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May 9, 1980

Docket Officer
Docket H-034
Room S6212
U.S. Department of Labor
200 Constitution Avenue, N. W.
Washington, D. C. 20210

Re: Docket H-034, Occupational Safety and Health Administration Request for Information on Vinyl Chloride and Polyvinyl Chloride - 44 Fed. Reg. 74928, December 18, 1979; 45 Fed. Reg. 668, January 29, 1980.

Dear Sir:

Pursuant to Section 4 of the Administrative Procedure Act, as amended, 5 U.S.C. Section 553(c), and the above-referenced U.S. Department of Labor - Occupational Safety and Health Administration (OSHA) Request for Information on Vinyl Chloride and Polyvinyl Chloride and the subsequent Notice extending the time during which interested persons are invited to submit written data, views, and comments with regard to the issues described in the original Notice, The Society of the Plastics Industry, Inc. (hereinafter referred to as "SPI" and/or "The Society"), by its attorneys, hereby submits in quadruplicate certain information which is intended to be responsive to the Agency's Request.

SCC
3-0792

I.

INTRODUCTION AND SUMMARY OF INFORMATION PRESENTED

SPI is a corporation organized under the Not-for-Profit Corporation law of the State of New York. Its 1500 member companies and individuals and 67 operating divisions include those who supply raw materials; process or manufacture plastics or plastics products; engineer or construct molds or similar accessory equipment for the plastics industry; and engage in the manufacture of machinery used to make plastics products and materials of all types. SPI is the major national trade association of the plastics industry. The majority of its members are the processors and converters of the plastics resins and end products which represent 75% of the dollar volume of sales of plastics in this country; SPI's membership also represents 95% of all plastics materials and machinery manufactured in the U.S.A.

The Society's concern with vinyl chloride and polyvinyl chloride begins with the manufacture of the ethylene dichloride (used to manufacture the monomer) and the manufacture of vinyl chloride monomer (VCM), carries forth through its polymerization into polyvinyl chloride (PVC), continues with the various processes which convert the PVC resin into its multitude of end uses, and includes, ultimately, the recycling or disposal of PVC products. In the United States there are 17 ethylene dichloride plants, 13 plants which produce vinyl chloride monomer and 42 polymerization plants.

Twenty-two companies representing over 95% of the domestic VCM and PVC capacity are active members of SPI's PVC Safety Group.

SPI's PVC Safety Group has prepared the information, data and views contained in what follows, and it is presented in a form which corresponds in large part to the explicit requests for information contained in the Agency's Notice.

In summary, The Society's responses to the questions asked are as follows:

1. OSHA is aware of the studies on vinyl chloride sponsored by Imperial Chemicals Industries, Limited; Montedison; Rhone-Poulenc Industries; and Solvay et Cie. in Dr. Maltoni's laboratory in Bologna, Italy.

   These most recent data of Maltoni indicate possible effects in rats exposed to 10 ppm of vinyl chloride but no increase in tumors was found in experimental rats exposed below 10 ppm. It is clearly evident that rodents are more susceptible than humans and that human experience should be given higher importance than animal studies. Animal data have predicted an angiosarcoma incidence rate over 500 times higher than that observed in man.

2. It has been reported in the literature that VCM can exert a carcinogenic effect transplacentally;

however, this phenomenon has only been demonstrated in pregnant rats exposed to very high concentrations. Additional work in this area needs to be done using lower, more realistic doses since available data indicate that the rates of metabolism in the rat shift as exposure concentrations of vinyl chloride change. Vinyl chloride did not cause significant embryonal or fetal toxicity and was not teratogenic in studies in rats, mice or rabbits. We know of no valid scientific paper associating vinyl chloride with human birth defects at any level of exposure.

3. While no carcinogenic effect of inhaled PVC has been demonstrated in animals or man, recent data indicate that like most nuisance dusts slight effects may occur in the lungs following exposure to high concentrations of the dust.

4. Although there are no new epidemiological data on health effects associated with vinyl chloride exposure which would appear to change the conclusions from those considered in the current standard, The Society has contracted for a complete, independent analysis of all available, relevant epidemiologic data. This is particularly important since it is not always made clear in subsequent papers reviewing them that many of

the studies which have been reported represent repeated investigations of the same population or subsets thereof. Considering the magnitude of this project, it will not be finished by the May 9, 1980 deadline associated with the Request but these materials will be submitted to the Agency when they have been completed.

5. The Society is not aware of any new, unpublished case reports on cancer associated with vinyl chloride or polyvinyl chloride but is submitting to the Agency the most recent, world-wide compilation of all liver angiosarcoma cases associated with VCM.

6. It has been demonstrated that if mutagenic effects occur as monitored by chromosomal analysis of circulating lymphocytes, they were the results of high exposures; the changes have not been permanent since they have been reversible. The changes did not occur at lower levels of exposure. The clinical significance of the reversible changes is not known.

7. Being a poorly soluble gas, vinyl chloride does not accumulate from chronic exposure, but, rather, is rapidly exhaled after exposure ceases. Hence, there can be no body burden in the usual meaning of the term.

8 and 9. An overview of PVC manufacturing operations, types of resin, residual monomer levels, and PVC dust experience is presented with information on monitoring results, control technology and personal protective devices. Variability in resin type and the resin type relationship to differing residual monomer content in the resin, varying VCM exposure potential from both resin and dust, and the type of PVC dust and other conditions which might be expected in both the polymerization and fabricating industries are explained.

10. SPI is unaware of any change in the types of occupations, jobs and industries which present a potential for exposure to VCM or PVC. The Society has no data on the numbers or breakdown by sex of employees exposed.

11. The VCM/PVC industry is engaging in the use of appropriate engineering controls, work practices and personal protective equipment and is trying to stay in compliance with the existing standard; the industry knows of no additional, feasible measures to reduce exposure below the present level. PVC dust is adequately regulated by OSHA's nuisance dust permissible exposure level (29 C.F.R. §1910.1000) and has not been shown to be hazardous at that level of exposure.

II.

SPECIFIC INDUSTRY RESPONSES TO INFORMATION REQUESTED

(1) Experimental Test Results for Carcinogenicity of Vinyl Chloride At Atmospheric Exposures Less Than 50 ppm

The current studies on vinyl chloride are those conducted in Dr. Maltoni's laboratory in Bologna, Italy. As OSHA is aware, these investigations are sponsored by Imperial Chemicals Industries, Limited; Montedison; Rhone-Poulenc Industries; and Solvay et Cie.

These most recent data of Maltoni indicate probable effects in rats exposed to 10 ppm of vinyl chloride but no increase in tumors was found in experimental rats exposed below 10 ppm. As to the mammary tumors Maltoni noted at lower levels, the data are not convincing because the experimental animals used are known to have a high incidence of spontaneous mammary tumors. As will be discussed below rodents are more susceptible than humans and human experience should be given higher importance than animal studies.

Dr. Maltoni's data were reported in detail at a recent OSHA/NIOSH/NIEHS sponsored Symposium, March 20 and 21, 1980 in Bethesda, Maryland. An unedited stenographic transcript of the entire Symposium and photographic copies of most of the slides are attached as Appendix A.

In Maltoni's studies, rats, mice and/or hamsters were exposed to concentrations ranging from 30,000 ppm to

1 ppm. The attached summary statement by the European sponsors quotes Dr. Maltoni and indicates that extensive statistical evaulations of Dr. Maltoni's data have been completed. Appendix B.

In that summary, Dr. Maltoni summarized his animal studies as follows:

> ...the following tumours should be given proper attention viz, extra-hepatic angiosarcomas, hepatomas, Zymbal gland carcinomas, liver angiosarcomas, neuroblastomas, nephroblastomas, forestomach papillomas and mammary carcinomas.
>
> In oncological terms, the meaning of the results at the lowest doses may be better evaluated by considering, not separately, but together, the tumours found to be VCM dependent. Thus the following results should be considered: -
>
> at 25 ppm - In 120 animals, 5 liver angiosarcomas, 4 Zymbal gland carcinomas and 1 nephroblastoma.
>
> at 10 ppm - In 120 animals, 1 liver angiosarcoma, 2 extra-hepatic angiosarcomas and 2 Zymbal gland carcinomas.
>
> at 1mg/kg - In 150 animals, 3 liver angiosarcomas, 1 extra-hepatic angiosarcoma, 1 hepatoma and 5 Zymbal gland carcinomas....

These data add to that previously published by Dr. Maltoni since tumors occurred in rats exposed to 25 or 10 ppm of vinyl chloride gas. Maltoni's European sponsors state,

> In addition, it is worthwhile emphasising that none of the previously cited tumours have been observed at 5 ppm by inhalation and 0.03 mg/kg by ingestion.

These data will be published as part of the proceedings of Club de Cancerogenese Chemique held at the Curie

Foundation in Paris, France, November 10, 1979. These proceedings, which will be supplied to OSHA as soon as available, will contain an even more complete report of Maltoni's studies than was presented in Bethesda on March 20, 1980.

In regard to the response of laboratory animals to vinyl chloride gas, the attached paper by Gehring, Watanabe and Park is extremely pertinent. Appendix C. This paper indicates the necessity of considering the rate of metabolism and body size of different species when comparing the response of different species to vinyl chloride.

Many factors, such as those considered by Gehring, Watanabe and Park, no doubt are involved in the wide difference between the high incidence of angiosarcoma in small rodents and the much lower incidence in man.

It is clearly evident that rodents are more susceptible than humans and that human experience should be given higher importance than animal studies. Dr. David Rall, Director of NIEHS, has commented on the wide difference between the susceptibility of man and rodents. Appendicies D and E. He stated that in regard to angiosarcoma the available data

> ...project a cancer incidence rate 500 times higher than has so far been reported in man by epidemiological studies for vinyl chloride. (Rall, 1977, Appendix D, p. 2.)

(2) Studies of Transplacental Carcinogenic and Teratogenic Effects in Humans or Animals at Any Level of Exposure to Vinyl Chloride

It has been reported that VCM can exert a carcinogenic effect transplacentally[1/2/]; however, the phenomenon has only been demonstrated in pregnant rats exposed to very high (10,000 or 6,000 ppm) concentrations of the material. Additional work needs to be done using lower, realistic doses since available data indicate that rates of metabolism in rats shift as exposure concentrations of vinyl chloride change and that some mechanisms of metabolism are saturable.[3/] Vinyl chloride did not cause significant embryonal or fetal toxicity and was not teratogenic in studies in rats, mice or rabbits[4/] even at levels which resulted in maternal toxicity. We know of no valid scientific paper associating vinyl

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1/ Maltoni, C., Vinyl Chloride Carcinogenecity: An Experimental Model for Carcinogenesis Studies. In "Origins of Human Cancer. Book A: Incidence of Cancer in Humans." H.H. Hiatt, J.D. Watson and J.A. Winsten, Cold Spring Harbor, Cold Spring University, 1977.

2/ Maltoni, C., Carcinogenicity Bioassays of Vinyl Chloride Monomer: A Model of Risk Assessment on Experimental Basis. Symposium Paper No. 1 (1980).

3/ Watanabe, P.G. and Gehring, P.J., Dose Dependent Fate of Vinyl Chloride and Its Possible Relationship to Oncogenicity In Rats. Environmental Health Perspectives 17:145-152 (1976).

4/ John, J.A., Smith, F.A., Leong, B.K.J., and Schwetz, B.A., The Effects of Maternally Inhaled Vinyl Chloride on Embryonal and Fetal Development in Mice, Rats, and Rabbits. Toxicol. and Appl. Pharmacol. 39:497-513 (1977).

chloride with human birth defects at any level of exposure.

1. TRANSPLACENTAL CARCINOGENESIS

Maltoni has reported that exposure of female rats to 10,000 and 6,000 ppm of vinyl chloride has resulted in transplacental effects in their offspring. No data have been found in which pregnant animals have been exposed to lower concentrations. Rice[5/], although not having actually studied vinyl chloride in his laboratory, reviewed the subject of transplacental carcinogenesis. In discussing the above-mentioned work of Maltoni, Rice refers to the important role of biotransformation of VC to the probable carcinogenic metabolite. This metabolic transformation in the pregnant female, the possibility of a similar transformation in the fetus, the kinetics of placental transfer (which processes can be bidirectional), and the metabolic degradation in both the gravid female and the fetus all illustrate the complexity of transplacental carcinogenesis.

Rice also rightly points out that in the case of VCM the probable carcinogenic metabolite has a relatively short half-life. This would limit the times for transfer across the placenta to the fetus and for distribution to the fetus. Although the data of Maltoni indicate that the metabolite either crosses the placental barrier or is formed in the fetus in sufficient concentration to elicit a carcinogenic

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5/ Rice, J., Transplacental Carcinogenic Effects. Symposium Paper No. 27 (1980).

response, the short half-life suggests that the effect would be minimized at lower levels of maternal exposure. Unfortunately, sufficient data are lacking on much of the foregoing, and, therefore, definitive conclusions cannot be reached.

Another complicating factor, even over and above those usually recognized in extrapolating from Maltoni's transplacental carcinogenic data to rats to a possible human situation, is the knowledge that rats are far more sensitive to VCM than humans. (See response to Request no. 1.) In addition, there are marked differences in placental structure between humans and rodents.

The following discussion of the possible effects of these differences in placentation with regard to teratogenesis is equally germane to the consideration of transplacental carcinogenesis:

> ...Both man and the test animals commonly used possess chorioallantoic placentae, but that of man is the hemochorial one consisting essentially of fetal villi hanging in a pool of maternal blood. The chorioallantoic placenta of rodents and lagomorphs (rabbits) is the complex hemoendothelial type which consists of closely juxtaposed and highly modified fetal and maternal cells, permeated by a labyrinth of blood sinuses. An even more significant difference between the two types is that man has only the chorioallantoic placenta, whereas the rodents and the lagomorphs have a yolk-sac placenta as well. In man, once the chorioallantoic placenta has formed, most if not all, drugs reach the fetus by this route. In the rodents, often used as test animals, the presence of two types of placentae in the same animal means that the drugs might reach the embryo in two different ways, and it seems probable that in the earliest most vulnerable stage, the drugs that enter

the embryo do so predominantly via the yolk-sac placenta6/....

The marked difference in the structure of the placenta between rats and humans, the alteration in metabolism at lower dosages, and the recognized higher susceptibility of the rat to angiosarcoma must all be taken into account in interpreting Maltoni's results at extremely high concentrations. The highly idealized scheme (Figure 1) may serve to highlight some of the complexities of these factors.

2. TERATOGENIC EFFECTS

The landmark study of the teratogenic potential of VCM remains the industry-supported study of three species by John et al., first reported in part at the 14th Annual Meeting of the Society of Toxicology in 1975. John and her co-workers subsequently published a detailed report of the work7/, and again reiterated the findings at this most recent Symposium. The authors' published summary of this important study is as follows:

> ...Groups of pregnant CF-1 mice, Sprague-Dawley rats and New Zealand white rabbits were exposed to 500 ppm of vinyl chloride 7 hr. daily during the period of major organogenesis. Subsequently, other groups of mice were similarly exposed to 50 ppm of vinyl

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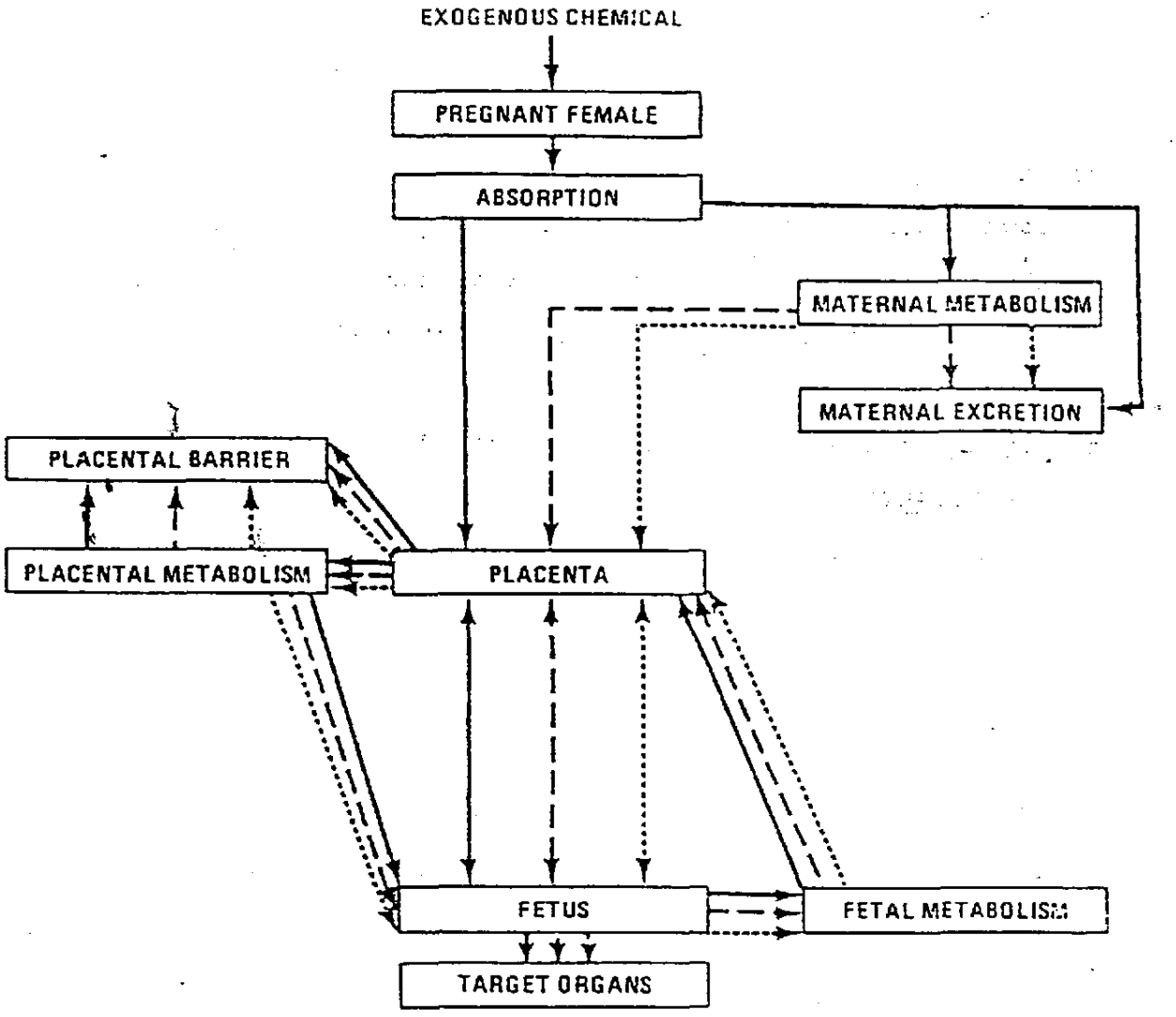
6/ "The Testing of Chemicals for Carcinogenicity, Mutagenicity and Teratogenicity," Minister of Health and Welfare, Canada, 1973.

7/ John, J.A., Smith, F.A., Leong, B.K.J. and Schwetz, B.A., The Effects of Maternally Inhaled Vinyl Chloride on Embryonal and Fetal Development in Mice, Rats, and Rabbits. Toxicol. and Appl. Pharmacol. 39:497-513 (1977).



LEGEND

——— CHEMICAL

– – – TOXIC (CARCINOGENIC) METABOLITE

------- NON-TOXIC (NON-CARCINOGENIC) METABOLITE

FIGURE 1. HIGHLY CONCEPTUALIZED SCHEME OF THE PATHWAYS FROM MATERNAL EXPOSURE TO A CHEMICAL TO THE EXPOSURE OF FETAL TARGET ORGANS. THE INFLUENCE OF THE PHYSICOCHEMICAL PROPERTIES OF THE SPECIFIC CHEMICAL, AS WELL AS MANY BIOLOGICAL CAPABILITIES OF THE ORGANISM, ON THESE PATHWAYS ARE NOT CONSIDERED IN THE DRAWING.

chloride and rats and rabbits were exposed to 2500 ppm of vinyl chloride. While maternal toxicity was observed, vinyl choride alone did not cause significant embryonal or fetal toxicity and was not teratogenic in any of the species at the concentrations tested. Maternal toxicity was more prominent among mice than among rats and rabbits. Simultaneous exposure of some of the pregnant animals to vinyl chloride by inhalation plus 15 percent ethanol in the drinking water resulted in toxic effects greater than those associated with exposure to vinyl chloride alone in the three species. The maternal toxicity was enhanced to an extent greater than the embryotoxicity. (Emphasis added.)

It should be noted that, to our knowledge, the conclusions reached by John et al. with regard to the teratogenic potential of VCM have not been seriously questioned and, in fact, have been confirmed.

Dr. John, in her presentation at the Symposium, discussed briefly the results of two additional studies of the teratogenic potential of VCM which have appeared in the recent literature. The first was a report of work carried out in Hungary,[8/] the findings of which were consistent with those reported by John et al. The second report, originating with the Institute of Hygiene and Occupational Health in Bulgaria, is still available only in abstract form. Although sufficient detail is not presented in the abstract to permit a critical evaluation of the study, the report alleges a teratogenic response at much lower levels of VCM exposure than those shown to have no effect by either Ungvary

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8/ Ungvary, G., The Teratogenic Effect of Vinyl Chloride. MUNKAVEDELEM (Work Safety) 25:29-33 (1979).

or John et al. A complete assessment of this Bulgarian study is essential before it can be given any credence.

Dr. John also mentioned the Symposium presentation of Hehir[9/]. As far as can be determined from the transcription of Dr. Hehir's talk, as well as what can be gleaned from a prepublication copy of the CPSC report[10/], the study did not address teratogenesis. All F_0 animals were exposed before mating but progeny were not. Thus, although effects on reproductive capacity or genetic changes in the germ cells (e.g., dominant lethal effects) could possibly be manifest, true teratogenic events dependent upon exposure of the fetus could not be revealed. It should be noted, however, that the F_0 exposure did not result in any alterations in the various parameters monitored in the F_1, F_2, and F_3 generations.

In summary, the most completely documented study of the teratogenic potential of VCM, viz., the study of John et al., reveals that under the high exposure conditions of that study inhalation of VCM by pregnant rats, mice or rabbits was neither embryo nor fetotoxic, nor was it teratogenic

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9/ Hehir, R., Cancer Induction Following Single and Multiple Exposures to a Constant Amount of Vinyl Chloride Monomer. Symposium Paper No. 5 (1980).

10/ Hehir, R.M., Bierbower, G., Willigan, D.A., Kolaja, G., Marrs, G.E., Hinton, D.E., Dimmick, R.L. and Wiles, J.S., Toxicology, Carcinogeniity and Reproductive Effects of Single and Multiple Exposures to Vinyl Chloride in Rats and Mice. CPSC Prepublication Release, April 1, 1979.

even at concentrations sufficient to cause maternal toxicity. While an additional published report basically confirms the latter conclusions, results available only in abstract form from the Bulgarian literature purport to reveal a teratogenic effect for VCM at lower levels. Although this latter information is contradictory of the study by John and her coworkers, as well as that of the Hungarian study, its full significance must await evaluation of the complete report when it becomes available.

Further, it should be noted that the maximum dose levels tested in the study of John et al., 2500 ppm in rats and rabbits and 500 ppm in mice, provide ample margin for safety extrapolation from these test species to the human situation.

Finally, to our knowledge, there are no reports which clearly relate birth defects in humans to maternal, or for that matter, paternal exposure to VCM. Two relevant studies which have been published[11/][12/] indicate no relationship between population exposure to VCM in the area surrounding PVC formulation plants and birth defects. Infante et al.,[13/]

11/ Edmonds, L.D., Anderson, C.D., Flynt, J.W. Jr., and Heath, C.W. Jr., Congenital Cental Nervous System Malformations, Kanawah County, West Virginia. U.S. Public Health Service, CDC, Atlanta, EPA-76-60-2 (1976).

12/ Edmonds, L.D., Falk, H. and Nissim, J.E., Congenital Malformations and Vinyl Chloride. Lancet ii:1098 (1975).

13/ Infante, P., Wagoner, J.K., McMichael, A.J., Waxweiler, R.J. and Falk, H., Genetic Risks of Vinyl Chloride. Lancet 11:734-35 (1976).

have suggested a causal relationship between paternal exposure to VCM in the industrial setting and pregnancy outcome. The adequacy of the data supplied by Infante et al. was almost immediately questioned[14/]. More recently, Haas and Schottenfeld[15/] have leveled severe criticisms at the Infante study, and the following is extracted from their paper:

> ...Analysis of questionnaire responses suggested that the number of fetal deaths per 100 conceptions was higher in the VCM-exposed group than in the comparison group. This difference was reported only for the period following vinyl chloride exposure. Adjustments removing women who were chronic aborters eliminated statistically significant differences. While the authors felt that these observations were likely to reflect a real difference in pregnancy outcome not attributable to either interviewer, or patient recall bias, the conclusions were based on indirect sources of information and could not take into account the multiplicity of maternal factors known to affect pregnancy outcome. The study design precluded documenting in even the crudest manner the validity of pregnancy histories. Without such adjustments and validation, the inferences made cannot be sustained and little light is shed on the possible association of abnormal pregnancy outcome with paternal occupational exposure to VCM. (Emphasis added.)

Likewise, others have raised serious doubts regarding the validity of the conclusions drawn by the authors of the Infante et al. study. For example, Downs et al.[16/] so com-

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14/ Paddle, G.M., Genetic Risks of Vinyl Chloride Lancet 1:1079 (1976). Appendix F.

15/ Haas, J.F. and Schottenfeld, D., Risks to the Offspring from Parental Occupational Exposures. J. Occup. Med. 21:607-13 (1979). Appendix G.

16/ Downs, T.D., Stallones, R.A., Frankowski, R.F. and Labarthe, D.R., Vinyl Chloride, Birth Defects, and Fetal Wastage: A Critical Review. Private Communication. Appendix H.

pletely discredited the study that their ultimate conclusion was as follows:

> ...It does not seem possible to salvage anything from this study....
>
> Among the more specific criticisms are the following:
>
> The authors gave no information on the distribution of workers' reported recall of time intervals since pregnancies. Such information would be useful in assessing the validity of this study, since it is well known that the reliability of recall of past events decreases with time elapsed since the events.
>
> The authors' analytical methods are invalid since the ...test requires that the two rates being compared be independent, and this is not the case....
>
> ...The methods used by the authors to test significance are inappropriate since pregnancies are clustered....
>
> ...The misleading conclusions drawn by the authors were brought about through the selection and use of their control group.
>
> ...The purpose of adjusting rates is to make them comparable. Adjusting a rate r to a population A and another rate to a population B, and then comparing the adjusted rates r and s is contrary to the purpose of adjusting rates, and the resulting comparison does not make any sense. But this is precisely what the authors do when they adjust the prior exposed rate to the prior control group, then adjust the subsequent exposed rate to the subsequent control group, and then compare the adjusted rates. When adjusted rates are to be compared the rates should be adjusted to the same population.
>
> ...The comparisons of rates made by the authors are irrelevant to the hypotheses tested by them and to their corresponding conclusions. (Emphasis added.)

Similarly, McMahon[17/] opens his review of the Infante paper with the following comment:

> ...It is disappointing to see an article of such poor quality as this published in the Lancet. The data are subject to serious criticism on several counts.

and, after detailed point-by-point analysis of the study, presents the following conclusory statement:

> In short, this paper deserves ...no consideration whatsoever in weighing the question of whether there is or is not a genetic risk associated with exposure to VCM. (Emphasis added.)

The foregoing reviews, two of which were available in 1977, completely rebut the scientific validity of the paper by Infante et al. in this matter. Absent a scientifically sound defense of this paper by the authors or their scientific peers, continued citation of this paper is inappropriate.

17/ MacMahon, B. (Professor of Epidemiology, Harvard School of Public Health) Vinyl Chloride and Human Reproduction. Private Communication, 1977. Appendix I.

(3) Experimental Studies of Carcinogenicity and Other Toxic Manifestations For Any Level of Polyvinyl Chloride Exposure. These Effects Should Include, But Are Not Limited to, Mutagenicity, Teratogenicity, Embryo Toxicity, and Other Transplacental Effects As Well As Cytotoxic and Cytogenic Effects on Sperm Cells. To the Greatest Extent Possible, Complete Information Concerning the Industrial Source of the Polyvinyl Chloride, the Size and Characteristics of the Particles, and Exposure Levels or Concentrations of PVC and Residual VC Should Be Included for Each Study.

While no carcinogenic or other toxic effect of inhaled PVC has been demonstrated in animals or man, recent data indicate that, like most nuisance dusts, slight effects may occur in the lungs following high exposures to PVC dust.[18/]

Two reports on PVC dust were presented during the

18/ The American Conference of Governmental Industrial Hygienists in the preface to the 1979 List of Threshold Limit Values for Chemical Substances in the Work Air makes the following statement concerning nuisance particles:

> Nuisance particulates. In contrast to fibrogenic dusts which cause scar tissue to be formed in lungs when inhaled in excessive amounts, so-called "nuisance" dusts have a long history of little adverse effect on lungs and do not produce significant organic disease or toxic effect when exposures are kept under reasonable control. The nuisance dusts have been called (biologically) "inert" dusts, but the latter term is inappropriate to the extent that there is no dust which does not evoke some cellular response in the lung when inhaled in sufficient amount. However, the lung-tissue reaction caused by inhalation of nuisance dusts has the following characteristics: (1) The architecture of the air spaces remains intact. (2) Collagen (scar tissue) is not formed to a significant extent. (3) The tissue reaction is potentially reversible.

course of the Symposium, that is, the papers by Groth and Wagner. The paper by Frongia, et. al., cited as reference no. 3 in the December 18, 1979 Notice, is one of many publications which deal with experimental exposure of test animals to vinyl chloride. In addition, SPI is aware of feeding studies conducted either with polyvinyl chloride resin, polyvinyl chloride copolymer resins, or low molecular weight fractions extracted from commercial PVC resins and/or copolymers. Two of these reports, one done at Harvard and one by Smith et al., are attached as Appendices J and K. Other reports are in the files of the Food and Drug Administration (FDA) and efforts are being made to obtain copies. As soon as they have been released to us, copies will be sent to the Docket Officer.

With regard to the papers presented by both Groth and Wagner, the specific nature and particle sizes of the samples used are not completely clear. It is essential that when written versions of their papers become available for study, these deficiencies be eliminated. For the present, we can only assume that fine particles (dispersion resins) were used. As noted by Wagner, these may contain surfactants or other suspending agents. Hence, their use may result in effects which were misattributed. As stated by Dr. Wagner, he has not been able to reproduce the positive results of his early work despite repeated attempts.

Nevertheless, taking the reports at face value, both studies appear to be consistent in demonstrating that exposure

of experimental animals to respirable PVC dust results only in a low level of toxic response and in the retention of dust particles in the lungs with the development of macrophage aggregates, a normal clearance mechanism used by the lung. It is significant to note that these experimental exposures (at least in the work of Groth with monkeys) did not result in impairment of pulmonary function. Although both authors considered that the dust induced a low-level pneumoconiosis[19/] in the exposed animals, their reports did not indicate the presence of fibrotic tissue which is observed in acute pneumoconiosis resulting from exposure to other substances such as silica.

The observations of Groth and Wagner appear to be quite consistent with the experimental work and observations reported by Frongia and his co-workers. Thus, it appears that in the experimental animals studied, exposure to respirable PVC dust particles results in retention of such dust particles for long periods of time, perhaps for the entire lifetime, and the development of macrophagic responses to these particles.

A major deficiency in all these studies is the ab-

19/ Pneumoconiosis, which literally means dust in the lung, can range from benign effects such as mere storage of particles in the lung to very severe effects such as fibrotic changes and death. It must be emphasized that as used in this response and by the speakers at the March 20-21 Symposium only lung storage and very minimal tissue response were seen. Fibrotic changes were not observed.

sence of a control using another inert "nuisance" dust. Thus, the observations made as a result of exposure to PVC dust may be a characteristic response of many dusts rather than a phenomenon unique to PVC.

As to the ingestion studies undertaken for the purpose of assuring the safety of PVC packaging materials, feeding either the whole resin or the low molecular weight (extractable) fractions showed no observed chronic effects up to the maximum quantities used, i.e. in the range of 5%-10% of the animals' diets. In this connection, one other study should be mentioned, i.e., that conducted by CIVO/TNO in Holland, a copy of which is attached as Appendix L. It is mentioned here only because a porous PVC powder was incorporated into the animals' diets as the vehicle to carry vinyl chloride monomer in a feeding study intended to measure the carcinogenic response of rats to vinyl chloride monomer. In that study, while it was not one directed at PVC, there was no observed effect attributed to exposure to PVC.

Summarizing, except for the feeding studies utilizing PVC and its copolymers, studies which showed no toxic effect attributable to PVC, and those studies discussed above, SPI is not aware of other experimental studies dealing with the toxicity of PVC.

Perhaps comment should be made on the speculative remarks made at the Bethesda Symposium concerning the impact of PVC dust on occupational exposure to vinyl chloride.

The hypothesis is that the dust is carried into the lung and thereby distributed throughout the body. Even were the hypothesis correct, and we think it is not, the quantity of monomer currently contained in PVC is so small that it would make this speculative exposure of no practical significance.

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(4) Epidemiologic Studies of Either Vinyl Chloride or of Polyvinyl Chloride (i.e., cohort, cross-sectional, or case-control)

Because of the importance of this area of information, The Society postponed preparing responsive commentary until the recent Conference to Reevaluate the Toxicity of Vinyl Chloride, Polyvinyl Chloride and Structural Analogs had been held. Prior to this Symposium it had been speculated that, perhaps, some new epidemiologic data or information might be presented concurrently with the industry-sponsored presentation of Dr. W. Clark Cooper. This, of course, did not happen. In fact, it is The Society's view that nothing new in this area was presented at all.

However, because of the Agency's interest in the subject area, SPI concluded that a detailed review of the existing epidemiology would be in order and appropriate for filing with this OSHA Docket in response to the request for this information. Accordingly, The Society has contracted for a complete, independent analysis of all the relevant epidemiologic data. Considering the magnitude of this project, it is obvious that it will not be finished by the May 9, 1980 deadline associated with the Request; hence these materials will be submitted when completed. It should be pointed out that, as is apparent from papers presented at the Bethesda Symposium, a factor complicating the preparation of this analysis involves the fact that it is not always

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made clear in subsequent papers reviewing them that many of the studies which have been reported represent repeated investigations of the same population or subsets thereof.

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(5) Case Reports and Case Series of Brain, Lympho-hematopoietic, Lung, and Liver Cancers By Facility and Relevant Demographic Variables, Such As Age, Sex, Race, Date of Diagnosis, Date of Death, Date of First Exposure, and Length of Exposure For Either Vinyl Chloride or Polyvinyl Chloride

At present, The Society is unaware of any information responsive to request #5 which is not already contained in the published literature.

However, since the papers prepared by Dr. John Stafford of Imperial Chemical Industries, Ltd. were not considered appropriate for presentation at the above-referenced Symposium, it has occurred to SPI that the Agency might care now to have a copy of the most recent compilation of data on the world-wide incidence of angiosarcoma of the liver. Dr. Stafford's tables, copies of which have already been supplied (and, indeed, have been supplied on an ongoing basis) to the Director of the National Institute for Occupational Safety and Health, are a complete compilation of all liver angiosarcoma deaths due to exposure to vinyl chloride monomer through January, 1980. Appendix M. As can be seen from a review of these data, there have been only 42 cases of liver angiosarcoma reported in the last five years and a total of 84 cases reported to date.

For background information on how Dr. Stafford came to be interested in keeping a register of the liver angiosarcoma cases refer to the attached correspondence between Dr. Stafford and Dr. Robbins of NIOSH. Appendix. N.

(6) Mutagenicity Study Results of Vinyl Chloride or Polyvinyl Chloride As Measured By Analysis of Human Body Fluids, e.g., Direct Mutagenic Testing With Peripheral Blood Lymphocytes, Non-Disjunction In Humans With YFF Sperm Test And *In Vivo* Cytogenetics

It has been demonstrated that if mutagenic effects occur, as measured by circulating lymphocytes, they were the results of high exposures; the measured effects have not been permanent changes since they were reversible. The effects did not occur at lower levels of exposure. The clinical significance is not known.

The attached report by Hansteen, Hillestad, Thiis-Evensen and Heldaas is extremely important. Appendix O. The mean chromosome-breakage frequency, which was significantly higher than controls when analyzed in 39 workers in 1974, was found on subsequent reexamination 2-2.5 years later to be no higher than the controls. Hence, whatever "effects" may have been measured or whether they were related to high exposure to vinyl chloride or not, the "effects" reverted to normal when exposures to vinyl chloride (and possibly other materials) were reduced. It is our understanding that similar results on a similar study are being prepared for publication by ICI in England and that they will supply a copy of the manuscript when it is available.

Picciano *et al.* found no effect in a study of men producing vinyl chloride monomer and those workers probably had lower levels of exposure than those above. Appendix P.

They summarized their data as follows:

> This report presents cytogenetic findings from a group of 209 workers employed for up to 28 years in the manufacture of vinyl chloride monomer at the Texas Division of Dow Chemical U.S.A. Cytogenetic evaluation results from this group were compared to results found in examination of individuals being considered for employment. Statistical analyses were performed on a group basis for chromatid aberrations, chromosome aberrations and proportion of abnormal cells; no statistical difference of significance was found between the two groups. Comparison of these results with reported studies suggests that the level of cytogenetic aberrations in vinyl chloride workers is probably related to the length and level of exposure, and that risk of adverse genetic effect can be avoided in controlled, minimal-exposure environments.

Fabricant attempted to summarize the mutagenic studies on vinyl chloride at the Symposium. This report merely reviewed the collected, previously-published information; nothing new was presented.

The Fabricant report pointed out that in the U.S. genetic anomalies resulting in infant mortality increased from 5% in 1915 to 15% in 1965. She alleged that 33% of pediatric hospital admissions are due to genetic defects and that 80% of all clinical mental retardation in the U.S. arises from genetic causes. She speculates that these observations are due to "an environmental component" creating birth defects. No mention is made of higher incidence as a result of the use of improved diagnostic tools or longer survivership of affected infants.

This author also relied on the discredited paper

by Infante et al. This publication (reference 12 in the December 18 Notice in the Federal Register) alleging reproductive effects in wives as a result of mutagenic changes in husbands who were exposed to vinyl chloride has been discredited because of faulty methodology. See Appendicies G, H, and I. The faulty nature of this study has been repeatedly called to OSHA's attention and, therefore, absent a sound scientific defense of this study by the authors or their scientific peers, Fabricant should not have cited it.

(7) Body Burden Measurments
of Vinyl Chloride In Humans

The meaning of this specific request for information is not clear. Being a poorly soluble gas, vinyl chloride does not accumulate from chronic exposure but, rather, is rapidly exhaled after exposure ceases. Hence, there can be no body burden of vinyl chloride as the term is usually applied to the accumulation of heavy metals, large organic compounds or radio nuclides.

Baretta, Stewart and Mutchler studied this subject and reported on "Monitoring Exposures to Vinyl Chloride Vapor: Breath Analysis and Continuous Air Sampling." American Industrial Hygiene Association Journal, Vol. 30, November-December 1969. The data in this study clearly shows a rapid decay in the expired air in industrially exposed employees and laboratory subjects when exposure to VCM ceases.

Published metabolic studies on laboratory animals[20/] and human subjects also indicate rapid excretion of vinyl chloride in expired air. This, again, is consistent with the high volatility and low solubility of vinyl chloride gas.

20/ Watanabe, P.G., McGowan, G.R. and Gehring, P.G., Toxicol. and Appli. Pharmacol. 36:339-352 (1976).

(8) For Operations Involving Polyvinyl Chloride, the Types of Resin in Use, the Concentration of Vinyl Chloride Trapped in the Resin, and the Concentration of VC and PVC Dust in the Atmosphere Where Individuals Are Working. This Information Should Include Estimates of the Particles Sizes and Concentrations of Particles That Fall Within the Respirable Range

and

(9) In PVC Bagging and Milling Operations, Atmospheric Levels of PVC and VC, Monitoring Devices Used to Detect VC, and the Type of Respirator Protection Program for Individuals Working in These Operations. This Should Include the Sensitivity and Validity of the Analytic Techniques in Use

Information supplied to answer the inquiries cited above is likely to create confusion if used out of context. Past regulatory effort on the part of OSHA has resulted in the promulgation of regulations that covered resins containing any amount of contained vinyl chloride and materials that contained little or no vinyl chloride monomer; therefore it appears necessary to call OSHA's attention to all aspects of the industry.

Operations involving polyvinyl chloride are broken down into the following sections:

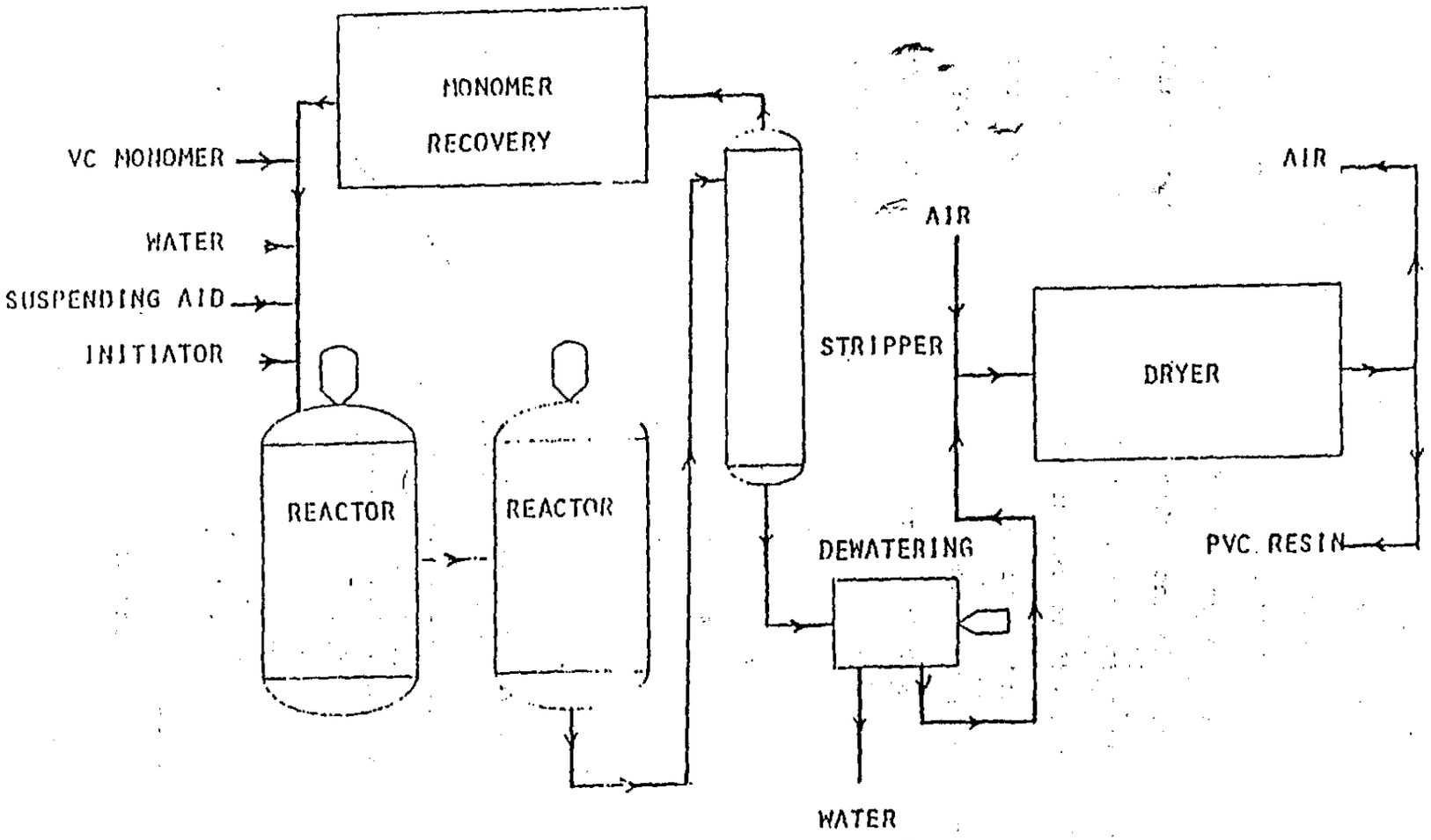
1. Manufacture of PVC
2. Distribution
3. Compounding
4. Fabrication into semi-finished materials
5. Conversion of semi-finished materials to finished products

The manufacture of synthetic resins (PVC) from vinyl chloride and other monomers involves reacting these monomers in agitated pressure vessels in the presence of catalysts in such a way that these liquids and/or gases are converted to solid resins. A considerable amount of heat is generated by the reaction. This is removed by cooling the vessel. As the monomer is converted to polymer during the reaction, the rate of reaction slows down. The unconverted monomers are removed from the reacting mass by heat and vacuum, and the resin (PVC) is recovered as a dried powder or as a latex or solution. The polymerization reaction may take place in pure monomer, in a solution, in a water-monomer emulsion or in a water-suspension of monomer. The nature of the polymerization process determines the nature of the subsequent recovery process and the nature of the resin particles produced.

Currently there are four basic vinyl chloride polymerization techniques;

1. Suspension
2. Emulsion/Dispersion
3. Bulk
4. Solution

Suspension polymerization is the major process used for the manufacture of PVC resins and is used for about 82-85% of U.S. production. (Figure 1). It involves the charging of one or two parts water and one part vinyl chloride



SUSPENSION POLYMERIZATION PROCESS

FIGURE 1.

monomer or co-monomer mixture to an agitated reactor along with initiator and suspending agents. The mass is reacted at 50 to 65°C until about 85-90% of the monomer is converted to resin. The resin-water mixture is then heated, sometimes under vacuum, until the unconverted monomer is substantially removed. The resin is then removed from the water and dried in rotary, flash or fluid bed dryers by exposure to heated air. The dried resin is transferred to storage silos prior to shipment in bulk containers or in paper bags. If only vinyl chloride monomer is reacted, the product is homopolymer PVC. If a monomer such as vinyl acetate is mixed with the vinyl chloride, then a co-polymer PVC is produced.

Resin products of the suspension resin process are many and varied in terms of molecular weight, composition, particle size, monomer retention, heat stability and ultimate use. A general description of the various grades of resins follows:

(a) General purpose homopolymer for flexible materials such as shower curtains and wire insulation are porous, large particle (~100 microns or more diameter) size, high molecular weight resins with good heat stability and low monomer retention characteristics. These resins contain essentially no particles less than 10 microns in diameter and the residual monomer content is in the range of non-detectable (N.D.) to 25 ppmw.21/ Particle porosity is a critical property.

21/ Particles 10 microns or larger in diameter are considered non-respirable.

(b) Homopolymer resins for pipe manufacture are dense, large particle size (~100 microns in diameter), high molecular weight resins with good heat stability. Monomer retention characteristics are medium to low. These resins contain essentially no particles less than 10 microns in diameter and residual monomer content is in the range of N.D. to 30 ppmw. Generally the lower the molecular weight the higher the monomer retention. Particle density is a critical property.

(c) Homopolymer resins for rigid extruder shapes and calendered rigid sheeting are dense, medium particle size (~80 microns in diameter), medium molecular weight resins with reasonable heat stability and medium monomer retention characteristics. These resins contain essentially no particles less than 10 microns in diameter and the residual monomer content ranges from N.D. to 50 ppmw. Particle density is desirable and porous particles are difficult to produce.

(d) General purpose copolymer resins for rigid sheeting, molding and flooring are medium particle size (~80 microns in diameter), glassy to low porosity, low to medium molecular weight, and high in monomer retention with poor heat stability. Comonomer content ranges up to 20%. The monomers most often used are vinyl acetate and vinylidene chloride. These resins contain essentially no particles less than 10 microns in diameter and residual monomer in the range of 100 to 300 ppmw. The poor heat stability coupled with the glassy particles make vinyl chloride monomer removal difficult. Copolymer resins offer ease of fabrication and have the ability to take up high loadings of fillers and pigments.

(e) Copolymer resins for coating applications are low molecular weight, medium particle size (~75 microns in diameter), glassy non-porous particles, poor in heat stability and high in monomer retention. Co-monomer content ranges up to 40%. These resins contain few particles less than 10 microns and residual vinyl chloride monomer contents of 25 to 100 ppmw.

(f) Plastisol additive resins for dispersion resin fabrication are medium molecular weight, small particle size (~25 microns in diameter), low in particle porosity, and medium in heat stability materials. The addition of these resins to a dispersion resin plastisol reduces the plastisol viscosity and reduces the product cost. The ability to perform uniformly in a plastisol is critical. These resins contain appreciable quantities of particles smaller than 10 microns in diameter and contain N.D. to 25 ppmw of residual vinyl chloride monomer.

Suspension process plants are generally meeting the permissible personal exposure limit of 1 ppmv TWA_8 imposed by the OSHA Standard but are having difficulty meeting the 5 ppmv ceiling value without the use of respirators. Certain tasks, such as maintenance, cleaning and correcting process upsets, involve personnel exposure to brief periods of high vinyl chloride concentrations. These personnel are protected by continuus air-supplied respirators. Elimination of all use of respirators cannot be considered a realistic requirement.

Suspension PVC, except for plastisol additive resins, presents no hazard from exposure to respirable dusts since these resins contain essentially no particles less than 10 microns in diameter. Indeed, except for the plastisol additive resins, the use of cyclone collectors on resin dryers and resin transfer systems obviates the presence of such particles in suspension resins, i.e., such particles are lost in the dryer exhaust.

SUSPENSION POLYMERIZED PVC
MAGNIFIED 50 TIMES

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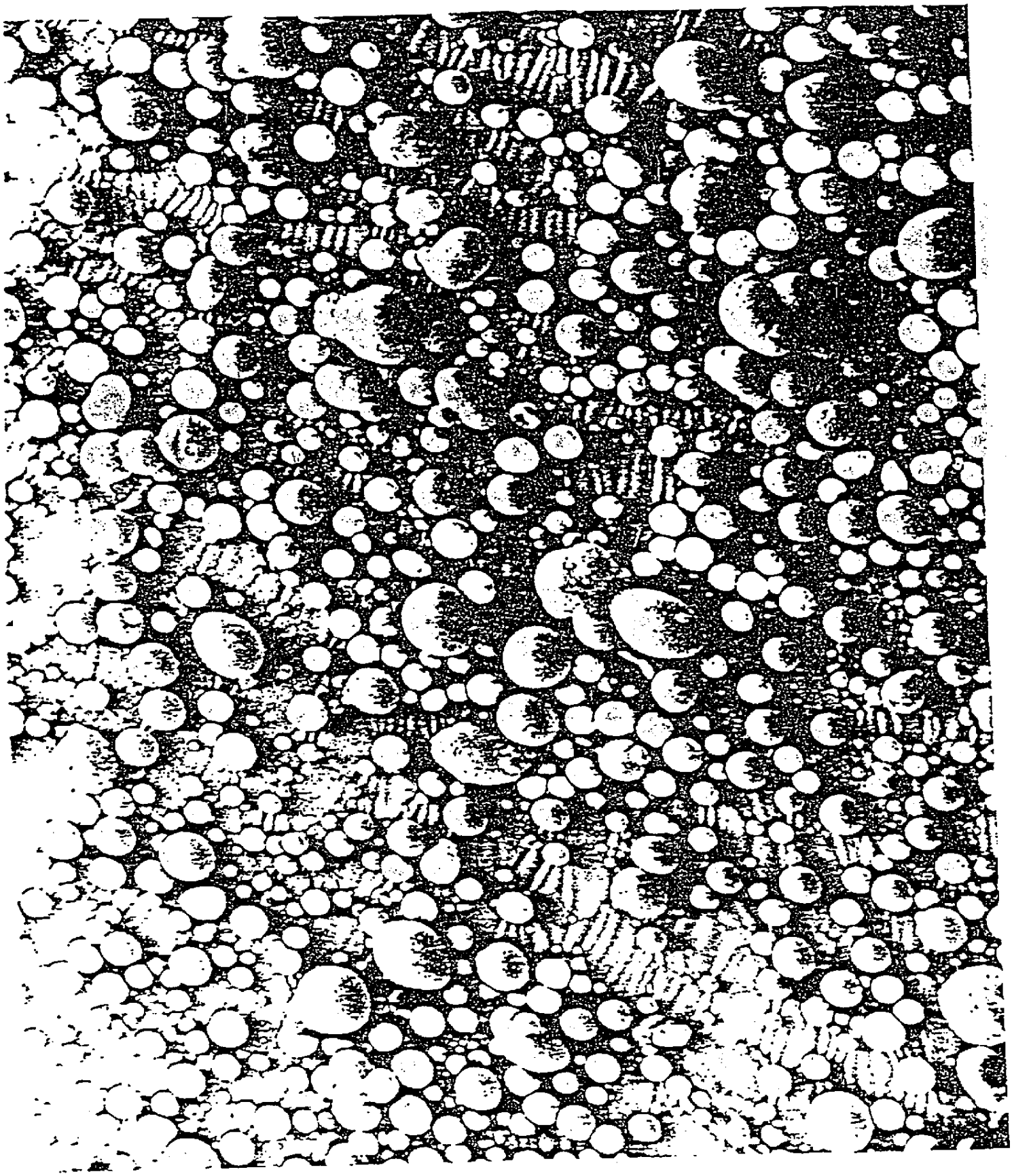

SUSPENSION POLYVINYL COPOLYMER
PVC RESIN MAGNIFIED 50 TIMES

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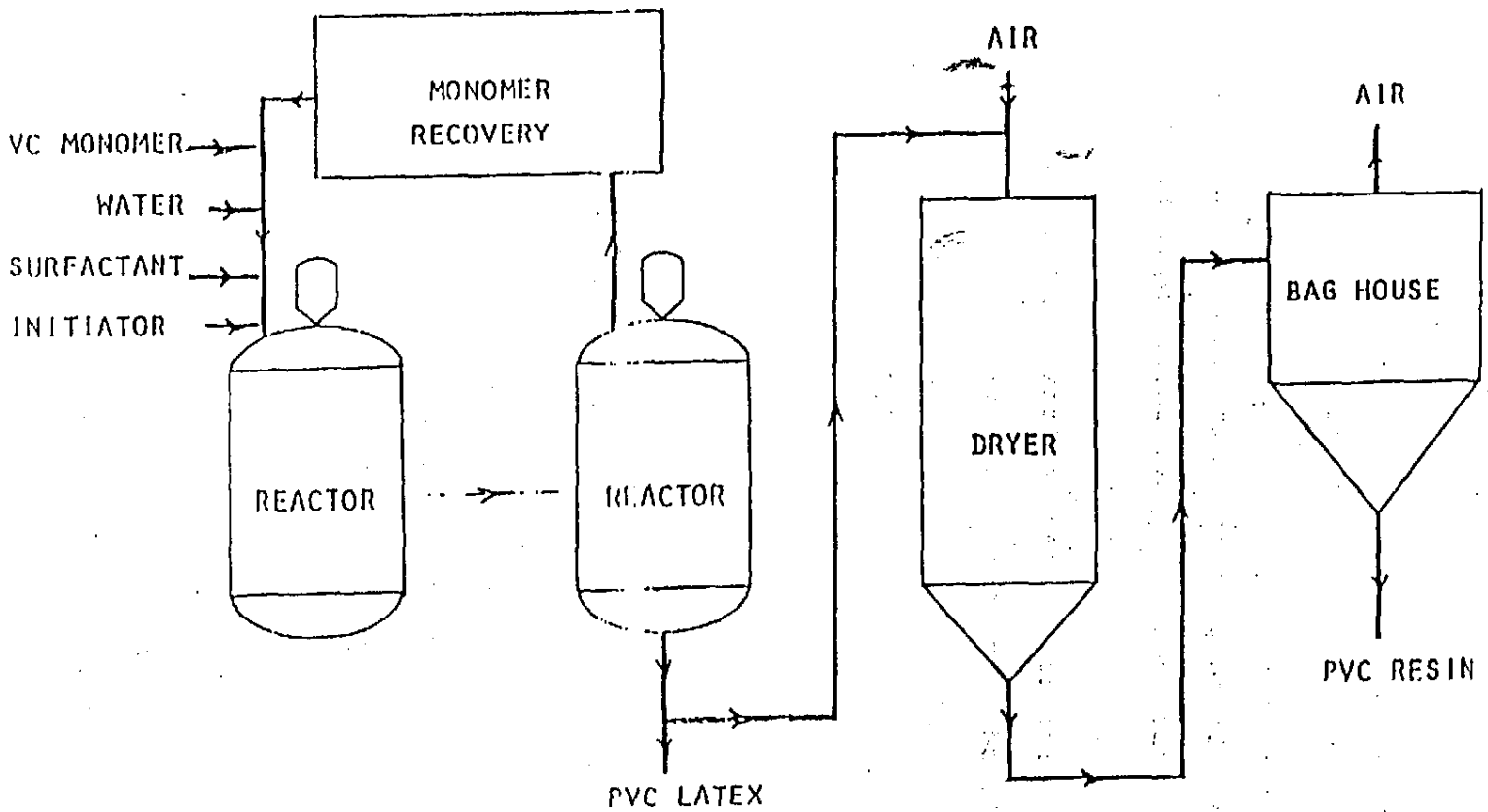
Emulsion/dispersion polymerization is the second most widely used process for the manufacture of PVC resins and amounts to 10-12% of total U.S. production. One of the important things to understand about emulsion polymerization is that it is not a single process but a family of processes, each producing specialized products that are defined or specified in terms of performance in a particular application. In the interest of brevity only the two major process families, the water soluble initiator system and the oil-soluble initiator system, will be discussed. (Figure 2).

In the water soluble initiator system one to two parts water, one part monomer, 0.01 to 0.03 parts surfactant and water-soluble initiator (a redox system or a persulfate salt) are charged or fed to an agitated reactor and reacted at 30 to 60°C to form a latex emulsion. The reactor agitation must be sufficiently vigorous to emulsify the monomer-water mixture but not so vigorous that the latex is destabilized. When 80 to 95% of the monomer is converted to polymer the latex may be gently stripped of the unconverted monomer with heat and vaccum or it may be subjected to a second initiator treatment and reacted to essentially 100% monomer conversion. The product of this polymerization may be simply filtered and shipped to consumers as a latex for use in the manufacture of coatings, mastics, laminations, and the like, or the polymer may be recovered as a dry resin. Recovery techniques vary. The most commonly used is to spray-dry



EMULSION POLYMERIZATION PROCESS

FIGURE 2

the latex, though some resins are recovered by coagulating the latex and dewatering with subsequent drying of the coagulum (resin).

In the oil soluble initiator system one part monomer containing an organic peroxide is emulsified in one to two parts water containing 0.01 to 0.03 parts surfactant. The resulting emulsion is reacted at 30 to 60°C to form a latex. From this point onward the oil soluble initiator system is similar to the water soluble initiator system.

The choice of process depends to a great extent on the ultimate use of the resin. If resin particle size is critical the oil soluble initiator system is preferred. The resin particles formed during emulsion polymerization range in size from 0.05 to 2 microns. In the course of recovery and drying these may be agglomerated to particles as large as 30 microns. For some uses the agglomerates may be ground to a median particle size of about two microns. The small particle size results in rapid loss of residual vinyl chloride so that the resin contains only about 1 ppm free monomer. Because of handling problems nearly all the resin is bagged. In this process, the small amount of monomer retained easily escapes before the resin arrives at a fabricating plant. Some proprietary dispersion resins, however, have material handling properties comparable to larger particle size suspension resins. These can be shipped, stored, conveyed and processed in bulk instead of in bags.

Resin products of the emulsion process are more difficult to list because most products are sold on a performance basis as opposed to suspension products which are sold largely on specification and price. A rough classification is as follows:

(a) Plastisol resins are the major emulsion process products. These are very small particle size (2 to 3 micron diameter), non-porous, very high molecular weight, 100% vinyl chloride resins with good heat stability and low monomer retention characteristics. These resins consist of particles less than 10 microns in diameter, contain significant amounts of surfactants and about 1 ppmw of residual vinyl chloride monomer. When stirred into approximately 60 parts of plasticizer per hundred parts resin, these resins yield a viscous liquid plastisol which, when baked, fuses to a flexible vinyl plastic.

(b) Foam resins are substantially the same as the plastisol resins except that they are medium molecular weight instead of high. The lower molecular weight yields a plastisol which fuses near the temperature at which a blowing agent yields up its gas. This is how a stable plastic foam is formed.

(c) Organosol resins are a special variety of plastisol resin primarily used for metal coating. If a high molecular weight PVC homopolymer dispersion resin is very finely ground and the remaining agglomerates are loosely bound together, it can be blended further with ketone, naphtha, pigments, stabilizers and adhesion promoters to yield a liquid coating material. This organosol is coated on metal and baked to provide a tough, abrasion resistant coating.

(d) Specialty resins are an assortment of materials that make use of the flexibility of the emulsion process for their manufacture. These are made from vinyl chloride in combination with other monomers. The resins are recovered from the latex by coagulation and subsequent drying. Uses of these materials range from film for food packaging to modifiers for other resin fabrication. Further characterization of these

materials is not possible, and they are mentioned here only to call attention to the fact that these important resins are impacted by vinyl chloride and polyvinyl chloride regulations.

(e) Latexes, too, are an assortment of materials generated by the flexibilty of the emulsion process. These are liquid materials containing 40 to 50% resin solids emulsified in water. Latexes containing resin solids high in vinyl chloride content are usually formulated with plasticizers and solvents and applied to a substrate. The substrate is heated or baked to yield a resin film. Latexes containing resin solids low in vinyl chloride content will yield a resin film when the latex coating is simply air dried. These latexes find major use in latex paints, impregnants, mastics and adhesives. Since both types of latexes are used as liquids, there is obviously no possibility of PVC dusts. Depending on the latex produced, most PVC latexes contain less than 10 ppmw of residual vinyl chloride monomer. This monomer is released slowly, and employee exposures in subsequent formulating and use is rarely over 0.5 ppmv.

As is the case with suspension PVC process plants, emulsion process PVC plants are generally meeting the OSHA permissible exposure limit of 1 ppmv TWA_8 for employees. However, these plants, too, are experiencing difficulty meeting the ceiling limit without reasonable use of respirators. The manufacture of dispersion PVC resins represents a source of respirable PVC dusts, and dust respirator use is usually required in dusty locations such as bagging. Certain proprietary dispersion resins do, however, have low dusting properties which greatly simplify control of the workplace dust normally associated with handling dispersion resins.

DISPERSION PVC RESIN PARTICLE
SIZE 1-2 MICRONS - MAGNIFIED
50 TIMES

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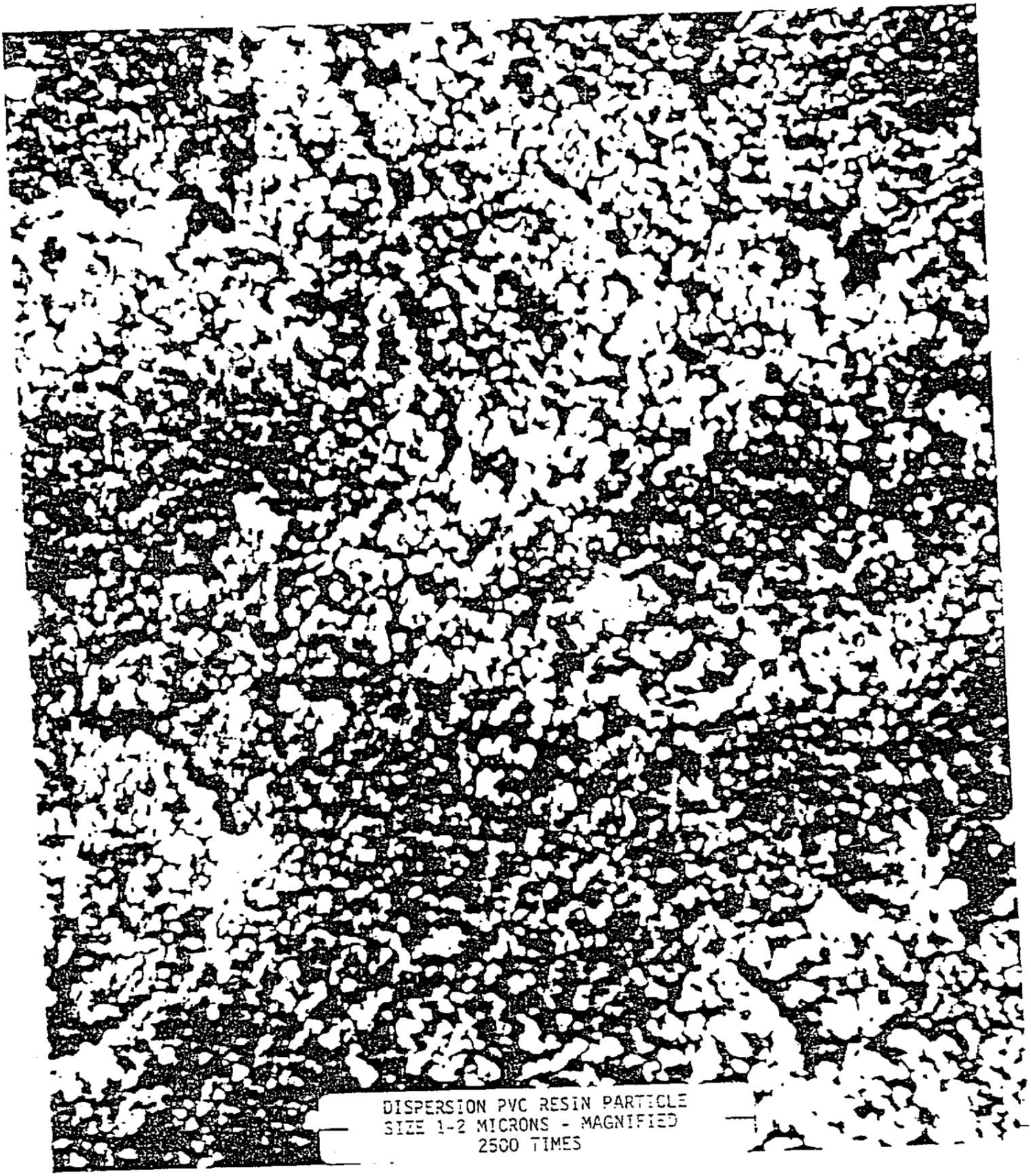
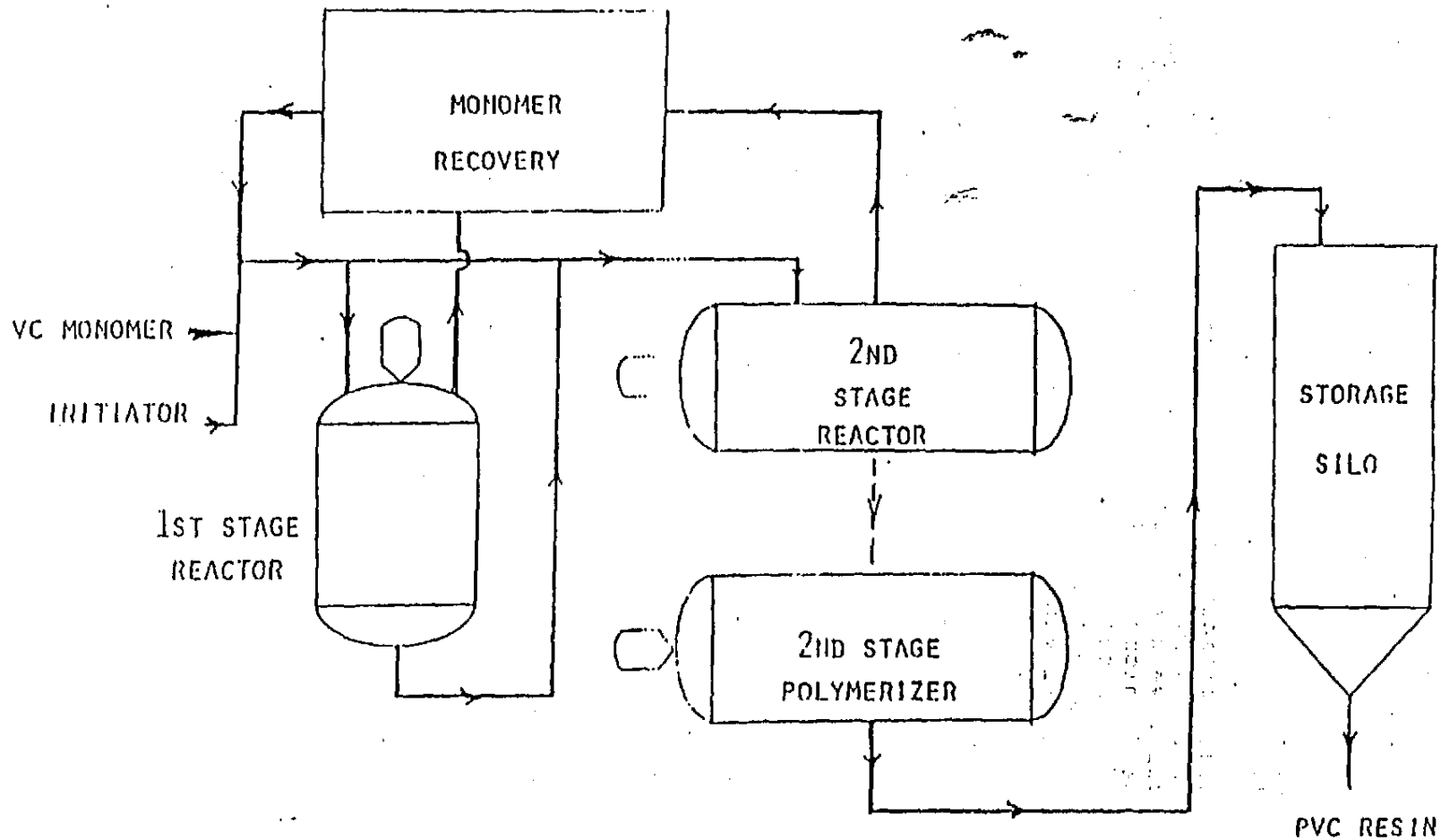


DISPERSION PVC RESIN PARTICLE
SIZE 1-2 MICRONS - MAGNIFIED
2500 TIMES

Bulk polymerization is the third major process in terms of volume for the manufacture of PVC resins but accounts for only about 5% of U.S. production. (Figure 3). The process involves the charging of vinyl chloride monomer and initiator to a first stage polymerizer where about 10% of the monomer is converted to polymer. This batch is then transferred to a second stage polymerizer where additional monomer and sometimes initiator are added. The polymerization is continued until about 80-85% of the monomer is converted to polymer. The unreacted vinyl chloride is removed by heat and vacuum and the finished resin product transferred to storage bins for later shipment to fabricating plants. The absence of water in the polymerization stage eliminates the need for the drying step.

The advantages of the bulk process are its simplicity, the uniformity of the resin particle size, the high porosity of the resin particles, the purity of the polymer (no soaps or suspending aids) and the granular, low dustiness of the product. The disadvantages are less flexibility in product mix (homopolymers only) than the suspension process and poorer removal of residual monomer. These bulk process resins are competitive with suspension process PVC homopolymers.

Bulk process PVC resins are used in applications where clarity of the finished plastic, uniform resin particle size and high resin particle porosity are desired. A simple classification of their uses is as follows:



BULK POLYMERIZATION PROCESS

FIGURE 3

(a) Rigid and flexible plastic materials produced by extrusion and calendering requires resins with large porous particles (100 microns in diameter), good heat stability and low monomer retention. These medium molecular weight homopolymer resins contain essentially no particles less than 10 microns and the residual VC monomer content is less than 50 ppmw.

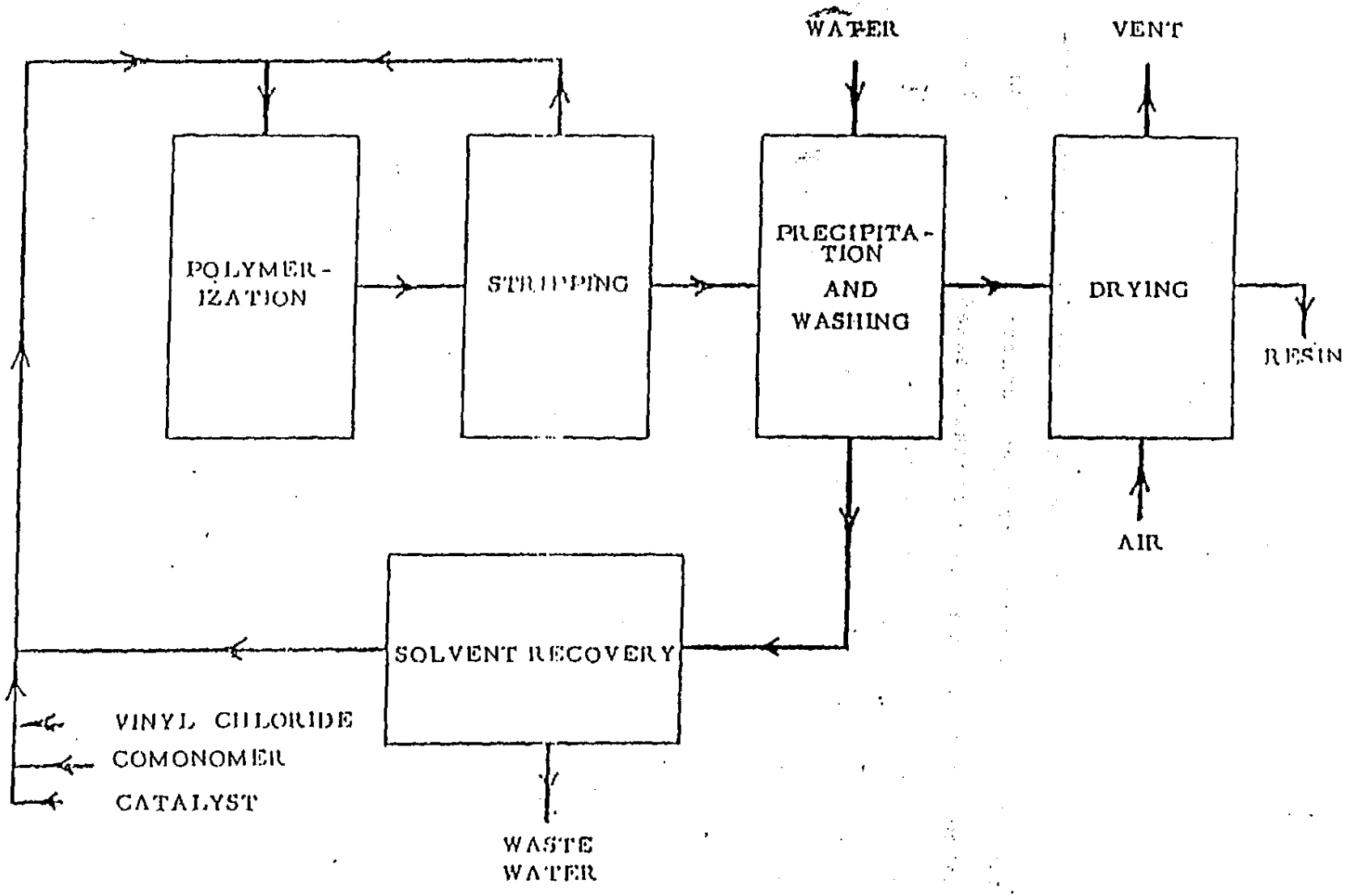
(b) Rigid plastic moldings, plastisol additive resins and powder coatings are made from low molecular weight homopolymers with a particle size of 75 microns. Particle porosity is good and monomer retention is low. There are few particles less than 10 microns in diameter and residual vinyl chloride content is less than 50 ppmw.

Bulk process plants have the same general types of personnel exposures to vinyl chloride noted for suspension process plants.

Solution polymerization is a process unique to one company and accounts for about two percent of the total resin produced. (Figure 4). Vinyl chloride monomer, co-monomer, solvent and initiator are fed to a continuous reactor system. The polymer formed is soluble in the reacting mass so that the reactor product is a viscous resin solution. This solution is distilled to remove the unconverted monomers and the resin is recovered by treating the resin solution with water and drying the product. The resin particle is very porous, is always a copolymer, is free of soaps and suspending agents, has a median particle size of 75 microns and contains less than 0.2 ppm residual vinyl chloride. Solution process PVC resins are very often confused with suspension process resins for solution application. Molecular weight

FIGURE 4

SOLVENT VINYL RESIN PROCESS



of these resins ranges from low to very low to achieve solubility in solvents. Essentially all these materials are used for coatings in combination with other resinous materials.

The solution PVC resin process, because of its nature, exposes personnel to vinyl chloride concentrations below 0.5 ppm. The resin contains essentially no particles less than 10 microns in diameter.

In addition to the resins produced by the four basic processes there are resins which are chemically modified in a second operation after the resin has been produced. The major product in this area is chlorinated PVC. In the coatings area copolymer resins are often modified through reactive sites in the co-monomer. These materials are mentioned only to note their existence and to point out that the very nature of chemical modification and subsequent processing removes all possibility of their release of vinyl chloride in subsequent applications.

Distribution of PVC resins poses some potential for personnel exposure to vinyl chloride monomer and to polyvinyl chloride. The structure and the economics of the distribution system have acted to cause such exposures to be minimal except for certain special situations.

Suspension and bulk process PVC resins, except for plastisol addition powder coating, solution coating and ex-

port sales move from the producing plant to the user via bulk carriers such as rail cars (containing approximately 150,000 pounds) or hopper trucks (containing approximately 35,000 pounds). These carriers are loaded via gravity from overhead bins or by pneumatic conveyor. The carrier is sealed during transit. On arrival at the delivery site, the carrier is unloaded by pneumatic conveyor to a storage silo. There is some vinyl chloride monomer emitted during the loading and unloading process, but personnel exposure is well below the OSHA Standard "action level". Monitoring of this operation when the resin contained 800 ppmw of residual vinyl chloride monomer showed personnel VCM exposures of approximately 1 ppmv TWA_8. Although there is no recent data, with current residual VCM levels of approximately 50 ppm, one would expect personnel exposures to be down around 0.1 ppm TWA_8. There is some resin dusting and spillage in the loading and unloading process, but the relatively large particle sizes and remote contact by personnel obviate any potential hazard.

The bagging of the 5 to 10% of suspension and bulk process resins that are handled in this fashion is highly automated and messy, but personnel exposure to dusts or VCM are negligible. Resin is delivered to the packaging machine via gravity or pneumatic conveyor. The packaging machine operator inserts the filling tube into a filling valve-type kraft paper bag and actuates the machine to fill the bag. The filled bag drops off the filling tube to a conveyor and

the operator repeats the operation with another bag. Filled bags are automatically or manually stacked on pallets which may be stored in a warehouse awaiting shipment or may be loaded on a truck or railcar immediately.

When the filled bag drops off the packing machine a small puff of resin and air is emitted from the bag valve as it closes. Subsequent handling of the bag causes some shifting of resin out of the valve. There are also faulty bags which may break on filling. Thus, the resin bagging area is usually messy. However, monitoring conducted in these areas indicates personnel vinyl chloride exposures are less than the OSHA Standard "action level." Dust measurements for suspension and bulk resin bagging show less than 1 mg/m^3 respirable dust.

Bagging and storage of suspension and bulk process resins causes a significant reduction in the residual vinyl chloride content of the resin. Tests of moderately porous medium molecular weight resins show a reduction of over 50% in free monomer after one week. Handling and transport of suspension and bulk PVC resin in bags via truck or rail car appears to present no unusual hazards if normal work space ventilation standards are met in storage, loading and unloading resins.

Except for some special situations where bulk transport or fiber drums are used, essentially all emulsion process PVC resins are packaged in kraft paper multi-wall bags. The bagging operation is essentially the same as that de-

scribed for suspension and bulk process resins. Vinyl chloride exposure is less than the OSHA Standard "action level." The NIOSH study by Mr. J.H. Jones in 1974 indicated that exposure is not likely unless it comes from emissions in adjacent operations.22/ Dust exposure is potentially a problem due to the resin's small particle size but local ventilation can control this readily. Current dust monitoring shows respirable dust less than 5 mg/m^3.

The storage and truck shipment of emulsion PVC resins offer minimal vinyl chloride exposure opportunities for personnel.

Warehouse and closed truck vinyl chloride monitoring show levels well below 0.5 ppmv vinyl chloride in the air.

Solution process PVC resins are also packaged primarily in kraft paper multi-wall bags. Bagging is essentially the same as described for suspension resins. The NIOSH study in 1974 indicated vinyl chloride exposure to baggers of N.D. to 0.7 ppmv and total dust of less than 2 mg/m^3. Vinyl chloride exposure currently is about 0.1 ppmv with no change in total dust exposure. Respirable dust is about 0.1 mg/m^3.

Monitoring of storage and truck shipment of solution process PVC resins usually shows undetectable amounts of

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22/ Jones, J.H., Worker Exposure to Vinyl Chloride During Production and Fabrication of Vinyl Chloride and Polyvinyl Chloride. Study conducted with the Bendix Corporation under NIOSH Contract CDC-99-74-50. Draft of this Report is at Appendix Q. See p. 36.

vinyl chloride monomer.

Polyvinyl chloride latexes are most often handled and shipped in tank cars or trucks and in fifty-five gallon drums. There is some emission of vinyl chloride during loading and unloading operations but, if the latex contains 10 ppmw residual vinyl chloride or less, the personnel exposures are less than 0.5 ppmv. Dust is not a problem with latex shipments.

Compounding a PVC resin or latex means the mixing of that material with pigments, stabilizers, plasticizers, fillers and other additives to yield an intermediate material for subsequent conversion into finished or semi-finished products. In major plastics operations this is simply a single process step in a large integrated factory, but in the specialty resin area, as well as with small plant operations, a compound is produced by one plant and is subsequently shipped in a container to the user. Thus, in terms of regulation, these materials are classified as polyvinyl chloride. Often, however, the properties of the material are completely dissimilar to the PVC resin raw material, i.e., the vinyl chloride monomer content is diluted or reduced by heating and dustiness is reduced sharply or eliminated completely. Some examples are pelletized PVC compounds, powder blends, organosols, plastisols, lacquers, dry blends, and latex paints.

The purpose of compounding is to combine the resin and additives into a homogeneous state suitable for further

processing. The level of compounding ingredients employed for PVC is substantially higher than for most plastics. Resin content of a compound may vary from 99% for rigid sheeting to 15% for floor tile. Some compounding ingredients introduced in PVC fabrication are as follows:

(a) Plasticizers are high boiling temperature esters of phthalic acid, adipic acid, azelaic acid, phosphoric acid and so on. Epoxidized oils, epoxy resins, and polyester resins are also used.

(b) Heat stabilizers are compounds of lead, barium, cadmium, tin and zinc. Epoxies and phosphites are also used.

(c) Fillers consist of clay, talc, mica, asbestos, calcium carbonate, titanium dioxide, diatomaceous earth and barytes.

(d) Pigments may be inorganic compounds, such as titanium dioxide, chromium oxide, ultramarine blue or molybdate orange, or they may be organic compounds, such as phthalocyanine, quinacridone and benzidine salts.

(e) Modifying resins improve processing characteristics and impact resistance. These are acrylate, ABS, and chlorinated polyethylene resins.

(f) Lubricants make subsequent processing easier. Some of these are waxes, stearic acid and metal stearates.

(g) Light stabilizers provide ultra-violet light resistance. Examples are benzotriazoles or benzophenones.

(h) Fungicides give resistance to fungal attack. Materials commonly used are amines, arsenates and organotins.

(i) Flame retardants, such as antimony oxide and the boranes, are used where plasticizer content is high.

(j) Anti-static agents reduce dust and dust pickup. These are often amine compounds.

(k) Brighteners yield a whiter, clearer film.

(l) Anti-oxidants, such as bisphenol A, are sometimes used.

Additionally, particularly in the coatings area, there are specialized additives such as surfactants, pigment wetting aids, solvents, and diluents.

These compounding ingredients and additives are mentioned in some detail to show the difficulty of isolating causative agents if an employee health problem is believed to exist in the PVC fabricating area. It is certainly a mistake to attribute dust problems solely to PVC dust and toxic vapor problems solely to vinyl chloride monomer.

Compounding PVC resins for subsequent fabricating via calendering, extrusion or molding involves two basic steps; dry or powder blending followed by melt compounding. The choice of equipment is important because variations in the homogeneity of the resin compound may cause serious deficiencies in processing and in the finished product. The calendering, extrusion and molding operations involve large scale operations with suspension and bulk process PVC resins which are almost always handled in bulk. Resin is charged from a bulk silo to a weigh hopper and dumped into the mixer. Plasticizers are metered into the charge. Other additives are charged by similar techniques though small volume additives may be first mixed into a portion of the plasticizer.

The mixer is closed and heat is applied externally or generated by the shear action of the mixer itself. The charge is subjected to mixing and heat (approximately 200-250°F) for some predetermined time or until the mass is a dry powder. Gases from the charge are vented outside the work area. On completion of the dry or powder blending, the mix may be cooled and stored for subsequent use or charged directly to a high intensity mixer or to an extruder.

The mixers and/or the extruder melt the dry or powder compound blend to secure a hot plastic mass. For calendering, the mixer or extruder charge is transferred to the calender where it is formed into flexible film or into rigid sheets as semi-finished products. For extruded products, the extruder may simply be fitted with a die, as in the manufacture of pipe, to form the finished product. The industrial hygiene survey carried out by NIOSH in 1974 and 1975 indicated that vinyl chloride monomer exposures for this type of operation in the range of N.D. to 0.6 ppm. (See attached Table 33 from the NIOSH report). With the current low levels of residual vinyl chloride monomer in suspension and bulk process PVC resins, vinyl chloride monomer exposure is negligible.

Dust is a problem in calendering, molding and extrusion processing but as Mr. Jones points out in his NIOSH report the dust is probably not all PVC since fillers and other additives are used. Mr. Jones does not mention, however, that plasticizer aerosols formed during processing