Fungitoxic Action of a Copper-Chromium-Arsenate Wood Preservative

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The technical assistance of R. K. White and D. Brain is gratefully acknowledged.

This research was supported by Hickson and Welch (Holdings) Ltd, Castleford, England.

Accepted for publication 13 September 1973.

ABSTRACT

The interactions between wood treated with copper-chromium-arsenate (CCA) and several wood-destroying fungi were studied to obtain a clearer understanding of the in vivo mode of action of the preservative. The fungi varied considerably in their tolerance to CCA. Subtoxic amounts of preservative had little effect on colonization of the treated wood. Toxic amounts drastically reduced colonization except with *Poria monticola*. Copper, chromium, and/or arsenic were solubilized by all the fungi at both subtoxic and toxic levels. All the fungi except *P. monticola* remained viable in veneers treated with toxic amounts of preservative. Microscopic examination of treated wood showed that many of the hyphae were dead. *P. monticola* absorbed copper, chromium, and arsenic from the secondary walls of tracheids into the hyphae. Absorption of solubilized fungitoxicants into hyphae is probably the primary mode of action of the CCA although there was evidence that inhibition of cellulose decomposing systems also may play a role. The contradictory behavior of *P. monticola* in colonization and viability tests may be due to differences in the rates of absorption of copper and arsenic by the fungus.

Phytopathology 64:335-341

Additional key words: Analytical electron microscope, brown-rot fungi, white-rot fungus.

Copper-chromium-arsenate (CCA) is one of the most successful wood preservatives developed in recent years. The first CCA was patented by Kamesan in 1933 (British Patent 404,855) based on an idea of Bruning that chromium has the ability to fix water soluble fungicides in wood (British Patent 2972). Since then several commercial formulations have been developed which differ in the proportions (as well as types) of copper, chromium and arsenic compounds used. An important feature of these preservatives is the ability of the water-soluble chemicals to react with wood so that when it has been dried the chemicals are no longer water-soluble. This "fixation" of the preservative in the wood is essential if treated wood is to remain durable in moist environments.

The chemistry of fixation is complex and not fully understood. It is thought that on contact with the wood there is a rapid increase in pH of the CCA as a result of ion-exchange and absorption reactions with the wood. Some of the copper reacts with carboxyl groups in the wood by ion-exchange and then a series of insoluble complexes are formed between copper, chromium, arsenic and the wood. The final products are basic copper arsenate, tertiary chrome arsenate and chromic acid, although basic chromic chromates persist for a long time (9). The resultant CCA complex is distributed through the cell walls and also forms a protective layer on the lumen walls (3).

Practically all the information available on how fungi affect, and are affected by CCA-treated wood concerns the amount of preservative required to prevent decay. In vitro studies have demonstrated the toxicity of soluble copper, chromium, and arsenical salts. These studies have led to the general assumption that the fungitoxic action of copper, arsenic, and to a lesser extent chromium, is responsible for the effectiveness of the preservative. Questions as to (i) how the highly insoluble preservative is mobilized, (ii) whether or not the CCA or portions of it are absorbed or detoxified by some or all wood-destroying fungi, and (iii) whether or not substrate protection or cellulase inhibition play a part in the mode of action of the preservative, have all largely been ignored.

Levi (15) has shown that highly fixed CCA can be extracted from treated wood by culture filtrates of several wood-destroying fungi grown on glucose-peptone media. This suggests that fungal solubilization of CCA is the first stage of toxic action. To confirm this hypothesis it is necessary to show that the CCA can be solubilized by the activity of fungi in CCA-treated wood.

The purpose of this paper is to answer some of the questions outlined above and to determine the effect of CCA on (i) the fungal colonization of wood, (ii) the viability of fungi in treated wood, and (iii) the fine structure of fungi, as well as the effects of the fungi on CCA.

MATERIALS AND METHODS. – The CCA used was Tanalith C (RTM Hickson's Timber Impregnation Co., Castleford, England) containing $K_2Cr_2O_7$, 45%; $CuSO_4 \cdot 5H_2O$, 35%; and $As_2O_5 \cdot 2H_2O$, 20%. The toxicity of a given retention of CCA in wood is dependent upon the species and strain of wood-destroying fungus, the length of exposure of the treated wood to the fungus, and the size of the exposed block in relation to the amount of fungus

inoculum (15). Thus, in the following experiments toxicity of CCA was determined whenever any of the above variables were changed. The toxic retention of CCA was determined as follows. A vacuum was drawn over weighed, oven-dried, sapwood veneers of *Pinus sylvestris* 2.0 X 2.0 X 0.2 cm. The veneers were immersed in a range of concentrations of CCA in water, whilst still under a vacuum. The vacuum was then released and the veneers allowed to soak for 2 h at atmospheric pressure. This treatment ensured complete penetration of preservative throughout the veneers. The amount of preservative in each veneer was calculated by weighing it immediately after removal from the treatment solution. The veneers were air-dried in the laboratory for at least ten days, sterilized in an autoclave at 1.05 kg/cm^2 for 30 min, and then in sets of five, were placed in petri dishes containing about 15 ml Oxoid malt agar previously inoculated and overgrown with one of the wood-decomposing fungi listed in Table 1.

The veneers were supported on glass rods so that they were not in contact with the agar. The exposed veneers were maintained at 25 C ± 1 C in a covered glass tank filled with vermiculite saturated with water. At the end of the test period, the veneers were removed, surface mycelium wiped off, the veneers oven-dried to constant weight, and the weight loss determined. The toxic limit was taken as the range between the highest retention of preservative which allowed measurable loss of weight and the lowest retention which prevented such weight loss.

Colonization of CCA-treated wood. – The extents of fungal growth in untreated veneers and veneers treated with toxic and subtoxic amounts of CCA were compared after exposure to fungi for 2.5 wk, by examining radial longitudinal sections of wood 20μm thick under phase contrast microscopy. At least 500 tracheid segments from nine different sections were examined for each treatment. Fungus colonization was expressed as the percentage of tracheid segments examined which contained hyphae.

Viability of fungi in CCA-treated wood. – CCA-treated and untreated veneers were exposed to fungal attack as already described. After two wk the colonized veneers were transferred for two additional wk to sterilized petri dishes containing water, and supporting glass rods. The veneers were then plated out on 5% malt agar. Five to ten veneers were used for each treatment. Viability was calculated as the percentage of veneers from which the fungus grew onto the malt-agar plates. The purpose of transfer to plates containing water was to ensure that all hyphae in or on the treated wood were exposed for at least two wk to the action of the CCA.

TABLE 1. Toxic limits of copper-chromium-arsenate (CCA) wood preservative for various wood-destroying fungi

Fungus species and isolate designation	Toxic limits (kg/m^3)
Poria monticola Murr. (Hicksons 12E)	11.2-40.0
Poria vaillantii (Fr.) Cke. (FPRL 14B)	> 40.0
Poria vaillantii (Fr.) Cke. (Hicksons 16C)	7.0-11.2
Coniophora cerebella (Schum.ex Fr.) (FPRL 11E)	≅ 5.9
Lentinus lepideus Fr. (FPRL 7B)	< 1.0
Gleophyllum trabeum (Pers. ex Fr.) Murr. (FPRL 108B)	1.0- 2.4
Polyporus versicolor L. ex Fr. (FPRL 28)	< 1.0

Electron microscope examination of fungus fine structure. – Untreated and CCA-treated veneers exposed to *Poria vaillantii* for different time periods were fixed in 6.5% glutaraldehyde in 0.1 M Sorensen's buffer at pH 7.16 for 15 h at 4C followed by three washings of 30 min each, with 0.2 M sucrose phosphate. They were postfixed in unbuffered 1% osmium tetroxide for 1 h at 4C and embedded in methacrylate/styrene (70% *n*-butyl-methacrylate, 30% styrene) (4). This method sometimes caused slight plasmolysis. For veneers exposed to *P. monticola* the fixation procedure was altered slightly. Veneers were fixed in 3% glutaraldehyde in 0.1 M Sorensen's buffer at pH 7.16 for 4 h at 4C and they were postfixed in 2% osmium tetroxide for 1 h at 4C. Other parts of the fixation procedure were the same as for *P. vaillantii*. Ultrathin sections were cut on an LKB Ultratome III, stained with uranyl acetate (1% for 20 min) followed by Reynold's lead citrate (5 min) and examined at 60 kV in Philips 200 electron microscope.

Solubilization of CCA by wood-destroying fungi. – The effect of fungal growth on the solubility of CCA was determined by analyzing treated veneers with and without extraction after exposure to decay fungi for 4 wk. After exposure, the veneers were dried and ground to pass a 1.5-mm-mesh sieve. Half the material was extracted with water at room temp for 16 h. The amounts of copper, chromium, and arsenic present in the extracted and unextracted wood was determined by X-ray analysis (1).

Fungal uptake of CCA. – The concentrations of copper, chromium, and arsenic in CCA-treated tracheid walls after attack by *P. monticola*, and in lumen and bore-hole hyphae of *P. monticola* in treated wood were determined using the AEI analytical electron microscope EMMA-4 (2). The method of quantitative analysis followed that developed by Hall (11,12, 13). Details of the method of analysis will be published elsewhere (3).

RESULTS. – *Effect of CCA on fungi.* – The fungi tested varied considerably in their tolerance to CCA (Table 1). *P. vaillantii* 14B was more than 40 times more tolerant than *Lentinus lepideus* and *Polyporus versicolor*. Not only were there large species differences, but also differences between the two isolates of *P. vaillantii* tested.

Colonization of CCA-treated wood. – The percentage of tracheids in nontreated wood colonized after 2.5 wk exposure to fungi ranged from 9% for *L. lepideus* to 54% for *Gleophyllum trabeum* (Table 2). The presence of subtoxic amounts of CCA in the

TABLE 2. Effect of copper-chromium-arsenate (CCA) wood preservative on the colonization of wood and the viability of hyphae of wood-destroying fungi in wood

Fungus (Isolate No.)	Preservative retention (kg/m^3)	Hyphal Population[a] after 2.5 wk	Surface colonization after 2.5 wk	Viability[b]
Poria monticola	untreated	38	moderate	100
	11.2	57	moderate	100
	40.0[c]	29	abundant	0
Poria vaillantii 14B	untreated	41	moderate	100
	11.2	41	moderate	100
	40.0	-	moderate	100
Poria vaillantii 16C	untreated	37	abundant	100
	5.9	37	sparse	100
	11.2[c]	4	sparse	100
Coniophora cerebella	untreated	-	-	100
	11.2[c]	-	-	40
Lentinus lepideus	untreated	9	abundant	100
	1.0[c]	-	-	100
	5.9[c]	1	negligible	60
Gleophyllum trabeum	untreated	54	moderate	100
	5.9[c]	6	sparse	100
Polyporus versicolor	untreated	23	moderate	100
	5.9[c]	4	very sparse	-
	11.2[c]	-	-	60

[a]Hyphal population is the percentage of tracheid segments examined which contained hyphae.
[b]Viability is the percentage of veneers from which the fungus grew into malt-agar.
[c]Toxic retention of preservative.

wood had little effect on colonization by *P. monticola, P. vaillantii* 14B and 16C, the only fungi examined at such levels. Toxic amounts of preservative, however, decreased colonization of tracheids by all the fungi tested. Similarly, growth of hyphae on the surface of exposed veneers diminished considerably in the presence of toxic amounts of CCA except with *P. monticola* where surface growth was greatest at the toxic amount of CCA.

The hyphae present in wood treated with toxic amounts of CCA were composed largely of highly vacuolated cells containing coagulated cytoplasm. This suggested that most of the cells were dead. This contrasted sharply with the appearance of hyphae in untreated wood, in which all the cells contained dense granular cytoplasm, regarded as a sign of vigorously growing cells.

Viability of fungi in CCA-treated wood. – The only fungus which completely failed to grow out of veneers treated with toxic amounts of CCA was *P. monticola.* This was surprising, because hyphal growth in tracheids and on the surface of veneers treated with toxic amounts of CCA was greater for *P. monticola* than for any of the other fungi tested. All the other fungi grew from veneers treated with toxic amounts of CCA, although with *Coniophora cerebella, L. lepideus,* and *P. versicolor,* most of the cells were either killed or inhibited because no hyphae grew from the veneers in 60, 40, and 40% of the veneers tested, respectively.

Ultrastructure of P. monticola and P. vaillantii 16C in CCA-treated wood. – Untreated veneers and veneers treated with subtoxic amounts of CCA were examined after *P. monticola* and *P. vaillantii* 16C had caused weight losses of 15-20%. Hyphae were also examined in wood treated with toxic amounts of CCA and exposed to *P. monticola.*

In sections of untreated wood, most hyphal cells were typical of actively growing plant cells. They were bounded by an intact plasmalemma with a characteristic three-layered structure (Fig. 1A). Cell contents included endoplasmic reticulum, mitochondria, golgi apparatus, microtubules, and lomasomes (Fig. 1A). Some of the cell organelles were not readily identifiable. *P. monticola* cells frequently contained unit membrane vesicles about 0.2 - 0.6 μm in diam with a matrix void of ribosomes and apparently different from the ground plasm. Inside the vesicles were numerous smaller vesicles which may have arisen from invagination of the vesicular membrane.

Wood treated with subtoxic amounts of CCA contained some normal hyphal cells in addition to structurally disorganized cells (Fig. 1B). All hyphal cells examined in wood treated with toxic amounts of CCA were badly disrupted (Fig. 1C). Cell disorganization was characterized by plasmolysis (Fig. 1D), disintegration of the plasmalemma (Fig. 1C), coagulation of cytoplasm (Fig. 1B), and loss of cell organelles; lysis of cells was indicated by recognizable cell debris (Fig. 1D).

Examination of bore-hole hyphae confirmed the sharp contrasts in structure of fungal cells in treated and nontreated veneers. Bore-hole hyphae in untreated wood were structurally intact with a full complement of organelles (Fig. 1E), whereas those in treated wood were plasmolyzed and almost devoid of cell contents (Fig. 1F). Another difference associated with bore-hole hyphae of *P. vaillantii* in treated and untreated wood was the ultrastructure of the tracheid

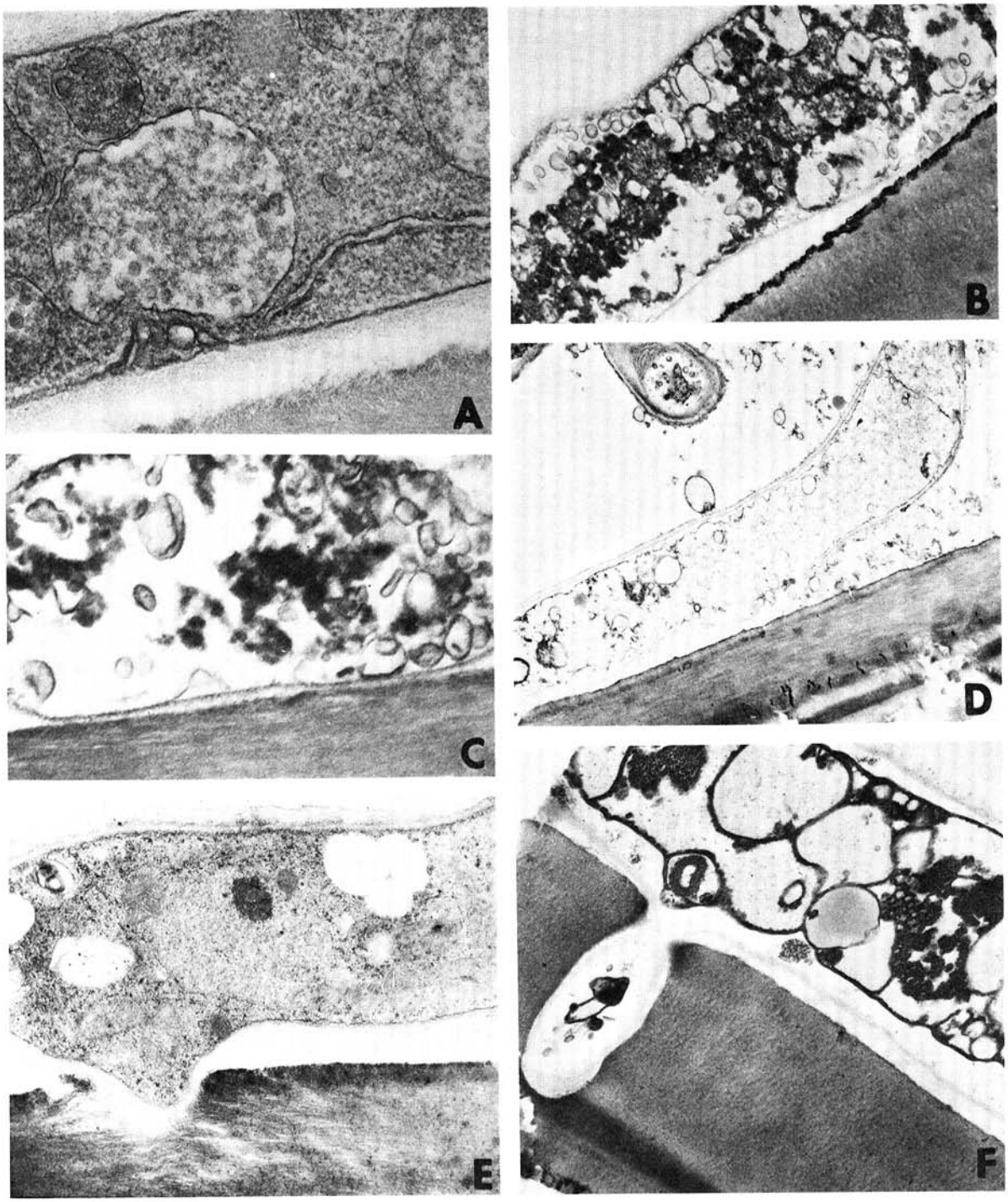

Fig. 1–(A to F). A) Longitudinal section of hypha of *Poria monticola* in untreated pine exposed to attack for 3.5 wk, showing organelles characteristic of actively growing normal hyphae (× 47,500). **B)** Longitudinal section of a hypha of *Poria monticola* in pine treated with a subtoxic amount of copper-chromium-arsenate wood preservative (CCA) and decayed to a weight loss of 10%, showing considerable disorganization of cell organelles. Note also, layer of CCA on tracheid lumen wall (× 15,000). **C)** Longitudinal section of a hypha of *Poria monticola* in pine treated with a toxic amount of CCA showing disintegration of plasmalemma and other cell organelles (× 47,500). **D)** Longitudinal section of hyphae of *Poria vaillantii* (Isolate 16C) in pine treated with a subtoxic loading of CCA and decayed to a weight loss of 20% showing distinct signs of wall degradation, lysis of hyphal cells and plasmolysis (× 7,200). **E)** Longitudinal section of hypha of *Poria vaillantii* (Isolate 16C) in untreated pine exposed for 5 wk showing organelles characteristic of normal hyphae, bore-hole formation, and accompanying dissolution of tracheid wall structure (× 15,000). **F)** Longitudinal section of hypha of *Poria vaillantii* (Isolate 16C) in pine treated with a subtoxic amount of CCA and decayed to a weight loss of 20% showing plasmolysed cells almost devoid of recognizable organelles, bore-hole formation, and an absence of any sign of tracheid wall dissolution around the bore hole (× 17,700).

wall. In untreated wood, the wall showed clear signs of dissolution around the bore-hole hyphae. In treated wood, there was no evidence of cell-wall dissolution.

Effect of fungi on CCA. – Fungal solubilization of CCA. – In the presence of subtoxic amounts of preservative, all the fungi tested were able to dissolve considerable quantities of copper, chromium, or arsenic (Table 3). *P. monticola* and *P. vaillantii* 14B dissolved over half the CCA in the wood. When the wood contained toxic amounts of preservative, copper, chromium, or arsenic were still dissolved by all the fungi, in spite of the fact that few hyphae were present in the wood and the wood was not decayed. However, less preservative was solubilized than in wood containing subtoxic amounts (Table 3).

The test fungi differed considerably in the extent to which they solubilized copper, chromium, and arsenic. There was no relationship between the amounts of each element solubilized by the fungi. Thus *P. monticola* dissolved copper, chromium, and arsenic, *P. vaillantii* dissolved chromium and arsenic, and *C. cerebella* dissolved copper and arsenic.

Fungal uptake of CCA.–Analytical electron microscopy showed that after 3.5-wk exposure in veneers treated with a subtoxic amount of CCA (4.3 kg/m^3), hyphal cells of *P. monticola* contained copper, chromium, and arsenic (Table 4). The total quantity of metal ions absorbed was approximately 4% of the dry wt of the mycelium. The concentration of the three elements varied greatly from hypha to hypha, as evidenced by the high coefficients of variability. This may have been due to differences in age of the hyphae, or to differences in the binding capacities of the various cell sites for copper, chromium, and arsenic.

In analyzing the preservative it was possible to determine two elements simultaneously for a single location. Thus arsenic and chromium were analyzed together and copper and chromium were analyzed together. The coefficients of variation were so large that it was difficult to draw exact conclusions about the relative proportions of the various elements in the hyphae. The approximate ratio of the elements was 1:1:1, however.

Analysis of hyphae in the lumina of wood cells containing subtoxic amounts of CCA (13.0 kg/m^3) exposed for 2.5 wk showed a completely different situation (Table 5). Hyphae contained considerably more copper than arsenic, the ratio being approximately 10:1. Hyphae in bore-holes also contained considerably more copper than arsenic. The amounts of copper and arsenic in the bore-hole hyphae were considerably less than in the lumen hyphae. Chromium was not analyzed in the hyphae examined after 2.5-wk exposure.

Analysis of tracheid walls adjacent to hyphae of *P. monticola* showed that copper and chromium were absorbed from the S2 layer of the walls (Table 6). Previous work has shown that copper and chromium are always present in the S2 layer of CCA-treated wood (3, 16). After exposure to *P. monticola*, however, no copper or chromium were present in several of the walls close to hyphae. Arsenic content of the tracheid walls was not determined.

DISCUSSION. – The wood-destroying fungi examined in this study varied considerably in their tolerance to CCA. The *Poria* spp. were the most tolerant, as has been demonstrated by other investigators (5, 7, 18). Variation in preservative tolerance among strains were also confirmed (5, 6, 10, 17).

Despite the variations in tolerance of the different fungi to CCA, a general understanding of the in vivo mode of action of the preservative can be constructed.

Subtoxic amounts of CCA had little effect on the colonization of treated wood. Many hyphal cells in

TABLE 3. Dissolution of copper, chromium, and arsenic by wood-destroying fungi growing for 4 wk in wood treated with subtoxic and toxic amounts of copper-chromium-arsenate (CCA) wood preservative

	Amount (%) of water-soluble element in wood treated with					
	Subtoxic amounts of CCA			toxic amounts of CCA		
Fungal agent	Cu	Cr	As	Cu	Cr	As
Unattacked control	18	0	0	0	0	0
Poria monticola	93	48	65	22	0	0
Poria vaillantii 14B	60	55	75	-	-	-
Poria vaillantii 16C	0	30	50	-	-	-
Gleophyllum trabeum	45	21	35	53	8	20
Coniophora cerebella	31	0	25	10	0	10
Lentinus lepideus	-	-	-	0	20	0

TABLE 4. Concentrations (% w/w) of copper, chromium, and arsenic in cells of *Poria monticola* colonizing veneers impregnated with subtoxic loading of copper-chromium-arsenate (CCA) wood preservative (4.3 kg/m^3) after 3.5-wk exposure to the fungus

	Concentration[a] (%, w/w)			
Element	Max.	Min.	Average	Coefficient of Variation
Arsenic[b]	4.3	0.05	1.45 ± 1.19	82
Chromium[b]	3.0	0.44	1.60 ± 0.77	47
Copper[c]	1.4	0.44	0.88 ± 0.31	38
Chromium[c]	2.7	0.26	1.14 ± 0.92	81

[a]Thirty randomly selected locations of approx. 0.2μm diam were analyzed for each element. Two elements at each location were determined simultaneously.

[b]Arsenic and chromium analyzed simultaneously.

[c]Copper and chromium analyzed simultaneously.

TABLE 5. Concentrations (%, w/w) of copper and arsenic after 2.5 wk in lumen and bore-hole hyphae of *Poria monticola* colonizing veneers impregnated with subtoxic amounts (13.0 kg/m³) of copper-chromium-arsenate (CCA) wood preservative.

	Concentration (%, w/w)							
	In lumen hyphae				In bore-hole hyphae			
Element	Max.	Min.	Average	Coefficient of variation	Max.	Min.	Average	Coefficient of variation
Arsenic	0.78	0.02	0.25 ± 0.23	95	0.06	0	0.03 ± 0.03	100
Copper	6.07	0.55	3.02 ± 2.03	67	2.00	1.61	1.71 ± 0.17	10

TABLE 6. Concentration (%, w/w) of copper and chromium in tracheids containing 4.3 kg/m³ of copper-chromium-arsenate(CCA) wood preservative exposed to *Poria monticola* for 3.5 wk

	Concentration (%, w/w) in S_2 layer of wall adjacent to hypha			
Element	Max.	Min.	Average	Coefficient of variability
Copper	0.40	0	0.20 ± 0.12	60
Chromium	0.29	0	0.12 ± 0.06	50

the treated wood were killed quickly and the rate of decay of the wood was decreased by the presence of preservative. Death of some cells in the presence of subtoxic amounts of preservative was probably due to solubilization of copper, chromium, and arsenic. With *P. monticola,* the solubilized copper, chromium, and arsenic were removed from the S2 layer of tracheids into the hyphae.

At toxic amounts of CCA, colonization of treated wood was considerably reduced, except with *P. monticola.* In this case numerous hyphae were observed in wood treated with toxic amounts of CCA. Some hyphal cells of all the fungi except *P. monticola* survived exposure to toxic amounts of CCA despite the facts that colonization was reduced, all hyphae appeared dead in the treated wood, and there was no wt loss in the wood. The failure of any hyphae to grow from some of the veneers treated with toxic amounts of CCA, and the absence of living cells in microscopical examination of wood, both suggest that only a small number of preservative-resistant cells were present in the wood. Examination of such cells possibly could give additional clues as to the nature of preservative tolerance in wood-destroying fungi.

The failure of *P. monticola* to grow out of veneers treated with toxic amounts of CCA was surprising in view of the fact that surface and internal colonization of the veneers was extensive. *P. monticola* is tolerant to copper but highly sensitive to arsenic (15). Analysis of hyphae in wood exposed to CCA for 2.5 wk showed that little arsenic was present in the hyphae whereas large amounts of copper were absorbed. After 3.5 wk the amounts of copper and arsenic in the hyphae were similar. Thus, differences in the rates of absorption of non-toxic copper and highly toxic arsenic may explain the presence of numerous, but non-viable, cells of *P. monticola.*

The protective action of CCA may be due not only to its fungistatic and fungicidal properties, but also to its ability to inhibit the decomposition of cellulose by cellulase or other catalytic systems such as peroxides (14). It has been suggested (4) that breakdown of the wall by bore-hole hyphae is the main pathway for decomposition of the cell wall by brown-rot fungi. Inhibition of catalyst action or production by bore-hole hyphae would effectively prevent decay. Wood cell walls surrounding bore-hole hyphae of *P. vaillantii* in CCA-treated wood showed no sign of dissolution, in sharp contrast to those in untreated wood. Thus, prevention of decay in CCA-treated wood may be due in part to inhibition of action or production of cell-wall dissolving catalysts. Levi (*unpublished*) has shown that CCA partially inhibits the decomposition of wood cellulose by cellulases. Further work is necessary to verify this hypothesis, however.

DaCosta and Osborne (8) have shown that decay occurred in wood treated with toxic amounts of CCA after exposure to wood-destroying fungi, both before and after extraction with water. The present study has demonstrated that this is almost certainly due to solubilization of CCA during the first exposure period and subsequent removal of the CCA during water extraction.

The practical significance of fungal solubilization of toxic amounts of CCA by micro-organisms cannot be estimated since insufficient data is available on solubilization of CCA in treated wood in service. It is well established that CCA performs well in ground contact but further work should be undertaken to determine the effects of fungi (and bacteria) on CCA in wood in contact with the ground.

Much thought has been given to the problem of preservative tolerance. Levi (15) found that *P. monticola* grown on a synthetic medium containing copper sulphate was able to precipitate the relatively nontoxic copper oxalate. *P. vaillantii* precipitated the copper in another form, later shown to be copper sulfide (Chou, *unpublished*). It was suggested that precipitation of the oxalate and sulfide at least partially explained the copper tolerance of these *Poria* spp. In the present study, no microscopic evidence was found of oxalate or sulfide production

by *Poria* spp. in CCA-treated wood. These phenomena therefore may be important in synthetic media, but not in wood treated with CCA. This demonstrates the dangers of predicting in vivo behavior on the basis of in vitro experiments.

Many questions remain unanswered concerning the mode of action of CCA. More information is needed on the nature of fungal metabolites which solubilize CCA, the nature of the toxicants released from the CCA which affect hyphal cells, and the effect of wood species on release of CCA. With such information for CCA and also for other wood preservatives, we will be better able to predict their performance in service.

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