GDP phosphorylation, NEM-treated and control chloroplasts equivalent to 25 μg of chlorophyll were used in the phosphorylation assay and the illumination time was 15 s. Phosphorylation rate is expressed as micromoles of Pi esterified per hour per mg of chlorophyll.

**Fig. 4.** Effect of 4'-deoxyphlorizin on the nucleotide concentration dependence of phosphorylation with ADP and GDP. Chloroplasts (10 μg of chlorophyll) were assayed for pyocyanine-dependent phosphorylation in the presence and absence of 0.1 mM 4'-deoxyphlorizin and at varying concentrations of ADP (A) or of GDP (B).

**Fig. 5.** Protection of phosphorylation from NEM inhibition by ADP or ATP. Chloroplasts were incubated for 90 s in the light in the presence or absence of 1 mM NEM and with the concentration of ADP or ATP shown. Arsenate was added to 1 mM concentration where indicated. Aliquots (0.1 ml) of these mixtures were assayed for pyocyanine-dependent phosphorylation under the standard conditions except that 5 mM Pi was used. A, effects of ADP in the presence or absence of HAsO₄²⁻. The rate of phosphorylation in the controls (minus NEM) was 600 μmol of Pi esterified per hour per mg of chlorophyll in chloroplasts illuminated without 1 mM HAsO₄²⁻ and 580 μmol of Pi esterified per hour per mg of chlorophyll in chloroplasts illuminated with HAsO₄²⁻. ADP has essentially no effect on phosphorylation rates in the controls. B, effects of ATP on the development of the NEM inhibition of phosphorylation. The control rates (minus NEM) were 650 μmol of Pi esterified per hour per mg of chlorophyll in chloroplasts illuminated in the absence of HAsO₄²⁻ and 570 μmol of Pi esterified per hour per mg of chlorophyll in chloroplasts illuminated with HAsO₄²⁻.
phosphorylation by NEM, the effects of these two nucleotides were quite different in the absence of Mg\(^{2+}\). As shown in Fig. 7A, ADP protected phosphorylation from inhibition by NEM in chloroplasts supplemented with 1 mM EDTA and in the absence of added Mg\(^{2+}\). However, under these same conditions, ATP actually enhanced the inhibition at the same concentrations which gave quite significant protection in the presence of Mg\(^{2+}\) (Fig. 7D). HAsO\(_4^{2-}\) or P\(_1\) did not enhance the protective effects of either ADP or ATP in the absence of Mg\(^{2+}\).

**DISCUSSION**

From our results it may be concluded that chloroplast-bound CF\(_1\) contains multiple sites which can recognize nucleotides.

The Influence of Mg\(^{2+}\), ATP, and ATP plus P\(_1\) on \([\text{H}]\)NEM Incorporation into CF\(_1\)—The close correlation (2) between the inhibition of phosphorylation by NEM and its incorporation into the gamma subunit of chloroplast-bound CF\(_1\) suggests that the inhibition is a consequence of the reaction of CF\(_1\) with NEM. To test this conclusion further and to assure that the effects of low concentrations of ATP on the inhibition of phosphorylation were also correlated with the reaction of CF\(_1\) with NEM, the influence of ATP on \([\text{H}]\)NEM incorporation was determined. As may be seen in Table I, \([\text{H}]\)NEM incorporation into CF\(_1\) was affected by Mg\(^{2+}\), 20 \(\mu\)M ATP, and 20 \(\mu\)M ATP in the presence of 1 mM P\(_1\); in a manner quite similar to the NEM inhibition of phosphorylation.

Lack of Effect of other Nucleotides on Onset of NEM Inhibition of Phosphorylation—Although nucleoside triphosphates other than ATP were reported to have no effect on the development of NEM inhibition (1), it was of interest to re-examine the effects of nucleotides in light of the enhancement of the effectiveness of ATP or of ADP by P\(_1\). A number of naturally occurring nucleotides including dATP, AMP, cyclic adenosine 3':5'-monophosphate; xanthosine diphosphate, IDP, and GDP were tested at 0.1 mM for their effects on the NEM inhibition in the presence and absence of 1 mM P\(_1\). With the exception of dATP, which gave results similar to ATP, these nucleotides gave little or no protection by themselves or in the presence of P\(_1\). Even in the presence of 0.4 mM GDP and 1 mM P\(_1\), the inhibition of phosphorylation by NEM was only diminished from 70% in the control to 30%. This slight effect could be a result of a minor contamination of the GDP with either ADP or ATP. As little as 2 \(\mu\)M ADP could have elicited the protective effect observed with the GDP. GDP (0.1 mM) did not diminish the ability of either ADP or ATP to protect.
Chloroplasts (20 mg of chlorophyll) were incubated for 5 min in the dark in a mixture (20 ml) which contained: 2 mM NEM, 0.4 M sucrose, 0.02 M Tricine-NaOH (pH 8.0), and 0.01 M NaCl. A slight excess of dithiothreitol was added and the mixture was diluted to 40 ml with the buffered sucrose solution. After centrifugation at 5000 × g for 10 min the chloroplast pellet was washed once with 40 ml of the buffered sucrose solution. Aliquots of the resuspended chloroplasts (2 mg of chlorophyll) were incubated in the light (2 × 10⁶ ergs/cm²-s), or dark at 20° for 90 s in a 20-ml reaction mixture which contained: 50 mM Tricine-NaOH (pH 8.0), 50 mM NaCl, 0.05 mM pyocyanine, 1 mM EDTA, 1 mM NEM which contained 61 μCi of [³H]NEM, and other additions as indicated. Dithiothreitol was then added to a concentration of 1 mM and the chloroplasts were centrifuged at 5000 × g for 10 min. CF, was extracted from the chloroplasts and was partially purified on DEAE-Sephadex A-50 (11). After dissociation of the CF, with 1% sodium dodecyl sulfate in the presence of 0.2% β-mercaptoethanol, sodium dodecyl sulfate gel electrophoresis (12) was run. After staining, bands corresponding to the γ subunit were cut out and radioactivity in the bands determined. The incorporation of [³H]NEM into the γ subunit in the sample illuminated with Mg²⁺ corresponds to about 0.63 nmol of NEM per nmol of CF,. MgCl₂ was present at 5 mM; ATP, at 20 μM; P₁ at 1 mM, where indicated.

Although GDP is phosphorylated by chloroplasts at high rates and with an apparent $K_m$ only slightly greater than that of ADP, GDP has little to no effect on either light-dependent H⁺ uptake or on the light-dependent inhibition of phosphorylation by NEM. Clearly then, the site which recognizes nucleotides for the phosphorylation reaction differs from that involved in the protection of phosphorylation from NEM inhibition. Not only are higher concentrations of nucleotides required for phosphorylation than for protection, but also the phosphorylation site appears to be less specific. Multiple nucleotide binding sites in soluble CF, have been demonstrated (16, 17).

The conformation of CF, in illuminated chloroplasts probably differs from that in darkened ones (18, 19). One consequence of this altered conformation could be an exposure of a group on the γ subunit of CF, to reaction with NEM. ATP or ADP could prevent the reaction of this group with NEM in at least two ways. First, the binding of ATP or ADP could occur at a site close enough to the NEM-reactive group to partially mask it. Second, the binding of ATP or ADP to a subunit(s) of CF, may affect the conformation of the enzyme in such a way that the NEM-sensitive group is inaccessible even in the light.

In view of the marked potentiation by P₁ of the protection of phosphorylation from NEM inhibition afforded by low concentrations of ADP or ATP, P₁ may alter the affinity of CF, for adenine nucleotides. It is possible however, that P₁ does not interact with CF, unless ADP or ATP is also present. This possibility is strengthened by the finding of Ryrie and Jagendorf (20) that for SO₂⁻ to cause a permanent inhibition of phosphorylation in the light, ADP was required. Since the effects of SO₂⁻ were overcome by HAsO₂⁻ it is possible that the SO₂⁻ interacts with a P₁ site. Since ATP could not replace the requirement for ADP, in the development of SO₂⁻ inhibition, ATP binding may induce a different state of CF₁ than ADP binding. Furthermore, GDP which also fails to protect phosphorylation from NEM inhibition, did not support the formation of the SO₂⁻ inhibition of phosphorylation in the light.

The effects of P₁ on the protection of phosphorylation from NEM inhibition by ADP or ATP cannot be explained in terms of rapid turnover of a catalytic phosphorylation site(s). Rapid phosphorylation of GDP did not markedly affect the NEM inhibition. Furthermore, quite significant protection was observed with 5 μM ATP and 1 mM P₁, and the phosphorylation rate under these conditions would be exceedingly low. However, slow turnover of a site cannot be ruled out. The Mg²⁺ requirement for the P₁ enhancement of the ADP or ATP protection from inhibition of phosphorylation by NEM could reflect a Mg²⁺ requirement for a catalytic event or for binding either the nucleotide or P₁ to the CF₁. The possibility that ADP and P₁ alter the conformation of CF, is underscored by the results of Ryrie and Jagendorf (19). The light-dependent tritiation of CF, in chloroplasts suspended in H₂O was diminished about 50% by 1 mM concentrations of ADP and P₁. Furthermore ADP and P₁ partially prevented the light-dependent release of H⁺ from CF₁ in chloroplasts previously incubated in the light with H₂O. Since the decrease in tritiation of CF₁ by ADP plus P₁ was not sensitive to Dio-9 and phlorizin at high concentrations, it is not likely that phosphorylation itself decreases the extent of tritiation. Rather, ADP plus P₁ may prevent part of the conformational changes in CF₁ which occur upon illumination of chloroplasts. It will be of interest to test the effects of low concentrations of ATP plus P₁ as well as GDP plus P₁ on the light-induced tritiation of CF₁ in chloroplasts.

In view of the Mg²⁺ requirement for the expression of the protective effects of ATP, but not of ADP, ATP may have to be converted to ADP by a Mg²⁺-dependent ATPase. However, the rate of ATPase activity with 15 μM ATP and 5 mM MgCl₂ was exceedingly low, less than 1 μmol of Pi formed per hour per mg of chlorophyll. Only a very small amount of the ATP would therefore be hydrolyzed in the protection experiments. Thus, Mg²⁺ may be required for the binding of ATP to CF₁ rather than a conversion of ATP to ADP.

Although the inhibition of phosphorylation in chloroplasts previously treated with NEM was independent of GDP concentration, the inhibition diminished as the concentration of ADP was decreased. This intriguing difference between GDP and ADP phosphorylation may be related to the inability of GDP to interact with the site(s) on CF₁ involved in the stimulation of H⁺ uptake or protection of phosphorylation from NEM inhibition. It is not inconceivable that this site(s) may be capable of catalyzing the phosphorylation of ADP, but not of GDP. Thus, CF₁ could contain two sites involved in the phosphorylation of ADP with differing ADP concentration requirements. If it is assumed that the site with higher apparent ADP affinity is less sensitive to inhibition by NEM, the fact that the inhibition of phosphorylation in chloroplasts pretreated with NEM in the light decreases with decreasing ADP concentrations may be explained.

1 R. P. Magnusson, unpublished observations.
If the phosphorylation of GDP were to be catalyzed only by the site with lower affinity for nucleotides, the observed constant extent of inhibition of GDP phosphorylation by the NEM treatment would be expected.

The properties of the stimulation of H+ uptake by ADP or ATP (4, 5) appear to differ somewhat from those of the protection by ADP or ATP of phosphorylation from NEM inhibition. For example, whereas the protective effect of ADP was observed in the absence of Mg2+, ADP had no effect on H+ uptake under these conditions. Furthermore, HAsO42- did not appear to enhance the effectiveness of low concentrations of ATP in the stimulation of H+ uptake. Although the same nucleotide binding site(s) may still be involved in these two processes, differing conditions were required for the effects of binding to be expressed.

Finally, it will be of interest to explore the relationship between the effects of adenine nucleotides on CF1 as revealed by their protection from inhibition of phosphorylation by NEM and the binding of adenine nucleotides to CF1. Tightly bound nucleotides have been shown to be present in CF1 (16).

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REFERENCES
Influence of adenine nucleotides on the inhibition of photophosphorylation in spinach chloroplasts by N-ethylmaleimide.

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