

INTERNATIONAL JOURNAL OF WHITE SEEDS
(WHITE SEEDS) 2007
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Histopathological alteration of white seabass (*Lates calcarifer*)
gills in subacute zinc exposure / Juliana Mohamed.



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PERPUSTAKAAN SULTANAH NUR ZAHIRAH UMT

**HISTOPATHOLOGICAL ALTERATION OF WHITE SEABASS (*LATES
CALCARIFER*) GILLS IN SUBACUTE ZINC EXPOSURE**

BY

JULIANA BT MOHAMED

**Research report submitted in partial fulfillment of the requirement for the degree of
Bachelor of Science (Marine Science)**

**Department of Marine Science
Faculty of Maritime Study and Marine Science
UNIVERSITY MALAYSIA TERENGGANU
2007**

1100054338

Juliana M., 2006. Histopathological of White Seabass (*Lates calcarifer*) Gills in Subacute Zinc Exposure. Undergraduate Thesis, Bachelor of Marine Science, Faculty Maritime Study and Marine Science, University Malaysia Terengganu, Terengganu.

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Adalah ini disahkan bahawa laporan penyelidikan bertajuk:

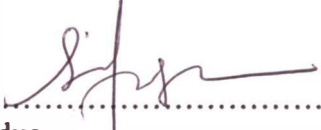
Histopathological Alteration of White Seabass (*Lates calcarifer*) gills in subacute zinc exposure oleh **Juliana Binti Mohamed**, No. Matrik **UK 10142** telah diperiksa dan semua pembetulan yang disarankan telah dilakukan. Laporan ini dikemukakan kepada Jabatan Sains Marin sebagai memenuhi sebahagian daripada keperluan memperoleh Ijazah Sarjana Muda (Sains Samudera) Fakulti Pengajian Maritim dan Sains Marin, Universiti Malaysia Terengganu.

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ACKNOWLEDGEMENTS

First and for most, I would like to take this golden opportunity to express my sincere gratitude and appreciation to my supervisor, Prof. Dr. Noor Azhar bin Mohd Shazili and also my co-supervisor Dr. Hing Lee Siang for their continuous comments and patience's throughout the course of the project.

I would also to thank to all Marine Hatchery staffs and laboratory assistances, En. Yaakob, En. Suhaimi, En. Kamari, En. Kamarul, En. Sulaiman who are always help me in completing my laboratory analysis. For all the assistance and help from the Institutes of Oceanography staffs En. Nasir, En. Subarjo and Puan Norita, I would like to extend my gratitude.

I wish to thank my parents and family for their support and loving encouragements as well as the tower of strength in my hour of needs.

Last but not least, I cannot adequately express my gratitude to all my course mates and all family of Student Representative Councils who made these years of study a most memorable one.

ABSTRACT

A study was conducted on seabass fingerlings (*Lates calcarifer*) to determine the histopathological alteration of subacute zinc exposures. The experiment was also to determine the lethal concentration of zinc to the seabass fingerlings and the bioaccumulation of zinc to white seabass fingerlings. Toxicity tests were conducted by seabass fingerlings were exposed to nominal zinc concentration of 0.56, 1.0, 3.2, 10.0, and 32.0 mgL⁻¹ with one control (no zinc exposure) in two replicates. The 96hr LC₅₀ values of zinc for seabass was found to be 5.8135±0.3019 mgL⁻¹. The subacute zinc exposures conducted in 0.01, 0.1 and 0.25 % of the 96h LC₅₀ value and control (not exposed to zinc) in two replicates. The bioaccumulation of zinc in seabass showed complex pattern due to high zinc concentration in the water sources. Gills histopathology changes showed the gills damage consist of tissue edema, clubbing of lamellar, swelling and enlargement of lamellar, hyperplasia of lamellar, tissue rupture and fusion of secondary lamellar become more prominent.

ABSTRAK

Kajian kesan histopatologi insang dijalankan ke atas anak ikan siakap putih (*Lates calcarifer*) di dalam zink separa akut. Kajian juga ini dijalankan untuk mengetahui kepekatan zink yang boleh membawa kematian kepada anak ikan siakap putih dan kadar bioakumulasi zinc oleh anak ikan siakap putih. Ujian ketoksikan telah dijalankan ke atas anak ikan siakap pada kepekatan zink 0.56, 1.0, 3.2, 10.0, 32.0 mgL⁻¹. Nilai 96hr LC₅₀ zink didapati ke atas anak ikan siakap ialah yang didapati adalah 5.8135 ± 0.3019 mgL⁻¹. Selepas itu, kajian zink separa akut dijalankan dengan menggunakan kepekatan zink 0.01, 0.1 and 0.25 % daripada nilai 96hr LC₅₀. Kadar bioakumulasi zink oleh anak ikan siakap menunjukkan satu keputusan yang kompleks kerana kandungan zink yang sedia ada di dalam punca air kajian. Kajian histopatologi pula menunjukkan bahawa pada pendedahan logam berat ke atas anak ikan siakap telah menunjukkan kerosakan pada insang anak ikan siakap dimana terdapat edema, perlekatan lamella, pembengkakan, pembesaran lamella dar saiz asal, hyperplasia pada lamella dan juga kecederaan serta kerosakan pada lamella sekunder.