Human monocytes incubated with phorbol myristate acetate (PMA) or opsonized zymosan particles can chlorinate the $\beta$-amino acid taurine to its monochloramine derivative. Taurine monochloramine can then be quantitated by its ability to oxidize 5-thio-2-nitrobenzoic acid to its disulfide or by its characteristic absorbance peak at 252 nm. Stimulated, but not resting, monocytes chlorinated taurine by a process dependent on time, cell concentration, and pH. The formation of taurine chloramine by stimulated monocytes could be inhibited by catalase, azide, or cyanide, was unaffected by superoxide dismutase, and was stimulated by exogenous myeloperoxidase. Thus, taurine chloramine generation by human monocytes appeared dependent on both $H_2O_2$ and myeloperoxidase. Compared to human neutrophils, the monocyte could generate similar amounts of chloramine when stimulated with phorbol myristate acetate, but far less if opsonized zymosan particles were used as the trigger. Based on the known ability of the $H_2O_2$-myeloperoxidase-Cl$^-$/ system to generate free HOCI, it would seem that this oxidant is the most likely species responsible for the monocyte-mediated chlorination reactions. Thus, we have used a simple quantitative assay to demonstrate the ability of the human monocyte to generate large quantities of a highly reactive and toxic oxygen metabolite.

**Human Monocytes or neutrophils can generate $H_2O_2$ and release myeloperoxidase to form a system capable of peroxidizing suitable halide substrates.**

The oxidants generated by the myeloperoxidase system are toxic to microorganisms or mammalian cells and may also inactivate soluble mediators involved in the inflammatory response. We have recently demonstrated that intact, human neutrophils use $H_2O_2$, myeloperoxidase, and Cl$^-$ to generate a species that can chlorinate the $\beta$-amino acid taurine to its stable monochloramine derivative. Based on the known ability of the $H_2O_2$-myeloperoxidase-Cl$^-$ systems to generate free HOCI, we concluded that neutrophils chlorinated taurine by producing this powerful oxidant. Thus:

$$H_2O_2 + Cl^- \xrightarrow{\text{Myeloperoxidase}} HOCI + H_2O$$

$$HOCI + H_2N - CH_2CH_2SO_3H \rightarrow \text{Taurine}$$

$$\xrightarrow{\text{Myeloperoxidase}} CINH - CH_2CH_2SO_3H + H_2O$$

Taurine chloramine

Monocytes also have the potential to generate HOCI, but this cell can produce only half as much $H_2O_2$ and contains one-third the myeloperoxidase content of the neutrophil. Despite those differences, several studies have demonstrated that human monocytes can effectively use the myeloperoxidase system to mediate target cell destruction. To our knowledge, nothing is known about the ability of intact monocytes to generate chloride oxidation products. In this study, we have used the taurine-trapping technique to detect and quantitate the chlorinating species generated by the human monocyte.

**MATERIALS AND METHODS**

**Cell Preparations**

Monocytes and neutrophils were obtained from the peripheral venous blood of normal healthy volunteers. Purified preparations of human monocytes were isolated as previously described. Briefly, mononuclear cells obtained by Ficoll-Hypaque separation were washed twice in Selegman's balanced salt solution (GIBCO Laboratories, Grand Island Biological Co., Grand Island, NY) with 13% autologous serum. An aliquot of $50 \times 10^6$ mononuclear cells was added to Falcon 3003 tissue culture dishes (Falcon Labware; Becton Dickinson and Co., Oxnard, CA) and incubated for 90 min at 37°C in a humidified 95% air/5% CO2 atmosphere. The dishes were washed 5 times with RPMI (GIBCO) supplemented with 10% fetal bovine serum (GIBCO) to remove nonadherent cells. The adherent cells were then incubated with Selegman’s balanced salt solution containing 0.2% bovine albumin (Sigma Chemical Co., St. Louis, MO) and 0.1% EDTA for 1 min, at which time the adherent cells were gently removed with a rubber policeman. The cells were washed and resuspended in Dulbecco's phosphate-buffered saline (pH 7.4, GIBCO) supplemented with 1 mg/ml glucose.

Cell preparations routinely contained >95% monocytes, as assessed by Wright's stain morphology or esterase staining. Contaminating cells consisted of no more than 3%–4% lymphocytes or 3% granulocytes.

Purified preparations of neutrophils were prepared as previously described by Ficoll-Hypaque separation and dextran sedimentation. Cell preparations contained >95% neutrophils and <5% eosinophils by Wright's stain morphology.
Incubation Conditions

Monocytes or neutrophils were suspended in Dulbecco's phosphate-buffered saline (pH 6.5, 7.0, 7.4, or 7.6) and placed in 24-well tissue culture plates (Falcon 3047) with 100 μg/ml fatty-acid-free human albumin (Sigma) in the presence or absence of 15 mM taurine (2-amino-methanesulfonic acid, Sigma). In selected experiments, the cells were added to 5-ml round-bottom, polypropylene tubes (Falcon 2005). In either case, the final volume was 1 ml. Cells were stimulated to generate oxygen metabolites and release lysosomal contents by the addition of phorbol myristate acetate (PMA; Consolidated Midland Corp., Forrester, NY) or opsonized zymosan particles (ICN Nutritional Biochemicals, Cleveland, OH), prepared as previously described. The plates (or tubes) were then placed in a humidified air incubator at 37°C for various lengths of time. At the end of the incubation period, the duplicate samples were assayed as described below. Other additions to the cell systems included bovine superoxide dismutase (3,200 U/mg, Boehringer Mannheim Biochemicals, Indianapolis, IN), bovine catalase (100,000 U/mg, Worthington Biochemical Corp., Freehold, NJ), sodium azide, sodium cyanide (Fisher Scientific Co., Pittsburgh, PA), or purified canine myeloperoxidase (5-5'-dithiobis (2-nitrobenzoic acid) (DTNB; see Fig. 1). Catalase was washed over an XM-100A ultrafiltration membrane (Amicon Corp., Lexington, MA) and assayed before use. Catalase was assayed according to the method of McCord and Fridovich, while myeloperoxidase activity was determined by the o-dianisidine method and activity expressed in international units. Catalase was washed over an XM-100A ultrafiltration membrane (Amicon Corp., Lexington, MA) and assayed before use.

Taurine Chloramine Assay

At the end of the incubation period, catalase (50 μg; Type C-40; Sigma) was added to the cell mixtures to reduce any residual H₂O₂. The samples were then incubated with an excess of 5-thio-2-nitrobenzoic acid (TNB) for 5 min at room temperature, spun at 500 g for 5 min, and the TNB concentration remaining in the clear supernatant determined. The chloramine concentration was calculated based on its ability to oxidize 2 mole of TNB to 1 mole of the disulfide, 5,5'-dithiobis (2-nitrobenzoic acid) (DTNB; see Fig. 1). TNB was prepared by reducing DTNB (Sigma), as previously described, and its concentration calculated based on an extinction coefficient of 1.36 x 10⁴ M⁻¹ cm⁻¹. Results are expressed as the mean ± 1 SD unless otherwise stated.

RESULTS

Chlorination of Taurine by Intact Monocytes

If monocytes generate HOCI or a species of similar reactivity, then stimulated cells incubated with an excess of taurine should form the N-chloro derivative.

Figure 1. The oxidation of TNB to DTNB by taurine chloramine.

Table 1. Oxidant Formation by Monocytes

<table>
<thead>
<tr>
<th>Additive</th>
<th>TNB Oxidized (nmole)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10⁶ Monocytes</td>
<td>0.0 ± 0.0</td>
</tr>
<tr>
<td>10⁶ Monocytes + PMA</td>
<td>111.8 ± 36.8</td>
</tr>
<tr>
<td>10⁶ Monocytes + opsonized zymosan</td>
<td>80.2 ± 29.8</td>
</tr>
</tbody>
</table>

*The complete system consisted of 10⁶ monocytes incubated with 15 mM taurine for 2 hr at 37°C in 1 ml of Dulbecco’s buffer (pH 7.4). Monocytes were stimulated with 30 μg/ml of PMA or 1.25 mg/ml of opsonized zymosan particles.

†Results are expressed as the mean ± 1 SD of 4 experiments for the resting cells and 16 experiments each for the stimulated cells.

Thus, 10⁶ monocytes were stimulated with either PMA or opsonized zymosan particles in the presence of 15 mM taurine for 2 hr. At the end of the incubation period, catalase was added to reduce residual H₂O₂, an excess of TNB was added, and its oxidation quantitated (Table 1). While resting monocytes did not form a detectable oxidant, stimulated cells generated a species capable of oxidizing TNB (Table 1).

The data presented in Table 1 represent the total amount of the oxidant detected in the entire incubation mixture. If the samples were first pelleted and the button and supernatant analyzed separately, >90% of the oxidant was found in the supernatant.

In the absence of exogenous taurine, stimulated monocytes could form small amounts of an oxidant that could be detected by the TNB technique. After a 2-hr incubation, 10⁶ PMA-triggered monocytes generated a compound(s) capable of oxidizing 22.4 ± 6.4 nmole of TNB (n = 3), while zymosan-stimulated cells oxidized 18.0 nmole (n = 2). In our previous study, taurine chloramine generated by the neutrophil could be detected by its characteristic absorption peak at 252 nm. A difference scan of the supernatants from 10⁶ resting versus 10⁶ PMA-stimulated monocytes in the presence of taurine revealed a peak with a λ-max of ~250 nm (Fig. 2). However, unlike the neutrophil study, if the amount of chloramine is calculated directly from this peak, almost twice as much taurine chloramine is detected when compared to the values obtained by the TNB technique. Because other species may absorb in this region, the sample was rescanned after the addition of a strong nuclease to reduce the chloramine. We recently noted that thioethers, like methionine, can rapidly reduce chloramines to their respective amines. For example, following the addition of an excess of methionine (100 nmole) to 50 nmole of authentic taurine chloramine, the 252-nm peak of the oxidant completely disappears and the mixture no longer oxidizes TNB (data not shown). As shown in Fig. 2, following the addition of methionine to the supernates from the resting and stimulated cells, the peak absorp-
Fig. 2. Optical spectrum of oxidant generated by PMA-stimulated monocytes in the presence of taurine. An aliquot of 10⁶ monocytes was incubated with 15 mM taurine in the absence or presence of PMA (30 ng/ml) for 2 hr at 37°C in 1 ml of Dulbecco’s buffer (pH 7.4). At the end of the incubation period, samples were centrifuged (500 g for 5 min) and the supernatants scanned. The reference cuvette contained supernatants from the resting monocytes. (A) Resting cells with taurine versus stimulated cells with taurine, and (B) difference spectrum following the addition of 1 mM methionine to the reference and sample cuvettes. (Inset) Optical spectrum of oxidant generated by PMA-stimulated monocytes in the absence of taurine. Samples were treated as above except taurine was omitted. (A) Resting cells versus stimulated cells, and (B) difference spectrum following the addition of 1 mM methionine to both cuvettes. A replicate sample contained an oxidant capable of oxidizing 16.0 nmole of TNB.

Fig. 3. (A) Taurine chloramine formation by monocytes as a function of time. An aliquot of 10⁶ monocytes was incubated with 15 mM taurine for varying periods of time at 37°C in 1 ml of Dulbecco’s buffer (pH 7.4). Monocytes were stimulated with 30 ng/ml of PMA (–––) or 1.25 mg/ml of opsonized zymosan particles (○–○). Results are expressed as the mean ± 1 SEM of 5 experiments. (B) Taurine chloramine formation as a function of monocyte number. Varying numbers of monocytes were incubated under conditions described in A for 2 hr. Monocytes were stimulated with either 30 ng/ml PMA (–––) or 1.25 mg/ml of opsonized zymosan particles (○–○). Results are expressed as the mean ± 1 SEM of 5 experiments.

The oxidizing potential was reduced from 0.0435 to 0.0190 and the oxidizing potential was lost. Based on an extinction coefficient of 429 M⁻¹ cm⁻¹ for taurine chloramine,⁹ 57 nmole of the chloramine were detected. In close agreement, replicate samples oxidized 108 nmole of TNB, corresponding to the presence of 54 nmole of taurine chloramine. Although taurine monochloramine can be chlorinated to the dichloramine, these species absorb at 300 nm¹⁹ and were not detected. Interestingly, difference spectra of resting versus stimulated cells in the absence of taurine also revealed the formation of a species that absorbed in the 250-nm region, which could be reduced by methionine (Fig. 2, insert). Thus, stimulated monocytes incubated in the presence of taurine generated the respective chloramine, while in the absence of taurine, an uncharacterized oxidant(s) with similar properties could be detected.
chloramine was also linear as a function of time. With either stimulus, chloramine formation was complete after a 2-hr incubation. Under the conditions used, chloramine generation was maximal in the presence of 15 mM taurine and 30 ng/ml PMA or 1.25 mg/ml opsonized zymosan (data not shown). Taurine chloramine was also linear as a function of monocyte number from 2.5 x 10^5 to 2 x 10^6 cells/ml (Fig. 3B). In 16 experiments, 10^6 PMA or zymosan-stimulated cells chlorinated 55.9 ± 18.4 nmole and 40.1 ± 14.9 nmole of taurine, respectively, during a 2-hr incubation. These data accurately reflect the total amount of chloramine generated, because known amounts of synthesized taurine chloramine could be almost completely recovered from suspensions of either resting or stimulated cells (Table 2).

### Mechanism of Taurine Chloramine Formation by Monocytes

If stimulated monocytes chlorinate taurine by generating HOCl or a species of similar reactivity then we would expect a requirement for both H_2O_2 and myeloperoxidase. Indeed, as shown in Fig. 4, catalase markedly inhibited chloramine generation, while superoxide dismutase had no significant effect. Heat-inactivated catalase did not inhibit the chlorination reaction with either stimulus (PMA system, 94.8% ± 6.7% of control; zymosan system, 93.4% ± 5.2% of control; n = 4). Azide or cyanide, two potent myeloperoxidase inhibitors, also significantly decreased chloramine generation (Fig. 4). Because monocytes do not contain large amounts of myeloperoxidase, we also examined the ability of exogenous myeloperoxidase to stimulate chloramine formation by the monocytes. In six experiments, 8 mU of purified myeloperoxidase stimulated chloramine generation by 10^6 PMA-stimulated monocytes from 54.3 ± 22.1 nmole to 82.0 ± 21.0 and from 38.2 ± 15.9 to 61.8 ± 24.0 nmole (p < 0.005; paired Student’s t test) for zymosan-stimulated cells. Apparently, monocytes incubated with either stimulus do not release sufficient myeloperoxidase to utilize all of the H_2O_2 generated.

Based on a requirement for H_2O_2 and myeloperoxidase in the chlorination reaction, chloramine formation was examined as a function of pH. Myeloperoxidase has an acid pH optimum, while maximal H_2O_2 generation seems to be favored at a more alkaline pH. As shown in Fig. 5, chloramine generation appears maximal with either stimulus at an approximate pH of 7.4.

### Taurine Chloramine Formation by Monocytes versus Neutrophils

Although both monocytes and neutrophils can generate H_2O_2 and release myeloperoxidase, the monocyte produces less H_2O_2 and has a lower myeloperoxidase content. Table 3 compares the ability of 10^6 monocytes from 54.3 ± 22.1 nmole to 82.0 ± 21.0 and from 38.2 ± 15.9 to 61.8 ± 24.0 nmole (p < 0.005; paired Student’s t test) for zymosan-stimulated cells. Apparently, monocytes incubated with either stimulus do not release sufficient myeloperoxidase to utilize all of the H_2O_2 generated.

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**Table 2. Recovery of Taurine Chloramine From the Monocyte System**

<table>
<thead>
<tr>
<th>Additive</th>
<th>Taurine Chloramine (nmole)</th>
<th>Percent Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monocytes</td>
<td>0.0</td>
<td>—</td>
</tr>
<tr>
<td>Monocytes + 50 nmole taurine chloramine</td>
<td>46.0</td>
<td>92</td>
</tr>
<tr>
<td>Monocytes + PMA</td>
<td>53.5</td>
<td>—</td>
</tr>
<tr>
<td>Monocytes + PMA + 50 nmole taurine chloramine</td>
<td>106.0</td>
<td>102</td>
</tr>
<tr>
<td>Monocytes + zymosan</td>
<td>42.5</td>
<td>—</td>
</tr>
<tr>
<td>Monocytes + zymosan + 50 nmole taurine chloramine</td>
<td>90.5</td>
<td>98</td>
</tr>
</tbody>
</table>

* A quantity of 10^6 monocytes was incubated alone or with 50 nmole taurine chloramine in the presence of 15 mM taurine for 2 hr at 37°C in Dulbecco’s buffer (pH 7.4). Monocytes were stimulated with either PMA (30 ng/ml) or opsonized zymosan (1.25 mg/ml). Taurine chloramine was synthesized as described in Materials and Methods.

† Results are expressed as the mean ± SD of 5 experiments.

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**Figure 4.** The effect of catalase (10 μg/ml), superoxide dismutase (10 μg/ml), azide (10^-4 M), and cyanide (5 x 10^-4) on taurine chloramine generation by 10^6 monocytes. Cells were incubated with 15 mM taurine for 2 hr at 37°C in 1 ml of Dulbecco’s buffer (pH 7.4) and stimulated with either 30 ng/ml PMA (□) or 1.25 mg/ml opsonized zymosan (●). Results are expressed as the mean percent of control ± 1 SD of 5 experiments.

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*This concentration of taurine did not inhibit O_2^- generation by PMA or zymosan-stimulated monocytes.
were stimulated with either 30 ng/ml PMA or 1.25 mg/ml of opsonized zymosan particles for 2 hr at 37°C in 1 ml of Dulbecco's buffer at varying pHs.

Monocytes were incubated with 15 mM taurine for 2 hr at 37°C to chlorinate taurine in the presence of PMA or opsonized zymosan particles. Interestingly, while both populations of PMA-stimulated cells generated similar amounts of the chloramine, zymosan uncovered the potential ability of the neutrophil to produce far larger amounts of the chloramine than the monocyte.

**DISCUSSION**

Human monocytes are armed with a plasma membrane-associated NADPH oxidase and the lysosomal enzyme myeloperoxidase, which together allow the cell to generate a variety of reactive oxygen metabolites. Recently, we reported that PMA-stimulated monocytes use the myeloperoxidase system to generate a tumoricidal species with characteristics similar if not identical with HOCI. In order to detect and quantitate chloride oxidation products generated by the intact monocyte, we have taken advantage of the ability of taurine to rapidly react with HOCI (or a species with similar reactivity) to form the stable, β-amino-acid chloramine. In this study, we have demonstrated that purified suspensions of human monocytes incubated with PMA or opsonized zymosan particles chlorinated taurine to its monochloramine. The generation of the chlorinating species was (1) inhibited by catalase, azide, or cyanide, (2) unaffected by superoxide dismutase, and (3) stimulated in the presence of exogenous myeloperoxidase. Thus, it appears that monocytes utilize H2O2, myeloperoxidase, and Cl− to form an oxidant capable of chlorinating taurine. Although we have not directly identified HOCI as the chloride oxidation product, the H2O2-myeloperoxidase-Cl− system is known to generate free HOCI. These data suggest that HOCI (or its anion hypochlorite) is the most likely species responsible for monocyte-mediated taurine chloramine generation.

HOCI is a powerful oxidant capable of reacting with a wide variety of biological substrates. Therefore, it would seem unlikely that unreacted HOCI would accumulate in a complex cell system. However, it is interesting to note that PMA- or zymosan-triggered monocytes (and neutrophils) accumulated an oxidant in the absence of exogenous taurine. The species could oxidize TNB, absorbed in the 250-nm region, and react with a thioether. Although further characterizations of this oxidant(s) are underway, it may well be the product of reactions between HOCI and cell-derived amines.

H2O2 generated by the monocyte may be metabolized along several different routes, including reduction by catalase, glutathione peroxidase, and myeloperoxidase, or it may react nonenzymatically with oxidizable substrates. Thus, the total amount of HOCI generated by the cell would depend on the ability of myeloperoxidase to complete with other pathways for the available H2O2. In this study, both PMA and opsonized zymosan triggered sufficient myeloperoxidase release to catalyze the chlorination reaction. However, the addition of exogenous myeloperoxidase to the triggered monocytes increased chloramine formation by ~50% with either stimulus. If the amount of taurine chloramine produced in the presence of excess myeloperoxidase is a true reflection of the total amount of H2O2 available for chlorination reactions, then monocytes are ~65% efficient in H2O2 utilization when stimulated with PMA (± myeloperoxidase) or myeloperoxidase ± 54 nmole/82 nmole or opsonized zymosan particles (38 nmole/62 nmole).

Monocytes and neutrophils can both effectively

![Fig. 5. Taurine chloramine formation by monocytes as a function of pH.](image)
mediate target cell destruction via the myeloperoxidase system, but the monocyte would seem to be at a disadvantage based on its ability to generate less H₂O₂ and its lower myeloperoxidase content. However, direct comparisons of the ability of the two cell populations to chlorinate taurine revealed that PMA-stimulated monocytes and neutrophils could generate similar amounts of the chloramine. Although these results are surprising, they are in close agreement with our earlier study demonstrating the ability of PMA-stimulated monocytes and neutrophils to mediate comparable myeloperoxidase-dependent cytotoxicity against a tumor cell target. In contrast to these findings, zymosan-stimulated neutrophils generated ∼4.5 times more taurine chloramine than comparably treated monocytes. We should note, however, that human neutrophils can internalize opsonized zymosan at faster rates than comparably treated monocytes. Nonetheless, it appears that neutrophils have a greater chlorinating potential than the monocyte, but the relative amounts generated by the two cell types are dependent on the particular stimulus employed.

Surprisingly, a comparison of the results obtained for chloramine generation by stimulated neutrophils in this study (Table 3) with our earlier report revealed a striking difference. While PMA-stimulated neutrophils generated comparable amounts of the chloramine in the two studies, the values reported for zymosan-triggered neutrophils are two times higher in the current report. Because the only major difference between these studies was the incubation vessel (i.e., flat-bottom wells in this study versus tubes in the first report), we directly examined this variable. In parallel experiments, 10⁶ PMA-stimulated neutrophils produced similar amounts of taurine chloramine whether incubated in tubes (61.8 ± 10.4 nmole, n = 5) or in wells (56.3 ± 5.4 nmole). However, the ability of zymosan-stimulated neutrophils to chlorinate taurine was markedly enhanced when the cells were incubated in wells (177.0 ± 19.2 nmole; n = 4) rather than in tubes (108.0 ± 17.4 nmole). Apparently, the plastic wells provide a superior environment for assessing the maximum chlorinating potential of the phagocyte. Although the reasons for this difference are unclear, adherent cells triggered with immune stimuli can release larger amounts of lysosomal enzymes than similarly treated cells in suspension.

Myeloperoxidase can use H₂O₂ to oxidize Cl⁻, Br⁻, or I⁻, but Cl⁻ is probably the preferred substrate in vivo, based on its relative concentration. Nonetheless, almost all studies attempting to assess the halogenating potential of phagocytes have used radiolabeled iodide, while only a single report used ¹²⁷Cl. Both of these assays are dependent on the incorporation of the radiolabeled halide into a stable covalent bond with a suitable acceptor molecule. Therefore, these techniques would not quantitate oxidized iodide or chloride products that were reduced back to free halides following their reaction with electron donors. For example, either oxidized halide would be reduced after reacting with thiols to form disulfides or thioethers to produce the respective sulfoxides. Because of these limitations, it is not surprising that ¹⁰² zymosan-stimulated neutrophils optimally incorporate ∼64 nmole of iodide/60 min into a protein target, while ¹⁰¹ neutrophils incubated with opsonized bacteria chlorinated only ∼3 nmole/60 min. Our results indicate that both monocytes and neutrophils have the ability to generate far larger amounts of oxyhalides than previously appreciated. But, are even these amounts biologically significant? Clearly, the answer is yes. Slivka et al. demonstrated that only 7.5 nmole of HOCl could destroy 10⁶ tumor cells, while Albrich and Hurst reported that as little as 1 nmole of HOCl could kill 4 × 10⁶ E. coli. These examples serve to highlight the powerful cytotoxic and microbicidal effects that intact phagocytes could exert by generating the quantities of HOCl reported in this study.

In conclusion, we have used a simple quantitative assay to demonstrate the ability of monocytes or neutrophils to form a chlorinating species with characteristics similar, if not identical, to HOCl. Although only those phagocytes that contain myeloperoxidase have been tested, it seems likely that other leukocyte populations will show similar activity. Eosinophil peroxidase can mediate chlorination reactions, and even peroxidase-negative macrophages may have access to these systems by ingesting senescent neutrophils or by utilizing extracellular myelo- or eosinophil peroxidase. This variety of leukocytes may have the potential to generate HOCl in vivo, either as a beneficial weapon in host defense or as a destructive mediator in inflammatory disease states.

REFERENCES

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The chlorinating potential of the human monocyte

MB Lampert and SJ Weiss