Redox Interactions between Catalase and Alcohol Dehydrogenase Pathways of Ethanol Metabolism in the Perfused Rat Liver*

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Alcohol metabolism via alcohol dehydrogenase (ADH) and catalase was studied in perfused rat livers by measuring the oxidation of methanol and butanol, selective substrates for catalase and ADH, respectively. In livers from fasted rats, basal rates of methanol uptake of 15 ± 1 μmol/g/h were decreased significantly to 8 ± 2 μmol/g/h by addition of butanol. Concomitantly, pyridine nucleotide fluorescence detected from the liver surface was increased by butanol but not methanol. Both effects of butanol were blocked by and an inhibitor of ADH, 4-methylpyrazole, consistent with the hypothesis that elevation of the NADH redox state by butanol inhibited H₂O₂ production via NAD⁺-requiring peroxisomal β-oxidation, leading indirectly to diminished rates of catalase-dependent methanol uptake. In support of this idea, both butanol and butyraldehyde inhibited H₂O₂ generation. The NADII redox state was also elevated by xylitol, causing a 75% decrease in rates of methanol uptake by livers from fasted rats. This effect was not observed in livers from fed rats unless malate-aspartate shuttle activity was restored (9). However, both xylitol and butanol increased malate fluorescence detected from the liver surface, suggesting that an increase in cytosol NADH-NAD⁺ ratio increased malate aspartate shuttle activity, leading indirectly to decreased rates of catalase-dependent methanol uptake.

It is generally accepted that alcohol dehydrogenase (ADH) is the predominant pathway for hepatic ethanol oxidation. This idea is based on the observation that ethanol metabolism was sensitive both in vitro and in vivo to inhibitors of ADH such as pyrazole and alkylpyrazoles (1, 2). However, alkylpyrazoles are also potent inhibitors of cytochrome P-450 (3), and the pyrazole metabolite 4-hydroxypyrazole inhibits catalase (4); thus, interpretation of results from studies on pathways of ethanol metabolism which employ such inhibitors is problematic.

Another common notion held until recently was that catalase plays only a minor role in ethanol metabolism (5). This idea was based on early reports that rates of H₂O₂ generation in perfused rat livers were more than an order of magnitude lower than rates of ethanol oxidation (6), and that ethanol elimination in vivo was not affected by treatment with the catalase inhibitor aminotriazole (7, 8). Three recent experiments (9-11) demonstrated that there are problems with these arguments. First, in perfused livers from fasted rats, rates of H₂O₂ production were increased significantly from 10 to about 80 μmol/g/h by the addition of fatty acid-albumin complexes (palmitate and oleate) which are substrates for H₂O₂ generation via the peroxisomal β-oxidation system (9). Thus, with the addition of appropriate physiological substrates, rates of H₂O₂ generation in the liver can be quite high. Albumin, which is critical for fatty acid uptake by liver cells, was either omitted or present in unphysiologically low concentrations in older work, accounting for low rates of H₂O₂ generation observed (9). Second, in carefully controlled studies in dermice lacking ADH, catalase was inhibited by treatment with aminotriazole and rates of ethanol elimination in vivo were decreased by about 75% (10). Thus, catalase can be a significant pathway of ethanol metabolism in vivo. Third, it was demonstrated recently in perfused livers from fasted rats that methanol uptake could be stimulated 4-fold by fatty acids (11). Taken together, these three results indicate that conditions exist under which catalase-H₂O₂ is a major pathway of alcohol metabolism.

Alcohol dehydrogenase will oxidize ethanol, propanol, and butanol at relatively higher velocities as the chain length is increased, but will not metabolize methanol (12). Conversely, catalase will metabolize methanol but not butanol (13). Thus, methanol and butanol are selective substrates for catalase and ADH, respectively. In a study which employed selective substrates, it was demonstrated that catalase-H₂O₂ was the predominant pathway of total alcohol metabolism in the fasted state in the presence of the physiological fatty acid oleate (11). While peroxidation of methanol by catalase-H₂O₂ was stimulated by oleate due to increased H₂O₂ generation from peroxisomal β-oxidation of fatty acyl-CoA compounds, rates of butanol uptake actually decreased in the presence of the fatty acids (11). Both alcohol oxidation via ADH and H₂O₂ generation via peroxisomal β-oxidation are NAD⁺-requiring reactions. Accordingly, the purpose of the present study was to investigate whether or not interactions between the ADH and catalase pathways of alcohol oxidation occur. The data indicate that increases in the NADH/NAD⁺ ratio caused by alcohol oxidation via ADH can inhibit peroxidation via catalase-H₂O₂ by decreasing rates of H₂O₂ generation.

MATERIALS AND METHODS

Liver Perfusion—Livers from fed or fasted (24 h) Sprague-Dawley rats were perfused in recirculating (65 ml volume) or nonrecirculating

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1 The abbreviation used is: ADH, alcohol dehydrogenase.
perfusion systems as described previously (14, 15). The concentration of oxygen in the effluent perfusate was monitored continuously with a Teflon-shielded, Clark-type O2 electrode.

Concentrations of butanol in the perfusate were determined enzymatically (10). Methanol in perfusate was determined either enzymatically (9) or by gas chromatography. Samples (1.0 ml) of effluent perfusate were placed in capped 25-ml Erlenmeyer flasks and incubated at 37 °C for 30 min. One ml of the headspace was sampled with a gas-tight syringe and injected onto a Carbobox 60/80 column. Operating conditions were: oven, 110 °C; detector, 250 °C; and carrier gas flow, 40 ml/min. Methanol was detected with a flame ionization detector and was quantitated with external methanol standards in a manner similar to the quantitation of ethanol described in detail elsewhere (17). Rates were calculated from the influent minus effluent concentration differences, the flow rate, and the liver wet weight.

Determination of Pyridine Nucleotide Fluorescence and the Steady-state Level of Catalase—The steady-state level of catalase-H2O2 was detected spectrophotometrically (660-640 nm) through a lobe of the liver as described by Sies and Chance (18) with a dual-channel, air-driven spectrophotometer (19). Pyridine nucleotide fluorescence from the surface of perfused livers was determined with a large tipped, bifurcated, fiber-optic light guide. One end of the light guide was connected to a 60-watt mercury arc lamp fitted with a 366-nm transmittance filter, and the other end was connected to a photomultiplier filtered to detect 450 nm light. The output from the photomultiplier was amplified and recorded as described elsewhere (18).

Oxidation of Methanol and Butanol by Cytosol and ADH in Vitro—Livers from fed rats were perfused for 1-2 min with Krebs-Henseleit buffer at 37 °C and washed twice with 0.15 M KCl. Supernatants were centrifuged at 100,000 x g for 60 min, and the high-speed supernatant containing ADH (cytosol) was collected.

The oxidation of butanol by supernatants containing ADH was measured by following the reduction of NAD+ to NADH at 386 nm (ε = 3.3 x 10^3 M^-1 cm^-1). Reaction mixtures containing 5 mg of cytosolic protein were incubated at 25 °C in 1-ml volume containing 500 mM phosphate buffer, pH 7.0, 1 mM NAD+, and 1 mM hydrazine hydrate, pH 9.0. Reactions were initiated by the addition of butanol (0-15 mM), and the reduction of NAD+ was measured spectrophotometrically and recorded for 3-5 min. Methanol (final concentration, 30 mM) was added subsequently, and reactions were incubated for 3 additional minutes.

The oxidation of butanol by catalase in mitochondrial/peroxisomal fractions was determined by measuring the formation of formaldehyde colorimetrically. Five mg of protein was incubated in 1 ml of 0.1 M phosphate buffer, pH 7.0, containing 100 mM semicarbazide and 10 mM glucose. Reactions were initiated by the addition of 10 units of glucose oxidase and were terminated by the addition of 100 µl of 6N HCl and 100 µl of glacial acid. Formaldehyde was measured colorimetrically using the method of Nash (20).

Statistics—Comparison between groups were made using Student’s t test (21).

RESULTS

Validation of the Selectivity of Methanol and Butanol for Catalase and ADH—To verify that methanol was a selective substrate for catalase, and that butanol was selective for ADH in rat liver, the oxidation of methanol and butanol was measured in subcellular fractions (Fig. 1). Rates of formation of formaldehyde from the peroxidation of methanol by 10,000 x g pellets incubated with an H2O2-generating system increased as the concentration of methanol was elevated up to 100 mM (Fig. 1A). Rates were half-maximal with about 35 mM methanol. Butanol (15 mM), which was not metabolized by the 10,000 x g pellete, had no effect on peroxidation of methanol (Fig. 1A).

In the presence of NAD+ and cytosol of rat liver homogenates, rates of NADH generation from the oxidation of butanol by ADH increased as the concentration of butanol was elevated (Fig. 1B). Maximal rates of NADH formation of about 4.5 nmol/min/mg protein were observed with about 5 mM butanol; rates were half-maximal with concentrations of butanol around 2 mM (Fig. 1B). As expected, the oxidation of butanol by cytosol was not affected by methanol (30 mM, Fig. 1B), and NADH was not formed by methanol alone (data not shown).

Methanol and butanol are both metabolized by isolated microsomes (22). In perfused rat liver, however, basal rates of methanol uptake were not affected by an inhibitor of mixed-function oxidation, 4-methylpyrazole (see below, Fig. 3). p-Nitrophenol is a good substrate for the isozyme of cytochrome P-450 (II.E.1) which is induced by ethanol and has a high affinity for butanol (23). It was oxidized at rates about 2 orders of magnitude lower than butanol (9), indicating that cytochrome P-450 plays only a minor role in oxidation of methanol and butanol in perfused liver. Taken together, these data verify that methanol and butanol are selective substrates for catalase and ADH, respectively.

Oxidation of Methanol and Butanol by Perfused Rat Liver—The results of a typical experiment employing the experimental design used in these studies is shown in Fig. 2. In a perfused liver from a fasted rat methanol (30 mM) did not affect basal rates of oxygen uptake (about 100 µmol/g/h) or steady-state levels of NADH fluorescence; however, the steady-state level of catalase-H2O2 measured spectrophotometrically (660-640 nm) was decreased sharply. The addition of butanol (30 mM) in the presence of methanol elevated oxygen uptake by about 10 µmol/g/h, increased absorbance (660-640 nm), and elevated pyridine nucleotide fluorescence to 115% of basal levels. The increase in absorbance (660-640 nm) due to butanol also occurred following inactivation of catalase by aminotriazole treatment, indicating that these changes in the 660-640 nm signal were not due to changes in the steady-state level of catalase-H2O2 under these conditions. The subsequent addition of oleate (1 mM) increased oxygen uptake significantly to about 150 µmol/g/h and elevated NADH fluorescence to 120% of basal levels, reflecting an increase in the intracellular
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NADH/NAD⁺ ratio (14). Absorbance at 660–640 nm was not affected by oleate (Fig. 2).

In order to assess the effect of nutritional state on rates of alcohol oxidation by catalase and ADH, livers from fed rats were perfused as in Fig. 2. Basal rates of uptake of methanol and butanol in the presence of both alcohols in livers from fed rats were 5 ± 1 and 95 ± 2 µmol/g/h, respectively (n = 4).

The addition of oleate (1 mM) increased rates of methanol uptake significantly to 9 ± 1 µmol/g/h (p < 0.05) but diminished rates of butanol uptake to about 70 µmol/g/h. Thus, it is concluded that ADH is the predominant pathway of alcohol oxidation in the fed state, as expected.

In the present study, rates of methanol uptake, which reflect rates of H₂O₂ generation, were 3–4 times greater in the fasted than in the fed state in the presence of oleate (Table I). Similar results were obtained in mice in vivo by van den Branden et al. (24). There are a number of possible explanations for this phenomenon, including uptake, activation, and peroxisomal metabolism of fatty acids. At present, however, it is not clear which of these factors is responsible for the effect of nutritional state on rates of peroxisomal H₂O₂ generation.

To quantitate alcohol oxidation by catalase and ADH in the absence of inhibitors, rates of uptake of methanol and butanol were measured simultaneously in perfused liver. Basal rates of methanol and butanol uptake in the presence of both alcohols were 8 ± 1 and 77 ± 15 µmol/g/h, respectively (Table I); total rates of alcohol oxidation were around 85 µmol/g/h. The subsequent addition of oleate (1 mM) diminished overall rates of alcohol uptake to about 65 µmol/g/h (Table I). Rates of methanol uptake increased significantly to 38 ± 4 µmol/g/h, while butanol uptake was diminished to about 26 µmol/g/h. When fructose (10 mM) was infused, overall rates of alcohol uptake were increased about 20%. Under these conditions, rates of methanol uptake were diminished to 5 ± 2 µmol/g/h while rates of butanol uptake were increased significantly to 101 ± 4 µmol/g/h (Table I). Fructose decreased rates of H₂O₂ generation measured spectrophotometrically in livers from fasted rats from 9 ± 2 to 4 ± 1 µmol/g/h (n = 4), consistent with observations in perfused livers from deermice genetically deficient in ADH (25). Peroxidation of alcohols is limited by the supply of H₂O₂ in rat liver (26); thus the data are consistent with the hypothesis that methanol uptake was mediated via catalase. In support of this hypothesis, methanol uptake was decreased below the limits of detection (<2 µmol/g/h) in livers from rats pretreated for 1.5 h with the catalase inhibitor aminotriazole (1.5 g/kg, intraperitoneally; data not shown), confirming that methanol uptake was mediated via catalase in intact cells.

**Redox Inhibition of Methanol Oxidation by Butanol**—Rates of methanol uptake of 15 ± 2 µmol/g/h in perfused livers from fasted rats (Fig. 3, left) were diminished to about 8 µmol/g/h by butanol (p < 0.05; Fig. 3, center). Under these conditions, rates of butanol uptake were about 75 µmol/g/h. When
an inhibitor of ADH, 4-methylpyrazole (4 mM), was added subsequently, rates of methanol uptake returned to basal values (Fig. 3, right). Concomitantly, rates of butanol uptake were below the limits of detection and pyridine nucleotide fluorescence, which was increased initially by about 20% when butanol was added (Fig. 2), decreased to basal levels (data not shown).

Since butanol did not affect the peroxidation of methanol by catalase from rat liver in vitro (Fig. 1), and because the supply of \( H_2O_2 \) is rate-limiting for the peroxidation of methanol and ethanol by catalase (26), one possible explanation for the observed inhibition of methanol uptake is that butanol oxidation decreased \( H_2O_2 \) generation. To test this hypothesis, rates of \( H_2O_2 \) generation were measured in livers from fasted rats perfused in a nonrecirculating system in the presence and absence of butanol and butyraldehyde plus 4-methylpyrazole. Butanol and butyraldehyde plus 4-methylpyrazole both increased pyridine nucleotide fluorescence by about 20% and diminished rates of \( H_2O_2 \) generation from about 8 \( \mu \)mol/g/h to between 2 and 3 \( \mu \)mol/g/h (Table II).

**Effect of Xylose on Methanol Uptake and Pyridine Nucleotide Fluorescence**—The fact that the inhibition of \( H_2O_2 \) generation observed with butanol and butyraldehyde, which are metabolized via NAD\(^+\)-linked dehydrogenases, was associated with an increase in pyridine nucleotide fluorescence (Table II) suggested that \( H_2O_2 \) generation could be inhibited by increasing the \( \text{NADH}/\text{NAD}^+ \) ratio. To test this hypothesis, livers from fasted rats were perfused in a nonrecirculating system and methanol (2 mM) was infused in the presence and absence of xylose (5 mM), a substrate for the cytosolic NAD\(^+\)-linked xylose dehydrogenase. Upon infusion of methanol, basal rates of \( O_2 \) uptake of 100-110 \( \mu \)mol/g/h were unaffected (Fig. 4, upper panel), while pyridine nucleotide fluorescence increased slightly (110% of basal), due most likely to dehydrogenase-dependent metabolism of formaldehyde (Fig. 4, center panel). Under these conditions, rates of methanol uptake were about 8 \( \mu \)mol/g/h (Fig. 4, bottom). Upon infusion of xylose, pyridine nucleotide fluorescence increased about 35% and rates of \( O_2 \) uptake increased to 130 \( \mu \)mol/g/h. Concomitantly, rates of methanol uptake declined to values around 3 \( \mu \)mol/g/h (Fig. 4). When xylose infusion was terminated, all parameters tended to return to basal values.

In livers from fed rats, methanol infusion increased pyridine nucleotide fluorescence to about 110% of basal values without affecting oxygen consumption. Basal rates of methanol uptake were about 3 \( \mu \)mol/g/h (Fig. 5). Infusion of xylose (6 mM) increased rates of \( O_2 \) uptake from about 95 to 115 \( \mu \)mol/g/h (Fig. 5) and pyridine nucleotide fluorescence to about 130% of basal values, but did not alter rates of methanol uptake.

**TABLE II**

Inhibition of \( H_2O_2 \) generation and elevation of pyridine nucleotide fluorescence by butanol and butyraldehyde in perfused rat livers from fasted rats.

<table>
<thead>
<tr>
<th>Addition</th>
<th>( H_2O_2 ) generation ( \mu )mol/g/h</th>
<th>Pyridine nucleotide fluorescence % of basal</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>7.1 ± 0.8</td>
<td>100</td>
</tr>
<tr>
<td>Butanol</td>
<td>2.2 ± 0.3( ^* )</td>
<td>119 ± 5( ^* )</td>
</tr>
<tr>
<td>Butyraldehyde</td>
<td>1.7 ± 0.6( ^* )</td>
<td>122 ± 6( ^* )</td>
</tr>
<tr>
<td>+ 4-methylpyrazole</td>
<td></td>
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\( ^* p < 0.01 \) as compared to no addition control values.

**FIG. 4.** Effect of methanol and xylose on oxygen and methanol uptake and NADH fluorescence in a perfused liver from a fasted rat. A liver from a fasted rat was perfused in a nonrecirculating system and oxygen uptake and surface fluorescence of NADH were measured as described under "Materials and Methods." Methanol was measured enzymatically in samples of perfusate collected every 2 min and rates of methanol uptake were calculated from influent minus effluent concentration differences, the flow rate, and the liver wet weight. Methanol (2 mM) and xylose (5 mM) were infused as denoted by the horizontal bars and vertical arrows. Typical experiment.

**FIG. 5.** Effect of methanol, xylose, and aminooxyacetate on oxygen and methanol uptake and NADH fluorescence in a perfused liver from a fed rat. A liver from a fed rat was perfused in a nonrecirculating system and oxygen uptake and surface fluorescence of NADH were measured as described in Fig. 4. Methanol (2 mM), xylose (5 mM), and aminooxyacetate (0.2 mM) were infused as denoted by the horizontal bars and vertical arrows. Typical experiment.
basal values, and methanol uptake decreased to about 1 μmol/g/h (Fig. 6).

**DISCUSSION**

**Catalase Is a Major Pathway of Ethanol Metabolism in the Fasted State**—As noted in the Introduction, the lack of specificity of inhibitors of pathways of ethanol metabolism makes interpretation of results difficult. This problem was circumvented by employing selective substrates for the major enzymes involved in ethanol oxidation. In the present study, methanol and butanol were verified as selective substrates for rodent catalase and ADH, respectively.

Rates of butanol uptake by livers from fed rats were about an order of magnitude greater than rates of methanol uptake. These data support the well accepted notion that ADH is the predominant pathway of alcohol metabolism in the fed state (27). In the fasted state, increasing concentrations of an albumin-oleate complex, a physiological substrate for peroxisomal β-oxidation, elevated rates of H₂O₂ generation by 400% (9). In the present study rates of methanol oxidation via catalase-H₂O₂ accounted for about 60% of total alcohol oxidation in the presence of fatty acids. Thus, in the fasted state in the presence of oleate, catalase-H₂O₂ is the predominant pathway of alcohol oxidation (11). This conclusion is supported by experiments with fructose, which decreases ATP (28) and thus would be expected to diminish activation of fatty acids and limit substrate supply for peroxisomal β-oxidation. Fructose decreased basal rates of methanol uptake by nearly 50% (Table I).

When carbohydrate is consumed, ADH is the predominant pathway of alcohol metabolism. The results of the present study indicate that in the fasted state where concentrations of fatty acids in the circulation and liver are elevated, rates of alcohol oxidation are decreased and oxidation is mediated predominantly via catalase. High concentrations of fatty acids, which are found in the fasted state in vivo, are necessary for maximal rates of catalase-dependent alcohol oxidation (9). Thus, in vivo one would predict a pattern of increasing and decreasing rates of alcohol metabolism resulting from changes in the contributions of catalase and ADH. Rhythmic increases and decreases in rates of ethanol metabolism have been observed by Sturtevant and Garber (29); nutritionally mediated alterations in the contributions of ADH and catalase-H₂O₂ may explain why these rhythms occur.

**Redox Interactions between ADH and Catalase**—The β-oxidation of fatty acyl-CoA compounds in peroxisomes proceeds via an acyl CoA oxidase which consumes O₂ and produces H₂O₂ and a dehydrogenase which requires NAD⁺ (Fig. 6). In fact, cyanide-insensitive reduction of NAD⁺ in the presence of fatty acyl-CoA compounds is used routinely to measure peroxisomal β-oxidation in vitro (30). In the perfused liver, increases in the NADH/NAD⁺ ratio caused by the metabolism of butanol, butyraldehyde, or xylitol inhibited H₂O₂ generation (Figs. 3–5, Table II). This inhibition of methanol uptake by butanol was reversed when oxidation of butanol via ADH was inhibited (Fig. 3). Thus, it is concluded that H₂O₂ generation via peroxisomal β-oxidation and methanol uptake were inhibited by elevation of the NADH/NAD⁺ ratio by these compounds, presumably at the NAD⁺-requiring β-hydroxyacyl-CoA dehydrogenase (Fig. 6). Based on these data, it is also concluded that interactions between the ADH and catalase pathways occur and are mediated via elevation of the pyridine nucleotide redox state.

Although it is known that peroxisomes are permeable to NAD⁺ (31), the mechanism(s) by which reducing equivalents generated via peroxisomal β-oxidation move into and out of peroxisomes is not clear. Redox interactions between catalase and ADH suggest that in intact cells the pyridine nucleotide redox state within the peroxisome and the cytosol are in equilibrium. It is generally accepted that reducing equivalents generated in the cytosol must be moved into the mitochondria to be combusted by the mitochondrial electron transport system.
chain. A major route of transport of reducing equivalents into and out of mitochondria is the malate-aspartate shuttle (Fig. 6). Reducing equivalents generated by the oxidation of both butanol and xylitol are transferred predominantly via this route (32). Since butanol and xylitol both inhibited H2O2 generation in the fasted state resulting in lower rates of methanol uptake, it is possible that reducing equivalents formed from the oxidation of butanol or xylitol compete with reducing equivalents generated via peroxisomal β-oxidation of fatty acyl-CoA compounds for transport into the mitochondria via the malate-aspartate shuttle. In support of this hypothesis, xylitol alone did not affect rates of methanol uptake in livers from fed rats (Table II), where the capacity of the malate-aspartate shuttle is greater in part due to increased levels of required intermediates involved in the transfer of reducing equivalents (i.e. malate, α-ketoglutarate; 33). On the other hand, when the transaminase inhibitor aminooxyacetate was infused to inhibit shuttle activity (Fig. 6), rates of methanol uptake decreased in the fed state (Fig. 5). These data are consistent with the possibility that transaminases are involved directly in the movement of reducing equivalents over the peroxisomal membrane; however, evidence in support of this hypothesis is lacking. The data do indicate clearly that the malate-aspartate shuttle is involved in the reoxidation of reducing equivalents generated in peroxisomes. This conclusion is further supported by the observation that butyraldehyde, which generates reducing equivalents in the mitochondria, also diminished rates of H2O2 generation (Table II).

Taken together, the data indicate that significant interactions exist between the ADH and catalase pathways of ethanol metabolism. The redox effects of ethanol metabolism via ADH on peroxidation via catalase may represent an important physiological form of regulation of ethanol metabolism in intact cells. Moreover, these results suggest that treatments which alter ADH activity or the ability of the cell to reoxidize NADH will significantly alter peroxidation of ethanol via catalase.

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