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BRONZE DISEASE : AN ELECTROCHEMICAL EXPLANATION

INTRODUCTION

A survey of the literature on the conservation of metals shows that although many papers describe 'Bronze Disease' (Chase, Hamilton, Lucey, Organ, Plenderleith, Sease) there is often a low level of understanding among many conservators regarding the fundamental chemistry behind this problem of accelerated corrosion. The following paper discusses the physical and chemical nature of the processes involved in 'Bronze Disease' and will hopefully serve to direct workers along appropriate avenues of research towards solutions to the problem.

Bronze disease can affect copper based metal objects ranging from pure copper through a wide range of brasses (alloys with zinc and other minor elements) and bronzes (alloys with tin and other minor elements). Because of the nature of the corrosion in many bronzes and brasses, a series of micropits can develop under specific conditions which lay the foundation for further active corrosion. The corrosion/patination of bronzes and brasses is dominated by the chemistry of the major alloying element, copper.

PATINA FORMATION

In moist ambient temperature air, copper will react with oxygen, an oxidant, to form a layer of cuprous oxide, Cu_2O (Mills). The speed of this reaction varies according to the previous environmental history of the object and depends markedly on temperature and relative humidity. Although the initial corrosion rate may be rapid, the cuprous oxide forms a coherent film over the underlying metal which effectively isolates it from the external environment. The object becomes covered with the familiar brown patina of bronzes, and the final corrosion rate is extremely slow.

Patination involves the transfer of electrons from the metal to the substance acting as the oxidant which in this case is oxygen. This can be represented by the following equations:

$2Cu \rightarrow 2Cu^{+} + 2e^{-}$ …….oxidation: anodic process ..... (1)

$\frac{1}{2}O_2 + 2e^{-} \rightarrow O^{2-}$ …….. reduction: cathodic process ..... (2)

which gives to overall reaction as

$$2Cu + \frac{1}{2}O_2 \rightarrow Cu_2O \quad \text{..... (3)}$$

The cuprous oxide layer is coherent and conforms to the original contours of the object. The rate of the oxidation reaction is controlled by the diffusion of copper through the Cu_2O layer. Copper oxide is a semiconductor of moderate electrical resistance and this plays an important role in bronze disease, as will be discussed later in this paper. The copper(I) oxide may be subsequently oxidized to form copper (II) compounds, which are characteristically blue-green. Basic copper nitrates, sulphates and carbonates are normally non-injurious to the underlying metal and are the end products of the combined effects of air, water, carbon dioxide and pollutants such as nitrogen and sulphur oxides on the corrosion of copper and its alloys. Such patinas once formed are stable for centuries.

EFFECTS OF CHLORIDE IONS

The normally protective film of cuprous oxide may fail in the presence of chloride ions because the chloride ion not only affects the relative stability of both oxidation states of the metal, it also changes the

speed of many of the electrode processes which are intimately involved with the corrosion/patination cycle. The source of chloride ions includes ground water, environmental sources such as aerosols and biological sources such as sweat from humans. In many cases, the addition of chloride ions to the environment may only result in the appearance of uniform blue-green copper (II) hydroxy chloride patina on the object, but bronze disease is suspected when localized outbreaks of mounds of the basic copper chlorides atacamite and paratacamite (α and δ – $Cu_2(OH)_3C1$, respectively) occur. Under the combined effects of ionic diffusion and electrical migration, chloride ions can penetrate the normally protective film of Cu_2O and react with the underlying metal to form a layer of cuprous chloride.

Bronze disease is a form of pitting corrosion where the inner region under the cuprite film is actively dissolving (anodic area) and the corrosion products are deposited in a mound (main cathodic area) above the Cu_2O film. The protective Cu_2O cannot reform on the metal in the pit because of limited access to oxygen. This may be due to the corrosion product mount or simply to adherent sand grains, etc. The conditions for pit growth include the presence of a layer of cuprous chloride under the cuprous oxide. The Cu_2O layer functions as a bipolar electrode viz. oxidation and reduction reactions occur on opposite sides. The establishment of the cuprous chloride (CuCl) layer will depend on the relative rates of copper corrosion

$$Cu + C1^- \rightarrow CuC1 + e^- \quad (4)$$

and the hydrolysis of cuprous chloride

$$2CuC1 + H_2O \rightarrow Cu_2O + 2H^+ + 2C1^- \quad (5)$$

The formation of Cu_2O according to equation (5) will be controlled by the pH and activity of the chloride ions. If the pit pH drops below its equilibrium value – pH 5.3 in sea water (Bianchi. 1973) – cuprous chloride will be the more stable phase.

The overall driving force in bronze disease is the oxidation of copper by molecular oxygen (see equation 3) and the effect of chloride ions on the kinetics of the pitting reaction is to help provide a concentration gradient of copper (I) species between the bottom of the pit and the corrosion mound above the Cu_2O film. In the absence of oxygen, the corrosion cell should, in time, stifle itself since the build up of copper(I) species on the surface of the object would remove the concentration gradient between the pit and the corrosion product mound. In the presence of chloride ions the normally insoluble CuCl can further react to form a series of soluble complexes such as $CuC1_2^-$, $CuC1_3^{2-}$ in the pit. High concentrations of chloride ions can arise because of (a) isolation from the immediate environment by the cuprite layer (b) the corrosion mound preventing ready exchange and (c) evaporative concentration. However, for buried and submersed objects evaporative concentration is not significant.

The increased concentration of chloride ions in the pits is due primarily to migration of anions from the cathodic to the anodic areas to satisfy charge neutrality. As a result of their higher concentration, the chloride rather than hydroxide ions tend to migrate into the pits. Because the corrosion of bronzes and brasses involves not only the oxidation of copper metal but also the oxidation of tin and zinc under the Cu_2O layer and on the base of the pit, there will be an extra force, namely electrical migration as distinct from simple diffusion, which will tend to concentrate chloride ions in the pit. Since the metallurgical structure of brasses and bronzes is often complex the results in the non-uniform distribution of the metals in the crystals, corrosion reactions can easily provide an extensive network of microscopic cracks.

Soluble copper(I) species from inside the pit can be transported through cracks in the Cu_2O membrane by diffusion, and these complexes are oxidized by molecular oxygen to cupric ion.

$$2CuC1_2^- \rightarrow 2CuC1_2 + 2e^- \quad (6)$$

while the oxygen is reduced to hydroxide ions.

$$O_2 + 2H^+ + 4e- \rightarrow 2OH- \quad (7)$$

The cathodic reaction (7) causes a localized increase in pH which tends to precipitate basic copper (II) compounds such as $Cu_2(OH)_3C1$ and $Cu_2(OH)_2(CO_3)$ – see Fig. 1. Experimentally, it is found that

approximately half the copper which has been corroded in the pit remains behind as Cu_2O in the pit itself. This is consistent with the densities of copper, copper (I) oxide and copper (I) chloride which are 8.92, 6.0 and 4.41g/cm^3 respectively.

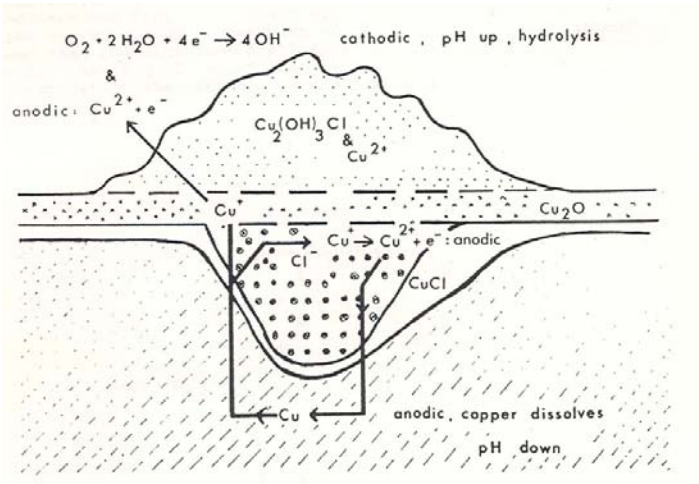


Figure 1.
Schematic representation of the major oxidation and reduction reactions involved in Bronze Disease.

"x" initial Cu_2O film, (x)Cu_2O crystals deposited as a result of CuC1 hydrolysis. The green Cu(II) corrosion products in the mound can be the basic chlorides $Cu_2(OH)_3C1$ or the basic carbonate $Cu_2(OH)_2(CO_3)$ depending on pH and carbonate activity. The chloride ions are associated with copper (I) complexes.

A major anodic reaction inside the pit, with the Cu2O membrane acting as an inert electrode, is the oxidation of copper (I) to copper (II) ions.

$$Cu^+ \rightarrow Cu^{2+} + e- \quad (8)$$

which in turn attack copper metal to form more cuprous ions

$$Cu^{2+} + Cu \rightarrow 2Cu^+ \quad (9)$$

which causes the pit to deepen. The precise function of the CuC1 layer at the bottom of the pit in the above reaction is not clear, but it may be an intermediate in the chemical attack of cupric ions on the metal. It is likely that the reaction in equation (8) is the slow step in the overall corrosion reaction.

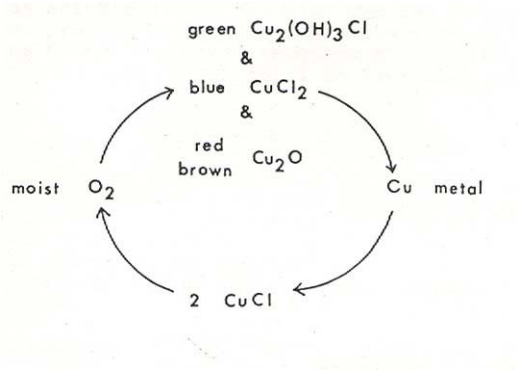
Table 1. The effect of chloride ion activity on the pitting corrosion mechanism.

Chloride ion concentration (ppm)*	**1**	**100**	**10,000**	**100,000**
Similar aqueous environment	**Deionized**	**Perth Tap**	**Dilute Sea**	**Pitt ** Solution**
Δ G kj/mole for $Cu^{2+} + Cu + 4C1^- \rightarrow 2CuC1_2^-$	**+75**	**+29**	**-17**	**-39**
Anodic reaction: $Cu + 2C1^- \rightarrow CuC1_2^- + e$				
Cathodic reaction: $Cu^{2+} + 2C1^- + e \rightarrow CuC1_2^-$				
Δ G kj/mole for $Cu^{2+} + Cu + 2C1^- \rightarrow 2CuC1$	**+12**	**-11**	**-34**	**-46**
Anodic reaction: $Cu + C1^- \rightarrow CuC1 + e$				
Cathodic reaction: $Cu^{2+} + C1^- + e \rightarrow CuC1$				

* The calculated standard free energies are made assuming that the chloride ion activity is equal to its concentration.
** Pit solution concentration is based on a concentrated brine.
(Data from Latimer, W.M. 'The Oxidation States of the Elements and their Potentials in Aqueous Solution' 2nd edn. Prentice Hall. New York 1952).

The effect of chloride ions on the leaching of copper metal by cupric ions can be seen in Table 1which shows how the free energy of the pitting reactions varies with chloride concentration. Positive values of

Δ G (the free energy) for a reaction means that the process will not proceed spontaneously in the direction as written but in fact the reverse reaction would be spontaneous. Negative values of Δ G mean that the reaction will proceed spontaneously but the sign and numerical size of Δ G does not indicate how fast the process will proceed. Since the sign of the free energy change varies with chloride concentration, it can be seen that chloride ions are intimately involved in the corrosion cycle – see Fig. 2. – and that by lowering the chloride concentration the pitting process can be stifled.



The BRONZE 'DISEASE' cycle

Figure 2. **Diagrammatic depiction of the cyclic nature of bronze disease.**

Since the oxidation of copper (I) ions which have diffused through the Cu2O layer occurs above the corrosion mound, the corrosion cell will not be stifled by the formation of basic copper chlorides. Most conservation sources state that the problem of bronze disease lies in the hydrolysis of the CuC1which produces HC1which, in turn, dissolves the copper and so deepens the pit, viz.

$$2CuC1 + H_2O \rightarrow Cu_2O + 2HC1 \quad \text{.....(5)}$$

$$2HC1 + 2Cu \rightarrow 2CuC1 + H_2 \quad \text{.....(10)}$$

The free energy change in Equation (5) under standard conditions is +64.48 kj $mole^{-1}$ and the reaction is in equilibrium at a pH of 5.65. Below this pH value the hydrolysis of cuprous chloride to form copper (I) oxide cannot occur at 25°C. At this pH the copper dissolution reaction, Equation (10) would produce an equilibrium pressure of approximately 10^{-27} atmospheres of hydrogen and so this reaction is not significant. Since chloride ions are known to move into the pit because of simple diffusion and migration to satisfy charge neutrality, the chloride concentration is not limited to that provided by Equation (5). High chloride activity mobilises the insoluble copper (I) chloride and so provides a mechanism for the removal of corrosion products without which the cell would tend to become stifled. The problem of high humidity is that apart from the hydrolysis reaction, Equation (5) releases chloride ions to help along the corrosion of the metal, Equation (4), the water vapour is sufficient to enable the $CuCl_2^-$ ions to move out to the region above the Cu_2O membrane.

If the hydrolysis of CuC1 is combined with oxidation to form the basic copper chloride $Cu_2(OH)_3C1$ as in Equation (11).

$$4CuC1 + 4H_20 + 0_2 \rightarrow 2Cu_2(OH)_3C1 + 2HC1 \quad \text{.....(11)}$$

the reaction will proceed spontaneously but the concentration of H+ cannot increase above approximately 4×10^{-5} M over the Cu_20 membrane since at lower pH values dissolution of the basic chloride occurs.

$$Cu_2(OH)_3C1 + 3H^+ \rightarrow 2Cu^{2+} + 3H_20 + C1^- \quad \text{.....(12)}$$

Similar arguments can be used for the formation/dissolution of the basic carbonate. Reaction is needed to establish the nature of the pH profile from the bottom of the pit to the top of the corrosion product mound.

CONTROL

Many artefacts which have bronze disease cannot be subjected to chemical treatment because of aesthetic constraints. In some collections the sheer volume of items, in itself, rules out any labour intensive treatment program. By lowering the relative humidity to 35%, bronze disease is normally controlled. As soon as a higher humidity environment is attained, this form of accelerated corrosion may recur. The lower humidity essentially prevents movement of ionic species from the pit to the surface and also removes the transport medium.

TREATMENT

The basic treatment procedures may involve one or more of the following (a) destruction of the cuprous chloride layer (b) removal of chloride ions and (c) isolating the metal ions from oxygen via film formation.

As mentioned in the discussion on pit formation, copper(I) chloride may be hydrolysed according to Equation (5) to produce copper(I) oxide plus hydrogen and chloride ions. If the pH of the pit is increased, the copper(I) chloride will hydrolyse to a greater extent. Removal of chloride ions will also cause the nantokite (CuC1) to decompose.

A combination of methods (a) and (b) forms the basis for the spot treatment of bronze disease by use of silver (I) oxide;

$$Ag_20 + 2CuC1 \rightarrow Cu_20 + 2AgC1 \quad \text{.....(13)}$$

Since the chloride ions are apparently removed by the formation of the less soluble silver chloride and the CuC1 layer is destroyed in the same process. Although this reaction is thermodynamically spontaneous there is another reaction to consider. Silver chloride can oxidize copper metal to form CuC1 and deposit Ag,

$$AgC1 + Cu \rightarrow CuC1 + Ag \quad \text{..... (14)}$$

For which ΔG is –9.2 kg mole $^{-1}$. If the corrosion rate is controlled by the cathodic process, Equation (7), then the silver metal may accelerate the corrosion rate since oxygen is reduced more rapidly on silver than on copper at room temperature. However, the overall effect of the reaction in Equation (14) is small since the finely divided silver would rapidly tarnish in air to form silver sulphide.

The procedure, (a), of destroying the CuC1 layer as a method of treating bronze disease can be attempted by chemical cleaning of all the corrosion products in various media described by Plenderleith and Werner (1971) and Merk (1978). Experience in these laboratories has shown that although the removal of surface material can speed up the chloride ion release rate on subsequent washing, the conventional chemical treatments such as soaking in citric acid (inhibited with thiourea) for several days does not penetrate into the deep cracks and voids in the copper, brass and bronze artefacts. Maritime material thus treated often shows signs of bronze disease several months after chemical removal of the bulk corrosion products.

The removal of chloride ions, method (b), is often affected by washing the artefacts in deionized water or in an aqueous solution of sodium sesquicarbonate (pH = 10). At a pH of 10 the copper(I) chloride is not stable and it will break down to form copper(I) oxide and release chloride ions between the corroded metal and the bulk of the wash solution, the chloride will be washed out of the artefact.

The hydrogen ions produced by the hydrolysis of CuC1 are removed by reaction with the carbonate to form more $HC0_3^-$. This neutralization reaction has little effect on the solution pH provided there is an adequate (0.5 wt%) concentration of the carbonate and bicarbonate to maintain the buffer system. Because of the high concentration of carbonate and bicarbonate, these ions can diffuse into the pit and precipitate more Cu_20 and/or basic copper (II) compounds. Since the chloride ion is one of the underlying causes of bronze disease its removal, as far as is practical, from the object is beneficial.

Washing of artefacts in deionized water is often an effective way of removing chloride ions and it is normally a diffusion-controlled process, which may take several years to complete. The rate of release of chlorides from both deionized water and sesqui-carbonate solutions should be plotted against time^{½} (time in hours, days, weeks etc.) and changes to wash solutions made after a period of no increase in the chloride concentrations. If marked variations occur in the characteristics of the $t^{½}$ plot e.g. conductivity increases more rapidly than $t^{½}$, then corrosion of the objects is suspected. When washing some bronze and brass artefacts in deionized water, care should be exercised to prevent surface blistering. I have found that corrosion of phases rich in alloying elements such as tin, lead and zinc can rapidly occur. It is advisable to ascertain the composition of brass and bronze artefacts prior to washing them in deionized water.

The third major method, (c), of bronze disease treatment is exemplified by the use of benzotriazole (BTA) – Sease (1978). The use of inhibitors such as benzotriazole has met with mixed success. The basis of the protection is the formation of a film of a copper (I) benzotriazole complex which consists of polymeric chains bonded to the surface. Benzotriazole reacts also with copper (II) compounds to form complexes which are readily reduced to the copper (I) species. The complexes of BTA involve BTA in its anionic form with copper (I) and a mixture of anionic and neutral for copper (II) viz. $Cu^{I}(BTA^{-})$ and $Cu^{II}(BTA^{-})_2(BTA)_2$ where the BTA^{-} is formed by loss of the hydrogen bonded to nitrogen (Pergola and Foresti, 1980). Benzotriazole is a weak acid, pK_a of 8.6 (Albert, 1968) and this facilitates the formation of such anionic complexes, as described above. The precise mechanism of inhibition is not completely understood.

We have found that for many bronzes and brasses recovered from a marine environment, treatment with aqueous and/or ethanolic solutions of BTA appears to be effective initially as no bronze disease occurs for about eighteen months. However, after this time the film appears to lose its effectiveness. The recurrence of bronze disease may be due to gradual movement of non-ionic species, such as oxygen, through the film which will reactivate the corrosion cycle. The bulk observations will naturally take time to appear. The underlying problem may be due to microcracks and pits deep within the structure of the apparently sound object. Intergranular corrosion, dezincification and destannification can penetrate several centimetres into an object and even extended soaking and vacuum impregnation in BTA and other wash solutions apparently fails to either remove enough chloride or to form a perfectly protective film to effectively prevent further accelerated corrosion.

The treatment of some bronzes with BTA can affect the colour of the patina, and in some cases the treatment cannot be used because of aesthetic constraints. This appears to be important when corrosion products from alloying elements such as lead form a significant proportion of the minerals in the original patina.

(N.B. Workers should be aware of the potentially carcinogenic nature of benzotriazole and related aromatic heterocyclic compounds).

RESEARCH

Many collections have thousands of similar artefacts to treat and thus labour – and materials – intensive/expensive processes such as the silver oxide treatment are ruled out. One promising avenue is the use of mixed solvent systems, which effectively leach out the harmful compounds such as cuprous chloride and yet leave the patina relatively untouched. The design of inorganic complexing agents, which will form soluble stable complexes with copper(I) and so remove unwanted CuC1 is a challenge to the conservator. The importance of having research chemists employed in conservation work cannot be overstated.

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