

# A STAIN UPON THE SEA

## THOUGHTS PROVOKED and CORRECTIONS

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On December 16, 2004, I attended the book launch hosted by the Friends of Clayoquot Sound (FOCS) for the anti-salmon aquaculture west coast salmon farming collage, "A Stain Upon the Sea".

The title of the book, especially as applied to salmon farming in BC by present day operators, is not descriptive of conditions and practices within the industry. In particular, there are blatant errors in fact, extrapolation and omission which should be corrected by the publisher before any further circulation occurs. I believe that the sentiments expressed by Terry Glavin and Stephen Hume are genuine in intent as is their concern for the well-being of wild salmon. Certainly it is obvious, however, that individuals of the stature of Glavin and Hume had little to do with the final editing.

Although the book is about west coast salmon farming, Don Staniford's contribution gives an over-dramatized historical world-wide view of the uses of carotenoids and therapeutic chemicals in salmon aquaculture. Of the ten compounds featured in "Silent Spring of the Sea" only three are of any concern in Clayoquot Sound. Creative Salmon Company Ltd., which produces farmed chinook according to organic principles, does not use antibiotics in market fish. It uses only the carotenoid astaxanthin in salmon rations. It has had, in sea lice monitoring, extremely low sea lice levels which do not trigger mandatory treatment with SLICE. As well, Creative Salmon has developed net cleaning protocols which have allowed the nearly complete phasing out of nets which have ever been treated with copper antifoulants.

Wild salmon get their pink and red colours from the wild food chain in the form of astaxanthin, which is the principal carotenoid in wild salmon flesh, as well as lesser amounts of a number of other natural carotenoids including canthaxanthin. These tetraterpenoid compounds are among the 300-odd closely related carotenes, carotenoids and xanthophylls known in nature, most of which are highly coloured. In the marine food chain these compounds are synthesized by marine algae where some of them play an essential role in photosynthesis. Carotenes and carotenoids enter the salmon food chain via crustaceans which consume marine algae or via forage fish which have consumed crustaceans.

In salmon nutrition, as in the case of all other animal forms, the carotenes, especially B-carotene are precursors of vitamin A, which is essential for life, including vision. The carotenoid pigments in salmonids are implicated in reproduction, and quality and survival of fry, as well as being natural antioxidants. In human diets, carotenoid pigments derived from the consumption of salmon are one of the natural sources of beneficial antioxidants.

In the preparation of fishmeal and other dry ingredients used in farmed salmon rations most of the coloured carotenoids in the raw feed ingredients are destroyed by heat and oxidation, necessitating supplementation of salmon diets with carotenoids to provide normal colouration in farmed market fish, and to meet the nutritional requirements of the fish. Canthaxanthin and astaxanthin can be supplied from natural sources or as synthetic product as in the case with vitamin supplements. The amount of dietary carotenoids supplied to farm salmon are designed to provide normal physiological concentrations (5-15 ppm) found in the flesh of wild salmon. The use of higher levels than these are physiologically unjustifiable. While some salmon production companies use only astaxanthin in their rations, other companies favour the use of a mixture of astaxanthin and canthaxanthin.

Canthaxanthin is an example of a physiological compound which is essential or at least benign within a normal, physiological concentration range, but can become toxic at higher concentrations. Vitamin A, Vitamin E, selenium, sodium chloride, many other nutrients, essential minerals and ethanol, are also in this category. Vitamin A is essential for life and

normal vision, but hypervitaminosis A toxicity can occur from excessive consumption of cod liver oil or other concentrated natural sources of Vitamin A such as seal or polar bear liver.

The consumption of wild or farmed salmon containing canthaxanthin and astaxanthin at normal flesh levels has never been shown to have adverse effects on human health. Retinal toxicity due to the use of mega-quantities of canthaxanthin for tanning purposes has nothing to do with the consumption of farmed salmon, and constitutes a substance abuse. Interestingly, some of the popular pharmaceutical W-3 fatty acid supplements made from wild sockeye oil contain enough astaxanthin to cause noticeable tanning.

With regard to non-antibiotic chemotherapeutants, the prime target here, of course, is SLICE, which is not needed for Chinook operations in BC. Although Atlantic operators in BC have tested various delousing compounds and protocols, sea lice infestations from an animal husbandry point of view seem to be inherently much less of a problem in BC than in the Atlantic basin. This may be due to differences between the Pacific and Atlantic strains of *L. salmonis*. An example of such differences in virulence is provided by VHS virus which is endemic in herring in both oceans. The VHS strain carried by North Sea herring is devastatingly virulent for rainbow trout, where the Pacific strain of VHS found on pilchards as well as herring has not been seen on farmed salmon and is very rarely found on wild Pacific salmonids. BC operators have learned to control lice by fallowing, "all-in" and "all-out" site management, and improved nutrition and other husbandry practices which decrease stress and produce shorter production cycles.

Over-use of SLICE in BC in order to adhere to arbitrary European standards is not environmentally sound, since this increases both the danger of resistance development by sea lice as well as the possibility of collateral damage. SLICE should be used sparingly as an adjunct to preventive management calibrated to BC conditions. This calibration is emerging from the objective analyses of the database developed from ongoing studies in the Broughton Archipelago and other areas such as Clayoquot Sound. There are also promising indications that significant control of sea lice on Atlantic salmon will be possible by vaccination.

Although Don Staniford did not discuss PCBs and related environmental contaminants in his contribution to "A Stain Upon the Sea", there is a chart in his section (page 197) which presents "Average PCB levels found in BC salmon". This chart, which shows PCB levels of 4 ppb and 34 ppb for wild and farmed salmon, respectively, summarizes data published in "Science" in 2004 from a study conducted in the US supported by the PEW Charitable Trusts. The control (wild) samples used included mature eviscerated pink and chum salmon which contain little fat compared to the farmed salmon which are harvested as more immature fish. As salmon mature, fat and fat-soluble contaminants are transferred to the maturing gonads which are removed in the viscera. Although the authors of the PEW study all appeared to be eminent scientists, they strangely did not review other published data on PCBs in Alaskan, Puget Sound and Columbia River wild coho, chinook and sockeye which revealed PCB levels similar or higher than the values reported in "Science" for farmed fish. None of these PCB levels represent a risk to human health, but are a wake-up call in that the world environment is now sufficiently polluted that low but detectable amounts of these contaminants are found in most human food stuffs, including salmon. Of course the misinformation displayed on page 297 of SUS has been used prominently in the CAAR campaign to discourage consumption of farmed salmon.

Mr. Staniford seems to consider all fish farm wastes as "contaminated waste". Unlike municipal sewage, salmon feces do not contain paper products, household cleaners, pharmaceutical residues, and detergents, some of which have estrogenic side effects on fish, and fecal coliforms which are not produced by salmon. Concern about the impacts of salmon farm waste in Clayoquot Sound should also include careful consideration of the composition and volume of the Tofino municipal effluent, which at present is completely untreated.

For sustainability, fecal production of the farms should equal the rate at which the fecal material is metabolized in seawater. Seawater has a large capacity to metabolize fish wastes, especially under aerobic conditions. This equality is achieved by careful feeding which is monitored with underwater cameras, correct feed formulation and benthic oxygenation, if natural flushing does not maintain aerobic conditions in sediments beneath salmon pens.

It is ridiculous to require that salmon farms guarantee fish farm waste is not released to the ocean. However, it is possible to not to have residues of noxious chemicals and to stay in equilibrium with the metabolic capacity of the environment. It should be remembered that wild salmon also produce waste. One million wild sockeye produce approximately 1000 tonnes of fecal dry matter and metabolic waste during their life cycle.

Benthic residues of copper and zinc are also mentioned in chapter 4 of SUS. Both copper and zinc are trace nutrients for salmon (as in humans) and copper takes the place of iron in the oxygen transport system used by crustaceans. Copper and zinc form insoluble carbonates in seawater, and very insoluble sulfides if they become part of an anaerobic benthos. Excess copper from net washing offshore can be trapped quite easily by settling and subsequent removal of settled net washing solids, including cuprous oxide from treated nets, for land disposal.

### **Diseases and Parasites**

The focal point of concepts of disease and parasite dynamics in BC is the Broughton Archipelago sea lice issue. Initially, it was very easy to relate the population crash of the 2000 year class pinks to the presence of farmed salmon (deemed to be the only possible sea lice reservoir) according to the popular curtain of death theory (COD), in which naïve pink and chum fry are overwhelmed by infective juvenile sea lice as they migrate past salmon farms. Subsequent study results suggest strongly that the primary determinants of pink and chum fry survival in the Broughton are stocking densities and the feed supply for wild smolts/fry in the inshore nursery pastures. Very high levels of lice can exacerbate the lethality of starvation when very high stocking densities of fry overwhelm the feed supply, as in the spring/summer of 2001. In contrast, in 2003, the progeny of the remnant 2002 year class adult pink escapement, which numbered less than 5% of the normal even-year class fry numbers, produced an astonishing 895,845 returning adult pinks in 2004. Similarly, pink and chum fry studied in 2004, which carried far more lice (warm, dry spring and summer, high salinity) than in 2003 appeared to be growing normally while carrying lice loads which were almost ten times greater than the "lethal" level (1.6 lice/gram) worked out

by Europeans with Atlantic salmon. The high level of lice in 2004 occurred in spite of rigorous monitoring and treatment of farmed fish for lice. Although farmed fish can be a reservoir for lice and are certainly a component of the environment, the levels of lice found on migrating pink and chum smolts suggest contacts with environmental sea louse reservoirs that were not being treated. DFO in 2003 identified a large standing biomass of marine sticklebacks as a reservoir of *Calligus Clemensii*, while more recent DFO studies have demonstrated groups of immature wild salmonids which over-winter in the inshore areas of the Broughton and which carry *Lepeophtherius salmonis*.

Although the Morton group has hailed the 2004 escapement rebound as evidence of the effects of fallowing the Broughton area in 2003, this is unlikely since the highest lice infestation rates seen on migrating pink and chum fry in 2003 were in the fallowed section. An examination of the standing biomass of farmed salmon in the Broughton archipelago in 2001, 2002, and 2003 revealed differences between years of less than  $\pm 2\%$ , whereas the sea entry-to-returning adult survivals of pink fry leaving archipelago rivers in 2001 and 2003 were 0.027% and more than 20%, respectively. This difference of approximately 3 orders of magnitude cannot be explained by fallowing or the presence of different numbers of sea lice. It is, however, energetically viable to suggest that these survival numbers are the expected result from a gross insufficiency of the feed supply relative to fry numbers in 2001 compared to an unlimited feed supply relative to fry numbers in 2003.

To summarize, an entirely louse-centered (curtain of death) approach to pink smolt dynamics in the Broughton area cannot explain the apparently conflicting results of 2001 and 2002 compared to 2003 and 2004. However, the data (including Alexandra Morton's) are very consistent with feed and density limiting primary dynamics where sea lice are an opportunistic secondary agent. The net conclusion is that pink and chum fry can grow normally in the presence of lice infestations much higher than those found to be lethal in European studies on Atlantic salmon and brown trout, provided they have a sufficient feed supply. This does not imply that there is no impact from a high infestation of sea lice, but rather that the fish can compensate for their presence as long as the feed supply is adequate.

One further anomaly not explained by the "curtain of death" theory is that while all of the studies suggest that the wild pink and chum fry/smolt acquire significant lice loads very soon after entering salt water, which increase as the migration proceeds seaward (and past the farming areas), cultured Atlantic smolts stocked in fallowed sites which are not very far from other operating farms typically require as much as a year to acquire a lice load sufficiently large to require mandatory treatment.

Although it is appropriate to manage farmed salmon in order to minimize the incidence of sea lice, pink salmon fry/smolt dynamics in the Broughton system demonstrate the need to manage and prevent excessive adult escapement in order to avoid density related population crashes in the future.

It is disappointing that, in her discussion of sea lice in the Broughton Archipelago beginning on page 224 of "A Stain Upon the Sea", Alexandra Morton was unable to mention, even in her epilogue for 2004, the DFO data of 2003 or the incredible rebound escapement of the 2002 year class which returned to the Broughton to spawn in 2004. Through these omissions, Morton has been able to maintain for gullible readers a gloom and extinction scenario for the Broughton system due to salmon farms. On page 234, she mentions that she has derived no joy from being right about the pink population crashes of 2001 and 2002. Had she been right, pinks and chums in the Broughton would now be truly extinct. Fortunately, she was wrong and the predicted extinction did not occur, in spite of the continued presence of about the same biomass of farmed salmon as in 2001. Furthermore, the data now in hand, including Morton's over the past four years, are leading to an understanding of the mechanisms of pink salmon population peaks, crashes and rebounds which have always characterized recorded pink salmon dynamics. This understanding can be the basis of integrated management of both farmed fish and wild pinks and chum in the Broughton system.

With respect to other diseases, IHN is endemic in wild fish on the BC coast, including Clayoquot and Barkley Sounds. IHN is carried by herring, sockeye, kokanee and chinook. (The Robertson Creek chinook have historically demonstrated a 2% incidence of IHNV carriers). Mainstream encountered an IHNV outbreak in the recent past but certainly did

not bring IHNV to Clayoquot Sound as announced by Mr. Stanford at the book launch. As in the case with other outbreaks of IHNV in farmed fish on the BC coast which have appeared to originate with an environmental challenge, such as carrier herring, chinook and sockeye, there has been no evidence that IHNV has been passed back to wild fish as a result of infection on farms. Since vaccines for IHNV have had variable efficacy and chemotherapeutants are ineffective, nutritional immune enhancement, density and stress management and rigid biosecurity protocols are the principal current mechanisms to date for prevention and control of IHNV. Biosecurity considerations are the principal reasons why farms have not encouraged tours of their facilities, especially in the case of individuals such as Mr. Staniford who have recently been active in other jurisdictions which have viral fish diseases (IPN and ISA in Scotland) that do not exist in BC. We do not want either wild or domestic salmon in BC to be contaminated by these exotic viral diseases. The devastation of the Fraser Valley Poultry industry by avian flu last spring is still a recent memory, and the Canadian beef industry still remembers the year 1952 when a German immigrant farm worker inadvertently brought foot and mouth disease to Saskatchewan on his shoes, resulting in the slaughter of thousands of head of cattle to contain the outbreak.

The discussion of bacterial diseases (pages 202-206 in SUS) draws very circumstantial and tenuous connections between outbreaks of furunculosis and bacterial kidney disease in farmed salmon and mortalities or disappearance of wild chum, coho and chinook in the Broughton area. I can give some perspective from personal experience.

Firstly, the description of the apparently precipitous, total loss of Viner River chum in 1989 does not fit the typical description of a BKD outbreak. The SUS description does not relate whether any of these chum mortalities were in fact checked for BKD, but it would have been much more likely that if these fish had been heavily infected with BKD at least some of them would have spawned successfully. A complete kill of broodstock is much more reminiscent of a heterosigma bloom, such as the one in 1986 which killed most of our near-mature domestic chinook at Genoa Bay (Cowichan Bay area), but was much less devastating to younger year classes held at the same site. In any case, chum infected environmentally with BKD do not all die prematurely. In November 1999, my curiosity about the incidence of BKD in local chum runs led to the collection of a small random



sample (five fish) of freshly dead, completely spawned, normal appearing chum from the lower Chemainus River and five more similar chum from Goldstream. These fish were delivered to a private diagnostic laboratory for examination for viral and bacterial pathology. No virus was detected, but 6/10 (3/5 from each river source) gave positive tests for BKD.

In the late 1970s, Apex Bio Resources Ltd. began to expand coho and chinook production with the establishment of farm sites in the Alberni Canal which is characterized by having fresh water surface lens due to the influence of the Somass River drainage. Coho and chinook from disease-screened wild eggs (Big Qualicum or Robertson Creek) were raised in ground water hatcheries at Great Central Lake and Westholme and transferred to small (6.5 m x 6.5 m) net pens in the Alberni sites at high commercial rainbow trout densities. Although the fish had no evidence of the diseases in their hatchery experience, they almost immediately began to die in the brackish water net pens. Marine vibrio infections responded well to antibiotics and then to the crude vaccine preparations. Furunculosis was initially responsive to oxytetracycline, but within three weeks of the initial treatments an oxytetracycline-resistant strain of furunculosis developed. This experience, repeated many times, demonstrated the futility of treating furunculosis in market fish with antibiotics and turned our attention to the use of vaccines which at that time were in the early stage of development. Although vertical transmission of BKD can be controlled by bacteriological screening of broodstock at spawning, horizontal transmission of BKD produced chronic mortality in the early Alberni Inlet sites. Mortality in market chinook and coho, due to all bacterial causes, decreased dramatically with the adoption of the much lower net pen stocking densities that are now standard practice.

Besides the demonstration of antibiotic-resistant furunculosis, the Apex experience also demonstrated the existence throughout the Alberni Canal and eastern Barkley Sound of an environmental challenge of both furunculosis and BKD due to the influence of the Somass River and other streams entering those waterways. Similarly, there is a natural furunculosis and BKD challenge in Clayoquot Sound due to the influence of the Kennedy River. It is almost certain that similar furunculosis and BKD challenges exist naturally in the Broughton system. Farmed chinook reared under present conditions in Clayoquot Sound are not

significantly susceptible to furunculosis and are not normally vaccinated against this organism. Atlantic salmon are much more susceptible to furunculosis, and successful production is entirely dependent on the vaccination of all Atlantic smolts used in the industry.

In hindsight, it is not surprising that furunculosis occurred in the first groups of Atlantic salmon reared in the Broughton area. Many of these fish were not vaccinated, as the high efficacy vaccines currently used were not available. It is also not surprising that antibiotic-resistant strains of furunculosis developed in farmed atlantics due to repeated treatments with antibiotics. These strains are not necessarily more virulent, but become untreatable with the antibiotics to which resistance has developed. When the use of antibiotic is terminated, there is no further selection pressure for the resistant strains and they eventually revert to pre-treatment background levels.

In the SUS story about furunculosis in the Scott Creek Hatchery in 1992, the coho broodstock responded, at least initially, to oxytetracycline which suggested that the strain of furunculosis was not one of the oxytetracycline-resistant strains which had occurred on the salmon farms, although one might expect a resistant strain to develop locally as a result of the treatment.

Similarly, given the low susceptibility of chinook for furunculosis, it is very unlikely that the described disappearance of Kingcome Inlet chinook in 1993 was due to furunculosis, antibiotic-resistant or not. If this had been the case, some fish would have been seen with some evidence of the disease.

Concerns about the establishment of Atlantic salmon in Pacific salmon habitat are expressed in several sections of SUS. Certainly the survival of escaped Atlantics, some to maturity, has been amply demonstrated, but with the exception of a very small number of apparently feral juvenile Atlantics found in two Vancouver Island streams by John Volpe, no feral Atlantics have been seen in the Atlantic salmon watch program which has operated for over ten years. The watch program has examined several thousand captured Atlantic

salmon, which is a highly valid statistical sample when integrated over the period of the program.

Oncorhynchus (Pacific salmon, including Pacific trout) began to evolve from the original *Salmo* approximately 20 million years ago, and the original *Salmo* genotype in the Pacific basin became extinct. Although it is not possible to say exactly why *Salmo* did not persist, comparisons between *Oncorhynchus* and cultured Atlantic salmon reveal differences which suggest why the Pacific salmon have been the more successful environmental competitors. The characteristics of the Pacific fish include shorter incubation periods in relation to temperature and much higher juvenile growth rates at ambient BC spring water temperatures. Their larger eggs lead to larger swim-up fry. Their one-way spawning migrations allow all body reserves to be expended in returning to the fresh water spawning areas, followed by post-spawning death in fresh water which results in massive transfer of marine nutrients into terrestrial watersheds, ensuring the feed supply for the next generation. This process probably resulted in the increased growth rates of juvenile *Oncorhynchus* compared with juvenile *Salmo*. A corollary of the one-way spawning runs and subsequent death is that very large challenges of endemic fish pathogens occur in Pacific rivers at spawning time. Exposure to these pathogens over millions of years has resulted in a higher resistance by Pacific than Atlantic to these pathogens. The Atlantic do not significantly fertilize their natural watersheds and have never had to adapt to these Pacific disease challenges. Although present day Atlantic salmon probably look different from the *Salmo* of 50-20 million years ago, the modern Atlantic genotype appears to be little different from that derived by retro extrapolation of present day *Oncorhynchus* genotypes which differ markedly from *Salmo*. The modern farmed Atlantic appears to be an ancient fish which, although a good domestic "broiler", has not made the environmentally necessary adaptations of the Pacific salmon for the whole life cycle survival in the Pacific basin. On the other hand, the Atlantic salmon evolved in its native range with fewer fresh water competitors, and certainly in the absence of Pacific salmonids.

There is absolutely no support for the statement that the cultured Atlantic salmon is the "most aggressive" of the salmonids. In fact, co-culture data at ambient temperatures with coho and Pacific trout, spawning competition trials between farmed Atlantic and wild

Pacific broodstock, and the inability of Atlantic to compete with introduced chinook and coho in the Great Lakes, equally suggest that farmed Atlantic are less environmentally aggressive than Pacific salmonids. In Europe, rehabilitation programs to reestablish wild Atlantic in rivers must first eradicate the naturalized rainbow trout before the Atlantic will reestablish. The terms "most aggressive", "invasive", and "voracious" are certainly a source of amazement to fish culturists who grow domestic Atlantic salmon, especially those who also grow Pacific salmon. Although there are many examples of truly invasive exotic species, the Atlantic salmon, in the Pacific Ocean at least, is an exception.

A concern expressed several times in SUS, as well as in the FOCS book launch presentation, is that the use of fish meal and fish oil in aquaculture feeds, especially for salmon, depletes the world supply of ocean-produced fish protein in order to produce a high-end product for the world's affluent. Salmon, and many of the other temperate zone aquaculture species, are unquestionably carnivores in their wild state, requiring 8-12 kg of wild forage fish to produce 1 kg of wild carnivore. This conversion includes the requirement to maintain a sustainable wild broodstock. Farmed salmon are also carnivores, but are subject to regimes of ration formulation which increasingly force them to become de facto herbivores. Also, due to high survival rates, brood stock requirements are only about 2-3% of production biomass compared to approximately 50% for the sustainability of wild salmonids. At the present time, with the present availability and price of fish meal and fish oil, the forage fish-to-live farmed salmon conversion is between 2 and 3 kg of forage fish/kg of farmed salmon. This represents a conversion of a fisheries resource which is not presently in demand directly as human food into a product that is very high quality human food. The production of farmed salmon is at present much more conservative of the world forage fish resource as human food than is the natural production of wild carnivores or for that matter, the use of fish meal in poultry or swine rations.

The trend in fish meal use in farmed salmon rations is that the levels presently in use are about 50% lower than levels used a decade ago, and recent nutritional research on the effective use of fish meals and increasing usage of terrestrial protein sources has demonstrated that forage fish-to-live farmed salmon conversion can be close to 1:1.

In the future, as the human population inexorably increases, it is certain that an increasing proportion of high quality fish meals and oils will be used directly in the human food supply and the expanding aquaculture industry will reach new and much more complex nutritional and environmental equilibria. A major component of this process which is consistently misunderstood and under-estimated by many environmentalists and biologists is the technical capability and adaptability of the European and North American feed manufacturing industries.

With respect to salmon aquaculture in BC the feed for farmed salmon does not impact the food chain of the wild BC salmon and the industry has the capability to expand carefully as long as separate marine and terrestrial nutrient supplies are not limiting. On the other hand, the sustainability of increased wild salmon production or even the maintenance of present stocks will probably be increasingly difficult, especially as global warming proceeds. It is also certain that the expansion of the wild fishery in other jurisdictions (such as Alaska) through increased ocean ranching will eventually, if not already presently, impact the food chain of the wild BC salmon.

### **Industry Obligations**

The salmon farms in Clayoquot Sound have an obligation to persist so that the myth that salmon aquaculture as practiced in BC and especially Clayoquot Sound is threatening to the wild fish resource is dispelled once and for all. We are obligated to demonstrate this to BC coastal residents, and particularly First Nations people who may very well be the primary aquaculturists within a generation.

In this country we have all grown up with a developing sister shellfish industry (remember, the BCSFA unilaterally banned TBT as soon as it was demonstrated that we were damaging shellfish in 1984-85), and we have all grown up with the wild commercial and sport fisheries, which are the other two essential components of the BC fishing industry. We must all remember that the wild salmon are a national heritage and are the mechanism by which Canada and BC harvest the free fish pastures of the North Pacific Ocean and our own territorial waters.

Salmon aquaculturists in BC are not fighting for excessive or inappropriate aquaculture in other jurisdictions, but are striving to develop a healthy, sustainable wild fishery and farmed industry on the BC coast. The aquaculture component of the BC salmon industry has not been shown to be deleterious to the wild component. Wild production, although variable, cannot be easily expanded, whereas farmed production can expand, at least moderately, in BC subject to the supply of local marine byproducts and marine and terrestrial nutrient sources separate from the food chain of wild BC salmon. It is not only possible, but necessary to have both healthy wild and farmed salmon production in BC in order to optimize a viable salmon production industry on the BC coast. Fine tuning of the relationship between farmed and wild components requires real solutions based on accurate environmental information, neither of which seems able to be contributed by the so-called E-NGOs. These anti-salmon farming groups who lack the ability to actually enhance wild salmon or to produce farmed ones, have developed a Lepeophtheiroid relationship to the salmon farming industry by which they have learned to earn a living by conducting anti-farming campaigns based on the scientifically unsupportable dogma that wild and farmed salmon production in BC are biologically incompatible. "A Stain Upon the Sea" was designed to be a component of this campaign. The preface and first two chapters provide a readable restatement of issues and history. This first part is not matched by the surreal attempts in the last three chapters to prove the dogma. Far from being a collage of brilliant essays as announced in its preface, the book ends up a misleading pot-boiler.