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Azide inhibition of chloroplast ATPase is prevented by a high protonmotive force

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Azide has no effect on photophosphorylation at normal light intensities, inhibits acid-base ATP synthesis in the dark weakly, and is a moderately strong inhibitor of the ATP-P_i exchange reaction occurring in the dark after activation of spinach thylakoids by light. However, azide is a potent inhibitor of the methanol-activated ATPase of thylakoids (which is not associated with proton pumping). When ATPase has been activated by light plus dithiothreitol it pumps protons during ATP hydrolysis, so the thylakoid high-energy state is maintained in darkness. Azide has no effect on this reaction. When the light + dithiothreitol-activated ATPase is maximally stimulated by the uncoupler, NH_4Cl, azide becomes a strong inhibitor. We suggest that inhibition of ATPase by azide occurs only when the net thylakoid protonmotive force differential is less than maximal.

Introduction

Azide is known to be a powerful inhibitor of mitochondrial F_1 function (see, for instance, Ref. 1 and references therein). Very little attention has been paid to its effect on chloroplasts' equivalent CF_1. It has no effect on photophosphorylation, and is added routinely to reaction mixtures demonstrating electron flow through Photosystem I to methyl viologen, in order to inhibit catalase [2]. Recently, however, Eric Larson in this laboratory (results in preparation) found that it did indeed inhibit sulfite-stimulated ATPase, in a fashion apparently competitive with sulfite. In this work the observation has been extended to other reactions of spinach thylakoids, and the surprising finding was made that the extent of inhibition is affected strongly by the energization state of the membranes.

Materials and Methods

Thylakoids were prepared by grinding spinach from the grocery in 300 mM sucrose, 200 mM choline-HCl, 20 mM Hepes, 5 mM $MgCl_2$, 2 mg/ml bovine serum albumen and 5 mM ascorbate (adjusted to pH 7.8 at 0 °C). The chloroplasts (naked thylakoids) were sedimented, washed once in 10 mM NaCl, then resuspended in a pH 8.5

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Abbreviations: CF_1, chloroplast coupling factor 1 (reversible ATP synthase); DTE, dithioerythritol; P_i, inorganic phosphate; DTT, dithiothreitol; Hepes, 4-(2-hydroxyethyl)-1-piperazineethanesulfonic acid; Taps, 3-{[2-hydroxy-1,1-bis(hydroxymethyl)ethyl]amino}-1-propanesulfonic acid; Tricine, *N*-[2-hydroxy-1,1-bis(hydroxymethyl)ethyl]glycine.

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buffer at 0 °C containing 300 mM sucrose, 25 mM Tricine, 5 mM $MgCl_2$ and 5 mM ascorbate.

The assay for methanol-stimulated ATPase of thylakoids was similar to that described in Ref. 3, in a reaction mixture containing 25 mM Taps (pH 8.5), 20 mM NaCl, 2 mM $MgCl_2$, 5 mM ATP and 35% methanol. Thylakoids (about 10 μg chlorophyll) were injected into the reaction mixture at 37 °C. After 2 min the reaction was stopped by adding the reagent developed by Lebel et al. [4] containing 2 M acetic acid, 0.37 M sodium acetate, 10 mM $CuSO_4$, 1% ammonium molybdate, 0.5% Elon, 1% Na_2SO_3 and 1% sodium dodecyl sulfate. 5 min later a 'color stop' reagent (10% sodium citrate) was added, and the absorbance at 750 nm determined.

Photophosphorylation was measured in reaction mixtures containing 50 mM Tricine-NaOH (pH 8.0 at 25 °C), 5 mM $MgCl_2$, 2 mM $Na_2H^{32}PO_4$, 1 mM ADP and either 1 mM ferricyanide or 50 μM phenazine methosulfate. Acid-base ATP synthesis was performed as described previously [5], with 20 mM succinate at pH 4.2 in the acid stage and 200 mM tricinate at pH 8.2 in the base stage. Separation of ATP from labeled inorganic phosphate was accomplished using the reagent of Sugino and Miyoshi [6] according to the previously described procedure [7]. Azide was added in the base stage only. The supernatant solution containing labeled ATP was used for counting incorporated radioactivity.

Thylakoid ATPase was activated by light and DTT in a procedure similar to that described earlier [8,9]. Thylakoids with about 50 μg of chlorophyll were illuminated for 2 min at about 4000 $\mu Einstein \cdot m^{-2} \cdot s^{-1}$ in 0.5 ml of a medium containing 50 mM Tricine-NaOH (pH8.0), 20 mM NaCl, 10 mM DTT, 2 mM $MgCl_2$ and 50 μM phenazine methosulfate; then injected immediately into 0.5 ml of 50 mM Tricine-NaOH (pH 8.0), 5 mM ATP, 2 mM $MgCl_2$. The reaction proceeded for 5 min in the dark at 37 °C, then was stopped with 0.1 ml 24% trichloroacetic acid, and inorganic phosphate measured as above.

The ATP-P_i exchange reaction was measured as in Ref. 10. Thylakoids with 50 μg of chlorophyll were activated by illumination with DTT as above, then injected into 0.5 ml containing 50 mM Tricine-NaOH (pH 7.6), 2 mM ATP, and 2 mM inorganic ^{32}P. The reaction proceeded in darkness for 5 min, then was terminated by adding 0.1 ml of 24% trichloroacetic acid.

Soluble CF_1 was prepared from spinach as described previously [11]. ATPase reactions were measured in 0.5 ml containing 25 mM Taps buffer (pH 8.5), 20 mM NaCl, 5 mM ATP, either 2.5 mM $CaCl_2$ or 2.0 mM $MgCl_2$, and 25% methanol for Ca^{2+}- or 30% methanol for Mg^{2+}-ATPase. The reaction was carried out at 37 °C, with inorganic phosphate measured as above.

Results

Photophosphorylation was measured with either ferricyanide as electron acceptor (for linear electron flow) or with phenazine methosulfate for cyclic electron flow. In both cases, azide up to 10 mM had no effect, as expected (Table I). However, in other experiments, when the linear electron flow phosphorylation was inhibited 92% by the uncoupler NH_4Cl at 20 mM, 10 mM azide inhibited about 15% of that residual ATP synthesis (data not shown). With the ATP formation driven by an acid-base transition, it took 10 mM azide added to the base stage to inhibit ATP yield 30–50% (Table II). Thus it appears that ATP synthesis is slightly sensitive to azide when the trans-membrane proton gradient is far below optimum.

ATPase of spinach thylakoids can be activated in several different ways. One is by light and

TABLE I

PHOTOPHOSPHORYLATION IN THE PRESENCE OF AZIDE

NaN_3 (mM)	Electron flow type (μmol ATP per mg Chl per h)	
	non-cyclic [a]	cyclic [b]
0	399	1312
0.5	353	1330
1.0	390	1352
3.0	420	1366
5.0	401	1368
10.0	448	1335

[a] Electron acceptor was ferricyanide at 1 mM.
[b] Mediated by *N*-methylphenazonium methosulfate (PMS) at 50 μM.

TABLE II

AZIDE EFFECT ON ACID-BASED ATP SYNTHESIS

The yield is measured in nmol ATP/mg chlorophyll.

Azide (mM)	Expt. 1		Expt. 2		Expt. 3	
	yield	inhib (%)	yield	inhib (%)	yield	inhib (%)
0	161	–	171	–	140	–
3	132	18	142	17	136	3
10	103	36	120	30	68	51

dithiothreitol [12]; the resulting ATP hydrolysis pumps protons inward, maintaining the high energy state and permitting ATPase to remain active. Another is by immersing thylakoids in 33% methanol [13]; in this situation ATPase is active without the high energy state, and does not pump protons into the lumen of the thylakoids [14]. There is a striking contrast between these two activities with respect to inhibition by azide (Fig. 1). The ATPase after activation by light plus dithiothreitol was unaffected by azide at all concentrations tested; while ATPase activity in methanol was extremely sensitive. Inhibition levels from 85 to 100% were achieved readily, between 0.3 and 1 mM azide. Half-maximal inhibition occurred at or below 0.1 mM NaN_3.

Low levels of azide had also been effective in inhibiting thylakoid ATPase after activation by sulfite (Larson, E., in preparation). In those

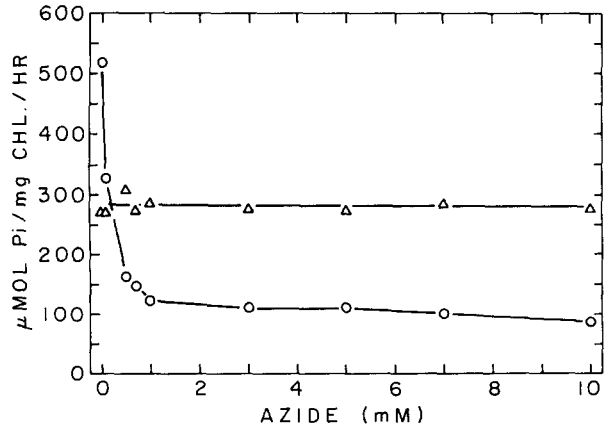

Fig. 1. Comparison of azide effects on the methanol-activated ATPase of spinach thylakoids (circles), with its lack of effect on the ATPase activated by light plus DTT (triangles).

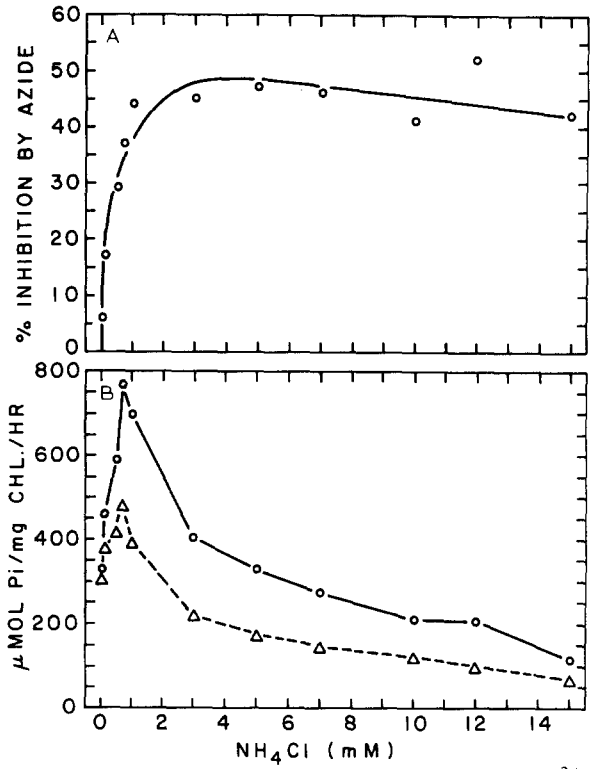

Fig. 2. Azide inhibition of the light plus DTE-activated Mg^{2+}-ATPase as NH_4Cl is added to uncouple the thylakoids. In (B): circles indicate the control rates; triangles are rates with azide added at 1.0 mM.

experiments, both NH_4Cl and gramicidin were present to insure maximal uncoupling. It seemed possible, therefore, that the critical difference in conditions permitting azide inhibition might be the absence of a strong protonmotive force, which is present with light + DTT-activated ATPase. To test this concept more critically, we measured azide sensitivity of the proton-pumping ATPase of thylakoids after activation by light and DTT, using increasing levels of NH_4Cl to bring about increasing degrees of uncoupling. Consistent with the concept of involvement of the proton gradient, azide did inhibit once NH_4Cl was added (Fig. 2). The uncoupler has the expected effect on the control rates; at first stimulating hydrolysis as it relieves inhibition by back-pressure from accumulated internal protons [15], then inhibiting as it collapses the energetic state so that ATPase activity is not adequately maintained. Inhibition by 1

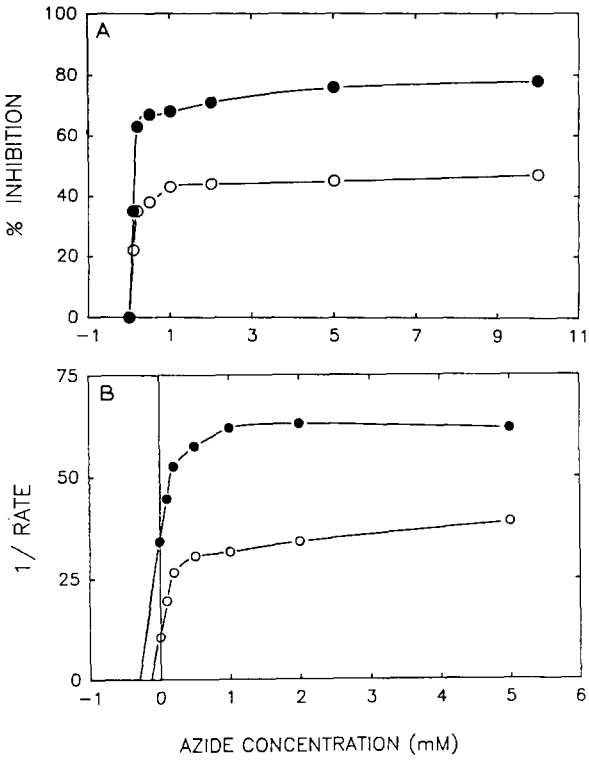

Fig. 3. Response of light + DTT activated ATPase with 2 mM NH_4Cl to increasing concentrations of NaN_3. (A) % inhibition as a function of azide concentration. Closed circles – reaction with 5 mM ATP and 5 mM $MgCl_2$. Open circles – 5 mM ATP and 3 mM $MgCl_2$. (B) Reciprocal rates of ATPase as a function of azide concentration. Data from the same experiment.

mM azide rose from 0 without NH_4Cl, to 45–50% at the maximally stimulating level of NH_4Cl.

The experiments described above were performed during the summer of 1987. On repeating the basic experiment in the fall, azide inhibitions from 20 to 40% occurred in some experiments, but not in others. We believe this may have been due to using partially uncoupled thylakoids, perhaps because of traces of detergent in the glassware, etc., used in their preparation. No matter what the effect of azide was on the control chloroplasts, the inhibition was always more severe when NH_4Cl was used for partial uncoupling.

With NH_4Cl present to insure some uncoupling, the inhibition by azide never reached 100%. A rapid onset of azide inhibition was seen between 0 about 0.2 mM azide; higher concentrations then caused only slowly increasing amounts of inhibition (Fig. 3A). In the region from 0 to 200 μM, reciprocal plots (Fig. 3B) showed K_i values for azide between 80 and 350 μM, in different experiments. While the inhibition was greater with a higher Mg^{2+} level in Fig. 3, in other experiments no consistent correlation was found between the extent of inhibition and the concentration of free Mg^{2+} in the reaction.

Attempts were made to study the effect of azide on the ATP kinetics of the ATPase activated by methanol. The results were quite variable, with the lines for control and azide-inhibited series in double reciprocal plots crossing sometimes at or before the ordinate, and with other thylakoid preparations crossing after the ordinate (Fig. 4, for instance). The azide effect is probably best described as showing mixed inhibition. Apparent K_i values were calculated in various experiments

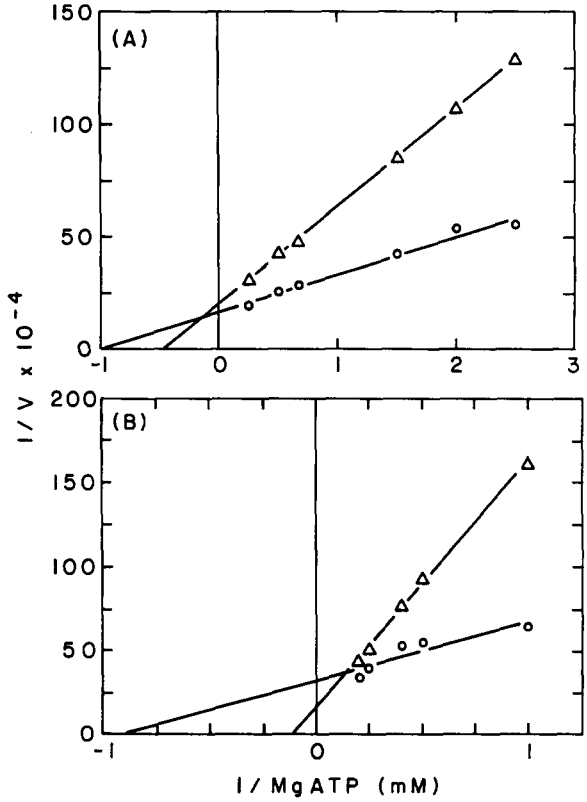

Fig. 4. Azide effect on kinetics of methanol-activated Mg^{2+}-ATPase with respect to ATP concentration. Two experiments are shown, illustrating the range of apparent kinetic results obtained using different preparations of thylakoids. Control rates: circles; with 0.1 mM (Fig. 4A) or 0.5 mM (Fig. 4B) NaN_3: triangles.

TABLE III

AZIDE FAILS TO INHIBIT Ca-ATPase OF SOLUBLE CF_1

Activation of CF_1 was by 25% methanol when Ca^{2+} was the cation, and by 30% when Mg^{2+} was the cation.

Cation	NaN_3 (mM)	ATPase (μmol P_i per mg protein per min)		
		Expt. 1	Expt. 2	Expt. 3
Ca^{2+}	0	5.20	5.67	–
Ca^{2+}	1	5.17	5.67	–
Mg^{2+}	0	15.7	16.3	12.3
Mg^{2+}	1	11.2	11.7	5.33

between 5 and 65 μM.

One consideration for the action of azide might be an effect on the 'regulatory' nucleotide site(s). However, regulatory effects are found with adenylates, but not guanylates [16]. It was thus of interest to see if the characteristics of azide inhibition were any different when measuring GTP hydrolysis by methanol-activated thylakoids. This was not the case; the curve for inhibition of GTPase as a function of NaN_3 concentration was identical to that for inhibition of ATPase. In a typical experiment, 0.5 mM inhibited ATPase aroused by methanol 78%, and GTPase under the same conditions 79%.

A relation between inhibition by N_3^- and Mg^{2+} is indicated in the mitochondrial literature [1]. This appears to be the case here as well, since the ATPase activity of thylakoids in methanol was not affected by 1 mM azide when Ca^{2+} was the cation, while this level inhibited 30–50% when Mg^{2+} was the cation used (Table III).

We examined the effect of azide on the ATP-P_i exchange reaction, which is thought to consist of

TABLE IV

EFFECT OF NaN_3 ON ATP-P_i EXCHANGE OF SPINACH THYLAKOIDS

The rates are measured in μmol ATP-P_i exchange per mg Chl per h.

Azide (mM)	Expt. 1		Expt. 2		Expt. 3	
	rate	inhib (%)	rate	inhib (%)	rate	inhib (%)
0	14.5	–	9.62	–	8.92	–
5	8.3	43	5.03	48	6.63	26
10	3.4	77	3.47	64	4.13	54

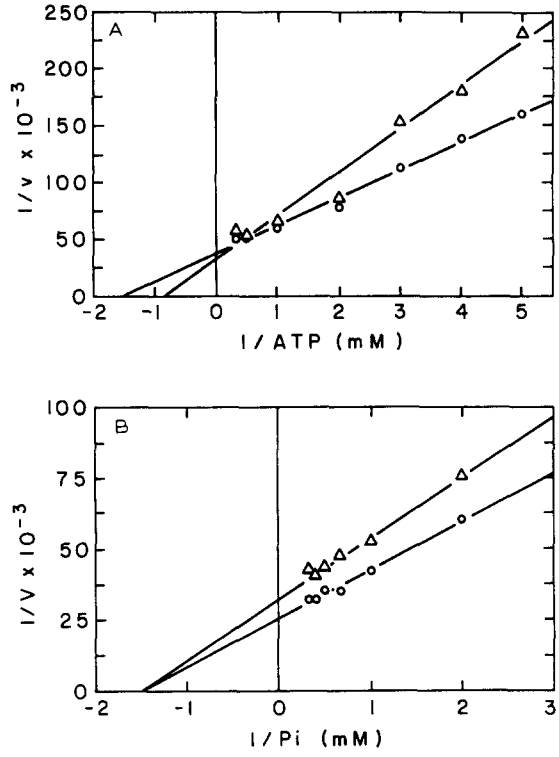

Fig. 5. Azide effect on kinetics of the ATP-P_i exchange reaction. Fig. 5A, ATP concentrations; Fig. 5B, P_i concentrations. Triangles, with 2.5 mM NaN_3; circles, no azide. Azide inhibition is close to competitive with the ATP, non-competitive with P_i.

complete cycles of ATP synthesis and hydrolysis [17]. Inhibition did occur (Table IV). In this reaction, azide was approximately competitive with ATP (Fig. 5), but clearly non-competitive with P_i. Thus the extent of the inhibition was a strong function of the concentration of ATP used in the reaction. However, azide was a rather weak inhibitor for the exchange reaction, with half-maximal inhibition occurring only between 5 and 10 mM (Table IV), instead of the 0.1 mM level as for thylakoid ATPase when activated by methanol.

Discussion

While the failure of azide to inhibit photophosphorylation is well known, we were not able to find any published reference to its use with the ATPase of thylakoid membranes or soluble CF_1 prior to the abstract from this laboratory [18]. One

reason for this may be that azide fails to inhibit either the tightly coupled, proton-pumping ATPase aroused by light and dithiothreitol (Fig. 1) or the Ca^{2+}-dependent activity of soluble CF_1 (Table III). These have been among the most commonly used assays for thylakoid ATPase in the past. On the other hand we can easily see that azide is a potent inhibitor of the non-proton pumping, methanol-activated Mg^{2+}-dependent ATPase (Fig. 1), of the sulfite-activated Mg^{2+}-ATPase when maximally uncoupled (Larson, E., in preparation) and of the Mg^{2+}-dependent ATPase of soluble CF_1 (Table III). The major factor preventing inhibition of thylakoid-bound light + dithiothreitol-activated ATPase appears to be the protonmotive force, since sensitivity to azide returns in the presence of uncoupling levels of ammonium chloride (Fig. 2).

The presence of a high protonmotive force can only be a part of the reason for the insensitivity to azide of photophosphorylation. The effect of azide was restricted to about 15% inhibition even with 20 mM NH_4Cl present, cutting the phosphorylation rate without azide to less than 10% of the control. The protonmotive force must have been very low indeed under these conditions, and it still took 5–10 mM azide to have even this minuscule effect. This is quite a contrast to the situation with uncoupled ATPase, where down to 0.1 mM azide can inhibit 50%.

Azide inhibition of acid-base phosphorylation seems more significant (Table II), perhaps because the pH gradient decays during ATP synthesis [19]. However, the extent of inhibition was erratic between experiments, and also did not change when the size of the artificially imposed pH gradient was varied between 2.8 and 4.2 pH units (data not shown). Overall, our data indicate that the relative insensitivity of ATP synthesis to azide must have some basis beyond that of the transmembrane protonmotive force.

The azide inhibition of P_i-ATP exchange has to be ascribed to the ATP hydrolysis portion, therefore. In this reaction, the initial illumination builds up a pmf and activated CF_1. Subsequent ATP hydrolysis in the dark pumps protons in, maintaining the pH gradient; however, some of this energy is used in rephosphorylation of the ADP formed, since the exchange reaction consists of a complete reversal of ATP synthesis and hydrolysis [17]. Use of energy for rephosphorylation of ADP means the pH gradient is less than optimal, and therefore some inhibition shows up. Without the inorganic phosphate added, during simple ATP hydrolysis after activation by light and DTT, the proton gradient is not consumed and no inhibition by azide is seen (Fig. 1).

Even the apparent competitive relationship between ATP and azide can be ascribed to this factor. At the lower levels of ATP, both ATP hydrolysis and the proton pumping to which it is coupled are limited. The leak rate for proton efflux from the thylakoids would not be affected, and therefore the steady-state pH gradient will be lower when ATP levels are low, leading to greater inhibition by azide. High ATP levels bring on faster ATP hydrolysis, faster proton pumping and therefore a higher pH gradient, and so azide will inhibit less. This effect of ATP would thus appear to be kinetically competitive with azide.

Inhibition by azide cannot be described as simply non-additive with that of the high-energy state, since rates are decreased to below those of the coupled state by sufficient azide (Figs. 1 and 2). Thus the azide inhibition must go beyond the constraints imposed on ATPase by internal protons.

We are left with the finding that a trans-membrane protonmotive force prevents azide inhibition of ATPase for still unknown reasons. Two speculative working hypotheses seem possible. The simpler one is that the high-energy state induces a conformation of CF_1 in which the binding site for azide is hidden. It is suggestive that the gamma subunit was essential for azide to inhibit ATPase of the reconstituted F_1 of thermophilic bacteria [20]. In CF_1 on thylakoid membranes it is clear that the gamma subunit has a very different exposure to the medium in the light than in the dark. Apparently, the gamma subunit disulfide bond is more exposed to reductants in the light than in the dark [21]; but the resulting reduced dithiols are less exposed to oxidants when the membranes are in the high-energy state [22]. The latter phenomenon might be analogous to our current results.

A more complex concept for the antagonism between the energized state and inhibition by azide is that, as for mitochondrial F_1 [1], azide inhibi-

tion may be synergistic with free (inhibitory) Mg^{2+}. If one function of the protonmotive force is to provide protection for CF_1 against inhibition by free Mg^{2+}, it could at the same time act against the effectiveness of azide as an inhibitor. Further tests of this concept are needed for a more complete evaluation.

It is not clear yet whether the protection of CF_1 ATPase by the transmembrane protonmotive force is a general phenomenon with all enzymes of this type. In experiments with the F_1/F_0 of the thermophilic bacterium incorporated into planar phospholipid bilayers [23] azide inhibited ATPase in spite of the presence of a considerable membrane potential. It would be interesting to see if the bacterial ATPase would be protected by a transmembrane pH gradient, which is the main component of the thylakoid protonmotive force.

It is of interest to speculate on the site on CF_1 where N_3^- binds, and has such strong effects on catalytic function. If azide binds to one of the regulatory sites, which are specific for adenylates, one might expect a stronger inhibition of GTPase than of ATPase, with no adenylates added to deflect azide binding. Since that was not the case, azide is more likely to affect catalysis directly than act at the level of enzyme regulation.

Reconstitution using purified subunits of the thermophilic bacterial F_1 indicated the need for the gamma subunit in order to have ATPase inhibitable by azide [20]. That implies the binding site for azide is likely to be on the gamma, not on the β subunit and therefore not on the catalytic site. (Alternatively, exposure of an azide binding site on β might depend on its interacting with the gamma subunit). On the other hand, evidence from altered sensitivity to azide induced by mutating an amino acid in the β subunit of the *Escherichia coli* F_1 led to the suggestion that β does have the azide binding site [24]. The latter work indicated that quercetin and azide may share the same binding site in *E. coli*. The situation seems rather different in chloroplasts. First, azide fails to inhibit the Ca^{2+}-dependent ATPase of chloroplasts (Table III), but does inhibit that of *E. coli* [24]. Secondly, quercetin and azide have differing modes of action for chloroplasts, since quercetin inhibits photophosphorylation [25] while azide does not.

With the F_1 ATPase from mitochondria, inhibition by azide has a slow onset, related to the presence of Mg^{2+} in the absence of ATP [1]. We found this was not the case with CF_1. When thylakoids were incubated for varying lengths of time (0–5 min) with 5 mM azide prior to initiating the ATPase reaction, whether with or without 5 mM $MgCl_2$, the % inhibition of ATPase was the same throughout (data not shown).

Nevertheless, the inhibition by azide is in some way related to the presence of free Mg^{2+}, since azide does not inhibit the Ca^{2+}-dependent ATPase of soluble CF_1 (Table III). It seems significant that sulfite shows a similar differential, in that it stimulates ATPase of soluble CF_1 with Mg^{2+} as divalent cation, much more than with Ca^{2+} as the cation. Also, we should note that Mg^{2+}, but not Ca^{2+}, is effective as the divalent cation for ATPase of light + DTT-activated thylakoid ATPase, and for photophosphorylation.

These differentials in azide (and sulfite) sensitivity for Mg^{2+} vs. Ca^{2+} with thylakoid ATPase add to a body of evidence for differences between the reactions facilitated by the two cations with CF_1. The variety of effects begins to support the possibility of fundamental differences in mechanism between the two. Earlier observations include the fact that sulfite does not stimulate the Ca^{2+}-ATPase of epsilon-deficient CF1 [26], the much higher rates with Mg^{2+} than with Ca^{2+} [14]; and the absence of the oxygen exchanges during Ca^{2+}-dependent ATP hydrolysis, but presence with the Mg^{2+}-ATPase [27]. Similarly, in the related F_1 of *E. coli,* site-directed mutagenesis showed serine 174 is critical for Mg^{2+}-ATPase, but not for Ca^{2+}-ATPase [28].

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