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"Most significant lesions" abbreviations in alphabetical order		
#	Abbreviation	Expanded text
1	AIB	Amphophilic inclusion bodies (liver); AIB might be remnants of ingested erythrocytes (this type of inclusion has not been described with any salmon virus).
2	BPC	Basophilic cytoplasm (hepatocytes); BPC is normal in mature females producing protein for deposition in their eggs. In juvenile salmon it might be related to increased protein needed as part an inflammatory response.
3	BPH	Biliary preductular cell hyperplasia (liver); BPH is evidence of exposure to toxins. The toxins could be produced inside the fish (e.g., bacterial toxins) or come from outside the fish (e.g., from the water or the feed).
4	CPL	Cholangitis/pericholangial leukocytes (liver); CPL is evidence of chronic immune stimulation. This type of inflammation can result from bacteria ascending from the intestine to the liver through the biliary system.
5	ECH	Endothelial cell hypertrophy (heart); ECH is evidence of systemic immune stimulation; probably resulting from inflammatory cell mediators released into the circulation (e.g., during a bacterial infection).
6	EGC	Eosinophilic granular cells/endothelial granules (kidney); EGCs are part of a specific type of inflammatory response that probably can be induced by more than one cause. I have seen these granules associated with <i>Piscirickettsia salmonis</i> infection, and these cells have been associated with experimental infection of rainbow trout with <i>Listonella anguillarum</i> (I have not seen this pattern of inflammation described in Atlantic salmon exposed to <i>Listonella anguillarum</i>).
7	EHP	Endothelial cell hypertrophy, with eosinophilic granules (spleen); EHP probably is the spleen equivalent of EGC; evidence of systemic immune stimulation; probably resulting from inflammatory cell mediators released into the circulation (e.g., with certain bacterial infections).
8	ENE	Endocarditis, eosinophilic (heart); ENE is part of a specific type of inflammatory response that probably can be induced by more than one cause. I have seen these granules associated with a <i>Piscirickettsia salmonis</i> , and these cells have been associated with experimental infection of rainbow trout with <i>Listonella anguillarum</i> (I have not seen this pattern of inflammation described in Atlantic salmon exposed to <i>Listonella anguillarum</i>).
9	ENH	Endocarditis, histiocytic (heart); histiocytic inflammation in the heart is evidence of chronic immune stimulation; differentials include a low grade bacterial infection and reaction to a vaccine.
10	ENL	Endocarditis, lymphohistiocytic (heart); ENL is evidence of chronic immune stimulation; differentials include a low grade bacterial infection and reaction to a vaccine.
11	EPH	Epicarditis, histiocytic (heart); epicarditis is evidence of chronic immune stimulation; differentials include a low grade bacterial infection and reaction to a vaccine.
12	EPL	Epicarditis, lymphohistiocytic (heart); EPL is evidence of chronic immune stimulation; differentials include a low grade bacterial infection and reaction to a vaccine.
13	EPN	Exocrine pancreas necrosis; EPN is a hallmark of infection with Infectious Pancreatic Necrosis virus (IPNV, Birnaviridae) and salmon pancreas disease (SPDV, salmonid alphavirus, Togaviridae). These diseases have not been identified in pen reared salmon in BC.
14	FEN	Fibrinocellular enteritis is evidence of local or systemic inflammation, it occurs with some viral and bacterial diseases.
15	FPL	Focal/multifocal parenchymal leukocytes (liver); FPL are foci of chronic immune stimulation (e.g., the result of a bacterial infection).
16	GLF	Gill lamellar fusion; GLF decreases the available surface area for gas transfer. Gill lamellar fusion may be a result of physical damage from exposure to a parasite or distoms (e.g., <i>Chaetoceros</i> spp.). GLF has been associated with exposure to heavy metals and with hypoxia (thought to be from fish gasping for oxygen at the water-air interface).
17	GLH	Gill lamellar hyperplasia/hypertrophy; GLH decreases the efficiency of gas exchange. Common causes of include physical and chemical irritants in the water, including parasites (e.g., <i>Paramoeba</i> spp.).
18	GLT	Gill lamellar telangiectasis is the permanent dilation of lamellar capillaries. It is usually associated with trauma.
19	GR	Granulomatous inflammation (no confirmed cause); differentials for granulomatous inflammation include a reaction to a vaccine and chronic bacterial disease (e.g., <i>Yersinia ruckeri</i> or <i>Renibacterium salmoninarum</i> infection).
20	GRP	Golden renal pigment (probably lipofuscin); GRP is a nonspecific change that can result from a variety of insults, including rancid feed, low levels of antioxidants in the feed, chronic infections, and exposure to organic contaminants. When tubular epithelial cells are involved, variation in size of nuclei and cytoplasm is evidence of cellular degeneration and regeneration, and it is consistent with persistent damage to the tubules.
21	GTH	Gill thrombosis; blood clots in the vascular spaces of the gill are evidence of increased coagulability. GTH can result from endothelial damage related to virus (e.g., VHSV), bacterial, or parasitic infection, or exposure to toxins.
22	HEM	Hemorrhage/congestion (interstitial, kidney); HEM probably is a nonspecific result of endothelial damage; HEM is often associated with VHSV and bacterial infections. Renal congestion and hemorrhage is one of the classic signs of infectious salmon anemia (ISA), but ISA/V has never been isolated from fish in BC.
23	HTH	Heart thrombosis; blood clots in the vascular spaces of the heart are evidence of increased coagulability. HTH can result from endothelial damage related to virus (e.g., VHSV), bacterial, or parasitic infection.
24	ICN	Interstitial cell necrosis (kidney); large numbers of necrotic interstitial cells is the hallmark lesion of Infectious Hematopoietic Necrosis (caused by IHNV). Differentials include infection with bacteria (e.g., <i>Yersinia ruckeri</i>) or other viruses.
25	IEA	Interstitial edema and/or interstitial cell atrophy is evidence of decreased immune function, usually a result of a viral infection (e.g., VHSV).
26	IFB	Interstitial fibrin (kidney); IFB is evidence of endothelial damage, probably from exposure to toxins. The toxins could be of bacterial origin or inflammatory cell origin; toxins in the water or feed are less likely.
27	IPC	Intratubular (luminal) protein casts (kidney); IPCs result from glomerular or tubular dysfunction; either excess protein leaks through glomeruli or tubules are unable to reabsorb protein. This lesion sometimes is associated with VHSV infection.
28	IPR	Intestinal peritonitis or peritonitis of adjacent mesenteries; IPR is consistent with a reaction to foreign material; it is common in fish that have been vaccinated. Vacuoles (when present) probably represent lipophilic vaccine material that was removed during tissue processing (alcohol and xylene remove lipid from tissues before staining). Peritonitis can also result from a bacterial infection (e.g., <i>Yersinia ruckeri</i> or <i>Aeromonas salmonicida</i>).
29	ISH	Interstitial (hematopoietic) cell hyperplasia (kidney); ISH is evidence of increased demand for erythrocytes or white blood cells somewhere in the body. In Chinook salmon, this lesion is often associated with the clinical diagnosis of "Marine anemia".
30	LFN	Liver focal/multifocal necrosis; hepatocellular necrosis can be caused by inadequate vascular perfusion (e.g., as occurs with harmful algal blooms or hypoxia) or direct cytotoxicity from viral or bacterial infections (e.g., viral hemorrhagic septicemia virus or <i>Piscirickettsia salmonis</i>).
31	LIP	Lipidosis (hepatocellular); LIP often occurs when fish are not feeding; it also occurs in cases of inadequate nutrition.
32	LKR	Leukocytic karyorrhexis (spleen); LKR is evidence of increased cell turnover, possibly as part on an active inflammatory response.
33	MCC	Mesenteric capillary congestion (intestine); distension of capillaries in the mesenteric adipose tissue is nonspecific evidence of circulating vasodilators; hemorrhage sometimes occurs in severe cases. MCC is most commonly associated with VHSV and bacterial infections.
34	MCH	Mucous cell hyperplasia (gill); excess mucus on the surface of the gill is a response to irritants in the water (e.g., toxins or parasites).
35	MEG	Megalocytosis/karyomegaly (hepatocellular); MEG results from sublethal injury to hepatocytes, and affected cells may be able to survive for several months. Hepatic megalocytosis can result from exposure to several types of toxins, including aflatoxins, pyrrolizidine alkaloids, complex chemical mixtures from marine sediment extracts, and the algal toxin microcystin-LR. In BC salmon, megalocytosis is one of the main diagnostic criteria for "netpen liver disease."
36	MGN	Membranous glomerulonephritis (kidney); MGN is fairly common in older salmonids, particularly Chinook salmon; it is often associated with infections in other parts of the fish, but a link to immune complex deposition has not been demonstrated. Membranous glomerulonephritis has been associated with cardiomyopathy syndrome (in Atlantic salmon), nephrocalcinosis, and infections with a number of bacteria and parasitic species.
37	MIN	Mineralization (kidney); MIN is common in cultured fish species; when severe, the condition is termed nephrocalcinosis. The lesion is not considered fatal, although feed conversion may be adversely affected. The pathogenesis is not fully understood, but renal mineralization has been experimentally reproduced through high carbon dioxide levels, magnesium deficiency, selenium toxicity, and a diet low in minerals (source, "Systemic Pathology of Fish", Second edition, 2006, edited by H. Ferguson). Clinically, renal mineralization is most commonly associated with high carbon dioxide levels.
38	MMK	Mycocardial karyomegaly/megalocytosis; MKM probably is an indication of multiple copies of DNA within the affected nuclei; this could represent a developmental anomaly or a response to toxicant exposure. Karyomegaly in other cell types has been associated with exposure to algal toxins (e.g., hepatocytes exposed to microcystin LR in netpen liver disease).
39	MSC	<i>Microsporidium cerebralis</i> (brain) infection.
40	none	No significant lesions
41	PER	Peritonitis (spleen); PER is consistent with a reaction to foreign material; it is common in fish that have been vaccinated; the primary differential, especially when peritonitis is fibrinous, is a bacterial infection (e.g., <i>Yersinia ruckeri</i> or <i>Aeromonas salmonicida</i>).
42	PFB	Parenchymal fibrin (spleen); PFB is evidence of endothelial damage, probably from exposure to toxins. The toxins could be of bacterial origin or inflammatory cell origin. I have seen this response in salmon that are PCR positive for VHSV.
43	PGP	Parenchymal golden pigment (probably lipofuscin, spleen); PGP is a nonspecific change that can result from a variety of insults, including rancid feed, low levels of antioxidants in the feed, chronic infections, and exposure to organic contaminants. Conditions that lead to moderate to abundant lipofuscin have been associated with decreased growth and survival in several studies.
44	PMP	Pigmented macrophages (liver); PMP contain lipofuscin, hemosiderin, or both (Melanin pigment occurs normally in some salmonids, so melanin is not included in this score). Accumulation of lipofuscin is a nonspecific change that can result from a variety of insults, including rancid feed, low levels of antioxidants in the feed, chronic infections, and exposure to organic contaminants. Conditions that lead to moderate to abundant hepatic lipofuscin have been associated with decreased growth and survival in several studies. Hemosiderin accumulation in the liver is evidence of increased turnover of red blood cells.
45	PMX	Presporogonic myxosporean (brain) infection
46	PS	<i>Piscirickettsia salmonis</i> infection.
47	PVL	Perivascular lymphocytes/leukocytes (liver); PVL probably are foci of chronic immune stimulation (e.g., bacterial infection).
48	RS	<i>Renibacterium salmoninarum</i> infection.
49	RTN	Renal tubular necrosis (epithelial); RTN is commonly associated with viral hemorrhagic septicemia virus (VHSV); differentials include exposure to toxins (e.g., bacterial toxins, heavy metals, or aminoglycoside antibiotics such as gentamicin).
50	SCN	Single cell necrosis (hepatocytes); SCN is evidence of cellular damage in the liver. Causes of hepatocellular single cell necrosis have not been well defined in fish. Possible differentials include exposure to toxins (endogenous or exogenous) or viral infection (VHSV); single cell necrosis is a common feature of "net pen liver disease".
51	SSC	Sinusoidal congestion (liver); SSC is a nonspecific result of sinusoidal damage. In BC Atlantic salmon, sinusoidal congestion is an uncommon feature of infection with viral hemorrhagic septicemia virus (VHSV) and <i>Listonella anguillarum</i> . Sinusoidal congestion is one of the classic lesions associated with infectious salmon anemia virus (ISAV) infection, but ISAV has never been identified in British Columbia.
52	SSF	Sinusoidal fibrin (liver); SSF is evidence of endothelial damage, probably from exposure to toxins. The toxins could be of viral (VHSV), bacterial, parasitic (e.g., <i>Loma salmonae</i> spores), or inflammatory cell origin; toxins in the water or feed are less likely.
53	TDI	Tubular dilation (of lumen, kidney); TDI is evidence that flow of urine is abnormal. The most probable cause is some type of flow blockage. Increased sectional area of the tubular lumen can also result from attenuation of epithelial cells (e.g., after necrosis of tubular epithelial cells).
54	TEP	Tubular epithelial protein droplets (intracytoplasmic, renal tubules); TEP are normal in some species, or they may be an indication of glomerular disease. Ferguson ("Systemic Pathology of Fish," second edition, 2006) reports an association of renal protein droplets and high ammonia levels in salmonids.
55	VAC	Vacuolation of hepatocyte cytoplasm; vacuoles might be normal for fish on diets with a high proportion of plant-based components. Vacuoles that contain eosinophilic granules (lysosomes filled with cellular debris?) might be a result of toxin exposure.