Determination of Oxalate Concentration in Blood
Jeremiah Costello and Douglas M. Landwehr

The methods used for determination of oxalate in blood are reviewed, and the advantages and disadvantages of the two basic approaches—direct methods and in vivo isotope-dilution techniques—are compared. Possible reasons for the previous discrepancies between direct and isotopic methods are discussed, as are the effects of protein binding, sample handling, and storage conditions on oxalate values in plasma. Necessary precautions for obtaining reproducible results are presented. We recommend and critically review several direct methods, and describe the application of a direct method for oxalate determination in some other biological fluids.

Additional Keyphrases: hemodialysis • conversion to oxalate, in vitro and in vivo • analytical methods compared • sex-related differences • protein binding

Knowledge of oxalate concentration in blood and other body fluids can be very important in certain clinical situations, such as primary hyperoxaluria (1, 2), chronic renal failure (1-5), calcium oxalate nephrolithiasis (6, 7), and poisoning with ethylene glycol (unpublished data). However, analysis of oxalate is difficult, and there is much controversy and uncertainty concerning the validity of its measurement in biological fluids. More than 30 different methods have been published, and proposed values for normal concentrations of oxalate in plasma range from less than 1 μmol/L to more than 400 μmol/L. Along with the absence of a recognized reference standard, this variability in the results and methodologies of previous studies makes it difficult to interpret much of the literature dealing with oxalate metabolism.

Our purpose here is to review the methods used to measure oxalate in biological samples. We will attempt to provide some insight into potential drawbacks of various methods and comment on the correctness of previous measurements of oxalate content in blood. We hope this will help in interpreting the literature and also will guide investigators to choose the most appropriate methodology for future studies.

Methods

Many methods have been used for measuring oxalate in blood and body fluids, both direct and indirect methods involving isotope-dilution studies. Table 1 lists the direct methods reported in the literature. One of the earliest methods, reported in 1981 by Merz and Mauger (8), yielded values for normal plasma oxalate of 222-444 μmol/L. Except for the reports of Sommoodar et al. (29) and Borland et al. (33), subsequent methods have yielded progressively lower values for blood oxalate concentration, which leads us to conclude that the earlier estimates of blood oxalate were erroneously high.

Direct techniques have involved enzymic, chromatographic, and fluorimetric methods. The most recent methods have been based on an enzymatic technique, probably because of its comparative ease of application and specificity. Either oxalate oxidase (EC 1.2.3.4) (28, 32) or oxalate decarboxylase (EC 4.1.1.2) (24, 27) has been used, and some methods include the use of a second enzyme, formate dehydrogenase (EC 1.2.1.2) linked to oxalate decarboxylase (25, 31). Reaction products are measured by spectrophotometric (25), colorimetric (32, 33), or potentiometric methods (28, 31). Although gas-chromatographic methods offer the advantage of smaller sample size, they require specialized equipment and prolonged column preparation (26, 30).

Table 2 lists studies in which isotope-dilution techniques were used for determining blood oxalate concentration (6, 7, 34-39). These undoubtedly have been developed because of the great variability of results obtained by the direct methods. The procedure involves either a single intravenous bolus injection or a constant infusion of [14C]oxalate for up to 6 h. Prenen et al. (39) collected blood samples at 30-min intervals and urine samples at 60-min intervals by spontaneous voiding. In the case of urine, both radioactivity and oxalate concentrations are determined; in plasma, only radioactivity is measured. It is assumed that in both fluids the specific activity of oxalate is the same, and plasma oxalate concentration is calculated by means of the formula (39):

\[
\text{Plasma oxalate (μmol/L)} = \frac{\text{urinary oxalate excretion (μmol/min)}}{\text{[14C]oxalate clearance (mL/min)}} \times 1000
\]

Values obtained with isotopic methods have shown little variability when used by different investigators, and have yielded uniformly low normal values for plasma oxalate. However, the use of radioactive isotopes limits their general applicability in human subjects, and the total volume of blood necessary for the procedure may exceed 100 mL (39). These methods also require direct measurement of urinary oxalate, and errors in this measurement will yield erroneous results for blood oxalate. Studies involving a bolus injection of isotope may be done relatively rapidly (6), but the lack of constant specific activity in the blood results in some degree of error, even if several hours are allowed for isotope distribution (39). On the other hand, a prolonged constant infusion of the isotope is required for constant specific activity to be achieved, a procedure that requires more than 6 h to complete (39).

Although direct methods appear to result in systematically higher values for blood oxalate than do isotope-dilution methods, a comparison of Table 1 and 2 shows relatively close agreement between values derived from isotope-dilution methods and all but two of the direct methods (29, 33) published since 1980. This agreement lends confidence to the validity of most of the recent estimates of oxalate concentrations in blood.

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Allegheny-Singer Research Institute and Allegheny General Hospital, 330 East North Avenue, Pittsburgh, PA 15212-3886.
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Table 1. Reported Normal Concentrations of Oxalate in Human Blood

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<td>1.25</td>
<td>0.47</td>
<td>23</td>
<td>Double enzymatic–spectrophotometric</td>
</tr>
</tbody>
</table>

*Plasma. †Serum. ‡Whole blood.

Variations in Oxalate Concentration Measured in Whole Blood, Plasma, and Serum

In comparing different methods for measurement of blood oxalate, one must also realize that, although oxalate concentrations in serum and plasma are similar (21, 25), its concentration in erythrocytes may exceed that in plasma (13, 15). Thus, we would expect values for whole blood to exceed those for plasma or serum. However, further studies with more recent methods are needed to confirm this expectation.

Sex-Related Difference

It has also been suggested that blood oxalate concentration may be different in males and females. Hatch et al. (19) reported higher concentrations of serum oxalate in women, but their method yielded overall concentrations considerably higher than recent studies, so their results must be viewed with caution. However, Kasidas and Rose (32), using a method that resulted in relatively low values for plasma, mean 2.03 μmol/L, also found that mean plasma oxalate is higher in women, 2.25 μmol/L, than in men, 1.87 μmol/L, (P<0.05). A study of 12 women and 11 men in our laboratory yielded similarly higher (P>0.10) concentrations of oxalate in plasma from men: 1.41 (SD 0.30) μmol/L vs 1.11 (SD 0.56) μmol/L for women. Studies of larger populations will be required to establish conclusively whether oxalate concentrations in plasma differ between the sexes.

In Vitro Oxalogenesis

Some workers have suggested that in vitro conversion of precursors to oxalate may explain the discrepancy between earlier direct measurements of oxalate, in which oxalate values for plasma were reported as approximately 17 μmol/L (13–23), and the values deduced from isotope-dilution studies (Table 2; 6, 7, 35). For example, Akay and Rose (24) showed that direct measurement of oxalate in blood collected in the presence of various enzyme inhibitors that block enzymatic oxidation of glyoxylate to oxalate gave values for plasma oxalate, 2.26 μmol/L, comparable with those derived from isotope-dilution studies. Allopurinol was used to inhibit xanthine oxidase (EC 1.1.3.22), boric acid to inhibit lactate dehydrogenase (EC 1.1.1.27), and phenylactic acid to inhibit glycolate oxidase ([5]-2-hydroxy-acid oxidase, EC 1.1.3.15).

However, several other observations fail to substantiate an important role for in vitro enzymatic oxidation of glyoxylate. Thus Buckle (40) and Zarembek and Hodgkinson (41), using methods capable of measuring 1.7 and 1.35 μmol/L, respectively, were unable to detect glyoxylate in blood. Concentrations lower than this would not likely result in significant in vitro oxalogenesis. Also, Maguire et al. (25) have shown that in vitro addition to blood of 6.7 μmol of glyoxylate per liter resulted in significant oxalogenesis, despite the presence of enzyme inhibitors. Thus in vitro conversion of glyoxylate to oxalate is probably not an

Table 2. Reported Normal Concentrations of Oxalate in Human Plasma, as Determined in In-Vivo Isotopic Studies

<table>
<thead>
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<th>SD</th>
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<td>1.83</td>
<td>—</td>
<td>6</td>
</tr>
<tr>
<td>35</td>
<td>0.83–2.11</td>
<td>1.31</td>
<td>—</td>
<td>5</td>
</tr>
<tr>
<td>36</td>
<td>0.10–0.9</td>
<td>—</td>
<td>—</td>
<td>3</td>
</tr>
<tr>
<td>37</td>
<td>0.65–1.45</td>
<td>0.94</td>
<td>0.36</td>
<td>5</td>
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<td>38</td>
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<td>1.25</td>
<td>0.36</td>
<td>13</td>
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<td>39</td>
<td>0.5–1.9</td>
<td>1.29</td>
<td>0.37</td>
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</table>

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important contributory factor to the higher oxalate concentrations found by assaying with direct methods.

Unlike the glyoxylate studies, there is convincing evidence for significant in vitro conversion of ascorbic acid to oxalate. Maguire et al. (25) were first to demonstrate substantial increases in plasma oxalate with storage. In their studies, plasma oxalate increased up to 750% in samples stored at −20 °C for 16 weeks. Further increases occurred when ascobic acid was added to plasma before storage, and storage of plasma ultrafiltrate showed greater oxalogenes than did plasma. Subsequent studies by Cole et al. (27), France et al. (42), and Kasidas and Rose (32) confirmed these findings.

We have recently performed studies in our laboratory in which we added [1-14C]ascorbic acid to plasma, then stored this at −20 °C for six, 11, and 21 days. Under those conditions 24%, 33%, and 45%, respectively, of [1-14C]ascorbic acid was converted to [14C]oxalate. These results are also consistent with the studies of Bradley et al. (43), who recovered only 44% of ascorbate in plasma stored at −20 °C for 21 days. Lastly, these investigators also showed that ascorbic acid was stable when stored at low pH, and other workers (26, 32, 42) have noted the stability of oxalate concentration in plasma stored at pH <3.8. Taken together, these observations suggest that ascorbate degradation may result in considerable in vitro generation of oxalate. This may explain a major part of the discrepancy between direct measurements of plasma oxalate in some earlier studies and the concentrations deduced from isotope-dilution studies. Also, it is important to acidify samples, to minimize ascorbate degradation.

Protein Binding

Oxalic acid binds significantly to plasma proteins at pH values less than 6.0 (15, 44, 45). This will lead to large losses of oxalate if plasma is acidified for storage and then ultrafiltered as part of an assay procedure. To demonstrate this, we ultrafiltered plasma with added [14C]oxalate at pH 5.7, 6.0, and 7.4, using an Amicon Centricon Ultrafiltration system, Type CF 50 membrane. Analytical recoveries were 26%, 44%, and 98%, respectively. In view of this, much oxalate presumably is lost to protein binding in assays such as that used by Kasidas and Rose (32), who ultrafiltered plasma at an acid pH. That Kasidas and Rose (32) did not find unusually low values may have been the result of offsetting errors. In addition, other workers not only have alkalized plasma for ultrafiltration, but also have failed to re-acidify it in storage. This probably explains the very high oxalate value, 55 μmol/L, found in normal plasma by Borland et al. (33), who alkalized plasma to pH 10.6 for ultrafiltration and then stored the plasma at −20 °C for several days before analysis.

Also, we have found that oxalate binding to plasma proteins is increased in patients with chronic renal failure. Thus, only 80.9% (SD 4.2%) of [14C]oxalate added to plasma from patients with renal failure was accounted for after ultrafiltration at pH 7.4. All of the above observations emphasize the importance of determining losses during extraction and ultrafiltration procedures in oxalate assays by use of [14C]oxalate internal standard.

Recommended Procedure for Determination of Oxalate in Plasma

Sample handling and preparation. Despite the reported lack of oxalate generation in blood during 1 h at 22 °C (26), we recommend removing plasma as quickly as possible after the blood sample is obtained. If the assay requires ultrafiltration (25, 32), this should be done without delay. For storage, the ultrafiltrate should be acidified. In our procedure we do this by collecting the ultrafiltrate directly in 3.5 mol/L HCl, 11 μL per milliliter of plasma ultrafiltrate. This gives a final pH of <3.0.

Analytical procedures. Several direct methods have been shown to yield oxalate values for normal plasma of <3.3 μmol/L, which is very similar to that found with isotope methods. By obviating the need to administer radioactive isotopes, these methods are easily applied to human subjects.

Wolthers and Hayer (26) developed a gas-chromatographic procedure for determining oxalate in plasma, analyzing the trimethylsilyl derivative. The method requires a number of extractions of acidified plasma with ethyl acetate; the combined extracts are dried under nitrogen before derivatization. Malonic acid is added to each plasma sample as an internal standard. With each sample batch, two control samples, without and with added ascorbic acid, are run and a correction factor is derived to compensate for losses. A single analysis requires 2 mL of plasma. A gas chromatograph and appropriate column are required. Analytical recoveries of oxalate added to serum were close to 100%; the within-assay CV was 5.8% for a serum sample with an oxalate concentration of 5.8 μmol/L. Wolthers and Hayer did not mention a detection limit. Their mean concentration of oxalate in normal plasma was 2.8 (SD 1.1) μmol/L.

Boer et al. (28) used oxalate oxidase and determined the CO2 released by the decrease in pH of an alkaline buffer. Their method requires a large blood sample, 50 mL, and an incubation period of 72 h. Analytical recovery of oxalate added to plasma was 105% (SD 8%), and the between-assay CV was 10.4% for a sample concentration of 7.7 μmol/L. The detection limit of the method is 0.47 μmol/L. The procedure has the advantage of requiring simple equipment. The normal value for oxalate in plasma was 3.3 (SD 1.3) μmol/L.

Parkinson et al. (31) used the coupled-enzyme method of Hatch et al. (19) but determined the NADH produced with a bioluminescent reaction. The assay requires 2 mL of plasma for a single analysis. The limit of detection of their method is 0.8 μmol/L. Analytical recovery of oxalate added to plasma averaged 99% (SD 3.2%). For a plasma sample with an oxalate concentration of 6.1 μmol/L, the within-run CV was 7.4%. The specialized bioluminescent equipment required is commercially available; however, the bioluminescent reagents are expensive. A major drawback of the assay is the very high blank values obtained, typically 140–200 mV on the potentiometer, with the oxalate content of a normal plasma increasing this response by only 4–5 mV. Parkinson et al. (31) attribute this very high blank to "apparent formate" contamination of their reagents. They also have reported the lowest oxalate values in plasma by a direct procedure, <0.8–1.5 μmol/L. However, because of the very high blanks involved, the reliability of the method at these low concentrations is questionable.

In our laboratory we use the coupled-enzyme method (19, 25) with oxalate decarboxylase and formate dehydrogenase and determine spectrophotometrically the NADH produced in the second reaction. The present procedure contains some modifications from our published method (19, 25). In brief, heparinized plasma is ultrafiltered at normal pH, 7.4, and the ultrafiltrate is collected in HCl (final pH <3.0). The oxalate is precipitated at pH 5.0 with saturated calcium
sulfate and ethanol (ultrafiltrate/calcium sulfate/ethanol, 6/2/13.5 by vol) and the precipitate is lyophilized. The lyophilized
material is extracted in 1.4 mL of 50 mmol/L citrate buffer, pH 3.2, at room temperature for 20 min and 0.20-mL aliquots are
assayed in duplicate as previously described (19). Tracer
quantities of [14C]oxalate added to plasma are used to
correct for possible losses on ultrafiltration, precipitation,
and extraction of dried precipitate. The assay requires about
20 mL of plasma from normal subjects for duplicate analysis
and 3 to 4 mL from patients with chronic renal failure.
Analytical recoveries of oxalate added to plasma were 94.2%
(SD 3.8%), and within-run CV was 6.9% for a sample
concentration of 3.5 μmol/L. Sensitivity of the assay is 0.55
μmol/L. The assay requires a spectrophotometer, lyophiliz-
er, and scintillation counting facilities. Both enzymes are
commercially available. Using this method, we determined
the oxalate content of normal plasma to be 1.25 (SD 0.47)
μmol/L (n = 23), in good agreement with values obtained by
isotope-dilution procedures (Table 2). That these values are
lower than our earlier results (25) is, we believe, ascribable
to decreased oxalogenesis resulting from more rapid plasma
separation, precipitation at pH 5.0 rather than 6.0, and
regulation of the plasma ultrafiltrate in HCl.

We have also recently applied our method (25) to determina-
tion of oxalate in dialysate, because review of the literature
suggests that similar methodological errors persist. In
some dialysis centers, ascorbate is added to the dialysis
solution as an antioxidant (46). Given the conversion of
ascorbate to oxalate described above, this could result in
considerable oxalate generation from ascorbate during
dialysis and so could invalidate any oxalate studies.

To prevent in vitro increases in oxalate concentration
during and after dialysis, dialysate should be collected in
HCl, a sufficient quantity to give a final pH of <3.0. To
obtain enough oxalate for easy determination by our assay,
we precipitate the oxalate in 24 mL of dialysate with ethanol
and calcium sulfate at pH 5.0, as described above for plasma
ultrafiltrate, and extract the lyophilized precipitate
with 2.0 mL of citrate buffer (50 mmol/L, pH 3.2). The
lyophilized precipitate is stable at −20°C for at least one
month.

In the case of dialysate from continuous ambulatory
peritoneal dialysis patients, where the fluid has a dwell time
of around 6 h, the possibility of oxalate generation from
ascorbate in the peritoneum should be considered when
studying oxalate removal or clearance in such patients.

The oxalate concentrations reported in plasma of hemodi-
asis patients by several investigators who used direct
methods agree well (Table 3), although the values are about
20-fold normal. The difference of about 1.5 μmol/L between
values for normal plasma oxalate derived by isotopic proce-
dures (6, 7, 35–39) and direct methods (25, 26, 28, 30) is less
important when oxalate is determined in patients with
chronic renal failure because of the markedly higher values
in these subjects. In one study (35), both methods, isotopic
and direct, were applied simultaneously and showed good
agreement for the higher concentrations in plasma.

Conclusions

The gap between plasma oxalate values obtained by in
vivo isotope-dilution procedures and direct methods has
narrowed considerably. Plasma oxalate concentrations
measured in the present study and by others (31, 32) demon-
strate that similar values can now be obtained by direct
methods and isotope-dilution procedures. However, many of

the recent direct methods have reported plasma oxalate
values approximately 1.5 μmol/L higher than those derived
by isotope-dilution procedures, 3.0 μmol/L (25, 26, 28, 30) vs
1.4 μmol/L (6, 7, 35–39). Further studies using direct
methods are necessary to confirm the lower values showing
similar concentrations to the isotopic procedure. Neverthe-
less, the difference in values obtained by the two methods is
now too small to be of clinical significance.

We thank Judy Brothius and Jeannette Humphries for excellent
technical assistance and Arline Schwartz for typing this manu-
script.

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| Table 3. Oxalate Concentrations Determined in Plasma of
<p>| Hemodialysis Patients |
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