Mechanism of the Reductive Activation of Succinate Dehydrogenase*

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SUMMARY

When succinate dehydrogenase contains oxalacetate in firmly bound form, activity cannot be expressed without special pretreatment ("activation") of the enzyme. Reduction of the enzyme results in dissociation of oxalacetate and activation of the enzyme. The course of reductive titrations appears the same whether or not the enzyme contains oxalacetate, and complete reduction as monitored by bleaching of chromophoric groups requires the incorporation of 6 to 7 reducing equivalents in either case. The stoichiometry is that expected from the non-heme iron and flavin content of the enzyme. Activation of the enzyme during reductive titrations occurs predominantly with the incorporation of the second pair of electrons, while determination of activation levels at various poised potentials shows that the group involved is reduced with the uptake of 2 H+ and 2 e−. These characteristics are consistent with titration of the flavin moiety rather than non-heme iron groups. Thus it appears that activation is concurrent with the reduction of flavin to the hydroquinone form. From the measured half-reduction potential for activation, that of the flavin in an oxalacetate-free enzyme has been estimated at −90 to −60 mv at pH 7.

The activation of succinate dehydrogenase, originally observed some 20 years ago (1), has more recently been related to the dissociation of tightly bound oxalacetate from the enzyme (2). Oxalacetate binds in an almost irreversible fashion to succinate dehydrogenase, and persists through treatments like dialysis or Sephadex passage; any oxalacetate already bound to the enzyme when it is extracted from tissue remains bound through most of the procedures used to isolate the enzyme (2). The catalytic activity of the enzyme cannot be expressed in this "deactivated" state and it is necessary to "activate" the enzyme and dissociate the bound oxalacetate for assay purposes. Activation can be accomplished in a number of ways (3), generally involving pretreatment of the enzyme with succinate prior to assay. The deactivated enzyme preparations, containing bound oxalacetate, were obtained by incubating activated enzyme with excess oxalacetate at 20° in 50 mM Hepes/NaOH/500 mM NaN303/100 mM semicarbazide-HCl, pH 7.0, at a protein concentration of 5 mg/ml. The oxalacetate dissociated thereby was removed by passage through Sephadex G-50 equilibrated with the same activating mixture. The activating anion mixture then was removed by passage through Sephadex G-50 equilibrated with 50 mM Hepes/NaOH, pH 7.8; preparations of type 3-NS enzyme were rendered free of extraneous salts in similar fashion.

Deactivated enzyme preparations, containing bound oxalacetate, were obtained by incubating activated enzyme with excess oxalacetate at 20° in 50 mM Hepes/NaOH, pH 7.0, until essentially no catalytic activity could be detected; unbound oxalacetate then was removed by Sephadex chromatography or, in the case of particulate preparations, by repeated washing and centrifugation. For preparations to be labeled with radioactive oxalacetate, the binding procedure was carried out with [14C]oxalacetate generated by glutamate-oxalacetate transaminase (Boehringer Corp.) from α-ketoglutarate and uniformly labeled L-[14C]glutamate (Amersham/Searle Corp.) (9).

Fully activated enzyme is defined as that obtained by incubation of the enzyme with succinate prior to assay. The activated fraction of the enzyme is equated with the difference between the activities observed in the catalytic assay at 15°, with and without this prior treatment. Determinations of activity and protein were

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ETP was isolated from beef heart mitochondria as previously described (6). Soluble succinate dehydrogenase was prepared either from an acetone powder of ETP according to Coles et al. (7) (type 1 enzyme) or by perchlorate extraction of Complex II (8), but in the absence of succinate (type 3-NS enzyme) (7).

Type 3-NS enzyme is fully activated as prepared. Type 1 enzyme was activated by incubation at 20° in 50 mM Hepes/NaOH/500 mM NaN303/100 mM semicarbazide-HCl, pH 7.0, at a protein concentration of 5 mg/ml. The oxalacetate dissociated thereby was removed by passing the reaction mixture through a Sephadex G-50 column equilibrated with the same activating mixture. The activating anion mixture then was removed by passage through Sephadex G-50 equilibrated with 50 mM Hepes/NaOH, pH 7.8; preparations of type 3-NS enzyme were rendered free of extraneous salts in similar fashion.

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The abbreviations used are: ETP, non-phosphorylating sub-mitochondrial particle preparation from beef heart; Complex II, succinate-ubiquinone reductase; Hepes, 4-aminoethylpiperazine-N′-2-ethanesulfonic acid.
done as in previous work (10). The concentration of enzyme was determined by analysis for histidyl flavin (11). Non-heme iron was measured by the method of Brumby and Massey (12) and oxalacetate and malate were measured fluorometrically, according to Williamson and Corkey (13).

All anaerobic experiments were performed in an atmosphere of helium that had been purified over hot copper and saturated with water in a gas train made of glass and butyl rubber tubing. Dithionite solutions, prepared anaerobically and standardized by anaerobic titration of a solution of 3-methylumbelliflavin, were stored in a gas-tight Hamilton syringe. Anaerobic titrations of succinate dehydrogenase with the standardized dithionite solution were performed in a glass cuvette, similar to that described by Burleigh et al. (14). Potentiometric titrations were carried out at 25°C in an apparatus essentially like that described by Dutton (15) under a continuous stream of water-saturated helium. Aliquots of enzyme taken for measurement of activity during both types of titrations were withdrawn from the respective reaction vessels under a stream of helium into a helium-flushed gas-tight syringe. A small amount was first ejected from the syringe and the requisite aliquot then was injected from the syringe directly into a prepared cuvette containing the otherwise complete reaction mixture for assay. This precaution was necessary because, after reduction with dithionite, the enzyme was very rapidly inactivated on exposure to air.

Absorption spectra were taken in a Cary 14 spectrophotometer thermostated at 25°C and corrected fluorescence spectra were determined with a Perkin-Elmer fluorescence spectrophotometer (MPF 3).

RESULTS

Spectrophotometric Changes—The oxidation-reduction groups in the enzyme which may be monitored spectrophotometrically during reductive titrations are the non-heme iron and flavin moieties. Directly relating the reduction of these groups to activation of the enzyme and dissociation of oxalacetate is complicated, however, by the fact that their contributions to the spectra are not clearly distinguished and by the fact that binding of oxalacetate itself is accompanied by spectral changes. The latter point is illustrated in Fig. 1, which shows the difference spectrum obtained when a sample of type 3-NS enzyme is treated with oxalacetate. The readily discernible changes are an increase in absorbance at wavelengths above 500 nm, an apparent bleaching with three minima at 370, 458, and 485 nm, and isosbestic points at 330, 410, and 500 nm. These observations are in essential agreement with those of DerVartanian and Veeger (16) with a different type of succinate dehydrogenase preparation. Absorbance measurements at the isosbestic wavelengths eliminate spectral interference by bound oxalacetate and permit a direct comparison of the reduction characteristics of the oxalacetate-bound and free enzyme. In Fig. 2, the loss of absorbance at 410 and 500 nm by both forms of the enzyme, when titrated anaerobically with sodium dithionite, has been correlated with the number of electrons added per molecule of enzyme.

It is evident that full reduction of the enzyme is effected by the incorporation of 6 to 7 electrons/mol of enzyme, and that this number is the same whether or not the enzyme is bound to oxalacetate. The activation of the enzyme induced during the titration is also plotted in Fig. 2. The activation profile is sigmoidal in shape; no activation resulted from the introduction of 1 electron/enzyme molecule, and only a 15% increase in activation occurred with the addition of the 2nd electron, despite considerable reduction of chromophoric groups (44% of the possible bleaching at 500 nm and 25% at 410 nm had occurred). Incorporation of the 3rd and 4th electrons induced full activation. The further addition of reducing equivalents is thus unnecessary for activation but needed for full reduction of the enzyme. The stoichiometry obtained in this experiment, 6 to 7 electrons/enzyme molecule, is compatible with its non-heme iron and flavin content (8.3 iron atoms and 1 flavin/mol), if one accepts that each pair of iron atoms requires 1 electron for reduction (17) and that the flavin is reduced to the hydroquinone form, with the uptake of 2 electrons and 2 protons. This agreement was not found in all preparations of the enzyme. The type 1 preparations examined, which usually contained 3 to 4 nmol of flavin and 18 to 24 ng atom of iron/mg of protein, as well as some type 3-NS

![Fig. 1. Oxalacetate-induced difference spectrum of succinate dehydrogenase. The enzyme was an oxalacetate-free type 3-NS preparation, dissolved in 50 mM Hepes/NaOH, pH 7.5, at 25°C (2.1 mg of protein/ml; 14.4 \( \mu \)M enzyme). A baseline was taken of enzyme (○), following which oxalacetate was added to the sample cuvette (final concentration = 100 \( \mu \)M), an equal volume of buffer was added to the reference cuvette, and the spectrum was again recorded (×).](http://www.jbc.org/content/jbc/247/3/7115.full)

![Fig. 2. Anaerobic titration of active and deactivated succinate dehydrogenase with dithionite: absorbance changes and activation. Both oxalacetate-free and oxalacetate-bound enzyme were titrated with sodium dithionite anaerobically in 50 mM Hepes/NaOH, pH 7.5, at 25°C, and at a protein concentration of 2.1 mg/ml (16.8 \( \mu \)M enzyme). The oxalacetate-bound enzyme contained 0.03 mol of oxalacetate/mol of enzyme. Spectra were recorded after each addition of dithionite and samples were taken for measurement of activity when absorbance changes were complete. The absorbance of the oxalacetate-bound enzyme at 410 and 500 nm is given by ○ and △, respectively, that of the oxalacetate-free enzyme by × and □, and the level of activation of the oxalacetate-bound enzyme by ●; all values are corrected for dilution and removal of samples during the titration. In another experiment the level of activation is plotted (■) during a similar titration of type 3-NS enzyme in the presence of excess oxalacetate (7.7 mol/mol enzyme; 1.52 mg of protein/ml, 10.4 \( \mu \)M enzyme).](http://www.jbc.org/content/jbc/247/3/7115.full)
prepared (6 to 8 nmol of flavin and 48 to 64 ng atom of iron/mg of protein), required up to 1.5 more electrons for full reduction than was anticipated from their non-heme iron and flavin content. This was attributed to the presence in these preparations of varying amounts of an impurity of relatively low oxidation-reduction potential, since the first part of the titration was unchanged, i.e. 3 to 4 electrons always induced activation, and the variability was related to the number of equivalents needed to finish the titration, and the total was the same whether or not the enzyme contained bound oxalacetate.

The identical titration stoichiometry in the presence and absence of oxalacetate imply that oxalacetate itself is not reduced. In agreement with this, the presence of free oxalacetate neither changed the characteristics of activation nor the reducing equivalents required for the purpose (Fig. 2, solid squares). Direct evidence that oxalacetate was not reduced to malate during the activation process was obtained by anaerobic addition of perchloric acid (5% final concentration) to the anaerobic cuvette contents after the enzyme had been activated by reduction with dithionite in a similar experiment. The original complement of oxalacetate was recovered in the protein-free supernatant solution (Table I), while assays for malate, sensitive enough to detect as little as 10% reduction of the oxalacetate originally bound to the enzyme, proved negative.

In the experiments detailed thus far, it has been assumed that oxalacetate is dissociated from the enzyme during reductive activation, as it is in activation by other means (2). In Fig. 3 this point is documented. Aliquots of enzyme, bound with [14C]-oxalacetate (0.9 mol/mol of enzyme), were passed through anaerobic columns of Sephadex G-50 in the presence and absence of 3 mM sodium dithionite. The elution profiles for each condition, Fig. 3, A and B, shows that in the absence of dithionite all counts were eluted with the protein, i.e. oxalacetate was bound in essentially irreversible fashion to the enzyme, whereas 90% of the oxalacetate was separated from the protein in the presence of dithionite. These data show that reduction of the enzyme decreases its affinity for oxalacetate by at least 1 order of magnitude.

**Oxidation-Reduction Titrations**—The nature of the component of the enzyme titrated with the incorporation of the 3rd and 4th electrons/ enzyme molecule, whose oxidized or reduced states so influence oxalacetate binding, was further studied in experiments where the effect of poisoning the oxidation-reduction potential on the level of activation was measured. Fig. 4 shows that the reductive activation of a deactivated type 1-soluble preparation at pH 7.6 is a sigmoidal function (crosses) of the oxidation-reduction potential relative to the standard hydrogen electrode (Eh). Back titration of the contents of the reaction vessel with ferricyanide (open circles) did not re-establish the original level of activation of the enzyme that had been observed at the various potentials during the reductive part of the experiment (crosses). A clear explanation for this is not apparent, although it may be attributable in part to some destruction of oxalacetate under the experience.

![Figure 3](http://www.jbc.org/)

**Figure 3.** Sephadex chromatography of [14C]oxalacetate-bound succinate dehydrogenase in oxidized and reduced form. One-milliliter aliquots of type 1 enzyme, containing 6.48 mg of protein, 26.6 nmol of enzyme, and 24 nmol of uniformly labeled [14C]oxalacetate, were applied to Sephadex G-50 columns, 0.9 × 20 cm, equilibrated at 4°C with oxygen-free 50 mM Hepes, pH 7.8, ±3 mM sodium dithionite. Fractions of 0.3 ml were collected and assayed for enzyme activity (●) and radioactivity (○). The specific activity of the oxalacetate was 5,348 cpm/nmol.

**Figure 4.** Potentiometric titration of deactivated succinate dehydrogenase. deactivated type 1 enzyme (2.15 mg of protein/ml, 9.0 μM enzyme) was titrated at 25°C in 50 mM Hepes/NaOH buffer, pH 7.5, in the presence of 60 μM concentrations of the oxidation-reduction mediators listed as follows, with their respective midpoint potentials given in parentheses: phenazine methosulfate (+80 mv), juglone (+30 mv), duroquinone (+5 mv), indigo tetrasulfonate (−46 mv), indigo disulfonate (−125 mv), and anthraquinone-2-sulfonate (−225 mv). Aliquots of the reaction mixture were taken anaerobically for assay of enzyme activity during the titrations, which were carried out on the same sample in the following sequence: X, plus dithionite; ○, plus ferricyanide; ▲, plus additional oxalacetate to give 67 μM and then dithionite again; ●, plus ferricyanide again.
mental conditions. A completely reversible system pertained only if the forward and back titrations were carried out with an excess of free oxalacetate present, as in the experiment represented by the closed symbols, where a 7-fold excess of oxalacetate (67 μM) over deactivated type 1 enzyme (9 μM) was used. All further experiments of this type were carried out in the presence of sufficient oxalacetate to ensure that the system was at equilibrium.

The half-reduction potential ($E_m$) of the component(s) of the enzyme influencing activation may be obtained from a plot of $E_3$ against log (oxidized component)/reduced component according to the Nernst equation, by equating the oxidized component with the deactuated fraction of the enzyme and the reduced component with the activated fraction. Fig. 5A shows such plots obtained with a Complex II preparation during reductive and reoxidative phases of titrations carried out in the presence of a 10-fold excess of oxalacetate, and, in addition, one reductive titration with still higher oxalacetate (30-fold excess).

The fact that the data from these three titrations can be expressed within experimental error as a single linear plot strongly implies that the reactivation of oxalacetate-bound succinate dehydrogenase stems from the reduction of only one of its several components. The $E_m$ value of this component was -100 mv at pH 7.5 and $n = 2$. Similar values were obtained with type 3-NS and type 1 enzymes and with the membrane-bound succinate dehydrogenase in ETP preparations. The half-reduction potential of this component in ETP was linearly dependent on pH in the range pH 6.0 to 9.0 (Fig. 5B) and was thus dependent on the transfer of protons as well as electrons. Since the slope of Fig. 5B was -0.056 volts/pH unit increase and $n = 2$, the number of protons involved was also 2. These results implicate the flavin component and imply that activation of the enzyme by dithionite occurs because the flavin is reduced to the hydroquinone form.

Spectrophotometric Evidence for Role of Flavin—Further evidence for the involvement of the flavin could be obtained by reconsideration of spectrophotometric data from anaerobic titration experiments (Fig. 2). Since flavin absorbs maximally at 450 nm but not at all at 550 nm, these data may be expressed for emphasis of the contribution of the flavin moiety, if the ratio of absorbance changes occurring at 450 and 550 nm (ΔA450/ΔA500) is plotted as a function of electron input per enzyme molecule as has been done with xanthine oxidase by Olson et al. (18). With succinate dehydrogenase this approach can be used only in the case of the oxalacetate-free enzyme since 450 and 550 nm are not isobestic wavelengths. This is permissible, however, since we have shown that oxalacetate does not affect the stoichiometry of the titration. Fig. 6 shows that the ratio is approximately 2.0 early in the titration, reflecting absorbance changes of the non-heme iron centers known to be reduced from EPR experiments (19). The ratio increases to a maximum value of 6.0 when 4 electrons have been incorporated. This is consistent with reduction of the flavin, which causes maximal loss of absorbance at 450 nm but no change at 550 nm, where flavins do not absorb.

The incorporation of 4 electrons is the exact complement required for a 90 to 100% activation of the oxalacetate-bound enzyme. The 2 extra electrons needed for full reduction of the enzyme caused the ratio to decrease to a final value of ~5.0. This last phase probably reflects the reduction of non-heme iron chromophores, known to be present in these enzyme preparations, which are of very low potential and the last oxidation-reduction components to be titrated (19). A preparation of type 1 enzyme, with lower non-heme iron content and an expected titer of 5 reducing equivalents/mol, gave similar data.

Fluorometric Evidence for Role of Flavin—As was reported earlier by Veeger (20) flavin fluorescence is observable in succinate dehydrogenase preparations. In type 3-NS enzyme, the fluorescence has the emission characteristics of flavin fluorescence (Fig. 7) and amounts to approximately 1% of that expected for a comparable concentration of free riboflavin. The relationship of the fluorescence to succinate dehydrogenase (rather than to trace impurities) is indicated by the fact that oxalacetate, on binding

![Figure 5](http://example.com/fig5.png)

**Fig. 5.** A, effect of oxidation-reduction potential on the activation level of deactivated Complex II in the presence of excess oxalacetate. The enzyme (2.3 mg of protein/ml) was titrated at pH 7.5 with dithionite in the presence of oxidation-reduction mediators as in Fig. 4. Activity was monitored during reduction titration with dithionite (X) in the presence of 67 μM oxalacetate, then during reoxidation with ferricyanide (O), and during reduction with dithionite (Δ) in the presence of 202 μM oxalacetate. B, pH dependence of the half-reduction potential in the presence of 67 μM oxalacetate. ETP (5.76 mg of protein/ml) were titrated as in Fig. 4, but in a medium containing, in addition to the mediators, 200 mM sucrose, antimycin A (1 μg/mg of protein), and one of the following buffers at 50 mM concentration: 2-(N-morpholino)ethanesulfonic acid/NaOH (pH 6.27), Hapes/NaOH (pH 6.0, 6.7, 7.5), glycylglycine/NaOH (pH 8.3) and Hapes/Tris (pH 8.8). Other half-reduction potentials similarly determined, which are included in this figure are for type 1 enzyme (O) and Complex II (Δ) in Hapes/NaOH at pH 7.6, and for type 3-NS enzyme (●) at pH 7.5.

![Figure 6](http://example.com/fig6.png)

**Fig. 6.** Ratio of absorbance changes at 450 and 550 nm during reductive titration. Data for the type 3-NS enzyme (○) were taken from the experiment of Fig. 2. Data for type 1 enzyme (3.4 mg of protein/ml, 12.7 μM enzyme, ratio of non-heme iron to flavin, 5:4:1) were similarly obtained (X).
wavelength was 445 nm. Curve A, no additions; Curve B, plus 85 

Fig. 7. (left). Fluorescence emission spectra of succinate dehydrogenase. Type 3-NS enzyme (1.21 mg of protein/ml, 9.7 μM enzyme, in 50 mM Hepes/NaOH, pH 7.5) was used. Excitation 

FIG. 460 540 600 0 50 100 

The half-reduction potential of the component associated with reductive activation of succinate dehydrogenase is one further point in favor of flavin reduction as the key step. The experimentally determined value, corrected for the effect of pH (by interpolation from Fig. 5B), is -120 mv at pH 7.0, when determined in the presence of oxalacetate. The half-reduction potential ($E_m$) of the couple, corrected for the effect of oxalacetate binding, may be estimated from the following equation:

$$E_m (observed) = E_m + \frac{RT}{nF} \log \frac{1 + K_R [OAA]}{1 + K_0 [OAA]}$$

where $K_R$ and $K_0$ are the equilibrium constants for the binding of oxalacetate to the reduced and oxidized forms of the enzyme, respectively, and [OAA] is the concentration of oxalacetate used. The dissociation constant ($K_0$) of the enzyme-oxalacetate complex has been reported to be $10^{-6}$ to $10^{-2}$ M (16, 21, 22) and it is apparent (Fig. 3) that the affinity of oxalacetate for the reduced enzyme is at least 1 order of magnitude lower than for the oxidized form. The true half-reduction potential then falls in the range of -90 mv. This value is probably the same for all types of enzyme preparations since the experimental data were the same with soluble enzymes of type 1 and 3-NS, with Complex II, and with ETP (Fig. 5B). The value is intermediate between the half-reduction potentials determined for the non-heme iron centers in EPR experiments and is clearly not coincident with any of them. Thus the enzyme in particular preparations is known to contain three types of iron-sulfur centers, one (Hipip, or S2) which resembles the bacterial high potential iron proteins (16) and two of the ferredoxin type, S1 and S3 (17, 23). The half-reduction potentials of these have been determined as +65, -5, and -250 mv, for the Hipip, S1, and S3 centers, respectively (23, 24). Reductive
titration of the enzyme would therefore be expected to proceed in the sequence Hipp, iron center S1, flavin (i.e. group involved in activation), and the low potential iron center S0.

If it is accepted that the half-reduction potential measured for the activation process is that of the flavin moiety, then it is also of interest that the value is significantly different from that of synthetic 8a-[N-3-histidylriboflavin (25) or of a histidyl-FAD peptide from sarcosine dehydrogenase (26), which are −160 and −167 mV, respectively. These compounds reproduce the main point of attachment of the flavin to the succinate dehydrogenase protein, namely, the covalent bond between a histidine residue and 8-riboflavine. This bond in itself modifies riboflavin or FAD so that the half-reduction potential is raised some 40 mV (E_{FAD} = −209 mV (27)). The complete protein environment thus would have a more pronounced effect, raising the E_m value to at least −90 mV.

The spectrophotometric and fluorometric data which we have obtained with the soluble enzyme are also in accord with this sequence of reduction, for the color changes observed early in the titration resemble bleaching of iron chromophores, whereas changes typical of flavin reduction become prominent as the 3rd and 4th electrons are incorporated. EPR data have shown that iron-sulfur center S1 present in soluble and particulate preparations of the enzyme, is one of the components titrated with the flavin. The lability of the Hipip center is therefore related to any known iron-sulfur center. The availability of the Hipip center, however, in terms of its EPR signal has been documented (19, 24), and it remains possible that some modification of the Hipip center might occur during solubilization of the enzyme that results in loss of the EPR signal without significant impairment of the chromophoric and oxidation-reduction properties.

The molecular details of the effect of flavin reduction on the oxalacetate binding site remain to be investigated. The quenching of fluorescence on binding oxalacetate to the substrate binding site is perhaps suggestive of a close relationship. It has been found that a free sulfhydryl group is required for binding oxalacetate (25), presumably forming a thiohemiacetal bond with the α-keto group of oxalacetate. Since these bonds are quite labile in model systems, stabilization is probably achieved through the enzyme environment. Reduction of the flavin could alter the protein conformation about the oxalacetate binding site and thereby stabilize the thiohemiacetal bond to solvent hydrolysis. This would explain the finding that the affinity of oxalacetate is much greater for the oxidized enzyme than for the reduced form.

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