REVIEWS IN Aquaculture

Reviews in Aquaculture (2014) **6**, 162–179 doi: 10.1111/raq.12039

Salmon welfare index model 2.0: an extended model for overall welfare assessment of caged Atlantic salmon, based on a review of selected welfare indicators and intended for fish health professionals

Jostein M. Pettersen[1], Marc B.M. Bracke[2], Paul J. Midtlyng[1], Ole Folkedal[3], Lars H. Stien[3], Håvard Steffenak[4] and Tore S. Kristiansen[3]

1 Norwegian School of Veterinary Science, Oslo, Norway
2 Wageningen UR Livestock Research, Lelystad, The Netherlands
3 Institute of Marine Research, Bergen, Norway
4 Norwegian Food Safety Authority, Brumunddal, Norway

Correspondence
Jostein M. Pettersen, Norwegian School of Veterinary Science, P.O. Box 8146 Dep, N-0033 Oslo, Norway.
Email: jostein.pettersen@nvh.no

Received 8 March 2013; accepted 12 May 2013.

Abstract

Here, we present an extended version of a semantic model for overall welfare assessment of Atlantic salmon reared in sea cages. The model, called SWIM 2.0, is designed to enable fish health professionals to make a formal and standardized assessment of fish welfare using a set of reviewed welfare indicators. SWIM 2.0 supplements SWIM 1.0, which was designed for application by fish farmers. We searched the literature for documented welfare indicators that could be used by fish health professionals. The selected indicators are eyes, cardiac condition, abdominal organs, gills, opercula, skeletal muscles, vaccine-related pathology, aberrant fish, necropsy of the dead fish and active euthanasia. Selection criteria for the SWIM 2.0 indicators were that they should be practical and measureable on salmon farms by fish health professionals and that each indicator could be divided into levels from good to poor welfare backed up by relevant scientific literature. To estimate each indicator's relative impact on welfare, all the indicators were weighted based on their respective literature reviews and according to weighting factors defined as part of the semantic modelling framework. This was ultimately amalgamated into an overall SWIM 2.0 model that can be used to calculate welfare indexes for salmon in sea cages, taking into account the available fish health expertise. Using this model, an example calculation based on recordings and samplings done from an Atlantic salmon sea cage containing 106 000 fish yielded an overall welfare index of 0.81 of a maximum of 1.0.

Key words: animal welfare score, aquaculture, diagnostic, fish health, sea cage, welfare indicator.

Introduction

In a previous study, we presented a welfare assessment model (SWIM 1.0) for Atlantic salmon (*Salmo salar* L.) reared in sea cages, primarily designed for salmon farmers (Stien *et al.* 2013). SWIM 1.0 was based on the biological and welfare needs of the species and formulated welfare indicators that could be scored separately, weighted and integrated into an overall welfare assessment (OWA). The selected SWIM 1.0 indicators were water temperature, salinity, oxygen saturation, water current, stocking density, lighting, disturbances, daily mortality, appetite, sea lice infestation, body condition, emaciated fish, vertebral deformation, maturation stage, smoltification state, fin condition and skin condition.

This paper describes an extended version of the model (SWIM 2.0), a health-oriented supplement to SWIM 1.0, designed for fish health professionals. The model's name SWIM is an acronym for Salmon Welfare Index Model. No 2. states that this is the fish health professionals' version of SWIM and .0 indicates that it is the first version that may be upgraded later. The main objective of SWIM 2.0 is to

supply a standardized methodology for fish health professionals and representatives of food and aquaculture authorities to assess salmon welfare in sea cages.

How to assess the welfare status of fish is an ongoing debate, and no consensus has been reached on definitions or assessment methodology (Huntingford & Kadri 2008). Both SWIM 1.0 (Stien *et al.* 2013) and 2.0 were constructed using the principles of semantic modelling introduced by Bracke *et al.* (1999a)Bracke *et al.* (1999b)Bracke *et al.* (1999c). In semantic modelling, welfare is defined as the quality of life as perceived by the animals themselves, and both positive and negative aspects of welfare are taken into account. Semantic modelling is designed for the purpose of formalized assessment of animal welfare based on available scientific information, including scientific knowledge and scientific descriptions of welfare-relevant aspects of holding systems in terms of both environment-based and animal-based measures (Bracke *et al.* 2008). It was originally developed for assessment of housing systems for dry sows (Bracke *et al.* 2002a,b), but it has also been applied to poultry (De Mol *et al.* 2006), to cattle (Ursinus *et al.* 2009), to tail-biting in pigs (Bracke *et al.* 2004) and to assess the welfare impacts of enrichment materials for pigs (Bracke 2008).

While SWIM 1.0 was designed for fish farmers, SWIM 2.0 supplements welfare indicators (WIs) that require the skills of fish health professionals, that is, expertise in pathophysiology of the fish, treatment and disease prevention in relation to ecology and technical aspects of salmon production. The objective was to the integrate fish health into the overall welfare assessment (OWA) of the SWIM model so as to further improve the accuracy of the welfare assessment. Health, as defined by the World Health Organization (1948), is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity. Health has been identified as a separate welfare need in semantic modelling (Bracke *et al.* 1999c), because it is related to a semibehavioural system, namely 'sickness behaviour' (Hart 1988). Health is clearly associated with welfare-relevant emotional states. In addition, when activated, sickness behaviour competes for time and motor output with other behaviour systems such as feeding (hunger) and the avoidance of danger (fear). Often sickness behaviour takes priority indicating that combating the disease is important for the animal and its welfare. Like other motivational systems, sickness behaviour is functional for survival (Hart 1988). It also involves learning processes, for example, food aversion learning and self-narcotization to alleviate pain (see e.g. Toates 1986).

The presence of disease may indicate an underlying problem with the environment or management, but diseases may also evolve in optimized rearing conditions (Huntingford *et al.* 2006). The welfare implications of disease should be interpreted carefully as the underlying pathophysiological mechanisms giving rise to disease are not always completely understood (Huntingford *et al.* 2006). As a consequence, we emphasize that diagnosis per se, although useful for predicting future development and the need for treatment (prognosis and cure), is not of prime relevance to welfare assessment. By contrast, we decided to take a symptom-oriented approach, reflecting the sensorial or emotional experience associated with good or poor health. Also, in practice, definitive diagnoses are often lacking at the time when welfare assessment is to be performed.

The welfare indicators reviewed in this paper are intended to be practical and feasible to be performed in conjunction with routine farm visits by fish health professionals and/or food authorities. Because the SWIM 2 model is primarily based on visual inspections, standardized gross necropsy procedures (Meyers 2009) and fish farm records, it should not be too time-consuming, costly or otherwise be too demanding (e.g. in requiring time, expertise/facilities for advanced diagnostics), either for the farmer or for the fish health professional. Further details regarding the methods are not outlined in this paper as these may depend on the application of the OWA (e.g. routine welfare monitoring, scientific or legislative use). However, to obtain a reliable result and allow a proper comparison between farms, it is essential to investigate a sufficient and representative number of fish per sea cage (we recommend minimum 20) and of sea cages (minimum 2) from each farm.

In the following, we first give a brief description of the semantic modelling process of creating the SWIM 2.0 model. We then present a review of the scientific statements collected for each newly formulated welfare indicator (WI) and how the WIs have been weighted using the semantic modelling calculation rules. We also present the final model, give examples of its use and finally discuss the concept of semantic modelling and the SWIM 2.0 model as a tool for objectively assessing overall salmon welfare in sea cages in accordance with the state of the art of knowledge in veterinary science.

The semantic modelling process

The first step of welfare assessment in semantic modelling is to draw up a list of the species' basic needs. This was already done for creating the SWIM 1.0 model (Table 1).

The second step is to collect a list of scientific statements (Bracke *et al.* 2002a) obtained from a systematic literature review. The main criterion here is that each statement must somehow be relevant to discriminate good welfare from poor welfare of farmed salmon in sea cages. A Web application (www.imr.no/swim) was constructed to facilitate author collaboration when updating the model's scientific

Table 1 List of basic welfare needs of Atlantic salmon (Stien *et al.* 2013)

Need	Explanation and relevance for salmon
Physical needs	
Respiration	Uptake of oxygen and release of carbon dioxide by pumping water over the gills.
Osmotic balance	Maintaining homoeostasis of body cell fluids
Nutrition	Intake of food containing the required energy, amino acids, minerals, vitamins, etc.
Health	Absence of disease, injury, illness and malfunction
Thermal regulation	Optimization of metabolism and temperature, including thermal comfort
Behavioural needs	
Behaviour control	Ability of the fish to freely position themselves (including regulation of buoyancy) and respond to stimuli
Feeding	Regular access to food.
Safety	Possibility to keep the body undamaged from physical injury and to avoid perceived danger
Social contact	Predictable interaction with conspecifics
Exploration	Possibility to search for resources and information.
Kinesis	Being able to swim (physical activity)
Rest	Possibility of reducing activity level or 'sleep'.
Sexual behaviour	Homeward migration, breeding behaviour, spawning, etc.
Body care	Scratching, parasite cleaning, etc.

database (statements from the literature) and application of the model itself. The Web application also supports updating the model with results from future research, such that the SWIM models are dynamic and can be kept up to date.

The third step is to formulate a list of measurable or observable welfare indicators (WIs). More specifically, we searched the literature for welfare indicators indicative of fish welfare and applicable by fish health professionals when visiting a farm. The selected new indicators were eyes, cardiac condition, abdominal organs, gills, opercula, skeletal muscle, vaccine-related pathology, aberrant fish, necropsy of the dead fish and active euthanasia. To ensure that the WIs were relevant to welfare from the animal's point of view, they all had to be linked to at least one need (Table 2).

We then divided the WIs into levels that could be ranked from best to worst for welfare. The levels are mutually exclusive and must cover the model's domain. As a result, all characteristics of a farming system, including the animals living there, are described by exactly one level of each attribute. This ensures that a generic calculation rule can be used such that any welfare advantage to, for instance, a cage system accrues to all systems with the same descriptive property, and only to them. Based on the ranking, each level was assigned an indicator score (IS):

$$\mathrm{IS}_{i,j} = \frac{\mathrm{NL}_i - \mathrm{RL}_{i,j}}{\mathrm{NL}_i - 1} \tag{1}$$

where $\mathrm{IS}_{i,j}$ is the score of the j th level of the i th WI in the model, NL_i is total number of levels of indicator i, and $\mathrm{RL}_{i,j}$ is the rank number of level j. Next, the scientific statements were used to assign weighting scores (WS) using weighting categories (WC) (Table 3). The WSs are experts' judgements based on the content of the scientific statements presented in each reviewed WI and within the framework of the weighting categories and the range of weighting scores defined in Table 3. This is a somewhat subjective, but systematic, scoring of the weighting category based on experts' assessment of the intensity, duration and incidence of the welfare impact as implied by each scientific statement that has been linked to the WI. The WCs classify welfare performance criteria, for example, pain, illness or reduced survival (Table 3). The weighting factor (WF) of each welfare indicator i in the model was subsequently calculated as proposed by De Mol *et al.* (2006):

$$\mathrm{WF}_i = \left(\sum_{wc} \max(\mathrm{WS}_{wcl})\right)_{ILbest,i} - \left(\sum_{wc} \min(\mathrm{WS}_{wcl})\right)_{ILworst,i} \tag{2}$$

where $\mathrm{IL}_{best,i}$ is the best indicator level, and $\mathrm{IL}_{worst,i}$ is the worst indicator level of the i th welfare indicator; WS_{wcl} is the weighting score assigned to the indicator level based on the scientific statements; wc identifies the weighting categories linked to the indicator level. A special case is made up of WI levels that are so detrimental for welfare that welfare is considered poor (minimum), no matter which levels are selected for the other indicators, that is, other WIs can no longer compensate for this welfare state, and consequently, the SWIM 2.0 model is not designed to assess this level of welfare any further. These levels are called knockout levels and are not included when calculating WFs.

Each section below reviews a WI, which applies at the level of either the individual fish or sea cage (group), and each review section includes a ranking and weighting paragraph. For each weighting, the weighting score (WS) is

Table 2 The most significant links between the selected welfare indicators in SWIM 2.0 and the welfare needs of Atlantic salmon in sea cages

Needs	Respiration	Osmotic balance	Nutrition	Health	Thermal regulation	Behaviour control	Feeding	Safety	Social contact	Exploration	Kinesis	Rest	Sexual behaviour	Body care
Welfare indicators														
Eyes			*	*		*	*			*				
Cardiac condition				*							*			
Abdominal organs			*	*			*							
Gills	*	*		*										
Opercula	*	*		*		*					*			
Skeletal muscle		*		*		*					*			
Vaccine-related pathology				*			*							
Aberrant fish	*	*	*	*			*	*	*					*
Necropsy of the dead fish	*	*	*	*		*	*	*	*	*	*			
Active euthanasia				*				*						

given in parenthesis behind its respective weighting category (WC). The WIs and WCs have been given capital first letters in these paragraphs to denote that these are technically defined terms. This is done in more detail for the first WI, that is, the eyes indicator, to help the reader understand the methodology. The WSs are expert's opinions based on the literature review and intended as a starting point for discussion about welfare assessment, but the reader is free to challenge these decisions.

Review of individual specific welfare indicators (WIs)

Eyes

The fish eye anatomy is very similar to the eyes of other vertebrates. However, when compared to most mammals, the fish eye is more exposed to the external environment. Eye disorders can induce physiological and behavioural stress that is considered to be detrimental for fish welfare (Thatcher 1979) and may occasionally lead to death (Noble *et al.* 2012).

Eye infections can be caused by several pathogens, which can enter the eye either through trauma (exogenous) or via the blood circulation (endogenous). After an infection, the eye may shrink and become dysfunctional. Factors such as chemical, thermal, toxic and UV light exposure may cause eye trauma, but it most frequently results from mechanical injuries (Jurk 2002; Koppang & Bjerkås 2006). Routine aquaculture practices such as pumping, grading and brailing are important risk factors for mechanical eye damage (Deng *et al.* 2005) and often result in intraocular haemorrhages and corneal damage. In addition, traumatic injury commonly results in secondary infections (Koppang & Bjerkås 2006).

The eyes can also be affected indirectly from a widespread non-eye-specific disease such as Parvicapsulosis, caused by the parasite *Parvicapsula pseudobranchicola*. This parasite affects the pseudobranchs (reduced first gill arch) inside the gills and leads to a reduced oxygen supply to the eyes, and infected fish can display haemorrhages inside the eyes (Karlsbakk *et al.* 2002; Anonymous 2011).

A well-studied eye disease in salmon aquaculture is cataracts and involves increased opacity and cloudiness of the lens, generally located bilaterally (two-sided; Poppe 2000). Chronic and severe cataracts are generally considered irreversible (Waagbø *et al.* 2003) and may result in poor vision, reduced growth, secondary infections and mortality (Breck & Sveier 2001; Ersdal *et al.* 2001; Bjerkås *et al.* 2003; Waagbø *et al.* 2010; Remø *et al.* 2011). The development of cataracts has been associated with nutritional deficiencies, toxic agents, parasites (e.g. flukes), exposure to ultraviolet light, hereditary factors, variation in water temperature and rapid growth (reviewed in Björnsson 2004). For salmon

Table 3 Weighting categories (WCs) used in the weighting procedure of semantic modelling with brief descriptions and ranges of weighting scores (WSs). Adapted from Bracke *et al.* (2002b)

Weighting category	Brief description	Range of WS
HPI	Evidence of activation of the HPI (hypothalamic pituitary interrenal) axis indicative of stress.	−5 to −1
Illness	Evidence of health problems, including increased mortality, but excluding skin lesions, fin damage and abnormalities in body shape (see 'pain').	−5 to −1
Pain	Evidence of unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage	−5 to −1
Reduced survival	Evidence of reduced survival related to physiological requirements (other than through specific health problems), for example, longevity, deprivation of food, poor environment	−5 to −1
Abnormal behaviour	Evidence of disturbed behaviour and/or apathy.	−3 to −1
Aggression	Evidence of aggression such as bite marks and attacks.	−3 to −1
Avoidance	Evidence of avoiding stimuli (which are perceived as dangerous/noxious).	−3 to −1
Frustration	Evidence of blocked behaviour or deprivation.	−3 to −1
Negative performance	Evidence of decreased performance (that is likely to indicate negative affect), including (re)production effects, but excluding specific survival aspects related to physiological necessities, HPI activation and illness.	−3 to −1
SAM	Evidence of SAM (sympathetic adrenal medullary) activation (indicative of negative affect), for example, increased heart rate and (nor)adrenaline levels.	−3 to −1
Demand	Evidence that the fish are willing to spend effort to obtain food or other recourses.	1–5
Natural behaviour	Evidence of (potential positive reward from) behaviour as seen in (semi) natural conditions.	1–3
Positive performance	Evidence of healthy, fit fish, which are growing well.	1–3
Preference	Evidence of choosing one resource over another (e.g. in a preference test).	1–3

aquaculture in particular, a deficiency of histidine in the feed (Breck *et al.* 2003, 2005; Waagbø *et al.* 2010), increased use of vegetable oil in the feed (Waagbø *et al.* 2003) and rapid increase in water temperature and growth (Bjerkås *et al.* 2001) have been associated with outbreaks of cataracts. A cataract classification scale proposed by Wall and Bjerkås (1999) characterizes cataracts from 0 to 4 based on the percentage cataract coverage of the lens, from healthy normal lens (0) to 10% (1), 10–50% (2), 50–75% (3) and total cataract (4). Cataracts covering more than 50% of the lens may be associated with impaired vision and reduced feed uptake (Poppe 2000).

Exophthalmia or 'popeye' describes the presence of protruding eyes. In severely affected fish, the stretching and compression of the optic nerve may lead to blindness (Jurk 2002). The condition may be caused by many factors including infections (Karlsbakk *et al.* 2002; Romalde *et al.* 2008), cardiovascular disorders (Tørud *et al.* 2006), gas supersaturation (Gültepe *et al.* 2011) and trauma (Jurk 2002). It may also be recognized as a sign of generalized illness (Koppang & Bjerkås 2006). Aggression in salmonids may cause physical injury, including eye damage (Turnbull *et al.* 1998). Eye snapping has also been reported to occur when sun reflections trigger conspecific attacks to the eye resulting in one-sided eye injury (Noble *et al.* 2012).

We suggest distinguishing between acute and chronic conditions. We consider chronic conditions to be more detrimental to welfare due to an increased time of potential suffering. Based on this consideration, the indicator is divided into five levels: 1) functional, healthy eyes, 2) unilateral (one-sided), traumatic injury, moderate exophthalmia or haemorrhages inside the eye, 3) bilateral (two-sided), traumatic injury, moderate exophthalmia or haemorrhages inside the eyes, 4) bilateral cataract (more than 50% of lens coverage) or chronic condition with impaired vision. Level 5 is a knockout level (K) and is given to individuals with severe exophthalmia and bilaterally blind individuals. Salmon need functional eyes for food consumption (Jurk 2002), and impaired vision is related to abnormal behaviour (−2), frustration (−1) and negative performance (−1). Eye injuries can lead to pain (−2), illness (−1) and reduced survival (−2). Healthy eyes are indicative of positive performance (1) and natural behaviour (1). Using Equation 2 presented above, we calculated a weighting factor (WF) of 11 for the WI 'eyes' (WF = (1 + 1)−(−2−1−1−2−1−2)).

Cardiac condition

The ventricle of salmonid fish is normally shaped as a triangular pyramid with the apex pointing caudoventrally. Its shape is positively correlated with the cardiac output. Farmed salmon appear to have rounder ventricles than wild salmon do. Furthermore, wild salmon exhibit a more acute angle between the ventricular axis and the axis of bulbus arteriosus compared with farmed salmon (Poppe *et al.* 2003; Farrell *et al.* 2006). Claireaux *et al.* (2005) found that

fish with rounder ventricles have lower cardiac outputs and can be characterized as less adapted swimmers.

Several anomalies of the heart are recognized in farmed salmon including arteriosclerosis (Farrell 2002), abnormal heart rate (Mercier *et al.* 2000), aplasia of septum transversus (Poppe *et al.* 1998), situs inversus (Kaada & Hopp 1995), hypoplasia of the outer, compact ventricular myocardium (Poppe & Taksdal 2000) and cardiac hernia with myocardial hypoplasia (Poppe *et al.* 2002). Abnormal heart morphology may be linked to genetics (Dunmall & Schreer 2003), nutrition (Seierstad *et al.* 2005) and environmental factors like egg or alevin incubation temperature (Takle *et al.* 2006).

A number of viral diseases such as cardiomyopathy syndrome (CMS), pancreatic disease (PD) and heart and skeletal muscle inflammation (HSMI) can cause infections in various compartments of the heart (Mcloughlin *et al.* 2002; Kongtorp *et al.* 2004a; Haugland *et al.* 2011). Cardiomyopathy syndrome is associated with sudden death in well-fed fish late in the production cycle, even though the infection itself may be regarded as chronic (Brun *et al.* 2003).

In a normal situation, caged salmon has limited space for movements and thus potentially reduced requirements for cardiac performance. However, abnormal heart morphology or a heart in poor condition may lead to reduced cardiac output with congestion in the abdominal organs; reduced ability to handle stressful events like handling, grading, transport and sea lice treatment; and increased mortality (Poppe *et al.* 2003; Farrell *et al.* 2006; Tørud *et al.* 2006).

We suggest dividing the 'cardiac condition' into the following two levels: 1) healthy cardiac condition and 2) severely deformed heart suggesting reduced cardiac output, congestion in the abdominal organs or signs of other gross heart pathology. Level 1 is linked to positive performance (1), while level 2 indicates illness (−2) and negative performance (−2) and reduced survival (−1). The WF is calculated to be 6.

Abdominal organs

Inflammation is an initiated protective response to any tissue damage commonly caused by factors like infectious microbes, parasites, mechanical trauma, heat, cold, radiation and cancerous cells (Roberts & Rodger 2012). Typical signs of internal acute inflammation are swollen and discoloured organs, exudates of various composition (i.e. fibrinous, serous, catarrhal, purulent), necrosis and haemorrhages. Chronic inflammatory responses are more related to a proliferative phase with manifestations like adherents, fibrosis, granulomas (Roberts & Rodger 2012) and more fish-specific, melanin deposits (Agius & Roberts 2003). In mammals, inflammatory responses are also typically linked to loss of function of the affected tissue and pain perception (McGavin & Zachary 2007).

Some fish may show abnormalities in the swim bladder (Poppe *et al.* 1997a), but the affected fish can often live normally and without external signs (Branson 2008). Another condition is nephrocalcinosis, a chronic, degenerative and inflammatory condition, often visible as white or grey spots in the kidney (Herman 1996). The condition is associated with excessive CO_2 concentrations in the freshwater phase (Fivelstad *et al.* 1999).

The intestine is, along with gills, an important entry site for pathogens, and salmon intestines may be subjected to inflammation and haemorrhages from infectious agents (Poppe 1999; Lumsden 2006). Inflammation of the intestines may also be caused by nutritional ingredients (e.g. soy bean: Baeverfjord & Krogdahl 1996). In addition, tape worms such as *Eubothrium* sp. may infest salmon intestines, even though affected fish generally remains clinically healthy (Anonymous 2011).

We suggest emphasizing chronic conditions as indicating persistent disease and reduced welfare. In this sense, it may also be useful to evaluate the amount of fat deposits in the abdominal cavity, which in a reduced state may indicate a long-term condition. In addition, pathological changes in the abdomen must be distinguished from the side effects of injected vaccines, as we decided to identify the latter as an independent welfare indicator (see under 'vaccine-related pathology').

Therefore, based on a visual inspection of the abdominal organs, we suggest to divide the indicator 'abdominal organs' into the following 3 levels: 1) normal healthy abdominal organs; 2) discoloured organs, exudates, bleeding or swelling; and 3) signs of severe chronic pathology including adherents, granulomas, melanin deposits or fibrosis. Symptoms of inflammation and dysfunctional organs are associated with illness (−3), pain (−3) and negative performance (−3), while normal organs are related to positive performance (1) implying a WF of 10.

Gills

Gill diseases have been associated with reduced performance and growth as well as large-scale mortality in salmon aquaculture (Rodger *et al.* 2011). Because the respiratory system of the fish is in intimate contact with the external environment, it is particularly vulnerable to waterborne agents. Consequently, the gills may act as an important site of antigen entry (Farrell *et al.* 2006; Huntingford *et al.* 2006). Gills also function as an important organ for maintaining osmotic balance and for the excretion of waste products such as ammonia (Farrell *et al.* 2006). Hence, gill tissue disturbances are likely to interfere with these functions (Albassam *et al.* 1987; Byrne *et al.* 1995). However,

careful interpretation of the systemic implications of a gill disease is necessary because the implications are complex and not fully understood (Ferguson & Speare 2006).

Noninfectious gill diseases may be caused by harmful algae, zooplankton and pollutants including jellyfish, nutritional, genetic and congenital factors (Rodger *et al.* 2011). A number of infectious agents of bacterial, viral and parasitical origin can infect the gills. Generally, however, gill diseases are multifactorial disorders where both environmental and infectious factors are involved (Mitchell & Rodger 2011).

Gill disease may result in petechial (small) bleedings, oedema, lamellae necrosis, increased mucus production and pale gills. It may lead to impaired gas exchange with respiratory distress, flared opercula, acid/base disturbances, cardiac dysfunction and ultimately death (Mitchell & Rodger 2011). Associated behavioural changes in severe cases include surface gasping, lethargy and anorexia (Mitchell & Rodger 2011).

We suggest dividing the indicator 'gills' into 3 levels: 1) normal healthy gills; 2) mild signs of focal inflammation, necrosis (dead tissue), lesions or trauma; and 3) severe signs of more generalized inflammation, necrosis, lesions or trauma. Healthy gills are associated with positive performance (1), while inflammation and reduced gill capacity may lead to abnormal behaviour (−2), activation of HPI (−1), frustration (−1), illness (−2) and reduced survival (−2) implying a total WF of 9.

Opercula

Opercula play an important part in fish respiration, including when fish 'cough' to clear debris from the gills, and shortened opercula may interfere with these mechanisms (Branson 2008; Davis 2010). A shortened operculum leads to exposure of the gill filaments which, as a consequence, may become shortened and thickened, making the fish more vulnerable to secondary infections (Branson 2008; Rodger *et al.* 2011). The condition may also result in a reduced ability to pump water over the gills, making the fish more susceptible to poor water quality and low levels of oxygen (Ferguson & Speare 2006). Severely affected fish must maintain an elevated swimming speed to ensure sufficient perfusion of the gills, and this makes affected fish particularly susceptible to handling procedures (Branson 2008). In addition, fish with shortened opercula may show reduced growth rates compared with fish with intact opercula (Ferguson & Speare 2006).

High temperatures during egg incubation have been associated with the development of shortened opercula, but nutrition and genetics may also play a role in its aetiology (Southgate 2006). Erosion of the opercula has also been observed in salmons affected by bacterial gill disease (BGD) (Ferguson & Speare 2006).

We suggest dividing the indicator 'opercula' into 5 levels; 1) normal opercula, 2) operculum only partly covering the gill on one side (unilateral), 3) opercula only partly covering the gills on both sides (bilateral), 4) operculum unilaterally absent and 5) opercula bilaterally absent. Intact opercula are associated with positive performance (1), while shortened opercula are related to abnormal behaviour (−2), reduced survival (−2) and illness (−2). Hence, the total WF is 7.

Skeletal muscles

Despite the importance of the muscular apparatus as the basis of animal movement, as a source of food for humans and as a site for several pathological entities, it is generally not given much attention during routine fish health examinations (Turnbull 2006).

The inflammatory, degenerative and reparative responses in muscular tissue are quite similar in fish and mammals. However, because fish are poikilothermic (cold blooded), the inflammatory activity is closely related to the water temperature (Roberts & Rodger 2012). Most fish species continue to grow throughout their life with a constant increase in the number of muscle fibres, which may positively influence the ability to repair muscular tissue after injury (Mommsen 2001).

Fungal, bacterial, viral, nutritional and parasitic aetiologies are often associated with muscular pathology, but also idiopathic (unknown) myopathies and mechanical and predatory causes may lead to injuries of muscular tissue (Turnbull 2006). The skeletal muscle is a common site for haemorrhages or necrosis following systemic viral infections. Pancreas disease and heart and skeletal muscle inflammation are two common viral diseases present in Norwegian salmon aquaculture that typically affect the skeletal musculature. However, their pathologies are often only visible when examined using histological techniques (Mcloughlin *et al.* 2002; Kongtorp *et al.* 2004b).

Some bacterial diseases can cause multifocal liquefactive necrosis of muscle tissues in the subacute or the chronic stages. These lesions are often characterized as pulpy and haemorrhagic. They may be visible on the skin surface and can even rupture through the skin (Turnbull 2006).

We suggest making a longitudinal incision through the muscles from the head to the tail, followed by a visual inspection of the revealed epi-axial muscular tissue. Based on this inspection, we suggest dividing this indicator into the following 3 levels: 1) normal healthy muscular tissue; 2) haemorrhages, necrosis (dead tissue) or scar tissue (fibrotic tissue) in the skeletal muscle; and 3) abscesses with pulpy liquefactive necrosis in the skeletal muscles. Haemorrhages in musculature are indicative of illness (−1). Muscular injuries caused from either abiotic or biotic factors induce

inflammation and pain (−1) and can reduce fish kinesis (mobility) leading to abnormal behaviour (−3). Normal musculature is related to positive performance (1). The WF calculated is 8.

Vaccine-related pathology

Oil-based polyvalent vaccines injected intraperitoneally (I. P.) are widely used in salmon aquaculture to prevent infectious diseases. Vaccines have proven highly efficacious against bacterial diseases, and vaccination programs have been the major contributing factor to the low consumption of antibiotics in the Norwegian salmon industry (Sommerset *et al.* 2005; Evensen 2009).

An oil-based adjuvant is normally included in multivalent salmon vaccines to enhance the response to certain antigens and to maintain a depot effect, that is, long-term antigen release, of the vaccine (Evensen 2009). However, the recipient fish can exhibit adverse reactions to the vaccine including inflammatory reactions in the abdominal cavity (Midtlyng *et al.* 1996a,b), impaired growth rate (Berg *et al.* 2006), decreased carcass quality (Poppe & Breck 1997b; Midtlyng & Lillehaug 1998), behavioural restrictions (Bjørge *et al.* 2011), spinal deformities (Aunsmo *et al.* 2008a), uveitis (Koppang *et al.* 2004) and systemic autoimmune reactions (Haugarvoll *et al.* 2010). Over time, most intraperitoneally vaccinated fish develop relatively moderate pathological changes in the abdominal cavity that can persist up to the time of harvest (Mutoloki *et al.* 2004). In more severe cases, the intra-abdominal pathology may be manifested as melanin deposits, fibrous granulation and strong adhesions between internal organs or between the organs and the peritoneal wall (Mutoloki *et al.* 2004). Occasionally side effects of vaccination can be so severe that normal organ functions are negatively affected (Poppe & Breck 1997b).

Midtlyng *et al.* (1996b) proposed a gross pathology scoring system (Speilberg's score) to evaluate the lesions caused by intraperitoneal administration of vaccines containing adjuvants. Aunsmo *et al.* (2008a) and Midtlyng and Lillehaug (1998) found, respectively, an increase in spinal deformity and reduction in growth related to intra-abdominal lesions at Speilberg's scores of approximately 3 and higher.

Based on this scoring system, we propose dividing the indicator 'vaccine-related pathology' into the following 7 levels: 1) no visible lesions; 2) slight adhesions, close to the injection site; 3) minor adhesions that may connect colon, spleen or caudal pyloric caeca to the abdominal wall; 4) moderate adhesions including more cranial parts of abdominal cavity, partly involving pyloric caeca, the liver or ventricle connecting them to the abdominal wall; 5) major adhesions with granuloma, extensively interconnecting internal organ, which appear as one unit; 6) extensive lesions affecting nearly every internal organ in the abdominal cavity. In large areas, the peritoneum is thickened and opaque, and the fillet may carry focal, prominent and/or heavily pigmented lesions or granulomas; 7) even more pronounced than 6, often with considerable amounts of melanin resulting in an inability to remove viscera without affecting fillet integrity. Vaccination is likely associated with a degree of stress and impairment of fish welfare, but may simultaneously constitute a prerequisite for freedom from disease and thus positive performance (1). On the other hand, the vaccine can induce a variety of side effects including inflammation with pain (−3), abnormal behaviour (−3) and negative performance (−3). The indicator is given a WF of 10.

Review of sea cage–specific welfare indicators (WIs)

In SWIM 1.0, the welfare indicator 'daily mortality rate' was used to score the mortality rate in the sea cage (Stien *et al.* 2013). The indicator was divided into 5 levels based on a mortality benchmark curve established by Soares *et al.* (2011). This benchmark curve was estimated from mortality rates in the marine stages from 88 salmon productions cycles in Scotland. Because the mortality values may differ between the salmon-producing countries, over time, the SWIM models may later be updated as new and more appropriate mortality benchmark curves arise. The indicators presented below may be regarded as a deeper investigation and more detailed classification of the mortalities in the 'daily mortality' WI in SWIM 1.0. The concept is to bring the welfare assessment a step further using the presented WIs to add additional weight to the mortalities depending on the individuals' welfare prior death.

Aberrant fish

Intensive salmon aquaculture is carried out in large sea cages with huge populations (up to 200 000 individuals; Anonymous 2008). A number of causes (e.g. diseases, injuries and congenital disorders) may reduce the ability of fish to cope under these conditions. As a result, some fish may become aberrant from the rest of the sea cage population. The expression of abnormal behaviour in sea cages has previously been used as a welfare indicator (Huntingford *et al.* 2006). Careful interpretation of the phenomenon, however, is necessary as the causes and consequences are only partially understood (Huntingford *et al.* 2006; Ashley 2007). In general, a notable proportion of aberrant fish, that is, fish suffering from disease showing an appearance and/or behaviour that markedly differs from the other fish in the sea cage ('looser fish', 'runts', 'pinheads', etc.), is indicative of reduced welfare (Huntingford *et al.* 2012).

Aberrant fish may display changed locomotion, external signs and/or rapid breathing (Getchell 2012). Clinically ill individuals may show abnormalities in skin colour (typically darker) as the pigmentation is under neuroendocrine control, that is, down-regulated by stress hormones such as epinephrine (Noga 2010). Abnormal behaviour such as bumping into the nets, circling or spiralling is sometimes observed (Stephen & Ribble 1995; Stien *et al.* 2009). Such fish tend to group with similar conspecifics either in the periphery of schooling groups or at the water surface forming subpopulations showing sickness behaviour (Stephen & Ribble 1995; Stien *et al.* 2009). They may live for months before either recovering (Getchell 2012) or dying from osmotic disturbance, anaemia or secondary diseases (Stien *et al.* 2009; Anonymous 2011).

Moreover, aberrant fish may experience a stressful state implying an impaired immune response against ubiquitous microbes and infectious diseases. As a consequence, they may serve as hosts for pathogen reproduction (Weyts *et al.* 1999; Conte 2004; Ashley 2007; Stien *et al.* 2009). A notable presence of aberrant fish in the sea cage may therefore be indicative of increased infection pressure in the sea cage as they may act as 'superspreaders', threatening the health and welfare of their conspecifics (Stien *et al.* 2009; Getchell 2012).

Estimating the number of aberrant fish in a sea cage can be quite a challenge. Visual detection of these individuals is dependent on several environmental factors such as clarity of the water, light reflection, wind, waves and the size of the sea cage. Despite this, we suggest that with a proper level of experience, it should be possible to make a reasonable estimate of up to 100–200 individuals accounting for approximately 1 ‰ of the population in a sea cage of 100 000 −200 000 individuals. The indicator is therefore divided into the following 4 levels based on the number of aberrant fish: 1) none, 2) single individuals (<1 ‰), 3) between 1 ‰ and 2% of the sea cage population. When more than approximately 2% (2000–4000) of the sea cage population are showing sickness behaviour, the welfare situation is considered to be very poor. Level 4 is therefore a knockout level, and the welfare assessment of the respective sea cage is discarded (i.e. the SWIM 2.0 model is not designed to assess this level of welfare any further). Absence of disease is associated with natural behaviour (2) and positive performance (1). Aberrant fish are associated with abnormal behaviour (−2), reduced survival (−2), illness (−3) and negative performance (−1). This implies a WF of 11.

Necropsy of the dead fish

Fish rapidly show post-mortem autolysis and necropsy should ideally be performed as soon as possible after death (Poppe 1999; Aunsmo *et al.* 2008b; Noga 2010). In the Norwegian salmon aquaculture, self-dead fish are normally removed from the sea cages on a daily basis resulting in a daily sample of mortalities being available from a period of approximately 24 h (Anonymous 2008). The importance of the clinical observations by fish health professionals when gathering information about the causes of mortality should not be underestimated. Aunsmo *et al.* (2008b) found that experienced fish health professionals could assign a likely cause of death in 1929 of 2088 (92.4%) dead fish using post-mortem evaluation supplemented with site and freshwater disease history and some laboratory diagnostic support.

Based on this, we suggest performing a visual inspection and a brief necropsy on the dead fish collected on the day of inspection to estimate the proportion of fish that died from chronic causes, presumably associated with impaired welfare. Dead fish showing severe acute organ lesions may presumably be indicative of intense acute pain. Conversely, fish with a chronic condition may live for several months with moderate but continuous pain or discomfort before dying (Stien *et al.* 2009) and thus experience a prolonged period of reduced welfare. When possible, we suggest taking into account the health history of the population, and the former confirmed diagnoses when evaluating the findings of the necropsied fish. While the other WIs have been given discrete levels, mainly for practical rather than theoretical reasons, we suggest assigning a continuous scale to the present WI (Necropsy of the dead fish) based on the proportion of fish presumably succumbing from chronic causes, ranging from 1.00 (when none of the fish subjected to necropsy are estimated to have died from chronic causes) to 0 (when 100% fish were classified as such). The 'necropsy of the dead fish' indicator's outcome is directly linked to the level of the sea cage mortality and therefore needs to be linked to the 'daily mortality rate' (see calculation rules under 'final model'). Fish suffering from chronic disease can be related to abnormal behaviour (−2), illness (−3), negative performance (−2) and pain (−2). Accordingly, the indicator has a WF of 9.

Active euthanasia (% per day calculated from the previous 7 days)

Removal of fish in poor condition (i.e. looser fish and ill fish that are not likely to recover in due course) from the sea cages for euthanasia is considered a good health management practice in aquaculture as it reduces the pool of potential disease carriers and spreading individuals (Ellis *et al.* 2012). In addition, active euthanasia is also beneficial for welfare. While the catching and killing procedure may present a brief negative experience to the fish (fear/pain), the practice is likely to contribute to considerable overall

reduction in poor welfare conditions in the affected individuals. This is also reflected in the current legislation (e.g. in Norway: Anonymous 2008).

The total number of fish dropping out of the production cycle is the sum of the number of fish that died spontaneously and fish that were euthanized actively. Because the majority of fish in poor condition are likely to die eventually (Stien *et al.* 2009), a larger proportion of euthanized fish indicates that fewer fish are experiencing a prolonged poor welfare condition.

Removing fish in a poor condition out of the sea cages may be challenging in a daily practice, because behaviourally unaffected individuals may still try to avoid the netting procedure. The indicator is divided into the following four levels based on the proportion of euthanized fish from the total number of dropouts: 1) more than 30% of the dropouts, 2) between 10% and 30% of the dropouts, 3) <10% of the dropouts and 4) 0% of the dropouts. Similar to the WI 'necropsy of the dead fish', the 'active euthanasia' indicator's outcome is directly linked to the level of the sea cage mortality and therefore needs to be linked to the 'daily mortality rate' (see calculation rules under 'final model'). As indicated above, euthanasia may have both pros and cons as perceived by the individual fish themselves. The euthanized fish is freed from illness (−3), pain (−3) and negative performance (−3), but consequently leads to reduced survival (−3), and therefore, we consider the overall evaluation of euthanasia to be a major welfare improvement. The WF is calculated to be 12.

Final model

The final step of the semantic modelling procedure (Bracke *et al.* 2002a; Stien *et al.* 2013) is to assemble the WIs, the levels and their associated ranks into an overall welfare assessment (OWA) model using the following three formulas for calculating relative weighting factors (RWFs), indicator welfare scores (IWSs) and the overall welfare index (OWI) on a scale from 0 to 1:

$$\mathrm{RWF}_i = \mathrm{WF}_i \cdot \left(\sum_{j=1}^{m} \mathrm{WF}_j \right)^{-1} \quad (3)$$

$$\mathrm{IWS}_i = \mathrm{IS}_i \cdot \mathrm{RWF}_i \quad (4)$$

$$\mathrm{OWI} = \sum_{j=1}^{m} \mathrm{IWS}_j \quad (5)$$

where m is the total number of indicators in the model, WF_i and WF_j (see Eqn 2) are the weighting factors of, respectively, indicator i and j, and IS_i (see Eqn 1) is the indicator score given by the assessor for indicator i. In case of one or more knockout levels, the OWI is discarded. Knockout levels are not included when calculating RWFs and IWSs.

Table 4 SWIM 1.0 welfare indicator 'daily mortality rate' with levels from best to worst and the associated mortality indicator score (IS_m)

WI	#	Levels	IS_m
Daily mortality rate (% per day)	1	At or below the 10 percentile	1.00
	2	Below benchmark curve	0.75
	3	At the benchmark curve	0.50
	4	Above the benchmark curve	0.25
	5	At or above the 90 percentile curve	0.00
	6	At or above the 90 percentile curve, long term	K

The IS_m is used to link the 'necropsy of the dead fish' and 'active euthanasia' indicators to the daily mortality in the sea cage.

The WIs 'necropsy of the dead fish' and 'active euthanasia' are measured on 'already' dead individuals and the outcome given as proportions. They must therefore be evaluated in association with the daily mortality rate because the WIs' welfare impact is directly linked to the level of sea cage mortality. In Table 4, the welfare indicator 'daily mortality rate' from SWIM 1.0 is divided into 5 levels with their respective indicator scores. Based on this, the ISs for the WIs 'necropsy of the dead fish' and 'active euthanasia' are in addition multiplied with the daily mortality indicator score, henceforth referred to as IS_m, given in Table 4 when calculating their IWS.

Table 5 shows the RWFs for the individual fish and the sea cage WIs. These RWFs together with their levels and their ISs in Table 6 give a model (or schema) for

Table 5 Relative weighting factors (RWFs) for the individual fish-specific welfare indicators (WIs) and for the sea cage–specific WIs in SWIM 2.0

Individual fish WIs	WF	RWF	Sea cage WIs	WF	RWF
Eyes	11	0.18	Aberrant fish	11	0.34
Cardiac condition	6	0.10	Necropsy of the dead fish	9	0.28
Abdominal organs	10	0.16	Active euthanasia	12	0.38
Gills	9	0.15			
Opercula	7	0.12			
Skeletal muscles	8	0.13			
Vaccine-related pathology	10	0.16			
SUM	61	1.00		32	1.00

RWFs are calculated by dividing the weighting factor (WF) with the sum of all WFs (61 and 32 for WIs applying at the level of the individual fish and of the sea cage, respectively (Eqn 3)).

Table 6 Welfare indicators (WIs) with levels from best to worst, the associated indicator level score (IS), the sum of the weighting score assigned to the best and worst level and the calculated weighting factor (WF), see Eqn 2

WI	#	Levels	IS	∑	WF
Individual					
Eyes	1	Functional, healthy eyes	1.00	2	11
	2	Unilateral (one-sided), traumatic injury, moderate exophthalmia or haemorrhages inside the eye	0.66		
	3	Bilateral (two-sided), traumatic injury, moderate exophthalmia or haemorrhages inside the eyes	0.33		
	4	Bilateral (two-sided) cataract (more than 50% of lens coverage) or chronic condition with impaired vision	0.00	−9	
	5	Severe exophthalmia or bilaterally blind individuals	K		
Cardiac condition	1	Healthy cardiac condition	1.00	1	6
	2	Severely deformed heart suggesting reduced cardiac output, congestion in the abdominal organs or signs of other gross hearth pathology	0.00	−5	
Abdominal organs	1	Normal healthy abdominal organs	1.00	1	10
	2	Discoloured organs, exudates, bleeding or swelling	0.50		
	3	Signs of severe chronic pathology like adherents, granulomas, melanin deposits or fibrosis	0.00	−9	
Gills	1	Normal healthy gills	1.00	1	9
	2	Mild signs of focal inflammation, necrosis (dead tissue), lesions or trauma	0.50		
	3	Severe signs of more generalized inflammation, necrosis, lesions or trauma	0.00	−8	
Opercula	1	Normal opercula	1.00	1	7
	2	Operculum only partly covering the gill on one side (unilateral)	0.75		
	3	Opercula only partly covering the gills on both sides (bilateral)	0.50		
	4	Operculum unilaterally absent	0.25		
	5	Opercula bilaterally absent	0.00	−6	
Skeletal muscles	1	Normal healthy muscular tissue	1.00	1	8
	2	Haemorrhages, necrosis (dead tissue) or scar tissue (fibrotic tissue) in the skeletal muscle	0.50		
	3	Abscesses with pulpy liquefactive necrosis in the skeletal muscle	0.00	−7	
Vaccine-related pathology	1	No visible lesions	1.00	1	10
	2	Slight adhesions, close to the injection site	0.83		
	3	Minor adhesions that may connect colon, spleen or caudal pyloric caeca to the abdominal wall	0.66		
	4	Moderate adhesions including more cranial parts of abdominal cavity, partly involving pyloric caeca, the liver or ventricle connecting them to the abdominal wall	0.50		
	5	Major adhesions with granuloma, extensively interconnecting internal organ, which appear as one unit.	0.33		
	6	Extensive lesions affecting nearly every internal organ in the abdominal cavity. In large areas, the peritoneum is thickened and opaque, and the fillet may carry focal, prominent and/or heavily pigmented lesions or granulomas	0.16		
	7	Even more pronounced than 6, often with considerable amounts of melanin resulting in an inability to remove viscera without affecting fillet integrity	0.00	−9	
Sea cage					
Aberrant fish	1	None	1.00	3	11
	2	Single individuals (<1 ‰)	0.50		
	3	Between 1 ‰ and 2% of the sea cage population	0.00	−8	
	4	More than approximately 2% of the sea cage population	K		
Necropsy of the dead fish		1.00 – (the proportion of fish subjected to necropsy and estimated to have died from chronic causes)	0 < IS<1	−9	9
Active euthanasia	1	More than 30% of the dropouts	1.00		12
	2	Between 10% and 30% of the dropouts	0.66		
	3	<10% of the dropouts	0.33		
	4	0% of the dropouts	0.00	−12	

Levels with indicator score K are knockout levels, that is, levels that result in severely reduced welfare regardless of other WIs.

Table 7 SWIM 2.0 applied on the example fish in the example scenario. The overall welfare index (OWI) is the sum of the indicator welfare score (IWS) (Eqn 5)

Individual WIs	RWF		Level	IS	IWS
Eyes	0.18	1	Functional healthy eyes	1.00	0.18
Cardiac condition	0.10	1	Healthy cardiac condition	1.00	0.10
Abdominal organs	0.16	1	Normal healthy abdominal organs	1.00	0.16
Gills	0.15	1	Normal healthy gills	1.00	0.15
Opercula	0.12	2	Operculum only partly covering the gill on one side (unilateral)	0.75	0.09
Skeletal muscles	0.13	1	Normal healthy muscular tissue	1.00	0.13
Vaccine-related pathology	0.16	3	Minor adhesions that may connect colon, spleen or caudal pyloric caeca to the abdominal wall	0.66	0.11
OWI					0.92

calculating an overall welfare index (OWI) score for an individual fish or sea cage.

To calculate an overall SWIM 2.0 OWI for the sea cage, the median OWI for the individual fish is combined with the sea cage OWI as described in the example scenario below.

Example scenario

In this section, we present an example of how the proposed SWIM 2.0 indicators may be used for welfare assessment to calculate an overall welfare index (OWI) score. We will also show how the SWIM 2.0 OWI may be combined with the OWI from SWIM 1.0. The calculated SWIM scores may be integrated further from sea cage to farm level, depending on the intended SWIM application.

A SWIM 2.0 trial was conducted in a sea cage at a fish farm in western Norway in the autumn of 2012. Six fish were sampled and euthanized for a SWIM 2.0 evaluation. We suggest using the median OWI of the investigated fish. Here, we present just one fish as an example. The fish had bilateral cataracts, but it was estimated to cover <50% of the lens implying that level 1 was given to the WI 'eyes'. There were no signs of pathology of the heart, skeletal muscles or abdominal organs. The operculum only partly covered the gill on one side, but the gills were looking healthy. The WI 'opercula' was therefore assigned to level 2. The vaccine-related pathology based on Speilberg's score was evaluated as level 3, that is, minor adhesions that may connect colon, spleen or caudal pyloric caeca to the abdominal wall. Based on Table 7, we calculate an individual OWI of 0.92 where 0 is worst and 1 is best welfare (Figs 1,2).

At the day of the visit (7 months after sea transfer), the sea cage population was estimated to be 106 122 fish. Some looser fish and fish showing sickness behaviour could be observed along the net of the sea cage, but their number was estimated to be <106 fish (i.e. <1‰) indicating level 2 in the WI 'aberrant fish'. Two of 6 dead fish (33%) subjected to necropsy were found to have suffered from chronic causes substantiated from emaciation, small body size and limited fat deposits in the abdomen giving an indicator score (IS) of 0.67 (1.00–0.33). The total number of dropouts calculated from the previous week was 63 fish.

Figure 1 Vaccine-related pathology level 3: minor adhesions that may connect colon, spleen or caudal pyloric caeca to the abdominal wall

Figure 2 Operculum only partly covering the gill on one side (unilateral).

Table 8 SWIM 2.0 applied on the sea cage from the example scenario

Sea cage WIs	RWF		Level	IS	IS_m	IWS
Aberrant fish	0.34	2	Single individuals (<1 ‰)	0.50		0.17
Necropsy of the dead fish	0.28		1.00–0.33	0.67	0.75	0.14
Active euthanasia	0.38	1	More than 30% of the dropouts	1.00	0.75	0.29
OWI						0.60

The indicator welfare score (IWS) is the product of the relative weighting factor (RWF), the indicator score (IS) and the mortality indicator score (IS_m). IS_m is retrieved from the SWIM 1.0 WI 'daily mortality rate' (Table 4) (Stien *et al.* 2013). The overall welfare index (OWI) is the sum of the IWSs (Eqn 5).

Two days before the visit, the farmers removed 42 aberrant fish from the sea cage using a dip net implying that 66% (42/63) of the total number of dropouts had been actively euthanized. This is more than 30% of the dropouts and thus level 1 in the WI 'active euthanasia'. The daily mortality rate was estimated to be 0.0084% (63 fish/7 days of 106.122 fish), that is, below the benchmark curve (Soares *et al.* 2011) and an IS_m of 0.75 (Table 4). The IS_m is included in the calculations of the IWS (=RWF*IS*IS_m) for the WIs 'necropsy of the dead fish' and 'active euthanasia' (Table 8). The sea cage WIs in Table 8 give a sea cage OWI of 0.60.

The individual scores of the sampled fish and the sea cage indicators scores may be combined into a SWIM 2.0 total OWI for the sea cage taking into account the summarized WF for each category: OWI = (0.60*32 + 0.92*61)/(32 + 61) = 0.81. This implies that the welfare scores calculated from all SWIM 2.0 indicators are 0.81 of maximum 1.00.

Considering that SWIM 2.0 is designed as a health-oriented supplement to SWIM 1.0, the OWI from SWIM 1.0 and SWIM 2.0 may also be combined. SWIM 1.0 has a total WF of 94 + 89 = 183 (Table 9). At the same day of visit, the SWIM 1.0 OWI was calculated to be 0.73. The total WF for SWIM 2.0 is 61 + 32 = 93 (Table 5). The OWI for the combined SWIM 1.0 and SWIM 2.0 models is then given by: OWI = (0.81*93 + 0.73*183)/(93 + 183) = 0.76. See Stien *et al.* (2013) for further details regarding the SWIM 1.0 OWI.

Table 9 SWIM 1.0 welfare indicators (WIs) and their respective weighting factors (WFs) (Stien *et al.* 2013)

Sea cage WIs	WF	Individual fish WIs	WF
Temperature(°C)	16	Sea lice	11
Salinity	3	Condition factor	6
Oxygen (%)	17	Emaciation state	16
Water current (BL s^{-1})	3	Vertebral deformation	10
Stocking density (kg m^{-3})	8	Sexual maturity stage	9
Lighting	4	Smoltification state	9
Disturbances	11	Fin condition	13
Mortality (% day^{-1})	21	Skin condition	15
Appetite	11		
SUM	94		89

Discussion

In a previous study (SWIM 1.0), basic welfare indicators for sea reared Atlantic salmon were reviewed (Stien *et al.* 2013). The objective then was to construct a semantic model, which enabled fish farmers to assess the welfare status of their respective sea cages. In this follow-up paper, we have presented a supplementing set of welfare indicators that can be provided by fish health professionals. As in SWIM 1.0, the indicators in SWIM 2.0 presented here are for Atlantic salmon (*Salmo salar L*) in sea cages. The WIs were selected based on feasibility and relevance for welfare, thus excluding associated ethical aspects. The presented model may be used independently, but we recommend using it in combination with SWIM 1.0 regardless of whether or not the scores of the models are combined. Importantly, combining SWIM 1 and 2 will also verify that all aspects of the welfare needs of salmon have been covered (see Table 2). Later, it should also be possible to supplement the overall welfare assessment (OWA) further with in-depth laboratory analysis (SWIM 3.0) in order to obtain the best possible OWA based on available scientific information from a wide range of disciplines including behaviour, (stress- and neuro-)physiology as well as veterinary science.

Although several papers have been published presenting welfare indicators for aquaculture (e.g. see Huntingford *et al.* 2006; Ashley 2007; Segner *et al.* 2012), to our knowledge, SWIM is the first model to systematically assess the overall welfare status of farmed fish based on the scientifically documented links to the basic needs of the fish and practical on-farm measurements.

As in SWIM 1.0, the semantic modelling procedure for SWIM 2.0 was derived from Bracke *et al.* (2002b) and De Mol *et al.* (2006). An important focus of semantic modelling is to systematically review the scientific literature in order to derive the welfare indicators and to weight these indicators based on knowledge about how they affect animal welfare. The procedure provides structured,

dedicated steps to analyse the available literature and derive WIs, weighting factors and welfare scores.

The process of creating the SWIM 2.0 model started with an extensive literature review for statements about fish health relevant to the welfare assessment of Atlantic salmon farmed in sea cages. This ensures that the formulation of WI levels and the calculation of WFs are based on independent scientific statements, that is, statements that have not been produced to confirm preconceived notions of the importance of different WIs and how welfare should be assessed.

It is also important to note that although the SWIM 2.0 model gives OWIs as output, its main purpose is to serve as a diagnostic tool to identify indicators of reduced welfare and encourage their targeted improvement. In addition, the SWIM WIs also put focus on the farmer's management and recognize that farmers are essential players in the animal welfare equation highlighted by Aerts *et al.* (2006) as an important aspect of an OWA. SWIM 2.0 now provides an important backup for farmers by incorporating the expertise of fish health professionals.

Health-related WIs have advantages. They are relatively straightforward to measure and are routinely monitored in modern aquaculture, thereby providing feasible tools to evaluate fish welfare under practical farming conditions (Segner *et al.* 2012). However, the link between health and welfare is not necessarily simple (Huntingford *et al.* 2006). In a health perspective, specific pathologies cannot always be ascribed to observed clinical signs, and often a range of external and internal factors may play a role, which may complicate the assessment (McGavin & Zachary 2007). Several deficiencies remain in our scientific knowledge about fish diseases, for example, links between stress, immune function and disease states; and relationships between health and welfare (Huntingford *et al.* 2006). When screening the literature, we encountered many studies where welfare-relevant parameters were excluded because their scientific objective was not welfare related. In addition, and despite its clear health relevance to the sea cage population, few published papers were dedicated to 'looser fish' in the salmon aquaculture.

A major criticism of semantic modelling is that it is subjective; that is, the modeller has to decide on how to divide the indicators into levels, which weighting categories are appropriate for each indicator and which indicator and weighting scores are to be assigned. These decisions are indeed based on a partly subjective interpretation of the meaning of the collected scientific information. This subjectivity is, however, decreasing with increasing quality and amount of available scientific information; more solid data reduce the freedom of the interpretation of the data. The model and the semantic modelling procedure itself are objective, that is, the information is scientifically valid, and the semantic modelling procedure is formalized and has been described and validated in detail elsewhere (Bracke *et al.* 2002b, 2008; Bracke 2008, 2011). It is designed to take the modeller's point of view, as much as possible, out of the equation (Bracke *et al.* 2002b, 2008; Bracke 2008).

One shortcoming of the SWIM models may relate to the challenge of obtaining a representative sample of fish from a sea cage containing thousands of individuals. Therefore, the number of sampled fish and the sampling method are important factors when evaluating the quality of the individual welfare scores. Farmed salmon are normally sampled from the surface using different types of nets. Some known factors are likely to influence the sample precision: size-dependent swimming depth in sea caged salmon (Folkedal *et al.* 2012), highest sea lice infestation rate of sea caged salmon in the upper cage meters (Hevrøy *et al.* 2003), and the fact that chronically ill and moribund salmon position themselves close to surface and net wall (Stephen & Ribble 1995). Moreover, time of day with regard to hunger level is likely to influence the sample as there is a positive correlation between hunger level and surface attraction (Juell *et al.* 1994).

A series of health inspections and observations are needed to determine the welfare status on the fish farm throughout the production cycle (Segner *et al.* 2012). Also, with frequent health inspections, in-depth evaluation of the historical data is less essential. However, we suggest including a retrospective and summary evaluation of the sea site population after the time of harvest. To a large extent, the salmon aquaculture industry is already recording environmental and health data using sophisticated software (Aunsmo *et al.* 2008b), and this may support the assessment of fish welfare. A retrospective welfare assessment should focus on analysis of production parameters such as incidents of disturbance, handling routines, disease outbreaks, cumulative and cause-specific mortality, obtained growth rates and also slaughter house reports. A model for a retrospective welfare evaluation is proposed in a continuation of the SWIM model concept.

Considering the semantic SWIM model in terms of its transparency, it is likely that existing indicators may have to be upgraded, and new indicators will be integrated as new technology or scientific evidence develops or when new health challenges arise. In particular, mortality benchmark studies on national levels are needed to support short- and medium-term goals for industrial improvement. Robust scientific backing of health- and function-related overall welfare assessments (OWAs) may also be important to receive necessary acceptance for legislation (Segner *et al.* 2012). Readers of this paper are encouraged to suggest improvements in the model in terms of new WIs, specific weightings, studies relevant to indicators or other improvements in the model, making it more accurate and

up to date. In addition, after the model has been tested in field, the procedure and weighting are to be optimized so that SWIM 2.0 users can rely on the model's reliability and validity. A first 'validation' of SWIM 1.0 and 2.0 will be presented in a separate paper (Folkedal *et al.* In prep).

In conclusion, we have proposed a semantic SWIM 2.0 model for welfare assessment of Atlantic salmon in sea cages. SWIM 2.0 contains 10 welfare indicators (WIs), which may be assessed by fish health professionals so as to improve the farmer-oriented SWIM 1.0 model. We anticipate that the SWIM 2.0 model can substantially contribute to a standardized objective welfare assessment of Atlantic salmon reared in sea cages.

Acknowledgement

SWIM is part of the Research Council of Norway (RCN) funded project 199728 SALMOWA.

References

Aerts S, Lips D, Spencer S, Decuypere E, De Tavernier J (2006) A new framework for the assessment of animal welfare: integrating existing knowledge from a practical ethics perspective. *Journal of Agricultural & Environmental Ethics* **19**: 67–76.

Agius C, Roberts RJ (2003) Melano-macrophage centres and their role in fish pathology. *Journal of Fish Diseases* **26**: 499–509.

Albassam M, Moore J, Sharma A (1987) Ultrastructural and clinicopathological studies on the toxicity of cationic acrylamide-based flocculant to rainbow trout. *Veterinary Pathology* **24**: 34–43.

Anonymous (2008) Forskrift om drift av akvakulturanlegg (In Norwegian). Available from: http://www.lovdata.no/cgi-wift/ldles?doc=/sf/sf/sf-20080617-0822.html

Anonymous (2011) Fiskehelserapporten (In Norwegian). Veterinærinstituttets rapport. Available from: http://www.vetinst.no/Publikasjoner/Fiskehelserapporten/Fiskehelserapporten-2011.

Ashley PJ (2007) Fish welfare: current issues in aquaculture. *Applied Animal Behaviour Science* **104**: 199–235.

Aunsmo A, Guttvik A, Midtlyng PJ, Larssen RB, Evensen Ø, Skjerve E (2008a) Association of spinal deformity and vaccine-induced abdominal lesions in harvest-sized Atlantic salmon, *Salmo salar* L. *Journal of Fish Diseases* **31**: 515–524.

Aunsmo A, Bruheim T, Sandberg M, Skjerve E, Romstad S, Larsen RB (2008b) Methods for investigating patterns of mortality and quantifying cause-specific mortality in sea-farmed Atlantic salmon *Salmo salar*. *Diseases of Aquatic Organisms* **81**: 99–107.

Baeverfjord G, Krogdahl A (1996) Development and regression of soybean meal induced enteritis in Atlantic salmon, *Salmo salar* L, distal intestine: a comparison with the intestines of fasted fish. *Journal of Fish Diseases* **19**: 375–387.

Berg A, Rødseth OM, Tangerås A, Hansen T (2006) Time of vaccination influences development of adhesions, growth and spinal deformities in Atlantic salmon *Salmo salar*. *Diseases of Aquatic Organisms* **69**: 239–248.

Bjerkås E, Bjørnestad E, Breck O, Waagbø R (2001) Water temperature regimes affect cataract development in smolting Atlantic salmon, *Salmo salar* L. *Journal of Fish Diseases* **24**: 281–291.

Bjerkås E, Holst JC, Bjerkås I, Ringvold A (2003) Osmotic cataract causes reduced vision in wild Atlantic salmon postsmolts. *Diseases of Aquatic Organisms* **55**: 151–159.

Bjørge MH, Nordgreen J, Janczak AM, Poppe T, Ranheim B, Horsberg TE (2011) Behavioural changes following intraperitoneal vaccination in Atlantic salmon (*Salmo salar*). *Applied Animal Behaviour Science* **133**: 127–135.

Björnsson B (2004) Can UV-treated seawater cause cataract in juvenile cod (*Gadus morhua* L.)? *Aquaculture* **240**: 187–199.

Bracke MBM (2008) RICHPIG: a semantic model to assess enrichment materials for pigs. *Animal Welfare* **17**: 289–304.

Bracke MBM (2011) Review of wallowing in pigs: description of the behaviour and its motivational basis. *Applied Animal Behaviour Science* **132**: 1–13.

Bracke MBM, Spruijt BM, Metz JHM (1999a) Overall animal welfare assessment reviewed. Part 1: is it possible? *Netherlands Journal of Agricultural Science* **47**: 279–291.

Bracke MBM, Metz JHM, Spruijt BM (1999b) Overall animal welfare reviewed. Part 2: assessment tables and schemes. *Netherlands Journal of Agricultural Science* **47**: 293–305.

Bracke MBM, Spruijt BM, Metz JHM (1999c) Overall animal welfare reviewed. Part 3: welfare assessment based on needs and supported by expert opinion. *Netherlands Journal of Agricultural Science* **47**: 307–322.

Bracke MBM, Spruijt BM, Metz JHM, Schouten WGP (2002a) Decision support system for overall welfare assessment in pregnant sows A: model structure and weighting procedure. *Journal of Animal Science* **80**: 1819–1834.

Bracke MBM, Metz JHM, Spruijt BM, Schouten WGP (2002b) Decision support system for overall welfare assessment in pregnant sows B: validation by expert opinion. *Journal of Animal Science* **80**: 1835–1845.

Bracke MBM, Hulsegge B, Keeling L, Blokhuis HJ (2004) Decision support system with semantic model to assess the risk of tail biting in pigs: 1. Modelling. *Applied Animal Behaviour Science* **87**: 31–44.

Bracke MBM, Edwards SA, Metz JHM, Noordhuizen JPTM, Algers B (2008) Synthesis of semantic modelling and risk analysis methodology applied to animal welfare. *Animal* **2**: 1061–1072.

Branson EJ (2008) *Fish Welfare*. Fish Veterinary Society, Oxford.

Breck O, Sveier H (2001) Growth and cataract development in two groups of Atlantic salmon (*Salmo salar* L.) post smolt transferred to sea with a four week interval. *Bulletin of the European Association of Fish Pathologists* **21**: 91–103.

Breck O, Bjerkås E, Campbell P, Arnesen P, Haldorsen P, Waagbø R (2003) Cataract preventative role of mammalian blood meal, histidine, iron and zinc in diets for Atlantic salmon

(*Salmo salar* L.) of different strains. *Aquaculture Nutrition* **9**: 341–350.

Breck O, Bjerkås E, Campbell P, Rhodes JD, Sanderson J, Waagbø R (2005) Histidine nutrition and genotype affect cataract development in Atlantic salmon, *Salmo salar* L. *Journal of Fish Diseases* **28**: 357–371.

Brun E, Poppe T, Skrudland A, Jarp J (2003) Cardiomyopathy syndrome in farmed Atlantic salmon *Salmo salar*: occurrence and direct financial losses for Norwegian aquaculture. *Diseases of Aquatic Organisms* **56**: 241–247.

Byrne PJ, Ostland VE, Lumsden JS, Macphee DD, Ferguson HW (1995) Blood chemistry and acid-base balance in rainbow trout *Oncorhynchus mykiss* with experimentally-induced acute bacterial gill disease. *Fish Physiology and Biochemistry* **14**: 509–518.

Claireaux G, Mckenzie DJ, Genge AG, Chatelier A, Aubin J, Farrell AP (2005) Linking swimming performance, cardiac pumping ability and cardiac anatomy in rainbow trout. *The Journal of experimental biology* **208**: 1775–1784.

Conte FS (2004) Stress and the welfare of cultured fish. *Applied Animal Behaviour Science* **86**: 205–223.

Davis MW (2010) Fish stress and mortality can be predicted using reflex impairment. *Fish and Fisheries* **11**: 1–11.

De Mol RM, Schouten WGP, Evers E, Drost H, Houwers HWJ, Smits AC (2006) A computer model for welfare assessment of poultry production systems for laying hens. *NJAS – Wageningen Journal of Life Sciences* **54**: 157–168.

Deng Z, Guensch GR, Mckinstry CA, Mueller RP, Dauble DD, Richmond MC (2005) Evaluation of fish-injury mechanisms during exposure to turbulent shear flow. *Canadian Journal of Fisheries and Aquatic Sciences* **62**: 1513–1522.

Dunmall KM, Schreer JF (2003) A comparison of the swimming and cardiac performance of farmed and wild Atlantic salmon, *Salmo salar*, before and after gamete stripping. *Aquaculture* **220**: 869–882.

Ellis T, Berrill I, Lines J, Turnbull J, Knowles T (2012) Mortality and fish welfare. *Fish Physiology and Biochemistry* **38**: 189–199.

Ersdal C, Midtlyng PJ, Jarp J (2001) An epidemiological study of cataracts in seawater farmed Atlantic salmon *Salmo salar*. *Diseases of Aquatic Organisms* **45**: 229–236.

Evensen O (2009) Development in fish vaccinology with focus on delivery methodologies, adjuvants and formulations. *Options Mediterraneennes Serie A, Seminaires Mediterraneens* **86**: 177–186.

Farrell AP (2002) Cardiorespiratory performance in salmonids during exercise at high temperature: insights into cardiovascular design limitations in fishes. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology* **132**: 797–810.

Farrell AP, Paige A, Ackerman PA, Iwama GK (2006) Disorders of the cardiovascular and respiratory systems. In: Woo PTK, Leatherland JF (eds) *Fish Diseases and Disorders*, pp. 287–322. CAB International, Wallingford.

Ferguson HW, Speare DJ (2006) Gills and Pseudobranchs. In: Ferguson HW (ed) *Systemic Pathology of Fish: A Text and Atlas of Normal Tissues in Teleosts and their Responses in Disease*. pp 25–63. Scotian Press, London.

Fivelstad S, Olsen AB, Kløften H, Ski H, Stefansson S (1999) Effects of carbon dioxide on Atlantic salmon (*Salmo salar* L.) smolts at constant pH in bicarbonate rich freshwater. *Aquaculture* **178**: 171–187.

Folkedal O, Stien LH, Nilsson J, Torgersen T, Fosseidengen JE, Oppedal F (2012) Sea caged Atlantic salmon display size-dependent swimming depth. *Aquatic Living Resources* **25**: 143–149.

Getchell R (2012) Separate out runts to reduce disease risk. *Fish Farming News* **18**: 6–7.

Gültepe N, Ateş O, Hisar O, Beydemir S (2011) Carbonic anhydrase activities from the rainbow trout lens correspond to the development of acute gas bubble disease. *Journal of aquatic animal health* **23**: 134–139.

Hart BL (1988) Biological basis of the behavior of sick animals. *Neuroscience & Biobehavioral Reviews* **12**: 123–137.

Haugarvoll E, Bjerkås I, Szabo NJ, Satoh M, Koppang EO (2010) Manifestations of systemic autoimmunity in vaccinated salmon. *Vaccine* **28**: 4961–4969.

Haugland O, Mikalsen AB, Nilsen P, Lindmo K, Thu BJ, Eliassen TM *et al.* (2011) Cardiomyopathy syndrome of atlantic salmon (*Salmo salar* L.) is caused by a double-stranded RNA virus of the Totiviridae family. *Journal of virology* **85**: 5275–5286.

Herman RL (1996) *Systemic Noninfectious Granulomatoses of Fishes*. US Department of the Interior, Fish and Wildlife Service, Research and Development, Washington, DC, USA.

Hevrøy EM, Boxaspen K, Oppedal F, Taranger GL, Holm JC (2003) The effect of artificial light treatment and depth on the infestation of the sea louse *Lepeophtheirus salmonis* on Atlantic salmon (*Salmo salar* L.) culture. *Aquaculture* **220**: 1–14.

Huntingford FA, Kadri S (2008) Welfare and fish. In: Branson EJ (ed) *Fish Welfare*, pp. 19–31. Blackwell Publishing Ltd, Oxford, UK.

Huntingford FA, Adams C, Braithwaite VA, Kadri S, Pottinger TG, Sandoe P *et al.* (2006) Current issues in fish welfare. *Journal of Fish Biology* **68**: 332–372.

Huntingford F, Kadri S, Jobling M (2012) Introduction: aquaculture and behaviour. In: Huntingford F, Kadri S, Jobling M (eds) *Aquaculture and Behaviour*, pp. 1–35. Blackwell Publishing Ltd, Oxford, UK.

Juell JE, Fernö A, Furevik D, Huse I (1994) Influence of hunger level and food availability on the spatial distribution of Atlantic salmon, *Salmo salar* L., in sea cages. *Aquaculture Research* **25**: 439–451.

Jurk I (2002) Ophthalmic disease of fish. *Veterinary Clinics of North America: Exotic Animal Practice* **5**: 243–260.

Kaada I, Hopp P (1995) Laks med deformerte hjerter og misdannede hjertesekker (in Norwegian) (English Abstract). *Norsk veterinærtidsskrift* **107**: 773–776.

Karlsbakk E, Saether PA, Hostlund C, Fjellsoy KR, Nylund A (2002) Parvicapsula pseudobranchicola n. sp (Myxozoa), a

myxosporidian infecting the pseudobranch of cultured Atlantic salmon (*Salmo salar*) in Norway. *Bulletin of the European Association of Fish Pathologists* **22**: 381–387.

Kongtorp RT, Kjerstad A, Taksdal T, Guttvik A, Falk K (2004a) Heart and skeletal muscle inflammation in Atlantic salmon, *Salmo salar* L.: a new infectious disease. *Journal of Fish Diseases* **27**: 351–358.

Kongtorp RT, Taksdal T, Lyngøy A (2004b) Pathology of heart and skeletal muscle inflammation (HSMI) in farmed Atlantic salmon *Salmo salar*. *Diseases of Aquatic Organisms* **59**: 217–224.

Koppang EO, Bjerkås E (2006) The eye. In: Ferguson HW (ed) *Systemic Pathology of Fish: A Text and Atlas of Normal Tissues in Teleosts and their Responses in Disease*, pp. 245–265. Scotian Press, London.

Koppang EO, Haugarvoll E, Hordvik I, Poppe TT, Bjerkås I (2004) Granulomatous uveitis associated with vaccination in the Atlantic Salmon. *Veterinary Pathology Online* **41**: 122–130.

Lumsden JS (2006) Gastrointestinal tract, swimbladder, pancreas and peritoneum. In: Ferguson HW (ed) *Systemic Pathology of Fish: A Text and Atlas of Normal Tissues in Teleosts and their Responses in Disease*, pp. 169–199. Scotian Press, London.

McGavin MD, Zachary JF (2007) *Pathologic Basis of Veterinary Disease*. Elsevier Mosby, St. Louis.

Mcloughlin MF, Nelson RN, Mccormick JI, Rowley HM, Bryson DB (2002) Clinical and histopathological features of naturally occurring pancreas disease in farmed Atlantic salmon, *Salmo salar* L. *Journal of Fish Diseases* **25**: 33–43.

Mercier C, Aubin J, Lefrançois C, Claireaux G (2000) Cardiac disorders in farmed adult brown trout, *Salmo trutta* L. *Journal of Fish Diseases* **23**: 243–249.

Meyers TR (2009) Standard necropsy procedures for finfish. In: Heil N (ed.) *National Wild Fish Health Survey – Laboratory Procedures Manual*. 5.0 edn, U.S. Fish and Wildlife Service, Warm Springs, GA

Midtlyng PJ, Lillehaug A (1998) Growth of Atlantic salmon *Salmo salar* after intraperitoneal administration of vaccines containing adjuvants. *Diseases of Aquatic Organisms* **32**: 91–97.

Midtlyng PJ, Reitan LJ, Lillehaug A, Ramstad A (1996a) Protection, immune responses and side effects in Atlantic salmon (*Salmo salar* L.) vaccinated against furunculosis by different procedures. *Fish & Shellfish Immunology* **6**: 599–613.

Midtlyng PJ, Reitan LJ, Speilberg L (1996b) Experimental studies on the efficacy and side-effects of intraperitoneal vaccination of Atlantic salmon (*Salmo salar* L.) against furunculosis. *Fish & Shellfish Immunology* **6**: 335–350.

Mitchell SO, Rodger HD (2011) A review of infectious gill disease in marine salmonid fish. *Journal of Fish Diseases* **34**: 411–432.

Mommsen TP (2001) Paradigms of growth in fish. *Comparative Biochemistry and Physiology Part B: Biochemistry and Molecular Biology* **129**: 207–219.

Mutoloki S, Alexandersen S, Evensen Ø (2004) Sequential study of antigen persistence and concomitant inflammatory reactions relative to side-effects and growth of Atlantic salmon (*Salmo salar* L.) following intraperitoneal injection with oil-adjuvanted vaccines. *Fish & Shellfish Immunology* **16**: 633–644.

Noble C, Cañon Jones H, Damsgård B, Flood M, Midling K, Roque A *et al.* (2012) Injuries and deformities in fish: their potential impacts upon aquacultural production and welfare. *Fish Physiology and Biochemistry* **38**: 61–83.

Noga EJ (2010) *Fish Disease: Diagnosis and Treatment*. Wiley-Blackwell, Ames, Iowa.

Poppe TT (1999) *Fiskehelse Og Fiskesykdommer (In Norwegian)*. Universitetsforlag, Oslo.

Poppe TT (2000) Produksjonsrelaterte lidelser i fiskeoppdrett–en etisk utfordring for veterinærene. (In Norwegian). *Norsk veterinærtidsskrift* **112** (2): 91–96.

Poppe TT, Breck O (1997b) Pathology of Atlantic salmon *Salmo salar* intraperitoneally immunized with oil-adjuvanted vaccine. A case report. *Diseases of Aquatic Organisms* **29**: 219–226.

Poppe TT, Taksdal T (2000) Ventricular hypoplasia in farmed Atlantic salmon *Salmo salar*. *Diseases of Aquatic Organisms* **42**: 35–40.

Poppe TT, Hellberg H, Griffiths D, Meldal H (1997a) Swimbladder abnormality in farmed Atlantic salmon *Salmo salar*. *Diseases of Aquatic Organisms* **30**: 73–76.

Poppe TT, Midtlyng PJ, Sande RD (1998) Examination of abdominal organs and diagnosis of deficient septum transversum in Atlantic salmon, *Salmo salar* L., using diagnostic ultrasound imaging. *Journal of Fish Diseases*, **21**: 67–72.

Poppe TT, Johansen R, Tørud B (2002) Cardiac abnormality with associated hernia in farmed rainbow trout *Oncorhynchus mykiss*. *Diseases of Aquatic Organisms* **50**: 153–155.

Poppe TT, Johansen R, Gunnes G, Tørud B (2003) Heart morphology in wild and farmed Atlantic salmon *Salmo salar* and rainbow trout *Oncorhynchus mykiss*. *Diseases of Aquatic Organisms* **57**: 103–108.

Remø SC, Olsvik PA, Torstensen BE, Amlund H, Breck O, Waagbø R (2011) Susceptibility of Atlantic salmon lenses to hydrogen peroxide oxidation ex vivo after being fed diets with vegetable oil and methylmercury. *Experimental Eye Research* **92**: 414–424.

Roberts RJ, Rodger HD (2012) The pathophysiology and systematic pathology of teleosts. In: Roberts RJ (ed) *Fish Pathology*, pp. 62–143. Wiley Blackwell, Hoboken, NJ.

Rodger H, Henry L, Mitchell S (2011) Non-infectious gill disorders of marine salmonid fish. *Reviews in Fish Biology and Fisheries* **21**: 423–440.

Romalde JL, Ravelo C, Valdes I, Magarinos B, De La Fuente E, Martin CS *et al.* (2008) *Streptococcus phocae*, an emerging pathogen for salmonid culture. *Veterinary Microbiology* **130**: 198–207.

Segner H, Sundh H, Buchmann K, Douxfils J, Sundell K, Mathieu C *et al.* (2012) Health of farmed fish: its relation to fish welfare and its utility as welfare indicator. *Fish Physiology and Biochemistry* **38**: 85–105.

Seierstad SL, Poppe TT, Koppang EO, Svindland A, Rosenlund G, Frøyland L *et al.* (2005) Influence of dietary lipid composition on cardiac pathology in farmed Atlantic salmon, *Salmo salar* L. *Journal of Fish Diseases* **28**: 677–690.

Soares S, Green DM, Turnbull JF, Crumlish M, Murray AG (2011) A baseline method for benchmarking mortality losses in Atlantic salmon (*Salmo salar*) production. *Aquaculture* **314**: 7–12.

Sommerset I, Krossøy B, Biering E, Frost P (2005) Vaccines for fish in aquaculture. *Expert review of vaccines* **4**: 89–101.

Southgate P (2006) Welfare and Farmed Fish. In: Woo PTK, Leatherland JF (eds) *Fish Diseases and Disorders*, pp. 357–370. CAB International, Wallingford.

Stephen C, Ribble CS (1995) An evaluation of surface moribund salmon as indicators of seapen disease status. *Aquaculture* **133**: 1–8.

Stien LH, Kristiansen T, Danielsen TL, Torgersen T, Oppedal F, Fosseidengen JE (2009) Fra utsett til slakt (In Norwegian). In: Agnalt AL, Bakketeig I, Haug T, Knutsen JA, Opstad I (eds) *Kyst og Havbruk 2009*, pp. 160–163. Institute of Marine Research, Bergen, Norway.

Stien LH, Bracke MBM, Folkedal O, Nilsson J, Oppedal F, Torgersen T *et al.* (2013) Salmon Welfare Index Model (SWIM-1.0): a semantic model for overall welfare assessment of caged Atlantic salmon – review of selected welfare indicators and model presentation. *Reviews in Aquaculture* **5**: 33–57.

Takle H, Baeverfjord G, Helland S, Kjorsvik E, Andersen O (2006) Hyperthermia induced atrial natriuretic peptide expression and deviant heart development in Atlantic salmon *Salmo salar* embryos. *General and Comparative Endocrinology* **147**: 118–125.

Thatcher TO (1979) A morphological defect in shiner perch resulting from chronic exposure to chlorinated sea water. *Bulletin of Environmental Contamination and Toxicology* **21**: 433–438.

Toates F (1986) *Motivational Systems*. Cambridge University Press, Cambridge.

Tørud B, Taksdal T, Dale OB, Kvellestad A, Poppe TT (2006) Myocardial glycogen storage disease in farmed rainbow trout, *Oncorhynchus mykiss* (Walbaum). *Journal of Fish Diseases* **29**: 535–540.

Turnbull J (2006) Musculoskeletal System. In: Ferguson HW (ed) *Systemic Pathology of Fish: A Text and Atlas of Normal Tissues in Teleosts and their Responses in Disease*, pp. 288–311. Scotian Press, London.

Turnbull JF, Adams CE, Richards RH, Robertson DA (1998) Attack site and resultant damage during aggressive encounters in Atlantic salmon (*Salmo salar* L.) parr. *Aquaculture* **159**: 345–353.

Ursinus WW, Schepers F, De Mol RM, Bracke MBM, Metz JHM, Koerkamp P (2009) COWEL: a decision support system to assess welfare of husbandry systems for dairy cattle. *Animal Welfare* **18**: 545–552.

Waagbø R, Hamre K, Bjerkås E, Berge R, Wathne E, Lie Ø *et al.* (2003) Cataract formation in Atlantic salmon, *Salmo salar* L., smolt relative to dietary pro- and antioxidants and lipid level. *Journal of Fish Diseases* **26**: 213–229.

Waagbø R, Tröße C, Koppe W, Fontanillas R, Breck O (2010) Dietary histidine supplementation prevents cataract development in adult Atlantic salmon, *Salmo salar* L., in seawater. *British Journal of Nutrition* **104**: 1460–1470.

Wall T, Bjerkås E (1999) A simplified method of scoring cataracts in fish. *Bulletin of the European Association of Fish Pathologists* **19**: 162–165.

Weyts F, Cohen N, Flik G, Verburg-Van Kemenade BML (1999) Interactions between the immune system and the hypothalamo-pituitary-interrenal axis in fish. *Fish & Shellfish Immunology* **9**: 1–20.

World Health Organization (1948) Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference, New York, 19–22 June, 1946; signed on 22 July 1946 by the representatives of 61 States (Official Records of the World Health Organization, no. 2, pp. 100) and entered into force on 7 April 1948.