	A	C	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE	AF	AG	AH	AI	AJ	AK	AL	AM	AN	AO	AP	AQ	AR	AS	AT	AU
1	Case 2009-0455 Histopathology (MH cases 7115)									LIVER -									KIDNEY -																											
2	Pathologist = Dr. Gary D. Marty										VAC = vacuoles in hepatocyte cytoplasm									ISH = interstitial (hematopoietic) cell hyperplasia															STOMACH/INTESTINE/EXOCRINE PANCREAS -													
3	Client # = writing on submitted cassette										LIP = lipidosis									TEP = tubular epithelial protein (intracytoplasmic)															IPR = peritonitis				GILL -									
4	AHC case number = # assigned by the Animal Health Centre, Abbotsford										BPH = biliary preductular cell hyperplasia									GRP = golden renal pigment																			GLH = lamellar hyperplasia/hypertrophy								
5	Slide # = number assigned at the Animal Health Centre (BC-MAL) for histopathology										LFN = focal/multifocal necrosis									TDI = tubular dilation (of lumen)																			GLF = lamellar fusion								
6	Clinical status = determined by submitter										BPC = basophilic cytoplasm (hepatocytes)									MIN = mineralization																			MCH = mucous cell hyperplasia								
7	NP = tissue not included for analysis										HHD = hepatocellular hydropic degeneration									HEM = interstitial hemorrhage/congestion																			EYE -								
8	Scoring = none (0), mild (1), moderate (2), or severe/abundant (3										LGR = granulomatous hepatitis/peritonitis,									EGC = eosinophilic granular cells/endothelial granules																			CPM = choroid plexus mineralization								
9	coordinates (e.g., 35.4 x 112.3) = location of a specific change on a slide;											vaccine reaction (?)								IPC = intratubular (luminal) protein casts																													
10	the structure/change can be found by placing the slide on the stage																			IFB = interstitial fibrin											SPLEEN -															
11	of the microscope in my office (AAC, room 106), with the white part on the left.																			ICN = interstitial cell necrosis												PER = peritonitis													
12	= Quality Control																			RTN = renal tubular necrosis												SGR = granulomatous inflammation (not SRS)													
13	1 score for all organs: artifact (Art), postfixation dehydration (PFD), acid hematin (AH)																																												
14	1 score for each organ: Autolysis (Atly)																																												
15																																												
16						Most	All organ																					HEART						Intestine/Stomach/								Skin/		
17		Client # on	AHC histopathology		Date	significant	Quality control							Liver																Spleen					Exocrine Pancreas					Gill				skeletal muscle		Brain
18	#	cassette	Slide #s		Sampled	Lesion	ART	PFD	AHT	LAtly	VAC	LIP	BPH	LFN	BPC	HHD	LGR		KAtly	ISH	TEP	GRP	TDI	MIN	HEM	RTN		HAtly		SAtly	PER	SGR		Iatly	IPR	MCC		Gatly	GLH	GLF	MCH		SkAtly		MEN
19	1	6	2009-0455-	6	2009-02-05	PER	1	0	0	0	3	0	0	0	0	0	0		0	0	1	0	0	0	0	0		0		0	3	0		1	2	1		NP	NP	NP	NP		0		1
20	2	7	2009-0455-	7	2009-02-05	PER	1	0	0	1	2	0	0	0	0	0	0		0	0	2	0	0	1	0	0		0		1	3	0		1	1	0		NP	NP	NP	NP		0		0
21	3	8	2009-0455-	8	2009-02-05	IPR	1	0	0	1	3	0	0	0	0	0	0		1	0	2	0	0	1	0	0		0		0	2	0		0	3	0		NP	NP	NP	NP		0		0
22	4	9	2009-0455-	9	2009-02-05	IPR	1	0	0	1	3	0	0	0	0	0	0		1	0	2	0	0	1	0	0		0		0	2	1		1	3	0		NP	NP	NP	NP		0		0
23	5	10	2009-0455-	10	2009-02-05	IPR	1	0	0	1	2	0	0	0	0	0	0		0	0	1	0	0	2	0	0		0		0	0	0		1	2	0		NP	NP	NP	NP		0		0
24	6	16	2009-0455-	16	2009-02-05	IPR	1	0	0	1	3	0	0	0	0	0	0		0	0	2	1	1	1	0	0		0		0	1	1		0	2	0		NP	NP	NP	NP		0		0
25	7	17	2009-0455-	17,17G	2009-02-05	IPR	1	0	1	1	1	0	0	0	0	0	0		1	0	1	0	0	0	0	0		0		0	1	1		1	2	0		1	0	0	0		0		0
26	8	18	2009-0455-	18	2009-02-05	PER	1	0	0	1	1	0	0	0	0	0	1		1	0	1	0	1	1	0	0		0		0	3	0		2	2	1		NP	NP	NP	NP		0		0
27	9	19	2009-0455-	19	2009-02-05	PER	1	0	0	1	2	0	0	0	0	0	0		1	0	2	0	0	1	0	0		0		0	3	0		1	1	0		NP	NP	NP	NP		0		NP
28	10	20	2009-0455-	20	2009-02-05	MIN	1	0	0	1	3	0	0	0	0	0	0		0	0	2	0	0	2	0	0		0		0	0	0		1	1	0		NP	NP	NP	NP		0		0
29		**Summary statistics:**																																											
30			count				10	10	10	10	10	10	10	10	10	10	10		10	10	10	10	10	10	10	10		10		10	10	10		10	10			1	1	1	1		10		9
31			n = 0				0	10	9	1	0	10	10	10	10	10	9		5	10	0	9	8	2	10	10		10		9	2	7		2	0			0	1	1	1		10		8
32			% = 0				0.0	100.0	90.0	10.0	0.0	100.0	100.0	100	100.0	100.0	90		50	100	0	90.0	80	20	100	100		100.0		90	20.0	70		20.0	0.0			0.0	100.0	100.0	100.0		100.0		88.9
33			**% > 0**				**100.0**	**0.0**	**10.0**	**90**	**100**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**10.0**		**50**	**0.0**	**100.0**	**10.0**	**20.0**	**80.0**	**0.0**	**0.0**		**0.0**		**10**	**80.0**	**30.0**		**80.0**	**100.0**			**100.0**	**0.0**	**0.0**	**0.0**		**0.0**		**11.1**

Lesion abbreviations in alphabetical order

#	Abbreviation	Expanded text
1	**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.
2	**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).
3	**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).
4	**FPL**	Focal/multifocal parenchymal leukocytes (liver); FPL are foci of chronic immune stimulation (e.g., the result of a bacterial infection).
5	**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 diatoms (e.g., *Chaetoceros* 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).
6	**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., *Paramoeba* spp.).
7	**GR**	(HGR or SGR) Granulomatous inflammation (no confirmed cause); differentials for granulomatous inflammation include a reaction to a vaccine and chronic bacterial disease (e.g., *Yersinia ruckeri* or *Renibacterium salmoninarum* infection).
8	**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.
9	**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.
10	**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., *Yersinia ruckeri* or *Aeromonas salmonicida*).
11	**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".
12	**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 *Piscirickettsia salmonis*).
13	**LIP**	Lipidosis (hepatocellular); LIP often occurs when fish are not feeding; it also occurs in cases of inadequate nutrition.
14	**MCC**	Mesenteric capillary congestion; 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.
15	**MCH**	Mucous cell hyperplasia (gill);excess mucus on the surface of the gill is a reponse to irritatants in the water (e.g., toxins or parasites).
16	**MEN**	Meningitis and encephalitis (brain); MEN is evidence of immune stimulation; differentials include viruses, bacteria, and parasites
17	**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.
18	**MKM**	Myocardial 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).
19	**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., Yersinia ruckeri or Aeromonas salmonicida).
20	**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).
21	**HHD**	hepatocellular hydropic degeneration; HHD is evidence of cellular damage in the liver. Possible differentials include exposure to toxins (endogenous or exogenous) or viral infection (VHSV).
22	**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).
23	**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.
24	**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.