APPENDIX F. BIOLOGICAL AND ENVIRONMENTAL UNCERTAINTIES

The vulnerability assessment presented here does not address two major uncertainties: (1) the effect of climate change on the marine environment and ecosystems, including critical migration corridors such as the Columbia and Snake rivers, and (2) the future epidemiology of fish pathogens and disease under the climates projected for the 2040s. Both factors could greatly affect the ability of hatcheries in the Pacific Northwest to propagate Pacific salmon and Steelhead through the 21st Century. These uncertainties are described below.

Effect of climate change on the marine environment

Predicting the future effects of climate change on marine ecosystems is extremely difficult, although many effects have been documented and postulated (see review by Hoegh-Guldberg and Bruno 2010). Global mean temperatures have risen approximately 0.2°C over the past 30 years with largest proportion of that heat energy absorbed by the oceans. The oceans have also absorbed approximately one-third of all anthropogenic CO_2, thus reducing the mean pH of the oceans globally.

Continued warming of the upper layers of the oceans is expected to increase temperature stratification, thus decreasing dissolved O_2 concentrations in deeper waters and potentially reducing nutrient availability in the phototrophic zone. Indeed, total phytoplankton production has decreased by more than 6% since 1980 with over 70% of this reduction concentrated at higher latitudes, particularly in the Pacific Ocean and Indian Ocean gyres.

Although the effects of sea-level rise and ocean acidification on nearshore estuarine ecosystems can be assessed to some extent, the overall effects of climate change on the trophic dynamics of marine ecosystems and salmon productivity are major uncertainties (Schindler et al. 2008). Modeling efforts do provide some insights regarding projected effects of climate change on marine survival and productivity of Pacific salmon in the North Pacific Ocean. Based on the A1B emission scenario, summer habitats in the North Pacific Ocean are projected to decrease by 86% for Chinook Salmon, 45% for Sockeye Salmon, 36% for Steelhead, and 30% for Coho Salmon by the year 2100 (Abdul-Aziz et al. 2011). A general decline since the mid-1970s in the marine abundance of Coho Salmon and Chinook Salmon in the eastern North Pacific Ocean has been attributed to climate-related changes (Irvine and Fukuwaka 2011). Peterson et al. (2010) report that the abundances of yearling Chinook Salmon (but not Coho Salmon) in the California Current were negatively correlated with marine temperatures which influence coastal upwelling and zooplankton abundance. In general, warm ocean conditions associated with the Pacific Decadal Oscillation suppress upwelling and reduce marine survival and productivity of Pacific salmon in the Pacific Northwest (Mantua et al. 1997; Scheuerell and Williams 2005; Mantua 2009). Conversely, climate change is projected to increase the incidence and intensity of Pacific storms in the Gulf of Alaska, and stronger onshore winds are projected to increase upwelling and nutrient turnover rates in the eastern North Pacific Ocean.

Interactions among climate-influenced increases in ocean temperature, ocean acidification, and onshore winds create additional uncertainties regarding marine primary production and marine food webs. These uncertainties confound attempts to assess the vulnerability of Pacific salmon populations to climate change in specific watersheds (e.g., Columbia River) and sub-basins (e.g., Wenatchee River). Consequently, much research and monitoring are necessary to understand the future effects of climate change on the marine survival and productivity of Pacific salmon and Steelhead originating from rivers in the Pacific Northwest. The intensive monitoring of hatchery-origin salmon and Steelhead along the west coast of North America provides one mechanism for assessing those effects and uncertainties.

Effect of climate change on pathogen prevalence and fish disease

The incidence and severity of infectious diseases in fish are affected by the immune status of the fish, the virulence of the pathogen within the fish, and the density of the pathogen in the environment. Serious disease outbreaks usually occur when the immune status of a fish is compromised at a time when (a) the pathogen is well within its optimal temperature range and (b) conditions are such that the density of the pathogen in the environment is high.

Basic metabolic rates, physiological homeostasis, and immune function of fish are direct functions of water temperatures. Water temperatures in the upper range of physiological tolerance will stress and ultimately compromise the immune system of fish (Wedemeyer 1970, 1996). At the same time, elevated temperatures may match the optimal ranges for (a) fish parasites like *Ichthyophthirius multifiliis* (Ich) or *Ceratonova shasta*, (b) bacterial pathogens like *Aeromonas salmonicida* (furunculosis) or *Flavobacterium columnare* (columnaris), and (c) pathogenic fungi. All species of salmonid fishes are particularly vulnerable to Ich at elevated water temperatures.

Reduced stream flows in summer, like those projected for most rivers in the Pacific Northwest due to reduced snow pack and less summer precipitation, can also increase disease risks to fish reared on surface waters in a hatchery. Reduced flows are expected to increase pathogen density by reducing dilution, increasing fish-to-fish contact by elevating density indexes, and by increasing nutrient loads to favor the growth of non-obligate bacterial and protozoan pathogens that can propagate in the environment without a fish host. Thus, while either increased temperatures or reduced flows might trigger an outbreak of infectious disease, the combination of both factors can often be devastating.

A recent example of this synergistic interaction among water temperature, flow, and disease occurred in 2002 on the Klamath River, California, where over 33,000 adult salmonids, primarily Chinook Salmon, died during their upstream migration at a time of low water flows and warm water temperatures. Pathology reports concluded that the fish died from Ich and columnaris (*Flavobacterium columnare*) and not directly from high water temperatures which reportedly did not exceed the physiological tolerances of the fish themselves but may have compromised their immune systems (CDFG 2004). Both Ich and columnaris are caused by warmer water pathogens

that are most devastating when salmon are exposed to temperatures above their physiological optima, as occurred in the Klamath River in 2002.

Although the potential for elevated water temperatures and decreased flows to increase the prevalence and severity of disease in Pacific salmon is difficult to quantify, recent parasitic and bacterial disease outbreaks at USFWS hatcheries – particularly those affected by low flows and high temperatures in the summers of 2015 and 2016 – clearly demonstrate the potential for climate change to increase risks of infectious disease and fish mortality. For example, in the summer of 2015, Chinook Salmon juveniles experiencing high mortality at 18 °C (despite disease treatments) at one hatchery (Warm Springs NFH) completely recovered within 10 days after they were transferred to another facility (Little White Salmon NFH) with water temperatures of 9 °C. At USFWS hatcheries in the Columbia-Pacific Northwest Region (Idaho, Oregon, and Washington), serious outbreaks of Ich, columnaris, furunculosis, fungal diseases, and ceratomyxosis strongly correlate with water temperatures in the range of 13 – 20 °C.

The effect of elevated water temperatures on viral pathogens of salmon and Steelhead is less clear than for bacteria, fungi, or parasites. The two most significant fish viruses in the Pacific Northwest, IHNV and VHSV[1], only produce disease at temperatures below 15 °C, and those viruses are unable to propagate at higher temperatures, even in vitro where host immunity is not a factor. However, low flows and high temperatures are expected to weaken fish physiologically during warm seasons, thus compromising their immune systems and potentially predisposing them to viral infections when temperatures cool.

In addition, organisms not currently impacting salmon health can become pathogenic whenever their fish hosts or environments are altered. For example, the parasite *C. shasta* has recently become much more problematic to salmon and Steelhead in the Deschutes River system, a circumstance that is likely attributable to recent changes in reservoir management that subsequently affected river flows and downstream water temperatures.[2]

In general, climate-induced increases in the prevalence of pathogens in existing habitats, and the spread of those pathogens to new habitats, are expected to reduce the abundance and viabilities of many fish species within their native geographic ranges (Harvell et al. 2009). Overall increases in water temperatures resulting from climate change are expected to (a) increase physiological stress and reduce immune effectiveness in salmon and Steelhead and (b) increase the propagation and transmission rates of aquatic parasites and pathogens (Marcogliese 2008). Despite these expectations, a great deal of uncertainty exists concerning the future magnitude and mechanisms of climate-induced changes to pathogen prevalence and disease throughout the Pacific Northwest.

Fish health and hatchery staff clearly recognize these increasing disease risks and uncertainties associated with climate change (e.g., Appendix A and Appendix D of this report). Additional

[1] Infectious hematopoietic necrosis virus (IHNV) and Viral hemorrhagic septicemia virus (VHSV)
[2] http://www.thedalleschronicle.com/news/2014/aug/23/parasite-driven-disease-hits-klamath-salmon-hard-f/

research and enhanced monitoring and evaluation efforts will most likely be needed to better understand how climate change will affect the future distribution, prevalence, and virulence of fish pathogens throughout the Pacific Northwest.

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