The chlorination of taurine by the myeloperoxidase-
H2O2-Cl- system was investigated under steady state
conditions. By systematically varying the pH and the
concentrations of H2O2, Cl-, and taurine such that chlo-
ride inhibition and the unwanted formation of inactive
compound II intermediate are minimized, rate data
were found to fit a mechanism involving an enzyme-
bound chlorinating intermediate. The mechanism we
propose is as follows.

\[
\begin{align*}
MPO + H_2O_2 \rightarrow MPO-I + H_2O \\
MPO-I + Cl^{-} \rightarrow MPO-I-Cl^{-} \\
H^{+} + \text{taurine} \rightarrow MPO + \text{taurine monochloramine} + H_2O
\end{align*}
\]

The kinetic parameters determined at pH 4.7 are: \(k_1 = (3.3 \pm 0.2) \times 10^7 M^{-1} s^{-1}\), \(k_2 = (2.8 \pm 1.2) \times 10^6 M^{-1} s^{-1}\), and \(k_3 = (4.4 \pm 0.2) \times 10^5 M^{-1} s^{-1}\). The rate constant for compound I formation \(k_1\) is of the same order of magnitude as the
value \((1.8 \times 10^7 M^{-1} s^{-1})\) obtained using transient state
techniques in a previous study by our group. The value of \(k_3\) is 2 orders of magnitude greater than the non-
enzymatic reaction between HOCl and taurine at the
same pH.

The results of this study indicate that the chlorination
reaction mediated by the myeloperoxidase system in
vivo may involve an enzyme intermediate species rather
than free HOCl. Not only does this mechanism offer
the advantage of substrate specificity but also of speed com-
pared to the non-enzymatic reaction. This mechanism
also allows for the indiscriminate oxidation reactions
by HOCl are prevented in the leukocyte. The fast
formation of taurine monochloramine, a relatively non-
toxic and stable compound compared to HOCl, is con-
sistent with the proposed role of taurine in the neu-
rophil, that of protecting certain targets including
myeloperoxidase from the attack by potent chlorinated
oxidants.

Myeloperoxidase (donor: H2O2 oxidoreductase, EC 1.11.1.7)
is present in high concentrations in the granules of polymo-
phonuclear leukocytes and monocytes (1). It is released during

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‡ On study leave from the University of the Philippines at Los Baios,
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§ To whom correspondence should be addressed. Tel.: 403-492-3818;
Fax: 403-492-8231.

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advantage of this chromophoric product to monitor the chlorination rate of the MPO-H$_2$O$_2$Cl$^-$ system under steady state conditions. This three-substrate reaction was analyzed in terms of the two most plausible mechanisms: one that involves free HOCl as chlorinating agent and another that involves an enzyme-bound chlorinating intermediate. Kinetic parameters were extracted from secondary plots of steady state data and compared with rate constants obtained using transient state techniques and non-enzymatic methods.

**MATERIALS AND METHODS**

Bovine spleen MPO was isolated and purified using a combination of modified procedures (29–31). Complete details are available (32). The enzyme preparation used in this study exhibited a Reinheitszahl ($A_{402}/A_{280}$) of 0.83. The MPO concentration was determined spectrophotometrically using $e_{402}$ of 178 M$^{-1}$ cm$^{-1}$ (33).

Hydrogen peroxide (–30% solution, BDH Chemicals) concentration was determined after appropriate dilution using the horseradish peroxidase assay (34) and was confirmed by absorbance measurements at 240 nm where $e_{240}$ is 38.9 M$^{-1}$ cm$^{-1}$ (35). Taurine (Sigma), KCl (Aldrich), and the chemicals used for the phosphate buffers (Fisher) were used without further purification. Aqueous solutions were prepared using deionized water obtained from the Milli Q system (Millipore Corp.), and concentrations were determined by weight. A stock solution of HOCl was prepared by bubbling Cl$_2$ through 0.1 M NaOH (Aldrich). The Cl$_2$ was prepared by dipping concentrated HCl (Anachem) on reagent-grade MnO$_2$ (Matheson, Coleman and Bell) (36). This stock solution was stored in the cold protected from light. Its concentration was determined by measuring the absorbance at 295 nm where $\epsilon_{295}$ is 0.35 M$^{-1}$ cm$^{-1}$ (37).

Routine spectral measurements were made on a Beckman DU-650 spectrophotometer. Stopped flow measurements were performed using the Photal RA-601 rapid reaction analyzer equipped with a 1-cm observation cell. All experiments were carried out at 25.0 ± 0.5°C.

Preliminary transient state experiments were conducted to see if the MPO enzyme intermediate compounds I and II react with taurine. Both stopped flow and rapid scan experiments were carried out with H$_2$O$_2$ in one reservoir and MPO (in the presence and absence of taurine) in the other reservoir.

Steady state experiments were performed using the stopped flow mode of the Photal instrument. The chlorinating activity of MPO was determined by following the formation of TauNHCl at 252 nm ($e_{252}$) (38). One reservoir contained H$_2$O$_2$ in water; the other, MPO, KCl and taurine in buffer. The solutions were buffered at the desired pH to a final ionic strength of 0.1 M. pH measurements were made using a Fisher Accumet model 2C digital pH meter.

The initial rate was determined from the slope of the first portion of the traces obtained after mixing all the components in about 4 ms. Usually 6–8 traces were recorded, and the mean values were used in the plots. A nonlinear regression data analysis program (Enzfitter) was used for curve fittings and calculations. The initial rate ($v$) and substrate concentration ([S]) were fitted to a typical steady state equation,

$$\frac{v}{[E]_{tot}} = \frac{k_{cat}[S]}{K_M + [S]} \quad (\text{Eq. 1})$$

where $[E]_{tot}$ is the total MPO concentration and $k_{cat}$ and $K_M$ are typical Michaelis-Menten parameters. The $k_{cat}$ parameters were further fitted to a hyperbolic equation as a function of chloride. Steady state rates were also measured at various taurine concentrations.

The nonenzymatic chlorination of taurine by HOCl was studied in the stopped flow apparatus at different pH levels by monitoring the increase in absorbance at 252 nm as TauNHCl forms. HOCI solutions in one reservoir (final concentration 0.15 mM) were prepared immediately before the experiment by diluting the stock solution in buffer of ionic strength 0.1 M. A large excess of taurine (final concentrations of 5–25 mM) was placed in the other reservoir. The stopped flow traces were single exponentials from which pseudo-first order rate constants were determined by a nonlinear least squares fit of seven or more traces. The mean $k_{cat}$ values were plotted against taurine concentration, and the apparent second order rate constant of the reaction was calculated from the slopes using linear regression analysis.

**RESULTS**

Prior to the steady state experiments, transient state studies were conducted (data not shown) to check if taurine could be

![FIG. 1. Formation of taurine monochloramine in the MPO-H$_2$O$_2$Cl$^-$ system. The lowest trace is for 10 mM taurine in 0.1 M phosphate buffer at pH 4.7. The middle traces were taken after 0.2 µM MPO and 4.0 mM KCl were added to the initial solution, and the upper trace was taken after adding 0.2 mM H$_2$O$_2$.](image-url)
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Fig. 2. Typical time course of the enzymatic formation of taurine monochloramine. The chlorination rate was determined from the slope of the initial linear portion of the trace. Reaction conditions were as follows: 0.1 μM MPO, 7 mM taurine, 0.03 M KCl, 2 mM H₂O₂ in 0.1 M phosphate buffer, pH 4.7.

Fig. 4. Chloride dependence of chlorination rate of taurine. Reaction conditions: 0.1 μM MPO, 5 mM taurine in 0.1 M phosphate buffer, pH 4.7. KCl concentrations (in mM) are: 0.5 (□), 0.75 (●), 1.0 (△), 1.5 (■), and 2.0 (○). The data were fitted using Equation 2 under "Results."
We have derived the rate equations for Mechanisms I and II (see "Appendix"). Mechanism I predicts a linear dependence of \( k_{\text{cat}} \) and \( K_M \) on chloride concentration, whereas Mechanism II predicts hyperbolic behavior. Thus, our data clearly support Mechanism II, which invokes the formation of an enzyme-bound chlorinating intermediate. We can extract kinetic parameters from secondary plots, particularly the plot of \( k_{\text{cat}} \) versus Cl\(^{-}\) concentration (Fig. 5). Using Equation 20A (see "Appendix"),

\[
k_{\text{cat}} = k_{\text{cat}}[\text{taurine}]
\]

we determined \( k_3 \) to be \((3.6 \pm 0.3) \times 10^5 \text{M}^{-1} \text{s}^{-1}\). Substituting this value to Equation 21A (see "Appendix"),

\[
K_{\text{Cl}^-} = \frac{k_3[\text{taurine}]}{k_2}
\]

minimized. Thus, using selected ranges for these three substrates, we were able to measure accurately rates of formation of TauNHCl. The catalytic amount of MPO used in this study did not interfere with the absorbance of TauNHCl (Fig. 1). Moreover, the linear portion of the traces on a short time scale (Fig. 2) allowed us to measure the first chlorination step before enough TauNHCl accumulates to be further chlorinated to TauNClt.

We then carried out the chlorination reaction at several pH levels to determine which pH would be most suitable (Fig. 3). Our choice of pH 4.7 for subsequent experiments was based on the following reasons. (a) At pH above 5.6, the rate of enzyme inactivation increases (46, 47) and H\(_2\)O\(_2\) inhibition becomes important (39) leading to a decrease in chlorinating activity. (b) At pH below 4, the inhibitory binding between Cl\(^{-}\) and native enzyme becomes stronger (31). (c) The pH under physiological conditions in the phagosome has been reported to fall in the range from 4.5 to 5.0 (48). Moreover, we obtained traces with good signal-to-noise ratio for the runs performed at this pH. Thus, subsequent measurements were conducted at pH 4.7.

The initial rate of chlorination of taurine was dependent upon both H\(_2\)O\(_2\) and Cl\(^{-}\) and showed saturation behavior (Fig. 4). After fitting the experimental data to Equation 1, parameters \( k_{\text{cat}} \) and \( K_M \) were obtained. Both of these parameters showed nonlinear dependence with Cl\(^{-}\) (Figs. 5 and 6). Of these parameters \( k_{\text{cat}} \) has the more straightforward meaning; it is the maximum turnover number for fixed concentrations of chloride and taurine. \( k_{\text{cat}} \) values were fitted to a rectangular hyperbola and a second set of parameters, \( k_{\text{Cl}^-} \) and \( K_{\text{Cl}^-} \), were obtained.

Both mechanisms involve the reversible formation of compound I, for which evidence has been presented recently.\(^2\) The reverse rate constant \( k_{-1} \) becomes insignificant when large excess of H\(_2\)O\(_2\) is used over the enzyme concentration as was the case for the steady state experiments in this work.

The two mechanisms also include the reversible binding of Cl\(^{-}\) to native enzyme. This binding has been documented in several reports (31, 39-43). In chlorination reactions, chloride not only acts as a substrate for MPO but also behaves as a competitive inhibitor of H\(_2\)O\(_2\). Chloride binding to MPO is pH-dependent. When the pH increases, the affinity of Cl\(^{-}\) for the enzyme decreases. In contrast, the formation of the inactive intermediate compound II increases with increasing pH (44). Thus, H\(_2\)O\(_2\) also inhibits the chlorination reaction. It is now evident that there is no fixed optimum pH for MPO-catalyzed chlorination. It depends on the relative concentrations of Cl\(^{-}\) and H\(_2\)O\(_2\) (45). Various combinations of pH, Cl\(^{-}\), taurine, and H\(_2\)O\(_2\) concentrations were tried so that the conditions were obtained in which the chlorination reaction was the rate-determining step and that inhibition by Cl\(^{-}\) and H\(_2\)O\(_2\) was at least

\(^2\) L. A. Marquez, J. T. Huang, and H. B. Dunford, submitted for publication.
we calculated \( k_2 \) to be \((2.8 \pm 1.2) \times 10^6 \text{ M}^{-1} \text{ s}^{-1}\).

Another criterion that can distinguish between Mechanisms I and II is the dependence of the initial rate of chlorination on taurine concentration. Mechanism I predicts no dependence, while Mechanism II does. Fig. 7 demonstrates a hyperbolic dependence of rate on taurine concentration. A curve fit of the data yields parameters \( k_{cat} \) and \( K_M \). The value of \( k_1 \) can be calculated from \( k_{cat} \) (Equation 25A under “Appendix”) using the value of \( k_2 \) previously determined. The value of \( K \) for the dissociation of the MPO-chlorite complex was taken from an average of previously reported values: 1.2 mM (43), 0.8 mM (interpolated from Fig. 9 of Ref. 40) and 1.0 mM (interpolated from Fig. 3 of Ref. 39). The \( k_1 \) value obtained, \((3.3 \pm 0.2) \times 10^7 \text{ M}^{-1} \text{ s}^{-1} \), is of the same order of magnitude as those obtained for compound I formation of MPO from canine pus (2.0 \times 10^7 \text{ M}^{-1} \text{ s}^{-1})(41), human blood (2.3 \times 10^7 \text{ M}^{-1} \text{ s}^{-1})(40), and bovine spleen (1.8 \times 10^7 \text{ M}^{-1} \text{ s}^{-1}).^2\ Equation 26A (see “Appendix”) gives a \( k_3 \) value of \((5.2 \pm 0.1) \times 10^3 \text{ M}^{-1} \text{ s}^{-1} \). The mean \( k_3 \) value from the chlorite dependence (Equation 20A) and taurine dependence (Equation 26A) is \((4.4 \pm 0.2) \times 10^3 \text{ M}^{-1} \text{ s}^{-1} \). Thus, we were able to satisfy three criteria supporting the existence of a MPO-chlorinating intermediate (Figs. 5–7). Such an enzyme-bound species has similarly been proposed for chloroperoxidase (26–28). We were also able to estimate rate constants for the different steps in the MPO mechanism. We show from the magnitudes of the rate constants that the formation of TauNHCl is indeed the rate-controlling step under the steady state conditions employed in this study. We also obtained a rate constant for the reaction between compound I and \( \text{Cl}^- \) to form the intermediate that cannot be determined using transient state kinetic techniques due to the instability of compound I.

In the presence of excess taurine, the non-enzymatic reaction between taurine and \( \text{HOCl} \) shows pseudo-first order kinetic behavior. Apparent second order rate constants were obtained from the slopes of the plot of \( k_{obs} \) versus taurine concentration at various \( \text{pH} \) levels. An interpolation from the \( \text{pH} \) rate profile gives a value of \( 3.5 \times 10^5 \text{ M}^{-1} \text{ s}^{-1} \) for the non-enzymatic reaction of taurine with \( \text{HOCl} \) at \( \text{pH} \) 4.7. This result indicates that a 100-fold increase in the rate of chlorination is obtained in the enzymatic reaction. Thus, a MPO-bound chlorinating intermediate offers not only the advantage of substrate specificity but also that of speed.

There have been numerous studies on MPO-catalyzed chlorination reactions (5, 6, 8, 39, 43, 45, 49), but it appears that the evaluation of steady state parameters has been complicated by peroxidase-like reactions (5, 6, 8, 39, 43, 45, 49). For example, monochlorodimethyldiamine, which is routinely used to measure chlorinating activity of peroxidases, was found to compete with \( \text{Cl}^- \) for compound I and promote the accumulation of compound II (48). These complications were circumvented when taurine was used as the substrate because it did not exhibit reactivity with either compound I or II.

The results of our study are physiologically relevant. Taurine concentration is higher than that of other free amino acids in the leukocyte (23). In fact, taurine accounts for a large part of the amines that are available for reaction with chlorinating reagents (25). Chloramines were found to accumulate in the extracellular medium when stimulated leukocytes are incubated in vitro (50). Apparently, taurine acts as a trap for chlorinated oxidants produced by the MPO-\( \text{H}_2\text{O}_2\)-\( \text{Cl}^- \) system. If \( \text{HOCl} \) were produced and allowed to diffuse freely, this potent oxidant could cause extensive damage to cellular components (51, 52). For example, it has been reported that when the amine concentration is low, \( \text{HOCl} \) reacts with and inactivates MPO (53). \( \text{HOCl} \) also reacts with \( \text{H}_2\text{O}_2 \) (54), and this results in the loss of oxidizing equivalents. Taurine has recently been found to be effective in protecting biomembranes against oxidant injury (55, 56). The formation of an enzyme-bound species that chlorinates taurine is consistent with taurine's protective role against oxidant damage, which could otherwise be indiscriminately brought about by free \( \text{HOCl} \). While it has been demonstrated that chloramines like TauNHCl can inactivate enzymes by attacking sulfhydryl groups, lyse erythrocytes, and kill bacteria (57), TauNHCl is much less reactive and less toxic than \( \text{HOCl} \) (25). Therefore, taurine may function as a moderator of neutrophil cytotoxicity.

**APPENDIX**

The derivation of the steady state equations for Mechanisms I and II are shown. The abbreviations are defined in Footnote 1.

**Mechanism I**

**Steady State—**

\[
-\frac{d[MPO]}{dt} = \frac{d[MPO-I]}{dt} = 0
\]

(Eq. 1A)

\[
k_1[MPO][H_2O_2] - k_2[MPO-I][H_2O] - k_2[Cl^-][MPO-I] = 0
\]

At large excess of \( \text{H}_2\text{O}_2 \) over MPO concentration, \( k_1 \sim 0 \).

(Eq. 2A)

\[
\frac{d[HOCl]}{dt} = k_2[Cl^-][MPO-I] - k_2[HOCl][taurine] = 0
\]

(Eq. 3A)

\[
k_2[Cl^-][MPO-I] = k_2[HOCl][taurine]
\]

(Eq. 4A)

**Equilibrium Equation for Binding of Chloride—**

\[
K = \frac{[MPO][Cl^-]}{[MPO-I][Cl^-]} \text{ or } [MPO][Cl^-] = K[MPO-I][Cl^-]
\]

(Eq. 5A)

**Mass Balance Equation—**

\[
[E]_{tot} = [MPO][Cl^-] + [MPO] + [MPO-I]
\]

(Eq. 6A)

Substituting from Equations 2A and 5A:

\[
[E]_{tot} = \frac{[Cl^-]}{K} + 1 + \frac{1}{k_2[Cl^-]}(1 + \frac{1}{k_2[H_2O_2]})[MPO-I]
\]

(Eq. 7A)

**Initial Rate Expression—**

\[
v = k_2[HOCl][taurine]
\]

(Eq. 8A)

From Equations 4A and 7A, Equation 8A is converted to Equation 9A.

\[
v = k_2[Cl^-][MPO-I] = \frac{k_1[Cl^-][E]_{tot}}{[(Cl^-)/K + 1] + \frac{1}{k_2[Cl^-]}[H_2O_2]}
\]

(Eq. 9A)

Rearranging this, we obtain Equation 10A,

\[
v = \frac{k_1[Cl^-][E]_{tot}}{[(Cl^-)/K + 1] + \frac{1}{k_2[Cl^-]}[H_2O_2]} \text{ or } \frac{v}{[E]_{tot}} = \frac{k_{cat}[H_2O_2]}{K_M[H_2O_2]}
\]

(Eq. 10A)

where \( k_{cat} = k_2[Cl^-] \) and \( K_M = (1/[Cl^-]/K + 1) \times (k_2[Cl^-]/k_1) \). The plots of \( k_{cat} \) and \( K_M/[Cl^-][K + 1] \) against \( [Cl^-] \) should be linear.

**Mechanism II**

**Steady State—**

\[
-\frac{d[MPO-I][Cl^-]}{dt} = k_2[Cl^-][MPO-I] - k_2[MPO-I][Cl^-][taurine] = 0
\]

(Eq. 11A)
This hyperbolic plot yields two parameters, $k_c'$ and $K_M'$, given by Equations 25A and 26A, respectively.

$$k_c' = \frac{1}{k_{[Cl^-]} + \frac{1}{k_{[H_2O_2]} + k_{[Cl^-]}}}$$  \hspace{1cm} (Eq. 25A)

$$K_M' = k_c' \cdot k_e$$  \hspace{1cm} (Eq. 26A)

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