

CIGUATERA POISONING, CORAL REEFS, AND CLIMATE OSCILLATIONS
IN RAROTONGA, SOUTHERN COOK ISLANDS

by

TEINA RONGO

B.A., University of Guam
M.S., University of Guam

A dissertation submitted to the Department of Biological Sciences
of Florida Institute of Technology in partial fulfillment
of the requirements for the degree of

DOCTOR OF PHILOSOPHY
in
BIOLOGICAL SCIENCES

Melbourne, Florida
December 2011

UMI Number: 3489037

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



UMI 3489037

Copyright 2011 by ProQuest LLC.

All rights reserved. This edition of the work is protected against unauthorized copying under Title 17, United States Code.



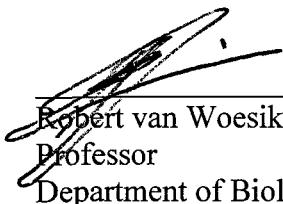
ProQuest LLC
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106-1346

CIGUATERA POISONING, CORAL REEFS, AND CLIMATE OSCILLATIONS
IN RAROTONGA, SOUTHERN COOK ISLANDS
A DISSERTATION

by

TEINA RONGO

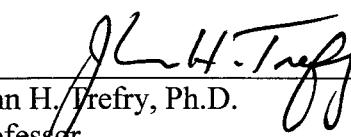
Approved as to style and content by:



Robert van Woesik, Ph.D., Chairperson
Professor
Department of Biological Sciences



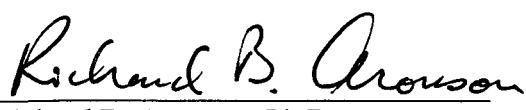
Mark B. Bush, Ph.D.
Professor
Department of Biological Sciences



John H. Trefry, Ph.D.
Professor
Department of Marine and Environmental Systems



Ralph G. Turingan, Ph.D.
Professor
Department of Biological Sciences



Richard B. Aronson, Ph.D.
Professor and Head
Department of Biological Sciences

December 2011

ABSTRACT

CIGUATERA POISONING, CORAL REEFS, AND CLIMATE OSCILLATIONS IN RAROTONGA, SOUTHERN COOK ISLANDS

By Teina Rongo, B.A., University of Guam;
M.S., University of Guam

Chairperson of Advisory Committee: Robert van Woesik, Ph.D.

The ocean and its resources have sustained island nations for millennia, yet an exclusive reliance on fishes can be catastrophic for an island population when those resources become inedible. Ciguatera poisoning is particularly problematic, because it not only causes health problems but also has serious socioeconomic repercussions. Ciguatera poisoning has been problematic in the southern Cook Islands for around 20 years, particularly in Rarotonga. This study examined the ciguatera problem by addressing the following questions: 1) Can we predict the spatial extent of ciguatera poisoning? 2) Can we use the concept of shifts in ciguatera-poisoning vectors to forecast the intensity of upcoming ciguatera outbreaks? 3) Is ciguatera poisoning in the southern Cook Islands linked to major climate cycles? 4) Is ciguatera poisoning influenced by reef disturbances, reef state, and herbivorous fish density? 5) What are the socioeconomic impacts of ciguatera poisoning? and 6) Did ciguatera poisoning occur in the past, and if so, did ciguatera poisoning prompt the late Holocene biogeographic distribution of humans across the Pacific Ocean? To examine these questions, I conducted reef surveys, obtained climate data from various sources, conducted questionnaire surveys among residents of Rarotonga, and carried out an extensive review of the archeological

literature, from the central Pacific, for evidence of ciguatera poisoning in the late Holocene between AD 1000 and 1450.

I found that coral cover is not a good predictor of ciguatera poisoning, but increased density of herbivorous fishes may be linked to increased cases of ciguatera poisoning. The surveys at the widest reefs of Rarotonga elicited the most cases of ciguatera poisoning, and vector shifts in fishes involved in poisonings can forecast the dynamics of the ciguatera-poisoning cycle. Wavelet analyses, used to determine the dominant climate cycle in this region, showed a link with ciguatera poisoning. Cases of ciguatera poisoning in Rarotonga were linked with El Niño years, the positive phase of the Pacific Decadal Oscillation, and periods with frequent reef disturbances. Ciguatera poisoning has also contributed to Rarotonga residents shifting away from a fish diet and towards a diet of imported meats and canned fish. The questionnaire surveys and the *cost savings and avoidance* valuation technique, used to determine the socioeconomic impacts of ciguatera poisoning on residents, showed that ciguatera poisoning cost the society ~NZD \$1.5 million per year. I suggested that the abrupt changes in fish middens from archeological studies in this region were the result of ciguatera poisoning, and that the celebrated Polynesian voyages across the Pacific Ocean may not have been random episodes of discovery to colonize new lands, but rather, voyages of necessity. With the recent shift in climate oscillations to the negative phase of the PDO, coupled with La Niña years, cases of ciguatera poisoning in Rarotonga are predicted to decline.

For O'Neal Isaia, Myria Moana, Konini, and Raetea Lyn

ACKNOWLEDGMENTS

I am very grateful to my advisor Dr. Robert van Woesik for giving me the opportunity to pursue a Ph.D. by accepting me into his lab, and for his invaluable guidance over the years. Thanks to my research committee members Dr. Richard Aronson, Dr. Mark Bush, Dr. John Trefry, and Dr. Ralph Turingan for their invaluable input. Reo akameitaki to my parents Ngatamaine-toko-toru and Tera-itirere-ki-avaiki Rongo, my brothers and sisters Teariki and Julia, Tauraki and Cathy, Pa and Nooroa, Grace and Mona, Raita and Ashleigh, Tangi, Mii and Moetu, and Tereapii and Dernice, and my nephews and nieces for their support. Much appreciation to my in-laws in Florida, Mark and Julie Zehr, for their generous support. I also thank Dr. Gustav Paulay and Dr. Robert Richmond for their guidance throughout my studies at the University of Guam. Meitaki maata to Tearoa Iorangi of the Cook Islands Ministry of Health, Arona Ngari and the staff of the Cook Islands Meteorological Service, and Davina Hosking-Ashford of New Zealand's National Institute of Water and Atmospheric Research for data assistance, as well as Howard Tangimetua of the Cook Islands Ministry of Health, Vavia Tangatataia of the Cook Islands National Environment Service, Tuaine Turua, Ian Bertrem, Kori Raumea, and Ben Ponia of the Ministry of Marine Resources, and the staff of the Cook Islands Statistics Office. Many thanks to Sandra van Woesik for her suggestions and editorial assistance. Dankolo na si yu`us ma`ase to Dr. Peter Houk for his friendship and help over the years. Thanks to the Cook Islands Human Resources Department for their financial assistance with partial tuition reimbursement, and Dr. Bush for a semester of financial support through Teaching Assistance. Meitaki maata to the residents of Rarotonga who participated in my research, and Enoa Rae & Rutaki Primary School, Papatua Papatua & Avarua Primary School, and Teariki Jacob Jr. & Titikaveka College for assistance in questionnaire surveys. Thanks to Dive Rarotonga and Cook Island Divers for tank rentals, and Teariki Charles Rongo, Nooroa Unuia, and my numerous boatmen for field assistance. Finally, I am especially grateful to my lovely wife Jackalyn Rongo for always being there for me through the ups and downs of this journey.

TABLE OF CONTENTS

ABSTRACT.....	iii
DEDICATION.....	v
ACKNOWLEDGMENTS.....	vi
TABLE OF CONTENTS.....	vii
LIST OF TABLES.....	x
LIST OF FIGURES.....	xii
CHAPTER I – INTRODUCTION.....	1
CAUSATIVE ORGANISMS.....	3
FACTORS AND INFLUENCE TOXIN PRODUCTION AND GROWTH OF CIGUATOXIC DINOFLAGELLATES.....	5
SPATIAL AND TEMPORAL DISTRIBUTION.....	8
CLIMATE, REEF DISTURBANCES, AND CIGUATERA POISONING.....	10
SOCIAL AND ECONOMIC IMPACTS.....	12
HISTORICAL ACCOUNTS.....	14
RESEARCH QUESTIONS.....	15
CHAPTER II – THE SPATIAL AND TEMPORAL DISTRIBUTION OF CIGUATERA POISONING IN RAROTONGA, SOUTHERN COOK ISLANDS.....	17
INTRODUCTION.....	17
MATERIALS AND METHODS.....	21
QUESTIONNAIRE SURVEY.....	21
DATA PREPARATION.....	22
DATA ANALYSIS.....	24
RESULTS.....	25
THE SPATIAL DISTRIBUTION OF FISHES AND INVERTEBRATES INVOLVED IN CIGUATERA POISONING.....	27
VECTOR SHIFTS OF CIGUATERA POISONING.....	31
DISCUSSION.....	33
SPATIAL DISTRIBUTION OF CIGUATERA POISONING.....	35
VECTOR SHIFTS OF CIGUATERA POISONING.....	36
ALGAL COMMUNITY AND CIGUATERA-CAUSING DINOFLAGELLATES.....	38

CHAPTER III – THE INFLUENCE ON CLIMATE OSCILLATIONS ON CIGUATERA POISONING IN RAROTONGA, SOUTHERN COOK ISLANDS.....	43
INTRODUCTION.....	43
MATERIALS AND METHODS.....	46
DATA ANALYSIS.....	46
RESULTS.....	49
DISCUSSION.....	55
CHAPTER IV – THE EFFECTS OF NATURAL DISTURBANCES ON REEF STATE AND HERBIVOROUS FISH DENSITY, AND THEIR INFLUENCE ON CIGUATERA POISONING IN RAROTONGA, SOUTHERN COOK ISLANDS.....	59
INTRODUCTION.....	59
MATERIALS AND METHODS.....	63
DATA ANALYSIS.....	63
RESULTS.....	65
REEF STATE: 1970s – 2011.....	65
HERBIVOROUS FISHES.....	68
DISCUSSION.....	70
CHAPTER V – SOCIOECONOMIC IMPACTS OF CIGUATERA POISONING IN RAROTONGA, SOUTHERN COOK ISLANDS.....	78
INTRODUCTION.....	78
METHODS AND MATERIALS.....	83
PROTEIN CONSUMPTION.....	83
COST SAVINGS AND AVOIDANCE ANALYSIS.....	84
GROSS VALUE OF FISH-STOCK LOSS.....	85
MONITORING AND MANAGEMENT COSTS.....	85
HEALTH-RELATED COSTS.....	86
DATA ANALYSIS.....	87
RESULTS.....	88
PROTEIN CONSUMPTION.....	88
FISHING FREQUENCY.....	92
COST SAVINGS AND AVOIDANCE ESTIMATES.....	92
GROSS VALUE OF FISH-STOCK LOSS.....	92
MONITORING AND MANAGEMENT COSTS.....	93
HEALTH-RELATED COSTS.....	94
CONTRIBUTION OF CLIMATE TO CIGUATERA POISONING.....	95
DISCUSSION.....	97

CHAPTER VI – DID CIGUATERA POISONING INFLUENCE THE LATE HOLOCENE BIOGEOGRAPHIC DISTRIBUTION OF HUMANS ACROSS THE PACIFIC OCEAN?.....	104
INTRODUCTION.....	104
OVERVIEW OF CIGUATERA.....	107
EFFECTS OF CIGUATERA IN THE SOUTHERN COOK ISLANDS..	108
PALAEOCLIMATE IN THE PACIFIC.....	109
CLIMATE AND CIGUATERA POISONING EVENTS.....	111
EVIDENCE OF CULTURAL ADAPTATIONS.....	114
DISCUSSION.....	117
CHAPTER VII – SYNTHESIS AND CONCLUSIONS.....	124
LITERATURE CITED.....	130
APPENDICES.....	147

LIST OF TABLES

Table 1. Cases of ciguatera poisoning on Rarotonga derived from the questionnaire survey conducted between December 2008 and January 2010

Table 2. Reef fishes and marine invertebrates involved in ciguatera poisoning that were reported in the questionnaire survey conducted between December 2008 and January 2010.

Table 3. Fishes currently considered edible (from local knowledge) in Rarotonga.

Table 4. Analysis of Similarity pair-wise test between exposures (leeward vs. windward) and among the three categories of lagoon widths (narrow, intermediate, and wide) in Rarotonga.

Table 5. Eigenvalues and eigenvectors of Principle Component Analysis of ciguatoxic reef fishes and marine invertebrates in Rarotonga for the three periods examined (1989 – 2000, 2001 – 2005, and 2006 – 2009).

Table 6. Natural disturbances (e.g., cyclones, *Acanthaster planci* outbreaks, coral bleaching, and harmful algal bloom) impacting Rarotonga between 1970 and 2011.

Table 7. Chi-square test comparing natural disturbances between the two ciguatera-poisoning events identified in Chapter II: (1) the 1970s event, and (2) from 1989 onwards.

Table 8. Chi-square tests comparing Rarotonga's hospital cases of ciguatera poisoning from 1994 to 2011 (August) during the positive and negative phases of the Southern Oscillation Index (SOI; La Niña and El Niño, respectively), using a 1-year lag period between climate data and case of ciguatera poisoning.

Table 9. The mean percentage cover of hard corals, turf algae, and macroalgae (\pm standard error) at fore-reef sites surveyed in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2003 (Lyon 2003), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011.

Table 10. Mean density of acanthurids, *Ctenochaetus striatus*, and scarids (\pm standard error) at fore reef sites surveyed in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011.

Table 11. Protein consumption of Rarotonga residents based on a questionnaire survey conducted in 2011, where n represents the number of households that consumed each food the week before the survey.

Table 12. Percentage (%) of meat (e.g., chicken, other meat, corned beef, lamb, and pork) and seafood (fresh fish, canned fish, and other seafood) consumed by households in 1989 & 2001 (Solomona et al. 2009), 2006 (Moore 2006), and 2011 (the present study).

Table 13. Average per-capita seafood consumption (g/person/day) for 1989 and 2001 (Solomona et al. 2009), 2006 (Moore 2006), 2007 (Pinca et al. 2007), and 2011.

Table 14. The percentage of reef, pelagic, and freshwater fishes consumed by 70 households the day before the survey in 2011.

Table 15. Consumption of potentially ciguotoxic reef fishes (kg/year) in Rarotonga from 1989 to 2011.

Table 16. Health-related costs per individual with ciguatera poisoning.

Table 17. Total economic impact (NZD per year) of ciguatera poisoning in Rarotonga for 2001, 2006, and 2011.

Table 18. Average annual economic impact of ciguatera poisoning at different climate phases.

Table 19. Summary of fish middens from archaeological records (limited to potentially ciguotoxic fishes taken by angling), and terrestrial resources consumed.

LIST OF FIGURES

- Figure 1. Map of the Pacific with the Cook Islands' Exclusive Economic Zone delineated.
- Figure 2. Rarotonga ($21^{\circ}14'15''S$ $159^{\circ}46'48''W$) indicating villages in windward and leeward exposures.
- Figure 3. Cases of ciguatera poisoning from Rarotonga's hospital data (thick line) and the questionnaire survey (thin line).
- Figure 4. An ordination of sites using total cases of ciguatera poisoning that was derived from the questionnaire survey conducted between December 2008 and January 2010.
- Figure 5. Principal Component Analysis of reef fish and marine invertebrate species involved in ten or more cases of ciguatera poisoning in Rarotonga plotted in relation to the three periods (1989 – 2000, 2001 – 2005, and 2006 – 2009).
- Figure 6. Natural disturbance events impacting Rarotonga's reefs from 1970 to 2011.
- Figure 7. (a) Changes in estimated sea-surface temperature anomalies spanning from AD 1726 to 1996 for Rarotonga using coral Sr/Ca (Linsley et al. 2000).
- Figure 8. (a) Wavelet analysis of the Southern Oscillation Index (SOI) and cases of ciguatera poisoning from 1994 to 2011.
- Figure 9. Rarotonga ($21^{\circ} 12' S$, $159^{\circ} 43' W$) depicting the fore-reef sites surveyed in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2003 (Lyon, 2003), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011.
- Figure 10. (a) Mean hard coral, (b) turf algal, and (c) macroalgal cover on fore reef sites in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2003 (Lyon 2003), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011.
- Figure 11. Hospital cases of ciguatera poisoning in Rarotonga from 1994 to 2011 (August), and mean fish density (per $200 m^2$) in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011.

Figure 12. Average per-capita consumption (g/person/day) of total seafood (diamond), fresh fish (triangle) and canned fish (circle) from 1989 to 2011, and cases of ciguatera poisoning reported at the Rarotonga Hospital (Cook Islands Ministry of Health) (dashed line).

Figure 13. Percentage of reef (grey bars) and pelagic (open bars) fishes consumed by households in 1989 & 2001 (Solomona et al. 2009), 2006 (Moore, 2006), and 2011 (the present study).

Figure 14. (a) Waves of migration (shaded grey arrows) originating from East Polynesia (dotted region; French Polynesia) with approximate migration dates to Rapa Nui (Hunt & Lipo, 2006), Hawai'i (Kirch & Kahn, 2007), and New Zealand (Wilmshurst et al. 2008).

Figure 15. The South Pacific region with red areas indicating warmer sea surface temperatures (SSTs) and blue areas indicating cooler SSTs during (a) El Niño; and (b) La Niña (modified from Hales et al. 1999, with permission from Blackwell Publishing).

Figure 16. (a) Pacific Decadal Oscillation (PDO) index from 1930 to 2007 with an 11-year running average (data from <http://jisao.washington.edu/pdo/PDO.latest>).

Figure 17. Schematic of the influence of climate oscillations on ciguatera poisoning in Rarotonga, southern Cook Islands, based on the results of this study.

CHAPTER I

INTRODUCTION

Ciguatera poisoning is a phenomenon that has been well-documented throughout the tropical and subtropical Pacific Ocean, Indian Ocean, and Caribbean Sea (Quod and Turquet 1996; Lewis 2001). Ciguatera poisoning stems from the consumption of reef fishes that have inadvertently ingested toxins produced by dinoflagellates (e.g., *Gambierdiscus toxicus*), but ciguatera-like poisonings have also been noted from the consumption of invertebrates such as the giant clam (Laurent et al. 2008; Yeeting 2009a; Rongo and van Woesik 2011). Dinoflagellates are typically epiphytic to macroalgae (see Cruz-Rivera and Villareal 2006). Ciguatoxins accumulate and transform as they pass through the marine food web, concentrating in large carnivorous fishes (Lewis 2006). Ciguatera poisoning is not limited to humans, but can also affect terrestrial mammals (e.g., cats and dogs; Forster 2009) and marine mammals (e.g., monk seals; Bottein et al. 2011). Ciguatoxins can also have adverse effects on the reef fishes that become intoxicated, and have been shown to reduce the survival of fish larvae (Lewis 1992a; Landsberg 1995; Edmunds et al. 1999; Ajuzie 2008).

In humans, ciguatera poisoning has clinical symptoms that are gastrointestinal (i.e., vomiting and diarrhoea), neurological (e.g., temperature dysesthesia and chronic fatigue) and cardiovascular (e.g., bradycardia and tachycardia) (Gillespie et al. 1986; Lehane and Lewis 2000; Hokama and

Yoshikawa-Ebesu 2001; Dickey and Plakas 2010). Typically, the onset of symptoms occurs within 24 to 48 hours after consumption of ciguatoxic fishes, but in severe cases it can occur within 30 minutes. Symptoms can last from weeks to months and even years (see Shoemaker et al. 2010). Recurrence of symptoms is common among victims, particularly after consuming another fish meal within days of the initial intoxication. Interestingly, certain food items such as peanuts, chicken, and pork can also trigger the recurrence of symptoms in individuals recovering from ciguatera poisoning (Lewis 2006). Death from ciguatera poisoning is rare, but can occur in severe cases through cardiovascular shock, dehydration, cardiac arrhythmia or through respiratory failure (Bagnis et al. 1979; Lehane and Lewis 2000; Kumar-Roiné et al. 2010). Most deaths involving ciguatera poisoning appear among individuals with underlying health conditions (e.g., Rongo and van Woesik 2011). In addition, Lewis (1992a) suggested that the lethal effects of ciguatoxins in reef fishes may impose an upper limit to what the fish can accumulate; those that have accumulated ciguatoxins above that limit (which would have lethal effects on humans) are rarely caught by fishers.

Presently, there is no cure for ciguatera poisoning and only supportive treatment of symptoms is provided. Intravenous mannitol was used for treatment of severe cases in the 1980s (Palafox et al. 1988), however Schnorf et al. (2002) found no significant difference between mannitol use and intravenous saline in a double-blind clinical trial in Rarotonga, Cook Islands. Recent studies suggest that brevenal, a natural non-toxic compound extracted from *Karenia brevis* (the dinoflagellate

that causes red tide), is a potent inhibitor of ciguatoxin and therefore has the potential to treat ciguatera poisoning (Mattei et al. 2008). Traditional herbal remedies are commonly used in many Pacific islands to alleviate symptoms of ciguatera poisoning, where over 90 plant species have been recorded for use in ciguatera-poisoning treatment (see Boydron-Le Garrec et al. 2005).

Although ciguatera poisoning is the most common marine food poisoning known, the severity of the problem is unknown largely because only around 10 – 20% of cases are reported (Lewis 1986). Various estimates have been proposed on the number of people experiencing ciguatera poisoning globally, which ranged from 10,000 – 500,000 people per year (Ragelis 1984; Quod and Turquet 1996; Bruslé 1997; Fleming et al. 1998). However, the range between 50,000 – 500,000 people per year is frequently cited in the literature. The effects of ciguatera poisoning are not limited to the tropics. The global transportation of protein has increased the extent of ciguatera poisoning beyond the geographic range of intoxicated fishes. For example, fishes caught in the tropical Pacific Ocean are often exported to Europe, Asia, and the USA (see Van Dolah 2000; Wong et al. 2005; Dickey and Plakas 2010).

CAUSATIVE ORGANISMS

Early studies of ciguatera poisoning postulated that fishes became toxic as a direct consequence of their diet. For example, Dawson et al. (1955) suggested that the source of ciguatoxins stemmed from benthic algae. Subsequently, Randall

(1958), working in French Polynesia, proposed that the toxin was produced by a benthic micro-organism, which was first ingested by herbivorous fishes and then transferred to larger carnivores. This transfer of toxins up the food web became known as the ‘food web concept of ciguatera’, but was not empirically validated for another 20 years. After examining the gut content of *Ctenochaetus striatus*, a fish commonly implicated in ciguatera poisoning, Yasumoto et al. (1977) proposed that the dinoflagellate *Gambierdiscus toxicus* Adachi and Fukuyo, 1979 was the primary causative agent producing ciguatoxin. Research on dinoflagellate toxins, and their transfer through the food chain to humans, is well-documented (Hashimoto 1979; Baden 1983; Bagnis et al. 1985; Anderson and Lobel 1987; Miller 1991; and Tosteson 1992). Other dinoflagellates have also been implicated in causing ciguatera poisoning. For example, dinoflagellates from the genera *Ostreopsis*, *Coolia*, *Prorocentrum* and *Amphidinium* have been reported in ciguatera-impacted areas (Ballantine et al. 1985; Carlson and Tindall 1985; Bomber and Aikman 1989; Bourdeau et al. 1995; Faust 1995), and have the potential to produce toxins. They can also co-occur with *Gambierdiscus* spp. (Yasumoto et al. 1980; Nakajima et al. 1981; Rhodes et al. 2009). In the Caribbean, Ballantine et al. (1985) and Carlson and Tindall (1985) suggested that *Ostreopsis* spp. and *Prorocentrum lima* were important contributors to ciguatera poisoning. A variety of dinoflagellates involved in ciguatera poisoning may explain why multiple toxins are found in most ciguotoxic fishes that have been examined in the Pacific Ocean and the Caribbean (Legrand et al. 1992; Lewis and Sellin 1992; Tosteson et al.

1992; Tosteson et al. 1995; Pottier et al. 2002). Moreover, the presence of multiple, toxic dinoflagellates may also explain the diversity of medical symptoms found among individuals suffering from ciguatera poisoning (e.g., Tosteson 2004).

Blooms of cyanobacteria have been also linked to ciguatera-like symptoms (Habekost et al. 1955; Dawson et al. 1955; Hashimoto et al. 1976; Laurent et al. 2008; Yeeting 2009a). For example, Laurent et al. (2008) suggested that cyanobacteria from the genus *Hydrocoleum* was the major cause of ciguatera-like poisonings in *Tridacna* clams that were consumed in New Caledonia. Moreover, several authors have also suggested that *Lyngbya* is involved in fish poisonings (e.g., Habekost et al. 1955; Dawson et al. 1955; Hashimoto et al. 1976; Osborne et al. 2001). Therefore, even after several decades of research, we seem far from understanding ciguatera poisoning. It may become apparent that ciguatera poisoning is merely a generic effect that stems from multiple underlying causes.

FACTORS THAT INFLUENCE TOXIN PRODUCTION AND GROWTH OF CIGUATOXIC DINOFLAGELLATES

The considerable variability in toxin production by dinoflagellates has been an obstacle for predicting ciguatera-poisoning outbreaks. To date, no studies have found a correlation between the toxicity and the density of *G. toxicus*. For example, Gillespie et al. (1985) studied *G. toxicus* blooms along the Queensland coast of Australia but found no relationship with ciguatoxin concentrations. Some studies have suggested that toxic *G. toxicus* are genetically predetermined, and that toxic

and non-toxic clones can co-occur during a single bloom (e.g., Tosteson et al. 1989; Chinain et al. 1999). Litaker et al. (2010) found that toxicity was more variable among than within species of the genus *Gambierdiscus*.

There is also some evidence that dinoflagellate toxicity is caused by symbiotic bacteria (Tosteson et al. 1989; Rausch de Traubenberg and Lassus 1991; Doucette 1995; Gonzalez et al. 1995). For example, Ashton et al. (2003) examined the toxicity level of *Ostreopsis lenticularis* in association with their symbiotic bacterium, and found that under high temperatures, bacterial densities were reduced and *O. lenticularis* became toxic, but when *Ostreopsis* and associated bacteria were subjected to lower temperatures, the toxicity was reduced. Ashton et al. (2003) suggested that bacteria consumed toxins produced by *O. lenticularis* at low temperatures and consumed toxins for a short duration (10 – 22 days) under elevated temperatures, but when the elevated temperatures were prolonged the bacterial densities decreased and the toxicity increased.

The role that nutrient enrichment plays in promoting ciguatera poisoning remains inconclusive, largely because only a few field studies have examined ciguatoxic dinoflagellates alongside nutrient concentrations (Anderson et al. 2008). Still, Yasumoto et al. (1980), Carlson (1984), and Carlson and Tindall (1985) all suggested that nutrients were significantly correlated with *G. toxicus* densities, whereas a recent study by Parsons and Preskitt (2007) found no significant correlation between nutrients and *G. toxicus* at six sites in Hawaii. Some authors even take the extra step and suggest that ciguatera poisoning and high nutrient

concentrations are related. The most likely source of nutrients that trigger such outbreaks would be from the reef itself, particularly following major disturbances (Randall 1958; Cooper 1964; Bagnis 1994; see also Anderson et al. 2008). Although large physical disturbances such as cyclones do generate high-nutrient concentrations by re-suspending sediments on coral reefs (Furnas et al. 2005), past studies on disturbances have not shown conclusive evidence of a definitive link between high-nutrient concentrations and ciguatera-poisoning outbreaks.

Several studies have examined links between ciguatoxic dinoflagellates and irradiance. *In vitro* experiments have shown that *G. toxicus* grew most rapidly in 11% full sunlight (e.g., Guillard and Keller 1984), while Yasumoto et al. (1980) found that the growth of *G. toxicus* was inhibited at depths less than 0.5 m, where light was greater than 19% (6500 lux) of full sunlight. Such requirements explain the preference of *G. toxicus* for shade among host algae (e.g., Villareal and Morton 2002). However, other potentially toxic dinoflagellates (e.g., *Prorocentrum micans*) can successfully grow in up to 30% full sunlight (e.g., Brand and Guillard 1981). While the influence of irradiance on dinoflagellate growth and toxin production remain inconclusive, toxin production by *G. toxicus* may be simply a stress response to high irradiance (e.g., Bomber et al. 1988).

Salinity also influences the distribution and growth of *G. toxicus*. *In vitro* experiments found that *G. toxicus* grew best at salinities between 25 and 40 % (e.g., Bomber et al. 1988). High densities of dinoflagellates have been reported in high-salinity waters, and low densities have been reported near areas of freshwater

discharge (Yasumoto et al. 1980; Taylor 1985; Hokama and Yoshikawa-Ebesu 2001). Taylor and Gustavson (1986) found that ciguatera poisoning frequently occurred on offshore islands with high oceanic exchange, and was uncommon on reefs with high freshwater inputs discharging from large land masses. In addition, Anderson et al. (1983) suggested that higher salinity, a consequence of low rainfall, explained the prevalence of ciguatera poisoning on the leeward exposure of Hawaii. On the contrary, Carlson and Tindall (1985) found a positive correlation between rainfall (i.e., low salinity) and *G. toxicus* density, although this correlation may have been influenced by other factors such as terrestrial sediment runoff – because soil extracts have been found to enhance the growth of *G. toxicus* (Yasumoto et al. 1984; Durand-Clement 1987).

SPATIAL AND TEMPORAL DISTRIBUTION

Although hospital records are biased toward extreme ciguatera-poisoning cases, it is suggested that only around 10 – 20% of cases are reported (Lewis 1986; Dalzell 1991). Yet, such records have been instrumental in shaping our understanding of the severity and spatio-temporal distribution of the problem (e.g., Bagnis et al. 1979; Lewis 1986; Chateau-Degat et al. 2007). The incidence of ciguatera poisoning in the Pacific Ocean has been estimated to be two-fold higher than in the Caribbean (24 versus 12 per 10,000 population per year; Tester et al. 2010). The highest incidence of ciguatera poisoning on record has been reported from Gambier Islands, French Polynesia, where 790 cases per 10,000 population

per year were estimated in 2004 (Chinain et al. 2009). Reasons for the higher incidence of ciguatera poisoning in the eastern Pacific is unknown. However, Randall (1981) suggested that the east-west dichotomy in ciguatera poisoning may be the result of the basic biogeographic phenomenon of reduced species diversity in the east. Similarly, Lewis (1983) suggested that low species diversity gave ciguatoxic dinoflagellates a competitive advantage, particularly after disturbances. A contrasting argument is that one would expect a competitive advantage following disruption of a high-diversity system because of competition release.

The spatial distribution of ciguatera poisoning within regions, and even within islands, is highly variable. For example, the average cases of ciguatera poisoning (per 10,000 population per year) in 14 countries in the Caribbean ranged from 58.6 to 0.003 from 1996 to 2006 (Tester et al. 2010). In the Pacific Ocean, the cases of ciguatera poisoning ranged from 65.3 to 0.1, from 1973 to 1983 (Lewis 1986). At a local scale, some studies have found that the occurrence of ciguatera poisoning was higher on windward reefs than on leeward reefs (e.g., Kaly et al. 1991), whereas other studies found a higher occurrence on leeward reefs (e.g., Carlson 1984; Taylor 1985). Ciguatera poisoning also varies temporally at any given locality (Chateau-Degat et al. 2007; Chinain et al. 2009). For example, from 1973 to 1983, the Cook Islands were considered to have the lowest incidence of ciguatera poisoning in the Pacific Ocean (Lewis 1986). Yet, from 1993 to 2006, Rarotonga became a ciguatera ‘hot spot’, with an average incidence of 176 per

10,000 population per year, which was among the highest in the world (Rongo et al. 2009a).

Evidence exists for a lag between the peak densities of the seemingly causative agents (i.e., *G. toxicus*) and reef-fish toxicity. Kaly and Jones (1994) suggested a one-year lag period between peak densities of *G. toxicus* and toxicity levels in reef fishes, whereas Chateau-Degat et al. (2005) found a lag period of 16 – 20 months between peak densities of *G. toxicus* and toxins appearing in the human population. Bagnis et al. (1988) suggested that changes in reef fishes involved in ciguatera poisoning can indicate whether an outbreak of ciguatera poisoning is on the rise or whether it is waning. During the early stages of an outbreak in French Polynesia, poisonings were mainly caused by herbivorous fishes, followed by herbivorous and carnivorous fishes at the peak of the outbreak, but when the outbreak declined only carnivorous fishes were causing ciguatera poisoning (Bagnis et al. 1988). However, no follow-up study has been conducted to examine this hypothesis.

CLIMATE, REEF DISTURBANCES, AND CIGUATERA POISONING

High anomalous sea surface temperatures (SST), associated with the inter-annual cycle of the El Niño Southern Oscillation (ENSO), have been frequently cited as the major factor inducing ciguatera-poisoning outbreaks (Tosteson et al. 1988; Epstein et al. 1993; Hales et al. 1999; Chateau-Degat et al. 2005; Tester et al. 2010). These relationships can be defined within a ‘climate oscillation hypothesis.’

From 1973 to 1994, in seven Pacific islands, the cases of ciguatera poisoning were positively correlated with positive SST anomalies associated with ENSO (Hales et al. 1999). If this is general, and since sea surface temperatures are predicted to increase by 3°C in the next century (Hegerl et al. 2007), then we would expect to see an increase in the incidence of ciguatera poisoning (de Sylva 1994; see World Bank, 2000). By contrast, Llewellyn (2009) proposed that the predicted scenario of high SST may instead suppress the incidence of ciguatera poisoning in tropical regions.

There is a general consensus that factors that disturb reefs often lead to outbreaks of ciguatera poisoning. The ‘new surface hypothesis’ suggests that reef disturbances provide space for opportunistic macroalgae, the preferred substrate for ciguatoxic dinoflagellates, which in turn increases the risk of ciguatera poisoning (Randall 1958; Bagnis et al. 1980). Although the ‘new surface hypothesis’ has gained much support in the literature, most accounts are largely anecdotal. Reef damage from boat channel construction (Tebano 1984), boat anchorage and wrecks (Cooper 1964), cyclones (Randall 1958; Banner 1976; Rongo and van Woesik 2011), *Acanthaster planci* outbreaks (Bagnis et al. 1988), and coral bleaching events (Kohler and Kohler 1992; Bagnis et al. 1992a) have all been associated with increased density of *G. toxicus* and often lead to increased cases of ciguatera poisoning. Alternatively, some reef disturbances occur without any increased risk of ciguatera poisoning. For example, the risk of ciguatera poisoning did not increase from the blasting of a channel in Tuvalu (Kaly and Jones 1994) or an *A.*

planci outbreak in the 1970s in Rarotonga, Cook Islands (Rongo and van Woesik 2011). In addition, Lewis et al. (1986) found that the toxicity in herbivorous fishes and the density of *Gambierdiscus toxicus* did not increase three months after cyclone damage to Sudbury Reef on the Great Barrier Reef. Continued monitoring, which would have accounted for the lag period (up to 18 months; Bagnis 1969) between a disturbance and ciguatoxins appearing in the food web may have provided different results.

SOCIAL AND ECONOMIC IMPACTS

Beyond health issues, ciguatera poisoning has also had socioeconomic impacts, including: 1) loss of a food source, 2) loss of labor productivity (Hajkowicz 2006), 3) increased health-related costs (Lewis 1986; Hajkowicz 2006), 4) loss of tourism (Lewis 1992b), 5) loss of foreign-exchange opportunities through fish exports (Lewis 1983; Yeeting 2009b), and 6) depopulation (Banner and Helfrich 1964; Cooper 1964; Rongo et al. 2009a). However, research into the socioeconomic impacts of ciguatera poisoning, and other harmful algal blooms, on human health are still in the early stages, and there is an urgent need to quantify these impacts (Kite-Powell et al. 2008).

Ciguatera poisoning has caused impacts to the economy of both developed and developing nations. For example, in U.S. tropical jurisdictions alone, the impact of ciguatera poisoning has been estimated at USD \$19 million annually (Hoagland et al. 2002). In addition, the impacts extend beyond the ciguatera-

affected regions. For example, in Canada, medical costs associated with an average of 300 cases per year between the 1960s and the 1980s due to ciguatera poisoning from tourism and food importation was estimated around CAD \$2.7 million (Todd 1985). Bagnis et al. (1992b) estimated that the economic loss associated with banned reef-fish sales and lost-labor productivity in Tahiti, French Polynesia, was over US \$2 million per year. In Kiribati, Yeeting (2009b) estimated that the closure of export fisheries to Hong Kong resulted in an annual revenue loss of AUD \$250,000 (~AUD \$8,000 per fishermen). Yet, the economic impact of ciguatera poisoning in Pacific-island countries is still poorly understood, largely because limited financial resources have been allocated to examine the problem (Dalzell 1993). For this reason, Dalzell (1993) suggested that management of ciguatera poisoning should be directed at industries that generate revenue, such as tourism and export fisheries. It is critical that socioeconomic studies are conducted on ciguatera poisoning to determine the extent of economic loss, and what actions may mitigate the problem (Lewis 1992b; Hoagland and Scatasta 2006).

Although extensive ciguatera poisoning leads to dietary shifts (Lewis 1983), the nature of the alternative protein source will depend on the economic status of the islands involved. More-developed communities would, theoretically, undergo dietary shifts more easily than less-developed communities (Lewis 1992b; Chateau-Degat et al. 2007). Developed communities, with ties to developed countries, tend to show shifts toward frozen meats such as chicken, beef, and lamb. By contrast, less developed communities with limited capacity to freeze food tend to shift their

diets toward canned foods, such as corned beef, with high fat content (Lewis 1983; Pinca et al. 2007). The shift away from a fresh-fish diet appears to have also contributed to the increased prevalence of non-communicable diseases among Pacific-island populations (Lewis and Ruff 1993; Li et al. 1994; Ulijaszek 2003).

Ciguatera poisoning has also caused the movement of people away from affected areas. For example, Banner and Helfrich (1964) noted that ciguatera poisoning caused people to move from one coast to another in Hiva Oa in the Marquesas, French Polynesia in the 1950s. Similarly in the 1950s, residents on Sydney Island in the Phoenix Islands were evacuated due partly to ciguatera poisoning rendering marine resources unusable (Cooper 1964). In the Cook Islands, ciguatera poisoning contributed to the migration of 18% of the resident population to New Zealand and Australia in the 1990s (Rongo et al. 2009a).

HISTORICAL ACCOUNTS

Historical accounts of ciguatera poisoning were based on symptoms described by early explorers after the consumption of reef fishes in tropical regions. For example, reports from the Pacific date back to 1606, where the Spanish explorer Pedro de Quiros reported fish poisoning in the New Hebrides (Dalrymple 1770). In 1774 from the same region, Captain James Cook recorded similar symptoms after his crew consumed a type of red snapper, while moored in Vanuatu (Anderson 1776; Doherty 2005). In the Caribbean, the earliest account of ciguatera poisoning was recorded in 1511 (Martyr 1555), although Columbus may have

encountered ciguatera poisoning earlier in 1492 (Deshpande 2002). In the Indian Ocean, the first account of ciguatera poisoning was reported by Harmansen in 1601 (Jones 1956). The presence of ciguatera poisoning was implied in archaeological records. For example, Kirch and Yen (1982) noted the abrupt disappearance of the moray eel around AD 1700 from midden records in Tikopia, Solomon Islands. The subsequent ‘taboo’ placed on this species may have been the direct result of ciguatera poisoning. Whether ciguatera poisoning had any impact on island inhabitants through the late Holocene period between AD 1000 and 1450 is unclear, but will be explored in the present study.

RESEARCH QUESTIONS

The ocean and its resources have sustained island nations for millennia, yet an exclusive reliance on fishes can be disastrous for an island population when those resources become inedible, particularly from ciguatera poisoning that can severely debilitate individuals after consuming intoxicated reef fishes. Indeed, the impact of ciguatera poisoning extends beyond the individual affected, and includes socioeconomic impacts. In the southern Cook Islands (Fig. 1), where ciguatera poisoning has been problematic for around 20 years, I conducted a study to further our understanding of the problem by addressing the following questions: 1) Can we predict the spatial extent of ciguatera poisoning? 2) Can we use the concept of shifts in ciguatera-poisoning vectors to forecast the intensity of upcoming ciguatera-poisoning outbreaks? 3) Is ciguatera poisoning in the southern Cook

Islands linked to major climate cycles? 4) Is ciguatera poisoning influenced by reef disturbances, reef state, and herbivorous fish density? 5) What are the socioeconomic impacts of ciguatera poisoning? and 6) Did ciguatera poisoning occur in the past, and if so, did ciguatera poisoning prompt the late Holocene biogeographic distribution of humans across the Pacific Ocean between AD 1000 and 1450?

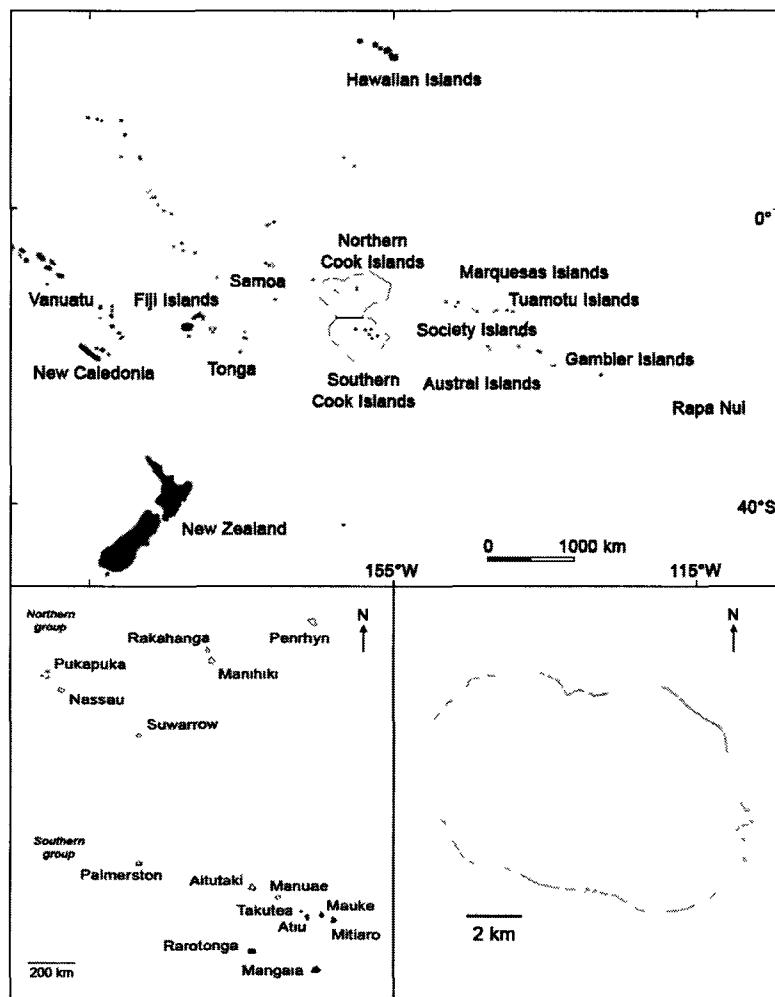


Figure 1. Map of the Pacific with the Cook Islands' Exclusive Economic Zone delineated. *Bottom left:* Cook Islands. *Bottom right:* Rarotonga ($21^{\circ}14'15''S$ $159^{\circ}46'48''W$).

CHAPTER II

THE SPATIAL AND TEMPORAL DISTRIBUTION OF CIGUATERA POISONING IN RAROTONGA, SOUTHERN COOK ISLANDS

INTRODUCTION

Ciguatera poisoning occurs in humans that have consumed reef fishes or invertebrates that have accumulated toxins produced by dinoflagellates (e.g., *Gambierdiscus toxicus*), which are typically epiphytic to macroalgae (see Cruz-Rivera and Villareal 2006). The symptoms include acute gastrointestinal, neurological, cardiovascular problems, and in extreme cases can lead to death. The global economy has increased the extent of ciguatera poisoning beyond the geographic range of natal fishes. For example, fishes caught in the tropical Pacific Ocean are frequently exported to Europe, Asia, and the USA (see Van Dolah 2000; Wong et al. 2005; Dickey and Plakas 2010). Therefore, ciguatera poisoning affects at least 50,000 – 500,000 people per year worldwide (Fleming et al. 1998).

Ciguatoxins bioaccumulate as they pass through the marine food web, consequently concentrating in large, carnivorous fishes (Lewis 2006). While *Gambierdiscus toxicus* has been implicated as the main dinoflagellate that causes ciguatera poisoning, other dinoflagellates have been also implicated (Faust 1995; Holmes 1998; Parsons and Preskitt 2007, Rhodes et al. 2009). In addition, some studies have linked outbreaks of cyanobacteria to ciguatera-poisoning-like symptoms (Habekost et al. 1955; Dawson et al. 1955; Hashimoto et al. 1976;

Laurent et al. 2008). Still, definitive links between ciguatera poisoning and the densities of microscopic life is tenuous, making predictions difficult.

To date, much of our understanding of ciguatera poisoning and its social impact stem from hospital records (e.g., Bagnis et al. 1979; Lewis 1986; Chateau-Degat et al. 2007). Yet, such results appear to underestimate the severity of ciguatera poisoning because only around 20% of cases are reported (Lewis 1986; Dalzell 1991), and therefore it has been difficult to comprehend the impact on societies, particularly on islands communities. Determining the true incidence of ciguatera poisoning will provide managers some indication of that economic impact so that the appropriate scale of action to mitigate the problem can be taken (Hoagland and Scatasta 2006).

The distribution of ciguatera poisoning is spatially variable (see Lewis 2006). For example, several studies from the Pacific showed that *G. toxicus* thrives in areas most exposed to oceanic waters, notably in high energy environments such as turbulent channel areas or on the windward side of islands (Yasumoto et al. 1979, 1980; Kaly and Jones 1994), while in Caribbean, Carlson (1984) showed that *G. toxicus* prefer protected lagoons and other inshore stations compared to reef stations. Temporal variations in ciguatera poisoning are also evident. For example, from the 1960s to the 1980s, ciguatera poisoning was problematic in the western Pacific (e.g., Tokelau, Samoa, Fiji; Bagnis et al. 1985), but declined in recent years, while during the same period, Rarotonga in the southern Cook Islands was an

anomaly with very low incidence (Lewis 1979), but cases have increased into the 1990s.

Understanding the spatial and temporal distribution of ciguatera poisoning is important to assist in management efforts of this problem, especially in Pacific island nations where fish is the main source of protein for inhabitants. I examined these distributions in Rarotonga (Fig. 2) in the southern Cook Islands (see Fig. 1, p. 16), an ideal location because it is currently a high-risk area for ciguatera poisoning. The average incidence rate of ciguatera poisoning in Rarotonga was estimated at 176 per 10,000 population per year between 1993 and 2006 (Rongo et al. 2009a). Although, Hajkowicz (2006) reported that ciguatera poisoning resulted in the avoidance of reef fishes in the diet of 71% of the residents, the fact that many residents continue to eat reef fishes despite the risk is important to further our understanding of the problem.

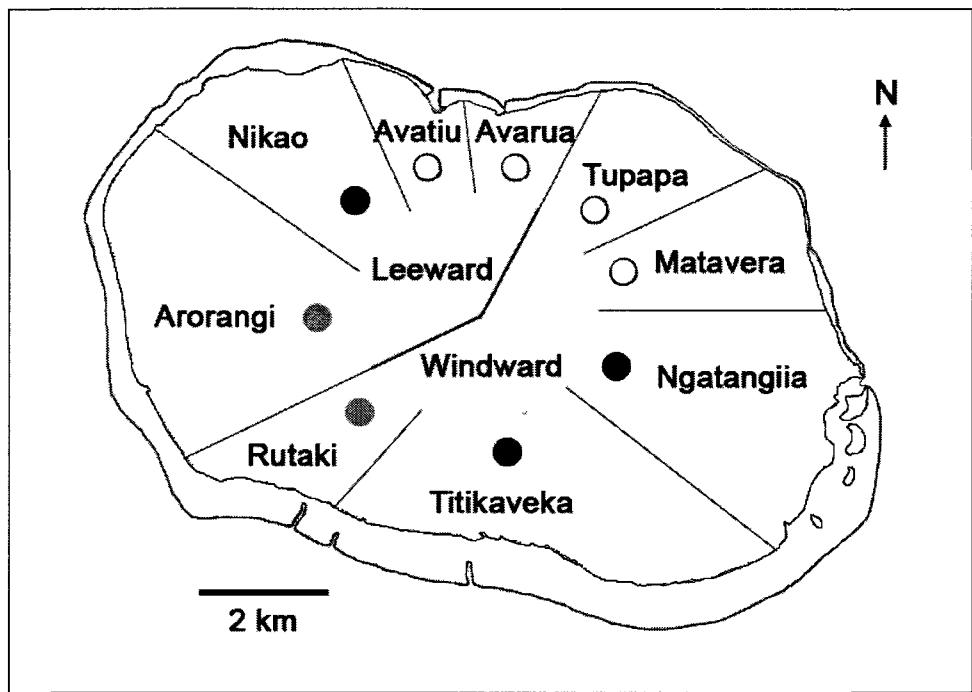


Figure 2. Rarotonga ($21^{\circ}14'15''S$ $159^{\circ}46'48''W$) indicating villages in windward and leeward exposures. Villages with wide lagoons (black circles), intermediate lagoons (grey circles), and narrow lagoons (open circles) indicated.

MATERIALS AND METHODS

QUESTIONNAIRE SURVEY

A questionnaire survey (Appendix A) was conducted from December 2008 to January 2010. The survey targeted individuals aged 15 and over because adults generally avoid feeding reef fishes to children. Using the total resident population of 2006 (10,226), 626 individuals were interviewed at random (i.e., over 6%; whereas 5% seems sufficient for a representative sample according to Zar 1999). Information collected included: (i) date of poisoning, (ii) identity of fish or invertebrate implicated in poisoning (to the lowest taxonomic level possible), (iii) location where each fish was caught, (iv) symptoms experienced, (v) whether ciguatera poisoning was reported to health officials, and (vi) current fish consumption preference (including reef fishes considered ‘safe’).

Hospital records on cases of ciguatera poisoning were obtained from the Cook Islands Ministry of Health, which is responsible for compiling information from the only public hospital in Rarotonga. While the reporting of cases of ciguatera poisoning to the public health database began in the Cook Islands in 1991, it was not until 1993 that cases were separated by island. Therefore, cases reported before 1994 were excluded from the analyses because of reporting inconsistencies in the early stages of the transition (T. Iorangi, pers. comm.). Although information on ciguatera poisoning collected at the Rarotonga hospital only recorded the year of poisoning, and the island where it was experienced, for

the purposes of this study, this type of information was sufficient to examine the timing of ciguatera-poisoning outbreaks.

Although most people in Rarotonga are familiar with the clinical symptoms of ciguatera poisoning (i.e., gastrointestinal and neurological), in order to avoid recording other types of food poisoning, unusual symptoms were verified with those described in the literature. The symptomatic effects of ciguatera poisoning usually last from 3 to 6 months, and the consumption of fishes, regardless of type, within this period can trigger the recurrence of symptoms (see Baden et al. 1995). To avoid using recurring cases of individuals with multiple poisonings, incidents were only recorded if they were at least 6 months apart.

DATA PREPARATION

Prior to any statistical analysis, sites where fishes were caught were categorized *a priori* as either (1) windward or leeward, and (2) in accordance with the width of the lagoon (i.e., narrow, intermediate, and wide) (see Fig. 2). The exposure of each site was based on the work of Thompson (1986). Windward villages were Tupapa, Matavera, Ngatangiia, Titikaveka, and Rutaki (which includes Kavera), and leeward villages were Arorangi, Nikao, Avatiu (which includes Panama), and Avarua (see Fig. 2). Using Google Earth, lagoons were categorized as narrow if ≤ 200 m, intermediate if > 200 m and ≤ 400 m, and wide if > 400 m.

Hospital and survey cases of ciguatera poisoning in Rarotonga were separated into three sampling periods based on the state of the reef prior to analysis: (i) 1989 – 2000 (although detailed reef surveys in Rarotonga commenced in 1994, prior years were included in this sampling period to encompass cases of ciguatera poisoning identified by the questionnaire survey), (ii) 2001 – 2005, and (iii) 2006 – 2009. Between 1989 and 2000, the shift towards a coral-depauperate state was largely the result of an outbreak by the coral-eating *Acanthaster planci* between 1995/96 and 2001 (Lyon 2000), and to a lesser extent, bleaching events in 1991 and 1994 (Miller et al. 1994; Goreau and Hayes 1995). The average hard-coral cover on the fore reef was reported to be around 30% in 2000 (Lyon 2000). Between 2001 and 2005, most coral reefs around Rarotonga supported less than 5% hard coral cover; *A. planci* density decreased dramatically by 2003 (Lyon 2003). In 2006, the average coral cover was around 1% on the fore reef (Rongo et al. 2006). In 2009, corals showed signs of recovery, with the fore reefs supporting on average 5% coral cover (Rongo et al. 2009b). Therefore, from 1989 to 2000 was defined as the ‘*Acanthaster planci*’ period, from 2001 to 2005 was defined as the ‘transitional’ period, and from 2006 to 2009 was defined as the ‘recovery’ period.

The incidence rate of ciguatera poisoning was calculated annually from hospital data, and the size of the resident population of Rarotonga was taken from census data (conducted every 5 years). Census populations of 1991 (10,886), 1996 (10,337), 2001 (9,424), and 2006 (10,226) were used in calculations of incidence rates for the years 1994 – 1995, 1996 – 2000, 2001 – 2005, and 2006 – 2010

respectively. Incidence rates were reported per 10,000 population per year (see Tester et al. 2010).

DATA ANALYSIS

Only reef fish and marine invertebrate species involved in ten or more cases of ciguatera poisoning were included in the analysis. The data were square-root transformed to reduce the influence of sites and fishes with the highest incidents. A similarity matrix was constructed using the Bray-Curtis similarity coefficient. The spatial distribution of cases of ciguatera poisoning was examined using Multi-Dimensional Scaling (MDS). A Principal Component Analysis (PCA) was used to examine whether reef fishes and marine invertebrates, implicated in ciguatera poisoning, differed between sampling periods.

An Analysis of Similarity (ANOSIM) was used to test for differences between exposure (windward and leeward) and between lagoon width (narrow, intermediate, and wide). The results of ANOSIM generated R-values that provided a confidence limit on the degree of similarity: 0 (similar) to 1 (different) at a 0.05 significance level (see Clarke and Warwick 1994). All multivariate analyses were conducted using *Primer 6®*.

A Chi-square test using *Statistica 6®* was conducted to examine the relationship between the observed and expected average incidence of ciguatera poisoning for the three periods examined (1994 – 2000, 2001 – 2005, and 2006 – 2010).

RESULTS

There were 626 Cook Islanders interviewed, of which 326 (52%) had experienced ciguatera poisoning at least once in their lifetime. Because 42% of these individuals had multiple poisonings, 509 cases were reported and summarized in Table 1. Overall, about 88% of the cases of ciguatera poisoning were from Rarotonga, whereas 12% were from the outer islands (primarily Aitutaki and Atiu; I note that incidents from the outer islands and those with no date were excluded). Two periods of ciguatera poisoning were identified: (1) the early 1970s, and (2) from 1989 onwards (Fig. 3). I estimated that 34% of the cases of ciguatera poisoning were reported to health officials. I also estimated that only 4% of cases were treated at private clinics (listed as unreported in this survey because they were not reported to the public health database). From 1994 to 2010, the annual incidence rate of ciguatera poisoning varied from 76 to 360 per 10,000 population per year, which equates to an actual incidence rate of 224 to 1,058 per 10,000 population per year (calculated from the 34% reporting to hospitals that was determined by the survey; see Table 1). I also estimated that the average incidence rate of ciguatera poisoning was significantly higher from 2001 to 2005 than in other periods ($\chi^2_2 = 28.7, p < 0.001$). The actual incidence rate for the three periods was estimated at 432, 670, and 404 per 10,000 population per year, respectively.

Table 1. Cases of ciguatera poisoning on Rarotonga derived from the questionnaire survey conducted between December 2008 and January 2010. Annual incidence rates (rounded to nearest whole numbers) were calculated for each period using hospital cases of ciguatera poisoning requested from the Cook Islands Ministry of Health, and the total resident population in census years: 1994 – 1995 (10,886 in 1991), 1996 – 2000 (10,337 in 1996), 2001 – 2005 (9,424 in 2001), and 2006 – 2010 (10,226 in 2006). Actual incidence rates were calculated based on the 34% reporting determined from the survey. Data in brackets indicates total hospital cases of ciguatera poisoning from 1994 to 2010.

Year	Survey cases	Annual incidence per 10,000 population per year	Actual annual incidence per 10,000 population per year	Period examined	Average incidence per 10,000 population per year	Actual average incidence per 10,000 population per year
1971	1					
1972	3					
1973	0					
1974	1					
1989	3					
1990	1					
1991	5					
1992	9					
1993	9					
1994	19	119	351			
1995	14	200	589			
1996	20	244	717			
1997	15	110	324			
1998	14	138	407			
1999	21	112	330			
2000	31	105	307	1994 – 2000	147 ± 20 SE	432 ± 60 SE
2001	31	132	387			
2002	24	128	378			
2003	20	179	527			
2004	28	360	1,058			
2005	37	341	1,002	2001 – 2005	228 ± 51 SE	670 ± 149 SE
2006	54	168	495			
2007	51	180	529			
2008	65	140	411			
2009	33	123	362			
2010		76	224	2006 – 2010	138 ± 18 SE	404 ± 54 SE
Total	509	[2,858]				

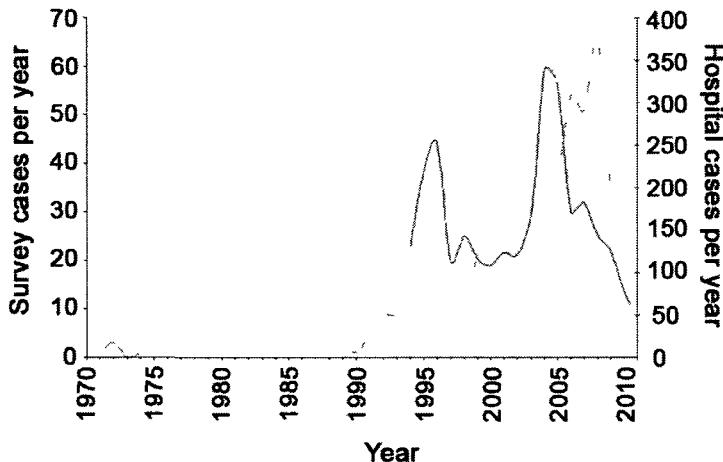


Figure 3. Cases of ciguatera poisoning from Rarotonga's hospital data (thick line) and the questionnaire survey (thin line). Shaded regions indicate the two ciguatera-poisoning events identified by the survey (in the early 1970s, and from 1989 onwards).

THE SPATIAL DISTRIBUTION OF FISHES AND INVERTEBRATES INVOLVED IN CIGUATERA POISONING

From this survey, a total of 48 fish species, representing 24 families, were implicated in ciguatera poisoning (Table 2). Forty-eight percent of the cases were reported from individuals eating the fishes *Scarus psittacus*, *Caranx melampygus*, *Ctenochaetus striatus*, *Crenimugil crenilabis*, *Chlorurus frontalis*, *Promethichthys prometheus*, *Mulloidess flavolineatus*, and *Epinephelus tauvina*. The giant clam (*Tridacna maxima*) was the most common invertebrate implicated in ciguatera poisoning, all of which were collected from the Titikaveka lagoon area. The survey also identified targeted reef fishes considered safe for consumption by locals; they included *Cirrhitus pinnulatus*, *Epinephelus hexagonatus*, *Kyphosus spp.*, *Myripristis spp.*, *Naso lituratus*, *Naso unicornis*, *Priacanthus hamrur*, *Siganus argenteus*, and *Siganus spinus* (Table 3).

Table 2. Reef fishes and marine invertebrates involved in ciguatera poisoning that were reported in the questionnaire survey conducted between December 2008 and January 2010. Species are ranked from highest to lowest in incidents of poisoning (*n*). Although 678 incidents were recorded in the survey, only 526 incidents of fish and invertebrate poisonings were reported because of identification difficulties involved with some fish species.

Scientific name	Common name	Local name	<i>n</i>
REEF FISHES			
<i>Scarus psittacus</i>	Palenose parrotfish	Pakati	72
<i>Caranx melampygus</i>	Bluefin trevally	Tīti ara	62
<i>Ctenochaetus striatus</i>	Striped bristletooth	Maito	56
<i>Crenimugil crenilabis</i>	Frungelip mullet	Kanae	43
<i>Chlorurus frontalis</i>	Tan-faced parrotfish	U u	32
<i>Promethichthys prometheus</i>	Snake mackerel	Mangā	31
<i>Mulloidess flavolineatus</i>	Yellowstripe goatfish	Vete	27
<i>Epinephelus tauvina</i>	Greasy grouper	Pātuki taraava	26
<i>Gymnothorax javanicus</i>	Giant moray	A a pata	17
<i>Lutjanus fulvus</i>	Flametail snapper	Tangau	14
<i>Cephalophalus argus</i>	Peacock grouper	Pātuki roi	13
<i>Epinephelus fasciatus</i>	Blacktip grouper	Ātea	10
<i>Naso unicornis</i>	Bluespine unicornfish	Ume	10
<i>Sarcocentron spp</i>	Squirrelfish	Kūtā	10
<i>Caranx ignobilis</i>	Giant trevally	Urua	8
<i>Lutjanus monostigma</i>	Onespot snapper	Kiriva	8
<i>Parapeneus spp</i>	Goatfish	Ka'uru	5
<i>Caranx lugubris</i>	Black trevally	Ru'i	4
<i>Variola louti</i>	Lyretail grouper	Oka	4
<i>Chaetodon auriga</i>	Threadfin butterflyfish	Taputapu	3
<i>Cheilinus undulatus</i>	Humphead wrasse, Napoleonfish	Maratea	3
<i>Fistularia commersonii</i>	Smooth cornetfish	Tātāvere	3
<i>Kyphosus spp</i>	Rudderfish	Pipi	3
<i>Lethrinus xanthochilus</i>	Yellowlip emperor	Īroa	3
<i>Monotaxis grandoculus</i>	Bigeye emperor	Mu	3
<i>Rhincanthus aculeatus</i>	Picasso triggerfish	Kōkiri	3
<i>Sargocentron spiniferum</i>	Sabre squirrelfish	Tarakī	3
<i>Siganus argenteus</i>	Forktail rabbitfish	Mōrava	3
<i>Scarus altipinnis</i>	Filament-finned parrotfish	Aumaori	3
<i>Acanthurus triostegus</i>	Convict surgeonfish	Manimi	2
<i>Caranx sexfasciatus</i>	Bigeye trevally	Kōmuri	2
<i>Epinephelus merra</i>	Dwarf spotted grouper	Pātuki marau	2
<i>Selar crumenophthalmus</i>	Bigeye scad	Ature	2
<i>Sphyraena spp</i>	Barracuda	Ono	2
<i>Symanceia verrucosa</i>	Stonefish	No'u	2
<i>Triakodon obesus</i>	Whitetip reef shark	Mangō maru	2
<i>Tylosurus crocodilis crocodilis</i>	Crocodile needlefish	Pāpā	2
<i>Acanthurus xanthopterus</i>	Yellowfin surgeonfish	Parangi	1
<i>Chaenomugil leuciscus</i>	Acute-jawed mullet	Ka'a	1
<i>Cheilio inermis</i>	Cigar wrasse	Kavakava	1
<i>Diodon hystrix</i>	Spot-fin porcupinefish	Tōtara	1
<i>Epinephelus hexagonatus</i>	Hexagon grouper	Pātuki paru	1
<i>Gerres spp</i>	Mojarra	A'ore	1
<i>Gymnosarda unicolor</i>	Dogtooth tuna	Varu	1
<i>Gymnothorax undulatus</i>	Undulated moray	Matakiva	1
<i>Hippocampus longiceps</i>	Pacific longnose parrotfish	Māmāringa	1

Table 2. Continued

Scientific name	Common name	Local name	N
<i>Liza vaigiensis</i>	Yellowtail mullet	Avake	1
<i>Lutjanus bohar</i>	Twinspot snapper, red snapper	Angamea	1
INVERTEBRATES			
<i>Tridacna maxima</i>	Giant clam	Pā ua	12
<i>Octopus scyanea</i>	Big blue octopus	Eke	2
<i>Percnon</i> spp	Nimble spray crab	Pāpaka akau	2
<i>Dendropoma maxima</i>	Large worm shell	Ungakoa	1

Table 3. Fishes currently considered edible (from local knowledge) in Rarotonga. Information obtained from survey data, with most targeted fishes indicated ✓.

Scientific name	Common name	Local name	Targeted
<i>Acanthurus achilles</i>	Achilles tang	Iku toto	
<i>Acanthurus guttatus</i>	Whitespotted surgeonfish	Api	
<i>Acanthurus leucopareius</i>	Whitebar surgeonfish	Maito	
<i>Acanthurus triostegus</i>	Convict surgeonfish	Manini	
<i>Carangoides orthogrammus</i>	Thicklip trevally	Pava	
<i>Caranx ignobilis</i>	Giant trevally	Kōkōkino (juvenile)	
<i>Caranx melampygus</i>	Bluefin trevally	Kōkōkino (juvenile)	
<i>Chaenomugil leuciscus</i>	Acute-jawed mullet	Aua (juvenile)	
<i>Chlororus frontalis</i>	Tanface parrotfish	Pakati/U u	
<i>Cirrhitus pinnulatus</i>	Stocky hawkfish	Pātuki toka	✓
<i>Epmephalus hexagonatus</i>	Hexagon grouper	Pātuki paru	✓
<i>Kyphosus</i> spp	Rudderfish	Pipi	✓
<i>Mulloidess flavolmeatus</i>	Yellowstripe goatfish	Kōma (juvenile)	
<i>Mulloidess vanicolensis</i>	Yellowfin goatfish	Takua (juvenile)	
<i>Myripristis</i> spp	Soldierfish	Kū	✓
<i>Naso lituratus</i>	Orangespine unicornfish	Marpo	✓
<i>Naso unicornis</i>	Bluespine unicornfish	Ume	✓
<i>Priacanthus hamrur</i>	Crescent-tail bigeye	Kū pā	✓
<i>Siganus argenteus</i>	Forktail rabbitfish	Mōrava	✓
<i>Siganus spinus</i>	Scribbled rabbitfish	Maemae	✓

There was no significant difference in the number of cases of ciguatera poisoning between windward and leeward lagoons. By contrast, narrow lagoons (i.e., Avatiu, Avarua, Tupapa, and Matavera) showed significantly fewer cases (Fig. 4; Table 4), whereas the widest lagoons (i.e., Ngatangiia, Titikaveka, and Nikao) showed the most cases of ciguatera poisoning (ANOSIM, $R = 0.796$, $p = 0.029$). However, narrow and intermediate lagoons ($R = 0.429$, $p = 0.267$), and

intermediate and wide lagoons ($R = 0.333, p = 0.200$) showed no significant differences in the number of ciguatera-poisoning cases.

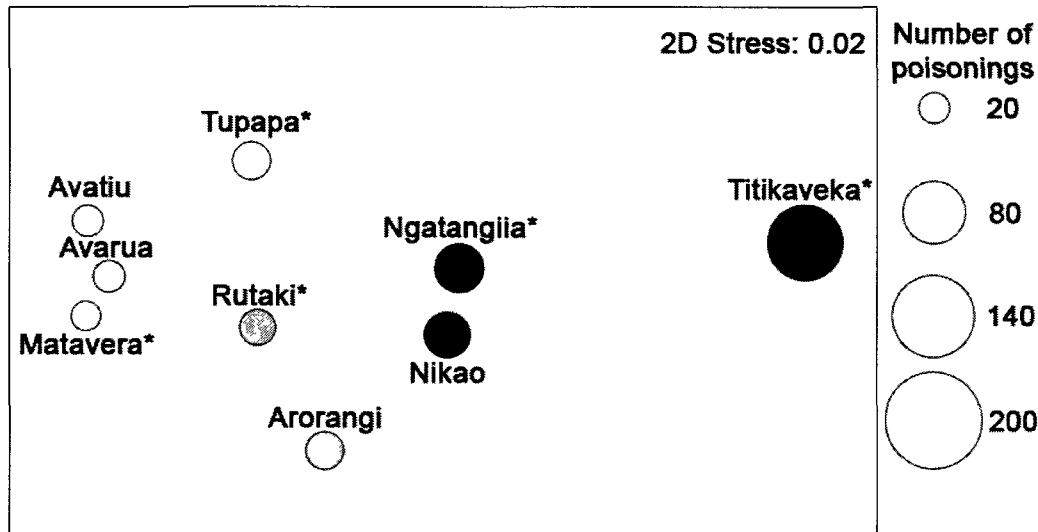


Figure 4. An ordination of sites using total cases of ciguatera poisoning that was derived from the questionnaire survey conducted between December 2008 and January 2010. Sites with narrow lagoons (open circles), intermediate lagoons (grey circles), and wide lagoons (black circles) were examined. Asterisk (*) indicates windward sites. The size of the bubbles reflects the relative cases of ciguatera poisoning.

Table 4. Analysis of Similarity pair-wise test between exposures (leeward vs. windward) and among the three categories of lagoon widths (narrow, intermediate, and wide) in Rarotonga. Asterisk (*) indicates significant difference at $p < 0.05$.

Exposure & Lagoon width	R-Statistic value	<i>p</i> -value
<i>Exposure</i>		
Leeward and Windward	0.175	0.889
<i>Lagoon width</i>		
Narrow and Wide	0.796	0.029*
Narrow and Intermediate	0.429	0.267
Wide and Intermediate	0.333	0.200

VECTOR SHIFTS OF CIGUATERA POISONING

Ordination analyses, using PCA, clearly separated the three periods examined (Fig. 5). Vector plots suggested that herbivore poisonings, primarily from *Ctenochaetus* spp. and to a lesser extent *Naso unicornis*, were most common between 1989 and 2000. Between 2001 and 2005, poisonings by *Epinephelus fasciatus* were common, but most interesting were the increased poisonings by soft-bottom benthic invertivores (i.e., *Mulloidess flavolineatus* and *Crenimugil crenilabis*) and filter feeders (i.e., *Tridacna maxima*) during this period (Table 5). Between 2006 and 2009, carnivorous fishes (i.e., *Promethichthys prometheus*, *Gymnothorax* spp., *Caranx melampygus*, and *Epinephelus tauvina*) and herbivorous fishes (i.e., *Scarus psittacus* and *Chlorurus frontalis*) caused most poisonings.

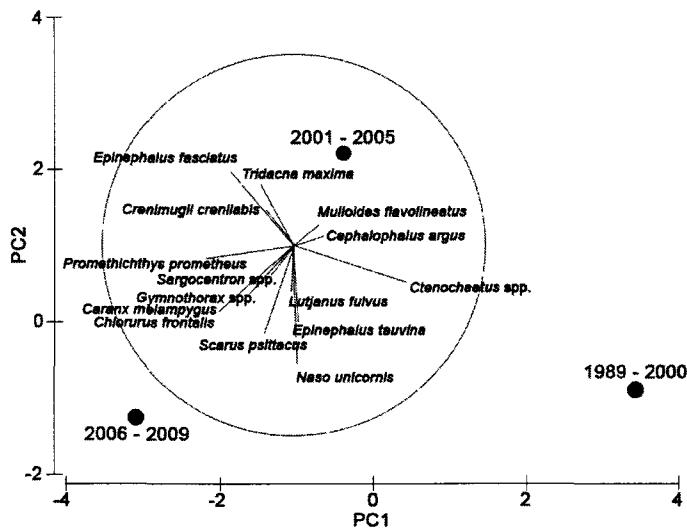


Figure 5. Principal Component Analysis of reef fish and marine invertebrate species involved in ten or more cases of ciguatera poisoning in Rarotonga plotted in relation to the three periods (1989 – 2000, 2001 – 2005, and 2006 – 2009).

Table 5. Eigenvalues and eigenvectors of Principle Component Analysis of ciguatoxic reef fishes and marine invertebrates in Rarotonga for the three periods examined (1989 – 2000, 2001 – 2005, and 2006 – 2009).

Eigenvalues		Eigenvalues	% Variation	Cum. %Variation
PC				
1		10.8	68.1	68.1
2		5.06	31.9	100
Eigenvectors				
SPECIES		PC1	PC2	
<i>Sargocentron</i> spp.		-0.117	-0.124	
<i>Naso unicornis</i>		0.002	-0.546	
<i>Epinephelus fasciatus</i>		-0.329	0.324	
<i>Tridacna maxima</i>		-0.172	0.270	
<i>Cephalophalus argus</i>		0.155	0.041	
<i>Lutjanus fulvus</i>		0.009	-0.129	
<i>Gymnothorax</i> spp.		-0.015	-0.213	
<i>Epinephelus tauvina</i>		0.024	-0.336	
<i>Mulloidess flavolineatus</i>		0.126	0.091	
<i>Promethichthys prometheus</i>		-0.464	-0.055	
<i>Chlorurus frontalis</i>		-0.391	-0.290	
<i>Crenimugil crenilabis</i>		-0.144	0.155	
<i>Ctenochaetus</i> spp.		0.587	-0.158	
<i>Caranx melampygus</i>		-0.231	-0.209	
<i>Scarus psittacus</i>		-0.148	-0.380	

DISCUSSION

Ciguatera poisoning has been problematic in Rarotonga since the late 1980s, and according to my survey a minor ciguatera-poisoning event also occurred in the early 1970s, which eluded previous studies (e.g., Lewis 1979, 1986). Consequently, I estimated that 52% (~5,318) of residents have experienced ciguatera poisoning at least once in their lifetime. Such estimates are considerably higher than the 1.8 – 2.5% reported from two Pacific island communities in Australia (Gillespie et al. 1985). Such differences may reflect island populations relying more on marine resources than their Australian counterparts. I also note that 34% of cases of ciguatera poisoning were reported to health officials, which was higher than the 20% generally assumed in the literature (e.g., Lewis 1986; Dalzell 1991; Chinain et al. 2009). Yet, over 80% of individuals surveyed in 2010 were consuming reef fish in Rarotonga, contrary to Hajkowicz's (2006) findings who reported 71% of the human population avoided reef fishes during a peak period of ciguatera poisoning. However, 96% of these individuals were selective in their consumption habits, and were particularly cautious of fishing locality (i.e., the Titikaveka area is generally avoided). In fact, 22% of these individuals only consumed reef fishes imported from islands in the northern Cook Islands (i.e., Manihiki and Penryhn), Palmerston in the southern group, and to a lesser extent from other islands in the southern group, (i.e., Mangaia, Mauke, and Mitiaro) that were thought to be largely free of ciguatera.

The survey indicated that in 2010, 75% of the resident population of Rarotonga was consuming more pelagic species than previously, even though the pelagic fishes were more expensive than reef fishes. This shift was also reported by Solomona et al. (2009) in 2001. I also estimated that 14% of individuals surveyed limited themselves to pelagic species, and only 1% completely excluded reef fish from their diet. Residents, especially those whom have had multiple poisonings over their lifetime, have accepted ciguatera poisoning as part of a reef-fish diet, and continue to consume reef fishes despite the risk. This risk-taking attitude is common among island populations, as was noted some 40 years ago in the Gilbert Islands (Cooper 1964), and recently on Raivavae of the Austral Islands in French Polynesia (Chinain et al. 2009).

Between 1973 and 1983, the Cook Islands were considered a ciguatera anomaly, with a very low reported incidence of ciguatera (Lewis 1986). However, over the past 18 years, Rarotonga has been a hotspot for ciguatera poisoning, alongside the neighboring islands of French Polynesia. For example, the average incidence rate from 1996 to 2006 in 14 countries of the Caribbean ranged from 58.6 to 0.003 per 10,000 population per year (see Tester et al. 2010), compared with 183 per 10,000 population per year in Rarotonga for the same period. On Raivavae, the average incidence from 2007 to 2008 was 140 per 10,000 population per year (Chinain et al. 2009), while my estimate for Rarotonga, for the same period, was 160 per 10,000 population per year. However, I noted that the actual annual incidence from 1994 to 2010 in Rarotonga ranged from 224 to 1,058 per 10,000

population per year, which suggests that ciguatera poisoning can affect up to 11% of the population in a year.

SPATIAL DISTRIBUTION OF CIGUATERA POISONING

Most cases of ciguatera poisoning were recorded from the Titikaveka section of the Rarotonga lagoon, yet my results showed that the degree of reef exposure did not contribute to the incidents of poisoning. Other studies examining *G. toxicus* densities with regard to wind and wave exposure have shown mixed results. Some studies found higher densities on windward reefs (e.g., Kaly et al. 1991), whereas other studies found higher densities on leeward reefs (e.g., Carlson 1984; Taylor 1985). By contrast, the width of the lagoon played a significant role in my study. More incidents of ciguatera poisoning were reported from individuals that consumed fishes caught in wide lagoons. It could be argued that narrow lagoons support fewer fishes in general, and therefore the probability of not having ciguatera poisoning from fishes caught in narrow lagoons is simply a statistical artifact. I suggest that these results are not artifacts, especially since most fishing activities on Rarotonga are carried out on the narrow reef locations, which are considered safe by residents. I propose that wide lagoons tend to have reduced water circulation that is ideal for the establishment of harmful algae populations. Constant water movement on fore reefs, and frequent flushing across narrow lagoons, might reduce the densities of toxic dinoflagellates. Turbulent conditions affect dinoflagellate growth (Smayda 1997), and certainly reduce their densities on

macroalgal hosts (see Nakahara et al. 1996). The present study may explain why reef fishes resident in surf zones (i.e., *Epinephelus hexagonatus* and *Cirrhitus pinnulatus*) were considered ‘safe’ in this survey (see Table 3), regardless of where they were caught. Toxicity studies of resident fish species in different reef zones may help clarify the relationship between toxic fishes and the distribution of ciguatera-causing dinoflagellates. In addition, narrow lagoons in Rarotonga are generally shallow (< 5 m), which may not be ideal for the growth of ciguatoxic dinoflagellates. For example, Yasumoto et al. (1980) found that the density and growth of *G. toxicus* were lower at depths less than 0.5 m where light intensity was greater than 19% full sunlight.

VECTOR SHIFTS OF CIGUATERA POISONING

Between 1989 and 2009, there were clear shifts in the types of reef fishes involved in ciguatera poisoning in Rarotonga. Firstly, between 1989 and 2000, herbivores (i.e., acanthurids) were primarily implicated in ciguatera poisoning. By contrast, ciguatera poisoning from benthic invertivores (e.g., goatfish and mullet) and filter feeders (i.e., *Tridacna* spp.) became more evident from 2001 to 2005. Herbivores (i.e., scarids) and notably carnivores were most frequently implicated in ciguatera poisoning from 2006 to 2009 (see Fig. 5). Similar temporal shifts in ciguatera-poisoning vectors were noted in the Gambier Islands over a 20-year period (Bagnis et al. 1988). Herbivores were mainly responsible for ciguatera poisoning from 1967 to 1975, whereas herbivores and carnivores were equally

involved from 1976 to 1982, and carnivores were mostly involved from 1983 to 1987 – with incidents of ciguatera poisoning declining after 1987 (Bagnis et al. 1988). My findings in Rarotonga agree with the Gambier Islands study that ironically showed an increased involvement of carnivorous fishes, in cases of ciguatera poisoning, before incidences in these same fishes declined.

Ciguatera poisoning was historically thought of as a ‘carnivore problem’ (Lewis 2006). Alternatively, I suggest that if carnivores are the *only* fishes involved in ciguatera poisoning, then the general incidence of ciguatera should decline in the near future. My argument stems from the fact that at low dinoflagellate densities, toxins only bioaccumulate in large carnivores, and remain low in other fishes. The shift in ciguatera-poisoning vectors from herbivorous fishes to benthic invertivores (i.e., *Mulloidess*), between 2001 and 2005, is also of interest. Although the transfer of toxins from specific dinoflagellates into the food web is mainly recognized through herbivorous fishes (Randall 1958), there is also some evidence that crustaceans transfer toxins (Lewis et al. 1994a), which may explain the involvement of soft-bottom invertivores in ciguatera poisoning in Rarotonga. In addition, my survey indicated that parrotfishes and surgeonfishes were edible in 2010, despite local knowledge of their involvement in many past cases of ciguatera poisoning.

Nevertheless, human adjustment to ciguatera poisoning is certainly evident in Rarotonga. Residents know that juveniles of known toxic species (e.g., *Mulloidess flavolineatus* and *Caranx* spp.) are safe to eat. However, ‘low-risk’

fishes are now being targeted (see Table 3). For example, *Kyphosus* spp., arguably the safest reef fish recorded in the survey, may be experiencing heavy fishing pressure. Moreover, *Naso unicornis* was in 2010 considered to be among the safest reef fish in Rarotonga, while in Raivavae (of the Austral Islands) this species is considered high-risk (Chinain et al. 2009).

ALGAL COMMUNITY AND CIGUATERA-CAUSING

DINOFLAGELLATES. Macroalgal cover has been uncommon on the fore reefs of Rarotonga in recent years. However, macroalgae such as *Sargassum*, *Dictyota*, *Jania*, and *Galaxaura*, known to host high densities of *G. toxicus* (e.g., Shimizu et al. 1982; Anderson and Lobel 1987; Cruz-Rivera and Villareal 2006), were common in the lagoon for most of the 1990s (T. Rongo, pers. obs.). Although we lack detailed information on algal species composition through time, the shift in (ciguatera-poisoning) vectors may reflect the dominant algae present on the reefs during these periods. For example, macroalgae grazers (e.g., *Ctenochaetus* spp.) and browsers (e.g., *Naso unicornis* and *Acanthurus* spp.) were prolific in the 1990s, and were potential ciguatera-poisoning vectors. Yet, in the 2000s, the ciguatera-poisoning vector shifted to benthic invertivores (e.g., goatfish and mullet) that feed among sediments and turf algae. Notably, turf algae were the dominant substrata during the 2000s (Rongo et al. 2006, 2009b), and have been reported to host among the highest densities of *G. toxicus* (see Cruz-Rivera and Villareal 2006). Because macroalgae and turf grazers such as *Ctenochaetus* spp. (the most implicated

herbivore in ciguatera poisoning in the early sampling) were avoided for much of the 2000s, it is difficult to distinguish whether this early shift in vectors is the result of algal community shifts or human adjustment to ciguatera poisoning.

Shifts in algal communities may also result from changes in herbivorous fish densities. I note that the average density of herbivorous fishes in Rarotonga increased from a mean of 0.26 (± 0.07 standard error) fishes per m^2 in 1999 (Ponia et al. 1999) to a mean of 1.40 (± 0.63 standard error) fishes per m^2 in 2006 (Rongo et al. 2006). Such increases in herbivorous fish densities may have been a consequence of coral loss (Sheppard et al. 2002; Wilson et al. 2006) (because of *Acanthaster planci* outbreaks) and declines in fishing pressure, because of ciguatera poisoning. Changes in fishing pressure may have also affected trophic cascades. For example, high fishing pressure in the early 1990s, particularly on herbivorous fishes (see Ponia et al. 1999), could have led to macroalgae dominance. But, when ciguatera poisoning became chronic in the early 2000s, fishing pressure declined and herbivorous fish densities would have increased; such increases would have reduced macroalgae cover, as was apparent for much of the 2000s.

Increased herbivorous fish densities may have also facilitated the transfer of ciguatoxins through the food web (an idea mentioned by Bagnis et al. [1988] in their study in French Polynesia), causing the ‘flare-up’ of ciguatera poisoning after 2003. Increased herbivore densities may also explain the increased number of fish species involved in ciguatera poisoning, especially during the transitional period, where some 40 species were involved compared with 33 species involved during

both the *A. planci* period and the recovery period. Notably, the average density of herbivorous fishes decreased from a mean of 1.40 (± 0.63 standard error) fishes per m^2 in 2006 to 0.51 (± 0.20 standard error) fishes per m^2 in 2009 (Rongo et al. 2009b), which also corresponded with a decline in hospital cases of ciguatera poisoning in Rarotonga.

The shift to turf algae in the 2000s may have altered the assemblages of toxic dinoflagellates in Rarotonga. For example, the Cook Islands Ministry of Marine Resources (who has occasionally monitored *G. toxicus* density since the early 1990s in Rarotonga) indicated that *G. toxicus* density decreased from 1000s per 100g (wet algae) in the 1990s to 10s per 100g in recent years; interestingly, the decline in *G. toxicus* density coincided with an increase in the density of *Ostreopsis* spp. (T. Turua, pers. comm.). Temporal shifts in dinoflagellate populations have been noted in the literature to relate with allelopathic interactions. For example, Taylor and Gustavson (1986) found an inverse relationship between abundance of *G. toxicus* and *Ostreopsis* spp. Similarly, Carlson (1984) found inverse correlations between *G. toxicus* and both *Prorocentrum rhathymum* and *Amphidinium carterae* abundance.

The involvement of other dinoflagellates in ciguatera poisoning is poorly understood (e.g., Lewis et al. 1998; Chinain et al. 1999), although Ballantine et al. (1985) and Carlson and Tindall (1985) suggested that *Ostreopsis* spp. and *Prorocentrum lima* were important contributors to ciguatera poisoning in the Caribbean. High densities of a *Prorocentrum* species, reported in Titikaveka lagoon

from sediments in 2003 (Skinner et al. unpublished data), coincided with the sharp increase in cases of ciguatera poisoning in 2004 and 2005 (see Fig. 3), involving benthic invertivores (e.g., goatfish and mullet) and filter feeders (i.e., *Tridacna* spp.) (Fig. 5). In addition, Rhodes et al. (2009) in their 2007 study in Rarotonga, found that dinoflagellates from the genus *Amphidinium*, *Ostreopsis*, and *Coolia* were toxic, and could co-occur with *Gambierdiscus*. However, further research is needed to examine if, in fact, other dinoflagellates are responsible for ciguatera poisoning in Rarotonga.

Laurent et al. (2008) identified cyanobacteria from the genus *Hydrocoleum* as the major cause of ciguatera-like poisonings in New Caledonia. Several authors have also identified the involvement of *Lyngbya* in fish poisonings in the Pacific Ocean (e.g., Habekost et al. 1955; Dawson et al. 1955; Hashimoto et al. 1976; see also Osborne et al. 2001). Although *Lyngbya* was implicated as the major source of eye and throat irritation and dermatitis in 2003 (called the Titikaveka Irritant Syndrome) (Eason and Hope 2005), their involvement in fish and *Tridacna* poisonings in Rarotonga cannot be ruled out. Van Dolah (2000) also suggested that factors such as nutrient loading, anomalous drought and storm events, and El Niño events can cause toxic cyanobacteria outbreaks. Therefore, it may be difficult to differentiate the effects of *Lyngbya* (the most common substrate on sandy lagoons around Rarotonga; T. Rongo, pers. obs.) uptake, from co-occurring ciguatoxic dinoflagellates in both reef fishes and marine invertebrates.

In summary, the widest reefs of Rarotonga elicited the most cases of ciguatera poisoning, but there was no relationship between ciguatera poisoning and reef exposure. In addition, I found that vector shifts in fishes involved in ciguatera poisoning are potential indicators for the waning of ciguatera poisoning.

CHAPTER III

THE INFLUENCE OF CLIMATE OSCILLATIONS ON CIGUATERA POISONING IN RAROTONGA, SOUTHERN COOK ISLANDS

INTRODUCTION

Ciguatera poisoning is the most reported seafood intoxication worldwide, estimated to affect around 50,000 – 500,000 people per year (Fleming et al. 1998). Symptoms of ciguatera poisoning include gastrointestinal, neurological, and cardiovascular disorders. Although ciguatera poisoning is limited to tropical and subtropical regions throughout the Pacific, Indian, and Caribbean, economic globalization has increased the extent of the problem beyond the ciguatera-endemic regions. For example, many Pacific countries export reef fishes to offshore markets in Europe, Asia, and the USA (see Van Dolah 2000; Wong et al. 2005; Dickey and Plakas 2010). In addition, through tourism, ciguatera poisoning is often reported in many temperate regions (e.g., Todd 1985, 1990).

The causative organism of ciguatera poisoning has been identified as *Gambierdiscus toxicus* Adachi and Fukuyo, 1979, however other dinoflagellates have been also implicated (Faust 1995; Holmes 1998; Parsons and Preskitt 2007, Rhodes et al. 2009). For example, *Ostreopsis* spp. and *Prorocentrum lima* have been suggested to be the main dinoflagellates causing ciguatera poisoning in the Caribbean (Ballantine et al. 1985; Carlson and Tindall 1985). There are also suggestions that outbreaks of cyanobacteria have been linked to ciguatera-

poisoning-like symptoms (Habekost et al. 1955; Dawson et al. 1955; Hashimoto et al. 1976; Laurent et al. 2008; Yeeting 2009a).

Environmental factors that may lead to outbreaks of ciguatera poisoning can be grouped into two broad categories. First, just as human-disease outbreaks are bio-indicators of ecosystem health (Spiegel and Yassi 1997; Cook et al. 2004), ciguatera-poisoning outbreaks have been linked to degraded coral-reef ecosystems. The ‘new surface hypothesis’ (Randall 1958) suggests that disturbances (i.e., cyclones, tsunamis, coral bleaching, *Acanthaster planci* outbreaks, dredging, boat channel construction, boat anchorage, and shipwrecks) provide freshly denuded surfaces for macroalgae to serve as substrate for toxic dinoflagellates (Cooper 1964; Banner 1976; Bagnis et al. 1988; Kohler and Kohler 1992; Bagnis 1994; Chinain et al. 1999). However, this rationale, although generally accepted, remains theoretical and correlations have been weak (see Kaly and Jones 1994; Bruslé et al. 1998; see Bienfang et al. 2008).

Alternatively, the ‘climate oscillation hypothesis’ suggests that ciguatera poisoning is primarily a consequence of specific phases of climate oscillations. For example, high, anomalous sea surface temperatures, associated with El Niño years of the inter-annual cycle of the El Niño Southern Oscillation (ENSO), have been frequently cited as the major factor inducing ciguatera-poisoning outbreaks (Tosteson et al. 1988; Epstein et al. 1993; Hales et al. 1999; Chateau-Degat et al. 2005; Tester et al. 2010). By contrast, Llewellyn (2009) proposed that the predicted scenario of high temperatures may instead suppress the incidence of ciguatera

poisoning in tropical regions. Therefore, the influence of climate oscillations on ciguatera poisoning remains uncertain.

Climate oscillations (i.e., ENSO and the Pacific Decadal Oscillation [PDO]) bring different climate conditions to different regions in the Pacific. Notably, El Niño years tends to bring cool sea surface temperatures to the western and central Pacific whereas La Niña years promote warm temperatures and high rainfall. In the southern Cook Islands, conditions are cool during El Niño and warm during La Niña years, while the opposite is observed in the northern Cook Islands (Baldi et al. 2009). There is also a coupling effect between ENSO and PDO; El Niño activities are frequent during the positive phase of the PDO while La Niña activities are frequent during the negative phase (Verdon and Franks 2006).

Predicting ciguatera poisoning is essential over both the short and long term, particularly in Rarotonga in the southern Cook Islands (see Fig. 1, p. 16), where ciguatera poisoning has been problematic for almost 20 years. I was able to test both the ‘new surface hypothesis’ and the ‘climate oscillation hypothesis’ by examining ciguatera poisoning through time and monitoring the reef condition in Rarotonga. More specifically, I examined (1) the frequency of disturbances and its influence on ciguatera-poisoning events, and (2) whether there were any links between cases of ciguatera poisoning and climate cycles (i.e., ENSO and PDO).

MATERIALS AND METHODS

In Chapter II, two periods where ciguatera-poisoning events occurred in Rarotonga were identified (see Fig. 3, p. 27): (1) a minor event in the early 1970s, and (2) from 1989 onwards. Hospital cases from 1994 to 2011 (obtained from the Cook Islands Ministry of Health) were used to examine the relationship between ciguatera poisoning and climate oscillations (e.g., ENSO and PDO).

DATA ANALYSIS

The frequency of disturbances was compared over time and in relation to the two ciguatera-poisoning events identified. For my purposes, years prior to 1989 were considered as the 1970s ciguatera-poisoning event (1970 – 1988). Various sources were used to compile data on natural disturbances impacting Rarotonga's reefs since the 1970s. Disturbances were included in the analysis only if they were observed to, or potentially have, impacted Rarotonga's reefs. They included: (1) major cyclones, cyclones, storms, and gales (Asian Development Bank 2005; de Scally et al. 2006; Baldi et al. 2009; New Zealand's National Institute of Water and Atmospheric Research database), (2) El Niño events (obtained from National Climate Center 2011a) assumed in this study to cause coral bleaching (based on observations by Goreau and Hayes [1995], Rongo et al. [2006, 2009], and T. Rongo [pers. obs.]), and (3) *Acanthaster planci* outbreaks (Devaney and Randall 1973; Dahl 1980; Lyon 2003). There were two *A. planci* outbreaks reported, one

within each ciguatera-poisoning event, lasting around seven years. In this study, each outbreak was considered one disturbance event. Subsequently, disturbances were combined within their respective ciguatera-poisoning event before conducting a Chi-square test to examine independence.

Yearly averages, using monthly climate data, were calculated for the El Niño Southern Oscillation (ENSO), using the Southern Oscillation Index (SOI) (National Climate Center 2011b). Because El Niño years occurred when the yearly SOI average was negative, and La Niña years occurred when the yearly SOI average was positive, in this study, a negative SOI value was referred to as an El Niño year while a positive SOI value was referred to as a La Niña year. With this method, climate neutral years would fall within an El Niño or La Niña year depending on the yearly SOI average.

Wavelet analysis was used to determine the dominant climate signature in Rarotonga, on a longer time scale, by re-analysing the ~270-year Sr/Ca raw data set derived from a Rarotonga coral by Linsley et al. (2000). The Sr/Ca is an accurate proxy of water temperature (McCulloch et al. 1994). Cross wavelet transform analysis was used to determine the regions (in time-frequency space) of significant common power between two time series data (see Grinsted et al. 2004): the yearly SOI average and hospital cases of ciguatera poisoning.

A Chi-square test was conducted to examine the relationship between observed and expected cases of ciguatera poisoning from hospital records (1994 – August 2011) for the positive and negative phases of SOI (with a 1-year lag period

between climate data and cases of ciguatera poisoning). The hypothesis was that more cases of ciguatera poisoning occurred during El Niño years.

Cross wavelet analysis and chi-square test was not conducted between PDO and hospital cases because of insufficient data available, as PDO has been predominantly in the positive phase since 1977.

RESULTS

Numerous disturbance events occurred in Rarotonga between 1970 and 2011 (Fig. 6; Table 6). There were significantly more disturbances during the recent ciguatera-poisoning event from 1989 to 2011 ($\chi^2_1 = 4.5, p < 0.034$), which corresponded to a higher number of ciguatera-poisoning cases (Table 7; see Fig. 6).

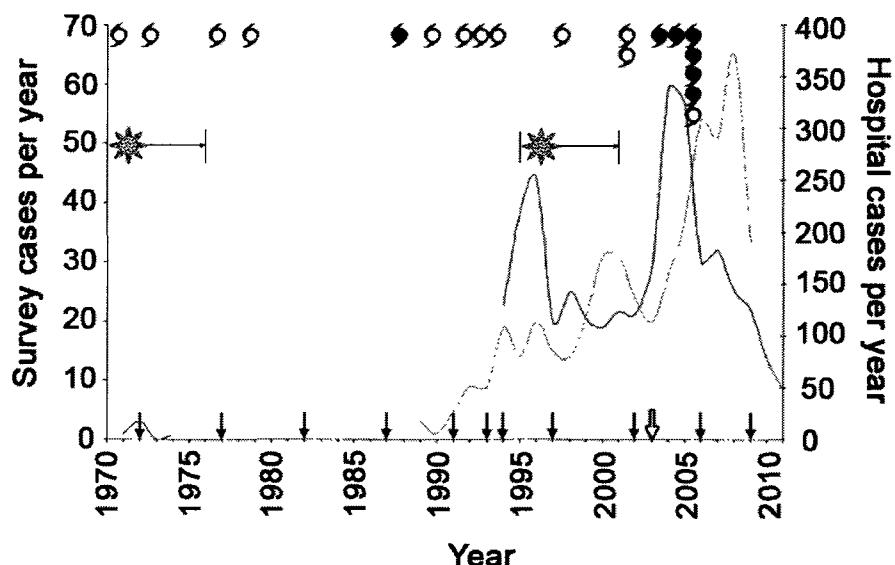


Figure 6. Natural disturbance events impacting Rarotonga's reefs from 1970 to 2011. Cases of ciguatera poisoning from Rarotonga's hospital data (thick line) and the questionnaire survey (thin line). The two ciguatera-poisoning events identified by the survey within 1970 – 1988 (white region) and 1989 – 2011 (grey region) indicated. Sun-shapes and horizontal arrows indicate the periods of two major *Acanthaster planci* outbreaks: ~1970 to 1976 (Devaney and Randall 1973; Dahl 1980), and ~1995 to 2001 (Lyon 2003; Rongo et al. 2006). Open symbols indicate years that Category ≤ 3 systems (cyclones, storms, and gales; 1970, 1972, 1976, 1978, 1989, 1991, 1992, 1993, 1997, 2001, 2005) impacted Rarotonga, and filled symbols indicate years that Categories 4 and 5 systems (major cyclones; 1987, 2003, 2004, 2005) impacted Rarotonga (Asian Development Bank 2005; de Scally et al. 2006; Baldi et al. 2009). Black arrows indicate El Niño years taken from the National Climate Center (2011a) (1972, 1977, 1982, 1987, 1991, 1993, 1994, 1997, 2002, 2006, 2009). White arrow marks the Titikaveka Irritant Syndrome in 2003 (Eason and Hope 2005).

Table 6. Natural disturbances (e.g., cyclones, *Acanthaster planci* outbreaks, coral bleaching, and harmful algal bloom) impacting Rarotonga between 1970 and 2011. Cyclone and wind data taken from Asian Development Bank (2005), de Scally et al. (2006), Baldi et al. (2009), and New Zealand's National Institute of Water and Atmospheric Research database (www.cliflo.niwa.co.nz). Cyclone, storm, and gale refer to Category ≤ 3 systems, and major cyclones refer to Category 4 and 5 systems. *Acanthaster planci* outbreak data and coral bleaching information taken from Devaney and Randall (1973), Dahl (1980), Goreau and Hayes (1995), Lyon (2003), Rongo et al. (2006, 2009b), and T. Rongo (pers. obs.).

Year	Natural disturbance	Description of impact
1970	<i>Acanthaster planci</i> outbreak begins, Cyclone (Dolly)	High winds damaged ~40% of Rarotonga's export banana crop, information on wave damage not available
1971	<i>A planci</i> outbreak	
1972	<i>A planci</i> outbreak, Cyclone (Agatha)	Extensive damage from <i>A planci</i> outbreak noted in lagoon areas on the northwestern exposure, <i>Agatha</i> wind speed up to 134 km/h that damaged ~75% of Rarotonga's export banana crop, information on wave damage not available
1973	<i>A planci</i> outbreak	
1974	<i>A planci</i> outbreak	
1975	<i>A planci</i> outbreak	
1976	<i>A planci</i> outbreak ends, Storm (Kim)	Lagoon coral cover declined from <i>A planci</i> outbreak, <i>Kim</i> wind speed up to 135 km/h, causing some fishing vessel damage
1978	Cyclone (Charles)	Wind speed up to 135 km/h, reported to have caused damage to the wharf
1987	Major cyclone (Sally)	<i>Sally</i> wind speed up to 148 km/h and wave height of 12 m
1989	Gale (unnamed)	Wind speed up to 105 km/h, no information on swells
1991	Cyclone (Val), Coral bleaching	<i>Val</i> wind speed up to 74 km/h and wave height of 14 m, severe bleaching of lagoon corals from extreme low tides
1992	Cyclone (Gene)	Wind speed up to 115 km/h, flooding and big swells with coastal damage on western exposure
1993	Cyclone (Nisha)	Wind speed up to 74 km/h, big swells
1994	Coral bleaching	Coral bleaching from high SSTs impacted corals on fore reef slopes on the northern to western exposure
1995	<i>A planci</i> outbreak begins	Large numbers noted on the northern fore reef exposure
1996	<i>A planci</i> outbreak	Extensive damage of fore reef slopes on the northern exposure
1997	Cyclone (Pam), <i>A planci</i> outbreak	Wind speed up to 150 km/h, wave height of 14 m, record rainfall of 107 mm in 6 hr, <i>A planci</i> damage on the northeastern exposure
1998	<i>A planci</i> outbreak, Coral bleaching	<i>A planci</i> damage on eastern and southeastern exposure, coral bleaching of lagoon corals from extreme low tides
1999	<i>A planci</i> outbreak	Extensive damage of fore reef slopes on the southern exposure
2000	<i>A planci</i> outbreak	Extensive damage of fore reef slopes on the southwestern and western exposure
2001	<i>A planci</i> outbreak ends, Storm (Oma, Trina)	<i>A planci</i> outbreak significantly reduced fore reef coral cover, <i>Oma</i> wind speed up to 130 km/h and heavy rain, <i>Trina</i> wind speed up to 102 km/h with big swells
2003	Major cyclone (Dovi), Titikaveka Irritant Syndrome (TIS)	Wind speed up to 66 km/h and wave height of 17 m, strong swell/surge along coastal areas, <i>TIS</i> harmful algal bloom causing eye and respiratory irritation in residents
2004	Major cyclone (Heta)	Wind speed up to 72 km/h and wave height of 17.4 m, major coastal damage
2005	Major cyclone (Meena, Nancy, Olaf, Percy), Gale (Rae)	Severe coastal damage from the four major cyclones, <i>Meena</i> wind speed up to 161 km/h, <i>Nancy</i> wind speed up to 163 km/h and wave height of 22 m, <i>Olaf</i> wind speed up to 95 km/h, <i>Percy</i> wind speed up to 76 km/h and wave height of 19 m, <i>Rae</i> wind speed up to 75 km/h
2006	Coral bleaching	Coral bleaching of lagoon corals observed in Ngatangia on the southeastern exposure from extreme low tides
2009	Coral bleaching	Minor bleaching observed on reef flats on the northern exposure from extreme low tides

Table 7. Chi-square test comparing natural disturbances between the two ciguatera-poisoning events identified in Chapter II: (1) the 1970s event, and (2) from 1989 onwards. Asterisk (*) indicates significant difference at $p < 0.05$.

Disturbances	1970s event	1989 onwards
Cyclone frequency	5	14
El Niño frequency	4	7
<i>Acanthaster planci</i> outbreak	1	1
Total	10	22
	$\chi^2_1 = 4.5, p < 0.034^*$	

Wavelet analysis on the ~270-year Sr/Ca raw data set derived from a Rarotonga coral revealed significant multi-decadal anomalous temperature variability recurring every 30 years (Fig. 7). Although results indicate some ENSO-like signatures around AD 1800 in the southern Cook Islands, clearly multi-decadal oscillations related to the Pacific Decadal Oscillation dominated temperature changes over the last 300 years (Linsley et al. 2000; Ren et al. 2002). Although the link between ENSO and PDO are poorly understood, Verdon and Franks (2006) indicated a coupling effect where El Niño is frequent during the positive PDO, and La Niña is frequent during the negative PDO.

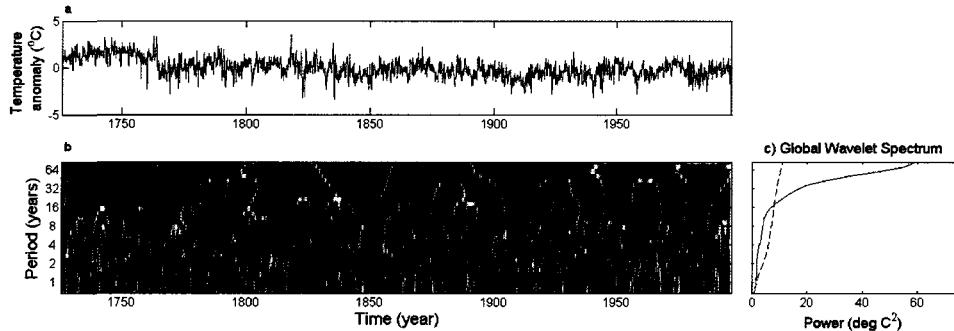


Figure 7. (a) Changes in estimated sea-surface temperature anomalies spanning from AD 1726 to 1996 for Rarotonga using coral Sr/Ca (Linsley et al. 2000); (b) local time-frequency spectrum, where dark-red shading indicates high sea-surface temperature variance and black contours enclose regions that are significantly above red noise at the 95% confidence level; and (c) the results of a formal chi-square test that displays return period (y-axis) (or 1/frequency) in relation to red noise; above-noise return periods are significant when displayed to the right of the global red-noise (indicated as a dashed line), which is only evident for Rarotonga at the multi-decadal timescale (Torrence and Compo 1998).

The wavelet analyses of the Southern Oscillation Index (SOI) and cases of ciguatera poisoning from 1994 to 2011, indicated some activity at the 4-year period (Fig. 8a). The cross wavelet analysis indicated that the SOI and cases of ciguatera poisoning showed common power at the 4-year period (Fig. 8b).

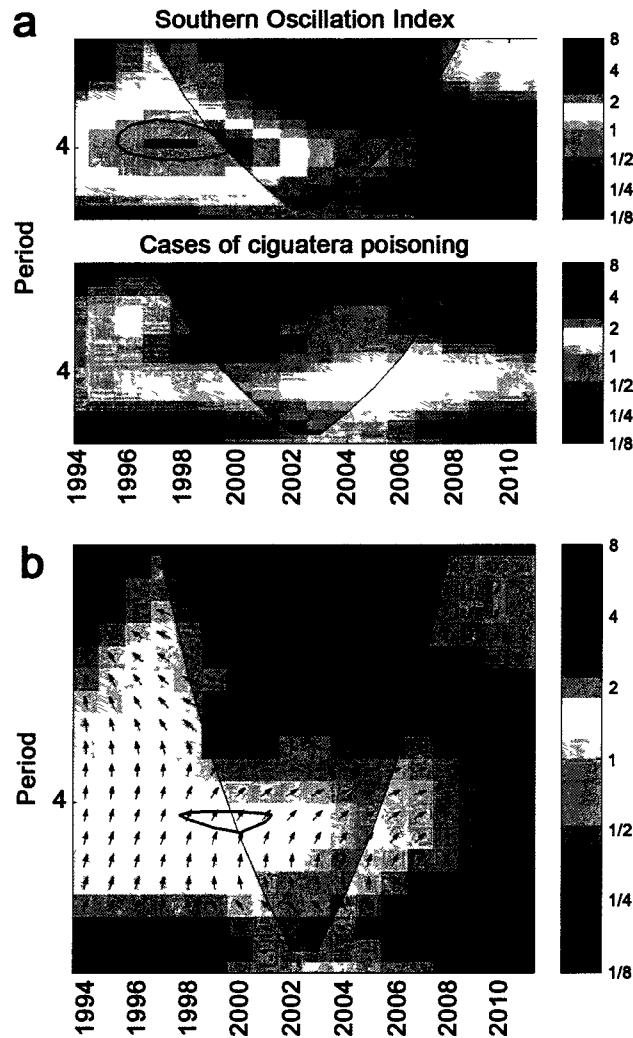


Figure 8. (a) Wavelet analysis of the Southern Oscillation Index (SOI) and cases of ciguatera poisoning from 1994 to 2011. (b) Cross wavelet transform of the SOI and ciguatera-poisoning cases. The thick, black line contours designate the 5% significance level against red noise, and the black line depicts the cone of influence in each subplot.

A chi-square test was conducted to examine the difference in cases of ciguatera poisoning between the positive and the negative phase of the SOI. Cases were significantly higher during El Niño years compared with La Niña years ($\chi^2_1 = 617, p < 0.001$), using a one-year lag period between climate data and cases (Table 8).

Table 8. Chi-square tests comparing Rarotonga's hospital cases of ciguatera poisoning from 1994 to 2011 (August) during the positive and negative phases of the Southern Oscillation Index (SOI; La Niña and El Niño, respectively), using a 1-year lag period between climate data and case of ciguatera poisoning. Asterisk (*) indicates significant difference at $p < 0.05$.

	El Niño	La Niña
Observed cases of ciguatera poisoning	2122	783
	$\chi^2_1 = 617, p < 0.001^*$	

DISCUSSION

This study showed that higher disturbance frequency associated with El Niño years, coupled with the positive phase of the Pacific Decadal Oscillation (PDO), were important in driving ciguatera poisoning in Rarotonga. Although my findings were consistent with Hales et al. (1999), who showed a significant correlation between El Niño events (using SST anomalies) and cases of ciguatera poisoning in Rarotonga from 1973 to 1994, I note that ciguatera poisoning did not become a problem in Rarotonga until the late 1980s to the early 1990s. I also note that such major differences in previous reporting of ciguatera poisoning involved the erroneous pooling of data that was obtained from the South Pacific Epidemiological and Health Information Service (SPEHIS) database. Cases of ciguatera poisoning reported to the SPEHIS database combined the southern and the northern Cook Islands, where climate conditions are different (see Baldi et al. 2009). Furthermore, my examination from 1994 to 2011, comparing hospital cases with SST anomalies (from the Cook Islands Meteorological Service) from Rarotonga, showed no significant correlation (Pearson's correlation, $r = -0.03$; $p = 0.90$).

Randall's 'new surface hypothesis' suggests that new surfaces become available usually after the loss of corals (Cooper 1964; Bagnis et al. 1992a; Bagnis, 1994; Kohler and Kohler 1992; Chinain et al. 1999). My data from Rarotonga 'cautiously' agrees with this hypothesis, but I add that the state of the reef is less

important than the generation of denuded carbonate substrate by disturbances.

Disturbances provide fresh, primary successional surfaces for algae to host opportunistic populations of ciguatoxic dinoflagellates. Indeed, turf algae has been the most dominant substrate on Rarotonga's reefs for over 10 years, and coral cover has been well below 10% at most localities (Rongo et al. 2006, 2009b). Yet, even in this algal-dominated system, outbreaks of ciguatera poisoning only occurred after several major disturbances to the reef.

The scale, intensity, and frequency of disturbances may play an important role in ciguatera-poisoning outbreaks (Kaly and Jones 1994). While small-scale, local disturbances might have little impact on outbreaks of ciguatera poisoning (Kaly and Jones 1994), large-scale disturbances, such as cyclones and coral-bleaching events, seem to be a prerequisite of outbreaks (de Sylva 1994; Chinain et al. 1999). I note that the frequency of reef disturbances and cases of ciguatera poisoning were low in Rarotonga between the 1970s and 1980s. By contrast, the cyclone frequency was high between 2001 and 2005, coinciding with the highest number of ciguatera-poisoning cases on record (see Fig. 6). The frequency of cyclones in the southern Cook Islands is influenced by the mean location of the South Pacific Convergence Zone that shifts in relation to the ENSO and PDO cycles (Folland et al. 2002); cyclone frequency in the southern Cook Islands is greater during El Niño and positive PDO years (de Scally 2008; Baldi et al. 2009).

Although the disturbance frequency was significantly different between the two ciguatera-poisoning events (see Table 7 and Fig. 6), I could not verify which

disturbance, if any, was responsible for the ciguatera-poisoning outbreak.

Rarotonga tends to be *cool and dry* during El Niño events (Baldi et al. 2009), with lower than average sea level (Pacific Country Report – Cook Islands 2003). Under such conditions, the reef edge is frequently exposed and lagoonal exchange with the adjacent ocean is reduced; such conditions might favor toxic algal blooms.

Alternatively, La Niña years that are *warm and wet* might be unfavorable for ciguatoxic dinoflagellates, which prefer higher salinity waters (Yasumoto et al. 1980; Taylor 1985; Bomber et al. 1988). While elevated temperatures have been identified as the cause of ciguatera poisoning (e.g., Hales et al. 1999), outbreaks of ciguatera poisoning in Rarotonga have historically occurred when temperatures were cooler. Therefore, warmer temperatures may not be driving ciguatera poisoning in Rarotonga. Although we are far from identifying one primary cause of ciguatera poisoning, this study suggests however that the combination of climate conditions during El Niño events and a positive phase of the PDO, cause a high frequency of storm activity that facilitates ciguatera poisoning in Rarotonga. Perhaps further examining this geographic region will aid our understanding of these factors, and assist in the development of predictive models of ciguatera-poisoning outbreaks.

In summary, my results agree with Rongo et al. (2009a), who proposed that ciguatera-poisoning events in the southern Cook Islands were linked to the positive phase of the Pacific Decadal Oscillation, a phase that is also accompanied by a high frequency of El Niño events and cyclones (see de Scally 2008). The recent decline

in the incidence of ciguatera poisoning in Rarotonga, and the continued decline through 2010, coincided with the recent shift of the Pacific Decadal Oscillation into a negative phase. I therefore predict that ciguatera poisoning will continue to decline in Rarotonga over the next decade, during this negative phase.

Over the longer term, however, ocean temperatures are predicted to increase by up to 3°C by the end of 2100 (Hegerl et al. 2007). If ciguatera poisoning is related to high, anomalous water temperatures, then the incidence of ciguatera poisoning is also expected to rise (de Sylva 1994; see World Bank 2000). However, Llewellyn (2009) proposed that a warmer climate would suppress ciguatera poisoning. The predicted warmer climate in the future will however increase the intensity of cyclones but potentially reduce their frequency (Hegerl et al. 2007). Therefore, and according to my findings, we should see fewer outbreaks of ciguatera poisoning during periods of low cyclone activity. Yet, with the pole-ward migration of species predicted under climate change (e.g., Parmesan and Yohe 2003), we may simply see the impact of toxic dinoflagellates shifting into higher latitudes, and potentially impacting fisheries in those regions (Rhodes et al. 2009).

CHAPTER IV

THE EFFECTS OF NATURAL DISTURBANCES ON REEF STATE AND HERBIVOROUS FISH DENSITY, AND THEIR INFLUENCE ON CIGUATERA POISONING IN RAROTONGA, SOUTHERN COOK ISLANDS

INTRODUCTION

Coral reefs around the world are experiencing shifts toward less desirable states because of a range of disturbances, which include eutrophication (Bell 1992; Lapointe 1997), *Acanthaster planci* infestations (Done 1987), disease (Lessios et al. 1984; Hughes 1994; Precht and Aronson 2006), overfishing (Jackson et al. 2001; Pandolfi et al. 2003; Bellwood et al. 2004), run-off from poor land-use practices (van Woesik et al. 1999), and global climate change (Glynn 1984; Hoegh-Guldberg 1999, Hoegh-Guldberg et al. 2007; Veron et al. 2009). Reef disturbances have also been linked to outbreaks of ciguatera poisoning (Bagnis 1994), which is the most common form of seafood intoxication globally (Baden et al. 1995).

The ‘new surface hypothesis’ suggests that reef disturbances provide space for opportunistic macroalgae, the preferred substrate for ciguatoxic dinoflagellates, which in turn increases the risk of ciguatera poisoning (Randall 1958; Bagnis et al. 1980). Ciguatoxins are produced by dinoflagellates (e.g. *Gambierdiscus toxicus*) epiphytic to macroalgae, and are inadvertently transferred into the food web via grazing of herbivorous fishes or invertebrates (Randall 1958; Lewis et al. 1994a,b). Although the ‘new surface hypothesis’ has gained much support in the literature,

most accounts are largely anecdotal. Reef damage from boat channel construction (Tebano 1984), boat anchorage and wrecks (Cooper 1964), cyclones (Randall 1958; Banner 1976; Rongo and van Woesik 2011), *Acanthaster planci* outbreaks (Bagnis et al. 1988), and bleaching events (Kohler and Kohler 1992; Bagnis et al. 1992a) have all been associated with increased cases of ciguatera poisoning. Alternatively, some reef disturbances occur without any increased risk of ciguatera poisoning. For example, the risk of ciguatera poisoning did not increase from the blasting of a channel in Tuvalu (Kaly and Jones 1994) or an *A. planci* outbreak in the 1970s in Rarotonga, Cook Islands (Rongo and van Woesik 2011). In addition, Lewis et al. (1986) found that the toxicity in herbivorous fishes and the density of *Gambierdiscus toxicus* did not increase three months after cyclone damage to Sudbury Reef on the Great Barrier Reef, although continued monitoring to account for the lag period (up to 18 months; Bagnis 1969) between a disturbance and ciguatoxins appearing in the food web may have provided different results.

The influence of reef disturbances on ciguatera poisoning may depend on the intensity, scale, and frequency of the disturbances (Kaly and Jones 1994), as these factors are known to affect reef state (Connell and Keough 1985; van Woesik et al. 1991; Gardner et al. 2005; Mumby et al. 2007). However, there is a general consensus that several factors, in combination, may interact synergistically with disturbances to drive outbreaks of ciguatera poisoning (e.g., Bagnis et al. 1988; Kaly and Jones, 1994). These factors include temperature (Tosteson et al. 1988; Hales et al. 1999; Chateau-Degat et al. 2005; Tester et al. 2010; but see Llewelyn

2009), salinity (Yasumoto et al. 1980; Carlson 1984; Taylor 1985), irradiance (Bomber et al. 1988), precipitation (Carlson and Tindall 1985), nutrient levels (Carlson 1984), the genetic nature of the dinoflagellate clones and their microbial symbionts (Tosteson et al. 1989), and the presence and density of vectors for toxin transfer (e.g., Bagnis et al. 1988; Bruslé 1997).

Based on the notion that increased grazing by herbivorous fishes will facilitate the transfer of ciguatoxins into the food web, Bagnis et al. (1988) suggested that increased density of herbivorous reef fishes also may play a key role in determining outbreaks of ciguatera poisoning in the Gambier Islands of French Polynesia. The 'fish-density hypothesis' has received no attention since it was first proposed over two decades ago. I conducted a study in Rarotonga (Fig. 9), in the southern Cook Islands (see Fig. 1, p. 16), to examine how changes in reef state (percent hard coral and algal cover) and fish density influenced the dynamics of ciguatera poisoning over an 18-year period.

The earliest surveys of the coral reefs of Rarotonga were conducted in 1994 (Miller et al. 1994). Miller et al. (1994) surveyed three sites on the southeastern exposure, and three sites on the northern exposure using the line-intercept transect (LIT) technique to assess the reef benthos, and using the Underwater Visual Census (UVC) technique for quantifying fish density. In 1999, Ponia et al. (1999) surveyed six sites using the video transect technique to assess the reef benthos, and the UVC technique to estimate fish densities. In 2003, Lyon (2003) surveyed 10 sites, but only examined the benthos using the LIT technique.

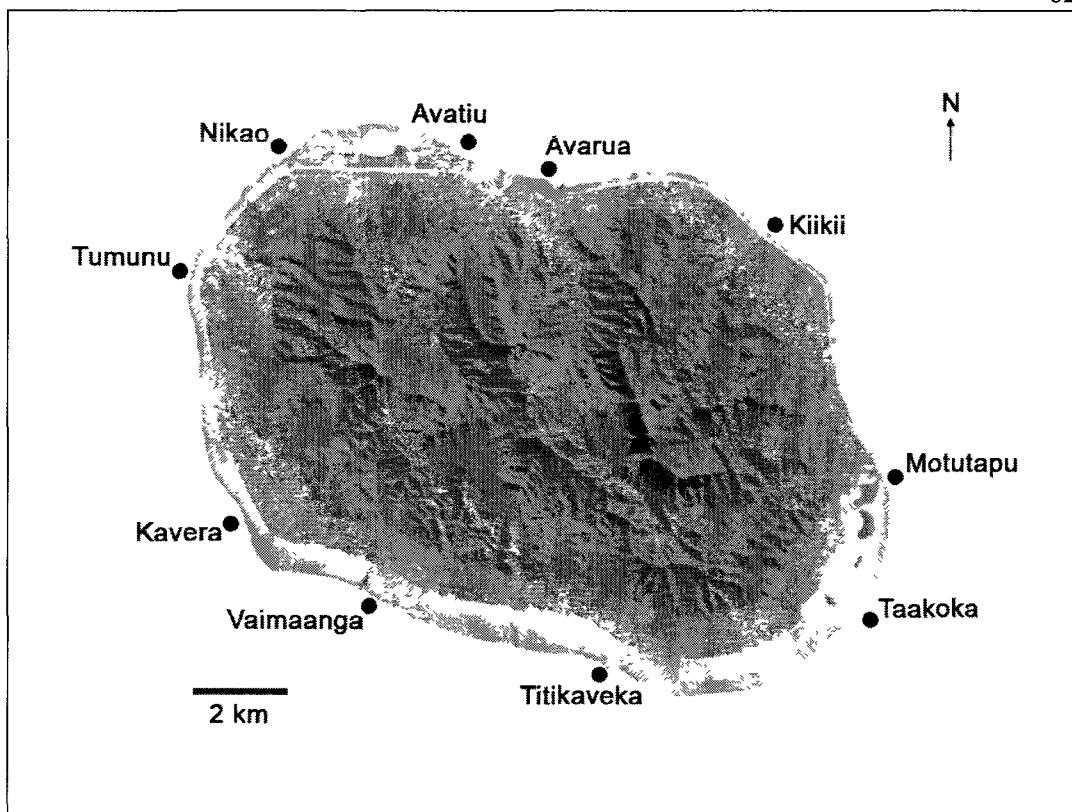


Figure 9. Rarotonga ($21^{\circ} 12' S$, $159^{\circ} 43' W$) depicting the fore-reef sites surveyed in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2003 (Lyon, 2003), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011; not all sites were surveyed in each survey year. Picture modified from Google Earth image.

MATERIALS AND METHODS

In 2006, 2009, and 2011, I revisited the general areas where previous surveys were conducted. At each site, four 50-m transects (replicates) were deployed consecutively, at 10 m intervals parallel to shore, between 7 – 9 m. Along each transect, I used the point-quadrat technique, where a 1-m² quadrat frame was tossed haphazardly every 5 m along the 50-m transect for a total of 10 quadrats (40 quadrats per site). The quadrat was partitioned into 25 sections with string, providing 16 points where the strings intercept. The survey focused on measuring hard coral cover, turf algae (less than 1 cm in height, see Steneck, 1988), and macroalgae (greater than 1 cm in height, see Steneck, 1988); turf and macroalgae have been reported to host high densities of ciguatoxic dinoflagellates (see Cruz-Rivera and Villareal 2006). Fish surveys only recorded the density of known ciguatoxic herbivorous fishes (i.e., acanthurids and scarids; see Rongo and van Woesik 2011) using a 50-m by 4-m wide belt transect.

DATA ANALYSIS

Because of the inconsistency in survey techniques employed and the low number of sites surveyed from 1994 to 2003, analysis was only conducted from data collected in 2006, 2009, and 2011, where the same sites were re-surveyed using the same techniques. Only general comparisons were made with data from survey years prior to 2006. Because hospital cases of ciguatera poisoning did not

have site specific information, and all sites around the island have been affected by ciguatera poisoning (based on data from the questionnaire survey; see Chapter II), I pooled hard coral, turf and macroalgae, and fish density data across all sites within years to examine changes over time.

A non-parametric Friedman test was used to examine differences in percent cover of hard coral, turf algae, and macroalgae among years (2006, 2009, and 2011). Information for macroalgae was not available for years prior to 2006. Herbivorous reef fishes were grouped into three main categories prior to analysis, which only included those fishes implicated in past ciguatera poisonings in Rarotonga. The first category was *Ctenochaetus striatus*, a common acanthurid considered high-risk in Rarotonga (Rongo and van Woesik 2011) and a key primary vector of ciguatoxin (Randall 1958; Yasumoto et al. 1971; Banner 1984; Lewis et al. 1994b; Lewis 2006). Other acanthurid species considered ‘low risk’ for ciguatera poisoning in Rarotonga (e.g., *Acanthurus triostegus*, *A. guttatus*, *Naso unicornis*, and *N. lituratus*) (see Rongo and van Woesik 2011) were placed into the second category. The third category consisted of scarids, which are also ‘high-risk’ species in Rarotonga (see Rongo and van Woesik 2011). A non-parametric Friedman test was used to examine differences in fish density among years. Plots were generated using *Statistica 6®*, and all comparative analyses were conducted using *SPSS 16.0®*.

RESULTS

REEF STATE: 1970s – 2011

In Chapter III, it was noted that Rarotonga has experienced several reef disturbances over the last few decades (see Fig. 6, p. 49). Four Category 3 or less systems (cyclones, storms, and gales) were known to have impacted Rarotonga in 1970, 1972, 1976, and 1978 (see Table 6, p. 50). In 1971, Devaney and Randall (1973) reported the first known *Acanthaster planci* outbreak in Rarotonga, which lasted from 1969/70 to around 1976/77. In 1976, Dahl (1980) indicated that the *A. planci* outbreak had decreased coral cover in the lagoon, but reefs had recovered by the late 1980s, to pre-*A. planci* conditions (G. Paulay, pers. comm.), over a 10-year period.

In 1987, a Category 5 system (major cyclone) impacted Rarotonga and seven Category 3 or less systems impacted in 1989, 1991, 1992, 1993, 1997, and 2001 (see Table 6, p. 50), but the damage to reefs were not assessed. Coral bleaching events were first reported in the 1990s in Rarotonga, predominantly during El Niño years. A severe bleaching event associated with extreme low tides that affected lagoon corals was observed in 1991 (Goreau and Hayes 1995), and subsequent bleaching of this nature was observed in 1998 and 2009 on the reef crest (T. Rongo, pers. obs.; Rongo et al. 2009b). Coral bleaching that was caused by high sea surface temperature anomalies was observed in 1994, with damage limited to fore-reef corals (Goreau and Hayes 1995). A second *A. planci* outbreak

occurred from 1995 to 2001, where coral cover declined from an average of ~31% in 1994 (Miller et al. 1994) to ~5% in 2003 (Lyon 2003) (Fig. 10a). Coral cover further declined to 1% in 2006, after six major cyclones and one gale impacted Rarotonga between 2003 and 2005 (Rongo et al. 2006). However, reef recovery was evident after 2006, with coral cover averaging 5% by 2009 (Rongo et al. 2009b), and 8% by 2011, which was a significant increase from 2006 and 2009 ($\chi^2 = 64, p < 0.001$; Table 9).

Turf algal cover was significantly different ($\chi^2 = 55, p < 0.001$) among years (2006, 2009, and 2011), with mean cover declining from 87% in 2006, to 69% in 2009, to 62% in 2011 (Fig. 10b). Turf algal cover in 1999 (76%) and 2003 (66%) was comparable to cover estimated for 2009 and 2011, whereas turf algal cover in 1994 was lower (49%). Macroalgal cover was also significantly different ($\chi^2 = 9, p = 0.01$) among years (2006, 2009, and 2011), with mean cover increasing from 1% in 2006, to 2% in 2009, to 4% in 2011 (Fig. 10c).

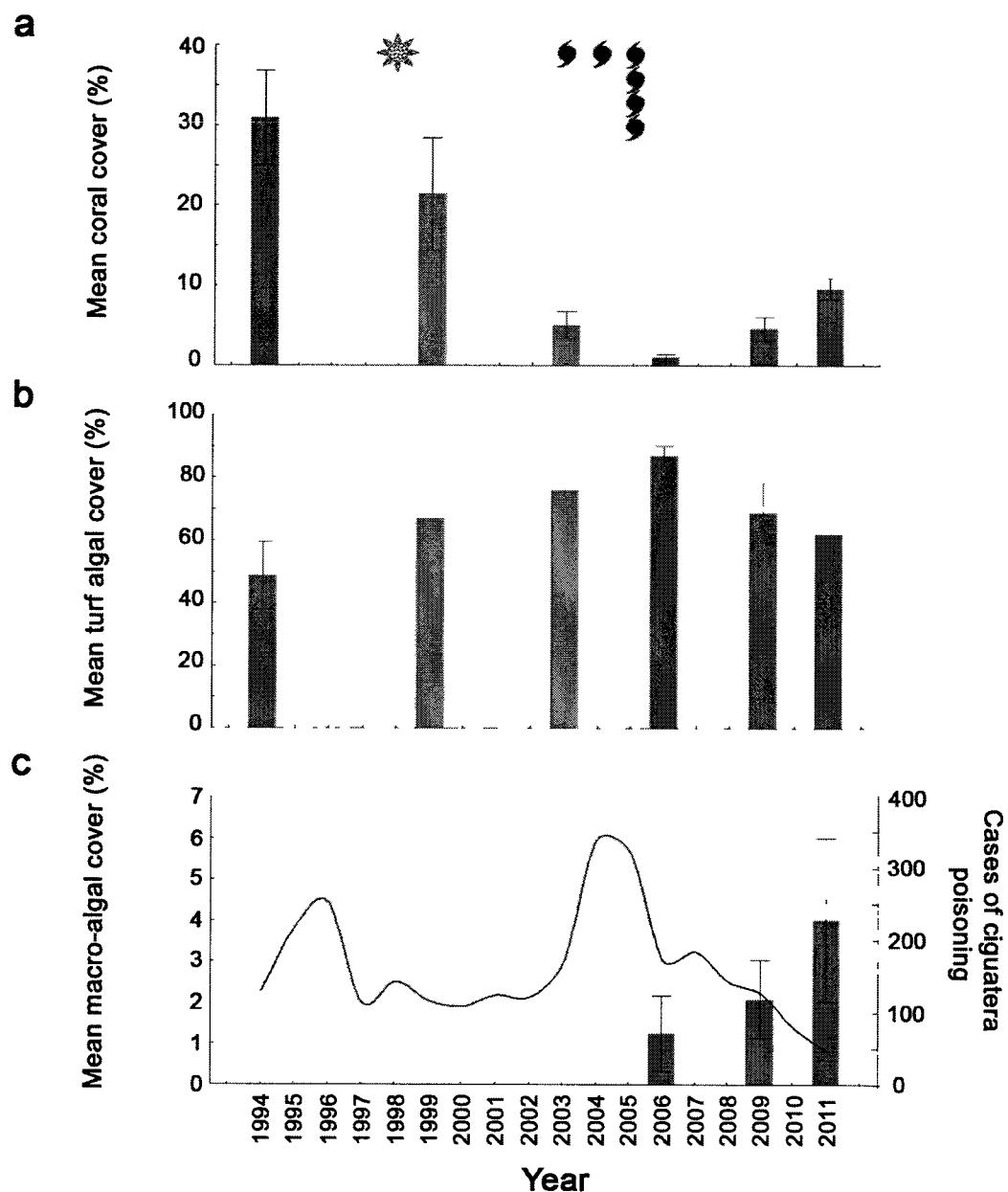


Figure 10. (a) Mean hard coral, (b) turf algal, and (c) macroalgal cover on fore reef sites in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2003 (Lyon, 2003), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011. Black line indicates hospital cases of ciguatera poisoning per year in Rarotonga from 1994 to 2011 (August). Shaded areas indicate the *Acanthaster planci* (star symbol) outbreak between 1995/96 and 2001, and major cyclones (Category 4 or 5; filled symbol) that impacted Rarotonga between 2003 and 2005. Error bars represent a 95% confidence interval.

Table 9. The mean percentage cover of hard corals, turf algae, and macroalgae (\pm standard error) at fore-reef sites surveyed in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2003 (Lyon 2003), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011. Friedman test was used to examine differences among years (2006, 2009, and 2011; grey shaded area). Asterisk (*) indicates significant difference at $p < 0.05$.

Year	Hard coral	Turf algae	Macroalgae
1994	31 \pm 3	49 \pm 5	
1999	21 \pm 3	66 \pm 3	
2003	5 \pm 1	76 \pm 1	
2006	1 \pm 0.2	87 \pm 2	1 \pm 0.5
2009	5 \pm 1	69 \pm 5	2 \pm 0.5
2011	8 \pm 1	62 \pm 4	4 \pm 1
<i>Friedman test</i>	$\chi^2 = 64, p < 0.001^*$	$\chi^2 = 55, p < 0.001^*$	$\chi^2 = 9, p = 0.01^*$

HERBIVOROUS FISHES

Acanthurid density was significantly different ($\chi^2 = 18, p < 0.001$) over time, with a mean density (fishes per 200 m²) of 133 in 2006, 73 in 2009, and 46 in 2011 (Fig. 11; Table 10). *Acanthurus nigrofasciatus* was consistently the most common acanthurid at all sites, whereas other acanthurids were recorded only occasionally, particularly schools of *A. triostegus*, *A. leucopareius*, and *A. olivaceus*. *Ctenochaetus striatus* density was also significantly different among years ($\chi^2 = 46, p < 0.001$), with a mean density (fishes per 200 m²) of 113 in 2006, 13 in 2009, and 4 in 2011. Scarid density did not differ among years ($\chi^2 = 0.1, p = 0.95$), with the mean density (fishes per 200 m²) of 17 in 2006, 15 in 2009, and 14 in 2011. Occasional schools of scarids were noted at some sites, particularly of *Chlorurus frontalis*, *C. sordidus*, *Scarus altipinnis*, *S. schlegeli*, and *S. psittacus*, but quite often schools consisted of several scarid species. In comparison with the

fish density in 1994 and 1999, acanthurid and scarid densities were less than the 2006, 2009, and 2011 surveys, except for *C. striatus* where the density was higher in 1999 than it was in the 2009 and 2011 surveys.

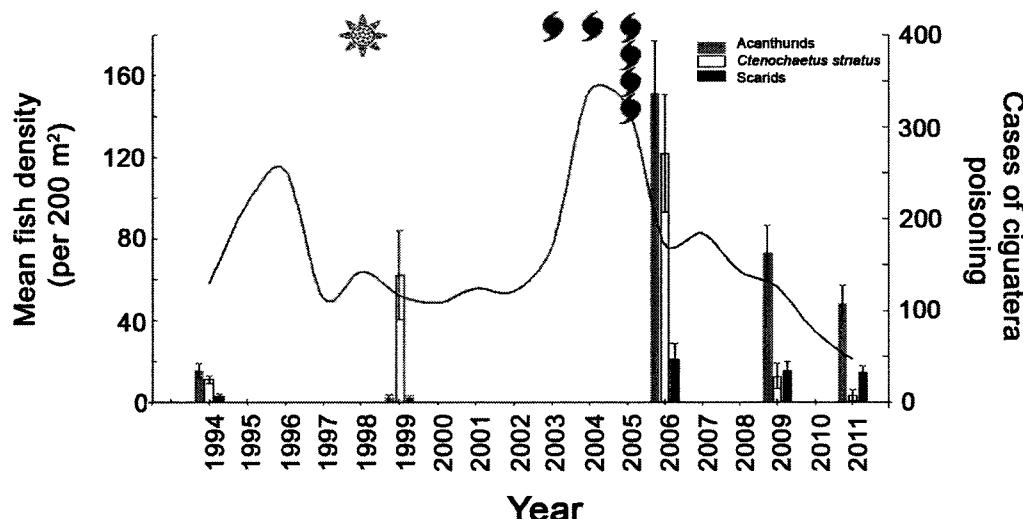


Figure 11. Hospital cases of ciguatera poisoning in Rarotonga from 1994 to 2011 (August), and mean fish density (per 200 m²) in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011. Shaded areas indicate the *Acanthaster planci* (star symbol) outbreak between 1995/96 and 2001, and major cyclones (Category 4 or 5; filled symbol) that impacted Rarotonga between 2003 and 2005. Error bars represent a 95% confidence interval.

Table 10. Mean density of acanthurids, *Ctenochaetus striatus*, and scarids (\pm standard error) at fore reef sites surveyed in 1994 (Miller et al. 1994), 1999 (Ponia et al. 1999), 2006 (Rongo et al. 2006), 2009 (Rongo et al. 2009b), and 2011. Friedman test was used to examine differences among years (2006, 2009, and 2011; grey shaded area). Asterisk (*) indicates significant difference at $p < 0.05$.

Year	Acanthurids	<i>Ctenochaetus striatus</i>	Scarids
1994	15 ± 2	11 ± 1	3 ± 1
1999	2 ± 1	62 ± 11	2 ± 1
2006	133 ± 14	113 ± 12	17 ± 3
2009	73 ± 7	13 ± 3	15 ± 2
2011	46 ± 5	4 ± 2	14 ± 2

Friedman test $\chi^2 = 18, p < 0.001^*$ $\chi^2 = 46, p < 0.001^*$ $\chi^2 = 0.1, p = 0.95$

DISCUSSION

Rarotonga has experienced several large-scale reef disturbances in the last 40 years. Although a minor outbreak of ciguatera poisoning occurred in the early 1970s, it was not until the 1990s when ciguatera poisoning became severe in Rarotonga (Rongo et al. 2009a; Rongo and van Woesik 2011). According to previous work in Rarotonga (Rongo and van Woesik 2011), the frequency of disturbance may have been pivotal in increasing the risk of ciguatera poisoning in recent years. Within a period of 15 years, from 1991 to 2005, Rarotonga has experienced, on average, one major disturbance per year. These disturbances included cyclone activity, *A. planci* outbreaks, and coral bleaching events. These disturbances were also important in influencing the trajectory of successional patterns of hard coral cover around Rarotonga. Coral cover was drastically reduced from 1994 to 2003, from 31% to 5%, which was primarily a consequence of an *A. planci* outbreak from 1995 to 2001, and coral bleaching events in 1991 and 1994. The passing of six major cyclones and a gale, between 2003 and 2005, may have caused a further decline in mean coral cover to 1% in 2006.

Although coral cover declined by 26% from 1994 to 2003, this decline may be an underestimate. The 1994 survey by Miller et al. was established to examine the effect of terrestrial run-off, and most survey sites were located near large channels and were therefore not representative of the reef in general. In the same year, Goreau and Hayes (1995) estimated that coral cover averaged around 30% on

the northwestern slopes, and over 90% on the southwestern slopes. Therefore, it is likely that the loss of corals, between 1994 and 2003, exceeded 26%. In addition, the extensive loss of corals from the *A. planci* outbreak made it difficult to determine the full extent of the damage that was caused by the six major cyclones, between 2003 and 2005, where only a 4% decline was noted in 2006. The impact of cyclones has been shown to reduce coral cover in several locations throughout the Pacific (van Woesik et al. 1991). In the Great Barrier Reef, extreme coral loss after a cyclone was reported at 70% between 1987 and 1990 (van Woesik et al. 1991; Halford et al. 2004). However since 2005, Rarotonga has not experienced any cyclone activity, and reefs in 2011 are recovering. While cyclones in the southern Cook Islands predominate during El Niño years (de Scally 2008), and El Niño events tend to be frequent during the positive phase of the Pacific Decadal Oscillation (PDO) (Verdon and Franks 2006), which is also the dominant climate cycle in this region (Linsley et al. 2000; Rongo et al. 2009a), the recent shift to the negative phase of the PDO suggests that cyclone frequency will likely decrease and reef recovery in Rarotonga will continue.

While the health of coral-reef ecosystems are often considered critical for understanding ciguatera poisoning, very few studies have quantitatively verified a link. Over two decades ago, Bagnis et al. (1988) showed that ciguatera poisoning followed several natural and anthropogenic disturbances on reefs. Bagnis et al. (1988) also suggested that incidence of ciguatera poisoning declined as coral cover increased. Similarly, cases of ciguatera poisoning increased when reefs around

Rarotonga were transitioning from a coral-dominated to an algal-dominated state between 1994 and 2000. The transition was mainly due to an *Acanthaster planci* infestation, which was preceded by cyclones and storms (Category ≤ 3) and two bleaching events between 1991 and 1994 (see Table 6, p. 50). In the last six years of my study (2006 - 2011), the decline in cases of ciguatera poisoning was congruent with increased coral cover. Still, I suggest that reef state is not a good indicator of the risk of ciguatera poisoning. For example, the $> 20\%$ coral cover noted in 1994 and 1999 was higher than the 8% noted in 2011, yet cases of ciguatera poisoning were higher during the former period. This study suggests that disturbances are perhaps more important than reef state when considering ciguatera poisoning on reefs that host ciguatoxic dinoflagellates. Yet, ciguatera poisoning is not a concern in all Pacific nations, and therefore more regional studies are necessary to thoroughly test the disturbance hypothesis, potentially examining ciguatera-poisoning dynamics inside and outside the doldrums – where cyclone activity differs.

This study showed that turf algal cover appeared to be declining congruently with the cases of ciguatera poisoning. But again, it is difficult to determine if turf algal cover had any direct influence on ciguatera poisoning because turf algal cover was still above 60%, suggesting that the potential to host ciguatoxic dinoflagellates still exists. The decline in turf algal cover since 2006 may be explained by the increased cover of other algae such as crustose coralline algae (Rongo et al. 2009b), as well as macroalgae that significantly increased on

the fore reef by 2011 (see Table 9) and were common at depths less than 5 m (T. Rongo, pers. obs.).

Often the most common algae on reefs are unpalatable to herbivorous fishes, and these unpalatable algae have been suggested to serve as reservoirs for toxic dinoflagellates (Cruz-Rivera and Villareal 2006). In Rarotonga, these unpalatable algae are primarily *Galaxaura* spp. (a calcified alga) and *Asparagopsis taxiformis* (a chemically-defended alga). However successional changes in host algae, following major disturbances (i.e., cyclones), can lead to an increase in palatable algae with the removal of unpalatable types, which in turn can increase the transfer of ciguatoxins into the food web through grazing by herbivorous fishes. Perhaps this scenario explains the peak in hospital cases of ciguatera poisoning in 2006, after the passing of the six major cyclones in Rarotonga between 2003 and 2005. In algal-dominated reefs that host ciguotoxic dinoflagellates, cyclone activity may be key drivers of ciguatera-poisoning outbreaks. In support, the absence of cyclone activity in the last six years in Rarotonga may have resulted in the increased cover of unpalatable algae, causing the decline in fish density as less food is available, leading to the reduction of ciguatoxin uptake into the food web, which subsequently contributed to the decline in the number of cases of ciguatera poisoning to the lowest that have been recorded in the last 20 years.

A meta-analysis of 17 independent studies, throughout coral-reef regions globally, showed an inconsistent response in the density of herbivorous reef fishes after the loss of corals (Wilson et al. 2006). However, other studies have noted an

increase in the density of herbivorous fishes in response to reef disturbance (e.g., Sheppard et al. 2002). This is consistent with the increased density of herbivores in this study, after the passing of six major cyclones between 2003 and 2005. The increase in fish density was evident in acanthurids, particularly *Ctenochaetus striatus*, a high-risk species in Rarotonga and infamous as a primary vector for ciguatoxin (Randall 1958; Yasumoto et al. 1971; Banner 1984; Lewis et al. 1994b; Lewis 2006), which coincided with the peak in cases of ciguatera poisoning. However, acanthurid density significantly declined in 2009 and onwards, coinciding with the decline in hospital cases. I suggest that these recruitment pulses that followed major disturbances will increase the probability of ciguatoxin transfer into the food web, especially when ciguotoxic dinoflagellates are known to be patchy and highly variable on reefs (Yasumoto et al. 1980; Lehane and Lewis, 2000). In addition, there is potentially a cascading societal effect, where the fear of ciguatera poisoning among residents in Rarotonga caused a decline in fishing activities (Solomona et al. 2009). This behavioral change in the society effectively generated a natural marine protected area that increased fish density, in turn increasing the potential for the transfer of toxins. A reduction in fishing pressure would have increased densities of larger fishes that contribute disproportionately more offspring to the gene pool, further boosting recruitment densities (e.g., Palumbi 2004). Indeed, a number of large pulse recruitments of *C. striatus* have been observed in the last 10 years in Rarotonga (T. Rongo, unpublished data).

Another factor that could increase toxin transfer is the adverse effect that ciguatoxins may have on reef fishes. Studies have shown that intoxicated fishes are affected behaviorally and morphologically (Lewis 1992a; Landsberg 1995; Edmunds et al. 1999; Ajuzie 2008), and are preferentially preyed upon (Lewis and Holmes 1993). This effect could increase the likelihood of toxins accumulating in higher trophic levels, also resulting in the increased diversity of intoxicated reef fishes. Rongo and van Woesik (2011) reported, in 2010, a total of 48 species of reef fishes, and four species of invertebrates that were implicated in ciguatera poisonings in Rarotonga, which is higher than those reported in other locations (e.g., 32 species in Tahiti, French Polynesia [Bagnis 1969], 10 species in Ryukyu, Japan [Hashimoto 1979], 16 species in Hawaii [Kodama and Hokama 1989], and 13 species in Australia [Lehane and Lewis 2000]). However, increased risk of ciguatera poisoning is unlikely in regions where overfishing is a problem and recruitment is compromised. This may explain the low incidence of ciguatera poisoning in some regions such as the Caribbean where overfishing of herbivorous fishes is a major problem.

Disturbances may also change the composition of dinoflagellates on reefs. If toxic dinoflagellates are opportunistic, then theoretically the frequency of disturbances may shift the dinoflagellate composition to favor toxic species. For example, Richlen and Lobel (2011) showed a positive correlation between water motion (i.e., reef exposure) and *Ostreopsis* spp. densities, whereas other species of dinoflagellates showed a negative correlation with reef exposure. It has been

suggested that in some regions, *Ostreopsis* spp. is the main dinoflagellate responsible for fish poisonings (Ballantine et al. 1985). In Rarotonga, there has been a noticeable increase in *Ostreopsis* spp. density, and a decrease in *Gambierdiscus toxicus* density since the early 1990s (see Rongo and van Woesik 2011). This shift in dinoflagellates may have been the result of the increased frequency of cyclones and other low pressure systems associated with El Niño years and the positive phase of the PDO. Perhaps examining factors that can influence water conditions (i.e., wind speed and wave height) in ciguatera-affected regions may be important for understanding the spatial and temporal distribution of ciguatera poisoning.

In summary, this study showed that coral cover, per se, is not a good predictor of ciguatera poisoning. Instead, I suggest that the decline in cases of ciguatera poisoning in Rarotonga was largely the result of a reduced frequency of disturbances, rather than a result of increased coral cover or decreased turf algal cover. However, I suggest that further work is necessary to understand the relationship between reef fishes transitioning between the lagoon and fore reef habitats. I found that a high-disturbance frequency preceded increased herbivorous fish density (in response to palatable algae that dominate during early successional stages), especially those fishes important in the transfer of ciguatoxins into the food web. Similarly, cases of ciguatera poisoning declined along with declines in herbivorous fish density that may be attributed to increases in unpalatable algae with decreased disturbance frequency (i.e., cyclones). With the recent shift in

climate oscillations to the negative phase of the PDO, coupled with La Niña years (where Rarotonga would experience less cyclones), not only will the effect of ciguatera decline but the reduced frequency of disturbances will also allow the recovery of reefs around Rarotonga.

CHAPTER V

SOCIOECONOMIC IMPACTS OF CIGUATERA POISONING IN RAROTONGA, SOUTHERN COOK ISLANDS

INTRODUCTION

The ocean and its resources have sustained island nations for millennia (Johannes 1997). Fish is the main source of protein, and most island communities consume well over 100 g per person per day (Lewis 1992b). Yet an exclusive reliance on fish can be disastrous for an island population when those resources become inedible, particularly from ciguatoxins that make fish poisonous (Lewis 1986; Rongo et al. 2009). This form of ichthyo-sarcotoxinism, referred to as ciguatera poisoning, occurs when reef fishes inadvertently ingest dinoflagellates that produce ciguatoxins. Ciguatera poisoning is characterized by a combination of gastrointestinal, neurological, and cardiovascular symptoms that can last from several weeks to several years (Lewis 2006). Although rarely fatal, ciguatera poisoning is the most common seafood poisoning worldwide, affecting around 50,000 to 500,000 people annually (Fleming et al. 1998). With increased globalization, ciguatera poisoning has become a concern well beyond the communities where the ciguotoxic fishes are caught (see van Dolah 2000; Wong et al. 2005; Dickey and Plakas 2010).

There are many theories that claim to explain outbreaks of ciguatera poisoning, including nutrient loading (Carlson 1984), natural and man-made destruction of reefs (Randall 1958; Cooper 1964; Banner 1976; Tebano 1992), loss of corals (Yasumoto et al. 1980; Kohler and Kohler 1992; Bagnis et al. 1992a), decadal climate oscillations (Rongo et al. 2009a), and elevated sea surface temperatures associated with global climate change (Tosteson et al. 1988; Hales et al. 1999; Chateau-Degat et al. 2005; Tester et al. 2010). Other environmental factors that influence the growth of ciguatoxic dinoflagellates include the affects of salinity (Bomber et al. 1988), where high densities of dinoflagellates were found in high-salinity waters, and low densities were found near areas of freshwater discharge (Yasumoto et al. 1980; Taylor 1985; Hokama and Yoshikawa-Ebesu 2001). Anderson et al. (1983) also suggested that high salinity, during periods of low rainfall, explained the prevalence of ciguatera poisoning on the leeward reefs of Hawaii.

Beyond the health issues, ciguatera poisoning has also had socioeconomic impacts, including: 1) loss of a food source, 2) loss of labor productivity, 3) increased health-related costs (Lewis 1986; Hajkowicz 2006), 4) loss of tourism (Lewis 1992b), 5) loss of foreign-exchange opportunities through fish exports (Lewis 1983), and 6) depopulation (Banner and Helfrich 1964; Cooper 1964; Rongo et al. 2009a). However, research into the socioeconomic impacts of ciguatera poisoning and other harmful algal blooms on human health are still in the early stages, and there is a need to quantify these impacts (Kite-Powell et al. 2008).

Ciguatera poisoning has caused impacts to the economy of both developed and developing nations. For example, in U.S. tropical jurisdictions alone, the impact of ciguatera poisoning has been estimated at USD \$19 million annually (Hoagland et al. 2002). In addition, the impacts extend beyond the ciguatera-affected regions. For example, in Canada, medical costs associated with an average of 300 cases per year between the 1960s and the 1980s due to ciguatera poisoning from tourism and food importation was estimated around CAD \$2.7 million (Todd 1985). Bagnis et al. (1992b) estimated that the economic loss associated with banned reef-fish sales and lost-labor productivity in Tahiti, French Polynesia, was over US \$2 million per year. In Kiribati, Yeeting (2009b) estimated that the closure of export fisheries to Hong Kong resulted in an annual revenue loss of AUD \$250,000 (~AUD \$8,000 per fishermen). Yet, the economic impact of ciguatera poisoning in Pacific-island countries is poorly understood, largely because limited financial resources have been allocated to examine the problem (Dalzell 1993). For this reason, Dalzell (1993) suggested that management of ciguatera poisoning should be directed at industries that generate revenue, such as tourism and export fisheries. It is critical that socioeconomic studies are conducted on ciguatera poisoning to determine the extent of economic loss, and what actions may mitigate the problem (Lewis 1992b; Hoagland and Scatasta 2006).

Although extensive ciguatera poisoning leads to dietary shifts (Lewis 1983), the nature of the alternative protein source will depend on the economic status of the islands involved. More-developed communities would, theoretically, undergo

dietary shifts more easily than less-developed communities (Lewis 1992b; Chateau-Degat et al. 2007). Developed communities, with ties to developed countries, tend to show shifts toward frozen meats such as chicken, beef, and lamb, whereas less developed communities tend to shift toward canned foods such as corned beef, with high fat content, in part because freezing facilities are limited (Lewis 1983; Pinca et al. 2007). The shift away from a fresh-fish diet appears to have also contributed to the increased prevalence of non-communicable diseases among Pacific-island populations (Lewis and Ruff 1993; Li et al. 1994).

In a previous study of resource use in Rarotonga, Solomona et al. (2009) found that ciguatera poisoning caused a decline in seafood consumption from 318 g (per capita per day) in 1989, to 271 g in 2001. Subsequently, similar surveys conducted in 2006 (Moore 2006), and in 2007 (Pinca et al. 2007), found that seafood consumption in Rarotonga continued to decline through the 2000s, and were reported at 176 g in 2006, and at 122 g per capita per day in 2007. In 2011, I conducted a survey to determine how protein-resource use has changed in Rarotonga (see Fig. 1, p. 16) over two decades of ciguatera poisoning, and whether decades of adjustment to the problem may have caused a shift away from a subsistence fishing lifestyle.

I employed the *cost-savings-and-avoidance* valuation technique (Hajkowicz 2006) to estimate the direct loss in value of marketable goods and services that were caused by ciguatera poisoning in Rarotonga. Previously, Hajkowicz (2006) estimated that from 1989 to 2001, the impact of ciguatera poisoning resulted in an

annual loss of revenue of (New Zealand Dollars, NZD) \$534,000. However, this estimate was based on an assumption that 50% of the loss was attributed to watershed degradation. In addition, the Hajkowicz's study provided 'cost scenarios' to give policy makers some indication of the problem in Rarotonga. In this study, I estimated the economic impact of ciguatera poisoning in 2011. In addition, I estimated the economic consequence of climate oscillations, in particular the El Niño Southern Oscillation (ENSO), on ciguatera poisoning, which have been linked to ciguatera poisoning in Rarotonga (Rongo and van Woesik 2011).

MATERIALS AND METHODS

I compared current food consumption and subsistence fishing in Rarotonga with previous studies by: (i) Solomona et al. (2009), who surveyed 100 households in 1989 and 2001, (ii) Moore (2006), who surveyed 90 households in 2006, and (iii) Pinca et al. (2007), who surveyed 59 households in 2007. Using a questionnaire survey (Appendix B) adapted from Moore (2006) (see also Zann and Aleta 1984), I randomly surveyed 179 households (6% of the most recent census; Cook Islands Statistics Office, 2006), between March and May 2011. Data that were collected included: (i) the average annual per-capita protein (seafood and meat) consumption, (ii) the frequency of subsistence fishing per week, and (iii) the socioeconomic impacts of ciguatera poisoning. Seafood items included fresh fish (i.e., reef, pelagic, and freshwater), marine invertebrates, and purchased imported canned fish. Meats included pork (including sausages and bacon), chicken, beef (e.g., steak and mince), lamb, and canned corn beef.

PROTEIN CONSUMPTION

Daily consumption of seafood (i.e., fresh fish, canned fish, and other seafood) per household and meat (i.e., chicken, other meat, corned beef, lamb, and pork) was determined from the weight (kg) of food items consumed per household per week. The per-capita consumption rate (g/person/day) was based on the number of individuals in the household (Zann et al. 1984). The annual consumption rate, in

metric tonnes/year, was calculated for each food item using the average annual per-capita consumption rate, which was multiplied by the total population of Rarotonga (13,890, for the 2006 census). Subsequently, the consumption rate of each protein category was compared with the rates that were estimated in 1989, 2001, 2006, and 2007.

Households were queried on the fishes that were consumed the day before the survey. The information allowed comparisons of reef fishes with pelagic and freshwater fishes, which were expressed as reef-pelagic-freshwater ratios. These ratios were used to determine the per-capita consumption of potentially ciguotoxic fishes as a proportion of the per-capita consumption rate of fresh fish.

COST SAVINGS AND AVOIDANCE ANALYSIS

The *cost savings and avoidance* technique was used to estimate the current economic impacts of ciguatera poisoning in Rarotonga (see Hajkowicz 2006 for detailed technique description). This technique essentially involves estimating the avoided revenue loss and costs in the absence of ciguatera poisoning. Estimates were limited to the direct market cost of goods (e.g., fishes) and services (e.g., hospital treatment and lost-labor productivity). Three categories were used in my analysis: 1) the gross value of reef-fish-stock loss, 2) the costs of monitoring and management, and 3) the health-related costs. The sum of these categories determined the total economic impact of ciguatera poisoning on the society.

GROSS VALUE OF FISH-STOCK LOSS. The per-capita consumption rates of fresh fish, which were determined by Solomona et al. (2009) in 1989, were considered ‘normal’ in this study, because ciguatera poisoning increased soon thereafter (Rongo and van Woesik 2011). The rates estimated in subsequent years were compared with the 1989 rate, to determine the fish-stock loss (in kg), but only potentially ciguatoxic reef fishes were included (see Section 3.1). The economic loss (in NZD \$) was determined as the product of fish-stock loss and the average market price of the reef fish. Because market prices did not change much since 1989, I used the same price of NZD \$7.80/kg that Hajkowicz (2006) used in his 2001 estimate. The total kilograms of potentially ciguatoxic fish consumed by the population were determined using the per-capita-consumption rate (converted to kilograms per person per year) multiplied by the total human population for each study year. For 1989, Hajkowicz used the 2001 total population of 12,188; however, the present study recalculated his estimates using the 1986 population of 9,826, which was a more accurate estimate of the population. For 2001 and 2011, the total population of 12,188 and 13,890 (i.e., based on the 2006 census) were used respectively.

MONITORING AND MANAGEMENT COSTS. The Ministry of Marine Resources in Rarotonga carries out monthly monitoring of potentially toxic dinoflagellates (e.g., *Gambierdiscus* spp. and *Ostreopsis* spp.) and monthly water-quality sampling at 15 lagoon sites around Rarotonga (see Anderson et al. 2004).

Costs associated with monitoring include staff time and materials for water quality and dinoflagellate analysis (T. Turua, pers. comm.). Public education and awareness, through radio and television, are regularly conducted by the Ministry of Health, and the costs of these media-outreach programs were estimated based on personal communications.

HEALTH-RELATED COSTS. Individual health-related costs were determined by summing the hospital and staff time involved in treatment, the price of pharmaceuticals, and lost-labor productivity. Hospital admittance for ciguatera poisoning varies from one day to six months, however, most patients are discharged after 5 days (T. Iorangi, pers. comm.); hospital fees were valued at NZD \$200 per day (see Hajkowicz and Okotai 2005), and staff fees for doctors and nurses (\$25/h at 1 hr, and \$14/h at 4 h respectively; M. Aunguna, pers. comm.) were estimated at NZD \$60 per day. Treatment costs in Rarotonga were based on commonly used medication to alleviate ciguatera-poisoning symptoms, which included intravenous infusion of saline solution and other medication (e.g., Phenergan, charcoal tablets, Paracetamol, anti-itch cream). Medication costs were obtained by consultation with local pharmacists (A.Y. Enjoy and S. Saunders, pers. comm.). The cost of lost-labor productivity, per person, was calculated as the product of the average hourly rate of NZD \$7.56 (obtained from Cook Islands Statistics Office, 2006) and the number of work hours lost because of hospitalization and recovery at home (10 days at 8 h/day).

Hospital cases of ciguatera poisoning (obtained from the Cook Islands Ministry of Health) were grouped into three, 5-year periods: 1997 – 2001, 2002 – 2006, and 2007 – 2011. These periods allowed calculations of the average status of ciguatera poisoning during (i) 2001 (Solomona et al. 2009), (ii) 2006 (Moore 2006), and (iii) 2011 (i.e., the present study). The health-related costs of reported cases of ciguatera poisoning were determined as the product of the total cost per individual and the average cases of ciguatera poisoning for each period. Because reported cases only represent around 34% of actual incidence in Rarotonga (Rongo and van Woesik 2011), the number of average unreported cases, for each period, was estimated at 66%. Health-related costs of unreported cases was determined as the product of the average unreported cases and the lost-labor productivity (\$605 per individual) only, because hospitalization is rare, and most unreported cases go untreated, or victims used traditional remedies to alleviate the symptoms (Rongo and van Woesik 2011).

DATA ANALYSIS

Results from the Chi-square test conducted in Chapter III (see Table 8, p. 54) between cases of ciguatera poisoning and the positive and negative phases of ENSO were used when determining the economic impact of ciguatera poisoning from climate oscillations.

RESULTS

PROTEIN CONSUMPTION

Household protein consumption was based on the amount of protein consumed in the week before the survey. In 2011, Rarotongans rely on chicken as their main source of protein. One hundred and sixty households had an average per capita consumption rate of 115.8 g of chicken per day (a 37% contribution to the total protein intake), or 587 metric tonnes of chicken per year, for a population of 13,890 (Table 11). Fresh fish was also an important source of protein. Fish was consumed by 132 households, at 102.7 g per day (33% of protein intake), which equates to 521 metric tonnes of fresh fish per year for the population. Other meats, which included minced beef and various sausages, contributed 14% to the total protein intake, with canned fish, corned beef, lamb, and pork contributing 6%, 4%, 3%, and 2%, respectively. Other seafood (i.e., marine invertebrates) had the lowest contribution at < 1%. In comparison with previous survey years, the percentage of meat items (not including seafood) from the total protein consumed has been increasing since 1989. Meat consumption was 36% of the total protein in 1989, increasing to 49% in 2001, and drastically increasing to 72% in 2006, but declined to 60% in 2011 (Table 12).

Table 11. Protein consumption of Rarotonga residents based on a questionnaire survey conducted in 2011, where *n* represents the number of households that consumed each food the week before the survey. Data on the average per-capita consumption rate* (g/per person/day), the annual consumption (metric tonnes per year), and the percent contribution for each food item to the total protein intake were included.

Food	<i>n</i>	Average per-capita rate (g/person/day)	Annual consumption (Mt/year)	Percent contribution (%)
Chicken	160	115.8	587	37
Fresh fish	132	102.7	521	33
Other meat	77	44.8	227	14
Canned fish	99	18.9	96	6
Corned beef	61	12.2	62	4
Lamb	26	8.3	42	3
Pork	22	6.9	35	2
Other seafood	6	0.6	3	< 1

**Per-capita calculations*: the daily weight (g) of protein consumed by a household was divided by the number of individuals per household. Annual consumption is the product of the average per-capita rate and the total population of 13,890 from the 2006 census (Cook Islands Statistics, 2006).

Table 12. Percentage (%) of meat (e.g., chicken, other meat, corned beef, lamb, and pork) and seafood (fresh fish, canned fish, and other seafood) consumed by households in 1989 & 2001 (Solomona et al. 2009), 2006 (Moore 2006), and 2011 (the present study).

Protein consumption	1989	2001	2006	2011
Meat	36	49	72	60
Seafood	64	51	28	40

The average per-capita seafood consumption (i.e., fresh fish, canned fish, and other seafood) in Rarotonga generally declined between 1989 and 2011. The most marked decrease in the consumption of seafood was apparent between 2001 and 2006 (Table 13). Although fresh fish consumption increased slightly from 1989 to 2001, there was a decrease from ~167 g in 2001 to ~75 g in 2006, with a slight increase after 2007. In contrast, canned fish consumption almost doubled between 2001 and 2006, but returned to 1989 levels after 2007. These major changes in

fresh fish and canned fish consumption coincided with a peak in cases of ciguatera poisoning at the Rarotonga Hospital (Fig. 12).

Table 13. Average per-capita seafood consumption (g/person/day) for 1989 and 2001 (Solomona et al. 2009), 2006 (Moore 2006), 2007 (Pinca et al. 2007), and 2011.

Seafood (g/person/day)	1989	2001	2006	2007	2011
Fresh fish	148.9	167.1	74.5	87.7	102.7
Canned fish	18.4	17.9	30.7	30.1	18.9
Other seafood	150.4	85.7	70.9	3.8	0.6
Total seafood consumption	317.7	270.7	176.1	121.6	122.2

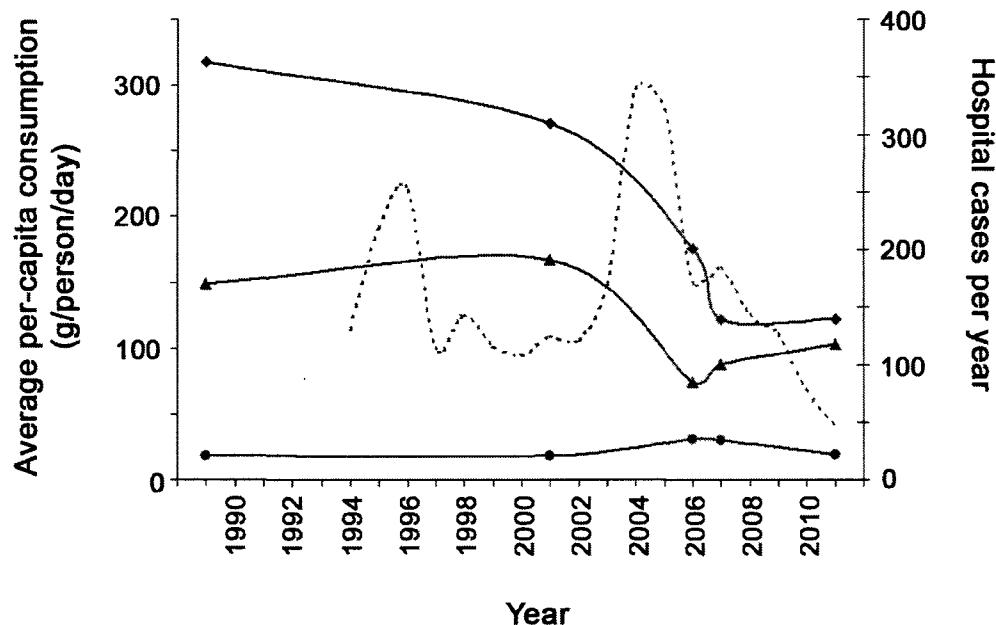


Figure 12. Average per-capita consumption (g/person/day) of total seafood (diamond), fresh fish (triangle) and canned fish (circle) from 1989 to 2011, and cases of ciguatera poisoning reported at the Rarotonga Hospital (Cook Islands Ministry of Health) (dashed line). 1989 and 2001 data were taken from Solomona et al. (2009), 2006 from Moore (2006), 2007 from Pinca et al. (2007), and 2011 from the present study. Shaded area indicates the period of major changes in seafood consumption in Rarotonga.

In 2011, fresh fish was consumed around 2.6 days per week by 132 households. Seventy households consumed fresh fish the day before the survey, with pelagic fishes (primarily tuna and flying fish) comprising 53% of the fresh fish consumed (Table 14). The percentage of reef fishes consumed was 46%, with 25% consisting of potentially ciguatoxic species; only 1% was freshwater fishes (i.e., tilapia). In 1989, more reef fishes were consumed than pelagic fishes (63% vs. 27%). However, this percentage shifted in 2001, with the consumption of pelagic fishes increasing to 70%, whereas reef fishes declined to 30% (Fig. 13). The amount of pelagic fishes consumed was only slightly higher than reef fishes consumed in 2006 (55% vs. 45%) and 2011 (53% vs. 46%), which differs from the 2001 ratio (of 70% to 30%).

Table 14. The percentage of reef, pelagic, and freshwater fishes consumed by 70 households the day before the survey in 2011. * indicates potentially ciguatoxic fishes in Rarotonga verified using Rongo and van Woesik (2011).

Reef fish	Percent (%)	Pelagic fish	Percent (%)	Freshwater fish	Percent (%)
Squirrelfish (ku)	9	Tuna (a`ai)	23	Tilapia	1
Moontail bullseye (ku pa)	6	Flying fish (maroro)	18		
Parrotfish (pakati)*	5	Bigeye scad (ature)	4		
Unicornfish (ume)*	4	Mahimahi (ma`ima`i)	4		
Parrotfish (u'u)*	4	Wahoo (paara)	2		
Snake mackerel (manga)*	4	Broadbill (akura)	1		
Small grouper (patuki)	3	Marlin (akura)	1		
Mullet (kanae)*	3				
Goatfish (ka`uru)*	3				
Trevally-juvenile (rupo)*	1				
Rudderfish (pipi)	1				
Rabbitfish (morava)	1				
Black trevally (rui)*	1				
Pacific threadfin (moi)	1				
Total	46			53	1
Potentially ciguatoxic fishes*	25				

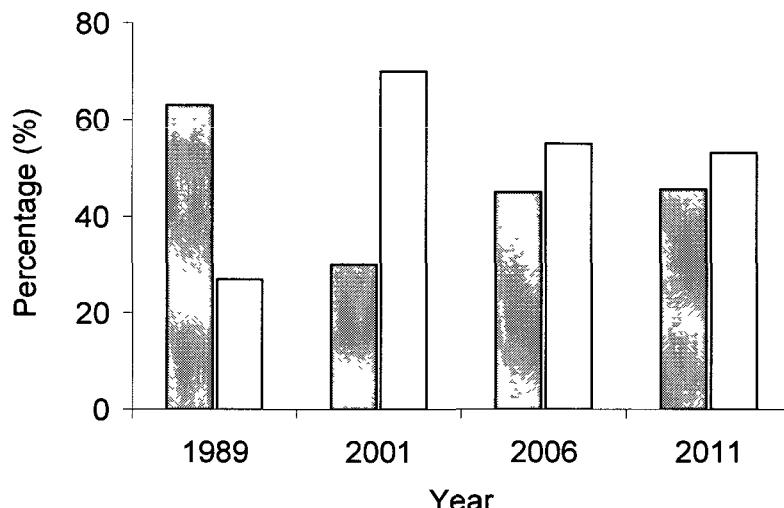


Figure 13. Percentage of reef (grey bars) and pelagic (open bars) fishes consumed by households in 1989 & 2001 (Solomona et al. 2009), 2006 (Moore 2006), and 2011 (the present study).

FISHING FREQUENCY

In 1989 and 2001, 14 and 17 households (out of 100) practiced subsistence fishing 3.4 and 2.2 days per week, respectively. In 2011, 32 households (out of 179; 21%) practiced subsistence fishing the week before the survey, with an average frequency of 2.7 days of fishing per week. Based on 55 households, 27% caught their own fish, whereas 40% purchased their fish, and 33% received fish from family members, primarily from the outer islands.

COST SAVINGS AND AVOIDANCE ESTIMATES

GROSS VALUE OF REEF-FISH-STOCK LOSS. In 2011, the per-capita consumption of potentially ciguatoxic reef fishes was ~25 g per day, or 125,225 kg per year for the total population (13,890 in 2006) (Table 15). When compared with

the ‘normal’ consumption rate of ~51 g per day in 1989, rates in 2001, 2006, and 2011 were more than two-fold lower, with the lowest rates occurring in 2006, when ciguatera poisoning peaked in Rarotonga (see Fig. 12). Recalculating Hajkowicz’s (2006) estimates of the kilograms of potentially ciguatoxic reef fish consumed in 1989, using the 1986 population instead of the 2001, the value was 184,346 kg/year instead of 228,659 kg/year. The difference between the 1989 recalculated value and kilograms consumed in subsequent study years, multiplied by the market value of reef fish (NZD \$7.80/kg), indicated that the gross value of lost reef-fish stock, because of ciguatera poisoning, was \$723,091 in 2001, \$702,367 in 2006, and \$461,144 in 2011.

Table 15. Consumption of potentially ciguatoxic reef fishes (kg/year) in Rarotonga from 1989 to 2011. Per-capita consumption (g/person/day) in 1989 and 2001 taken from Hajkowicz (2006), 2006 from Moore (2006), and 2011 from the present study. * indicates the corrected total population (1986 population census; Cook Islands Statistics Office, 2006) used to recalculate the 1989 estimate.

Potentially ciguatoxic reef fishes	1989	1989	2001	2006	2011
Per-capita consumption (g/person/day)	51.4	51.4	20.6	18.6	24.7
Total population of Rarotonga	12,188	*9,826	12,188	13,890	13,890
Kilograms per year	228,659	184,346	91,642	94,299	125,225

MONITORING AND MANAGEMENT COSTS. Costs associated with dinoflagellate and water-quality monitoring (including staff time) is approximately NZD \$17,100 per year (T. Turua, Ministry of Marine Resources, pers. comm.). Public education and awareness through radio and television were regularly conducted by the Ministry of Health (costing ~NZD \$1,000/year) when cases of

ciguatera poisoning at the Rarotonga Hospital peaked in the early to mid-2000s, however, in the last two years, only ~NZD \$100/yr has been spent on four radio advertisements per year (\$25 each) in Rarotonga because the problem of ciguatera poisoning has been declining (H. Tangimetua, pers. comm.).

HEALTH-RELATED COSTS. The total health-related cost for an individual with ciguatera poisoning was estimated at \$2,090 (Table 16). Using the health-related cost for an individual hospitalized with ciguatera poisoning, the costs that could have been avoided in the absence of ciguatera poisoning were \$252,890 in 2001, \$468,160 in 2006, and \$242,440 in 2011 (Table 17). Monitoring and management costs were estimated at \$18,600 in 2001 and 2006, and at \$17,300 in 2011. Considering that reported cases of ciguatera poisoning only represent 34% of the actual cases in Rarotonga (Rongo and van Woesik 2011), we estimated that health-related costs of the 66% of unreported cases were \$142,104 in 2001, \$263,068 in 2006, and \$136,232 in 2011 (see Table 17). The total economic impact of ciguatera poisoning (NZD/year) in Rarotonga, using the sum of the estimated gross value of fish-stock lost, monitoring and management costs, and health-related costs, was ~\$1.1 million in 2001, ~\$1.5 million in 2006, and ~\$857,000 in 2011 (see Table 17).

Table 16. Health-related costs per individual with ciguatera poisoning.

	Price per unit (NZD)	Quantity	Total (NZD)
<i>Hospital & staff time</i>			
Hospital fee (per day)	\$200.00	5	\$1000.00
Staff fees (per day)	\$81.00	5	\$405.00
<i>Pharmaceutical treatments</i>			
Intravenous infusion of saline	\$12.00	2	\$24.00
Other medication	\$56.00	1	\$56.00
<i>Lost labor productivity</i>			
Lost wages (\$7.56/h at 8 h/day)	\$60.50	10	\$605.00
Total cost per individual			\$2,090.00

Table 17. Total economic impact (NZD per year) of ciguatera poisoning in Rarotonga for 2001, 2006, and 2011. The average cases of ciguatera poisoning were based on three periods that corresponded with food consumption study years: 2001 (1997 - 2001), 2006 (2002 – 2006), 2007 and 2011 (2007 – 2011). The total economic impact was the sum of the gross value of fish-stock loss, monitoring and management costs, and health-related cost of reported and unreported cases (NZD per year). Unreported cases of ciguatera poisoning were determined at 66% based on Rongo and van Woesik's (2011) 34% reporting, and health-related costs only using lost-labor productivity estimates.

Study year	Average cases of ciguatera poisoning	Gross value of fish stock loss	Monitoring and management costs	Health-related costs (reported cases)	Health-related costs (unreported cases)	Total economic impact (NZD/year)
2001	121	\$723,091	\$18,100	\$252,890	\$142,104	\$1,136,185
2006	224	\$702,367	\$18,100	\$468,160	\$263,068	\$1,451,695
2011	116	\$461,144	\$17,200	\$242,440	\$136,232	\$857,016

CONTRIBUTION OF CLIMATE TO CIGUATERA POISONING. In Chapter III, it was determined that cases of ciguatera poisoning were significantly higher during El Niño years compared with La Niña years ($\chi^2_1 = 617, p < 0.001$), using a one-year lag period between cases and climate data (1993 – 2010) (see Table 8, p. 54).

Using similar calculations carried out to determine the economic impact of ciguatera poisoning for the three different periods, in 2001, 2006, and 2011 (see Table 17), the average economic impact was determined for the different climate phases of ENSO (Table 18). The reef fish stock loss, and monitoring and management costs for 2006 (when average cases of ciguatera poisoning were the highest), were used for estimates during El Niño years, whereas the 2011 costs (when average cases of ciguatera poisoning were the lowest) were used for La Niña years. The average annual economic impact of ciguatera poisoning during El Niño years was estimated at ~NZD \$1.4 million, while the impact during La Niña years was estimated at ~NZD \$843,000.

Table 18. Average annual economic impact of ciguatera poisoning at different climate phases of ENSO. Reef fish stock loss and monitoring & management costs for 2006 were used for El Niño years, while costs for 2011 were used for La Niña years; each cost was multiplied by the number of years within each climate phase. Health-related costs for reported cases were estimated by multiplying the observed cases within each climate phase (between 1994 and 2011) by individual costs (\$2,090). Unreported cases were determined at 66% based on Rongo and van Woesik's (2011) 34% reporting, and health-related costs only used lost-labor productivity estimates (\$605 per individual). The average annual economic impact was calculated by dividing the total estimate for each climate phase by the number of years within each phase.

Climate phase (# of years in phase)	Observed cases of ciguatera poisoning	Reef fish stock loss	Monitoring & management costs	Health-related costs (reported cases)	Health-related costs (unreported cases)	Total estimate for climate phase	Average annual economic impact
El Niño (11)	2122	\$7,726,037	\$199,100	\$4,434,980	\$2,492,102	\$14,852,219	\$1,350,202
La Niña (7)	783	\$3,228,008	\$120,400	\$1,636,470	\$919,564	\$5,904,442	\$843,492

DISCUSSION

Imported meats, especially chicken, were the primary source of protein in Rarotonga in 2011. Over 580 metric tonnes of chicken were consumed per year by a population of 13,890, which is equivalent to approximately 100 shipping containers of chicken per year. My estimate is ~92% of the amount of chicken that was actually imported into the Cook Islands (637 metric tonnes) in 2009 (Food and Agriculture Organization 2011). My estimate therefore seems reasonable, considering that 67% of the Cook Islands population resides in Rarotonga (the most developed island in the Cook Islands).

A shift away from a fresh fish diet in the last 20 years may be in part a consequence of modernization. However, ciguatera poisoning may also explain a large proportion of the shift in meat consumption (Rongo et al. 2009; Rongo and van Woesik 2011). The decline in seafood consumption from 1989 to 2007, by 62%, coincided with the increased incidence of ciguatera poisoning throughout the 1990s and 2000s. Notably, the contribution of meat to total protein consumption increased sharply from 49% in 2001 to 72% in 2006, a period when hospital cases of ciguatera poisoning peaked. In addition, canned-fish consumption increased by 72% between 2001 and 2006. Canned fish became the alternative, safer, fish source, and were also cheaper than pelagic fishes (~NZD \$4.50 per can [425 g] or NZD \$10.60 per kg, compared with pelagic fishes that were priced from NZD \$15 – \$23 per kg).

Adjustment to ciguatera poisoning was also evident in the ratio of reef to pelagic fishes consumed. In 1989, 63% of fresh fish consumed were reef fishes, and 27% were pelagic fishes (ratio of 2.3 : 1). In 2001, this ratio declined to 0.4 : 1. Despite their high cost, pelagic fishes have remained the main fresh fish consumed since 2001. Reef-fish consumption increased by 15% in 2006, and has remained stable up to 2011. Rongo and van Woesik (2011) noted that the increase in reef-fish consumption was the result of residents (> 30 years old) learning that certain reef fish were ‘safe’ to eat. Alternatively, changes in reef-fish consumption appear to be a social response to changes in the severity of ciguatera poisoning. Once an individual is poisoned, the species and location of the intoxicated fish spreads through social networks (i.e., the ‘coconut wireless’, a vernacular phrase used in Rarotonga to describe communication by word-of-mouth), and the information is quickly disseminated to the wider community. In ciguatera-affected islands, especially where the media is ineffective, the ‘coconut wireless’ is the most efficient form of managing ciguatera poisoning.

The impact of ciguatera poisoning in Rarotonga clearly extends beyond the individual. I showed that when ciguatera poisoning is severe, such as the period between 2004 and 2006, the economic impact can amount to ~NZD \$1.5 million, which equates to ~30% of the country’s annual export revenue from pearls and pelagic fisheries of \$4.9 million. To put this amount into context, the economic impact of ciguatera poisoning on this small island is more than the highest estimates for Florida (~NZD \$933,000), Virgin Islands (~NZD \$509,000), Guam

(~NZD \$423,000), and American Samoa (~NZD \$264,000) between 1987 and 1992 (Hoagland et al. 2002). However, as Hoagland and colleagues had limited information on the ratio of reported to unreported cases, their estimates were subjective, and comparisons between my results and their results should be treated somewhat cautiously.

The loss of reef-fish stock by ciguatera poisoning, which would have either provided consumable protein for subsistence fishing families or generated revenue for artisanal fishers, had the greatest economic impact on Rarotonga. My estimates showed that loss of reef-fish stock can range from ~NZD \$461,000 to \$723,000 per annum. Hajkowicz (2006) indicated that in 2005, the fear of ciguatera poisoning among Rarotonga residents resulted in 71% of the population entirely avoiding the consumption of reef fish. This estimate was consistent with hospital cases of ciguatera poisoning that peaked around that same period. In contrast, Rongo and van Woesik (2011) indicated that in 2010, 88% of residents were cautiously including reef fishes into their diet. This period coincided with a decline in hospital cases that were related to ciguatera poisoning.

The link between ciguatera poisoning and human-induced causes is complex and poorly understood. However, land-based activities in Rarotonga such as 1) nutrient-loading from poor sewage systems and animal farms, and 2) increased runoff of soil erosion from hillside development, have increased in the last 20 years. According to Anderson et al. (2004), nutrient levels in the lagoon of Rarotonga are well above World Health Organization standards. The result of these

poor land-use practices can potentially lead to increased ciguatera poisoning (Carlson 1984; Yasumoto et al. 1984; Durand-Clement 1987). For example, Carlson (1984) found that *Gambierdiscus toxicus* densities were positively correlated with all the nutrient parameters measured (i.e., nitrite, nitrate, ammonia, phosphate, and total Phosphorus). In addition, soil extracts were found to stimulate the growth of *G. toxicus* (Carlson et al. 1984; Yasumoto et al. 1984; Durand-Clement 1987). Although both nutrient and soil runoff are influenced by precipitation, the effect of high precipitation, through lowering ocean salinity, has been shown to have adverse effects on ciguatoxic dinoflagellates (Yasumoto et al. 1980; Taylor 1985; Hokama and Yoshikawa-Ebesu 2001). Human activities may indeed have played a role in causing ciguatera poisoning in Rarotonga when climate conditions were favorable (i.e., positive phase of the PDO coupled with El Niño years) (Rongo and van Woesik 2011).

The influence of ENSO and PDO appear to have a significant influence on ciguatera poisoning in Rarotonga. Cases of ciguatera poisoning were significantly higher during El Niño years, which are frequent during the positive phase of the PDO. Explanations for this link may include factors such as a higher frequency of cyclones (de Scally 2008), and low precipitation (Baldi et al. 2009), which have been shown to influence the colonization and growth of ciguatoxic dinoflagellates (Yasumoto et al. 1980; Taylor 1985; Hokama and Yoshikawa-Ebesu 2001).

Alternatively, La Niña years (i.e., lower cyclone frequency and higher precipitation) have adverse effects on ciguatoxic dinoflagellate colonization and

growth. In this study, I estimated that the average annual economic impact of ciguatera poisoning during El Niño years was ~NZD \$1.4 million, while the impact during La Niña years was estimated at ~NZD \$843,000.

According to Rongo et al. (2009a), the recent shift into the negative phase of the PDO, coupled with frequent La Niña activity, will likely reduce the effect of ciguatera poisoning. This shift was realized in 2010 (Rongo and van Woesik 2011). In 2011, hospital records showed a decline in ciguatera-poisoning cases to the lowest records in 20 years. However, human activities involved in land-use change that most likely contributed to ciguatera poisonings, may compromise the integrity of reefs around Rarotonga that are already showing signs of recovery (Rongo et al. 2009b). Notably, the shift to the negative phase of the PDO coupled with La Niña events will increase precipitation, which will increase terrestrial runoff, nutrients, and sedimentation (e.g., Rogers 1990; Lapointe 1997). In addition, as reef fish return to the menu of Rarotongan residents, the potential for overfishing may further compromise reef health.

Whether reef-fish consumption in Rarotonga will return to consumption rates that were reported in 1989 may depend on not only reductions in ciguatera poisoning but also on lifestyle changes. Two decades of ciguatera poisoning in Rarotonga may have eroded traditional and cultural values that were closely tied to a basic subsistence fishing lifestyle (Johannes 1990). Modernization can affect the social transmission of traditional knowledge associated with subsistence fishing (see Turner et al. 2007), which can lead to a shift away from a fish diet. In

Rarotonga, I suggest that in addition to the effect of modernization, ciguatera poisoning expedited a shift away from a fish diet, particularly for those citizens raised in the 1990s who were generally not fed reef fish when they were children (referred to here as the ‘ciguatera’ generation). The ‘ciguatera’ generation (29 years and under) comprises ~52% of Rarotonga’s population (Cook Islands Statistics Office 2006). Although, by 2006, there was a slight increase in the ratio of reef fish to pelagic fish consumed, as well as a slight increase in fishing frequency in 2011, returning to 1989 consumption levels may take some time because reef fishes are primarily consumed by the ‘pre-ciguatera’ generation. Despite ciguatera poisoning declining in recent years, I suggest that two decades of severe ciguatera poisoning has disrupted the transmission of traditional subsistence fishing knowledge between the ‘pre-ciguatera’ generation and the ‘ciguatera’ generation. Such knowledge has been shown to be critical for managing marine resources (Alcala and Russ 1990; Johannes 1997).

In summary, this study showed that when ciguatera poisoning was severe in Rarotonga, people shifted their diets toward readily available, alternative proteins that included imported meats and canned goods. This trend is alarming because a shift away from a seafood diet, towards imported goods, has been shown to contribute to an increase in non-communicable diseases in the Pacific (Lewis and Ruff 1993; Li et al. 1994). Hospital cases of ciguatera poisoning and the loss of reef-fish stock were key variables in determining the economic impacts of ciguatera poisoning. While cases of ciguatera poisoning in Rarotonga continue to decline

(Rongo and van Woesik 2011), and as reef fish return to the menu of residents, we should also expect the direct economic impacts of ciguatera poisoning to decline. However, such a scenario may result in overfishing, and the economic impact would be similar to the loss of reef fish stock to ciguatera poisoning. Alternatively, if the shift away from a fish diet becomes the norm, then costs related to non-communicable diseases will increase (i.e., long-term costs that are not accounted for in my study). If the appropriate management strategies are not implemented in both scenarios of reef fish consumption, then economic impacts will continue even in the absence of ciguatera poisoning.

CHAPTER VI

DID CIGUATERA POISONING INFLUENCE THE LATE HOLOCENE
BIOGEOGRAPHIC DISTRIBUTION OF HUMANS ACROSS THE PACIFIC
OCEAN?

INTRODUCTION

The period between AD 1000 and 1450 was characterized by extensive cultural exchanges in Polynesia, which declined once marginal regions of Polynesia (e.g., Rapa Nui, Hawaii and New Zealand; Fig. 14a) were colonized (Hunt and Lipo 2006; Kirch and Kahn 2007; Wilmshurst et al. 2008). The collapse of the voyaging network between island archipelagos led to complete isolation of marginal regions, and the development of distinct cultures. However, low-level inter-island communications continued until AD 1800 (Rolett 1998).

The early ‘great fleet theory’ (Smith 1921), suggested that a single wave of seven canoes colonized New Zealand around AD 1350. It is now generally accepted that multiple waves of migrations occurred, originating from the East Polynesian ‘homeland’ (Rolett 2002), with the first canoe arriving in New Zealand around AD 1280 (Wilmshurst et al. 2008). But why would large groups of people leave their homelands to voyage into the unknown? Oceanic voyages are risky, albeit less so today than in the past. Landfalls were not guaranteed improvements over ports of departure. Explanations for migration range from overpopulation, resource

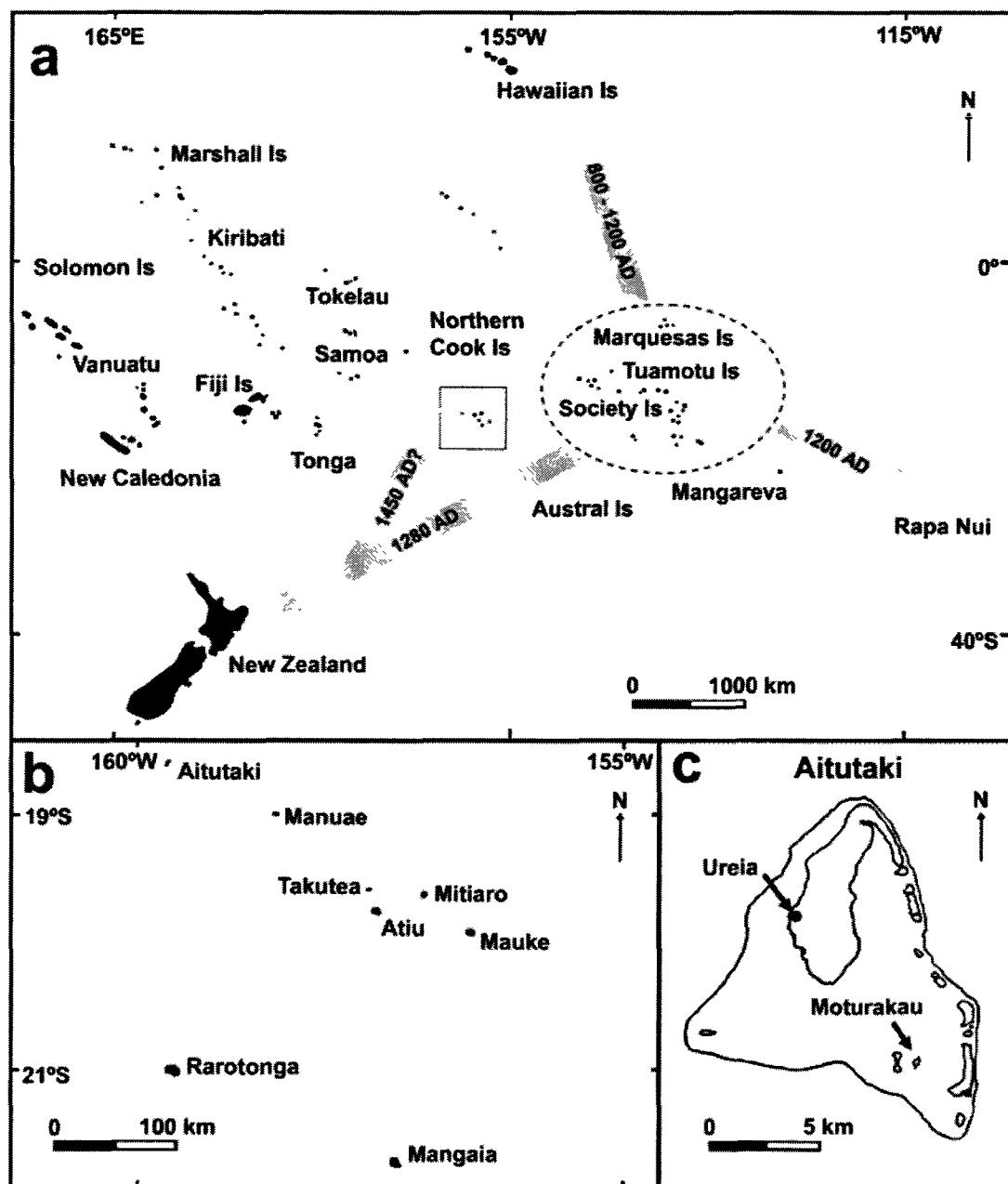


Figure 14. (a) Waves of migration (shaded grey arrows) originating from East Polynesia (dotted region; French Polynesia) with approximate migration dates to Rapa Nui (Hunt and Lipo 2006), Hawai'i (Kirch and Kahn 2007), and New Zealand (Wilmshurst et al. 2008). A possible subsequent migration from the southern Cook Islands (boxed region) to New Zealand after AD 1450 is noted. (b) Southern Cook Islands. (c) Map of Aitutaki indicating archaeological sites (Allen 1992, 2002).

overexploitation, warfare, environmental change, advances in canoe technology (e.g., double-hull canoe), and the adventurous spirit of Polynesians combined with favourable winds associated with El Niño–Southern Oscillation (Bridgman 1983; Kirch 1996; Finney 1998; Rolett 2002; Anderson et al. 2006). However, factors affecting marine resources, the main source of protein in Polynesia, have been ignored. I suggest that some migrations occurred when fish resources were unusable.

For millennia, Polynesians have had the technology to capture large pelagic and coral-reef fishes. However, a heavy reliance on a fish-based diet makes Pacific Islanders particularly vulnerable to harmful algal blooms that cause fishes to become toxic. Examining palaeoclimate and archaeological records, I ask if harmful algal blooms that result in ciguatera poisoning could have prompted waves of migrations of peoples across Polynesia between AD 1000 and 1450, a period when climatic conditions may have been favourable for ciguatera poisoning in the ‘homeland’. I suggest that after the collapse of cultural exchanges around AD 1450, societal adaptations to ciguatera poisoning resulted in dietary shifts away from ciguatoxic predatory fishes. I draw a parallel with the contemporary 1990s migration of Cook Islanders to New Zealand and Australia during ciguatera-poisoning events.

OVERVIEW OF CIGUATERA

Early historical accounts of ciguatera poisoning in the Pacific Ocean are found in Captain James Cook's journal. While moored at Vanuatu in 1774, Captain Cook recorded a fish poisoning incident, which has been interpreted as ciguatera poisoning (Doherty 2005). To date, ciguatera poisoning is the most common form of toxic seafood poisoning globally (Baden et al. 1995), affecting between 50,000 and 500,000 people annually, in tropical to subtropical regions (Fleming et al. 1998). Yet, for cultural reasons about 80 – 90% of ciguatera poisoning cases go unreported (Dalzell 1991).

Ciguatera poisoning is generally considered a consequence of fishes inadvertently ingesting toxic dinoflagellates (e.g., *Gambierdiscus toxicus* Adachi and Fukuyo, 1979; Yasumoto et al. 1977) that are epiphytic to macroalgae (see Cruz-Rivera and Villareal 2006). Recently, cyanobacteria have also been proposed to have a role as an inducer of ciguatera poisoning (Laurent et al. 2008).

Ciguatoxins bioaccumulate within the food web and are highly concentrated in large, carnivorous fishes (Lewis 2006). Because these toxigenic dinoflagellates rarely bloom, and only a low percentage of the strains produce detectable levels of toxin (Holmes et al. 1991), the distribution of ciguotoxic fishes is patchy, and therefore difficult to quantify in the field (Lewis 2006). Ciguatera poisoning is characterized by gastrointestinal (i.e., vomiting and diarrhoea), neurological (i.e., temperature dysesthesia and chronic fatigue) and cardiovascular symptoms (Lehane and Lewis 2000). Extreme cases can lead to paralysis or death. During ciguatera

poisoning events, most large reef fishes are rendered unusable in the food supply (Lehane and Lewis 2000). Because these toxigenic dinoflagellates are limited to shallow coastal waters, pelagic migratory species, such as bonito, are not affected.

EFFECTS OF CIGUATERA IN THE SOUTHERN COOK ISLANDS

Anecdotal reports of ciguatera poisoning were noted from Aitutaki in the mid-1940s, but the first recorded cases in the southern Cook Islands (Fig. 14b) were in 1984 (Losacker 1992), however a few cases were noted in the 1970s (see Fig. 3, p. 27). Based on the resident population in Rarotonga for 2006 (Cook Islands Statistics Office), the average incidence of ciguatera reported for Rarotonga from 1994 to 2010 was 168 cases per 10,000 persons per year. Because most reef fishes are potentially ciguatoxic, about 71 % of Rarotonga residents excluded fish from their diet in 2005 (Hajkowicz 2006). Residents who still eat fish only eat pelagic species, which are costly, and to a lesser extent also eat those reef fishes considered ‘safe’. Occasionally, residents import reef fishes from the northern Cook Islands, which according to locals are currently unaffected by ciguatera. This restriction contributed to the emigration of 18% of the resident Cook Islands’ population (from 1994 to 2000; Cook Islands Statistics Office 2007) to New Zealand and Australia during the 1990s, in part a consequence of the high cost of imported foods. Most emigrant families were from the lower income strata of Rarotonga, many of whom supplemented their daily protein with coastal marine resources (R. Crocombe, pers. comm.). In addition, the status of free association

between Cook Islands and New Zealand facilitated relocation. Ciguatera poisoning prompting the movement of people away from affected islands has been noted elsewhere. For example, Banner and Helfrich (1964) noted that ciguatera poisoning caused people to move from one coast to another in Hiva Oa in the Marquesas, French Polynesia, in the 1950s. Similarly in the 1950s, residents on Sydney Island in the Phoenix Islands were evacuated due partly to ciguatera poisoning rendering marine resources unusable (Cooper 1964). A similar movement is not improbable in the past because the areas least likely to be influenced by ciguatera poisoning were (and are) the islands amid cool waters, such as Rapa Nui and New Zealand.

PALAEOCLIMATE IN THE PACIFIC

A variety of proxy records based on ice cores, lake records, oceanic clastic deposition and coral cores shed light on Pacific Ocean sea surface temperatures (SSTs) during the period of Polynesian migrations. The largest single cause of inter-annual variability in Pacific climates is El Niño–Southern Oscillation (ENSO). El Niño tends to bring cool SSTs to the western and central Pacific while La Niña episodes promote warm SSTs and higher rainfall. However, regional differences in SSTs during El Niño have been noted (Hales et al. 1999); for example, the southern Cook Islands are cooler during El Niño conditions and warmer than usual during La Niña, while the opposite is apparent in the northern Cook Islands and French Polynesia (i.e., the Society, Marquesas, and Tuamotu Islands) (Figs. 15a & b).

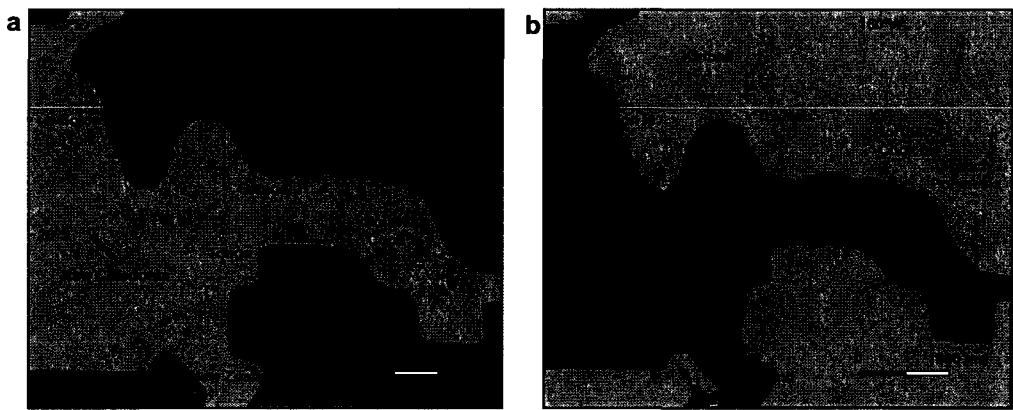


Figure 15. The South Pacific region with red areas indicating warmer sea surface temperatures (SSTs) and blue areas indicating cooler SSTs during (a) El Niño; and (b) La Niña (modified from Hales et al. 1999, with permission from Blackwell Publishing). These regional differences noted during the El Niño–Southern Oscillation are maintained during the Pacific Decadal Oscillation (PDO), but are less intense, with positive PDO following El Niño (a), and negative PDO following La Niña (b). Dotted lines delineate regions of interest.

Palaeo-records consistently indicate that the period from AD 900 to 1400 was one of relatively low El Niño activity (Thompson et al. 1984, 2000; Hendy et al. 2002; Moy et al. 2002; Cobb et al. 2003) with sporadic bursts of La Niña activity. Of the proxy records available, the Quelccaya ice cap provides the most direct quantification of ENSO intensity with low precipitation corresponding to strong La Niña events (Thompson et al. 1984, 2000). Between c. AD 1250 and 1300 drought reduced Andean precipitation at the Quelccaya ice cap by > 20%, compared with the long-term mean (Thompson et al. 1984). The period from AD 1300 to 1720 was a time in which La Niña activity subsided and El Niño became dominant. Drought again gripped the Andes between AD 1720 and 1860.

CLIMATE AND CIGUATERA POISONING EVENTS

Environmental factors influencing ciguatera poisoning remain complex and no single factor seems to consistently drive these events. For example, cases of ciguatera poisoning were positively correlated with SST anomalies associated with El Niño years in Rarotonga from 1973 to 1994 (Hales et al. 1999), yet my examination from 1994 to 2011 showed no significant correlation (see Chapter III, pg. 55). Instead, I found that disturbance frequency associated with El Niño years increased the risk for ciguatera poisoning in Rarotonga. With the coupling between SOI and PDO (see Verdon and Franks 2006), and PDO being the dominant climate signature in Rarotonga (see Fig. 7, p 52), this suggests that both cycles are important for driving ciguatera poisoning in this region.

Based on literature, anecdotal reports, and available hospital records of ciguatera-poisoning events from the Cook Islands and French Polynesia, I examined long-term relationships of ciguatera-poisoning events with climate forcing using the multi-decadal cycle of the PDO. Although the Inter-decadal Pacific Oscillation (IPO) is normally used to describe decadal oscillations in the South Pacific, I used PDO instead of IPO because there is an extended time-series data available into the late Holocene (back to AD 1000). In addition, the two cycles are highly correlated and equivalent in describing Pacific-wide variations in ocean climate (Power et al. 1999; Verdon and Franks 2006). For my purposes, I refer to periods when ciguatera poisoning begins to cause nuisance to the human population

as ‘initial’ ciguatera-poisoning events, which I consider less biased because humans tend to remember these periods.

Over the last 70 years, I note that ciguatera-poisoning cases in regions examined coincided with different PDO phases. For example in the southern Cook Islands, anecdotal reports were noted in Aitutaki in the mid-1940s (Losacker 1992) during the positive PDO (1925 – 1946). There were no reports in the southern Cook Islands during the 1950s when PDO shifted to negative (1947 – 1976), but extensive ciguatera reports were evident in the 1980s during the recent positive PDO (1977 – 1998; Fig. 16a). In the northern Cook Islands (Penrhyn Atoll), (initial) ciguatera-poisoning events occurred in the late 1950s (Losacker 1992; J. Williams, pers. comm.), during the negative phase, but declined in the mid 1990s (T. Rongo, pers. comm. with local inhabitants of the northern Cook Islands) during a predominantly positive phase (see Fig. 16a). Early reports of ciguatera poisoning in French Polynesia were noted in the early 1960s during the negative PDO (1947 – 1976), but generally declined toward the end of the study period in 1984 (Bagnis et al. 1985), as PDO shifted to positive. Therefore, French Polynesia and the northern Cook Islands experienced ciguatera-poisoning events during the negative PDO, when conditions were similar to those in the southern Cook Islands during a positive PDO (see Fig. 15). In light of these examples, I suggest that PDO played a role in driving ciguatera-poisoning events in these regions. Drawing from my proposed link to ciguatera-poisoning events in East Polynesia, I examine whether PDO phase shifts during the late Holocene (Fig. 16b) coincided with dietary and

technological shifts noted in southern Cook Island archaeological records (after AD 1450).

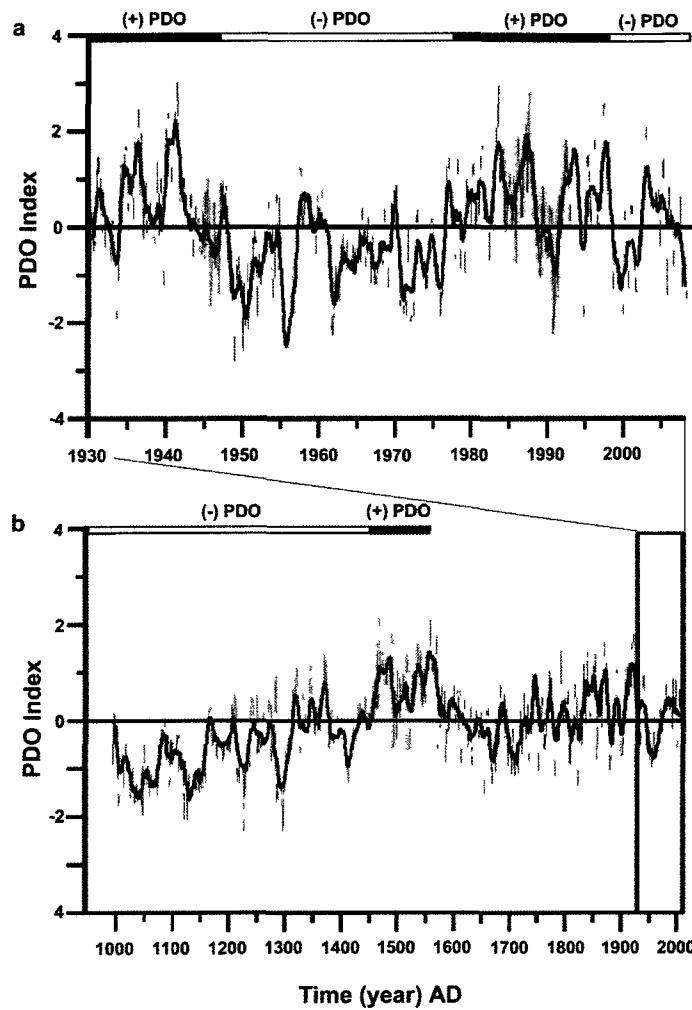


Figure 16. (a) Pacific Decadal Oscillation (PDO) index from 1930 to 2007 with an 11-year running average (data from <http://jisao.washington.edu/pdo/PDO.latest>). Blue shaded regions: poisoning events in the southern Cook Islands. Green shaded region: poisoning events in the northern Cook Islands and French Polynesia. Horizontal bars: the predominant PDO phase (+ or -). (b) PDO index from AD 993 to 2007 with an 11-year running average (AD 993–1996 data taken from MacDonald and Case 2006, and AD 1997–2007 from ‘*a*’). Green shaded region: heightened voyaging in Polynesia (AD 1000–1450). Blue shaded region: voyaging network collapse and dietary shifts in archaeological records (AD 1450–1550).

EVIDENCE OF CULTURAL ADAPTATIONS

Archaeological evidence and primary dietary indicators derived from middens and site excavations show temporal changes in both diet and technology. While other explanations have been offered, these archaeological findings are consistent with my hypothesis of ciguatera poisoning shaping Polynesian cultures. The colonization of East Polynesia was marked by a rapid decline of terrestrial resources such as bird populations (Steadman 1989; Steadman and Kirch 1990; Steadman and Rolett 1996; Grayson 2008). Midden records reveal that as terrestrial resources became scarce, coastal people relied more on fishes as their primary protein resource (Broughton 1994). Archaeological indicators of dietary shifts are drawn from middens and site excavations. Archaeo-fish assemblages provide direct evidence of food remains, and hooks provide an indication of the size of target prey. Various fishing methods such as traps, nets, spears and poisoning were practiced throughout the Pacific. Angling was not important until people reached East Polynesia where use of the pearl-shell (*Pinctada margaritifera* Linnaeus) hook became widespread, primarily because of its abundance, strength and efficacy as a lure (Allen 1992).

Archaeological evidence of bonito lure shanks from Mo'orea, French Polynesia, suggests that offshore fishing was also important in this region (Green et al. 1967). Furthermore, bone assemblages recovered from Huahine, Society Islands, indicated that pelagic fishes formed a high proportion of islanders' protein source (Leach et al. 1984). While the use of other shell material such as *Turbo* was

limited to small one-piece hooks, pearl-shell was used for most fish hooks (Green et al. 1967). Their extensive use suggests angling or trolling for pelagic fishes were important to past populations in East Polynesia.

In the southern Cook Islands, the collapse of the voyaging network after AD 1450 was marked by a shift from the preferred pearl-shell to the weaker *Turbo* hooks on Aitutaki, Mangaia, Mitiaro and Ma'uke (Steadman and Kirch 1990; Allen 1992; Walter and Campbell 1996) (see Fig. 14b; Table 19). The shift also coincided with dietary shifts in Aitutaki (Allen 1992) and Mangaia middens (Steadman and Kirch 1990; Butler 2001), indicating a transition from large to small inshore fishes [e.g., small cirrhitids (hawkfish) and small serranids (grouper) were consistent throughout the middens; Butler 2001; Allen 2002] (see Table 19). Fishing in general declined on Mangaia and Aitutaki (at Ureia and Moturakau; Fig. 14c) after AD 1450; declines were most noticeable in lutjanids (snapper), lethrinids (emperor), belonids (crocodile needlefish) and sphyraenids (barracuda) (Butler 2001; Allen 2002) (see Table 19), species that are considered potentially highly ciguatoxic or ‘high-risk’. While carangids also declined in both Ureia and Moturakau middens on Aitutaki after AD 1450, there was a slight increase around the European Contact period (post-AD 1650). In contrast, muraenids (moray eels; a high-risk family) increased in general after AD 1450 at Moturakau (see Table 19).

Fishes became eclipsed in the diet by domesticates (e.g., chickens, dogs, and pigs) and commensals (e.g., rats) on Aitutaki and Mangaia after AD 1450 (Steadman and Kirch 1990; Allen 2002; see Table 19). Anguillids (freshwater eels)

and eleotrids (sleeper gobies) also became conspicuously common in the Mangaia midden (cal AD 1000–1700). Most interesting was the seemingly sequential reduction in the relative abundance of anguillids that followed the initial increase (Steadman and Kirch 1990; Butler 2001), suggesting that this terrestrial resource (unaffected by ciguatera poisoning) was subjected to progressive overharvesting.

Table 19. Summary of fish middens from archaeological records (limited to potentially ciguotoxic fishes taken by angling), and terrestrial resources consumed. * indicates fish that declined in the record after AD 1450; ** indicates an increase in abundance; bold font indicates consistency throughout record. Records for Aitutaki were taken from Allen (2002), and the Mangaia record from Steadman and Kirch (1990) and Butler (2001). Contact is defined as post-AD 1650.

ISLAND	TIME (AD) <i>Dominant hook material</i>	FISH TARGETED BY ANGLING		CIGUATOXIC FISH AVOIDED <i>Declined* or absent between 1450 – 1650</i>	TERRESTRIAL RESOURCES
		OFFSHORE & REEF EDGE	INSHORE		
Aitutaki (Ureia)	950 – 1450 <i>Pearl-shell</i>	belonids (large) carangids (large) lutjanids (large)	lethrinids (large) serranids (small)		domesticates non-fish vertebrates
	1450 – Contact <i>Turbo</i>		serranids (small)	carangids (large)* lutjanids (large)* lethrinids (large)* belonids (large)	domesticates** non-fish vertebrates**
Aitutaki (Moturakau)	1250 – 1450 <i>Pearl-shell</i>	belonids (large) carangids (large) lutjanids (large) sphyraenid (large)	lethrinids (large) muraenids (large) serranids (small)		domesticates commensals
	1450 – Contact <i>Turbo</i>	carangids (large)**	muraenids (large)** serranids (small)	belonids (large)* lutjanids (large)* lethrinids (large)* sphyraenid (large)	domesticates commensals**
Mangaia	1000 – 1450 <i>Pearl-shell</i>	muraenids serranids (large) serranids (small) carangids lutjanids cirrhitids (small)			land birds (rapidly depleted) domesticates commensals anguillids (large) <i>Eleotris</i>
	1450 – 1650 <i>Turbo</i>	serranids (small) cirrhitids (small)		muraenids* carangids lutjanids serranids (large)	domesticates** commensals** anguillids (small)** <i>Eleotris</i> **

DISCUSSION

Although ciguatera-poisoning cases were linked to elevated SST anomalies on Rarotonga from 1973 to 1994 (Hales et al. 1999), I found no significant association with SST from 1994 to 2011. The inconsistency may be attributed to the time periods examined, where SST anomalies were important from 1973 to 1994, but not from 1994 to 2011. While under-reporting is likely to bias both analyses, data obtained from 1973 to 1994 included the entire Cook Islands (including the northern group where ciguatera poisoning was chronic during that period). According to the Cook Islands Ministry of Health, cases prior to 1993 were not separated by island and were therefore excluded from my analysis (1993 was also excluded from the analyses because of reporting inconsistencies in the early stages of the transition; T. Iorangi, pers. comm.). I note clear differences between the climatic conditions in the northern and southern Cook Islands under ENSO and PDO conditions (see Figs. 15a & b). While the southern Cook Islands are bathed in cool waters during El Niño conditions, the northern Cook Islands are warm. Therefore, combining ciguatera cases without considering locality may mask regional ciguatera interpretations.

Studies linking ciguatera-poisoning events to inter-annual cycles have been ambiguous (but see Chapter III); perhaps relationships may be better understood at inter-decadal cycles. The impact of inter-decadal cycles has been noted in the Pacific Ocean, where variability in fisheries was linked to PDO phase shifts (Lluch-

Belda et al. 1989; Chavez et al. 2003). For example, dramatic declines in the production of Alaskan salmon in the northeast Pacific Ocean occurred during the negative phase of the PDO, but increased during the positive phase (Hare and Francis 1995; Mantua et al. 1997). Similarly, recruitment of yellowfin tuna in the eastern Pacific Ocean declined during the negative phase of the PDO and increased during the positive phase (Maunder and Watters 2001). Based on initial ciguatera-poisoning events that coincided with different PDO phases, I suggest that positive phases are linked to ciguatera-poisoning events in the southern Cook Islands, while events in the northern Cook Islands and French Polynesia are linked to negative phases, although some ciguatera-poisoning cases were reported outside these climatic windows (T. Rongo, pers. comm. with local inhabitants of the northern Cook Islands; Chateau-Degat et al. 2007). Most important, however, is that these different phases (i.e., negative PDO in the northern Cook Islands and French Polynesia, and positive PDO in the southern Cook Islands) elicit the same climatic circumstances (see Fig. 15). Determining the importance of PDO and the timing of initial ciguatera-poisoning events in different regions of the South Pacific will be important to further examine this possible link.

The heightened voyaging network noted from AD 1000 to 1450 in eastern Polynesia may have been prompted by ciguatera poisoning. As terrestrial resources were depleted, Polynesians resorted to fishing. But when ciguatera poisoning was encountered, few alternatives were available. Advanced canoe technology and steady north-easterly La Niña winds (Anderson et al. 2006) during this negative

PDO period may have increased the probability of voyaging in the direction of New Zealand. These favourable winds were tested by a modern replicate of a traditional double-hull long-distance voyaging canoe (*Hokule'a*), where a crossing from Rarotonga to New Zealand was achieved in 16 days (Finney 1998). The adventurous spirit and superb navigational skills of Polynesians, who also always had return routes planned (as observed by Captain Cook in Tahiti; see Finney 1998) when venturing into uncharted waters (Michael Tavioni, pers. comm.), also made voyaging less daunting.

Though much of the native forest of Aitutaki was removed by AD 1100, the '*tamamu*' tree (genus *Calopyllum*), important for canoe-building, remained throughout the record (Allen 1992), indicating that long-distance voyaging and offshore fishing was still possible after AD 1100. The increase in offshore fishing in eastern Polynesia has been attributed to the availability of pearl-shell (see Allen 1992). While pearl-shell trading was probably critical to offshore pelagic fishing, the threat of ciguatera poisoning from reef fish may have been an additional factor that prompted exploitation of these offshore fish. The shift to offshore fishing may have been driven by an understanding that migratory pelagic fishes were 'safe' to eat. Contemporary examples of the shift to pelagic species, because of ciguatera poisoning, are noted on Raivavae, Austral Islands (M. Chinain, pers. comm.) and in the southern Cook Islands. Consequently, the collapse of voyaging and of the pearl-shell trade after AD 1450 would have reduced offshore fishing in the southern Cook

Islands. Such circumstances would have been particularly consequential when PDO became positive around AD 1450 (see Fig. 16b), increasing the likelihood of ciguatera-poisoning events in the southern Cook Islands.

The collapse of the voyaging network after AD 1450 would have led to the isolation of populations in the southern Cook Islands where adjustment to ciguatera poisoning was inevitable. Consequently, the dietary shift from large carnivorous fishes to smaller fishes and terrestrial resources may indicate a human behavioural adjustment (see Table 19). Small serranids and cirrhitids were consistent throughout middens despite the decline in overall fishing. Even today, in the southern Cook Islands, small serranids and cirrhitids are caught in the lagoon and off the reef edge using bamboo rods (*takiri*) with baited hook (T. Rongo, pers. obs.). However, for most of the 1990s, because of the ciguatera poisoning scare, *takiri* and fishing in general declined. Recently, Cook Islanders have learned, or as it may turn out re-learned, that small serranids (e.g., *Epinephelus merra* and *E. hexagonatus*) and cirrhitids (*Cirrhitus pinnulatus*) are among the few reef fishes considered ‘safe’. The decline in lutjanids on Aitutaki has been attributed to the loss of pearl-shell hooks (Allen 2002). Alternatively, I suggest that lutjanids were purposefully avoided. In the southern Cook Islands today, it is common knowledge that lutjanids (i.e., *Lutjanus fulvus* and *L. monostigma*) are off-limit because they are considered high-risk.

Reasons offered for the shift toward small fishes in Aitutaki and Mangaia middens were shifts in fishing technology, environmental change, overfishing, or a

preference for smaller fishes (Butler 2001; Allen 2002); I support the latter scenario. While contemporary signatures of overfishing include sequential reductions in average size frequency, this is not apparent on Rarotonga today; rather the size-frequency distributions are skewed to the left (T. Rongo, unpublished data), an apparent result of preference for small, low-risk fishes. The interpretation of archaeo-fish assemblages from the Pacific has been challenging because certain fish species do not preserve well (particularly small species; Nagaoka 1994). In addition, reference collections are lacking (Nagaoka 1994). Consequently, the highest taxonomic resolution has been to fish family [for the Aitutaki (Allen 2002) and Mangaia middens (Butler 2001)]. Identification to species level and information on size-frequency distribution may facilitate the interpretation of ciguotoxic prevalence, since large individuals bio-accumulate toxins, while small and juvenile individuals are often ‘safe’. Clearly, archaeological evidence in the southern Cook Islands points to a shift to ‘safer’ fishes. Because ciguatera exists on these islands today, I suggest that instantaneous shifts in fish use were probably a consequence of ciguatera poisoning.

In summary, counter to the general trend across the Pacific Ocean, where fishing was important over time (Allen 2002), a general decline in fishing was noted in the southern Cook Islands after AD 1450. This can be compared with recent ciguatera-poisoning incidents in the 1980s that led to a sudden decline in fishing in the 1990s. However, ciguatera poisoning may have had a greater impact in the past, when imported alternatives were unavailable. Terrestrial resources were

limited on small islands, and ciguatera poisoning rendered marine resources unusable. The impact on past human populations may have been life-threatening and options were few.

Clearly, ciguatera poisoning influenced the migration of Cook Islanders to New Zealand and Australia in the 1990s, and may have also induced late Holocene human population migrations between AD 1000 and 1450. Notwithstanding the adventurous spirit of people of the distant past, I suggest that when ciguatera poisoning became chronic, people migrated out of necessity. The inference that migration originating from French Polynesia was associated with ciguatera-poisoning events and negative PDOs is consistent with the timing of heightened voyages and the colonization of marginal regions of Polynesia (see Fig. 14a). I suggest that the importance of offshore fishing and the dietary shifts noted in middens were indications of adjustments to ciguatera poisoning. I also infer that the collapse of voyaging after AD 1450 possibly marked the decline of ciguatera poisoning in French Polynesia, and its increase in the southern Cook Islands when the PDO shifted from negative to positive. Therefore, any migration originating from the southern Cook Islands to New Zealand could have occurred after AD 1450 (see Fig. 14a) when ciguatera poisoning became chronic.

Although my inferences are circumstantial and drawn from available climate and archaeological records, the possibility of ciguatera poisoning affecting late Holocene human populations throughout the Pacific Basin should not be overlooked, and future archaeological studies should consider its impact. My

examination used the longer PDO reconstruction available from the northern Pacific Ocean (MacDonald and Case 2006); perhaps a reconstruction from the South Pacific region would allow a rigorous test of my proposed hypothesis. Furthermore, we need a more thorough biogeographical examination of the impact of interdecadal climate fluctuations that may trigger ciguatera poisoning events, which in turn may have critically influenced the behavioural patterns of past human populations. Detecting ciguatera-poisoning events that happened in the past will not only aid our understanding of the influence of climate on human history, but will also guide us in the future.

CHAPTER VII

SYNTHESIS AND CONCLUSIONS

This study showed that climate oscillations, particularly the inter-annual cycle of the El Niño Southern Oscillation and the multi-decadal cycle of the Pacific Decadal Oscillation (PDO), play a significant role in driving ciguatera poisoning in Rarotonga (Fig. 17). This study linked the ‘new surface hypothesis’ with the ‘climate oscillation hypothesis’, which were, in the past, two supposedly mutually exclusive hypotheses. I found that cases of ciguatera poisoning significantly increased during El Niño years and during the positive phase of the PDO, when disturbance frequency was significantly higher, than during La Niña years and during the negative phase of the PDO. Disturbances appeared to create new surfaces on reefs for the establishment of opportunistic ciguatoxic dinoflagellates.

I found that the climate phases not only influenced the disturbance frequency but also influenced the rate of reef recovery. Anecdotal reports showed that Rarotonga’s reefs, after a major *Acanthaster planci* outbreak in the 1970s, during a predominantly negative phase of the PDO, recovered after 10 years. In contrast, after a second *A. planci* outbreak in the 1990s, and other major reef disturbances during a predominantly positive phase of the PDO, reefs recovered slowly and have still not recovered in 2011, even after 12 years. With the recent shift into the negative phase of the PDO in 2008, reef recovery in Rarotonga should increase. There is recent evidence supporting this conjecture. This study showed

that coral cover was approximately 8% in 2011, which increased from the 1% and 5% cover recorded in 2006 and 2009, respectively.

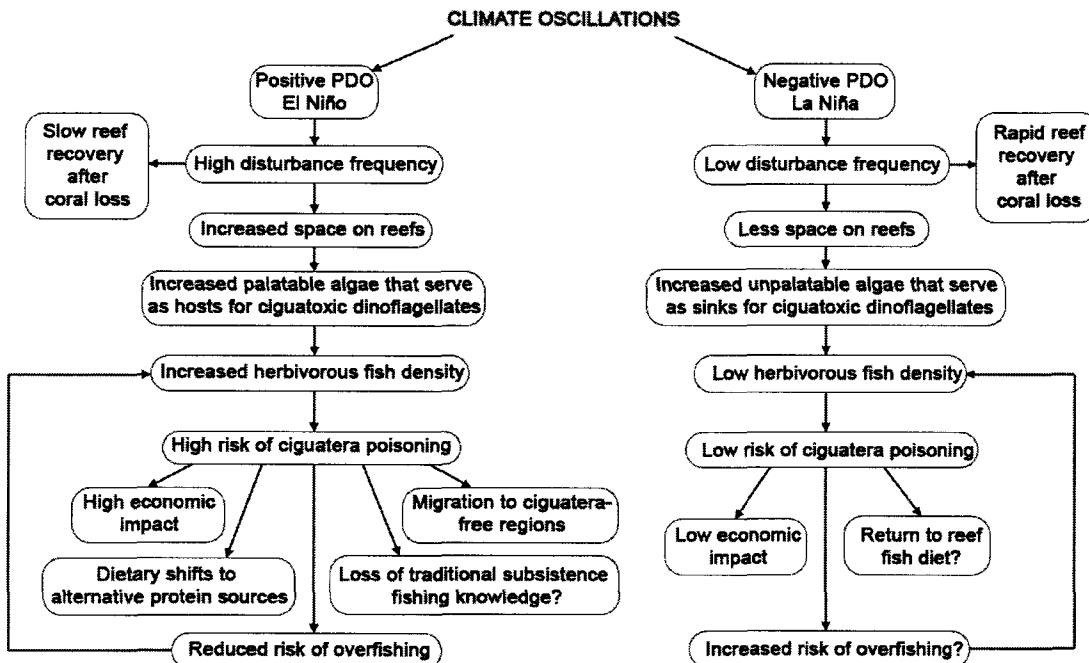


Figure 17. Schematic of the influence of climate oscillations on ciguatera poisoning in Rarotonga, southern Cook Islands, based on the results of this study. The positive phase of the Pacific Decadal Oscillation (PDO) is coupled with El Niño years, whereas the negative phase of the PDO is coupled with La Niña years. Disturbance frequency refers to cyclones and coral bleaching events that can impact coral reefs by reducing coral cover, and creating new surfaces for the establishment of host algae of ciguatoxic dinoflagellates. Herbivorous fishes refer to the primary vectors of ciguatoxins, such as acanthurids (primarily *Ctenochaetus striatus*) and scarids. High and low economic impacts refer to reef fish stock loss, monitoring and management costs, and health-related costs of ciguatera poisoning. Alternative protein sources refer primarily to imported meats and canned foods. The loss of traditional subsistence fishing knowledge or migration to ciguatera-free regions may occur when ciguatera poisoning is severe for decades. Under a climate phase with a low risk of ciguatera poisoning, a return to a reef fish diet and increased risk of overfishing are likely.

This study found that the increased density of herbivorous fishes after major cyclones coincided with increased cases of ciguatera poisoning. I suggest that increased herbivorous fishes, in particular *Ctenochaetus striatus* and other acanthurids, increased the transfer of ciguatoxins into the food web as food availability increased. In contrast, during non-El Niño years, and during the negative phase of the PDO, the frequency of cyclone disturbances was low as was the density of herbivorous fishes, but unpalatable macroalgae increased. I suggest that the increase of unpalatable algae contributed to the decline in herbivorous fish density, which decreased the transfer of ciguatoxins into the food web. In support, non-El Niño years and the negative phase of the PDO coincided with a significant decline in the cases of ciguatera poisoning, especially in recent years (see Fig. 17).

There is a strong consensus in the literature that the loss of corals (creating space on reefs for opportunistic macroalgae to establish and host ciguotoxic dinoflagellates) can lead to outbreaks of ciguatera poisoning. If so, then theoretically, coral cover should be a good proxy of the severity of a ciguatera outbreak. However, this study showed that coral cover was not a good predictor of ciguatera poisoning. I found that the decline in cases of ciguatera poisoning in Rarotonga, in recent years, was largely the result of a reduced frequency of disturbances, rather than a result of increased coral cover or decreased algal cover. Because reefs around Rarotonga have been dominated by algae in the last 10 years and algal communities have shifted toward unpalatable types, I suggest that space

can also be created with the removal of unpalatable algae during cyclone disturbances, which increases the risk of ciguatera poisoning.

Although ciguatera poisoning in Rarotonga was reported from all locations around the island, the widest reefs elicited the most cases of ciguatera poisoning, but there was no relationship between cases of ciguatera poisoning and reef exposure. The latter results contrast with other studies in the Pacific that found that the occurrence of ciguatera poisoning was higher on windward reefs.

My study agrees with work by Bagnis et al. (1988), who showed that vector shifts can be useful for understanding the dynamics of the ciguatera-poisoning cycle. I found that the increased involvement of herbivorous fishes in ciguatera poisonings signaled the early stage of an outbreak, whereas a shift to the increased involvement of carnivorous reef fishes signaled the decline of ciguatera poisoning.

The socioeconomic impacts of ciguatera poisoning have been difficult to quantify largely because of the underreported nature of the problem. Based on the questionnaire surveys that I conducted, the reported cases of ciguatera poisoning to health officials represented only 34% of the true incidence of ciguatera poisoning in Rarotonga. These estimates were instrumental in my calculations of the economic impact of ciguatera poisoning. When cases of ciguatera poisoning in Rarotonga peaked in 2004, the annual incidence was estimated at 1,058 per 10,000 population per year, which is the highest ever reported in the literature. I found that the average annual economic impact of ciguatera poisoning was greater during El Niño years and during the positive phase of the PDO (which frequently co-occurred)

than during non-El Niño years and during the negative phase of the PDO. The average annual economic impact in El Niño years and the positive phase of the PDO was estimated at ~NZD \$1.4 million for each cycle, while La Niña years and the negative phase of the PDO was estimated at ~NZD \$843,000 and ~\$872,000 respectively.

Since 1989, ciguatera poisoning has also contributed to the increased consumption of alternative proteins, particularly imported meats and canned fish. I found that reef fish was mostly consumed by individuals 30 years and older, who were raised before the outbreak of ciguatera poisoning in the 1990s (referred to in this study as the ‘pre-ciguatera generation’). Individuals raised during the 1990s were generally not fed reef fish when they were children (referred to in this study as the ‘ciguatera generation’). Although hospital records confirm that ciguatera poisoning is declining in Rarotonga in 2011, its impact over the last two decades may have disrupted the transmission of traditional subsistence fishing knowledge between the ‘pre-ciguatera’ generation and the ‘ciguatera’ generation.

Yet ciguatera poisoning may not be simply a contemporary phenomenon, but rather a dilemma that past human populations have encountered. By examining archaeological evidence and climate and temperature oscillations, using palaeo-datasets, I argue that the celebrated Polynesian voyages across the Pacific Ocean may not have been random episodes of discovery to colonize new lands, but rather, voyages of necessity because of ciguatera poisoning. A modern analogue (in the

1990s) was the shift towards processed foods in the Cook Islands during ciguatera-poisoning events, and mass emigration of islanders to New Zealand and Australia.

With the recent shift in climate oscillations to the negative phase of the PDO, coupled with La Niña years, not only will the incidence of ciguatera poisoning most likely continue to decline, but the reduced frequency of disturbances will also allow the recovery of reefs around Rarotonga. As reef fish return to the menu of residents in Rarotonga, we should also expect the direct economic impacts of ciguatera poisoning to decline. However, as in other nations that do not suffer ciguatera poisoning, such a scenario may result in overfishing, and the economic impact would be similar to the loss of reef-fish stock that was a consequence of ciguatera poisoning. Alternatively, if the shift away from a fish diet becomes the norm, then costs related to non-communicable diseases will most likely increase. If the appropriate management strategies are not implemented, then economic impacts will continue even in the absence of ciguatera poisoning.

Although the findings of my study have added to our understanding of ciguatera poisoning, a more comprehensive spatial examination is necessary. In particular, we need to examine whether climate oscillations influence ciguatera-poisoning dynamics in other regions of the Pacific. Furthermore, a reconstruction of climate oscillations from the South Pacific region would allow a rigorous test of my proposed late Holocene migration hypothesis, especially when combined with archaeological studies.

LITERATURE CITED

- Ajuzie CC. 2008. Toxic *Prorocentrum lima* induces abnormal behaviour in juvenile sea bass. *J Appl Phycol* 20:19-27.
- Alcala AC, Russ GR. 1990. A direct test of the effects of protective management on abundance and yield of tropical marine resources. *J Conserv Internat Explor Mer* 46:40-47.
- Allen MS. 1992. Temporal variation in Polynesian fishing strategies: the Southern Cook Islands in regional perspective. *Asian Perspectives* 31:183-204.
- Allen MS. 2002. Resolving long-term changes in Polynesian marine fisheries. *Asian Perspectives* 41:195-212.
- Anderson J. 1776. An account of some poisonous fish of the South Seas. *Philos Trans R Soc Lond* 66:544-62.
- Anderson DM, Lobel PS. 1987. The continuing enigma of ciguatera. *Biol Bull* 172:89-107.
- Anderson BS, Sims JK, Wiebenga NH, Sugi M. 1983. The epidemiology of ciguatera fish poisoning in Hawaii, 1975 – 1981. *Hawaii Med J* 42:326-34.
- Anderson J, Brider J, Makikiriti N, Solomona D, Tuatai T, Turua T. 2004. Water quality programme, Rarotonga and Aitutaki lagoons, 2004 annual summary. Ministry of Marine Resources. Misc. report 05/01. 42 p.
- Anderson A, Chappell J, Gagan M, Grove R. 2006. Prehistoric maritime migration in the Pacific Islands: an hypothesis of ENSO forcing. *The Holocene* 16:1-6.
- Anderson DM., Burkholder JM, Cochlan WP, Glibert PM, Gobler CJ, Heil CA, Kudela RM, Parsons ML, Jack Rensel JE, Townsend DW, Trainer VL, Vargo, GA. 2008. Harmful algal blooms and eutrophication: Examining linkages from selected coastal regions of the United States. *Harmful Algae* 8:39-53.
- Asian Development Bank. 2005. Pacific studies series: climate proofing – a risk-based approach to adaptation. Library of Congress, Philippines, 219 p.
- Ashton M, Tosteson T, Tosteson C. 2003. The effect of elevated temperature on the toxicity of the laboratory cultured dinoflagellates *Ostreopsis lenticularis* (Dinophyceae). *Rev Biol Trop* 51 (S4):1-6.
- Baden DG. 1983. Marine Food-Borne Dinoflagellate Toxins. In: Bourne GH, Danielli JF, Jeon KW, editors. *International Review of Cytology*. Academic Press. Volume 82. p 99-150.
- Baden D, Fleming LE, Bean JA. 1995. Marine toxins. In: deWolff FA, editor. *Handbook of Clinical Neurology: Intoxications of the Nervous System Part II. Natural Toxins and Drugs*. Amsterdam: Elsevier Press 21(65). p 141-75.
- Bagnis R. 1969. Naissance et développement d'une flambée de ciguatera dans un atoll des Tuamotu. *Rev Corps Santé Armees Terre Mer Air* 10:783-95.

- Bagnis R. 1994. Natural versus anthropogenic disturbances to coral reefs: comparison in epidemiological patterns of ciguatera. *Mem Queensl Mus* 34:455-60.
- Bagnis R, Kuberski T, Laugier S. 1979. Clinical observations on 3,009 cases of ciguatera (fish poisoning) in the South Pacific. *Am J Trop Med Hyg* 28 (6): 1067-73.
- Bagnis R, Chanteau S, Chungue E, Hurtel JM, Yasumoto T, Inoue A. 1980. Origins of ciguatera fish poisoning: a new dinoflagellate, *Gambierdiscus toxicus* Adachi and Fukuyo, definitely involved as a causal agent. *Toxicon* 18:199-208.
- Bagnis R, Bennett J, Barsinas M, Chebret M, Jacquet G, Lechat I, Mitermite Y, Perolat PH, Rongeras S. 1985. Epidemiology of ciguatera in French Polynesia from 1960 to 1984. In: Gabrie SB, editor. *Proceedings of the Fifth International Coral Reef Congress, Tahiti, Vol. 4. Moorea, French Polynesia: Antenne Museum-Ephe, Moorea.* p 475-82.
- Bagnis R, Bennett J, Barsinas M, Drollet JH, Jacquet G, Lecrand AM, Cruchet PH, Pascal H. 1988. Correlation between ciguateric fish and damage to reefs in the Gambier Islands (French Polynesia). In: Choat JH, Barnes D, Borowitzka MA, Coll JC, Davies PJ, Flood P, Hatcher BG, Hopley D, Hutchings PA, Kinsey D, Orme GR, Pichon M, Sale PF, Sammarco P, Wallace CC, Wilkinson C, Wolanski E, Bellwood O, editors. *Proceedings of the 6th International Coral Reef Symposium, Vol. 2. Townsville, Australia.* p 195-200.
- Bagnis R, Rougerie F, Orempuller J, Jardin C. 1992a. Coral bleaching as a cause of potential proliferation of *Gambierdiscus toxicus*. *Bull Soc Pathol Exot* 85:525.
- Bagnis R, Spiegel A, Nguyen L, Plritchard R. 1992b. Public health, epidemiological and socioeconomic patterns of ciguatera in Tahiti. In: Tosteson T, editor. *Proceedings of the Third International Conference on Ciguatera Fish Poisoning. Puerto Rico: Polyscience Publications Inc., Quebec.* p 131-143.
- Baldi M, Mullan B, Salinger J, Hosking D. 2009. Module 3: The Cook Islands Climate – Variation and Change. Prepared for the Cook Islands National Environment Service and Cook Islands Meteorological Service. NIWA Client Report: AKL2009-032. NIWA Project: CIN09101.
- Ballantine DL, Bardales AT, Tosteson TR. 1985. Seasonal abundance of *Gambierdiscus toxicus* and *Ostreopsis* sp. In: Gabrie C, Salvat B, editors. *Proceedings of the Fifth International Coral Reef Congress, vol. 4. Moorea: Antenne Museum-EPHE.* p 417-22.
- Banner AH. 1976. Ciguatera: a disease from coral reefs. In: Jones NA, Endean R, editors. *Biology and Geology of Coral Reefs, Vol. 3. New York: Academic Press.* p 177-213.

- Banner AH. 1984. The biological origin and transmission of ciguatoxin. In Humm HJ, Lane CE, editors. Bioactive compounds from the sea. New York: Marcel Dekker. p 15-36.
- Banner A, Helfrich P. 1964. The distribution of ciguatera in the tropical Pacific. Hawaii Marine Laboratory Technical Report No. 3. Honolulu (Hawaii): University of Hawaii. 48 p.
- Bell RE. 1992. Eutrophication and coral reefs: Some examples in the Great Barrier Reef lagoon. Water Res. 26:553-68.
- Bellwood DR, Hughes TP, Folke C, Nyström M. 2004. Confronting the coral reef crisis. Nature 429:827-33.
- Biensfang PK, Parsons ML, Bidigare RR, Laws EA, Moeller PDR. 2008. Ciguatera fish poisoning: a synopsis from ecology to toxicity. In: Walsh PJ, Smith SL, Fleming LE, Solo-Gabrielle HM, Gerwick WH, editors. Oceans and Human Health. Burlington (MA): Elsevier, Inc., Academic Press. p 257-70.
- Bomber JW, Aikman KE. 1989. The ciguatera dinoflagellates. Biol. Ocean. 6:291-311.
- Bomber JW, Guillard RRL, Nelson WG. 1988. Roles of temperature, salinity, and light in seasonality, growth, and toxicity of ciguatera causing *Gambierdiscus toxicus* Adachi et Fukuyo (Dinophyceae). J Exp Mar Biol Ecol 115:53-65.
- Bottein MYD, Kashinsky L, Wang Z, Littnan C, Ramsdell JS. 2011 Identification of ciguatoxins in Hawaiian monk seals *Monachus schauinslandi* from the Northwestern and main Hawaiian Islands. Environ Sci Technol 45:5403-9.
- Bourdeau P, Durand-Clement M, Ammar M, Fessard V. 1995. Ecological and toxicological characteristics of benthic dinoflagellates in a ciguateric area (Saint Barthelemy: French West Indies). In: Lassus P, Arzul G, Erard E, Gentien P, Marcaillou C, editors. Harmful marine algal blooms. Lavoisier (London). p 133-7.
- Boydrone-Le Garrac R, Benoit E, Sauviat M-P, Lewis RJ, Molgó J, Laurent D. 2005. Ability of some plant extracts, traditionally used to treat ciguatera fish poisoning, to prevent the in vitro neurotoxicity produced by sodium channel activators. Toxicon 46:625-34.
- Brand LE, Guillard RRL. 1981. The effects of continuous light and light intensity on the reproduction rates of twenty-two species of marine phytoplankton. J Exp Mar Biol Ecol 50:119-132.
- Bridgeman HA. 1983. Could climatic change have had an influence on the Polynesian migrations? Palaeogeography, Palaeoclimatology, Palaeoecology 41:193-206.
- Broughton JM. 1994. Declines in mammalian foraging efficiency during the Late Holocene, San Francisco Bay, California. Journal of Anthropological Archaeology 13:371-401.
- Bruslé J. 1997. Ciguatera Fish Poisoning – A Review. Sanitary and Economic Aspects. Les éditions INSERM, Paris, France. 147 p.

- Bruslé J, Bruslé-Sicard S, Fourcault B. 1998. Gonads of groupers (Serranidae from Mururoa and Hao, French Polynesia) used as indicators of the quality of the coral reef environment: Histological and ultrastructural aspects. *Cybium* 22:371-82.
- Butler VL. 2001. Changing fish use on Mangaia, southern Cook Islands: resource depression and the prey choice model. *International Journal of Osteoarchaeology* 11:88-100.
- Carlson RD. 1984. Distribution, periodicity, and culture of benthic/epiphytic dinoflagellates in a ciguatera endemic region of the Caribbean [dissertation]. Illinois: Southern Illinois University. 308 p.
- Carlson RD, Tindall DR. 1985. Distribution and periodicity of toxic dinoflagellates in the Virgin Islands. In: Anderson DM, White AW, Baden DG, editors. *Toxic Dinoflagellates*. New York: Elsevier Scientific Publications. p 171-76.
- Carlson RD, Morey-Gaines G, Tindall DR, Dickey RW. 1984. Ecology of toxic dinoflagellates from the Caribbean Sea: effects of macroalgal extracts on growth in culture. In: Ragelis EP, editor. *Seafood Toxins*. Amer Chem Soc Symp Ser, No. 262. Washington, D.C. p 271-87.
- Chavez FP, Ryan J, Lluch-Cota SE, Niñen CM. 2003. From anchovies to sardines and back: multidecadal change in the Pacific Ocean. *Science* 299:217-21.
- Chateau-Degat ML, Chinain M, Cerf N, Gingras S, Hubert B, Dewailly E. 2005. Sea water temperature, *Gambierdiscus* spp. variability and incidence of ciguatera poisoning in French Polynesia. *Harmful Algae* 4:1053-62.
- Chateau-Degat ML, Dewailly E, Cerf N, Nguyen NL, Huin-Blondey MO, Hubert B, Laudon F, Chansin R. 2007. Temporal trends and epidemiological aspects of ciguatera in French Polynesia: a 10-year analysis. *Trop Med Int Health* 12:485-92.
- Chinain M, Germain M, Deparis X, Pauillac S, Legrand AM. 1999. Seasonal abundance and toxicity of the dinoflagellates *Gambierdiscus* spp. (Dinophyceae), the causative agent of ciguatera in Tahiti, French Polynesia. *Mar Biol* 135:259-67.
- Chinain M, Darius HT, Ung A, Fouc MT, Revel T, Cruchet P, Pauillac S, Laurent D. 2009. Ciguatera risk management in French Polynesia: The case study of Raivavae Island (Australes Archipelago). *Toxicon* 56:674-90.
- Clarke KR, Warwick RM. 1994. Change in Marine Communities: An Approach to Statistical Analysis and Interpretation. Plymouth Marine Laboratory. 144 p.
- Cobb KM, Charles CD, Cheng H, Lawrence Edwards R. 2003. El Niño/Southern Oscillation and tropical Pacific climate during the last millennium. *Nature* 424: 271-76.
- Cook Islands Statistics Office. 2006. Cook Islands census of population and housing: an analytical report. 195 pp.
- Cook Islands Statistics Office. 2007. Cook Islands Annual Statistical Bulletin. Last accessed: 1 September 2011. Available at:
<http://www.stats.gov.ck/Resources/Publications/Annual/AnnualBul.htm>

- Cook A, Jardine A, Weinstein P. 2004. Using human disease outbreaks as a guide to multilevel ecosystem interventions. *Environ Health Perspect* 112:1143-6.
- Connell JH, Keough MJ. 1985. Disturbance and patch dynamics of subtidal marine animals on hard substrata. In: Pickett STA, White PS, editors. *The ecology of natural disturbance and patch dynamics*. New York (USA): Academic. pp. 125-152.
- Cooper MJ. 1964. Ciguatera and other marine poisoning in the Gilbert Islands. *Pac Sci* 18:411-40.
- Cruz-Rivera E, Villareal TA. 2006. Macroalgal palatability and the flux of ciguatera toxins through marine food webs. *Harmful Algae* 5:497-525.
- Dahl AL. 1980. Report on marine surveys of Rarotonga and Aitutaki. Noumea (New Caledonia): South Pacific Commission. 13 p.
- Dalrymple A. 1770. An historical collection of the several voyages and discoveries in the South Pacific Ocean. Vol. I : being chiefly a literal translation from the Spanish writers. Printed for the author, and sold by Nourse, J., Payne, T., Elmsley, P. London. p 140-1.
- Dalzell, P. 1991. Improvement of ciguatera case history reporting. *SPC Ciguatera Information Bulletin* 1:9-10.
- Dalzell P. 1993. Management of ciguatera fish poisoning in the South Pacific. *Inshore Fisheries Research Project Meeting Report*. Noumea (New Caledonia): South Pacific Commission. 26 p.
- Dawson E, Aleem A, Halstead B. 1955. Marine algae from Palmyra Islands with special reference to the feeding habits and toxicology of reef fishes. Allan Hancock Publications of the University of Southern California. Occasional paper 17.
- de Scally FA. 2008. Historical tropical cyclone activity and impacts in the Cook Islands. *Pacific Science* 18:411-440.
- de Scally FA, Wood GV, Maguire LK, Fournier-Beck MA, Silcocks D. 2006. A history of tropical cyclones and their impacts in the Cook Islands. Report for the Cook Islands Meteorological Service. 384 pp.
- de Sylva DP. 1994. Distribution and ecology of ciguatera fish poisoning in Florida, with emphasis on the Florida Keys. *Bull Mar Sci* 54:944-54.
- Deshpande SS. 2002. *Handbook of Food Toxicology*. New York: Marcel Dekker, Inc. 903 pp.
- Devaney DM, Randall JE. 1973. Investigations of *Acanthaster planci* in Southeastern Polynesia during 1970 – 1971. *Atoll Res Bull* 169:1-35.
- Dickey RM, Plakas SM. 2010. Ciguatera: A public health perspective. *Toxicon* 56:123-36.
- Doherty MD. 2005. Captain Cook on poison fish. *Neurology* 65:1788-91.
- Done TJ. 1987. Simulation of the effects of *Acanthaster planci* on the population structure of massive corals in the genus *Porites*: evidence of population resilience? *Coral Reefs* 6:75-90.

- Doucette GJ. 1995. Interactions between bacteria and harmful algae: a review. *Nat Toxins* 3:65-74.
- Durand-Clement M. 1987. A study of production and toxicity of cultural *Gambierdiscus toxicus*. *Biol Bull* 172:108-21.
- Eason B, Hope V. 2005. A detailed risk assessment of potential causes of the Irritant Syndrome in Titikaveka. November 2003 – July 2004. Landcare Research Contract Report LC0405/075, Landcare Research.
- Edmunds JSG, McCarthy RA, Ramsdell JS. 1999. Ciguatoxin reduces larval survivability in finfish. *Toxicon* 37:1827-32.
- Epstein P, Ford TE, Colwell RR. 1993. Marine ecosystems. *Lancet* 342:1216-19.
- Faust MA. 1995. Observation of sand-dwelling toxic dinoflagellates (Dinophyceae) from widely differing sites, including two new species. *J Phycol* 31:996-1003.
- Finney BR. 1998. Experimental voyaging, oral traditions and long-distance interaction in Polynesia. In: Weisler ME, editor. Prehistoric long distance in Oceania: an interdisciplinary approach. NZ Archaeological Association Monograph no. 21. Auckland (NZ): New Zealand Association. p 38–52.
- Fleming LE, Baden DG, Bean JA, Weisman R, Blythe DG. 1998. Seafood toxin diseases: issues in epidemiology and community outreach. In: Reguera B, Blanco J, Fernandez ML, Wyatt T, editors. Harmful Algae. Xunta de Galicia and Intergovernmental Oceanographic Commission of UNESCO. p 245-8.
- Folland CF, Renwick JA, Salinger MJ, Mullan AB. 2002. Relative influence of the Interdecadal Pacific Oscillation and ENSO on the South Pacific Convergence Zone. *Geophys Res Lett* 28(13):21-1–21-4.
- Food and Agriculture Organization (FAO). 2011. FAOSTAT Database: <http://faostat.fao.org/>. Accessed: October 2011.
- Forster D. 2009. Problematic Pacific poisonings. *Clinical Toxicology: Veterinary Times*. p 34-6.
- Furnas M, Mitchell A, Skuza M, Brodie J. 2005. In the other 90%: phytoplankton responses to enhanced nutrient availability in the Great Barrier Reef Lagoon. *Marine Pollution Bulletin* 51:253-65.
- Gardner TA, Côté IM, Gill JA, Grant A, Watkinson AR. 2005. Hurricanes and Caribbean coral reefs: impacts, recovery, patterns, and role in long-term decline. *Ecology* 86:174-84.
- Gillespie NC, Holmes NJ, Burke J. 1985. Distribution and periodicity of *Gambierdiscus toxicus* in Queensland, Australia. In Anderson DM, White A, Baden DG, editors. *Toxic Dinoflagellates*. London (UK): Elsevier. p 183 – 188.
- Gillespie NC, Lewis RJ, Pearn JH, Bourke ATC, Holmes MJ, Bourke JB, Shields WJ. 1986. Ciguatera in Australia: occurrence, clinical features, pathophysiology and management. *Med J Aust* 145:584-90.
- Glynn PW. 1984. Widespread coral mortality and the 1982–83 El Niño warming event. *Environmental Conservation* 11(2):133-46.

- Gonzalez I, Tosteson CG, Hensley V, Tosteson TR. 1995. Associated bacteria and toxicity development in cultured *Ostreopsis lenticularis*. In: Lassus P, Arzul G, Erard-Le Denn E, Gentien P, Marcaillou-Le Baut C, editors. Harmful marine algal blooms. Paris (France): Lavoisier. p 451-56.
- Goreau TJ, Hayes RL. 1995. Coral reef bleaching in the south central Pacific during 1994. Domestic Coral Reef Initiative Report, US Dept of State. 202 p.
- Grayson DK. 2008. Holocene underkill. Proceedings of the National Academy of Sciences USA 105:4077-78.
- Green RC, Green K, Rappaport RA, Rappaport A, Davidson JM. 1967. Archeology on the island of Mo'orea, French Polynesia. Anthropological Papers of the American Museum of Natural History, Vol. 51: Part 2. New York. p 1-136.
- Grinsted A, Moore JC, Jevrejeva S. 2004. Application of the cross wavelet transform and wavelet coherence to geophysical time series. Nonlinear Processes in Geophysics 11:561-66.
- Guillard RRL, Keller MD. 1984. Culturing dinoflagellates, Chapter 13. In: Spector DM, editor. Dinoflagellates. New York: Academic Press. p 391 – 441.
- Habekost R, Fraser I, Halstead B. 1955. Toxicology: observations on toxic marine algae. J Wash Acad Sci 45:101-3.
- Hajkowicz S. 2006. Cost scenarios for coastal water pollution in a small island nation: a case study from the Cook Islands. Coastal Management 34:369-86.
- Hajkowicz SA, Okotai P. 2005. An Economic Valuation of Watershed Management in Rarotonga, the Cook Islands. CSIRO Sustainable Ecosystems, Brisbane, Australia. 62 p.
- Hales S, Weinstein P, Woodward A. 1999. Ciguatera (fish poisoning), El Niño, and Pacific sea surface temperature. Ecosyst. Health 5:19-25.
- Halford A, Cheal AJ, Ryan D, Williams DMC. 2004. Resilience to large-scale disturbance in coral and fish assemblages on the Great Barrier Reef. Ecology 85:1892-1905.
- Hare SR, Francis RC. 1995. Climate change and salmon production in the northeast Pacific Ocean. In: Beamish RJ, editor. Climate change and northern fish populations. Canadian Special Publication of Fisheries and Aquatic Sciences 121. p 357-372.
- Hashimoto Y. 1979. Marine organisms which cause food poisoning. In: Marine Toxins and Other Bioactive Marine Metabolites. Japan Scientific Societies Press, Tokyo, 91 – 105.
- Hashimoto Y, Kamiy H, Yamazato K, Nozawa K. 1976. Occurrence of toxic bluegreen alga inducing skin dermatitis in Okinawa. In: Ohsaka A, Hayashi K, Sawai Y, editors. Animal, plant and microbial toxins. New York: Plenum. p 333-8.

- Hegerl GC, Zwiers FW, Braconnot P, Gillett NP, Luo Y, Marengo Orsini JA, Nicholls N, Penner JE, Stott PA. 2007. Understanding and Attributing Climate Change. In: Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, Tignor M, Miller HL, editors. *Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*. Cambridge (UK) and New York (USA): Cambridge University Press. 84 p.
- Hendy EJ, Gagan MK, Alibert CA, McCulloch MT, Lough JM, Isdale PJ. 2002. Abrupt decrease in tropical Pacific sea surface salinity at end of Little Ice Age. *Science* 295:1511-4.
- Hoagland P, Scatasta S. 2006. The economic effects of harmful algal blooms. In: Graneli E, Turner J, editors. *Ecology of Harmful Algae*. Ecological Studies 189. Germany: Springer-Verlag. p 391-402.
- Hoagland P, Anderson DM, Kaoru Y, White AW. 2002. The economic effects of harmful algal blooms in the United States: estimates, assessment issues, and information needs. *Estuaries* 25:819-37.
- Hokama Y, Yoshikawa-Ebesu JSM. 2001. Ciguatera Fish Poisoning: a Foodborne Disease. *J Toxicol—Toxin Reviews* 20(2):85-139.
- Hoegh-Guldberg O. 1999. Climate change, coral bleaching and the future of the world's coral reefs. *Marine and Freshwater Research* 50:839-66.
- Hoegh-Guldberg O, Mumby PJ, Hooten AJ, Steneck RS, Greenfield P, Gomez E, Harvell CD, Sale PF, Edwards AJ, Caldeira K, Knowlton N, Eakin CM, Iglesias-Prieto R, Muthiga N, Bradbury RH, Dubi A, Hatziolos ME. 2007. Coral reefs under rapid climate change and ocean acidification. *Science* 318:1737-42.
- Holmes MJ. 1998. *Gambierdiscus yasumotoi* sp. nov. (Dinophyceae), a toxic benthic dinoflagellate from Southeastern Asia. *J Phycol* 34:661-8.
- Holmes MJ, Lewis RJ, Gillespie NC. 1991. Toxicity of Australian and French Polynesian strains of *Gambierdiscus toxicus* (Dinophyceae) grown in culture: characterisation of a new type of maitotoxin. *Toxicon* 28:1159-72.
- Hughes TP. 1994. Catastrophes, phase shifts, and large-scale degradation of a Caribbean coral reef. *Science* 265: 1547-51.
- Hunt TL, Lipo CP. 2006. Late colonization of Easter Island. *Science* 311:1603-6.
- Jackson JBC, Kirby MX, Berger WH, Bjorndal KA, Botsford LW, Bourque BJ, Bradbury RH, Cooke R, Erlandson J, Estes JA, Hughes TP, Kidwell S, Lange CB, Lenihan HS, Pandolfi JM, Peterson CH, Steneck RS, Tegner MJ, Warner RR. 2001. Historical overfishing and the recent collapse of coastal ecosystems. *Science* 293, 629 – 638.
- Johannes RE. 1990. Managing small-scale fisheries in Oceania: unusual constraints and opportunities. In: Campbell H, Menz K, Waugh G, editors. *Economics of Fishery Management in the Pacific Island Region*. ACIAR, Canberra. Proceeding 25. p 68-76.

- Johannes RE. Traditional coral-reef fisheries management. 1997. In: Birkeland C, editor. *Life and death of coral reefs*. United States of America: Chapman and Hall. p 380-5.
- Jones JD. 1956. Observations on fish poisoning in Mauritius. *Proc R Soc Arts Sci Mauritius* 1:367-85.
- Kaly UL, Jones GP. 1994. Test of the effect of disturbance on ciguatera in Tuvalu. *Mem Queensl Mus* 34: 523-32.
- Kaly UL, Jones GP, Tricklebank K. 1991. Preliminary assessment of a severe outbreak of ciguatera at Niutao, Tuvalu. *S Pac J Nat Sci* 11:62-81.
- Kirch PV. 1996. Late Holocene human-induced modifications to a central Polynesian island ecosystem. *Proceedings of the National Academy of Sciences USA* 93:5296-5300.
- Kirch PV, Kahn JG. 2007. Advances in Polynesian prehistory: a review and assessment of the past decade (1993–2004). *Journal of Archaeological Resources* 15:191–238.
- Kirch PV, Yen DE. 1982. *Tikopia: the Prehistory and Ecology of a Polynesian outlier*. Hawaii: Bernice P Bishop Museum Bulletin 238. Honolulu, Bishop Museum Press. 396 p.
- Kite-Powell HL, Fleming LE, Backer LC, Faustman EM, Hoagland P, Tsuchiya A, Younglove LR, Wilcox BA, Gast RJ. 2008. Linking the oceans to public health: current efforts and future directions. *Environmental Health* 7(S2):S6. doi:10.1186/1476-069X-7-S2-S6
- Kodama, AM, Hokama Y. 1989. Variations in symptomology of ciguatera poisoning. *Toxicon* 27:593-5.
- Kohler ST, Kohler CC. 1992. Dead bleached coral provides new surfaces for dinoflagellates implicated in ciguatera fish poisonings. *Environ Biol Fish* 35:413-6.
- Kumar-Roiné S, Matsui M, Pauillac S, Laurent D. 2010. Ciguatera fish poisoning and other seafood intoxication syndromes: a revisit and a review of the existing treatments employed in ciguatera fish poisoning. *The South Pacific Journal of Natural and Applied Sciences* 28:1-26.
- Landsberg JH. 1995. Tropical reef-fish disease outbreaks and mass mortalities in Florida, USA: what is the role of biological dietary toxins? *Diseases of Aquatic Organisms* 22:83-100.
- Lapointe BE. 1997. Nutrient thresholds for bottom-up control of macroalgal blooms on coral reefs in Jamaica and southeast Florida. *Limnology and Oceanography* 42(5 part 2):1119-1131.
- Laurent D, Kerbrat A, Darius HT, Girard E, Golubic S, Benoit E, Sauviat MP, Chinain M, Molgo J, Pauillac S. 2008. Are cyanobacteria involved in Ciguatera Fish Poisoning-like outbreaks in New Caledonia? *Harmful Algae* 7:827-38.

- Leach BF, Intoh M, Smith IWG. 1984. Fishing, turtle hunting, and mammal exploitation at Fa'ahia, Huahine, French Polynesia. *Journal de la Société des Océanistes* 79:183-97.
- Legrand AM, Fukui M, Cruchet P, Ishibashi Y, Yasumoto T. 1992. Characterization of ciguatoxins from different fish species and wild *Gambierdiscus toxicus*. In: Tosteson TP, editor. *Proceedings of the Third International Conference on Ciguatera Fish Poisoning*, Puerto Rico. Québec: Polyscience Publications. p 25-32.
- Lehane L, Lewis RJ. 2000. Ciguatera: recent advances but the risk remains. *International Journal of Food Microbiology* 61:91-125.
- Lessios HA, Robertson DR, Cubit JD. 1984. Spread of diadema mass mortality through the Caribbean. *Science* 226:335-7.
- Lewis ND. 1979. The impact of ciguatera fish poisoning in the Pacific. *South Pacific Bulletin Third Quarter*. p 8-12.
- Lewis ND. 1983. Ciguatera - Implications for nutrition and marine resource development in the Pacific Islands. *Journal de la Societe des oceanistes* 77:89-104.
- Lewis, N.D. 1986. Epidemiology and impact of ciguatera in the Pacific: a review. *Mar Fish Rev* 48:6-13.
- Lewis RJ. 1992a. Ciguatoxins are potent ichthyotoxins. *Toxicon* 30:207-11.
- Lewis RJ. 1992b. Socioeconomic impacts and management of ciguatera in the Pacific. *Bull Soc Path Ex* 85:427-34.
- Lewis RJ. 2001. The changing face of ciguatera. *Toxicon* 39:97-106.
- Lewis RJ. 2006. Ciguatera: Australian perspectives on a global problem. *Toxicon* 48:799-809.
- Lewis RJ, Sellin M. 1992. Multiple ciguatoxins in the flesh of fish. *Toxicon* 30:915-19.
- Lewis RJ, Holmes MJ. 1993. Origin and transfer of toxins involved in ciguatera. *Comp Biochem Physiol* 160C(3):615-28.
- Lewis RJ, Ruff TA. 1993. Ciguatera: ecological, clinical and socioeconomic perspectives. *Crit Rev Environ Sci Technol* 23:137-56.
- Lewis RJ, Burke JB, Gillespie NC. 1986. Possible effects of Cyclone Winifred on ciguatera endemicity of Sudbury Reef, North Queensland. In: Dutton IM, editor. *Workshop on the offshore effects of Cyclone Winifred*. Great Barrier Reef Marine Park Authority Workshop Series No. 7. 111 p.
- Lewis RJ, Holmes MJ, Sellin M. 1994a. Invertebrates implicated in the transfer of gambioxins to the benthic carnivore *Pomadasys maculatus*. *Mem Queensl Mus* 34:561-4.
- Lewis RJ, Sellin M, Gillespie NC, Holmes HJ, Keys A, Street R, Smythe H, Thaggard H, Bryce S. 1994b. Ciguatera and herbivores: uptake and accumulation of ciguatoxins in *Ctenochaetus striatus* on the Great Barrier Reef. *Memoirs of the Queensland Museum* 34:565-70.

- Lewis RJ, Vernoux JP, Brereton IM. 1998. Structure of Caribbean ciguatoxin isolated from *Caranx latus*. J Am Chem Soc 120:5914-20.
- Li N, Tuomilehto J, Dowse G, Virtala R, Zimmet P. 1994. Prevalence of coronary heart disease indicated by electrocardiogram abnormalities and risk factors in developing countries. Journal of Clinical Epidemiology 47:599-611.
- Linsley BK, Wellington GM, Schrag DP. 2000. Decadal sea surface temperature variability in the subtropical South Pacific from 1726 to 1997 A.D. Science 290:1145-8.
- Litaker RW, Vandersea MW, Faust MA, Kibler SR, Nau AW, Holland WC, Chinain M, Holmes MJ, Tester PA. 2010. Global distribution of ciguatera causing dinoflagellates in the genus *Gambierdiscus*. Toxicon 56:711-30.
- Llewellyn LE. 2009. Revisiting the association between sea surface temperature and the epidemiology of fish poisoning in the South Pacific: Reassessing the link between ciguatera and climate change. Toxicon 56:691-7.
- Lluch-Belda D, Crawford RJM, Kawasaki T, MacCall AD, Parrish RH, Schwartzlose RA, Smith PE. 1989. World-wide fluctuations of sardine and anchovy stocks: the regime problem. South African Journal of Marine Science, 8:195-205.
- Losacker W. 1992. Ciguatera fish poisoning in the Cook Islands. Bulletin of the Exotic Pathology Society 85:447-8.
- Lyon SJ. 2000. Base line survey and long term monitoring programme of the outer reef, Rarotonga, Cook Islands. Prepared for the Cook Islands National Environment Service. 17 pp.
- Lyon SJ. 2003. Rarotonga fringing reef survey. Prepared for the Cook Islands National Environment Service, Tu'anga Taporoporo, Cook Islands. 20 pp.
- MacDonald GM, Case RA. 2006. Pacific Decadal Oscillation reconstruction for the past millennium. IGBP PAGES/World Data Center for Paleoclimatology Data Contribution Series no. 2006-023. NOAA/NCDC Paleoclimatology Program, Boulder, CO.
- Mantua NJ, Hare SR, Zhang Y, Wallace JM, Francis RC. 1997 A Pacific interdecadal climate oscillation with impacts on salmon production. Bulletin of the American Meteorological Society 78:1069-79.
- Martyr P. 1555. De orbo novo, the eight decades of Peter Martyr, 2 Vols. Engl. transl. by F. A. MacNutt (1912) G. A. Putnam Sons, NY.
- Mattei C, Wen PJ, Nguyen-Huu TD, Alvarez M, Benoit E, Bourdelais AJ, Lewis RJ, Baden DG, Molgó J, Meunier FA. 2008. Brevenal inhibits Pacific ciguatoxin-1B-induced neurosecretion from bovine chromaffin cells. PLoS ONE 3: 1 – 9.
- Maunder MN, Watters GM. 2001. Status of yellowfin tuna in the eastern Pacific Ocean. Inter-American Tropical Tuna Commission Stock Assessment Report 1:5-86.

- McCulloch MT, Gagan MK, Mortimer GE, Chivas AR, Isdale PJ. 1994. A high-resolution Sr/Ca and d₁₈O coral record from the Great Barrier Reef, Australia, and the 1982–1983 El Niño. *Geochimica et Cosmochimica Acta* 58:2747-54.
- Miller DM. 1991. Ciguatera seafood toxins. Boca Raton (FL): CRC Press. 176 p.
- Miller I, Thompson A, Loo M. 1994. Report on baseline surveys for monitoring the fringing reef of Rarotonga, Cook Islands. Prepared for the Cook Islands National Environment Service. 29 pp.
- Moore J. 2006. Seafood consumption survey, Rarotonga [thesis]. Tauranga (NZ): Bay of Plenty Polytechnic, Windemere Campus. 33 p.
- Moy CM, Seltzer GO, Rodbell DT, Anderson DM. 2002. Variability of El Niño/Southern Oscillation activity at millennial timescale during the Holocene epoch. *Nature* 420:162-5.
- Mumby PJ, Hastings A, Edwards HJ. 2007. Thresholds and the resilience of Caribbean coral reefs. *Nature* 450:98-101.
- Nagaoka L. 1994. Differential recovery of Pacific Island fish remains: evidence from the Moturakau Rockshelter, Aitutaki, Cook Islands. *Asian Perspectives* 33:1-17.
- Nakahara H, Sakami T, Chinain M, Ishida Y. 1996. The role of macroalgae in epiphytism of the toxic dinoflagellates *Gambierdiscus toxicus* (Dinophyceae). *Psychol. Res.* 44:113-7.
- Nakajima I, Oshima Y, Yasumoto T. 1981. Toxicity of benthic dinoflagellates in Okinawa. *Jpn Soc Sci Fish* 47: 1029-33.
- National Climate Centre. 2011a. Bureau of Meteorology. El Niño – Detailed Australian Analysis. Available at:
<http://www.bom.gov.au/climate/enso/enlist/index.shtml>
 Last accessed: 1 October 2011.
- National Climate Centre. 2011b. Bureau of Meteorology: Climate Analysis Section. Monthly Southern Oscillation Index. Available at:
<ftp://ftp.bom.gov.au/anon/home/ncc/www/sco/soi/soplaintext.html>
 Last accessed: 1 October 2011.
- Osborne NJT, Webb PM, Shaw GR. 2001. The toxins of *Lyngbya majuscula* and their human and ecological health effects. *Environ. Int.* 27:381-92.
- Pacific Country Report – Cook Islands. 2003. Sea level and climate: their present state. Report for the Cook Islands Government. Sponsored by the Australian Agency for International Development, managed by Australian Marine Science and Technology Ltd, and supported by NTF Australia at the Flinders University of South Australia. 21 pp.
- Palafox NA, Jain LG, Pinano AZ, Gulick TM, Williams RK, Schatz IJ. 1988. Successful treatment of ciguatera fish poisoning with intravenous mannitol. *JAMA* 259:2740-2.
- Palumbi, SR. 2004. Why mothers matter. *Nature* 430:621-2.

- Pandolfi JM, Bradbury RH, Sala E, Hughes TP, Bjorndal KA, Cooke RG, McArdle D, McClenachan L, Newman MJH, Paredes G, Warner RR, Jackson JBC. 2003. Global trajectories of the long-term decline of coral reef ecosystems. *Science* 301:955-8.
- Parmesan C, Yohe G. 2003. A globally coherent fingerprint of climate change impacts across natural systems. *Nature* 421:37-42.
- Parsons ML, Preskitt LB. 2007. A survey of epiphytic dinoflagellates from the coastal waters of the island of Hawai'i. *Harmful Algae* 6:658-69.
- Pinca S, Awira R, Kronen M, Chapman L, Lasi F, Pakoa K, Boblin P, Friedman K, Magron F, Tardy E. 2007. Cook Islands country report: profiles and results from Surrey work at Aitutaki, Palmerston, Mangaia, and Rarotonga (February and October 2007). Pacific Regional Oceanic and Coastal Fisheries Development Programme (PROCFish/C/CoFish), Secretariat of the Pacific Community. ISBN: 978-982-00-0389-7.
- Ponia B, Raumea K, Turua T, Clippingdale M. 1999. Rarotonga fringing reef fish and coral monitoring survey. Ministry of Marine Resources. Misc. Report: 99/20. Rarotonga, Cook Islands. 23 p.
- Pottier I, Vernoux JP, Jones A, Lewis RJ. 2002. Characterization of multiple Caribbean Ciguatoxins and congeners in individual specimens of horse-eye jack (*Caranx latus*) by high performance liquid chromatography/mass spectrometry. *Toxicon* 40: 929-40.
- Power S, Casey T, Folland C, Colman A, Mehta V. 1999. Inter-decadal modulation of the impact of ENSO on Australia. *Climate Dynamics* 15:319-24.
- Precht WF, Aronson RB. 2006. Death and resurrection of Caribbean coral reefs: a paleoecological perspective. In: Côté I, Reynolds J, editors. *Coral Reef Conservation*. Cambridge (UK): Cambridge University Press. p 40-77.
- Quod JP, Turquet J. 1996. Ciguatera in Reunion Island (SW Indian Ocean): Epidemiology and clinical patterns. *Toxicon* 34:779-85.
- Ragelis EP. 1984. Ciguatera seafood poisoning. In: Ragelis EP, editor. *Seafood Toxins*. ACS Symp Ser 262. Washington, DC (USA): American Chemical Society. p 25-36.
- Randall JE. 1958. A review of ciguatera tropical fish poisoning with a tentative explanation of its cause. *Bull Mar Sci Gulf Caribb* 8:236-67.
- Randall JE. 1981. National Marine Fisheries Service. Pacific Ciguatera Workshop. Honolulu (HI): Honolulu Lab.
- Rausch de Traubenberg C, Lassus P. 1991. Dinoflagellate toxicity: are marine bacteria involved? Evidence from the literature. *Mar Microb Fd Webs* 5:205-26.
- Ren L, Linsley BK, Wellington GM, Schrag DP, Hoegh-Gulberg O. 2002. Deconvolving the d₁₈O seawater component from subseasonal coral d₁₈O and Sr/Ca at Rarotonga in the southwestern subtropical Pacific for the period 1726 to 1997. *Geochemica et Cosmochimica Acta*, 67:1609-21.

- Rhodes LL, Smith KF, Munday R, Selwood AI, McNabb PS, Holland PT, Bottein, M. 2009. Toxic dinoflagellates (Dinophyceae) from Rarotonga, Cook Islands. *Toxicon* 56:751-8.
- Richlan ML, Lobel PS. 2011. Effects of depth, habitat, and water motion on the abundance and distribution of ciguatera dinoflagellates at Johnston Atoll, Pacific Ocean. *Marine Ecology Progress Series* 421:51-66.
- Rogers CS. 1990. Response of coral reefs and reef organisms to sedimentation. *Marine Ecology Progress Series* 62:185-202.
- Rolett BV. 1998. Hanamiai: Prehistoric Colonization and Cultural Change in the Marquesas Islands (East Polynesia). Yale University Publications in Anthropology Number 81. New Haven: Department of Anthropology and The Peabody Museum: Yale University. 277 p.
- Rolett BV. 2002. Voyaging and interaction in ancient East Polynesia. *Asian Perspectives* 41: 182-92.
- Rongo T, van Woesik R. 2011. Ciguatera poisoning in Rarotonga, southern Cook Islands. *Harmful Algae* 10:345-55.
- Rongo T, Holbrook J, Rongo TC. 2006. A survey of Rarotonga. Report for the Cook Islands National Environment Service. 81 pp.
- Rongo T, Bush M, van Woesik R, 2009a. Did ciguatera prompt the late Holocene Polynesian voyages of discovery? *J Biogeogr* 36:1423-32.
- Rongo T, Rongo TC, Rongo J. 2009b. Rarotonga fore reef community survey for 2009. Report for the Cook Islands National Environment Service. 36 pp.
- Schnorf H, Taurarii M, Cundy T. 2002. Ciguatera fish poisoning: a double-blind randomized trial of mannitol therapy. *Neurology* 58:873-80.
- Sheppard CRC, Spalding S, Bradshaw C, Wilson, S. 2002. Erosion vs. recovery of coral reefs after 1998 El Niño: Chagos reefs, Indian Ocean. *Ambio* 31:40-8.
- Shimizu Y, Shimizu H, Scheuer PJ, Hokama Y, Oyama M, Miyahara JT. 1982. *Gambierdiscus toxicus*, a ciguatera-causing dinoflagellate from Hawaii. *Bull Jpn Soc Sci Fish* 48(6):811-3.
- Shoemaker RC, House D, Ryan JC. 2010. Defining the neurotoxin derived illness chronic ciguatera using markers of chronic systemic inflammatory disturbances: a case/control study. *Neurotoxicology and Teratology* 32:633-9.
- Smayda TJ. 1997. Harmful algal blooms: their ecophysiology and general relevance to phytoplankton blooms in the sea. *Limnol Oceanogr* 42:1137-53.
- Smith SP. 1921. Hawaiki: the original home of the Maori: with a sketch of Polynesian history, 4th ed. Auckland (NZ): Whitcombe and Tombs. 288 p.
- Solomona DM, Tuatai T, Vuki V, Koroa M. 2009. Decadal changes in subsistence fishing and seafood consumption patterns on Rarotonga, Cook Islands. *SPC Women in Fisheries Information Bulletin* 19:19-27.
- Spiegel J, Yassi A. 1997. The use of health indicators in environmental assessment. *J Med Syst* 21:275-89.

- Steadman DW. 1989. Extinction of birds in Eastern Polynesia: a review of the record and comparison with other Pacific island groups. *Journal of Archaeological Science* 16:177-205.
- Steadman DW, Kirch PV. 1990. Prehistoric extinction of birds on Mangaia, Cook Islands, Polynesia. *Proceedings of the National Academy of Sciences USA*, 87:9605-9.
- Steadman DW, Rolett B. 1996. A chronostratigraphic analysis of landbird extinction on Tahuata, Marquesas Islands. *Journal of Archaeological Science*, 23:81-94.
- Steneck RS. 1988. Herbivory on coral reefs: a synthesis. *Proc. 6th International Coral Reef Symposium, Australia* 1:37-49.
- Taylor FJR. 1985. The distribution of the dinoflagellates *Gambierdiscus toxicus* in the eastern Caribbean. In: Delesalle B, Galzin B, Repsilon D, Salvat B, editors. *Proceedings of the Fifth International Coral Reef Congress*, Vol. 4. Moorea (French Polynesia): Antenne Museum-EPHE. p 423-8.
- Taylor FJR, Gustavson MS. 1986. An underwater survey of the organism chiefly responsible for “ciguatera” fish poisoning in the eastern Caribbean region: the benthic dinoflagellate *Gambierdiscus toxicus*. In: Stefanon A, Flemming NJ, editors. *Proceedings of 7th International Diving Science Symposium*. Padova (Italy): Padova University. p 95-111.
- Tebano T. 1984. Population density study on a toxic dinoflagellate responsible for ciguatera fish poisoning on South Tarawa atoll, Republic of Kiribati. Atoll Research and Development Unit, Tarawa. 53 p.
- Tebano T. 1992. Ciguatera poisoning and reef disturbance in South Tarawa, Kiribati. Atoll Research Programme. *SPC Ciguatera Information Bulletin 2: Fisheries Information Project*, p 7.
- Tester PA, Feldman RL, Naua AW, Kibler SR, Litaker RW. 2010. Ciguatera fish poisoning and sea surface temperatures in the Caribbean Sea and the West Indies. *Toxicon* 56:731-8.
- Thompson CS. 1986. The Climate and Weather for the Southern Cook Islands. NZ Meteorological Service Miscellaneous Publication 188 (2). 69 p.
- Thompson LE, Mosley-Thompson E, Arnao BM. 1984. El Niño-Southern Oscillation events recorded in the stratigraphy of the tropical Quelccaya Ice Cap, Peru. *Science* 226:50-3.
- Thompson LE, Mosley-Thompson E, Henderson KA. 2000. Ice-core palaeoclimate records in tropical South America since the Last Glacial Maximum. *Journal of Quaternary Science* 15:377-94.
- Todd ECD. 1985. Ciguatera poisoning in Canada. In: Anderson DM, White AW, Baden DG, editors. *Toxic Dinoflagellates*. *Proceedings of the Third International Conference*. New York (USA): Elsevier/North Holland. p 505-10.

- Torrence C, Compo GP. 1998. A practical guide to wavelet analysis. *Bulletin of the American Meteorological Society* 79: 61-78.
- Tosteson T. 1992. Proceedings on the Third International Conference on ciguatera fish poisoning: Puerto Rico, 1990. Quebec (Canada): Polyscience Pub. 204 p.
- Tosteson T. 2004. Caribbean ciguatera: a changing paradigm. *Revista de Biología Tropical* 52(1):109-13.
- Tosteson TR, Ballantine DL, Durst HD. 1988. Seasonal frequency of ciguatoxic barracuda in southwest Puerto Rico. *Toxicon* 26:795-801.
- Tosteson TR, Ballantine DL, Tosteson CG, Hensley V, Bardales AT. 1989. Associated bacterial flora, growth and toxicity of cultured benthic dinoflagellates *Ostreopsis lenticularis* and *Gambierdiscus toxicus*. *Appl Environ Microbiol* 55:137-141.
- Tosteson TR, Edwards RA, Baden DG. 1992. Caribbean ciguatera and polyether dinoflagellate toxins: correlation of ciguatoxin with standard toxins. In: Tosteson TR, editor. *Proceedings of the Third International Conference on Ciguatera Fish Poisoning*, Puerto Rico. Québec: Polyscience Publications. p 89-102.
- Tosteson TR, Bard RF, Gonzalez IM, Ballantine DL, Bignami GS. 1995. Toxin diversity in the ciguatera food chain. In: Hokama Y, Scheuer PJ, Takeshi TY, editors. *Proceedings of the International Symposium on Ciguatera and Marine Natural Products*. Honolulu (HI): Asian Pacific Research Foundation. p 73-87.
- Turner RA, Cakacaka A, Graham NAJ, Polunin NVC, Pratchett MS, Stead SM, Wilson SK. 2007. Declining reliance on marine resources in remote South Pacific societies: ecological versus socio-economic drivers. *Coral Reefs* 26:997 – 1008.
- Ulijaszek SJ. 2003. Trends in body size, diet and food availability in the Cook Islands in the second half of the 20th century. *Economics and Human Biology* 1:123-137.
- Van Dolah FM. 2000. Marine algal toxins: origins, health effects, and their increased occurrence. *Environ. Health Persp* 108 (S1):133-141.
- van Woesik R, Ayling AM, Mapstone B. 1991. Impact of tropical cyclone 'Ivor' on the Great Barrier Reef, Australia. *Journal of Coastal Research* 7:551-8.
- van Woesik R, Tomascik T, Blake S. 1999. Coral assemblages and physico-chemical characteristics of the Whitsunday Islands: evidence of recent community changes. *Mar Freshwater Res* 50:427-40
- Verdon DC, Franks SW. 2006. Long-term behaviour of ENSO: Interactions with the PDO over the past 400 years inferred from paleoclimate records. *Geophysical Research Letters* 33: L06712. doi:10.1029/2005GL025052.
- Veron JEN, Hoegh-Guldberg O, Lenton TM, Lough JM, Obura DO, Pearce-Kelly P, Sheppard CRC, Spalding M, Stafford-Smith MG, Rogers AD. 2009. The coral reef crisis: the critical importance of <350 ppm CO₂. *Marine Pollution Bulletin* 58:1428-36.

- Villareal TA, Morton SL. 2002. Use of cell-specific PAM-fluorometry to characterize host shading in the epiphytic dinoflagellates *Gambierdiscus toxicus*. P.S.Z.N.: Marine Ecology 23 (2):127-40.
- Walter R, Campbell M. 1996. The Paraoa Site: fishing and fishhooks in 16th Century Mitiaro, Southern Cook Islands. Man & Culture in Oceania 12:47-60.
- Wilmshurst J, Anderson AJ, Highman TFG, Worthy TH. 2008. Dating the late prehistoric dispersal of Polynesians to New Zealand using the commensal Pacific rat. PNAS USA 105:7676-80.
- Wilson SK, Graham NAJ, Pratchett MS, Jones GP, Polunin NVC. 2006. Multiple disturbances and the global degradation of coral reefs: are reef fishes at risk or resilient? Global Change Biology 12:2220-34.
- Wong C-K, Hung P, Lee KLH, Kam K-M. 2005. Study of an outbreak of ciguatera fish poisoning in Hong Kong. Toxicon 46:563-71.
- World Bank. 2000. Cities, Seas and Storms: Managing Change in Pacific Island Economies, Vol. 4: Adapting to Climate Change. Washington, DC (USA): World Bank, Papua New Guinea and Pacific Island Country Unit. 72 p.
- Yasumoto T, Hashimoto Y, Bagnis R, Randall JE, Banner AH. 1971. Toxicity of the surgeonfishes. Bulletin of the Japanese Society of Scientific Fisheries 37:724-34.
- Yasumoto T, Nakajima I, Bagnis R, Adachi R. 1977. Finding of a dinoflagellate as a likely culprit of ciguatera. B Jpn Soc Sci Fish 43:1021-26.
- Yasumoto T, Inoue A, Bagnis R, Garcon M. 1979. Ecological survey on a dinoflagellate possibly responsible for the induction of ciguatera. Bull Jap Soc Sci Fish 45(3):395-9.
- Yasumoto T, Inoue A, Ochi T, Fujimoto K, Oshima Y, Fukuyo Y, Adachi R, Bagnis R. 1980. Environmental studies on a toxic dinoflagellate responsible for ciguatera. Bulletin of the Japanese Society of Scientific Fisheries 46:1397-1404.
- Yasumoto T, Raj U, Bagnis R. 1984. Seafood Poisoning in Tropical Regions. Lab. Food Sci. Hyg., Faculty of Agriculture, Tohoku, Japan. p 1-74.
- Yeeting B. 2009a. Ciguatera-like fish poisoning from giant clams on Emao Island, Vanuatu. SPC Fisheries Newsletter 129:13-6.
- Yeeting B. 2009b. Ciguatera and related biotoxins conference 2008: outcomes of the meeting. Background paper 8, 6th SPC Heads of Fisheries Meeting, Secretariat of the Pacific Community. 4 pp.
- Zann LP, Aleta S. 1984. A preliminary survey of fish consumption in Tokelau. Suva (Fiji Islands): Institute of Marine Resources, University of the South Pacific. 11 p.
- Zann LP, Bell L, Sua T. 1984. A preliminary survey of the inshore fisheries of Upolu Island, Western Samoa. Suva (Fiji Islands): Institute of Marine Resources, University of the South Pacific. 17 p.
- Zar JH. 1999. Biostatistical Analysis: fourth edition. Englewood Cliffs, New Jersey (USA): Prentice Hall, Inc. ISBN 0-16-081542-X

APPENDIX A. CIGUATERA QUESTIONNAIRE SURVEY

Name _____ Age _____ Gender _____ Data collector _____ Date _____

Never been poisoned (tick box and go to CURRENT STATE OF FISH CONSUMPTION)

No.	Date (year & month if possible)	Fish or invertebrate (see card for ID)	Where fish was caught (as close as possible, e.g., Titikaveka beach cargo)	Reported (A = admitted to hospital, O = outpatient, P = private, NR = not reported)	Degree (M = mild, S = severe)	Medication taken or prescribed (e.g., IV drip, phenegran, charcoal tablets, panadol), or Maori medicine taken (e.g., nono)	Symptoms (check all that apply)											
							Diarrhea (ake)	Vomiting (ruaki)	Dizziness (takarimi te kau)	Cold & shivering (anu tuketeke)	Joint Pain (mamae paunanga)	Sore gums (mamae te tuke no o)	Fatigue (ro i ro i)	Fainted (mata pain)	Needles & pins (putaputa)	Hallucination (a nevaneva)	Blurry vision (av-eave)	Itchiness (mango)
1						<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
2						<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
3						<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
4						<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
5						<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
6						<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

For more incidents, please use back of page

CURRENT STATE OF FISH CONSUMPTION (check all that apply)

Don't eat fish at all

Rarely eat fish

Only eat pelagics (e.g., tuna, marlin, broadbill, flying fish)

Stay away from known toxic fishes

Eat any reef fish

Only eat fish from the outer islands (indicate which island) _____

Eat more reef fish than pelagics

Eat more pelagics than reef fish

FISHES YOU CONSIDER SAFE _____

ADDITIONAL COMMENTS _____

APPENDIX B

RAROTONGA SEAFOOD CONSUMPTION QUESTIONNAIRE

1. DATE: _____
2. Name of person interviewed: _____
- 3: VILLAGE: _____
4. Occupation: _____
5. Number of people in household: _____
6. Income earned per week: Tick () *one only*.

<input type="radio"/> <\$50	<input type="radio"/> \$201 - \$300
<input type="radio"/> \$50 - \$100	<input type="radio"/> \$301 - \$400
<input type="radio"/> \$101 - \$200	<input type="radio"/> >\$400
7. Did the household consume any fresh fish yesterday? Circle one only. YES / NO
8. How much whole fish did the household consume yesterday? _____ kg
9. What kind of fish?

<input type="radio"/> tuna / a'ai, au'opu	<input type="radio"/> cod / patuki	<input type="radio"/> mullet / kanae
<input type="radio"/> moray / a'a pata	<input type="radio"/> goatfish / vete	<input type="radio"/> surgeonfish/maito
Others _____		
10. Where did you get the fish?

<input type="radio"/> Caught personally	<input type="radio"/> Purchased	<input type="radio"/> Received
---	---------------------------------	--------------------------------
11. Did the household consume any shellfish yesterday? Circle one only. YES / NO
12. How much shellfish did the household consume yesterday? _____ kg
13. What kind of shellfish?

<input type="radio"/> mussels / ka'i	<input type="radio"/> trochus / torokati
<input type="radio"/> giant clams/pa'ua	<input type="radio"/> turban snail/ariri
<input type="radio"/> pearl oyster / korori	
Others _____	
14. Where did you get the shellfish?

<input type="radio"/> Caught personally	<input type="radio"/> Purchased	<input type="radio"/> Received
---	---------------------------------	--------------------------------
15. What other seafood did the household consume yesterday?

<input type="radio"/> sea cucumber gonads / matu rori	<input type="radio"/> sea urchin / kina, atuke, avake
<input type="radio"/> lobster / koura tai	
<input type="radio"/> None of the above	
Others _____	

CANNED FISH

16. Did the household eat any canned fish yesterday? YES/NO last week? YES/NO
IF NO GO TO QUESTION 19.
17. How many days did household eat canned fish last week (including yesterday) _____
18. How many tins of canned fish did household eat last week (including yesterday)?

_____ tins	_____ weight (g)
_____ tins	_____ weight (g)
_____ tins	_____ weight (g)
Total tins _____	Total weight _____ (kg)

MEAT CONSUMPTION

19. Did the household eat any meat yesterday? YES / NO last week? YES / NO

IF NO GO TO QUESTION 23.

20. How many days did household eat meat last week (including yesterday)? _____

21. What meats did the household eat last week? Circle Pork Chicken Beef Lamb

22. What was the approximate kg of meat eaten by the household last week?

pork _____ chicken _____ red meat _____ corned beef _____ lamb _____

23. Did the household eat any fresh fish yesterday? YES/NO last week? YES/NO

24. How many days last week did household eat fresh fish (including yesterday)? _____

25. What was the approximate kg of fresh fish eaten by the household last week? _____

FISHING HOUSEHOLD

26. How many members of the household went fishing yesterday? _____

27. How long did they spend fishing yesterday? (TOTAL) _____ hrs

28. How did they fish?

Outboard Motor Canoe Reef collecting Free dive

Others _____

29. What did they use?

Fishing line Spear gun Fishing net

Scuba gear Snorkelling gear

Others _____

30. Where did they fish?

Open Sea Reef Lagoon

Other places _____

If on the reef and/or lagoon, which area(s) or village(s) _____

31. When did they go fishing?

Morning Evening Afternoon Night

32. List names and approximate weight of fish caught yesterday:

Name	Number caught	Length (cm)	Total weight (kg)

33. List names and approximate weight of shellfish caught yesterday:

Name	Number caught	Length (cm)	Total weight (kg)

34. Each week, how much fish did the household

sell? _____ kg

give away? _____ kg

receive? _____ kg

35. How many days last week did the household members go fishing? _____

Seafood Consumption Weight Estimates

Tin Fish

Wonderful Mackerel (Tomato) 425g
 Wonderful Mackerel (Green) 425g
 Sea Lord Fish Fillets chunky smoked 340g
 Pams Sandwich Tuna 95g/185g/210g/415g
 Pams Pink Salmon 105g/185g/210g/415g
 Tarragon Spanish Sardines 250g
 Brunswick Sardines (soya/olive/lemon/spring water) 106g/215g
 Tuna 185g/210g/425g

Corned Beef

Tapered Pams 340g
 Pacific 340g (12oz), 453g (1lb)
 Corned beef 340g
 Palm 340g/453g 1.36kg.72kg
 Ox & Pacific 340g

Chicken

Tegel & Ingham
 Size 20kg -2.1kg, Size 18 –1.9kg, Size 16 – 1.7kg, Size 14 - 1.35kg, Size 13 - 1.25kg, Size 12 1.14kg, Size 11-1.05kg, Size 10 - 1.05kg
 Brinks 1.2kg
 Mixed cut 2.0kg
 Carton 10kg
 Chicken nibbles/drumsticks/thighs 1kg
 Chicken pieces 2kg
 Turkey 3.5kg
 Nuggets 1kg
 Burger patties 800g
 Steak 1kg
 Chicken Pieces 5kg
 Chicken franks/sausage 454g
 Chicken pieces 12kg/2kg (CITC)

Pork

Bacon 1.094kg (local)
 Kiwi 200g/450g
 Precook Hutton sausages/skinless 2.5kg
 Gilmours bacon 1kg
 Specialty meat (Danish/bacon/hot pork) 180g
 Pastrami/Pepperoni 100g
 Luncheon Hutton 500g/800g
 Verkers Bacon 230/500g
 Hellers frankfurts 3kg

Lamb

Chops 2kg (pre-weighed)
 Off cuts 2kg
 Goat meat 1.4kg (Meatco)
 Neck chops 1.7kg (Meatco)1.5kg JIMCO
 Lamb shanks \$13.50kg

Beef

Kiwi Precooked 2.5kg
 Sizzlers cheese 450g
 Saveloys 400g
 Frankfurters 1kg
 Watties burger patties 600g
 Mince 500g/1kg
 Steak 16.90/kg
 Brisket \$18.95kg

Fish

Broadbill \$18.95/kg
 Marlin \$17.95/kg
 Mahimahi 180cm=28kg 210cm=40kg \$19.95/kg
 Tuna/Yellowfin \$22.90/kg
 Yellowfin 239cm=200kg 15-20 kg whole
 Albacore \$17.95/kg; 20kg whole
 Offcuts \$15.00/kg
 Tuna Steaks 200g
 Flying Fish (maroro) 27cm 7 fish (tui)=2.5kg
 Auopu (Skipjack tuna) 108cm=35kg (3kg avg.)
 Ku (Squirlfish) \$30/kg; 15 fish (tui)=1kg
 Black Jack (Ru'i) 100cm=17.9kg
 Mullet 76cm=5kg 30cm Avg=1kg
 Vena (castor oil fish) 100cm=63.5kg 30cm a avg=1.5kg
 Grouper (Punga) 90cm Avg=7kg
 Parrotfish 75 cm Avg=2kg