

POSITION DESCRIPTION
PLANT CHIEF CHEMIST
ANNISTON PLANT
FEBRUARY 1970

I. FUNCTION

Plant Chief Chemist is directly responsible for management of the plant laboratory and pollution control functions. Maintains an adequately trained staff of professional employees and assistants capable of providing Process Research, Pollution Control Engineering and Analytical services in support of plant manufacturing, cost and pollution goals.

II. RESPONSIBILITIES	INDICATORS	OBJECTIVES	GOALS 1970
A. Safety	1. Accident Reports Safety Statistics	1.a. Disabling injury frequency 0.0. b. Serious injury frequency 0.0. c. All injury frequency <1.0.	1.a. Disabling injury frequency 0.0. b. Serious injury frequency 0.0. c. All injury frequency <1.0.
	2. Plant Dispensary First Aid Service	2.a. First aid services reflect expertise and sound judgment. b. Test to determine individual proficiency at first aid.	2.a. Plan and conduct first aid review seminar for all analytical employees by 9-1-70. b. Conduct three emergency tests to determine profi- ciency in treating burns, heart attack, electrical shock.
	3. Safety Meetings	3.a. All employees attend 100% safety meetings.	3.a. Minimum of 90% attendance at all safety meetings.
	4. Safety Review	4.a. Safety review conducted on all major lab projects.	4.a. Formal safety review conducted for: 1. Trailer lab facility prior to occupancy. 2. Biphenyl tube prior to start-up. 3. Plant laboratories by 10-1-70.

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II. RESPONSIBILITIES	INDICATORS	OBJECTIVES	GOALS 1970
A. Safety (cont'd)	4. Safety Review (cont'd)	4.b. All research project work plans include safety review section.	4.b. All research project work plans include safety review section.
	5. Accident Investigation	5.a. Reduce lab injuries to 0.0. 5.b. Solutions and investigation reflect knowledge of chemical and physical causes.	5.a. All lab injuries reviewed by three men to generate recommendations. 5.b. Lab personnel serve and/or chair AI committees as appointed.
	6. Housekeeping Reports	6.a. No safety hazards due to poor housekeeping.	6.a. Minimum housekeeping score 240.
B. Cost	1. Budgets	1.a. Minimize service dept. cost. b. Pollution control cost under control and accurate. c. Minimize cost and need for outside consulting services.	1.a. Gross 914.35-914.46 1970 actual minimum 10M below budget. b. Review cost distribution to 913.41, 914.23 and 914.24 by 7-1-70. Publish guidelines for charges. c. Review present use of consultants and employ only on sound justification by 4-1-70.
	2. Special Cost Reports	2.a. Minimum cost methods to comply with pollution legislation and community needs.	2.a. Develop two alternates for compliance with PCB and SO ₂ guidelines report by 10-1-70.
	3. 914.35 Overtime Report	3.a. Control overtime below 8.0%.	3.a. 1970 overtime 5% maximum.
	4. Research and Development Program	4.a. Cost improvement program contributions \$200,000/yr.	4.a. Cost improvement of \$100,000 minimum.

II. RESPONSIBILITIES	INDICATORS	OBJECTIVES	GOALS 1970										
C. Planning & Control of Resources	1. Manufacturing Improvement Plan	1.a. All Process Research contributions to MIP achieve target. b. Pollution control and research programs current for product MIP.	1.a. 85% PR&D projects completed within time and achieve target. b. Pollution and research sections of MIP due as follows: <table> <tr> <td>PNP</td><td>4-30</td></tr> <tr> <td>HCl</td><td>5-15</td></tr> <tr> <td>Parathion</td><td>6-16</td></tr> <tr> <td>Aroclor</td><td>7-1</td></tr> <tr> <td>Bi, Santowax, HB-40</td><td>6-1</td></tr> </table>	PNP	4-30	HCl	5-15	Parathion	6-16	Aroclor	7-1	Bi, Santowax, HB-40	6-1
PNP	4-30												
HCl	5-15												
Parathion	6-16												
Aroclor	7-1												
Bi, Santowax, HB-40	6-1												
	2. 1971 Annual Programs	2.a. PR&D Program issued by 12-1-70. b. Pollution Control Program issued by 12-1-70. c. All program revisions are formally recorded and assigned a Revision No.	2.a. PR&D Program issued 12-1-70. b. Pollution Control Program issued by 12-1-70. c. All program revisions are formally recorded and assigned a Revision No.										
	3. Five-year Manpower Plan	3.a. Combined R&D-Pollution Control Manpower Plan published by 12-1-70.	3.a. Combined R&D-Pollution Control Manpower Plan published by 12-1-70.										
	4. Work Plans	4.a. Minimum 70% manpower directed toward research program and pollution abatement. Review semiannually. b. Go - No Go decision points included for each quarter of work plan.	4.a. Minimum 70% manpower directed toward research program and pollution abatement. Review semiannually. b. Go - No Go decision points included for each quarter of work plan.										
	5. Analytical Manpower Utilization	5.a. Hours/sample analytical index at 0.35.	5.a. Hours/sample analytical index at 0.38 YTD.										

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II. RESPONSIBILITIES	INDICATORS	OBJECTIVES	GOALS 1970
D. Pollution	1. Control of plant discharge to eliminate adverse effect on operations.	1.a. Reduce PCB discharges to 10 ppb in all plant water.	1.a. Complete research and install facility for 70-90% reduction in PCB discharges by 12/31/70.
		b. Comply with governmental regulations for SO ₂ ground level concentration.	b. Complete evaluation of alternates and develop premise for compliance with regulations by 12-31-70.
		c. Front ditch under control pH 3.5 Min. HCl - NDA.	c. Publish HCl neutralization guidelines by 3-1-70. Assist production in operating per guideline.
		d. Snow Creek quality pH - 4.0 Min., PNP - 1 ppm Max., Parathion - < 0.1 ppm.	d. Publish Parathion waste treatment guidelines by 6-1-70. Assist production in operating per guideline.
	2. Pollution Control Board	2.a. Monthly review with staff of present trends, legislation and programs underway.	2.a. Establish Pollution Control Board by 4-70 to a) inform, b) decide, c) take action on pollution issues.
	3. Pollution Control Program	3.a. Annual program to coordinate activities of Production - TSD - Maintenance.	3.a. 1970 program published by 3-1-70.
	4. PCB Analytical Services	4.a. PCB lab and FDA-5460 equipment installed and on line by 5-15-70.	4.a. PCB lab and FDA-5460 equipment installed and on line by 5-15-70.
E. Research	1. PR&D Reports	1.a. 1970 program and revisions completed with target deadlines.	1.a. 1970 program and revisions completed with target deadlines.
		b. Biphenyl Pilot Tube start-up 4-15-70.	b. Biphenyl Pilot Tube start-up 4-15-70.
		c. Biphenyl Pilot project generates cost improvement of 200M in 1970.	c. Biphenyl Pilot project generates cost improvement of 100M in 1970.

II. RESPONSIBILITIES	INDICATORS	OBJECTIVES	GOALS 1970
E. Research (cont'd)	1. PR&D Reports (cont'd)	1.d. Minimum 250 mandays on Pilot Tube program in 1970. e. Comprehensive evaluation of Santowax C/HB-40 completed by 9-23-70. Go - No Go by 8-14-70.	1.d. 250 mandays effort on project in 1970 by J. L. Brown, J. T. Bell, and G. L. Arnett. e. Comprehensive evaluation of Santowax C/HB-40 completed by 9-23-70. Go - No Go by 8-14-70.
F. Community and Governmental Relations	1. Knowledge of pollution law trends 2. Complaint Reports	1.a. Pollution control plans developed with sufficient lead time prior to enforcement to allow compliance. c. Fully utilize the corporate governmental relations manager for the Anniston Plant to be up-to-date on legislation. 2.a. No air or water complaints.	1.a. Evaluate and communicate plans of AWIC and AIA through personal contact with staff at least quarterly. b. Maintain current legal opinion on Alabama law and potential effects on plant. Publish. c. Invite governmental relations manager to plant for semi-annual status review. 2.a. No more than 10 air pollution complaints. b. No water pollution complaints. c. Aid to develop program allowing 700 ppm Max. solids to city. d. Maintain "good neighbor" relations with all plant neighbors. Provide prompt investigation for all complaints. e. Develop formal report system for complaints and follow-up. Review monthly.

II. RESPONSIBILITIES	INDICATORS	OBJECTIVES	GOALS 1970
G. Personnel	1. Performance Appraisals, Development Plans	1.a. Current, appraised RIG documents exist and training profile for all personnel.	1.a. All RIG based appraisals are < 14 months old. b. Training and development profiles are reviewed each six months for all employees. c. All position descriptions are reviewed annually by 12-1-70.
		2.a. Analytical employees have access to ICS type instruction in analytical chemistry.	2.a. Develop ICS program to allow home study for analysts and technicians by 4-1-70.
H. Relationships	1. Reports to TSD Superintendent. Appraised by TSD Superintendent.	1.a. Minimum of above adequate performance	1.a. Minimum of above adequate performance.
Administers:			
Process Research and Development			
Pollution Control Engineering			
Analytical Lab Service			

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No. 1

INFORMATION BULLETIN B.I.B.R.A.

CURRENT DEVELOPMENTS IN
FOOD ADULTERATION
AND TECHNOLOGY

BRITISH INDUSTRIAL
BIOLOGICAL RESEARCH
ASSOCIATION

in deciding where priorities lie. It is surely no more important to study the possibility of poisoning by food additives than to keep a watch on fried potatoes.

THE PCB STORY UNFOLDS

It has only recently been recognised that polychlorinated biphenyls (PCBs), widely used as plasticisers, lubricants and fire retardants and as vapour suppressors to prolong the residual life of insecticides, are extremely stable compounds which resemble the organochlorine insecticides in their ability to persist in the environment. The similarity does not end there, for their retention times on the gas chromatograph are close to those of some structurally-related organochlorine pesticides such as DDT, and their unsuspected presence interfered in the past with analyses of DDT in biological material (BIRRA Bull. 1970, 2, 363). The realization of their persistence in the environment was reflected last October in an order from the US Department of Agriculture's Pesticides Regulation Division that the use of PCBs and polychlorinated terphenyls as active or inactive ingredients in pesticide formulations should be phased out within 6 months, since any benefits derived from this application were "outweighed by the resulting environmental contamination and the possible long range adverse effects on fish and wildlife" (Food Chemical News 1970, 12 (35), 20). A few weeks later the FDA asked its 17 District Offices to survey foodstuffs for contamination with PCBs, giving particular attention to liquid milk (*ibid* 1970, 12 (35), 5).

The FDA announcement noted that studies on the effects of PCBs on growth, mortality and reproduction were in progress and referred to indications that these chemicals might increase the toxicity of pesticides (BIRRA Bull. 1970, 2, 90). Comparatively little is known about PCB toxicity and a recent study has shown that its assessment is considerably confused by other compounds commonly present in commercial PCBs (*ibid* 1970, 2, 377; Vos *et al.* *Food Cosmet. Toxicol.* 1970, 8, 625). Earlier reports have associated ingestion of PCBs with liver damage in rats and guinea-pigs and lesions resembling 'chick-oedema' in the chick and quail, but Vos *et al.* (*loc.cit.*), comparing the oral toxicities and analyses of three commercial samples, found that high mortality, subcutaneous and abdominal oedema and centrilobular liver necrosis were produced in chicks by only two of the samples, and hydropic oedema, a common effect of these two samples, was only occasionally associated with the third. The analytical results and other toxicological evidence indicated that these toxic effects were in fact due not to the PCB itself, but to chlorinated dibenzofurans, principally the tetra- and pentachloro compounds, present as impurities in only two of the commercial PCB products.

Just how widespread are PCB residues in wildlife? At the Nature Conservancy's Mins Wood Experimental Station, wild birds and their eggs have for some years been analysed for PCB as well as pesticide residues (BIRRA Bull. 1970, 2, 470) and Presti *et al.* (*Envir. Pollut.* 1970, 1, 3) have now presented the results obtained so far. Analyses have been completed for a total of 196 livers from 33 species and 363 eggs from 28 species, obtained between April 1966 and August 1968. PCBs have been detected in terrestrial species from most regions of Britain, in seabirds and all seabird eggs from one west coast and two east coast colonies, and in most of the freshwater species, which were collected principally from

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the Midlands and eastern and southern England. Liver residues were high-
est in freshwater fish-eating birds, levels of 500 and 900 ppm having been
found in two herons from Dorsetshire and Yorkshire, and in bird-eating
vaptors, in which levels ranged from 0-70 ppm. Birds that fed on mammals
generally contained somewhat lower levels (0-50 ppm), while those on a
mixed diet of carrion, birds and mammals and those feeding on insects had
maximum levels of 15 and 1 ppm respectively. The same pattern was observed
in the egg residue figures, the highest level detected being 80 ppm in a
heron's egg. The eggs of this species also contained a maximum of 26.5 ppm
p,p'-DDE and 6.5 ppm dieldrin, but there were more eggs with <1 ppm PCBs
than with <1 ppm p,p'-DDE or dieldrin, suggesting that of the three, PCBs
were generally present in the smallest amount. Residue levels of the three
compounds in kestrel livers followed the same distribution, except that
PCBs occurred at <2 ppm less frequently than did dieldrin.

Could the higher residues detected have caused the death of the birds
concerned, without any contribution from other factors? Preat *et al.*
(1969, *ibid.*) obtained an indication of the avian toxicity of PCB by feeding
Aroclor 1254 to Bengalese finches at dose levels ranging from 26-1940
mg/day for 56 days. No dose level produced 100% mortality over this
period, but the estimated dose causing 50% mortality was equivalent to
254 mg/kg/day, although one bird died on less than 12 mg/kg/day. In
contrast, the level of p,p'-DDE required to cause 50% mortality over the
same period was only about 19 mg/kg/day, but the mortality curve was much
steeper than with PCB, suggesting that the latter may be the more toxic at
low dose levels. Some birds dying from PCB poisoning displayed apparent
leg paralysis or body and wing trembling, and on autopsy all had enlarged
kidneys and two showed considerable amounts of fluid in the pericardial
sac. The liver concentration of PCB increased with increasing dose levels,
although great variation was observed at each level; at the dose causing
50% mortality the mean liver content was 345 ppm with a range of 59-3070
ppm, while one bird survived with a liver concentration of 654 ppm. Birds
starved at the end of the feeding period had a significantly increased
liver concentration of PCB. In normally fed birds, however, PCB was
stored mainly in the depot fat, as was shown in one bird which was found
to have accumulated 9% of the total dose by the end of the experiment.

The relationship between liver PCB concentration and death was so
vague that no definite conclusions could be drawn as to the high levels
found in certain predatory birds. A high ratio of brain-to-liver con-
centrations was much more closely correlated with mortality in the finch,
but unfortunately brain levels were not measured in any of the wild birds
studied. Assuming a similar sensitivity to the finch, roughly 3% of birds
could have died with liver residues of 5-360 ppm, 34% with 22-1780 ppm and
50% with 39-3070 ppm. On this basis it was thought unlikely that PCBs
could have caused widespread lethal toxicity in wild predatory birds in
Britain, but no reference was made to the mass deaths of scabids in the
autumn of 1969 (BIRRA Bull. 1969, 8, 554; Bourne & Moad, B.T.O. News
1969, no. 36). It was considered, however, that PCBs could be a component
cause of the present breeding failure reported in several species.

Work by Linoer & Peakall (Nature, Lond. 1970, 228, 783) confirmed
that PCBs affect hormone metabolism in birds at very low dose levels.
Kestrels were fed for 5 months on dead cockerels which had been injected
with 0.5 or 5.0 ppm Aroclor 1254 or Aroclor 1262 dissolved in sesame oil,
the higher dose being roughly equivalent to a dose for the kestrel of 2 mg
PCB/kg. When at the end of this period the microsomal fraction from the
kestrel liver was incubated with oestradiol *in vitro*, a dose-dependent

breakdown of the oestradial to a more polar metabolite took place. There was no apparent difference between the effects of the two PCBs, and no such conversion occurred with the livers of control birds fed cockerels injected with sesame oil alone. In a further study on kestrels given 5 ppm Aroclor 1254, this increase in hepatic-enzyme activity was correlated with an increase in cytoplasmic RNA, suggesting that the action of PCB was similar to that of DDT and its metabolites.

PCB residues in seabirds appear commonplace (Prestt et al. loc.cit.) and high residues have been reported in birds from the Irish Sea (BIRDA Bull. 1969, 8, 554) and the Baltic (ibid 1970, 2, 120). The sources of such residues in the marine environment has been something of a mystery, for which Haldon (Nature, Lond. 1970, 228, 1220) now offers a probable solution. High PCB concentrations have been detected in water from the Firth of Clyde in recent years, and levels of up to 2.6 ppm have been found in clupeoid fish from that area. In the autumn of 1969 analysis of samples of surface and sub-surface water and of trunk-sewer effluents between the estuary mouth and Glasgow failed to reveal any local discharges of PCB. A close resemblance was found, however, between the gas-liquid chromatograms of extracts from crude sewage sludge, normally dumped in deep water 6 miles south of the estuary mouth at the rate of about 1 million wet tons/year, and those of some commercial PCBs. Estimated PCB concentrations were in the range <0.1-14 ppm (wet weight) or 1-185 ppm (dry weight). A simple calculation revealed that the mean PCB level of 1 ppm (found in 15 samples of wet sludge weighted according to the proportions dumped) would lead to an annual PCB discharge into the Clyde estuary of about 1 ton. Crude sludge from Manchester and London contained respectively about 2 and 0.2 ppm PCB, suggesting that about 1 ton of PCB isomers is similarly discharged into both the northern Irish Sea and the Thames estuary every year.

The PCBs in this sludge come from industrial waste which is normally combined with domestic sewage for treatment. When it is considered that some coastal towns discharge untreated wastes directly into the sea, while in inland areas treated sludge is commonly spread on the land, the ease with which PCBs can enter both the marine and terrestrial ecosystems becomes obvious.

That PCBs may also enter foodstuffs directly as a result of contamination from packaging is indicated by the findings of Bailey et al. (Chem Ind. 1970, 20, 705). One sample of ground cashew nuts, analysed at the Pest Infestation Control Laboratory as part of a routine survey for pesticide residues in imported foodstuffs, was found to contain about 10 ppm of a PCB containing 40% chlorine. Unlike other samples from the same shipment which were free from PCB, this sample had been received in a lacquered cardboard drum, which on analysis proved to contain 200 ppm of the same PCB. Similar drums were used in the laboratory to store empty gelatine capsules intended for toxicological feeding, but fortunately no PCB could be detected in the capsules. PCBs were found, however, in most other samples of cardboard analysed, and the ease with which contamination from this source could occur is emphasised.

The gradual slope of the avian mortality curve for PCBs presented by Prestt et al. (loc.cit.), indicating that these compounds may cause deaths at low dose levels (albeit in only a small proportion of birds), and the ability of PCBs to induce microsomal enzymes at a dietary level of only 0.5 ppm (Lincoer & Peakall, loc.cit.) suggests that even low residues of PCBs in the environment could present some threat to wildlife. Whether a

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ban like that imposed on certain organochlorine pesticides will be necessary is another matter. In the USA, Monsanto Chemical Corp., one of the major PCB manufacturers, is reported to have discontinued sale of PCB products for uses other than in 'closed systems' such as electrical transformers (Food Chemical News 1970, 12 (35), 6) and in the UK a similar move is afoot to introduce a voluntary ban on uncontrolled use of these compounds in products like paints and varnishes (Sunday Times, 3 January 1971). If this public-spirited move succeeds the need for legislative measures will be obviated.

AFATOXIN—A BARDY ANNUAL

Reports on aflatoxin (AF) have continued to be published at a considerable rate since our last review (BIRRA Bull. 1970, 2, 273) but progress in our understanding of the health hazard presented by this toxin remains fairly slow. Many strains of the *Aspergillus flavus* group can produce AF, which has been shown to be, for some animals, one of the most potent carcinogens known. In a study of some 28 of these strains, Besseltino et al. (Mycologia 1970, 62, 123) were unable to distinguish between them on grounds of gross morphology or biochemistry and found that the presence and ratios of the various types of AF appeared to be the best basis for classification.

One of the more notable of the recent contributions to the ever-growing AF literature is a topical survey of the problem given by Dr. J.M. Barnes as the Second Stenhouse-Williams Memorial Lecture to the Society for Applied Bacteriology (Barnes, J. appl. Bact. 1970, 22, 285). Dr. Barnes points out that there is little point in trying to analyse food eaten at any particular time in order to find out whether it contains AF because chance plays too great a part in determining the degree to which any particular harvest or batches of stored food may be contaminated. Information must be obtained at a much earlier stage, with adequate surveys of food harvesting and storage, so that the populations that may be most heavily exposed to food regularly or intermittently contaminated with fungi may be identified.

However, many reports on the levels of AF contamination in food products are still compiled and remain of some passing interest. Cheese has always seemed a likely candidate to play host to AF and, indeed, AF production by *Aspergillus* has been demonstrated in Cheddar cheese by Lie & Marth (J. Dairy Sci. 1967, 50, 1708). However, more recently, samples of Roquefort, Camembert and domestic blue cheese were all found to be free from AF (Shih & Marth, *ibid* 1969, 52, 1681). Similarly in a German study (Klemmeier & Groll, Z. Lebensmittelunters. u. -Forsch. 1970, 143, 81; *idem*, *ibid* 1970, 142, 120), no AF was found in 169 samples of 19 cheese varieties, but artificial infection with *A. flavus* resulted in large amounts of AF by on Tilsit cheese under favourable environmental conditions, although on Camembert and Boursart the growth of the fungus was inhibited. Clearly the right conditions for AF contamination are present in some hot and humid cheese factories. A further German paper (Spicher, Brot Gebäck 1969, 23, 149) has shown that AF may occasionally be formed in some wholemeal and mixed breads. However, the state of deterioration of the bread was such that it was unlikely to have been eaten.

The potential formation of AF during the storage of meat in display