



Chevron Environmental Health Center, Inc.

Richmond, California
November 14, 1985

FILE COPY

**CHEMICAL INVOLVEMENT/PARKINSON'S
DISEASE**

105,3 PARAQUAT TECH. - 62J,
* 81.0

J. N. SULLIVAN
Chevron Corporation

Attached please find the Environmental Health Center's response on Parkinson's Disease for transmittal to Mr. Follis. If you have any further questions, please feel free to contact me.


J. N. OSPENSON

KDM-1/NBI

Attachment

cc: R. D. Cavalli
J. A. MacGregor
Files - w/attachment

CHEMICAL INVOLVEMENT/PARKINSON'S DISEASE

The article in the July 19, 1985 issue of Science raised the general allegation that environmental factors, primarily pesticides, may be implicated in causing Parkinson's Disease. Paraquat was specifically named in the article because of its structural similarity to a chemical known to cause Parkinson's Disease, MPTP. There is no scientific evidence to support a cause and effect relationship between exposure to paraquat and development of Parkinson's Disease. In fact, considerable evidence is available to refute such a claim. Several important facts are summarized below:

1. Entry of paraquat into the nerve cells of the portion of brain effected by MPTP is impeded by two mechanisms. First, positively charged chemicals like paraquat are excluded from most regions of the brain by a specialized layer of cells called colloquially, the "blood-brain barrier". On the contrary, a neutral molecule like MPTP passes easily through this "barrier" into the brain.

Secondly, after entering the brain, MPTP is metabolized to a chemical which can be selectively accumulated by the brain cells through a monoamine uptake system. Because paraquat is a diamine compound it cannot be selectively accumulated by that cell type.

2. Lifetime feeding studies with paraquat in rats and mice have not illicited any clinical signs of Parkinson's Disease in the test animals. A one-year feeding study in dogs also failed to induce Parkinson-like behavioral changes so easily seen in MPTP treated animals.
3. In epidemiology studies of people occupationally exposed to paraquat, no Parkinson-like syndrome has ever been reported.
4. Ingestion of paraquat by individuals in quantities sufficient to cause renal and pulmonary toxicity has not illicited any signs of Parkinson's Disease.
5. The distribution of Parkinson's Disease in Canada is thought to not be coincident with the pattern of paraquat usage. Currently, further work is being funded by ICI in this area. ICI will map paraquat usage in Canada and compare this usage pattern to that reported for Parkinson's Disease.

Several investigators are doing further experiments with paraquat in animal systems which are known to be able to produce Parkinson's Disease from MPTP. Chevron will continue to track this research.

JAM/kdm-1/NBI
sullivan
11/14/85

Case ID: 220300429
Control No.: 26064959

CUSA-00343003

Memorandum

FILE COPY

San Francisco, California
October 14, 1985

MS. JUDY MacGREGOR: ✓

CHEVRON
ENVIRONMENTAL HEALTH CENTER

As discussed, after the ICI Meeting in early November, please prepare a response for me to Mr. Keller that would be a suitable transmittal to Mr. Follis regarding his concern.

OCT 21 '85

J. N. SULLIVAN
10-18-85

105.3 PARAQUAT
TECH-600
+ BHO

RTE	NAME	INIT	HDLE	CC
	J. N. OSPENSON			
	R. D. CAVALLI			
	J. T. KERNS			
	J. K. KODAMA			
	J. A. MacGREGOR			
	J. McKINDLEY			
	J. C. SINCLAIR			
RETURN	CIRC.		FILE	

cc: Mr. J. N. Ospenson

MR. G. M. KELLER:

Dear George:

I read the article in Chevron World that outlines our distribution of paraquat and the great care which is used to guard against poisoning by those handling this product. The attached outlines an aspect of the problem which differs from the normal dangers in dealing with poisons--namely, long-term effects that might not become apparent except after many years.

Since we don't want to take any chance of facing an asbestos situation down the road, I am sure your people are following this aspect of the matter most closely. However, I thought I would pass this on to you as I cannot think of anything more horrible for us to bequeath to our successors than an asbestos problem.

Regards,

R. Gwin Follis
R. GWIN FOLLIS

Attach.

Case ID: 220300429
Control No.: 26064959

CUSA-00343004

Memorandum

**Chevron's Paraquat and
Parkinson's Disease**

October 8, 1985

Attached are an article from the July 19 issue of Science, discussing recent evidence of environmental causes of Parkinson's and portions of an article from Chevron World's summer 1985 edition focused on the important economic and ecological advantages of conservation tillage using the herbicide, Paraquat. Chevron holds sole U.S. marketing rights for the product which is produced by ICI. Conservation tillage is expected to become the major tillage method in the country.

The article raises concern that Paraquat may be implicated because:

Paraquat is chemically very similar to the by-product of synthetic heroin manufacture, MTPT, which produces almost instant Parkinson's, by killing dopaminergic neurons in the brain, and

Paraquat is among the agricultural chemicals used in the area of Canada in which an extraordinarily high correlation of .967 was found between levels of pesticide use and Parkinson's cases. The incidence of the disease in the area was about 7 times the rate in areas where use was low.

The bankruptcy of the asbestos manufacturer, The Manville Corporation, has highlighted the especially severe financial risks involved in selling a product which contributes to a chronic disease. Parkinson's can go on for decades.

For the present, we can hope that another chemical or cause will explain the correlation found in the Barbeau study, but I trust that Chevron is watching this closely, and, perhaps, doing a little testing, for the sake of its customers and stockholders.

JGF

to improving the mathematical techniques for extracting measurements from the raw data to developing new models. A conceptually simple experiment has been to mount a radiometer called VAS on the geostationary GOES-5 satellite rather than a polar-orbiting satellite. That increased the horizontal resolution and provided nearly continuous rather than twice daily coverage.

John Le Marshall of the Cooperative Institute for Meteorological Satellite Studies in Madison, Wisconsin, and William Smith and Geary Callan of the National Oceanic and Atmospheric Administration in Madison found that the VAS data reduced the mean position error in

two 12-hour forecasts of Hurricane Debby from 139 to 50 kilometers (4). Analysis results from 12 storms are still encouraging, says Smith, but still do not represent strong statistical evidence of a consistent positive effect.

Such evidence of a positive effect in the Northern Hemisphere is something that all satellite observing system experiments still lack. Pre-FGGE computer experiments had indicated that the effect of satellites would be far more evident, probably because of a flaw in experiments designed to run on simulated data in the absence of real observations. In part to acquire such statistically valid evidence, an international field experiment will be conducted over the North

Atlantic beginning in 1986. The observing systems tested during FGGE—including satellites, aircraft, ships, and buoys—will operate for 1 year to produce a sufficient number of cases for observing system experiments.

—RICHARD A. KERR

References

1. R. A. Kerr, *Science* 228, 704 (1985).
2. *Data Assimilation Systems and Observing System Experiments with Particular Emphasis on FGGE, Vol. 1*, proceedings of a seminar and workshop held 3–11 September 1984 in Reading, United Kingdom (European Center for Medium Range Weather Forecasts, Reading, England, 1985).
3. *Proceedings of the First National Workshop on the Global Weather Experiment*, held July 1984 at Woods Hole, Massachusetts (National Academy of Science, Washington, D.C., in press).
4. J. F. Le Marshall, W. L. Smith, G. M. Callan, *Bull. Am. Meteorol. Soc.* 66, 258 (1985).

Parkinson's Disease: An Environmental Cause?

As the pace of research on drug-induced Parkinsonism increases, data are produced, and disputed, that identify pesticides as causative agents

It is little more than 2 years since researchers in several laboratories discovered that a relatively simple pyridine compound, 1-methyl-4-phenyl-1,2,3-tetrahydropyridine (MPTP), induces a parkinsonian-like state in humans and some animals (1). The discovery stemmed from an incident in which a "synthetic heroin" was contaminated with by-product MPTP that produced severe neuropathies in several dozen young people in southern California (2). As a consequence of this unfortunate episode, however, there has been a tremendous surge of research in the United States and throughout Europe on the mechanism of action of the chemical, which some consider might give insights into the etiology of the natural disease. Four separate conferences on the topic within the past several weeks attest to the intensity of research activity, especially as most of the data presented are yet to reach the printed page.

Parkinson's disease, which involves degeneration of certain dopaminergic neurons in the substantia nigra of the midbrain, typically develops late in life. Treatment with L-dopa usually relieves the neurological symptoms, which include tremor, partial or complete paralysis, and characteristic posture, but with time the condition usually deteriorates.

People who have been exposed to MPTP develop within a few days clinical symptoms that are virtually indistinguishable from those of the natural dis-

ease. On the neuropathological level, however, there are some differences. Although the neurons of the substantia nigra are affected in both conditions, some groups of cells that look pathological in the natural disease are apparently unaffected in the drug-induced state. Most significantly, characteristic inclusions derived from neurofilaments, known as Lewy bodies, are present in the natural disease but absent in the MPTP syndrome.

One argument offered to explain the differences at the cellular level is that the MPTP syndrome is an acute condition brought about by the acceleration of naturally occurring processes. The more diffuse pathology, including the Lewy bodies, of the natural disease might simply be the product of time, it is suggested.

The striking chemical similarity of MPTP and its metabolites with certain industrial chemicals, specifically the herbicide paraquat, is sufficient to persuade some observers that Parkinson's disease is environmentally caused and that the MPTP syndrome is therefore a very direct model of the natural disease. Opponents of this view say that the natural condition simply does not have the appearance of paraquat-like toxicity at the cellular level and that evidence on the involvement of environmental agents is negative. The balance of opinion at a recent meeting held at the National Institutes of Health (NIH)* supported the

position that Parkinson's is not an environmental disease.

Therefore, evidence presented a few days later by Andre Barbeau and his colleagues at a subsequent meeting in New York came as something of a surprise.† Barbeau, who is at the Clinical Research Institute of Montreal, collected data on the incidence of Parkinson's disease in the nine hydrographic regions of Quebec Province and matched it with pesticide use. The Montreal team used four independent methods to track the disease incidence and came up with more than 5000 cases in all. The correlation between disease incidence and level of pesticide use was very strong—0.967.

An area southwest of Montreal, Quebec's "breadbasket," recorded the highest rural incidence, 0.89 per thousand of population compared with 0.13 per thousand in regions where pesticide use was low. In Montreal itself there was a similar disparity between the industrialized east of the island and the residential west. All comparisons were adjusted for age structure of the populations.

Barbeau and his colleagues stress that pesticides are just one class of potential environmental neurotoxin that might be important in the etiology of Parkinson's

*Symposium on MPTP, Uniformed Services University of the Health Sciences, Bethesda, Maryland, 6–7 June.

†The Eighth International Symposium on Parkinson's Disease, New York, 9–12 June.

disease. There are in fact scores of pyridine compounds in the environment, most of them products of the chemical industry. Barbeau notes that Parkinson's disease was unknown before the industrial revolution, and its incidence rose rapidly through the 19th century, apparently to plateau in the early decades of this century. He does, however, expect a further upsurge in incidence as a consequence of the relatively recent introduction of the paraquat-like pesticides.

The Montreal researchers further support their case by demonstrating that 67 percent of Parkinson's disease patients have a genetic deficiency in certain detoxifying enzymes of the liver, the P450 monooxygenases, which render harmless foreign chemicals such as paraquat. This compares with 18 percent in the normal population.

All these data contrast with the arguments presented at the NIH meeting by Roswell Eldridge, of the neuroepidemiology branch of the National Institute of Neurological and Communicative Disorders and Stroke. He noted that comparisons between nations of different degrees of industrialization reveal no significant difference in disease incidence, an observation that Barbeau disputes. An analysis of British data by Roger Duvoisin and R.C. Schweitzer, of Rutgers Medical School, has shown no increase in incidence since the 1940's, he said, which was paralleled by a study in Rochester, Minnesota. Eldridge also cited studies on 43 identical twin pairs from the United States, which showed only two sets to be concordant for the disease. Again, studies in the United Kingdom and Finland are in line with this result.

None of this makes a strong case for the involvement of either a heritable genetic defect or an environmental agent. Instead, Eldridge proposes developmental differences that might endow some people with lower numbers of dopaminergic neurons than others. Those with low numbers would be more likely to be at risk from the effects of the steady loss of neurons throughout life. This suggestion accords with the inference from the U.S. twin study that the disease sufferers were united by a more subdued, inward-looking personality compared with their sibling, a difference that could usually be identified from childhood. There is some indication from observations on animals that subdued behavior correlates with low levels of dopamine neurons.

The direct conflict between the data offered by Barbeau and the position argued by Eldridge is unresolvable as yet,

it is clearly crucial to the development of further ideas.

One of the main messages of the NIH meeting was the consolidation of a finding reported earlier this year by Solomon Snyder and his group at the Johns Hopkins University School of Medicine (3), which relates to the site of metabolism of the MPTP once it has entered the brain.

One of the first major advances in the study of the mode of action of MPTP was the discovery that MPTP itself was not the toxin: toxicity depended on the metabolism by monoamine oxidase B to 1-methyl-4-phenyl-2,3-dihydropyridine (MPDP+), which is then transformed to the major stable metabolite 1-methyl-4-phenylpyridinium ion (MPP+). For instance, administration of monoamine oxidase B inhibitors protects an animal from subsequent treatment with MPTP. The question was, where was this taking place? And, more specifically, why were the dopaminergic cells of the substantia nigra so selectively destroyed?

In a Canadian study, the correlation between disease incidence and level of pesticide use was very strong—0.967.

The similarities between dopamine and MPTP were obvious and it was assumed that the substantia nigra neurons selectively took in MPTP, which was then converted to the toxic MPP+. Being a charged species, it was further assumed that MPP+ would then be unable to escape from the nigral neuron, whose fate would then be sealed.

As Snyder demonstrated, however, and as several groups have since confirmed, the conversion of MPTP to MPP+ appears to go on in the supporting glial cells, the astrocytes, not in the neurons. The MPP+ is then highly selectively taken up by the dopamine uptake system of the nigral cell neurons. Block the dopamine uptake system, using any one of several amphetamine-like compounds, and the toxic effect of MPTP administration is prevented, at least in mice.

If this description is indeed correct, it must probably spell the end of one of the popular explanations of the mode of toxicity, which was proposed principally by Neal Castagnoli and Anthony Trevor of the University of California, San Fran-

ce. They argued that the transformation of MPTP to MPP+ might generate many toxic species, perhaps involving interactions with dopamine, which would be lethal to the cell. If the astrocyte is the site of transformation, however, this argument in its basic form can no longer apply.

There was a lot of support at the NIH meeting for the notion that MPP+ is the toxic agent, which is perhaps not surprising in view of its similarity to paraquat. Several laboratories, including that of William Nicklas and Richard Heikkila at Rutgers Medical School, presented evidence that the species interferes with the first step of the electron transport chain of mitochondria, thus blocking respiration and killing the cell. The ion can also cause chemical havoc by generating oxidative stress, which results from the continuous formation of oxygen radicals, as Markey and co-workers have emphasized.

The general model as it stands at present, with MPTP conversion to MPP+ occurring in glia, followed by selective uptake by dopaminergic neurons, might explain the delayed onset of symptoms in patients exposed to MPTP, as noted by William Langston of Stanford University. MPP+ is known to remain in the brains of primates for some days after MPTP administration, so perhaps there is a slow shift from glia and subsequent concentration in striatal neurons. The scheme might also explain some of the species differences in susceptibility to MPTP. Rats and mice, for instance, are much less vulnerable than are primates, which might be related to a demonstrably much faster loss of MPP+ from the brain. And, in contrast with the results on mice, Langston and his colleagues have as yet been unable to demonstrate the protective effect of dopamine uptake blockers in monkeys dosed with MPTP. The meaning of this difference is as yet unclear.

For all its advances, the new scheme still does not explain adequately why the neurons of the substantia nigra suffer most while other dopaminergic neurons remain relatively unscathed. The answer may be simply quantitative: that the nigral cells accumulate much more MPP+ than do others. But that would seem be too simple a conclusion to what is turning out to be a very complex story.

—ROGER LEWIN

References

1. J. W. Langston, P. Ballard, J. W. Tetrud, I. Irwin, *Science* 219, 979 (1983); R. S. Burns *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* 80, 4546 (1983).
2. R. Lewin, *Science* 224, 1083 (1984).
3. J. A. Javitch *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* 82, 2173 (1985).

To a farmer, soil is money. Ray Tucker knew he had trouble the day he watched a spring downpour wash away tons of topsoil from one of his freshly plowed fields. "I was standing in my barn and I could see the water rushing down the slope as brown as mud," he recalls. "Everything settled beyond my fence line at the bottom of the hill." The rich soil that minutes earlier had stood ready to grow a new crop was now a useless ankle-deep quagmire in a ravine. Tucker was shaken. He'd farmed this rolling land in Knox County, Ohio, all his life. His father had farmed it before him. Plowing on hills inevitably leads to some erosion, he knew. But he also knew his fields couldn't take this kind of beating much longer. A few more ill-timed gully-washers and he would, quite literally, lose his land. On that soggy day 10 years ago, Tucker vowed to quit plowing. He didn't quit farming, however. Instead, he switched to no-till farming, a new way of planting and growing crops.

EROSION IS A DIRTY WORD

Ortho Paraquat is
helping farmers to keep soil
—and profits— from
washing away

Farmer Ray Tucker (left) and Chevron's Mike Dailey stroll through a recently planted no-till field. Hilly areas, particularly vulnerable to soil runoff, are ideal for no-till farming techniques.



Paraquat in perspective

The skull and crossbones so prominently displayed on containers of Ortho PARAQUAT PLUS® Herbicide are there for good reason. The chemical concentrate can be extremely toxic.

Paraquat also is one of the most widely used and versatile herbicides in the world, and since its introduction in the United States nearly 20 years ago, no poisonings or deaths have occurred from its proper use.

Many Americans are familiar with Paraquat because of the publicity it received several years ago when U.S. and foreign governments used it to destroy illegally grown marijuana plants. Erroneous reports stated that the treated marijuana could be lethal if smoked. In truth, no known illnesses were caused by smoking the treated marijuana.

Chevron, ICI Americas and various formulators carry on a product stewardship program aimed at protecting users and the public from possible health and safety risks. Among the steps taken:

- Paraquat has been registered domestically as a restricted-use product that must be applied by trained, licensed applicators or persons under their supervision.
- Paraquat is carefully packaged and clearly labeled to protect users by instructing them on safe handling procedures. Paraquat also is formulated with a stench agent, which creates a distinctive odor intended to discourage swallowing, and an emetic to induce vomiting in case the product is swallowed.
- Medical research has developed treatments for Paraquat poisoning, and Chevron has established an Emergency Information Center telephone-number (415-233-3737, collect) to answer questions about possible poisonings.

absorption and creates additional nutrients for the plants growing on the surface.

No-till farming, with its minimal disruption of the soil, helps maintain this natural balance, according to Chevron AgChem Sales Representative Bill Haddad. A wiry, energetic storehouse of no-till knowledge, Haddad (or "No-Till Bill," as customers and friends call him) carries a spade and penknife with him to demonstrate his point.

"Look at this," he says, pulling a softball-size chunk of earth from a Paraquat customer's no-till field. The soil is moist and a rich, dark brown color. In his hands, the earth crumbles easily to reveal four wriggling earthworms and their passageways.

"These are the veins of the soil. You won't find this many worms in conventionally tilled soil. Those that haven't been cut in half by plowing, have probably found the habitat too warm or too dry," says Haddad.

Another AgChem Sales Representative, Mike Dailey, agrees that no-till farming with Paraquat actually is a relatively natural system. "Sure, we use chemicals, but Paraquat doesn't disturb the structure of the soil or the micro-organisms in it," he says.

Dailey explains that any Paraquat that falls to the ground during spraying is biologically deactivated as soon as it bonds with clay particles found in nearly all soil. Paraquat in the soil remains inert and is gradually decomposed; it cannot be reactivated or accumulated in the food chain.

There's an added benefit to no-till farming with Paraquat, notes Dailey. It helps combat pollution. No-tilling so effectively prevents soil runoff that other herbicides and pesticides used on the crops, even though they may remain active in the soil (unlike Paraquat), stand a lesser chance of being swept into waterways through erosion.

"This isn't always an easy concept to explain," Dailey says. "However, when you realize that soil erosion is a serious environmental by-product of farming, you see that chemicals actually can help preserve the environment."

Paraquat is used in more than 100 countries around the world, and its applications extend beyond no-till farming. In orchards and vineyards, for example, it kills moisture-robbing weeds. In cotton fields, Paraquat becomes a harvest aid, defoliating the plants and opening the mature cotton bolls.

Since 1966, when Chevron gained U.S. marketing rights for Paraquat, the product has been used on more than 100 million acres of domestic cropland. (Paraquat is manufactured by a British firm, ICI Americas.) Ohio is the center of its strongest U.S. market, which extends from Virginia, north to Pennsylvania and west into Indiana.

About 14 million acres of U.S. cropland now are farmed the no-till way, compared with 1 million acres in the early 1970s.

Last year, Paraquat was used on 9 million acres and accounted for a sizable percentage of the Agricultural Chemical Division's domestic profits. The future looks even brighter. The U.S. Department of Agriculture predicts that within 25 years, 90 percent of all domestic cropland will use conservation tillage methods.

Nevertheless, selling Paraquat is a challenge, according to Haddad. "What we're really selling is the no-till concept," he says. "We're asking farmers to break tradition, and that's tough for them."

Haddad explains that no-till planting makes farmers rethink their farm management practices. "No-till is an entire program with many variables. You have to consider the types of crops you'll grow, the hybrids you'll plant, and the fertilizers and insecticides you'll use. You have to monitor your crops more closely.

"Most no-tillers," he concludes, "become better farmers."

Neal and Nelly Springer are good examples. When they bought their second farm 10 years ago, it was rumored to be one of the worst in Knox County. The soil was thin and severely depleted as a result of past erosion caused by conventional tillage. It also was full of rocks.

When the Springers declared that they wanted to convert the farm to no-till, even their pro-no-till friends balked because they knew how hard this farm would be to salvage. If it failed, neighboring farmers might blame no-till.

Alternatively, if it is later determined by a court of competent jurisdiction that the Copyright Office is an "agency" subject to the Regulatory Flexibility Act, the Register of Copyrights has determined that this proposed regulation will have no significant impact on small businesses because the proposed regulation does not change the application process by merely modifies the certificates issued by the Copyright Office.

List of Subjects in 37 CFR Part 201

Copyright, jukeboxes.

Proposed Regulations

PART 201—[AMENDED]

In consideration of the foregoing, the Copyright Office proposes to amend Part 201 of 37 CFR, Chapter II.

1. The authority citation for Part 201 is revised to read as follows:

Authority: Sec. 702, 90 Stat. 2541, 17 U.S.C. 702; § 201.16 is also issued under 17 U.S.C. 119.

2. Section 201.16(c)(1) and (c)(2) is revised to read as follows:

§ 201.16 Recordation and certification of coin-operated phonorecord players.

(c) *Certificate.* (1)(a) After receipt of the prescribed form and fee, the Copyright Office will issue a certificate containing the information set forth in paragraphs (b)(1)(i) and (ii) of this section, together with a unique licensing number, the date of issuance of the certificate and the date of expiration of the license. The date of expiration of the license will be December 31st of the year in which the certificate is issued. Certificates issued upon payment of a half-year fee will be valid only after July 1 of the year in which they are issued and will be so identified.

(2) The certificate may be affixed in the record selection (title strip) panel of a player or in another position on the player where it can be readily examined by the public, but in any case it must be clearly visible.

Dated: October 16, 1985.

Ralph Oman,
Register of Copyrights.

Daniel J. Boerstin,
The Librarian of Congress.
[FR Doc. 85-25273 Filed 10-22-85; 8:45 am]
BILLING CODE 1410-07-M

POSTAL SERVICE

39 CFR Part 10

International Mail Manual; Proposed Express Mail International Service to Cyprus

AGENCY: Postal Service.

ACTION: Proposed rule.

SUMMARY: Pursuant to an agreement with the postal administration of Cyprus, the Postal Service intends to begin Express Mail International Service with Cyprus at postal rates indicated in the tables below. The proposed service is scheduled to begin on December 27, 1985.

DATE: Comments must be received on or before November 27, 1985.

ADDRESS: Written comments should be directed to the General Manager, Rate Development Division, Office of Rates, Rates and Classification Department, U.S. Postal Service, Washington, DC 20260-5350. Copies of all written comments will be available for public inspection and photocopying between 9 a.m. and 4 p.m., Monday through Friday, in room 8620, 475 E. Enfant Plaza West, SW., Washington, DC 20260-5350.

FOR FURTHER INFORMATION CONTACT: Leon W. Perlinn, [202] 268-2673.

SUPPLEMENTARY INFORMATION: The International Mail Manual is incorporated by reference in the Code of Federal Regulations, 39 CFR 10.1. Additions to the manual concerning the proposed new services, including the rate tables reproduced below, will be made in due course. Accordingly, although 39 U.S.C. 407 does not require advance notice and the opportunity for submission of comments on international service, and the provisions of the Administrative Procedure Act regarding proposed rulemaking [5 U.S.C. 553] do not apply [39 U.S.C. 410 (a)], the Postal Service invites interested persons to submit written data, views or arguments concerning the proposed Express Mail International Service to Cyprus at the rates indicated in the table below.

Lists of Subjects in 39 CFR Part 10—

Postal Service, Foreign relations.

PART 10—[Amended]

The authority citation for Part 10 continues to read as follows:

Authority: 5 U.S.C. 552(a), 39 U.S.C. 401, 404, 407, 408.

CYPRUS—EXPRESS MAIL INTERNATIONAL SERVICE

Custom designed service ¹		On demand service ²	
Up to and including		Up to and including	
Pounds	Rate	Pounds	Rate
1	\$31.00	1	\$23.00
2	35.90	2	27.90
3	40.80	3	32.80
4	45.70	4	37.70
5	50.60	5	42.60
6	55.50	6	47.50
7	60.40	7	52.40
8	65.30	8	57.30
9	70.20	9	62.20
10	75.10	10	67.10
11	80.00	11	72.00
12	84.90	12	76.90
13	89.80	13	81.80
14	94.70	14	86.70
15	99.60	15	91.60
16	104.50	16	96.50
17	109.40	17	101.40
18	114.30	18	106.30
19	119.20	19	111.20
20	124.10	20	116.10
21	129.00	21	121.00
22	133.90	22	125.90
23	138.80	23	130.80
24	143.70	24	135.70
25	148.60	25	140.60
26	153.50	26	145.50
27	158.40	27	150.40
28	163.30	28	155.30
29	168.20	29	160.20
30	173.10	30	165.10
31	178.00	31	170.00
32	182.90	32	174.90
33	187.80	33	179.80
34	192.70	34	184.70
35	197.60	35	189.60
36	202.50	36	194.50
37	207.40	37	199.40
38	212.30	38	204.30
39	217.20	39	209.20
40	222.10	40	214.10
41	227.00	41	219.00
42	231.90	42	223.90
43	236.80	43	228.80
44	241.70	44	233.70

¹ Rates in this table are applicable to each piece of International Custom Designed Express Mail shipped under a Service Agreement providing for tender by the customer at a designated Post Office.

² Pickup is available under a Service Agreement at an added charge of \$5.60 for each pickup stop, regardless of the number of pieces picked up. Domestic and International Express Mail picked up together under the same Service Agreement incurs only one pickup charge.

An appropriate amendment to 39 CFR 10.3 to reflect these changes will be published when the final rule is adopted.

W. Allen Sanders,

Associate General Counsel, Office of General Law and Administration.

[FR Doc. 85-25264 Filed 10-22-85; 8:45 am]

BILLING CODE 7710-12-M

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 716

[OPTS-84014; FRL-2905-2]

Health and Safety Data Reporting; Submission of Lists and Copies

Correction

In FR Doc. 85-23259 beginning on page 39715 in the issue of Monday, September 30, 1985 make the following corrections: