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EXECUTIVE SUMMARY

Throughout the Caribbean, concerns exist about the levels and effects of contaminants, including but not limited to organochlorine pesticides used in agriculture. Despite such concerns, there are few data to substantiate the threat of environmental contamination for people or wildlife. Such is the case for the highly toxic pesticide, chlordecone, which has been used historically (but not in recent years or at present) in the French Antilles for the control of banana weevils. Although there are multiple reasons for concern with regard to possible health effects of chlordecone use, this study was stimulated by the possibility that manatees (*Trichechus manatus*) may be reintroduced to the waters of Guadeloupe and concerns that residual chlordecone levels may represent a threat to the introduced animals.

In fact, there are no data to available to link levels of environmental contaminants to lethal or sublethal effects on manatees or most other marine mammals. It is possible, but unwise to make unsupported assumptions that measured effects in laboratory animals would be similar to those experienced by free-ranging animals of different species. Thus, the current study would, at best, be able to identify contaminants levels of concern for manatee health. To understand and document effects of stressors such as contaminants on significant biological parameters such as immune system function or reproductive potential, there is a need to start: (a) using available technology (e.g., carefully validated biomarkers of effects of exposure), and (b) conducting formal health risk assessments

using the best available data from future studies. These parameters are important components of comprehensive conservation status assessments.

In our study we found that chlordecone levels in the Grand Cul-de-Sac Marin are either undetectable or minimal in sediment and seagrass samples. These findings suggest that chlordecone does not constitute a threat to manatees. Dithiocarbamate fungicides, on the other hand, were present ubiquitously, albeit at levels that may not create a threat for manatee or human health. We recommend monitoring of dithiocarbamate levels in the waters, sediments, and organisms of the Grand Cul-de-Sac Marin. Finally, we found low levels of certain polycyclic aromatic hydrocarbons in sediments and seagrasses; we believe that the current levels do not represent a threat to manatees, if the reintroduction takes place.

It should be noted that our study did not do a complete assessment of contaminants that could be present in the Grand Cul-de-Sac Marin. C. Bouchon and colleagues have assessed some other inorganic and organic chemicals of concern (and also have found that threats to manatees would be minimal) but certain classes of compounds have not been assessed at all (e.g., flame retardants).

In summary, our study detected a number of organic contaminants in the sediments and seagrasses of the Grand Cul-de-Sac Marin, Guadeloupe. Current contaminant levels suggest that the classes and specific types of organic compounds we

assessed do not presently constitute a sufficient environmental threat to prevent a possible introduction of manatees to the waters of this area.

INTRODUCTION:

In May, 2008, Reynolds and Wetzel submitted a report to the Parc National de la Guadeloupe. The topic of the report focused on the feasibility of reintroducing West Indian manatees (*Trichechus manatus*) into the waters of Guadeloupe, specifically into the waters of the Grand Cul-de-Sac Marin where they were once relatively common. Reynolds and Wetzel (2008) concluded that the project was, indeed, feasible and could be of great conservation value to the species in the wider Caribbean. However, the authors expressed concern that certain potential risk factors, including contaminants, needed study and, possibly, mitigative actions.

One of the first steps that was taken was to develop a study of persistent organic contaminants in seagrasses and sediments of the Grand Cul-de-Sac Marin. The lead scientist in Guadeloupe was Dr. Claude Bouchon; the lead at Mote Marine Laboratory was Dr. Dana Wetzel. The study was motivated generally by the need to understand background levels of contaminants in the area, prior to moving forward with the manatee project, but there was also specific interest in levels of the insecticide chlordecone, since persistent residues of this toxic chemical had been found in terrestrial and marine environments of the French West Indies.

Thus, this study set out specifically to assess levels of chlordecone, but also included assessments of other organic molecules of concern. The report that follows focuses primarily on chlordecone, but also discusses the possibility of health risk to wildlife and people associated with the other contaminants found.

THE POSSIBLE REINTRODUCTION OF MANATEES IN GUADELOUPE

The Government of France and the Parc Nacional de la Guadeloupe have proposed an interesting experiment, namely the possible reintroduction of West Indian manatees (*Trichechus manatus*) into the waters of the Grand Cul-de-Sac Marin, where the species was extirpated by hunting many decades ago (Reynolds and Wetzel, 2008; UNEP 2010; Marsh et al. in review). Assuming that potential threats to manatees in Guadeloupe waters are identified and under control, this process could have important, and positive, consequences for the status of manatees (specifically, the Antillean subspecies of the West Indian manatee: *T.m. manatus*) throughout the Caribbean. The potential threats are outlined by Reynolds and Wetzel (2008); one is the topic of this study, namely the potential threat posed by levels of organic contaminants in the environment of the Grand Cul-de-Sac Marin.

CONTAMINANT RISKS

The Caribbean Marine Mammal Action Plan (UNEP 2008) describes a number of putative threats to marine mammals in the wider Caribbean. On a more specific level

(i.e., for Antillean manatees) the Regional Management Plan for the West Indian Manatee (UNEP 2010) raises the same general types of concerns. In both documents, contaminants of a variety of types and sources are included as threats of great concern, albeit without sufficient empirical data on either the levels or effects of those chemicals with which to assess exactly how serious the true threat is.

In fact, for manatees there are no data with which to judge the effects of specific contaminants on either manatee mortality or sublethal consequences for important biological functions such as reproductive potential, immune system function, or energetic fitness (Wetzel et al, in press). Although there are experimental data on effects of contaminants on certain laboratory animals and in some cases, humans, the fact that different species may respond very differently to particular doses of a specific contaminant means that it is risky to assume similar responses to similar exposures or body burdens. Since it is impossible (for legal and ethical reasons) to conduct dose response experiments on manatees, it is important to use other means (e.g., well validated biomarkers) to quantitatively measure effects of contaminants on species of concern such as West Indian manatees (Wetzel et al. in press). Until that is done, it would be premature to assume that particular body burdens of contaminants, or particular environmental levels, are or are not having certain consequences for manatee health or survival. Nonetheless, in locations where environmental levels of contaminants are extremely high and are known to pose a threat to human health, it would be prudent to monitor and assess possible effects on other species of concern and as a precautionary measure to mitigate those worrisome levels prior to a crisis.

CHLORDECONE

History of Chlordecone Development and Use in the United States

Chlordecone, also commonly known as kepone or curlone, has been used for a relatively short period of time, but its environmental persistence and effects make it a cause for concern in some locations both now and in the future. Chlordecone was first synthesized in 1951, patented in 1952, and first marketed in 1958 by Allied Chemical. However, its production and use in the United States were terminated less than 20 years later, in 1977 (Epstein 1978; Huff and Gerstner 1978). In contrast, in other parts of the world the use of chlordecone persisted for some time, and in the French West Indies, the use of this chemical to control banana weevils (*Cosmopolites sordidus*), began in 1972 and continued until September, 1993 (e.g., Cabidoche et al. 2009; Coat et al. 2006).

This truncated use pattern occurred due to attributes of the chlordecone molecule and its effects on both humans and wildlife (Epstein 1978). Chlordecone is an extremely toxic chlorinated hydrocarbon pesticide, similar in its structure to mirex. Chlordecone is very persistent in the environment (with an estimated half life of approximately 10 years; Dubuisson et al. 2007), where it can induce a wide range of pathological effects on birds, non-human mammals and humans (see section below on Documented Levels and Effects). In 2009, the production and use of chlordecone were banned at the global scale, when the chemical was included on the list of priority pollutants by the Stockholm Convention.

Although chlordecone production and use did not elicit great concern through the 1960s and early 1970s (Huff and Gerstner 1978), by the middle of 1975 that situation changed when workers at the Life Science Products Company (LSPC) plant (Hopewell, Virginia, USA), the only site where the chemical was produced, began to show severe and diverse pathology associated with their exposure. That plant produced approximately 3,000 pounds (~1,365 kilograms) of chlordecone powder per day; it was closed in 1975.

However, it was not only the human workers in the plant (and their families) that whose health was adversely affected by exposure to chlordecone. The local environment and wildlife were also impacted because the plant released chlordecone into the sewage system of Hopewell. Around that time, atmospheric concentrations of chlordecone were as high as 20.7 ng/m^3 nearly 10 miles from the LSPC, and concentrations in nearby waters associated with sewage treatment exceeded 3 ppb (Epstein 1978). Perhaps most worrisome were levels as high as 4 ppm in river bottom sediments of nearby Bailey's Creek, and as high as 20,000 ppm in soils outside LSPC (Epstein 1978).

Local fish and shellfish were shown to have accumulated high levels of chlordecone. Bivalves several miles from Hopewell had concentrations as high as 0.8 ppm. Levels in fish depended on species and body tissue sampled, with levels as high as 14 ppm reported in entrails of bass and bream (Epstein 1978).

Concerns about effects of fish and shellfish consumption on human health led to a ban on all fishing in the James River in December, 1975. The following month, the U.S. Environmental Protection Agency recommended to the Food and Drug Administration a chlordecone shellfish action level of 0.3 ppm.

In a more recent publication, Nichols (1990; cited by Coats et al. 2006) found astonishing levels of chlordecone in organisms sampled in the James River, near the Hopewell LSPC plant, in 1977. In zooplankton, the mean concentration Nichols found was 4,800 µg/kg wet weight; in freshwater fishes, the mean value was 2,500 µg/kg wet weight; and in benthic mollusks and phytoplankton, the means were 1,500 µg/kg wet weight and 1,300 µg/kg wet weight, respectively. Even migratory fish species had, on average 400 µg/kg wet weight (Nichols 1990).

Some Documented Levels and Effects of Chlordecone

As noted above, workers at the Hopewell, Virginia production plant experienced a variety of pathological conditions including brain and liver damage and sterility (Epstein 1978; Huff and Gerstner 1978). Of the 110 individuals who worked at the plant, more than 50% displayed "high blood levels" of the chemical, and in some instances family members of workers also had high blood levels. Patients suffering from chlordecone exposure presented with a characteristic suite of symptoms, including anxiety, irritability, memory loss, headaches, slurred speech, stuttering, tremors, abnormal gait, opsoclonus

(uncontrolled eye movements), abnormal liver function, neurotoxicity, and low sperm counts (oligospermy) and motility. Effects on the gonads or other components of the human reproductive system occurred in both sexes (Huff and Gerstner 1978).

In fact, a report of the National Academy of Sciences indicated that kepone cause sterility through its action as an estrogen mimic (Anon. 1980). In female mice, kepone elicited a state of "constant estrus" by competing with the body's endogenous estrogen for receptor sites in the uterus. More recently, Johnson (1996) challenged this conclusion when he showed that chlordecone has a low affinity for the estrogen receptor in ovariectomized rats. Johnson (1996) attempted to clarify the uterotropic effects of chlordecone in the presence or absence of estradiol. He concluded that chlordecone does NOT function as an estrogen antagonist for those functions that involve uterine estrogen receptors, but indicated that the mechanisms for chlordecone action on reproduction remain uncertain. Nonetheless, possible interactions between xenoestrogens and natural estrogens in the body need to be considered when assessing the risks of contaminants such as chlordecone.

Estrogen antagonists can affect other organs besides the uterus, including the central nervous system and hypophyseal gonadotrophic cells (Johnson 1996). In fact, Wang et al. (2008) recently showed that chlordecone exposure has an overall estrogenic effect on autoimmune response development (not unlike those caused by some other organochlorine pesticides), but that splenic T-cells respond somewhat differently to chlordecone and to 17-β estradiol.

Studies of laboratory mammals have linked chlordecone exposures to hepatocellular carcinomas (Reuber 1977, cited in Huff and Gerstner 1978). The liver has been shown (Fariss et al. 1980) to be the site where chlordecone metabolism (via reduction reactions, followed by glucuronidation) occurs. In fact, not surprisingly the ability of hepatic cells to metabolize chlordecone appears to be species specific (Fariss et al. 1980).

Chlordecone's toxicity corresponds to that of heptachlor (Brooks 1974, cited in Huff and Gerstner 1978). LD50 levels reported in the literature (summarized by Huff and Gerstner 1978) suggested that concentration levels as low as 65 mg/kg could be lethal to laboratory mammals, and in one study of chicks, an LD 50 of 480 mg/kg was determined. In several species of marine and freshwater fishes exposed to chlordecone for 48-96 hours, LD 50 concentrations were less than 100 µg/liter, and as low as 20 µg/liter.

A concern with regard to chlordecone exposure is the presence of delayed or otherwise subtle effects. In laboratory rats given a single dose, abnormal physiological responses, such as tremors, intensified startle response, and abnormal gait, lasted at least two weeks, but muscle weakness lasted six months (Egle et al. 1979).

Nor are effects of exposure to chlordecone limited simply to direct effects on exposed individuals. In sheepshead minnows (*Cyprinodon variegates*) for example,

Goodman et al. (1982) showed that the maximum acceptable concentration of the chemical was between 0.074 and 0.12 µg chlordecone/liter of sea water, and that at higher concentrations, a number of problems arose, including: external signs of poisoning ($\geq$ 0.074 µg/l; fatty degeneration of the liver (0.78 µg/l); reduced growth of adults (between 0.39 and 0.78 µg/l); lower fecundity and fertility of eggs (0.78 µg/l); and reduced survival of embryos of produced by fish exposed to 0.78 µg/l. Such life history level consequences mean that multiple generations may be affected by exposures of adult individuals.

Six years after the use of chlordecone was banned in Guadeloupe, the chemical was detected Multigner et al. 2006) in 88/100 men who were tested (detection limit ~ 1ng/ml) and quantified in 78/100 (quantification limit ~ 3ng/ml). Banana workers with "occupational exposure" had slightly higher levels than did men from non-agricultural sectors of the population; thus occupation exposure was important but not the only mechanism by which people were exposed. In this study, the higher blood values were not statistically related to impairment in terms of sperm numbers, motility or morphology.

Chlordecone in the French West Indies

In Guadeloupe and Martinique, widespread use of chlordecone occurred starting in 1972 in order to effectively control the banana weevil, a pest that affects the commercially important banana industry of those islands. Persistent organochlorines have long been prohibited in France due to their propensity to bioaccumulate, and the sale of chlordecone, specifically, was banned in 1990 (Coat et al. 2006). However, the lack of an effective alternative at that time to control the weevils led to an exemption on the 1990 ban until September 1993. Between 1972 and 1978, chlordecone was acquired (as kepone) from LSPC, but after that plant closed, the chemical was purchased from 1982-1993 (as curlone) from Calliope S.A. (Port-la-Nouvelle, France; Cabidoche et al. 2009).

A number of recent papers (Cabidoche et al. 2009; Coat et al. 2006; Dubuisson et al. 2007; and Guldner et al. 2010) have documented the persistence of chlordecone in soils, aquatic organisms, and people of Guadeloupe and Martinique. Cabidoche et al. (2009) provided especially worrisome prognostications: these authors used empirical data on levels of chlordecone in volcanic soils of the islands, and then applied a leaching model that indicated that depending on soil type, pollution may last anywhere from several decades (for nitisol) to centuries (ferralsol), to approximately half a millennium (for andosol).

Coat et al. (2006) assessed chlordecone levels in a variety of freshwater and marine fishes and invertebrates in Martinique; their sampling occurred in January-February 2002, approximately a decade after the use of chlordecone ceased. Not

surprisingly, these authors noted that highest concentrations of chlordecone occurred in carnivorous and detritivorous species of fish and prawns, due to magnification up the food chain. Reef-dwelling species such as spiny lobster (13-31 µg/kg) and surgeonfish (4.1 µg/kg wet weight) demonstrated measurable levels of chlordecone, but contained less of the chemical than did many other species. The highest levels observed in this study were in wild and farm-reared tilapia (*Oreochromis spp*.), where values as high as 386 µg/kg wet weight were found; those very high levels were from red tilapia caught in an area where the Lézarde River traverses a banana plantation.

Multigner et al. (2006) noted the persistence of chlordecone levels in human serum. Guldner et al. (2010) went so far as to use the word "permanent" to describe the duration of chlordecone pollution in Guadeloupe; these authors also documented currently-high concentrations of chlordecone in human blood, which they attributed primarily to dietary, rather than occupational exposures. Blood chlordecone concentrations (BCC) in Guldner et al.'s study were significantly correlated ($r = 0.47$; $p<0.0001$) with food exposure predicted from empirical weight models, and BCC averaged 0.86ng/ML among 191 subjects. The main dietary contributors were root vegetables, seafood, and cucurbitaceous plants such as melons and squashes; among the subjects tested, the mean per capita intake of chlordecone was 3.3µg/day (range = 0.1-22.2 µg/day).

In Martinique, consumption of subsistence-produced foods also places the human population at risk of consuming chlordecone in excess of recommended value (the so-

called chronic health-based guidance value or CHGV; Dubuisson et al. 2007) of 0.5 µg/kg body weight/day. In this study, the likelihood of exceeding the CHGV was more than 20% for children and nearly 16% for adults.

Allowable Levels and Limits in Food

Coat et al. (2006) summarize information with regard to allowable levels of chlordecone or related organochlorine pesticides (OCPs) in food. The toxicity reference value (i.e., the maximum OCP concentration tolerated in food) is determined in France by the Institute Français National de veille Sanitaire (INVS) and in the United States by the Food and Drug Administration (FDA). The toxicity reference values for these two organizations are identical: 0.5 µg/kg/day, or 30 µg/day for a 60 kg adult. In order to exceed the acceptable daily intake (ADI) based on the data from Coat et al. (2006) for Martinique species of fish and shellfish, a person weighing 60 kg would have to consume 77g of wild tilapia, 227 g of farmed tilapia, 1000 g of spiny lobster, or 1300 g of prawn each day. Some of these values easily fall within the possible daily consumption for an individual: 77 g of wild tilapia, for example, is 0.16 pounds of meat, and even 277 g of farmed tilapia represents a little more than ½ pound of meat. The amount of lobster (2.2 pounds) by itself is a relatively large amount, but when combined with other possible sources of chlordecone (e.g., root vegetables; melons), could be reached.

The FDA determined that chlordecone concentrations of 0.3 mg/kg of edible fish tissue and 0.4 mg/kg of crab flesh represented the intervention limit, a point which is

designed to provide some leeway before consumption of these foods is prohibited. Coat et al. (2006) were prudent to note that other items in a person's diet also may contain chlordecone or other OCPs; for example, root vegetables grown in soil contaminated by banana plantation run off could contribute significantly to someone's overall OCP intake on a daily basis.

Results of This Study

This study detected and assessed the levels of 10 different chemical contaminants of concern in samples of seagrasses and marine sediments from the Grand Cul-de-Sac Marin, Guadeloupe, French West Indies (see Figure 1 for sampling locations). Levels of chlordecone were of greatest interest.

The results of this study appear in Table 1. Although the presence of chlordecone was indicated in two sediment samples, there was only a single sample (i.e., sample # 4b-sed (Figure 1) in which the measured amount of chlordecone (i.e., 11 µg/kg) exceeded the detection limits of the instrument.

In contrast, the contaminants (or metabolites thereof) that were present at the highest levels were certain polycyclic aromatic hydrocarbons (PAHs) and dithiocarbamate. The latter was found in almost all samples of seagrasses and sediments from the Grand Cul-de-Sac Marin, whereas the PAHs were at detectable levels in very few samples.

Interpretation and Implications of Our Results

The measured levels of chlordecone in marine angiosperms and marine sediments associated with the Grand Cul-de-Sac Marin were extremely low; absent other information to the contrary, the measured levels do not appear to pose a health risk to humans or to other species, including manatees which might be re-introduced to that location.

We are somewhat surprised at how little chlordecone was detected in our study. In terrestrial sediments (Cabidoche et al. 2009) and in certain foods such as root vegetables, melons and seafood on Guadeloupe (Guldner et al. 2010), measured chlordecone levels were sufficiently high as to cause concerns about human health and reproductive capacity. Similarly, in fish and shellfish sampled off nearby Martinique, chlordecone levels are sufficiently high (Coat et al. 2006) that a person could easily exceed the acceptable daily intake amount.

The low amounts we measured in our samples may be explained by at least a couple of alternatives. First, the use of chlordecone in the latter part of the 10th century was focused on banana farms, so if the drainage into the Grand Cul-de-Sac Marin includes fewer banana farms than do other locations in Guadeloupe, then the watershed and embayments should have reduced concentrations of the chemical.

In addition, our study involved plants and sediments, whereas the Martinique assessments of chlordecone (Coat et al. 2006) involved fish and shellfish. It is possible that organisms in the Grand Cul-de-Sac Marine at a trophic level above plants would have measurable, and perhaps significant body burdens of chlordecone.

In fact, Connolly and Tonelli (1985) developed a mathematical model to attempt to clarify the relationship between kepone levels in striped bass and other teleosts, and those measured in the water column and sediments. In organisms at higher trophic levels, diet is the most important route of contamination (as it is in people on Guadleoupe; see Guldner et al. 2010). The model indicated that for kepone concentrations to remain at or below 0.3 µg/g in fish tissues, the concentrations in the water column and sediments would be between 3-9 ng/l and 13-39 ng/g, respectively. Since these sediment values are considerably less than what we found in our study, we are inclined to believe that fish from the Grand Cul-de-Sac Marin are safe to eat, with regard to chlordecone levels; nonetheless it would be prudent to assess levels in some representative fish species consumed by people.

OTHER CONTAMINANTS OF CONCERN:

Although the primary purpose of the study was to examine levels of chlordecone in the shallow marine environment of the Grand Cul-de-Sac Marin, our data suggest that other chemical contaminants may be of greater concern than chlordecone in that location

(Table 1). Among the contaminants that were found were the dithiocarbamate fungicides (DTCs) and a number of polycyclic aromatic hydrocarbons (PAHs).

Dithiocarbamate:

This chemical was easily the most ubiquitous contaminant in the samples we examined, with levels equaling or exceeding 100µg/kg in three of 29 samples. The DTCs were introduced approximately 40-70 years ago, primarily for use in agriculture to control a wide range of plant pathogens; Crnogoroc and Schwack 2009 list more than a dozen of the more important dithiocarbamate fungicides. In general, the DTCs are considered to be of low cost to produce and of low acute mammalian toxicity (Crnogorac and Schwack 2009). Interestingly, among other things, DTCs are used clinically for the treatment of chronic alcoholism and as anticancer and antitoxic drug agents (Crnogorac and Schwack 2009).

The DTCs are organosulfur compounds that form polymers with transition metals such as manganese or zinc. The diversity and high activity of DTCs causes them to be used to control nearly 400 pathogens for 70 different crops. The presence of the heavy metal ion in the molecule increases the potential toxicity of that molecule, and exposures to DTCs have been reported to cause problems such as eye, skin or respiratory tract irritation and dermatitis in people (Kazos et al. 2007). Furthermore the metabolites of DTCs can produce neurotoxic effects or impact thyroid function. Despite the controlled use of DTCs as anticancer agents noted above, Kazos et al. (2007) indicate that these compounds may have carcinogenic, mutagenic and teratogenic effects. For example,

Irons and Pyatt (1998) implicated DTCs in causation of hematopoietic neoplasms and in hematotoxicity and immunotoxicity.

Domico et al. (2007) conducted studies of the extent to and mechanism by which one specific compound (ethylene-*bis*-dithiocarbamate (EBDC) found in the product, mancozeb (which contains manganese), contributed to neuronal toxicity in the mesencephalon; their findings are of interest because of the increasing awareness of neurodegenerative diseases and possible environmental (non-genetic) causes. Inasmuch as DTCs are used very widely (residential lawns; golf courses; agricultural areas), the health risks could be substantial. Domico et al. (2007) demonstrated that mancozeb-treated neuronal cells produced large amounts of reactive oxygen species, and that the induction of these species contributes to neurotoxicity.

Recent papers (e.g., Kazos et al. 2007; Crnogorac and Schwack 2009) describe current efforts to develop improved analytical methods for the DTCs. In fact, the possibility, or likelihood of effects of DTCs on human health need further assessment, resulting in the creation of toxicity reference values. In that regard, the recent study of toxicity of the fungicide, Tricyclazole, on a common, regional food fish (*Ophiocephalus leucopunctatus*; a type of snakehead) grown in rice fields, found an LD50 value of 15ppm; interestingly, the fungicide was used at concentrations of 1000 ppm in the field (Vivek et al. 2009). These authors also related specific organ damage to particular DTC concentrations, and they concluded that DTC levels could affect both aquatic species and the humans that consume them.

Since DTC levels ranged between 50 and 124 µg/kg (between 0.050 and 0.124 ppm) in 25 of 29 samples in our study, and since LD 50 levels in snakehead fish were at 15 ppm, we believe that dithiocarbamate does not represent a great potential problem for inshore marine ecosystem and human health in Guadeloupe. However, the fact that it is found so ubiquitously in the sediments and sea grasses of the Grand Cul-de-Sac Marin and that the LD 50 values apply to just one fish species, we suggest that dithiocarbamate may be of more concern in the inshore marine environment than chlordecone.

Polycyclic aromatic hydrocarbons (PAHs):

In contrast to the dithiocarbamates, which were found rather ubiquitously in our samples, PAHs were found in relatively few (sometimes only 1) samples (Table 1). Sample 4c-sed was especially contaminated in this study, containing five of the eight PAHs identified in this study. Four of the eight PAHs present at detectable levels in samples from the study area (namely benzo(a)anthracene, benzo(a)pyrene, benzo(a)phenanthrene; and indeno(1,2,3-cd)pyrene) are considered toxic (http:///www.mass.gov/dep/toxics/pahs.htm) and one (benzo(a) pyrene) is sufficiently toxic to be considered one of the most hazardous chemical compounds (among the worst 10%) in existence to both human and ecosystem health (www.**scorecard.org**/chemical-profiles/summary.tcl?edf). The Stockholm Convention (signed in 2001) considered benzo(a)pyrene to be a priority chemical contaminant, one of a dozen for which the Convention prioritized establishment of control and phase-out measures.

Benzo(a)pyrene is the most studied PAH, among the hundreds of PAH molecules in existence (Scientific Committee on Food 2002).

In fact, different PAHs can have a wide range of effects on humans, including carcinogenesis, genotoxicity, immunosuppression, and reproductive and developmental toxicity (Scientific Committee on Food 2002).

Long et al. (1995) considered whether, and if so to what extent, a variety of chemical contaminants in sediments affected biology of local organisms (fishes and invertebrates). Among the chemicals tested in the study were 13 PAHs, including two (benzo(a)anthracene and benzo(a)pyrene) found in sediments of Guadeloupe. The PAHs were among the chemicals for which incidence of effects was directly correlated with sediment concentrations.

In order to assess the incidence of biological effects, Long et al. (1995) defined two "guideline values": concentrations below the ERL (Effects range-low) value were those for which effects would "rarely" be observed; whereas concentrations at the ERM (Effects range-median) value were considered those that produced "frequent" effects. For the two PAHs of particular interest in terms of the Guadeloupe sediment/seagrass study, the ERLs for benzo(a)pyrene and benzo(a)anthracene were 430 ppb (dry weight) and 261 ppb (dry weight) respectively. For concentrations below these ERL levels, the percentage of incidence of effects was 10.3% for benzo(a)pyrene and 21.1% for benzo(a)anthracene. It should be noted that in sample 4c-sed, the values we determined

for these two chemicals were 13 µg/kg (= 13 ppb) for benzo(a)anthracene and 12 µg/kg (= 12 ppb) for benz(a)pyrene, well below the ERL levels suggested by Long et al. (1995), but still with a capacity to elicit biological effects.

With regard to effects on the health of wildlife, Martineau et al. (2002) stated that high levels of benzo(a)pyrene in the St. Lawrence Seaway were the cause of the very high incidence of cancer in local beluga whales (*Delphinapterus leucas*).

As we have indicated for chlordecone and the DTCs, it is difficult to relate sediment levels of contaminants with particular problems for human or environmental health. For humans, the major routes of exposure to PAHs are via food and inhalation (including cigarette smoking), although contaminated drinking water can also contribute to PAH intake (Scientific Committee on Food 2002). It is important to realize that PAHs in food can arise from a number of possible sources: environmental contamination (e.g., deposition following combustion, oil spills) or via preparation and cooking (e.g., drying, smoking, grilling). Thus, as noted, there is often not a clear relationship between PAH levels in the environment and those to which humans are exposed.

With regard to dietary intakes of PAHs, a number of study results from Europe are summarized by the Scientific Committee on Food (2002). Although it is beyond the scope of this report to describe the results in detail, for benzo(a)pyrene, the maximum intakes were around 0.42 µg/day (in the Netherlands) and the maximum for benzo(a)anthracene was slightly higher, at 0.65 µg/day (also in the Netherlands).

Interestingly, for these two toxic PAHs, the upper bound intake levels in 2000 were lower than the lower bound intakes in 1979 (Scientific Committee on Food 2002).

The presence of particularly toxic PAHs in certain sediment samples in our study probably warrants monitoring, and it would be useful to assess why certain locations have both more-diverse and more-abundant PAHs present so that mitigation might occur. As an interesting and related observation, in sediments we have examined in Cook Inlet, Alaska, PAH diversity is markedly higher, as are the concentrations of most specific PAHs, including especially toxic components such as benzo(a)pyrene (Wetzel and Reynolds, unpublished); in Cook Inlet, concerns exist that environmental contamination by PAHs and other organics may be impairing recovery of endangered beluga whales.

SUMMARY AND CONCLUSIONS:

In Guadeloupe (French West Indies), an ambitious idea has been developed, namely to re-introduce manatees to the waters of the Grand Cul-de-Sac Marin. If such a project occurs, its success will be dependent to an extent on identification and mitigation of potential threats or risk factors, one of which is environmental contamination.

Of particular concern in the French West Indies is residual levels of the insecticide, chlordecone. Our study sampled sediments and seagrasses at a number of

locations in the area of the Grand Cul-de-Sac Marin to assess levels of chlordecone and other organic contaminants.

Our results indicate that chlordecone levels are undetectable in most samples and very low in the two samples where the presence of chlordecone was documented. In addition, although dithiocarbamates were found in almost all samples, the levels do not appear to be sufficiently high to warrant concerns for human health, or likely for manatee health, although that remains an uncertainty. Finally, only eight PAHs were found in a small number of samples, but four of the eight are notably toxic (especially benzo(a)pyrene).

Our study suggests that chlordecone in the Grand Cul-de-Sac Marin is not a problem for manatee or human health. Other chemical contaminants are of greater concern there, and even though their levels may not be sufficient to cause health effects, we recommend monitoring studies to (a) assess possible trends in contaminant levels of time, and (b) to determine sources of those chemicals and as possible reduce them.

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Figure 1. Sampling station map

Table 1. Detectable levels of organic contaminants by station and matrix in ug/kg.

Station	phenanthrene	anthracene	pyrene	benzo(a) anthracene	benzo(a)pyrene	benzo(a) fluoranthene	benzo(ghi) perylene	indeno(1,2,3-cd) pyrene
1Bis b-sed								
1Bis c-sed								
1Bis b-thal						10		
2c –thal			70					
2b-sed								
2c-sed								
3a-sed								
3b-sed								
3c-sed								
4a-sed					126	16		
4b-sed								
4c-sed				13	12	14	22	10
5a-sed								
5b-sed								
5c-sed								
6a-sed								
6b-sed								
6c-sed					24			
7a-sed								
7b-sed								
7b-thal		40						

8a-sed									
8b-sed									
8c-sed									
11c-thal	60								
14a-sed									
14b-sed									
14c-sed									
15c-sed									