

## STAIN AND WHAT CAN BE DONE TO PREVENT IT

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As all seasoning superintendents know, if anything occurs to degrade the quality of the lumber, the cause will be laid on either the dry kiln or air yard. This may even include thick and thin to say nothing about log-borne defects. From the standpoint of stain control one has to grapple not only with log-borne defects but the exposure problems that develop between falling and milling as well as the seasoning process. Factors of the total exposure are time (or age off of the stump and saw), temperature and humidity (or the season of the year) plus pond vs. decking (wet or dry log storage) and seasoning. It is easy to claim that the previously proven anti-stain chemical is no longer effective. The fact is overlooked that the summer mix was used when cutting "our eighteen month old dry deck" during the winter. Naturally the performance did not compare with that of last summer's fresh logs. Not only was the stock totally different but so was the exposure prior to and in the air seasoning yard. It is the problems resulting from the total exposure that we have to wrestle with. The effects of exposure can be mitigated with management procedures and/or the proper use of anti-stain chemicals.

Before one can prescribe a regime of either prevention or control of a stain it is necessary to identify the cause and if possible how the staining system functions. To provide some structure to this presentation, I shall review the several classes of stain on the basis of cause and effect. Before doing that I would like to review the  $Q^{10}$  law which is fundamental to all biology and chemistry. This states that for each ten degree centigrade rise in temperature, there is a doubling of an enzyme's reaction rate. This is true between freezing and the thermal inactivation temperature of each enzyme. The arithmetic plot of this effect is shown in Figure 1. Assuming an activity of one at the water-ice interface the curve progresses through three orders of magnitude between freezing and boiling. Since all simple enzymatic reactions obey this law, it follows that all bacterial, mold and fungal growth is subject to it. The circle at 85°C is the inactivation temperature for a 15-minute exposure of the peroxidase that causes sugar pine brown stain. More about this thermally tough enzyme later. The table in the lower right hand corner gives the data for the curve. Ten degrees Celsius is equal to 18°F. A three cycle semi-log plot of the same data is shown in Figure 2. The concept of the  $Q^{10}$  law will be referred to in several contexts.

### CHEMICAL STAINS

From the standpoint of mechanism, a chemical stain, such as iron stain, is the simplest. To produce this stain iron reacts with the tannins in the wood forming a black pigment similar to iron pyro-gallate inks. Particles of metallic iron or rust are

picked up by the lumber during manufacturing or transportation. If the moisture conditions are right, a pigment will be formed. Redwood is particularly susceptible, both unseasoned and kiln dried, followed by unseasoned fir and hemlock packaged dimension and most hardwoods. Aqueous oxalic acid has long been used to remove these discolorations in the furniture industry. Weak phosphoric acid or ammonium-phosphates should not be used on unseasoned stock because the phosphate ion (as well as ammonia ion) is a nutrient for mold and fungi. Highway and railway dust are common sources of iron.

#### BACTERIAL STAINS

The prime example is sour log brown stain and the related porosity problem sometimes seen in the pines. At one time all the brown stains in the pines were assumed to have a single cause (1). The only difference was that the heartwood of the yellow pines didn't stain while that of the white pines did.

Shortly after I went to the Western Pine lab, Al Stout and I went to a mill out of Madras, Oregon to investigate a ponderosa brown stain problem. On entering the mill the bad silage odor was overpowering. The obvious presence of the lower aliphatic acids (propionic, butyric and caproic or caprylic) (1) suggested the action of a bacteria. The rough KD lumber just out of the kiln showed the typical streaked stain in the sapwood. We learned that the logs had been stored in their new hot spring heated pond which had eliminated the sawing of frozen logs.

On returning to the lab the stained KD samples were found to give an uneven pattern of solvent uptake in the sapwood. This is the test for porosity, the problem that plagued the millwork and pine furniture industries. Ununiform uptake of finishing systems and solvent borne preservative water repellents is encountered on sour stock.

On squeezing some of the sap from the fresh samples, a curved spore forming bacillus was found in what appeared to be a pure culture. We could not identify this bacteria although morphologically it seemed to be a vibrio. It was subsequently shown to be Desulfovibrio desulfuricans an acid forming bacteria common to marine muds, hot springs and oil wells (2). Clearly bacteria were producing stain and porosity while following the  $Q^{10}$  rule in its growth within the logs stored in the warm pond. Over the years it has been found that salt or brackish water storage promotes bacterial growth in Douglas fir and hemlock logs. The bacteria invade the sapwood via the inner bark through breaks in the bark as trimmed limbs. The wood is penetrated via the ray parenchyma as the bacteria live off of the starch, pectin and hemicellulose producing the porosity seen when dry. The presumption that these curved rods were the cause of sour log brown stain and porosity was reported to the Western Pine Association membership at their 1957 spring meeting. With this portion of the brown stain problem defined it was now possible to concentrate on the cause and effect of the white pine brown stain --particularly in sugar pine in the presence of sour log or bacterial brown stain.

After the fact nothing can be done to correct this log borne damage. Fast turnover of logs is the best preventative procedure

while very cold water storage provides the maximum storage life after falling. Decks sprinkled with warm recycled water in the summer can approach the staining pattern of a warm pond. This water carries a heavy inoculum. Since there is no escape from the  $Q^{10}$  law for this problem, the best protective strategy is low temperature storage for the shortest practical time between stump and saw. Don't inventory two years' supply of logs on tidal flats or in a shallow stagnate pond.

In a study from the Richmond lab Ellwood (3) showed that the retting bacteria Bacillus polymixa produces stain and porosity without the odorous acids. Knuth and McCoy's extensive study (2) indicates that B. polymixa is the primary invader giving rise to the porosity while producing ethanol and acetic acid as metabolic by products. D. desulfuricans is thought to be an incidental organism living off of the ethanol and acetic acid produced by B. polymixa and not contributing to increased porosity. Several studies on the addition of bacteriostatic chemicals to pond water to control these bacteria have been carried out. None were either environmentally acceptable or commercially feasible. All conifer saw logs are subject to bacterial infections. In the worse cases a virtual zoo of microorganisms can be expressed from infected log ends--various types of bacteria, yeast and protozoa. In extremely sour samples, the D. sulfuricans percentage of the population is so high that it appears to be the sole bacteria present.

In recent years some of the fir and hemlock operations have experienced a heart-sap boundary stain in logs harvested from some wet sites (4). This system is reported to be heat sensitive indicating a biological cause. Although no causative organism has been reported, the action of a bacteria, fungi or their extra-cellular enzymes may be involved.

#### BLUE STAIN AND MOLD

These two classes of oxygen requiring (aerobic) ascomycetes are usually found together. With a few exceptions they are generally susceptible to the same fungicides. It is easy to deal with them as one while pointing out the most obvious differences. Blue stain refers to the grey-blue to black discoloration that develops in the hyphae of this group of fungi as the wood dries slowly. In the wide sapwood of second growth redwood the stain is a grey-brown approaching the color of the low extractive heartwood. This in fact can add to customer confusion at the local lumber yard. Redwood sapwood is as vulnerable to decay at the ground line as any. Fence posts from this type of stock are short lived.

The green, black and grey molds that accompany the stainers are usually aspergillus or penicillium. Very colorful thermophilic molds may occasionally be encountered under very moist and warm exposure conditions. The bright red, orange and yellow surface discoloration produced by the thermophiles is usually photo degraded or lost as the lumber dries. An effective chemical anti-stain control program provides some luxury in time for management of the seasoning program. There is time for the slow sorts to accumulate and stain build up over a three-day weekend in the summer is not as critical. It still is advisable

to get everything off of the chain and onto sticks as soon as possible. The drying mode is safer.

Stain in the core of otherwise bright stock, is caused by log borne infection, which was prevented from blooming through the surface by the anti-stain treatment. To my knowledge there is no fungicide, which applied to the surface of freshly sawn lumber, can penetrate the board sufficiently to eliminate internal stain. Nor can stain, which is already visible, be readily bleached. Lumber cut from aged and infected decks should go directly to the dry kiln. Internal stain is guaranteed in the dry yard if the infected stock is not topped off in the kiln before the core reaches fiber saturation. At this point the level of available oxygen will be high enough to support the growth of the stainer in the core. The bright shell and blue core will be found under an otherwise adequate anti-stain treatment. Internal stain is not a problem during the rapid air seasoning of thick stock from bright freshly felled logs, although sufficient fungicide must be used to prevent sticker stain. Effective anti-stain treatment imparts a protective envelope of treated wood over a susceptible core. Hence the treatment must cover all surfaces.

It must not be forgotten that the unprotected sapwood of any species will stain if the moisture content and temperature are high enough to permit growth in an aerobic state. Rewet kiln dried lumber is as vulnerable as an untreated board fresh off of the saw.

The much too common local lumber yard scene of a well picked cover pile of dirty stained, moldy and somewhat decayed studs is unnecessary. The sapwood is totally stained and all faces of the boards are moldy--this includes the heartwood. If conditions have been optimum for growth, there will be a large fraction of the pieces in the core of the package stuck together by the mycelial pads of decay. Again the  $Q^{10}$  law is predictably running its course. This surface degradation can be prevented with proper anti-stain treatment even if the lumber was cut from infected logs and the unseasoned studs are 6 to 12 months off of the saw.

Occasionally seemingly bright unseasoned studs will be put into place and partially collapse on drying. In these cases the stock had been adequately treated to control the surface evidence of decay. Meanwhile the decay fungi continued to grow in the core during the period of solid package green storage. Such studs are often a mere shell of their former selves after they dry. Two-year-old packages of studs are the most common source of this problem.

As to the question of how well the new non-chlorophenol and mercury-free fungicide formulations will perform under severe exposure conditions, one can only follow the supplier's recommendation. A fungicide with very little or no fish or mammalian toxicity plus being totally biodegradable must be carefully checked under severe exposure conditions to determine its real cost effectiveness. Since all organisms on this earth share many identical critical metabolic steps, one must reflect that it is the dose that makes the poison. About 460 years ago Paracelsus (1493-1541) taught that "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy."

A more modern description of the phenomena that Paracelsus recognized can be visualized in the bell-shaped diagram seen in Figure 3. Metabolically essential elements, such as copper and zinc, fit this growth response pattern for most organisms. The sensitivities vary but the pattern is the same for bacteria, mold, fungi, green plants, fish, fowl and mammals including man. The sequence of death from too little, a clinical and subclinical deficiency, to an optimum growth or healthiness at the top, is only half of the story. This first segment is often forgotten when dealing with copper and zinc toxicity. The descending part of the bell encompasses the toxic range where the fungistatic properties of these elements can be seen. At concentrations greater than that required for optimum growth, there is a region of subclinical toxicity, followed by a clearly defined region of clinical toxicity, which crosses the LD 50 boundary and finally 100% kill at all increasing concentrations. The LD 50 is that magic number where 50% of the population dies under defined exposure conditions. The 100% plus kill range of a fungicide is the level that must be present on a board's surface to prevent stain, mold and decay spore germination or hyphal invasion. Less could stimulate growth if the exposure was right.

There are several mold blue stain relationships which bear special mention. The first is the pentachlorophenol tolerance of the molds Cephalosporium fragrans and Pullularia pullians. Both can grow in the presence of the chlorophenols which these molds immobilize and/or degrade. Blue stain spores can germinate on cephalosporium mycelial pads and stain the underlying wood below the surrounding bright shell. This situation is most severe when the level of penta is barely enough to suppress blue-stain development but permitted a lush growth of brown mold. Cephalosporium is also sensitive to the phenyl mercurials. The second example is the tolerance of Trichoderma verdi to the organic mercurial--ethyl-mercuric-phosphate--the active ingredient in DuPont's original Lignasan. The lush green felts, that grew on Lignasan-treated ponderosa in the air-seasoning yards, suppressed blue-stain--probably through the production of an anti-biotic. The mold felts simultaneously retarded the drying sufficiently to leave wet areas. This stock could be so moldy that the dry lumber had to be dressed to be sold.

Historically, by reducing the availability of oxygen through water storage of logs, blue stain was greatly reduced when compared with dry deck logs. The advent of effective stain inhibiting chemicals such as the chlorophenols, greatly improved the operational freedom of a mill. With the present environmental restrictions on the use of chlorophenols for stain control, it may become economically desirable to review the pattern of lumber processing. If the time delays between stump and mill were minimized and the drying operation tightly controlled, the need for stain controlling chemicals can be eliminated. My father-in-law, Heber Radcliffe, never used anti-stain chemicals to protect the sawn lumber at McCloud River. The mill had the kiln capacity and steam to accommodate the cut. Even with their large pond, they did have sour log stain in the spring on last summer's logs as well as some log-borne blue stain from the high floaters in the bundles. There was no fresh water circulation in the pond. To produce the brightest possible lumber, it was just a matter of

making the  $Q^{10}$  law work for you rather than ignoring it-- particularly if one is to successfully dry lumber without anti-stain chemicals.

#### ENZYMATIC BROWN STAIN OF THE WHITE PINES

In the absence of bacterial brown stain in the sapwood, the brown stain found in the white pines is caused by a peroxidase. This enzyme is an iron hematoporphyrin with a pair of active sulphydryl groups on the protein chain. The sulphydryl is usually portrayed as R-SH where R represents the protein or enzyme. R-SH can be reversibly oxidized or reduced by the removal or addition of a molecule of hydrogen as:  $2 \text{ R-SH} \rightleftharpoons \text{R-S-S-R} + \text{H}_2$ . The sulphydryl specificity towards the organic mercurials is evidence of structural differences in the peroxidase of the white pines.

The hemoglobin of blood is a well known iron hematoporphyrin while chlorophyll is the magnesium hematoporphyrin of plants. All organisms have peroxidases. The use of buffered sodium azide in conjunction with the alkaline anti-stain chemicals in use during the 1960s could eliminate all but log-borne brown stain in sugar pine. The loss of azide from the market and the replacement of the alkaline with acidic anti-stain formulations puts brown stain control back with the low temperature open vent kiln schedules of thirty years ago. Because of this it is necessary to examine the character of the enzyme as it affects the seasoning strategies that may now be required.

Examination of the staining mechanism points up the basis of the unique problems confronting seasoning of lumber from the white pines. Since peroxidases require an abundance of oxygen for their activity, the staining process cannot start until the log is converted to lumber with the exception of dry log ends, falling cracks and breaks in the bark. In their natural environment the action of peroxidases is blocked by the presence of natural inhibitors and the lack of sufficient oxygen. The inhibitors must either be metabolized or removed for activity to be demonstrated. In sugar pine the staining process is first detected at the heart sap boundary, then builds up in the sapwood and later in the heartwood. As time goes on the intensity of stain will be bleached out in the outer sapwood while increasing in color and depth in the heartwood. This process is accelerated by a moist atmosphere, and elevated temperatures, but blocked by still higher inactivating temperatures and drying.

A creamy tan coloration develops as incubation progresses. If this lumber is kiln dried, it will stain. This second chemical step is the oxidative polymerization of the creamy leuco pigment formed by the peroxidase. Slightly off color air seasoned sugar pine can be shipped as bright and put in place only to have the stain develop over a period of years in a heated building. There is a story of the special run of wide air seasoned sugar pine paneling used in a lumber company's board room. The streaked black walnut discoloration that developed after a few steam heated winters led to its removal. In all fairness the leuco pigment may not have been present when this paneling was put in place. The peroxidase is still capable of activity after air seasoning. It is only the absence of sufficient moisture that inactivates the peroxidase. All that is needed is an adequate supply of moisture

and heat to restart the staining process which may then run its course. Topping off air seasoned stock in the kiln can prevent this problem by inactivating the enzyme. Topping off will also complete the staining process for any unnoticed leuco pigment. Thus topping off can constitute a real operational dilemma.

The strategy of chemical control was to inhibit the active site on the enzyme. At the start of the Western Pine Association studies the identity of the enzyme was not known. The staining system was known to be heat sensitive which presumes the action of an enzyme. Since it could be either a peroxidase or a copper containing tyrosinase, the action of inhibitors for both were tested. The basis of the assay in the lab were small pieces of wood from the heart-sap boundary. Buffered azide was found to be the most effective and compatible with the anti-stain dips then in use. The effective use of azide is limited by the fact that the inhibition is reversed by the movement of wood acids to the surface. These are carried by the evaporating moisture stream as drying progresses. A large portion of the sodium azide is converted into gaseous hydroazotic acid thus gradually reducing the level of control. This loss of hydroazotic acid during drying can be a real problem for partially air seasoned lumber caught in a heavy fall rain. If possible, such stock should immediately be topped off in the kiln. Unfortunately, there are no good irreversible inhibitors for the iron hematoporphyrin site on the enzyme.

In the course of the WPA studies it was demonstrated that when azide was used in conjunction with Lignasan, half the normal amount of azide was required to attain the same level of control. The formation of ethyl-mercuric-azide could be demonstrated in these ready-to-use solutions. This led to the assumption that the combination was acting on the iron hematoporphyrin even though ethyl-mercuric-phosphate, EMP, alone showed noticeable activity. At this time it was not recognized that a pair of sulfhydryl groups were required for peroxidase activity. Other workers later demonstrated the -SH activity in a bacterial peroxidase. The WPA studies also showed that pentachlorophenol, as used in the sap stain formulations, provided some suppression of brown stain. Presumably this was via substrate competition from an essentially unmetabolizable phenol not inhibition of a key reaction.

In 1975 I carried out a study on *Pinus strobus* in Ontario, Canada. In this study our commercial anti-stain containing penta-60, borate and phenyl-mercuric-lactate, (PML) was used for blue stain control. To my surprise the PML controlled the white pine coffee stain that developed in the absence of PML or azide. An irreversible enzyme inhibitor complex can be formed between the eastern white pine sulfhydryls and PML. This effect cannot be shown for PML with sugar pine. Presumably there is enough space in the sugar pine peroxidase to bind the smaller ethyl-mercurial ion but not the larger phenyl-mercurial. Irreversible inhibition of sulfhydryl enzymes by both organic and inorganic mercurials as corrosive sublimate-- $\text{HgCl}_2$ --is well known. This Canadian mill was using sodium fluoride, at about 5 lb. per 100 gal. Imp. for coffee stain control. Fluoride has no effect on sugar pine brown stain.

Peroxidases do not follow the one reaction one product path common to most enzymes. Instead peroxidases are product activated

with the hydrogen peroxide produced being used to further degrade susceptible substrate and product, i.e., phenols and tannins. The initial oxidative action of a peroxidase is to remove a pair of hydrogen atoms from a substrate through the action of a pair of oxidized sulfhydryls. The two hydrogen atoms are then attached to an oxygen molecule forming hydrogen peroxide or  $H_2O_2$  and regenerating the oxidized sulfhydryls. The sites on the phenolic substrate, which were formally occupied by hydrogen, are replaced by water thus oxidizing it. A plot of such a mixed up reaction pattern is far more complex than a  $Q^{10}$  plot. A log log plot is required to produce a fairly straight line when oxygen uptake is being measured. Under optimum incubation conditions the  $Q^{10}$  law is being vastly exceeded.

The extreme heat stability of the peroxidase may well be the largest problem in the kiln drying of sugar pine in the absence of azide. The extracted enzyme requires 15 minutes at  $85^{\circ}C$  to be inactivated. Activity in a 1/4 inch slice of wood was only reduced 50% by a 20 minute immersion in boiling water. This is one tough enzyme. It is no wonder that a tightly closed kiln becomes a giant incubation chamber during a rapid high temperature warm up. If the surface of the lumber is not kept dry until inactivation has been achieved, the charge will be brown. The activity of the peroxidase is more than doubling for each  $10^{\circ}C$  increase in temperature. The  $Q^{10}$  is being exceeded. On the other side of this seemingly dismal picture is the fact that the higher the temperature the faster the rate of inactivation--or cooking of the enzyme protein. In the absence of the inhibitor, sugar pine fresh from the saw must be kept dry on the surface through proper use of the vents and a judicious warm up rate. This is costly of calories and kiln time. A little azide and a fast schedule will do the job for less. The activation vs. inactivation obstacle must be dealt with intelligently. Properly kiln dried sugar pine will never brown stain if it is bright when it comes out of the kiln.

The strategy that seems to best alleviate the problems raised by absence of azide is to keep the surface of the lumber dry and free of puddling as the kiln temperature is raised. Apparently both a saturated environment and oxygen are required for the auto-catalytic product activation step to become fully operational. These drying conditions, which are wasteful of heat and kiln time, may be more easily achieved in the air seasoning yard during the summer although there is some risk of brown sticker stain. The kiln operator has to stage a balancing act between the  $Q^{10}$  thermal activation and inactivation while trying to dry sugar pine lumber. As soon as the newly sawn board is exposed to air the staining process starts. Even at temperatures as low as  $50^{\circ}C$  or  $40^{\circ}F$  stain will develop if given sufficient time under otherwise good incubation conditions. In the absence of azide, Heber Radcliffe's mild and vented kiln schedules may be a viable solution when coupled with getting the drying process underway the day the board is milled.

A second strategy, which could be investigated, would be to quickly cook the surface 1/8-1/4 inch of the board. By destroying the peroxidase in this part of the board, the startup of the product activation system should be blocked. Perhaps this could be accomplished in a microwave tunnel with the boards passing



through one at a time as they do through a planer. The energy cost might be prohibitive. Cross sectional differences could be managed by a computer programmed to provide uniform treatment.

There are some thermally sensitive stains in the hardwoods which presumably are of an enzymatic nature. The orange-red stain, which develops on western red alder on milling, may be caused by a tyrosinase. It is not sensitive to azide. The grey discoloration seen in the core of ripped hard maple may have an enzymatic or blue stain origin. This pattern is faithfully reproduced in photo overlays of maple drain boards. The grey stain of southern red oak is probably caused by a peroxidase. Azide will block stain development on the surface, but as this very porous wood dries in the seasoning yard, the stain develops under the bright surface. There are other non-microbial staining systems in the southern hardwoods with which I am less familiar.

#### FUNGAL STAINS

There is the very colorful red-purple stain occasionally encountered in Douglas fir which is the incipient stage of Fomes pini. Since this decay occurs in standing timber, there is nothing that can be done to correct the defect. I have occasionally encountered it in S4S unseasoned stud studies on stock that was bright when treated. White speck will also continue to grow in solid packages of studs.

One final caution on "safe, non-toxic and biodegradable" fungicides. Determine how they will perform under the most severe exposure. Summer in the dry yard is a poor time to check out fungicides for next winter's wet weather. Dipping in water alone will give a 15-20% reduction in stain. Even aspirin can be made to look good after a short, mild exposure.

If you have any questions, I'll be glad to try to answer them. Please remember--I've never run a dry kiln--not even the little experimental one at the old WPA lab. I can only discuss sugar pine schedules in respect to activating and inactivating temperatures plus the fundamentals of the enzyme. To consistently produce stain-free lumber, all of the components of the exposure must be taken into consideration and the necessary compensatory action must be taken to fit the total exposure.

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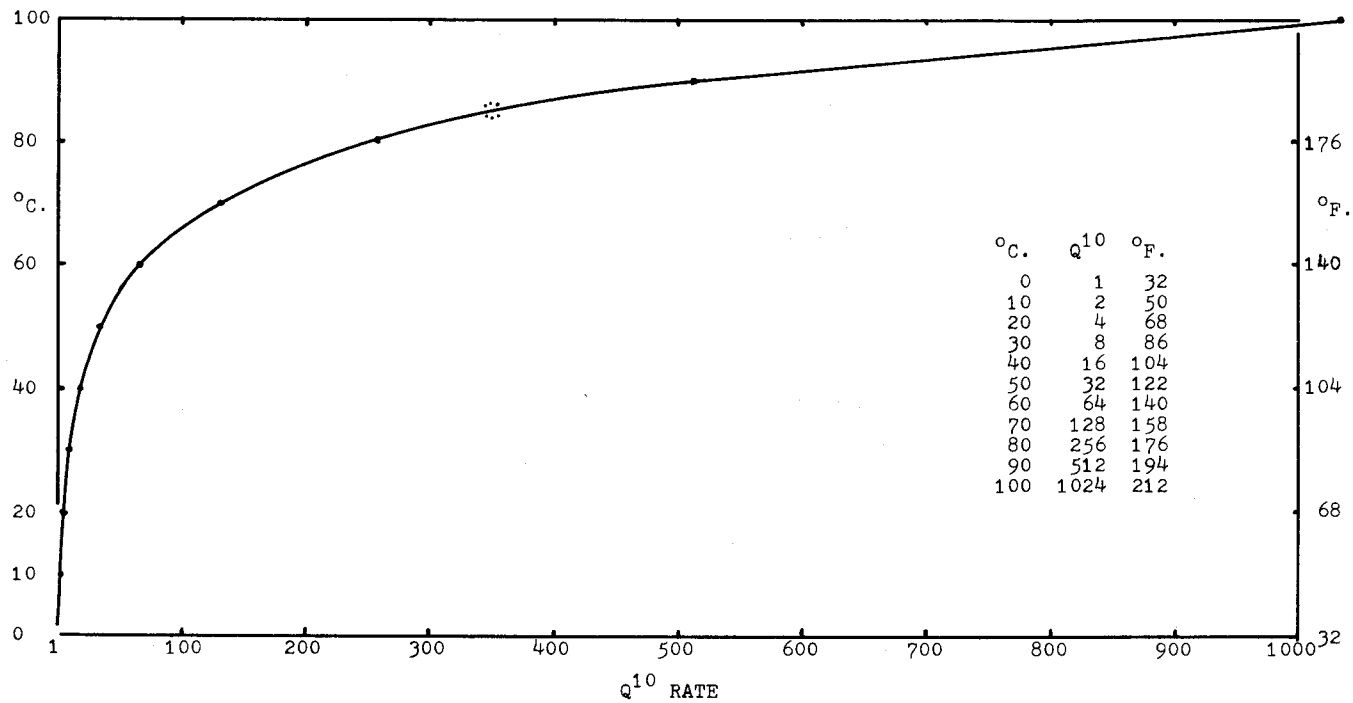


Figure 1

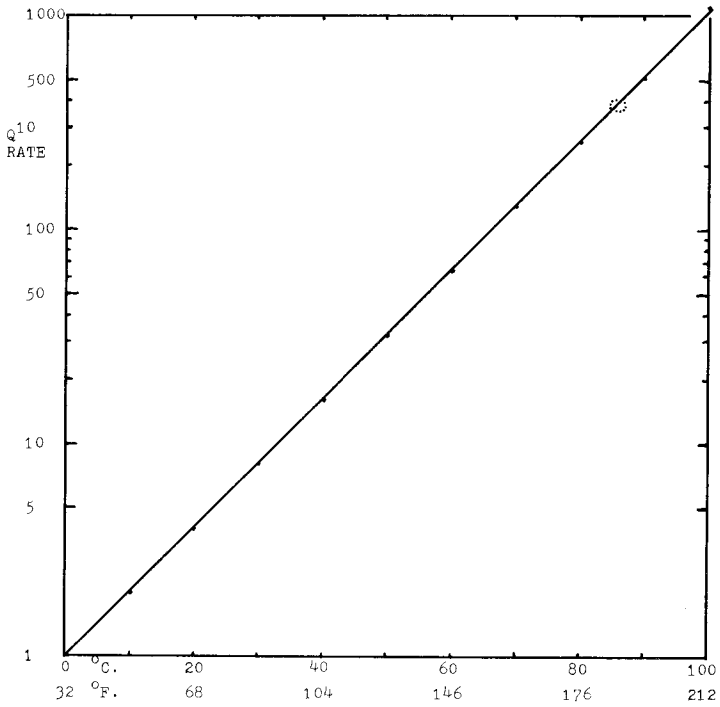


Figure 2

"ITS THE DOSE THAT MAKES THE POISON."

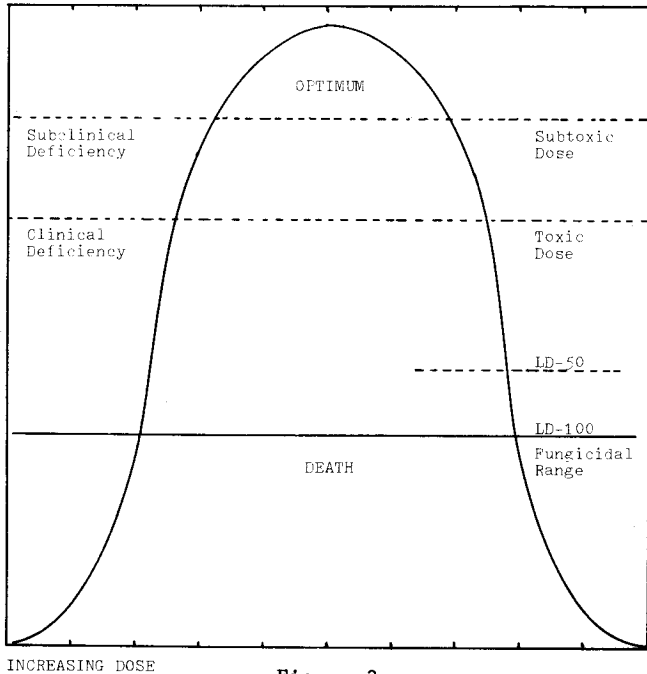


Figure 3