
LIBRARY
Te Tumu Herenga
THE UNIVERSITY OF AUCKLAND
RESEARCHSPACE@AUCKLAND

Copyright Statement

The digital copy of this thesis is protected by the Copyright Act 1994 (New Zealand). This thesis may be consulted by you, provided you comply with the provisions of the Act and the following conditions of use:

- Any use you make of these documents or images must be for research or private study purposes only, and you may not make them available to any other person.
- Authors control the copyright of their thesis. You will recognise the author's right to be identified as the author of this thesis, and due acknowledgement will be made to the author where appropriate.
- You will obtain the author's permission before publishing any material from their thesis.

To request permissions please use the Feedback form on our webpage. http://researchspace.auckland.ac.nz/feedback

General copyright and disclaimer

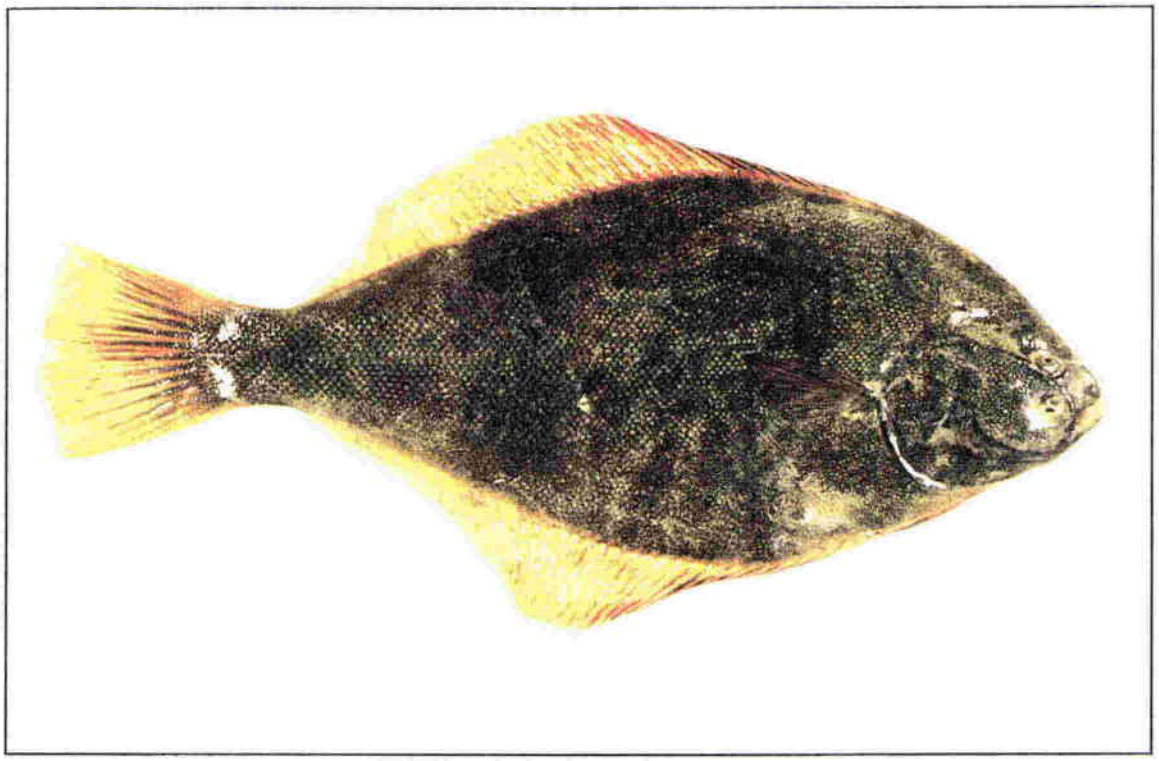
In addition to the above conditions, authors give their consent for the digital copy of their work to be used subject to the conditions specified on the Library Thesis Consent Form

THE HEALTH OF YELLOWBELLY FLOUNDER (*Rhombosolea leporina*) FROM THE WAITEMATA HARBOUR

AJRIN NENADIC

A thesis submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Biological Sciences, University of Auckland, June 1998.

UNIVERSITY OF ICELAND
1995
LIBRARY



Yellowbelly flounder
(*Rhombosolea leporina*)

ABSTRACT

This study focuses on an assessment of the health status of the yellowbelly flounder (*Rhombosolea leporina*) from two estuarine locations (site 1-the mouth of the Henderson Creek; site 2-the mouth of the Whau Creek) in the Waitemata Harbour. This harbour borders the highly urbanised and industrialised Auckland City metropolitan area. Whangaparaoa Peninsula, located approximately 30 km north of the other two collecting sites (away from the main urban area), was chosen as a reference site for comparative purposes.

Physico-chemical analyses revealed differences in water quality at the sampling sites. A lower pH, oxygen deficiency and higher temperature were recorded in both the Waitemata Harbour locations in comparison with the reference site.

Histopathological analyses revealed significantly higher prevalences and severity of pathological changes in the gills, blood, liver, kidney and gonads of the yellowbelly flounder from both harbour locations in comparison with fish from the reference site. In addition, some types of lesions (eg. neoplasms) were observed in fish from the two harbour locations only. Abnormalities in the gill structure of fish from both harbour sites included: epithelial swelling (hyperplasia and hypertrophy), necrosis, and lifting with oedema; the fusion of secondary lamellae; aneurysms; filamental deformities; mucous cell proliferation; and infestation by *Trichodina*. The abnormalities found in the blood of these

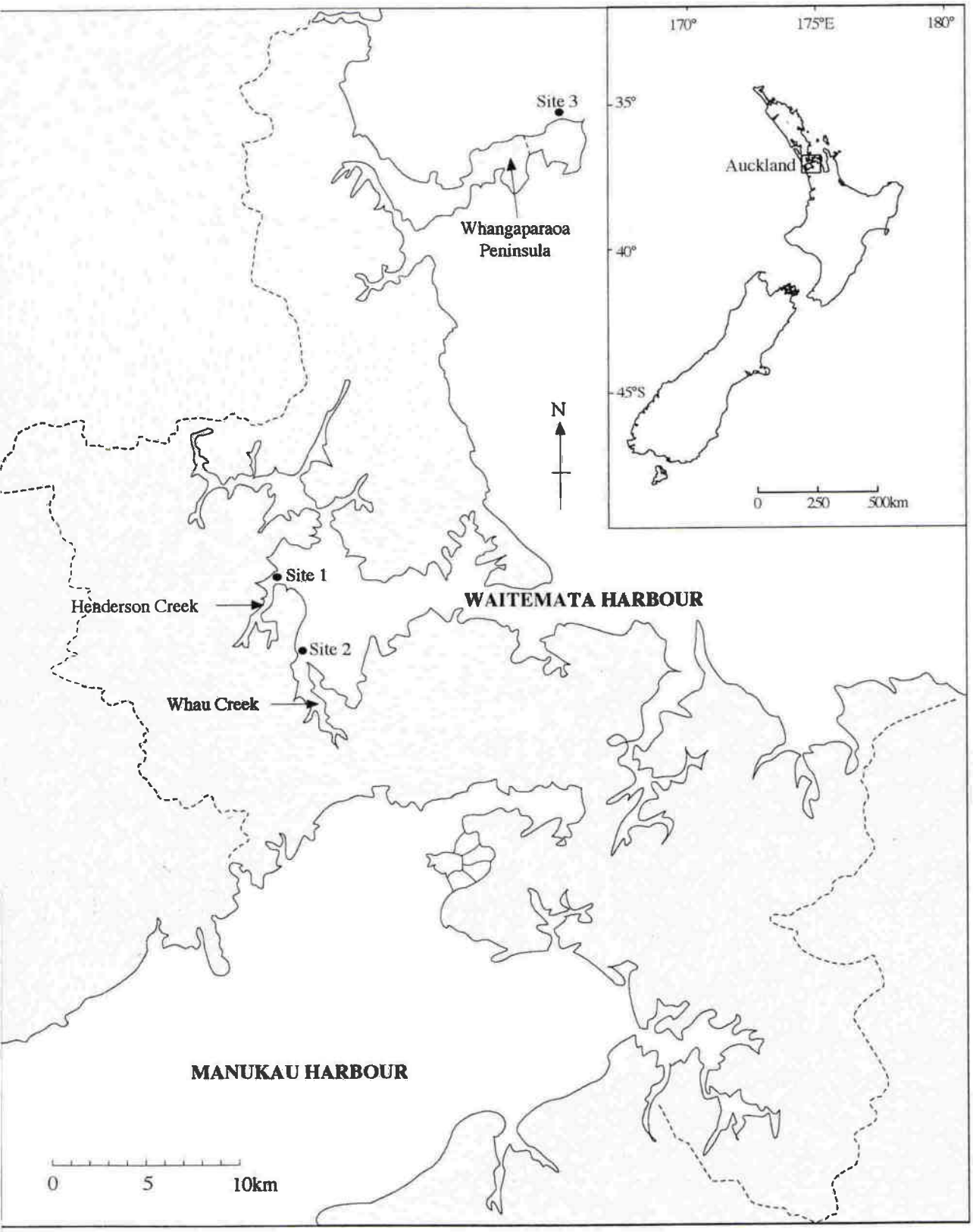
fish were manifested as: polycythaemia; erythrocytosis; erythroblastosis; leucocytosis (increased neutrophils); poikilocytosis; anisocytosis and an increased prevalence of erythrocytes undergoing necrosis. Vacuolar degeneration of the hepatocellular parenchyma due to lipid or glycogen accumulation was the most prominent liver change observed in fish from all sampling sites. The other liver abnormalities observed in flounder from the two harbour sites included: foci of cellular alterations (clear, basophilic and necrotic), congestion of the sinusoids, infestation by nematodes, and anaplastic growths (cholangiocellular carcinoma and teratoma). Pathological changes found in the kidneys of flounder collected at both harbour sites were classified as glomerular abnormalities (atrophy and dilatation of the glomerular tuft; enlargement of Bowman's space) and tubular vacuolar degeneration and necrosis. The presence of myxosporean parasites was also a common finding in the kidneys of harbour fish. Follicular atresia was the most prevalent change observed in the ovaries of flounder from the two harbour sites.

Biochemical analysis of plasma proteins and electrolytes of flounder from the three sampling sites revealed hyperbilirubinaemia, hypoalbuminaemia and uraemia in those inhabiting both harbour sites. In addition, the concentration of total liver microsomal proteins was significantly depressed in flounder from harbour sites 1 and 2 when compared to that of fish from the reference site. Concentrations of heavy metals in the livers of flounder from different sampling localities were found to decrease in the order: site 2 > reference site > site 1, and did not correlate with the prevalences of liver abnormalities.

Significantly more prominent pathological changes were thus observed in fish from both estuarine harbour sites in comparison with those from the reference open water site. The pathological changes noted are believed to occur in response to environmental changes. Contamination by different xenobiotics in the Whau and Henderson Creeks, which have been recorded in previous studies, suggest the possibility of direct toxic effects of the water contaminants on flounder from the estuarine parts of these creeks. In addition, the relatively high temperature, low pH and low oxygen levels recorded at the two sites in the

Waitemata Harbour are believed to have induced oxygen deficiency-related tissue hypoxia which could then have led to the expression of a variety of diseases of which some have been detected in this study. However, the possibility that some unknown and unmeasured causal factors may have produced the observed pattern of flounder diseases cannot be eliminated.

Figure 1 Auckland Region Map



---------- border of the Auckland metropolitan area

ACKNOWLEDGMENTS

My sincere gratitude is extended to my supervisor, Associate Professor Clive W. Evans for his excellent guidance, encouragement and immense patience he showed throughout this research.

I would also like to thank all the members of the Developmental Biology and Cancer Research Group, School of Biological Sciences for their support and friendship during the years. I am especially grateful to Jenny Rains for all her advice and understanding during my time at the School. Thanks are also extended to Kenny Goldie and Beryl Davies for their technical assistance in the pathohistological analyses, and to Mira Stojkovic for her help with microsomal protein analysis. I would like to thank Vivian Ward and Iain McDonald for their contribution in the making of the TEM and SEM photos. Special thanks to Dr Dianne Brunton for her advice and help with the statistical analyses.

Thanks to Dr Mike Timperley for his contribution towards the heavy metal analysis.

In the end, I would like to thank my family for all their support without which this thesis would not have been possible.

LIST OF ABBREVIATIONS

Abs. alc.	absolute alcohol
ATP	adenosine tri phosphate
ALT	alanine transaminase
ALB	albumin
AMY	amylase
α	alpha
AST	aspartate transaminase
β	beta
BIL	bilirubin
BSA	bovine serum albumin
Ca^{++}	calcium
CH	cholesterol
CF	condition factor
CK	creatine kinase
CR	creatinine
°C	degrees Celsius
DNA	deoxyribonucleic acid
DO	dissolved oxygen
EDTA	ethylene diamino tetra acetate
ER	endoplasmic reticulum
E	erythrocytes
FACs	fluorescent aromatic compounds
γ	gamma
GGT	gamma glutamyl transferase
g	gram
mm	millimetres
min	minute
M	molar
N	neutrophils
n	number of observations
%	percent
PO_4	phosphate
PCBs	polychlorinated biphenyls
PAHs	polycyclic aromatic hydrocarbons
K^+	potassium
RBCC	red blood cell count
rpm	revolutions per minute
RNA	ribonucleic acid
RER	rough endoplasmic reticulum
sec	seconds
S.T.W.S.	Scott's tap water substitute
SEM	scanning electron microscopy
SER	smooth endoplasmic reticulum
Na^+	sodium

H/E	haematoxylin/eosin
Hct	haematocrit
hr	hour
l	litre
LSI	liver somatic index
M/H	Mallory/Heidenhain
µg	micrograms
µm	micrometers
mg	milligrams
ml	millilitres
NaCl	sodium chloride
s. d.	standard deviation
TEM	transmission electron microscopy
TR	tryglicerides
Tris HCl	tris hydrochloric acid
TPP	total plasma proteins
UA	uric acid
WBCC	white blood cell count

TABLE OF CONTENTS

LIST OF TABLES

2. 0. MATERIALS AND METHODS

Appendix 6. Coomasie blue protein assay (Bradford, 1976)

3. 0. RESULTS

3.1 Physico-chemical water parameters

3.2. Biological parameters

3.3. Histopathological analyses of the gills, blood, liver, kidney, gonads and the gall bladder

3. 3. 1. Gills

3.3.2. Blood

3. 3. 3. Liver

3. 3. 4. Kidney

3. 4. Biochemical analyses

3. 4. 1. Plasma proteins and electrolytes

3. 4. 2. Liver microsomal proteins

3.4.3. Heavy metals

LIST OF FIGURES

3.3.2. Blood

3.3.3. Liver