The transport of ATP out of mitochondria and uptake of ADP and Pi into the matrix are coupled to the uptake of one proton (Klingenberg, M., and Rottenberg, H. (1977) Eur. J. Biochem. 73, 125-130). According to the chemiosmotic hypothesis of oxidative phosphorylation this coupling of nucleotide and Pi transport to proton transport implies that the P/O ratio for the synthesis and transport of ATP to the external medium is less than the P/O ratio for the synthesis of ATP inside mitochondria. A survey of previous determinations of the P/O ratio of intact mitochondria showed little convincing evidence in support of the currently accepted P/O ratio of 3 with NADH-linked substrates and 2 with succinate thiokinase. We have measured P/O ratios in rat liver mitochondria by the ADP pulse method and by 32P, e2P, esterification, measuring oxygen uptake with an oxygen electrode, and find values close to 2 with \( \beta \)-hydroxybutyrate as substrate and 1.3 with succinate as substrate in the presence of rotenone to inhibit NADH oxidation. These values were largely independent of pH, temperature, Mg\(^2+\) ion concentration, ADP concentration, ADP pulse size, or amount of mitochondria used. We suggest that these are the true values of the P/O ratio for ATP synthesis and transport by mitochondria, and that previously reported higher values resulted from errors in the determination of oxygen uptake and the use of substrates which lead to ATP synthesis by succinate thiokinase.

The P/O ratio of mitochondrial oxidative phosphorylation was first determined to be 3 by Ochoa (1), who made a correction for anaerobic ATPase activity. Since then a wide range of values for the P/O ratio have been reported using a variety of assay methods. Although most reported values are below 3 it is usually assumed that the true value is never reached because of unavoidable losses of energy. The concept of three “coupling sites” was also supported by cross-over point studies of the respiratory chain (2), and thermodynamic calculations (3) showed the feasibility of synthesis of three ATP per pair of electrons traversing the respiratory chain. In early studies of the P/O ratio it was assumed that all substrates should give integral values of one ATP per coupling site. Since the proposal of the chemiosmotic hypothesis (4, 5), however, it has been possible to accommodate fractional P/O ratios because the unit of energy coupling is the proton rather than ATP. In fact recent studies of photophosphorylation have shown that the P/2e ratio is 1.33 (6), consistent with the transport of 4 protons inwards by photoelectron transport from \( \text{H}_2\text{O} \) to NADP (7, 8) and outward movement of 3 protons in the synthesis of ATP (9).

A complication in the energetics of ATP synthesis by mitochondria has also arisen. Klingenberg and co-workers (10-12) have shown that the transport of ADP into mitochondria in exchange for ATP is coupled to the membrane potential, indicating that ATP crosses the membrane with one more negative charge than ADP. This effect, together with the entrance of Pi with a proton (or in exchange for an hydroxide ion) (13-19) is equivalent to the entrance of 1 proton and one positive charge during the transport of ADP and Pi into the mitochondrion and ATP out during oxidative phosphorylation. In terms of the chemiosmotic hypothesis this coupling of nucleotide and Pi transport should lower the P/O ratio for synthesis and transport of ATP by mitochondria below the value expected for synthesis of ATP inside. For these reasons we have re-examined the P/O ratio in rat liver mitochondria by two established methods with substrates which are oxidized by clearly defined pathways, taking care to exclude possible systematic errors.

**EXPERIMENTAL PROCEDURES**

**Materials**—ADP, NADH, NADP, phosphoenolpyruvate, hexokinase, glucose-6-phosphate dehydrogenase, pyruvate kinase, adenylate kinase, and lactate dehydrogenase were purchased from Sigma. Sucrose (special enzyme grade) was purchased from Schwarz/Mann.

**Preparation of Mitochondria**—Rat liver mitochondria were prepared in 250 mM sucrose, using gentle homogenization. The respiratory control ratios immediately after preparation were 6 with \( \beta \)-hydroxybutyrate, 9 with succinate, and 15 with glutamate plus malate as substrates.

**Measurement of Oxygen Uptake**—Oxygen uptake was measured with an oxygen electrode (Yellow Springs Instrument Co., model 4004) using a polarizing voltage of 0.65 V and a potentiometric chart recorder in a circuit similar to that described by Estabrook (20). The electrode was inserted in a glass chamber (Gibson) of 1.25 ml volume with temperature controlled by a water jacket and circulating water bath. The chamber was stirred with a 7-mm Teflon-covered bar driven at 600 rpm by a magnet. Additions were made with Hamilton syringes through a 2-mm port in a ground glass top. There is some diffusion of oxygen from the port to the chamber during the experiments which depends on the extent of oxygen uptake in the port compared to the chamber. Modification of the port by inserting a small piece of polyethylene tubing to decrease the diameter at the chamber entrance to 0.5 mm did not measurably alter the results of a typical ADP pulse experiment, so no correction was made for diffusion of oxygen into the cell during such experiments. The oxygen concentration of air-saturated 250 mM sucrose at 25°C and 760 mm Hg was 480 \( \mu \text{M oxygen} \) as measured by oxidation of known amounts of NADH in the presence of 0.5 mg/ml of submitochondrial particles (ETP, (21)) and 1 \( \mu \text{M carbonylcyanide-m-chlorophenylhydrazone} \). This value is slightly higher than a previous determination (22) and is significantly lower than the oxygen content of air-saturated water (520 \( \mu \text{M oxygen} \) (23)). Air-equilibrated 150 mM KCl contains 470 \( \mu \text{M oxygen} \) at 25°C (24). Media were equilibrated with air by shaking 30

\[^1\]Oxygen concentrations and amounts are given as molar or moles of oxygen, not \( \text{O}_2 \).
Measurement of ADP/O Ratio—The ratio of added ADP to the amount of oxygen taken up in the resulting burst of respiration is called the ADP/O ratio and is different from the P/O ratio only to the extent that ADP is incompletely phosphorylated. The ADP/O ratio was calculated by the method of Chance and Williams (25) using significant amounts of oxygen since the ADP solution is cold and contains much oxygen. The exact concentration of the ADP solutions was determined by enzymatic assay of ADP and AMP (26). Solutions contained 2 to 5% AMP, and the effective ADP concentration was taken to be [ADP] + 2x [AMP] since AMP is phosphorylated by mitochondrial adenylic kinase when Mg2+ ions are present. Measurement of the absorbance of ADP solutions at 259 nm (E259 = 15.4) confirmed the ADP + AMP concentration to within 5%. Measurement of P/O Ratio by 32P Esterification—The procedure of Schatz and Racker (27) was used, with hexokinase and glucose present. The reaction was carried out in the oxygen electrode chamber and was initiated by addition of mitochondria and stopped with trichloroacetic acid (2%) just as the oxygen concentration reached zero. Esterified 32P, was counted with a Beckman LS-100 scintillation counter using the 14C + 3H window. The sample was diluted to 10 ml with water and 1 ml of 28% NH4OH. When β-hydroxybutyrate was the substrate, formation of acetoacetate was measured as described (28).

RESULTS

β-Hydroxybutyrate and succinate (plus rotenone) are oxidized rapidly by mitochondria by simple pathways which do not involve substrate level phosphorylation of α-ketoglutarate oxidation. Measurements of P/O ratios with these substrates by the ADP pulse method are shown in Fig. 1. Addition of ADP to mitochondria oxidizing β-hydroxybutyrate increased the rate of respiration 6-fold until the ADP concentration became low. The decrease in respiration rate at the end of an ADP pulse is more abrupt (Trace A) when the phosphate concentration is above 2 mM and Mg2+ ions are present. Mg2+ ions lower the ΔGATP, and it is apparently the approach to equilibrium between the electrochemical proton gradient and phosphorylation which determines the kinetics of the transition to the controlled rate of respiration. The addition of ATP also slows the transition (29, 30). If the P/0 ratio is low and Mg2+ absent, the ADP/O ratio is the same, as shown in Trace B, but the end point extrapolation is less accurate. Calculations of the P/O ratio are often made under conditions where the end point is poorly defined, leading to possible underestimation of the amount of oxygen uptake. The ADP/O ratio measured at high P, with Mg2+ ions present is clearly 2 in Fig. 1. Trace A, where β-hydroxybutyrate was the substrate. The ADP/O ratio with succinate as substrate was 1.36 (Fig. 1, Trace C). The addition of ADP to mitochondria with no added substrate caused no stimulation of the slow rate of oxygen uptake (not shown), indicating that endogenous substrates did not contribute significantly to the results. The ADP/O ratios were found to be only slightly changed by temperature (Fig. 2a), pH (Fig. 2b), or ADP pulse size (Fig. 2c). Similar results were also obtained in a medium of 150 mM KCl in place of 250 mM sucrose (Table I). Variation of the amount of mitochondria used from 0.2 to 2 mg/ml did not change the ADP/O ratio (not shown), indicating that the oxygen electrode kinetics did not influence the result. Variation of the phosphate concentration from 0.8 to 25 mM did not change the ADP/O ratio. Addition of 0.5 mM ethylene glycol bis(β-aminoethyl ether)N,N'-tetracetic acid to chelate calcium ions had no effect on the ADP/O ratio or the controlled respiration rate.

A summary of results of ADP pulse experiments is shown in Table I. The ADP/O ratio was close to 2 with β-hydroxybutyrate, α-ketoglutarate, or glutamate plus malate as substrates, but was higher with α-ketoglutarate plus malonate to inhibit succinate oxidation (Fig. 3). The fact that respiration was inhibited by low levels of malonate which had only a small effect in raising the ADP/O ratio may be accounted for by the inhibition of α-ketoglutarate oxidation during the experiment. α-Ketoglutarate is transported into mitochondria in exchange for malonate (31). When malate formation from succinate is inhibited by malonate, succinate could leave the mitochondria in exchange for small amounts of malate which could in turn exchange for α-ketoglutarate, but this indirect process would be slower than the direct α-ketoglutarate-malate exchange in the absence of malonate. In addition the concentration of succinyl-CoA may be increased in the presence of malonate, inhibiting α-ketoglutarate dehydrogenase (32). The ADP/O ratio in the presence of malonate reached a maximum value of about 2.7, consistent with the synthesis of ATP by succinate thiokinase in addition to ATP synthesis during the oxidation of NADH (see “Discussion”). The respiratory control ratio, also shown in Table I, was reproducibly characteristic of the different substrates, and was highest with glutamate plus malate. In general it appears that substrates which are oxidized rapidly have higher respiratory control, although probably many factors contribute to the extent of control observed.

The ADP/O ratios shown are not corrected. Subtraction of the unstimulated rate of respiration from the total oxygen uptake would increase the ratio from 2 to 2.3. The background respiration is presumably proportional to the proton leak rate through the membrane which has been shown to be a nonlinear function of the electrochemical proton gradient, increasing rapidly at higher values of the gradient (33). The proton gradient is smaller during ATP synthesis than before addition of ADP, and the rate of the proton leak is less during phosphorylation than before addition of ADP. The value corrected for the proton leak is probably about half-way between the
Mitochondrial P/O Ratios

Fig. 2. Effect of temperature, pH, and ADP pulse size on the ADP/O ratio. ADP/O ratios were measured as described under “Experimental Procedures” with 8.3 mM succinate plus 0.8 μM rotenone ( ), or with 16.7 mM β-hydroxybutyrate ( ) as substrate. The medium contained 250 mM sucrose, 8.3 mM KPi, 4.2 mM MgCl₂, 0.08 mM EDTA, 0.17 mg/ml of bovine serum albumin, and 1.6 mg/ml of mitochondria, pH 7.2, 25°C. In A, the temperature was varied and the oxygen electrode calibrated at each temperature used. In B, the pH was varied and 5 mM 2-(N-morpholino)ethanesulfonate (pH 6.4 to 7) or 5 mM Tris (pH 7.5 to 8.4) was present. In C, the ADP pulse size was varied. The dashed lines are drawn at ADP/O = 2.0 and 1.33.

Table I
Summary of ADP/O ratios

<table>
<thead>
<tr>
<th>Substrate</th>
<th>No. of experiments</th>
<th>ADP/O</th>
<th>Rate of oxygen uptake (nmol O/ min/mg)</th>
<th>Respiration control ratio (nmol nmol O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. β-Hydroxybutyrate</td>
<td>26</td>
<td>2.12 ± 0.11</td>
<td>63 ± 12</td>
<td>5.6 ± 0.6</td>
</tr>
<tr>
<td>Succinate + rotenone</td>
<td>16</td>
<td>1.40 ± 0.08</td>
<td>141 ± 04</td>
<td>7.26 ± 1.5</td>
</tr>
<tr>
<td>α-Ketoglutarate</td>
<td>6</td>
<td>2.02 ± 0.07</td>
<td>67 ± 16</td>
<td>5.0 ± 1</td>
</tr>
<tr>
<td>α-Ketoglutarate + malonate</td>
<td>9</td>
<td>2.62 ± 0.1</td>
<td>34 ± 8</td>
<td>4.3 ± 0.8</td>
</tr>
<tr>
<td>Glutamate + malate</td>
<td>7</td>
<td>2.21 ± 0.09</td>
<td>135 ± 20</td>
<td>12.5 ± 1.2</td>
</tr>
<tr>
<td>B. β-Hydroxybutyrate</td>
<td>5</td>
<td>2.11 ± 0.13</td>
<td>43 ± 7</td>
<td>5.24 ± 1.2</td>
</tr>
<tr>
<td>Succinate + rotenone</td>
<td>3</td>
<td>1.39 ± 0.1</td>
<td>121 ± 7</td>
<td>5.6 ± 1.4</td>
</tr>
</tbody>
</table>

Fig. 3. Malonate titration of ADP/O ratio with α-ketoglutarate as substrate. ADP/O ratios were measured as described under “Experimental Procedures.” The medium contained 250 mM sucrose, 8.3 mM KPi, 4.2 mM MgCl₂, 0.08 mM EDTA, 0.18 mg/ml of bovine serum albumin, and 4.2 mM α-ketoglutarate. The pH was 7.2 and the temperature 25°C. The dashed line is drawn at ADP/O = 2.66.

Table II
Measurement of P/O ratio by 32P-esterification

<table>
<thead>
<tr>
<th>Substrate</th>
<th>Oxygen uptake</th>
<th>32P uptake</th>
<th>P/O</th>
<th>P/O av</th>
<th>P/AcAc</th>
</tr>
</thead>
<tbody>
<tr>
<td>β-Hydroxybutyrate</td>
<td>579</td>
<td>1140</td>
<td>1.97</td>
<td>2.04 ± 0.09</td>
<td>2.13 ± 0.03</td>
</tr>
<tr>
<td>Succinate + rotenone</td>
<td>590</td>
<td>754</td>
<td>1.28</td>
<td>1.34 ± 0.05</td>
<td>1.43 ± 0.07</td>
</tr>
<tr>
<td>Succinate</td>
<td>589</td>
<td>818</td>
<td>1.39</td>
<td>1.43 ± 0.07</td>
<td></td>
</tr>
</tbody>
</table>

Values obtained from total and stimulated oxygen uptake, or about 7% higher than measured. Another correction arises from the incomplete phosphorylation of added ADP. Respiration returns to the controlled rate when the ADP concentration is about 10 μM (29). We have repeated measurements of ADP levels immediately after respiration slows to the controlled rate and find about 20 μM following a pulse of 200 μM ADP. This represents an overestimation of the P/O ratio by 10%. The corrections for the proton leak (basal respiration), and incomplete phosphorylation of added ADP approximately cancel each other, and the measured values of ADP/O should be considered to be correct to about ±10%.

Measurement of P/O ratios by the esterification of 32P, in the presence of hexokinase and glucose to maintain a low ΔG_ATP is shown in Table II. The results were similar to those obtained by the ADP pulse method. The reaction was stopped as soon as the medium became anaerobic. If the reaction is stopped before becoming anaerobic (e.g. Ref. 34) the amount of oxygen uptake is underestimated due to the slow response of the oxygen electrode which causes the trace to lag behind the actual oxygen uptake. The P/O ratio determined with succinate in the absence of rotenone to inhibit NADH oxidation was higher than in the presence of rotenone. Determination of acetoacetate in addition to oxygen when β-hydroxy-
ybutyrate was the substrate gave a P/acetoacetate ratio similar to the P/O ratio (Table II).

**DISCUSSION**

The results indicate that the P/O ratio of rat liver mitochondria is close to 2.0 with NADH-linked substrates and 1.33 with succinate as substrate although the ADP/O ratios reported were slightly higher. The most likely explanation for these low values is the coupling of nucleotide and phosphate transport to the electrochemical proton gradient (10, 11). Other possible reactions which could account for low coupling ratios, such as Ca2+ ion transport, transhydrogenase, or proton permeability of the membrane would be expected to occur continuously rather than only following an ADP pulse and thus to lower the respiratory control ratio. Our preparations of mitochondria showed unusually high respiratory control, however, and the subtraction of the controlled rate of oxygen uptake from the total would increase the calculated P/O ratio.

The results indicate that the P/O ratio of rat liver mitochondria is close to 2.0 with NADH-linked substrates and 1.33 with succinate as substrate although the ADP/O ratios reported were slightly higher. The most likely explanation for these low values is the coupling of nucleotide and phosphate transport to the electrochemical proton gradient (10, 11). Other possible reactions which could account for low coupling ratios, such as Ca2+ ion transport, transhydrogenase, or proton permeability of the membrane would be expected to occur continuously rather than only following an ADP pulse and thus to lower the respiratory control ratio. Our preparations of mitochondria showed unusually high respiratory control, however, and the subtraction of the controlled rate of oxygen uptake from the total would increase the calculated P/O ratio only slightly. In fact consideration of the systematic errors in the ADP pulse method showed a small underestimation from the basal respiration proton leak and a small overestimation of the P/O ratio by the incomplete phosphorylation of ADP. These two errors approximately cancel out, and it seems unlikely that the true P/O ratio with β-hydroxybutyrate as substrate could differ from two by more than 10%.

A survey of some previous reports of the P/O ratios in mitochondria is shown in Table III. The first three reports were obtained with crude homogenates and are included for historical interest. The next series of six reports were studies of mitochondria using a Warburg manometer to measure oxygen uptake, and represent typical data on which the currently accepted value of 3.0 is based. Slater (48) has discussed the potential errors in measurements of oxygen uptake with a Warburg manometer, and concluded that “It is unfortunate that errors in measuring P/O ratios usually lead to high values.” Most reports also showed considerable variation and it was commonly assumed that the higher values were more correct and that the true value was even higher. The average value, however, was often similar to our results. Slater and Holton (41) reported a P/O ratio of 2.7 with α-ketoglutarate plus malonate as substrate, and criticized the practice of emphasizing a few high values to support the expected ratio of 4 with this substrate. The reports of P/O ratios (Table III) close to 0.7 with ascorbate-TMPD (44) or reduced cytochrome c (43) as substrate are consistent with our results.

Six studies of P/O ratios by the ADP pulse method are shown in Table III. They range from 1.7 with β-hydroxybutyrate (29) to 3.2 with glutamate plus malate (30). The latter value was obtained with ischemic dog heart mitochondria and may represent an unusual metabolic pattern. Some possible errors which can lead to an overestimation of the P/O measured by this method are diffusion of oxygen into an open cell or from a plastic cell into the aqueous solution, addition of oxygen with the ADP solution, incomplete phosphorylation of ADP, and use of conditions which lead to a slow transition to the controlled state as the ADP is expended.

The P/O ratios of 2 with β-hydroxybutyrate and 1.33 with succinate are consistent with the chemiosmotic scheme for oxidative phosphorylation shown in Fig. 4. The stoichiometry of proton transport by the respiratory chain was originally found to be 6H+/O with β-hydroxybutyrate and 4H+/O with succinate as substrate (49). These values were also found with inverted submitochondrial particles (50) and are consistent with H/O ratios determined with reconstituted segments of the chain (51, 52). Higher values have been reported using mitochondria in the presence of N-ethylmaleimide (53, 54), at low temperature (55), or in phosphate-depleted mitochondria (55) to prevent phosphate transport. Phosphate transport could not have interfered with the determinations in reconstituted segments of the chain, however, and this problem was

<table>
<thead>
<tr>
<th>Table III</th>
<th>Survey of some reported P/O ratios in mitochondria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Authors</td>
<td>Method‡</td>
</tr>
<tr>
<td>Kalckar, 1937 (35)</td>
<td>A</td>
</tr>
<tr>
<td>Beltzer and Taisakowa, 1930 (36)</td>
<td>A</td>
</tr>
<tr>
<td>Ochoa, 1943 (1)</td>
<td>A</td>
</tr>
<tr>
<td>Cross et al., 1949 (37)</td>
<td>A</td>
</tr>
<tr>
<td>Lehninger and Smith, 1949 (38)</td>
<td>A</td>
</tr>
<tr>
<td>Hunter, 1951 (39)</td>
<td>A</td>
</tr>
<tr>
<td>Coopenhaver and Lardy, 1952 (40)</td>
<td>A</td>
</tr>
<tr>
<td>Slater and Holton, 1954 (41)</td>
<td>A</td>
</tr>
<tr>
<td>Greenberg et al., 1959 (42)</td>
<td>A</td>
</tr>
<tr>
<td>Lehninger, 1955 (43)</td>
<td>D</td>
</tr>
<tr>
<td>Howland, 1963 (44)</td>
<td>A</td>
</tr>
<tr>
<td>Lenaz and Beyer, 1965 (45)</td>
<td>B</td>
</tr>
<tr>
<td>Lee et al., 1967 (34)</td>
<td>B</td>
</tr>
<tr>
<td>Chance, 1965 (46)</td>
<td>C</td>
</tr>
<tr>
<td>Estabrook, 1967 (20)</td>
<td>C</td>
</tr>
<tr>
<td>Slater et al., 1973 (29)</td>
<td>C</td>
</tr>
<tr>
<td>Wilson et al., 1978 (30)</td>
<td>C</td>
</tr>
<tr>
<td>Davis et al., 1974 (47)</td>
<td>C</td>
</tr>
</tbody>
</table>

*Methods: A, Warburg manometer; B, oxygen electrode, phosphorylation measured by 32P esterification; C, oxygen electrode, ADP pulse; D, spectrophotometric.
‡No inhibitors present unless stated.
§Average after correction for anaerobic ATPase activity.
*Average.

The abbreviation used is: TMPD, N,N′,N″,N‴-tetramethyl-p-phenylenediamine.
remains to be resolved (56). The higher H⁺/O ratios of 9 or 12 with NADH-linked substrates would allow the use of a proton in nucleotide and Pi transport while maintaining the currently accepted P/O ratio of 3 (57, 58). The P/O ratio of 2, which we have determined, is accounted for by an H⁺/O of 6 with NADH-linked substrates, as shown in Fig. 4.

The stoichiometry of proton transport by the F₁, Fₒ ATPase complex has been determined to be 2H⁺/ATP in inverted submitochondrial particles (59). Studies of the magnitude of the electrochemical proton gradient formed by mixtures of ATP, ADP, and Pᵢ in submitochondrial particles also indicate that ΔG₅ₐ₅ = twice the electrochemical proton gradient (Δμ₁₉), consistent with a coupling ratio of 2H⁺/ATP (60, 61). Studies of the H⁺/ATP ratio for the F₁, Fₒ complex in mitochondria are complicated by the requirement for ATP transport, but have also indicated a ratio of 2 (57, 62). In mitochondria where an extra proton is coupled to the uptake of Pᵢ and ADP the overall H⁺/ATP ratio for synthesis of external ATP should be 3, as shown in Fig. 4. This proton stoichiometry is consistent with the P/O values reported here and with studies of the energetics of oxidative phosphorylation. The value of ΔG₅ₐ₅ formed in the external medium is about 3 times Δμ₁₉, and the value of ΔG₅ₐ₅ inside mitochondria is about 2 times the value of Δμ₁₉ (10, 11, 29, 33, 63-65).

The scheme shown in Fig. 4 is consistent with a P/O ratio with succinate as substrate of 1.3 because 4 protons are driven outwards during succinate oxidation and one ATP is made and transported out for every 3 protons entering. α-Ketoglutarate in the presence of malonate would lead to synthesis of one internal ATP by substrate level phosphorylation, which would use 1 proton when it is transported out, leaving 5 protons from NADH oxidation. These protons would make 1.66 external ATP by oxidative phosphorylation giving a total yield of 2.66 ATP per oxygen atom reduced (compare with Fig. 3). In the absence of malonate the overall P/O ratio of α-ketoglutarate oxidation would be the average of 2.66 and 1.33 or 2.0.

Regardless of the proton stoichiometries, however, we wish to emphasize that the measured P/O ratio in intact rat liver mitochondria is near 2 during β-hydroxybutyrate oxidation and 1.3 during succinate oxidation.

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P C Hinkle and M L Yu


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