Sensitivity to SO$_2$ of Various Metabolic Processes in an Epiphytic Lichen, *Evernia mesomorpha*

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Key Term Index: sulphur dioxide effects, protein biosynthesis, photosynthetic CO$_2$-fixation, lipid biosynthesis, sulphur uptake; *Evernia mesomorpha*

**Summary**

In the epiphytic lichen *Evernia mesomorpha* Ny1., metabolic processes such as photosynthetic CO$_2$-fixation and protein and lipid biosyntheses were found to be very sensitive to SO$_2$. Exposure of lichens to 0.1 ppm of gaseous SO$_2$ for increasing durations produced a progressive reduction in these processes. Protein biosynthesis appeared to be the process most sensitive to SO$_2$. Fumigation of lichen tissues at 0.34 ppm of SO$_2$ for increasing durations caused an increased phytotoxic effect on all three metabolic processes. Such fumigations also inhibited acid phosphatase activity and caused an increase in the sulphur content of the tissues. During an SO$_2$-free period after the fumigations, these metabolic processes recovered partially or completely in lichens exposed to 0.1 ppm SO$_2$ but showed little or no recovery in lichens exposed to 0.34 ppm SO$_2$.

**Introduction**

Numerous lichen distribution and transplant studies around industrial developments or polluted areas have indicated a decline in the density and composition of species sensitive to SO$_2$, a major air pollutant in such areas. In view of this it was believed that SO$_2$ was responsible for the reduction in growth of sensitive lichens in such areas (Skye 1968; Gilbert 1973; Hawksworth 1973; Ferry and Coppins 1979); however, little work has been done at the metabolic level to determine the effects and mechanism of SO$_2$ phytotoxicity in lichens.

Rao and Le Blanc (1965) showed that there was a breakdown of algal chlorophyll to phaeophytin after exposure of lichen thalli to lethal doses of SO$_2$ (5 ppm for 24 h). Such exposures also caused plasmolysis of cells and produced brown spots on the chloroplasts of phycobiont cells. In vascular plants, exposure to aqueous SO$_2$ caused chlorophyll breakdown (Malhotra 1977) and alteration in chloroplast ultrastructure (Malhotra 1978). Aqueous solutions of SO$_2$ have also been used to demonstrate the effects of SO$_2$ on lichen photosynthetic CO$_2$-fixation and respiration (Hill 1971; Baddeley et al. 1972; Puckett et al. 1973); however, at present it is not known how the controlled exposure of lichens to low concentrations of gaseous SO$_2$ affects the above responses.

**Abbreviations:** TAG, triacyl glycerol; PVP-10, polyvinyl pyrrolidone; PC, phosphatidyl choline; PE, phosphatidyl ethanolamine; DPDG, diphosphatidyl diglycerol
In addition, little is known about the effect of gaseous SO₂ on the biosynthesis of proteins and lipids, two important membrane and cellular constituents.

On the basis of limited experimentation, O'Hare and Williams (1975) suggested that in certain lichens the degree of damage and sulphur accumulation was proportional to the rate of airflow. One may therefore expect that a low rate of airflow would limit the uptake of SO₂ from the atmosphere and that the small amounts of SO₂ thus absorbed would be quickly metabolized by lichen tissues into nontoxic forms. On the other hand, at the high flow rates that generally occur under field conditions, drying of lichen thalli would occur, thus limiting SO₂ absorption. In the field, the lichens absorb atmospheric SO₂ mainly during periods of high relative humidity. Hawksworth (1973) suggested that a strong correlation could be made between lichen distribution and average SO₂ concentration during periods of high relative humidity. Moist atmospheric conditions may thus result in longer metabolically active periods during which lichens can absorb SO₂ and suffer more damage. In the field, however, lichens experience both wetting and drying cycles, and little study has been done on the effect SO₂ has on lichen metabolism under such conditions.

The present paper describes for the first time the protein and lipid biosyntheses and the effect of gaseous SO₂ on photosynthetic CO₂-fixation and the 2 biosyntheses in the epiphytic lichen Evernia mesomorpha Nyl. Recovery of various metabolic processes in an SO₂-free atmosphere was also studied in lichen tissues initially exposed to gaseous SO₂. Such information is lacking and must be obtained in order to explain the metabolic tolerance of lichen species to air pollution stress.

In this study we also attempted to simulate some important field environmental conditions in order to monitor the effects of different concentrations of SO₂ on a number of metabolic processes in E. mesomorpha. This species was selected because it appeared to be one of the species that was most sensitive to air pollution in the Athabasca oil sands area of northeastern Alberta (Addison and Baker 1979).

**Materials and Methods**

*Chemicals*

[U-¹⁴C]-leucine (spec. act. 350 mCi/mmol) and Omnifluor were purchased from New England Nuclear Corporation, and [1-¹⁴C]-acetate (spec. act. 59 mCi/mmol) and [¹⁴C]-sodium bicarbonate (adjusted to spec. act. 150 µCi/mmol) were purchased from Amersham Searle Corporation. Other reagents were of the highest quality commercially available.

*Lichen collection*

Branches laden with epiphytic lichens were collected from jack pine (*Pinus banksiana* Lamb.) stands in a relatively pollutant-free area near Fort MacKay in northeastern Alberta. When collected in the field the lichen samples were almost dry; they were brought to the laboratory in the same condition in polyethylene bags and were stored at -15 °C in darkness until used. The lichen communities remained undisturbed during controlled fumigations in the laboratory. Only samples of *Evernia mesomorpha* Nyl. were harvested for analyses.

*SO₂ fumigation*

Small branches supporting *E. mesomorpha* were removed from cold storage and left overnight in a controlled environment chamber for preconditioning. The branches were divided into 2 groups:
1 group served as a control (untreated), and the other received SO$_2$ treatment. Both groups were moistened by a fine spray of distilled water at the start of the experiment. Branches supporting moistened lichen samples were mounted horizontally in cuvettes for both the control and the SO$_2$ samples. The cuvettes were placed in a controlled environment chamber and were maintained at 20 °C and 4,800-lx light intensity (16 h photoperiod). Humidified air (relative humidity 80—85%) was passed through the cuvettes at a flow rate of 101/min. The dimensions and operation of the cuvette have been described elsewhere (MALHOTRA and KUMAR 1978). The low light intensity and reduced airflow rate were used to prevent rapid drying of the lichen samples. The concentration of SO$_2$ inside the cuvette was maintained by a feedback controller and was continuously monitored by a Phillips PW 9700 SO$_2$ analyser. In the experiments studying recovery of metabolic functions in an SO$_2$-free atmosphere, the supply of SO$_2$ to the cuvette was discontinued after a specified time, and the system was flushed with clean air. The lichens were then allowed to recover in clean air. The lichen samples were sprayed with distilled water at 8.00 and 16.00 during the treatment and recovery periods. Representative samples of the lichen were harvested and divided into about 1 cm segments before being assayed for various metabolic functions.

For all metabolic experiments, lichen material (0.1 g) was brought to full saturation by an initial incubation in distilled water (3—5 ml) for 10 min at 30 °C.

**Protein biosynthesis**

Protein biosynthesis in the lichen tissues was measured by the incorporation of [U-$^{14}$C]-leucine into proteins. This was done by incubating the wet tissues in 5 ml of [U-$^{14}$C]-L-leucine (1.5 μCi) for 4 h at 30 °C with constant shaking under a 9,000-lx light intensity. The tissue was separated by filtration and was washed thoroughly with distilled water. The labeled tissue was then homogenized for 1 min in 10 ml of cold 0.1 M K-phosphate buffer (pH 7.0) using a Brinkman Polytron homogenizer (Model PT-10). Duplicate aliquots of each homogenate were mixed with trichloroacetic acid (final concentration 10%, v/v) to precipitate proteins. The suspension was mixed and allowed to stand in ice for several hours. It was then centrifuged at 4 °C, and the residue was washed twice with cold 10% (w/v) trichloroacetic acid and once with cold 80% (v/v) acetone. The washed residue was then suspended in 0.5 N NaOH and left for 10 min in a hot-water bath (60—70 °C) to solubilize the proteins. The insoluble material was removed by centrifugation, and an aliquot from the supernatant was assayed for [$^{14}$C] incorporation into proteins.

**Photosynthetic CO$_2$-fixation**

For photosynthetic CO$_2$-fixation, the lichen tissue was incubated in 3 ml of NaH$_{14}$CO$_3$ (3.0 μCi) for 2 h at 9,000-lx light intensity. The reaction was then stopped by adding 1 ml of formic acid (88%, v/v) and 16 ml of absolute ethanol. The contents were brought to a boil, cooled, and homogenized for 1 min with a Polytron homogenizer. The suspension was filtered through a Büchner funnel, and the residue was washed several times with hot 80% (v/v) ethanol. Aliquots of the filtrate were mixed with 0.1 ml of formic acid in scintillation counting vials and were flushed with a stream of N$_2$ in a hot-water bath to almost dryness. The residue was then dissolved in 0.1 ml of distilled water, and the fixed CO$_2$ was measured by counting [$^{14}$C].

**Lipid biosynthesis**

Lipid biosynthesis was measured by assaying the incorporation of [1-$^{14}$C]-acetate into lipids. For this, the samples were incubated with 5 ml of [1-$^{14}$C]-acetate (10 μCi) under 9,000-lx light intensity for 2 h at 30 °C with gentle shaking. The tissue then was removed by filtration, washed several times with distilled water, and homogenized for 1 min in 20 ml of chloroform: methanol (2: 1, v/v) using a Polytron homogenizer. The suspension was filtered through a Büchner funnel, and the residue was washed several times with chloroform: methanol (2: 1, v/v). From the combined filtrate, total lipids were extracted by the method described by FOLCH et al. (1957).

Thin-layer chromatography of the labeled lipids was done using 0.5 mm silica gel H plates. Phospholipids were separated by developing the plates in lined tanks containing chloroform: me-
thanol: acetic acid: water (100: 25: 10: 4, v/v). For TAG, the plates were developed in a hexane: ethyl ether: formic acid (40: 10: 1, v/v) solvent system (Malhotra and Khan 1978). Radioactive spots on the thin-layer chromatograms were identified by scanning the plates using a radioisotope scanner (Searle Actigraph III). These spots were matched with the standards, which were run beside or added to the sample. The various products in the chromatograms, including the standards, were visualized by exposure to iodine vapours. Silica gel from the radioactive regions was removed and counted for [14C].

Acid phosphatase (EC 3.1.3.2.)

Lichen tissue (0.1 to 0.2 g) was incubated with 5 ml of distilled water for 10 min at 30 °C. It was then mixed with 5 ml of a chilled solution of 0.1 M Tris-HCl buffer (pH 7.0) containing 0.5 mM dithioerythritol, 1 mM MgCl₂, and 2 % (w/v) PVP-10. The contents were homogenized at 4 °C with a Polytron homogenizer for 1 min, and the homogenate was filtered through two layers of cheesecloth. The filtrate was used immediately for assaying the enzyme activity (Malhotra and Khan 1980).

Measurement of radioactivity

The incorporation of [14C] was measured by mixing the [14C] products in a scintillation vial with a counting fluid (0.4 g Omnifluor dissolved in a mixture of 30 ml of absolute ethanol and 70 ml of toluene). Counting was done with a Nuclear Chicago Isocap/300 liquid scintillation spectrometer.

Other estimations

The dry weight of a sample was measured by oven-drying the fresh tissues at 70 °C for 24 h. For sulphur analysis, the dried sample was ground to a fine powder with a mortar and pestle, and a portion of the powder was used for oxygen flask combustion (Chen 1975). Total sulphur content of the combusted sample was determined by the method of Carson et al. (1972).

All experiments were repeated several times, and the trends were found to be highly reproducible. The data in the tables are taken from representative experiments. To compensate for biological variability on a day-to-day basis, an untreated control was analyzed simultaneously in each experiment and the results were expressed as a percent of the control. Mean values and the standard deviation for the controls (4—8 estimations) are provided in the tables.

Results and Discussion

De novo biosynthesis of proteins, lipids, and photosynthates in E. mesomorpha

Incubation of E. mesomorpha tissue segments in solutions of [14C] labeled compounds resulted in a de novo synthesis of proteins, lipids, and total photosynthates (Table 1). The rates of these processes were linear with respect to reaction time under the assay conditions.

[U-14C]-leucine was absorbed into the lichen tissues and incorporated into tissue proteins. About 10% of the absorbed label was incorporated into the proteins, while the remainder was in the form of free leucine.

The lichens incorporated [1-14C]-acetate into various lipid fractions. Radio thin-layer chromatography showed that a major portion of the label was present in the TAG fraction. Among the phospholipid fractions, the incorporation was highest in PC, followed by PE, and then DPDG. Occasionally, an unidentified radioactive peak representing incorporation almost equal to the DPDG fraction was found in the hexane: ethyl ether: formic acid (40: 10: 1, v/v) solvent system. This fraction was
Table 1. Biosynthesis of various metabolites in E. mesomorpha

<table>
<thead>
<tr>
<th>Metabolites</th>
<th>Incorporation(^1) (10^3 cpm/g dry wt. of tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proteins</td>
<td>19.3 ± 2.6(^2).</td>
</tr>
<tr>
<td>Lipids</td>
<td></td>
</tr>
<tr>
<td>PC</td>
<td>11.7 ± 0.9.</td>
</tr>
<tr>
<td>PE</td>
<td>7.1 ± 1.1.</td>
</tr>
<tr>
<td>DPDG</td>
<td>5.1 ± 0.7.</td>
</tr>
<tr>
<td>TAG</td>
<td>57.5 ± 6.5.</td>
</tr>
<tr>
<td>Photosynthates</td>
<td>15.6 ± 1.2.</td>
</tr>
</tbody>
</table>

\(^1\) Labeled precursors as listed in materials and methods.
\(^2\) Standard deviation.

located on the thin-layer plate just below the free fatty acid band. It has not been identified yet and is therefore not included in the table. Further analyses of these labeled lipid fractions after either alkaline hydrolysis or transmethylation showed that all the radioactivity was associated with the acyl moieties of the lipid fraction. These results indicate that E. mesomorpha contain a complement of the enzymes involved in the biosynthesis and acylation of fatty acyl moieties.

During photosynthesis, [\(^{14}\)C]-bicarbonate was also fixed by the lichen tissue into acid-stable and alcohol-soluble products. The relative distribution of the label into various photosynthates and the activity of various enzymes involved remains to be determined.

**Effects of SO\(_2\) on protein biosynthesis and photosynthetic CO\(_2\)-fixation**

Increasing lengths of exposure of E. mesomorpha to 0.1 ppm SO\(_2\) resulted in progressive reduction of protein biosynthesis and photosynthetic CO\(_2\)-fixation (Table 2, expt. 1). Protein biosynthesis appeared to be more sensitive to SO\(_2\) than did CO\(_2\)-fixation, especially after long exposures. It is important to note that even 2 days of exposure at such a low concentration resulted in about 15% inhibition in the rates of both processes. At a concentration of 0.34 ppm SO\(_2\), the inhibitory effects on these processes occurred much faster and were more pronounced than at 0.1 ppm SO\(_2\) (Table 2, expt. 2). Again, protein biosynthesis was inhibited more severely than photosynthetic CO\(_2\)-fixation. There was a considerable decline in the activity of both processes even after 2 h exposure time; 7 d of fumigation resulted in almost total inhibition of both processes.

The results in Table 2 also show the extent of recovery in the 2 metabolic processes when the fumigated lichens were transferred to an SO\(_2\)-free atmosphere. In lichens initially exposed to 0.1 ppm SO\(_2\) a partial recovery was observed in both processes during the SO\(_2\)-free period. There was, however, no appreciable recovery of these processes in lichens initially exposed to 0.34 ppm SO\(_2\) for 7 d (Table 2, expt. 2). This suggests that, under field conditions, if an exposure to low levels of SO\(_2\) is followed by an SO\(_2\)-free period lichens may recover at least partially from the SO\(_2\) stress, but
due to their high metabolic sensitivity an exposure to high concentrations could cause irreversible injury. This would lead to severe reduction in lichen growth and their eventual death.

In vascular species, SO$_3^-$ has been reported to cause a decrease in protein content (Malhotra and Khan 1982). Such an effect is possible due to either an inhibition in the de novo synthesis of the cellular proteins or an increase in the breakdown of existing proteins, or both. In E. mesomorpha exposed to SO$_2$, a marked reduction in the biosynthesis of proteins indicates that SO$_3^-$ inhibited at least the de novo synthesis of cellular proteins. Such a direct effect of SO$_3^-$ has not been previously demonstrated in either lichens or vascular species. Recently we also observed similar effects on vascular species (Khan and Malhotra, unpublished results).

Similarly, there have been no reports of a direct effect of gaseous SO$_2$ on the photosynthetic CO$_2$-fixation in lichens. Most of the earlier work in this area was with lichens treated with solutions of HSO$_3^-$, SO$_3^{2-}$, and SO$_2$. For example, Hill (1971) showed there was a reduction of photosynthetic CO$_2$-fixation in Usnea subfloridana treated with solutions of HSO$_3^-$ . The effect was highly pronounced at pH 4.0 and lower. Similar reductions in photosynthetic CO$_2$-fixation have been reported by others (Richardson and Puckett 1973; Hallgren and Huss 1975) following treatment of lichens with aqueous solutions of SO$_2$ or HSO$_3^-$.

Photosynthetic CO$_2$-fixation is catalyzed mostly by ribulose bisphosphate carboxylase (RuBPCase), and sulphite has been shown to inhibit the activity of partially purified (Khan and Malhotra 1982) or impure (Ziegler 1972) preparations of this enzyme by competing for HCO$_3^-$/CO$_2$ binding sites. If metabolic processes in lichens are more sensitive to SO$_2$ or SO$_3^{2-}$ than are those in vascular plants, one would expect that the RuBPCase activity of lichens would show lower $K_i$ for SO$_3^{2-}$. Ziegler (1977), however, found that RuBPCase preparations isolated from Pseudovernia furfuracea, a lichen very sensitive to SO$_2$ (Le Blanc and Rao 1975), had higher $K_i$ values for SO$_3^{2-}$ than did a

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**Table 2. Effects of SO$_2$ fumigation on protein biosynthesis and photosynthetic CO$_2$-fixation in E. mesomorpha and their recovery in an SO$_2$-free atmosphere**

<table>
<thead>
<tr>
<th>Exposure time</th>
<th>Protein biosynthesis (% of control)</th>
<th>Photosynthetic CO$_2$-fixation (% of control)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expt. 1 (0.1 ppm SO$_2$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 d</td>
<td>85.5</td>
<td>85.2</td>
</tr>
<tr>
<td>15 d</td>
<td>35.2</td>
<td>65.3</td>
</tr>
<tr>
<td>29 d</td>
<td>11.6</td>
<td>32.6</td>
</tr>
<tr>
<td>29 d + 8 d SO$_2$-free</td>
<td>38.6</td>
<td>62.2</td>
</tr>
<tr>
<td>Expt. 2 (0.34 ppm SO$_2$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 h</td>
<td>40.7</td>
<td>72.4</td>
</tr>
<tr>
<td>1 d</td>
<td>28.3</td>
<td>65.0</td>
</tr>
<tr>
<td>3 d</td>
<td>9.2</td>
<td>27.6</td>
</tr>
<tr>
<td>7 d</td>
<td>1.8</td>
<td>5.4</td>
</tr>
<tr>
<td>7 d + 7 d SO$_2$-free</td>
<td>3.6</td>
<td>16.5</td>
</tr>
</tbody>
</table>

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On d 3 biosynthesis of all lipids was inhibited by more than 50%, and on d 7 inhibition was 90 to 95%. In spite of such severe effects, there was some recovery in lipid biosynthetic capacity when the fumigated plants were transferred to an SO₂-free atmosphere for 7 d. The maximum recovery was in PC. These results suggest that various acylation reactions in E. mesomorpha have different sensitivities to SO₂. It is also possible that the biosynthesis of fatty acid chains, required for the acylation and synthesis of lipid species, was inhibited by SO₂. This would limit the availability of fatty acyl chains for acylation reactions in tissues and thus would inhibit the biosynthesis of various lipid species. Transmethylation or hydrolysis of the total lipids showed that reduced incorporation was due to a decrease in fatty acid synthesis. This indicates an inhibition of fatty acid synthetase activity.

Lipid biosynthesis in jack pine needles has also been shown to be markedly inhibited by low SO₂ concentrations that usually do not produce any visual symptoms of toxicity (MALHOTRA and KHAN 1978). Since lipids are essential constituents in the structure and function of cellular membranes, changes in their composition by SO₂ (KHAN and MALHOTRA 1977) were considered to be the primary reasons for adverse effects of SO₂ on pine needle cellular membranes (MALHOTRA 1976). Similar adverse effects could be anticipated in the membranes of E. mesomorpha as a result of SO₂ inhibition of lipid biosynthesis. Such alterations in the composition of lipids in the membranes would in turn affect important metabolic processes of the cellular membranes such as permeability, enzyme activity, and energy metabolism.

**Effects of SO₂ on acid phosphatase**

Treatment of E. mesomorpha with 0.34 ppm SO₂ for various lengths of time caused a decrease in lichen acid phosphatase activity, but the reduction was only 10% after 7 d of fumigation. The acid phosphatase activity of this lichen, therefore, appeared to be considerably less sensitive to SO₂ than were other cellular reactions. SCHMID and KREEB (1975) found that in another sensitive lichen species, Parnelia physodes, brief exposure to an unspecified low concentration of SO₂ neither affected the acid phosphatase activity nor caused any visual injury, but an unspecified high concentration produced marked reduction in the enzyme activity and discoloration of the thallus. There was also a wide scatter in the activity curve. In E. mesomorpha, a small decrease

<table>
<thead>
<tr>
<th>Exposure time</th>
<th>Sulphur uptake (% of control)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expt. 1 (0.1 ppm SO₂)</td>
<td></td>
</tr>
<tr>
<td>15 d</td>
<td>115.4</td>
</tr>
<tr>
<td>29 d</td>
<td>133.6</td>
</tr>
<tr>
<td>Expt. 2 (0.34 ppm SO₂)</td>
<td></td>
</tr>
<tr>
<td>1 d</td>
<td>132.5</td>
</tr>
<tr>
<td>7 d</td>
<td>178.0</td>
</tr>
</tbody>
</table>

1) Sulphur content of the control = 1.10 ± 0.05.
in the enzyme activity at a level that caused a marked reduction in other metabolic processes could be explained if SO₂ influenced the enzyme activity by differentially affecting the fungal and algal components of the lichen tissues. SO₂ may inhibit certain metabolic activity in one lichen component while stimulating activity in another component. In such a situation a composite assay involving the total tissues could be a misleading index of the tissue metabolic activity of individual components. If the metabolic processes in the algal cells are inhibited by SO₂ (which appeared to be the case, because photosynthetic CO₂-fixation was severely reduced), their sensitive symbiotic relationship with the fungal component would be disturbed.

**Sulphur uptake of SO₂-fumigated lichens**

Sulphur content of the lichen tissues increased appreciably upon fumigation at both levels of gaseous SO₂ (Table 4). The uptake was greater in lichens exposed to 0.34 ppm SO₂ for shorter periods than in lichens exposed to 0.1 ppm SO₂ for a longer period. This suggests that the metabolic turnover of the absorbed SO₂ was higher in the lichens exposed to a low SO₂ concentration for a longer duration. The fact that there was increased metabolic inhibition in treatments (Tables 2 and 3) involving exposure to high concentrations of SO₂ for shorter periods (Tables 2 and 3) supports this suggestion. It therefore appears that metabolic injuries are related to accumulation of absorbed SO₂ in the tissues.

**Conclusions**

The results of this study clearly indicate that 0.1 ppm of gaseous SO₂ can produce deleterious effects on lichen metabolism, but that such effects could be reversed in an SO₂-free atmosphere. The lichens appear to have an excellent ability to detoxify low levels of SO₄ (Turk et al. 1974). At 0.34 ppm of SO₂, however, the injury to various metabolic processes can be much more severe and not fully recoverable. Thus exposure of lichens to relatively high SO₄ concentrations in moist field conditions for even a short time can be expected to cause severe phytotoxic effects.

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