The Common Swift: A Synanthropic Bird Species for Monitoring Airborne Microcontaminants?

Fabrizio Rodriguez,[a] Claudio Carere,[b] Giacomo Dell'Omo,[b] Nicola Iacovella,[a] Luigi Turrio Baldassarri,[a] Fabrizio Volpi,[a] Alessandro di Domenico[a,*]

(a) Laboratory of Comparative Toxicology and Ecotoxicology and *(b)* Laboratory of Organ and System Pathophysiology, Istituto Superiore di Sanità, 00161 Rome, Italy

INTRODUCTION

The ubiquitous polychlorobiphenyls (PCBs), polychlorodibenzo-*p*-dioxins (PCDDs), polychlorodibenzofurans (PCDFs), and chlorinated pesticides such as DDE, DDT, and HCB are lipophilic organic microcontaminants of the environment characterized by remarkable environmental persistence, potential for bioaccumulation, and possible adverse effects on wildlife populations. Furthermore, in humans several of these compounds exhibit a strong toxic action — including carcinogenity — even at very low exposure levels.[1-4] The common swift (*Apus apus, Apodiformes*) is a specialized long-lived bird that is known to spend all of its active time flying, feeding exclusively on aerial arthropods within ≈ 0.1 km from ground level;[5] it is also a long-distance migratory bird, whose western Palearctic populations move regularly from sub-Saharan Africa over to Europe where they breed in colonies, preferentially in urban environments.[6] At our latitudes, they arrive in early spring to spend approximately five months; normally, some 70 days will elapse from the establishment of colonies — shortly after arrival — until offspring fledging, during which period birds experience limited mobility as they are motivated to stay by their nests.[7,8] The present investigation is part of a study, currently in progress, aimed at assessing *(a)* whether the cited species may be used as a bioindicator of the microcontaminants of interest and *(b)* how exposure relates to body burden and distribution in different organs. Preliminary findings are here briefly reported upon and discussed.

EXPERIMENTAL

Forty dead or fatally injured grounded adult swifts were collected in the City of Rome (Italy), during the breeding period, between April 29 and July 14, 1993; live birds were sacrificed with ethyl ether once it was ascertained that they had no chance of survival. The plumage and skin of each body were carefully and immediately removed upon delivery to the laboratory, and so were the brain, breast muscle, heart, liver, and lungs. Excision of tissues was performed rapidly, using scalpels; individual specimens were wrapped in aluminum foil, properly labelled, and stored at -80 °C awaiting further treatments. When analytical activities were resumed several months later, 14 randomly-selected specimens of each tissue type were allowed to thaw out in the laboratory; then, five 14-specimen pools (samples) were obtained by combining specimens of the same type. Samples were spiked with fully C^{13}-labelled standards — along with laboratory policies[9,10] — and homogenized and extracted by means of an Omni-Mixer homogenizer (OCI Instruments, Waterbury, CT) after addition of a mixture of *n*-hexane-acetone (1:1, v/v); details of lipid extraction from biotic matrices have been reported elsewhere.[11,12] The crude organic extract was subjected to a number of cleanup steps — including liquid-liquid partition, elution on a multilayer column (primarily, a treatment with concentrated sulfuric acid), and chromatographic filtration on an activated alumina column — also previously described.[9,10,13,14] The two main eluates obtained from alumina cleanup were used for determination of PCBs, DDE, DDT, and HCB (Fraction 1), and PCDDs and PCDFs (Fraction 2) by HRGC-HRMS(SIM).[9,10] GLP and QA/QC protocols were applied throughout;[9-12] blanks were run at the beginning and at the end of a sample sequence, and between samples.

RESULTS AND DISCUSSION

The fresh tissue pools utilized for analytical determination weighed between 4.12 and 10.4 g. The lipid amounts extracted ranged from 67.2 to 487 mg — 1.6 to 4.7 % of the original matrix, in agreement with the literature:[15] since chemical levels are reported below on a lipid base exclusively, the relative quantity of lipids may be used to convert analytical figures to the pertinent fresh tissue-based concentrations. For the PCB C^{13}-tracers selected — T_3CB[28], T_4CB[52], P_5CB[101], H_6CB[138], H_7CB[178], and O_8CB[202] — recovery rates were seen to fall between 51 and 107 %, with the sole exceptions of T_3CB[28] and T_4CB[52] in the breast muscle tissue matrix (respectively, 12 and 23 %) and T_3CB[28] in the heart tissue matrix (40 %); recovery rates of C^{13}-2,3,7,8-PCDDs and -PCDFs — one tracer for each homologous group — covered the range 44—87 %; C^{13}-DDE, -DDT, and -HCB recovery rates ranged from 67 to 95 %. A synopsis of the relevant findings available, which includes some of the above, follows hereafter.

ANALYTE LEVELS PER TISSUE TYPE
(ng/g, lipid base)

ANALYTES	*BRAIN*	*BREAST MUSCLE*	*HEART*	*LIVER*	*LUNGS*
PCBs[a]	1,100[b]	4,100	3,000	9,700	7,500
PCDDs+PCDFs[c]	0.35[d]	1.4	3.5	37	11
PCDDs+PCDFs *(I-TE)*[e]	0.038[d]	0.16	0.20	0.98	0.25
DDE	2,300	5,100	7,300	6,500	6,400
DDT	8.1	22	30	<1[f]	24
HCB	110	170	180	230	250
POOL SIZE[g] *(g)*	6.65	10.4	5.83	9.80	4.12
EXTRACTED LIPIDS *(mg, %)*	288, 4.3	487, 4.7	224, 3.8	304, 3.1	67.2, 1.6

(a) Cumulative results of analytically relevant congeners of tri- to octachlorosubstituted homologous groups.

(b) All values rounded off to two or three figures.

(c) Cumulative results of 2,3,7,8-chlorosubstituted congeners.

(d) Lacking representativeness.[16,17]

(e) Cumulative results of 2,3,7,8-chlorosubstituted congeners expressed in 2,3,7,8-T_4CDD toxicity equivalency units.[18]

(f) The sign < indicates below limit of quantification.

(g) Fresh tissue weights. Pools were used completely, each providing a single sample for analysis.

From the table, it may be observed that all the analytes reach a distinct concentration minimum in the brain — in particular, PCDD and PCDF cumulative levels are not representative.[16,17] Not considering the uncertainty affecting analytical measurements,[9,16,19] maximum levels seem to be present in the liver for PCBs and PCDDs and PCDFs, in the heart for DDE and DDT, and in the lungs for HCB: however, for pesticides maxima cannot be as convincingly singled out as minima (see below). Furthermore, it should be pointed out that PCBs and PCDDs and PCDFs cover concentration ranges of approximately one to two orders of magnitude, contrary to the pesticide patterns which — with the exclusion of DDT disappearance in the liver — exhibit minimum-to-maximum variations not greater than a factor of ≈3. In other words, the distribution of pesticides appears to be much less

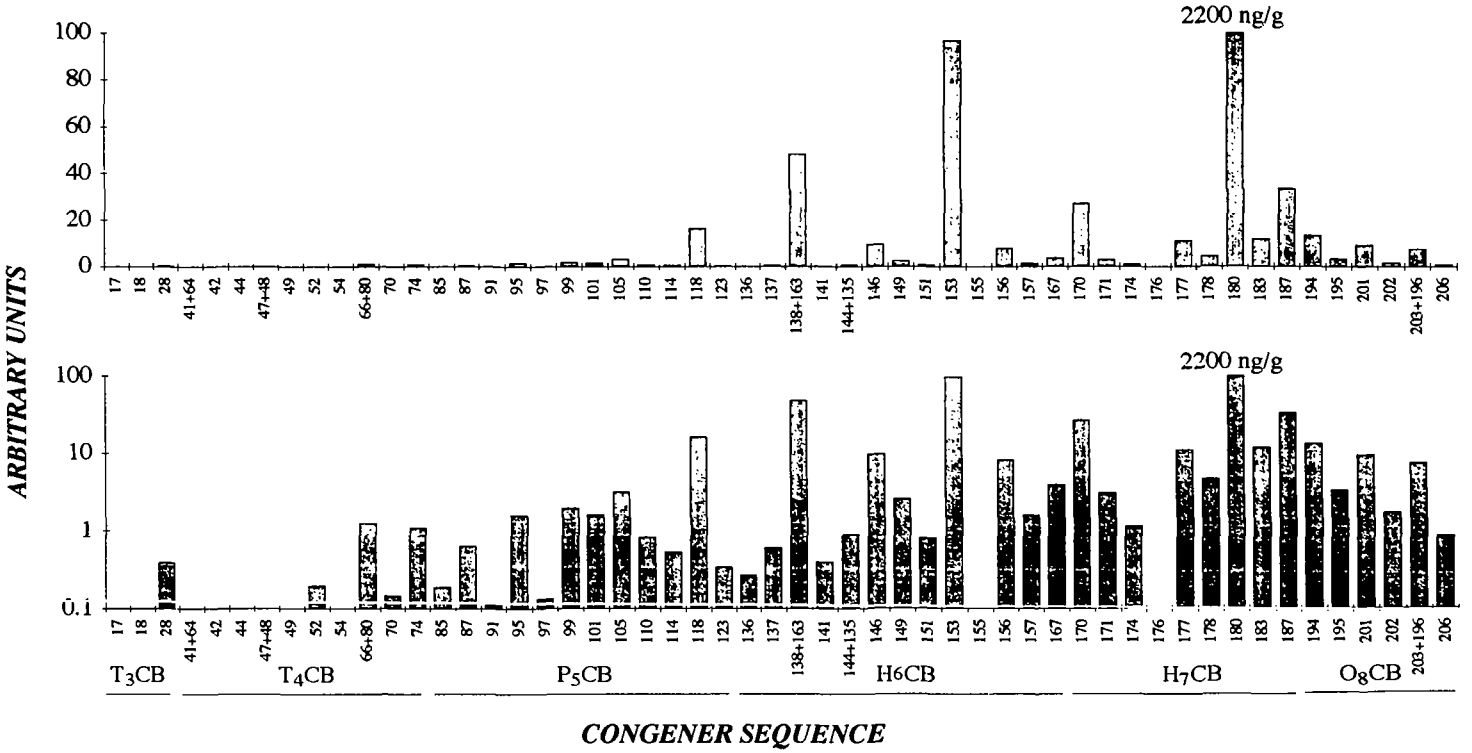

Figure 1. PCB congener analytical profile as detected in swift hepatic tissue, normalized on the base congener H_7CB[180] (chlorosubstitution pattern: 2, 2', 3, 4, 4', 5, 5'). The upper box exhibits the distribution referred to on a 0—100% linear scale; in the lower box, the Y-axis is a three-decade logarithmic reference to allow for more graphic sensitivity and expand the representation of congeners providing a lesser contribution. When compared with literature data, PCB cumulative levels detected in the swift tissues — and specifically in the liver (total PCBs, 9700 ng/g, lipid base) — appear to be in agreement with similar data obtained from areas under general anthropic impact,[20] an observation which also applies to the chlorinated pesticides quantified. However, the PCB levels reported here are much lower than those found, for instance, in a fish-eating bird species.[21,22] The congener sequence on the X-axis follows the IUPAC ranking.

uneven than that of the other chemicals dealt with — a feature which remains, even disregarding the findings reported for brain. In fact, DDE, DDT, and HCB show concentration ranges respectively of 5100—7300, 22—30 (level in the liver omitted), and 170—250 ng/g — variations which are comparable with analytical measurement uncertainty[9,16,19] — where PCB and PCDD and PCDF levels are comprised between 3000 and 9700 and 1.4 and 37 (0.16—0.98, TE units) ng/g, respectively.

Figures 1 and 2 contain examples of PCB, PCDD, and PCDF congener profiles (specifically, in the hepatic tissue); for the latter two families, the distribution pattern of TE[18] contributions is also exhibited.

If a 20% reference line is set on the Y-axis of Figure 1, only the following five PCB congeners appear to provide significant individual contributions: H_6CB[138+163], H_6CB[153], H_7CB[170], H_7CB[180], and H_7CB[187]; taking into account the profiles from remaining pools (unshown), P_5CB[118] could also be included in the group. This five- or six-congener cluster — where H_6CB[153] and H_7CB[180] may be seen to compete for being base congener (100 %) — is a recurrent and remarkably stable primary feature typical of all PCB distributions assessed in the available pools and, more in general, typical of animals sharing high trophic levels.[21-23] However, perceivable differences characterize the occurrence in the different tissues of several T_3CB, T_4CB, and P_5CB congeners of minor importance: whether this is to be ascribed to exposure or metabolic factors, or both, cannot be said as yet.

From Figure 2 and the other available profiles (unshown), it can be readily observed that PCDFs consistently provide only a minor contribution — <30 %, mostly accounted for by 2,3,4,7,8-P_5CDF — to the total TE level. This is primarily determined by the 2,3,7,8-PCDD component, in particular that penta- and hexachlorosubstituted (1,2,3,7,8-P_5CDD is the base congener, followed by 1,2,3,6,7,8-H_6CDD); however, 2,3,7,8-T_4CDD may also occasionally become significant. Therefore, although perceptible deviations from the above pattern can be observed in the unshown profiles, again a five- or six-congener cluster may be taken as a primary feature capable of a reasonably reliable fingerprinting. In Figure 2 analytical profile, the presence of 1,2,3,4,6,7,8-H_7CDD and O_8CDD stands out, the latter being unequivocally the base congener; however, as already pointed out for the TE figures, the analytical profiles associated with the remaining pools also exhibit sensible variations, to the extent that in one case (breast muscle) the aforesaid primary feature appears to be significantly altered: again, whether this is to be ascribed to exposure or metabolic factors, or both, cannot be said as yet.

Considering that the age of the birds collected could not be ascertained — as estimated, from one to several years — the experimental evidence may suggest that pesticide contamination levels and distribution patterns in the pools examined reflect (relative) periodical long-term exposures, primarily in Africa,[24] and the reaching or approaching of a steady-state condition in the organisms exposed. On the contrary, exposure to PCDDs and PCDFs could be mostly limited to the relatively short Italian period(s),[25,26] this determining a chemical distribution in the swifts collected that did not have a time long enough to develop into the features proper of a steady state. PCB situation seems to follow this latter trend, or else could fall between the two extreme cases.

ACKNOWLEDGEMENTS

This work has been supported by funds from Tuscany Region, Contract No. 4029/3A of June 6, 1991. We are grateful to F. Ferri for her editorial help and preparing the drawings, and to D. Henegan for language assistance in writing the manuscript.

REFERENCES

1) WHO (1989). *DDT and its Derivates - Environmental Aspects.* Environmental Health Criteria No. 83, IPCS, World Health Organization (Geneva).
2) WHO (1989). *Polychlorinated Dibenzo*-para-*dioxins and Dibenzofurans.* Environmental Health Criteria No. 88, IPCS, World Health Organization (Geneva).
3) WHO (1993). *Polychlorinated Biphenyls and Terphenyls (Second Edition).* Environmental Health Criteria No. 140, IPCS, World Health Organization (Geneva).
4) Kutz F.W., S.C. Strassman, and J.F. Sperling (1979). Survey of selected organochlorine pesticides in the general population of the United States: Fiscal years 1970-1975. In: *Health Effects of Halogenated Aromatic Hydrocarbons*, pp. 60-68. Annals of the New York Academy of Sciences No. 320, W.J. Nicholson and J.A. Moore, Eds., The New York Academy of Sciences (New York).
5) Lack D. and D.F. Owen (1955). J. Animal Ecol. *24*, 120-136.

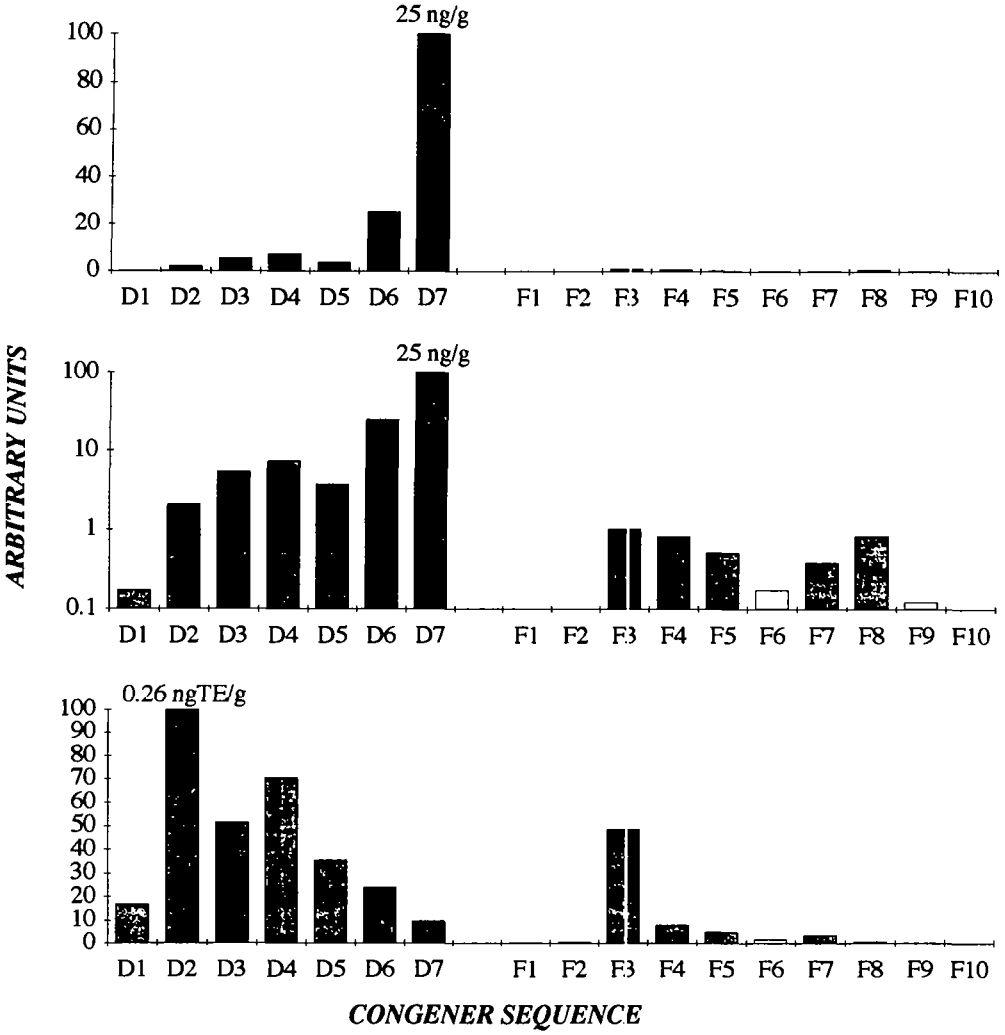

Figure 2. PCDD and PCDF congener analytical and I-TE profiles as detected in swift hepatic tissue, normalized on base congeners. The upper box exhibits the distribution of the analytical findings on a 0—100% linear scale; in the middle box, the Y-axis is a three-decade logarithmic reference to allow for more graphic sensitivity and expand the representation of congeners providing a lesser contribution: in both cases, the base congener is O_8CDD. In the lower box, the congener profile is shown on a 0—100% linear scale after conversion to 2,3,7,8-T_4CDD toxicity equivalency (I-TE) units:[18] here, the base congener turns out to be 1,2,3,7,8-P_5CDD. When compared with literature data, PCDD and PCDF cumulative levels detected in the swift tissues — and specifically in the liver (total PCDDs and PCDFs, 0.98 ngI-TE/g, lipid base) — appear to be higher than those reported, for instance, for a wildlife fish-eating species such as the common tern (*Sterna hirundo*).[21,22] The congener sequence on the X-axis follows the IUPAC ranking, in particular labels are paired as follows: D3, D4, and D5 with 1,2,3,4,7,8-, 1,2,3,6,7,8-, and 1,2,3,7,8,9-H_6CDD; F2 and F3 with 1,2,3,7,8- and 2,3,4,7,8-P_5CDF; F4, F5, F6, and F7 with 1,2,3,4,7,8-, 1,2,3,6,7,8-, 1,2,3,7,8,9-, and 2,3,4,6,7,8-H_6CDF; F8 and F9 with 1,2,3,4,6,7,8-, and 1,2,3,4,7,8,9-H_7CDF. White bars indicate limit of quantification.

6) Cramp S., Ed. (1985). *Handbook of the Birds of Europe, the Middle East, and North Africa - The Birds of the Western Palearctic* (Volume IV - Terns to Woodpeckers), pp. 657-669. Oxford University Press (Oxford).
7) Lack D. and E. Lack (1952). Brit. Birds *45*, 186-215.
8) Carere C., G. Dell'Omo, and E. Alleva (1995). Ruoli parentali e presenza al nido durante il periodo d'incubazione nel rondone comune (*Apus apus*): Dati preliminari. In: *Atti del VII Convegno Nazionale di Ornitologia*, pp. 125-129. Supplemento alle Ricerche di Biologia della Selvaggina No. 22, M. Pandolfi and U.F. Foschi, Eds., Istituto Nazionale per la Fauna Selvatica (Bologna).
9) di Domenico A., E. De Felip, F. Ferri, N. Iacovella, R. Miniero, E. Scotto di Tella, P. Tafani, and L. Turrio Baldassarri (1992). Microchem. J. *46*, 48-81.
10) Turrio Baldassarri L., A. di Domenico, A. Fulgenzi, N. Iacovella, A. Bocca, and B.R. Larsen (1993). Fresenius Environ. Bull. *2*, 370-374.
11) di Domenico A., C. La Rocca, C. Lintas, and L. Turrio Baldassarri. (1989). Bull. Environ. Contam. Toxicol. *43*, 556-563.
12) La Rocca C., A. di Domenico, N. Iacovella, R. Miniero, and L. Turrio Baldassarri (1991). Presence of microcontaminants in molluscs and sediments of the north Adriatic sea. In: *Proceedings of the 3rd International Workshop on Study and Prediction of Pesticide Behaviour in Soils, Plants, and Aquatic Systems*, pp. 403-410. M. Mansour, Ed., GSF - Forschungszentrum für Umwelt und Gesundheit (Neuherberg).
13) De Felip E., A. di Domenico, M. Grande, R. Pazzaglia, and M. Falleni (1990). Intern. J. Environ. Anal. Chem. *38*, 607-616.
14) De Felip E., A. di Domenico, L. Turrio, F. Volpi, and F. Merli (1990). Toxicol. Environ. Chem. *29*, 37-46.
15) Södergren A., and S. Ulfstrand (1972). Ambio *1*, 36-40.
16) De Felip E., A. di Domenico, M. Falleni, F. Ferri, N. Iacovella, G. Menale, P. Tafani, G. Tommasino, and L. Turrio Baldassarri (1994). Toxicol. Environ. Chem. *46*, 239-260.
17) di Domenico A., C. Lupi, E. De Felip, F. Ferri, N. Iacovella, R. Miniero, E. Scotto di Tella, P. Tafani, L. Turrio Baldassarri, and F. Volpi (1995). Clusters of kin analytes: Detection thresholds of individual components and representativeness of cumulative results. In: *Organohalogen Compounds 23*, pp. 165-170. D. Bolt, R. Clement, H. Fiedler, B. Harrison, S. Ramamoorthy, and E. Reiner, Eds. (Edmonton).
18) US EPA (1989). *Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Polychlorinated Dibenzo*-p-*dioxins and Dibenzofurans (CDDs and CDFs) and 1989 Update*. EPA/625/3-89/016. Risk Assessment Forum, US Environmental Protection Agency (Washington).
19) Turrio Baldassarri L., A. di Domenico, A.R. Fulgenzi, C. La Rocca, N. Iacovella, F. Rodriguez, and F. Volpi (1996). Influence of relative response factor in the determination of organic microcontaminants with isotopically labelled standards. Mikrochim. Acta, April issue.
20) Bishop C.A., M.D. Koster, A.A. Chek, D.J.T. Hussell, and K. Jock (1995). Environ. Toxicol. Chem. *14*, 491-501.
21) Bosveld A.T.C., J. Gradener, M. van Kampen, A.J. Murk, E.H.G. Evers, and M. Van den Berg (1993). Chemosphere *27*, 419-427.
22) Bosveld A.T.C., J. Gradener, A.J. Murk, A. Brouwer, M. van Kampen, E.H.G. Evers, and M. Van den Berg (1995). Environ. Toxicol. Chem. *14*, 99-115.
23) Boumphrey R.S., S.J. Harrad, K.C. Jones, and D. Osborn (1993). Arch. Environ. Contam. Toxicol. *25*, 346-352.
24) Zaranyika M.F., E. Mambo, and J.M. Makhubalo (1994). Sci. Tot. Environ. *142*, 221-226.
25) Turrio Baldassarri L., A. Carere, A. di Domenico, S. Fuselli, N. Iacovella, and F. Rodriguez (1994). Fresenius' J. Anal. Chem. *348*, 144-147.
26) Berlincioni M., G. Croce, F. Ferri, N. Iacovella, C. La Rocca, M. Lolini, A. Megli, M. Pupp, L. Rizzi, L. Turrio Baldassarri, and A. di Domenico (1995). Fresenius Environ. Bull. *4*, 169-174.