	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
1	Case 2008-3591 Histopathology (MH case 6861)									LIVER -																			KIDNEY -		HKN = head kidney		
2	Pathologist = Dr. Gary D. Marty										VAC = vacuoles in hepatocyte cytoplasm											SCN = single cell necrosis/apoptosis/hydropic degeneration								TKN = trunk kidney		
3	Client # = writing on submitted cassette										LIP = lipidosis											LGR = granulomatous hepatitis/peritonitis, vaccine reaction (?)								ISH = interstitial (hematop		
4	AHC case number = # assigned by the Animal Health Centre, Abbotsford										BPH = biliary preductular cell hyperplasia											LRS = *Renibacterium salmoninarum*								TEP = tubular epithelial pr		
5	Slide # = number assigned at the Animal Health Centre (BC-MAL) for histopathology										LFN = focal/multifocal necrosis											LPS = *Piscirickettsia salmonis*								GRP = golden renal pigm		
6	Clinical status = determined by submitter										BPC = basophilic cytoplasm (hepatocytes)																				TDI = tubular dilation (of l		
7	NP = tissue not included for analysis										CPL = cholangitis/pericholangial leukocytes																				MIN = mineralization		
8	Scoring = none (0), mild (1), moderate (2), or severe/abundant (3);										PMP = pigmented macrophages																				HEM = interstitial hemorrh		
9	coordinates (e.g., 35.4 x 112.3) = location of a specific change on a slide;										FPL = focal/multifocal parenchymal leukocytes																				EGC = eosinophilic granu		
10	the structure/change can be found by placing the slide on the stage										PVL = perivascular lymphocytes/leukocytes																				IPC = intratubular (lumina		
11	of the microscope in my office (AAC, room 106), with the white part on the left.										SSC = sinusoidal congestion																				IFB = interstitial fibrin		
12		= Quality Control									AIB = amphophilic/eosinophilic inclusion bodies																				ICN = interstitial cell necro		
13		1 score for all organs: artifact (Art), postfixation dehydration (PFD), acid hematin (AH)									SSF = sinusoidal fibrin																				RTN = renal tubular necro		
14		1 score for each organ: Autolysis (Atly)									MEG = hepatocellular megalocytosis/karyomegaly																							
15																																	
16						Most	All organ																										
17			AHC case number		Clinical	significant	Quality control							Liver																				
18	#	Client # on cassette	Histopathology slide #s		status	Lesion	ART	PFD	AHT	LAtly	VAC	LIP	BPH	LFN	BPC	CPL	PMP	FPL	PVL	SSC	AIB	SSF	MEG	SCN	LGR	LRS	LPS	Liver comments	HKN	TKN	KAtly	ISH	TEP
19	1	6861-1	2008-3591-	1A,1G	moribund	GTH	1	0	0	1	1	2	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0		Y	Y	0	0	0
20	2	6861-2	2008-3591-	2A,2G	moribund	GTH	1	0	0	1	0	2	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0		Y	Y	0	1	0
21	3	6861-3	2008-3591-	3A,3G	moribund	GTH	1	0	0	1	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		Y	Y	0	0	0
22	4	6861-4	2008-3591-	4A,4G	moribund	LFN	1	0	1	1	2	0	0	2	0	1	1	0	0	0	0	0	0	0	0	0	0	LFN is multifocal; cytoplasm of affected cells is slightly hypereosinophilic (differential diagnosis = autolysis, but autolysis is usually not multifocal); CPL involves eosinophilic granular cells;	Y	Y	0	0	0
23	5	6861-5	2008-3591-	5A,5G	moribund	GTH	1	0	0	1	2	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0		Y	Y	0	0	0
24	6	6861-6	2008-3591-	6A,6G	moribund	GTH	1	0	0	1	2	0	0	0	0	0	1	1	1	0	0	0	0	0	0	0	0		Y	Y	0	0	0
25	7	6861-7	2008-3591-	7A,7G	moribund	LFN	1	0	0	0	1	2	0	2	0	0	1	0	0	0	0	0	0	0	0	0	0		Y	Y	0	0	0
26	8	6861-8	2008-3591-	8A,8G	moribund	LFN	1	0	0	1	0	2	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0		Y	Y	0	0	0
27		**Summary statistics:**																															
28			count				8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8				8	8	8
29			n = 0				0	8	7	1	3	3	8	5	8	7	3	7	7	6	8	8	8	8	8	8	8				8	7	8
30			% = 0				0.0	100.0	87.5	12.5	37.5	37.5	100.0	62.5	100.0	87.5	37.5	87.5	87.5	75	100	100.0	100.0	100.0	100	100.0	100.0				100	87.5	100
31			**% > 0**				**100.0**	**0.0**	**12.5**	**87.5**	**62.5**	**62.5**	**0.0**	**37.5**	**0.0**	**12.5**	**62.5**	**12.5**	**12.5**	**25.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**				**0**	**12.5**	**0.0**

	AJ	AK	AL	AM	AN	AO	AP	AQ	AR	AS	AT	AU	AV	AW	AX	AY	AZ	BA	BB	BC	BD	BE	BF	BG	BH	BI	BJ	BK	BL	BM	BN	BO	BP	BQ	BR	BS
1	/; Y = yes, examined; NP = not present on the slide														HEART -													SPLEEN -								
2	/; Y = yes, examined; NP = not present on the slide															MKM = myocardial karyomegaly/megalocytosis													PER = peritonitis								
3	oietic) cell hyperplasia															EPL = epicarditis, lymphohistiocytic													LKR = leukocytic karyorrhexis								
4	otein (intracytoplasmic)															EPH = epicarditis, histiocytic													PGP = parenchymal golden pigment (lipofuscin)								
5	ent															ENL = endocarditis, lymphohistiocytic													PFB = parenchymal fibrin								
6	umen)															ENE = endocarditis, eosinophilic													EHP = endothelial cell hypertrophy (with eosinophilic								
7																ENH = endocarditis, histiocytic													SGR = granulomatous inflammation (not SRS)								
8	age/congestion															ECH = endothelial cell hypertrophy													SRS = *Renibacterium salmoninarum*								
9	ar cells/endothelial granules															HTH = thrombosis													SPS = *Piscirickettsia salmonis*								
10	) protein casts									MGN = membranous glomerulonephritis						HGR = granulomatous inflammation																					
11										KGR = granulomatous inflammation						HRS = *Renibacterium salmoninarum*																					
12	sis									KRS = *Renibacterium salmoninarum*						HPS = *Piscirickettsia salmonis*																					
13	sis									KPS = *Piscirickettsia salmonis*																											
14																																				
15																																				
16																																				
17				Kidney																	Heart									Spleen							
18	GRP	TDI	MIN	HEM	EGC	IPC	IFB	ICN	RTN	MGN	KGR	KRS	KPS	Kidney Comments	HAtly	MKM	EPL	EPH	ENL	ENE	ENH	ECH	HTH	HGR	HRS	HPS	Heart Comments	SAtly	PER	LKR	PGP	PFB	EHP	SGR	SRS	SPS
19	0	0	0	0	0	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
20	0	0	0	0	0	0	0	0	0	0	0	0	0	ISH in head kidney;	0	1	0	0	0	0	0	0	0	0	0	0		0	1	0	1	0	0	0	0	0
21	0	0	0	0	0	0	0	0	0	0	0	0	0		0	0	0	0	1	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
22	0	0	0	0	0	0	0	0	0	0	0	0	0		0	1	0	0	0	0	0	0	0	0	0	0		0	1	0	0	0	0	0	0	0
23	0	0	0	0	0	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	0		0	1	0	0	0	0	0	0	0
24	0	0	0	0	0	0	0	0	0	0	0	0	0		0	1	0	0	0	0	0	0	0	0	0	0		0	1	0	0	0	0	0	0	0
25	0	0	0	0	0	0	0	0	0	0	1	0	0	KGR in head kidney includes a multinucleate giant cell;	0	0	1	0	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0
26	0	0	0	0	0	0	0	0	0	0	0	0	0		0	1	1	0	0	0	0	0	0	0	0	0		0	1	0	1	0	0	0	0	0
27																																				
28	8	8	8	8	8	8	8	8	8	8	8	8	8	count	8	8	8	8	8	8	8	8	8	8	8	8	count	8	8	8	8	8	8	8	8	8
29	8	8	8	8	8	8	8	8	8	8	7	8	8	n = 0	8	4	6	8	7	8	8	8	8	8	8	8	n = 0	8	3	8	6	8	8	8	8	8
30	100.0	100	100	100	100	100	100	100.0	100	100	87.5	100.0	100.0	% = 0	100.0	50.0	75.0	100.0	87.5	100.0	100.0	100.0	100.0	100.0	100.0	100.0	% = 0	100	37.5	100.0	75.0	100.0	100.0	100	100.0	100.0
31	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**12.5**	**0.0**	**0.0**	**% > 0**	**0.0**	**50.0**	**25.0**	**0.0**	**12.5**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**% > 0**	**0**	**62.5**	**0.0**	**25.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**

	BT	BU	BV	BW	BX	BY	BZ	CA	CB	CC	CD	CE	CF	CG	CH	CI	CJ	CK	CL	CM	CN	CO
1		INTESTINE/EXOCRINE PANCREAS -							GILL -										SKIN/SKELETAL MUSCLE -			
2		IPR = peritonitis							GLH = lamellar hyperplasia/hypertrophy										MDN = myodegeneration and necrosis			
3		EPN = exocrine pancreas necrosis							GLF = lamellar fusion													
4		MCC = mesenteric capillary congestion							LSE = lamellar subepithelial edema													
5		IGR = granulomatous inflammation (not IRS)							GLT =lamellar telangiectasis													
6	granules	IRS = *Renibacterium salmoninarum*							GLB = thrombosis													
7									MCH = mucous cell hyperplasia													
8									GGR = granulomatous inflammation (not GRS)													
9									GRS = *Renibacterium salmoninarum*													
10																						
11																						
12																						
13																						
14																						
15																						
16																						
17		Stomach/Intestine/Exocrine Pancreas										Gill							Skin/skeletal muscle			
18	Spleen Comments	Iatly	IPR	EPN	MCC	IGR	IRS	Intestine/Pancreas comments	Gatly	GLH	GLF	LSE	GLT	GTH	MCH	GGR	GRS	Gill Comments	SkAtly	MDN		
19		0	0	0	0	0	0	no intestine in section	0	1	1	0	1	2	0	0	0	GLH @ 38 x 106.5; GLB @ 32 x 118; GLT @ 31 x 109;	0	0		
20		0	0	0	0	0	0		1	1	1	0	0	1	0	0	0		0	0		
21		0	0	0	0	0	0		1	0	0	0	1	1	0	0	0		0	1		
22		0	0	0	0	0	0		1	0	0	0	1	1	1	0	0		0	0		
23		0	0	0	0	0	0		1	0	0	0	0	1	0	0	0		0	0		
24		0	0	0	0	0	0		1	1	1	0	1	1	0	0	0		0	0		
25		0	2	0	0	0	0		0	0	0	0	1	1	0	0	0		0	0		
26		1	0	0	0	0	0		1	0	1	0	1	1	0	0	0		0	1		
27																						
28		8	8	8	8	8	8		8	8	8	8	8	8	8	8	8		8	8		
29		7	7	8	8	8	8		2	5	4	8	2	0	7	8	8		8	6		
30		87.5	87.5	100.0	100.0	100.0	100.0		25.0	62.5	50.0	100.0	25.0	0.0	87.5	100.0	100.0		100.0	75.0		
31		**12.5**	**12.5**	**0.0**	**0.0**	**0.0**	**0.0**		**75.0**	**37.5**	**50.0**	**0.0**	**75.0**	**100.0**	**12.5**	**0.0**	**0.0**		**0.0**	**25.0**		

"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 *Piscirickettsia salmonis* infection, and these cells have been associated with experimental infection of rainbow trout with *Listonella anguillarum* (I have not seen this pattern of inflammation described in Atlantic salmon exposed to *Listonella anguillarum*).
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*Piscirickettsia salmonis* , and these cells have been associated with experimental infection of rainbow trout with *Listonella anguillarum* (I have not seen this pattern of inflammation described in Atlantic salmon exposed to *Listonella anguillarum*).
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	**FPL**	Focal/multifocal parenchymal leukocytes (liver); FPL are foci of chronic immune stimulation (e.g., the result of a bacterial infection).
15	**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).
16	**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.).
17	**GLT**	Gill lamellar telangiectasis is the permanent dilation of lamellar capillaries. It is usually associated with trauma.
18	**GR**	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).
19	**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.
20	**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.
21	**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 ISAV has never been isolated from fish in BC.
22	**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.
23	**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., Yersinia ruckeri) or other viruses.
24	**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.
25	**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.
26	**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*).
27	**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".
28	**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*).
29	**LIP**	Lipidosis (hepatocellular); LIP often occurs when fish are not feeding; it also occurs in cases of inadequate nutrition.
30	**LKR**	Leukocytic karyorrhexis (spleen); LKR is evidence of increased cell turnover, possibly as part on an active inflammatory response.
31	**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.
32	**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).
33	**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."
34	**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.
35	**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.
36	**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).
37	**MSC**	*Microsporidium cerebralis* (brain) infection.
38	**none**	No significant lesions
39	**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).
40	**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.
41	**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.
42	**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.
43	**PMX**	Presporogonic myxosporean (brain) infection
44	**PS**	*Piscirickettsia salmonis* infection.
45	**PVL**	Perivascular lymphocytes/leukocytes (liver); PVL probably are foci of chronic immune stimulation (e.g., bacterial infection).
46	**RS**	*Renibacterium salmoninarum* infection.
47	**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).
48	**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".
49	**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*Listonella anguillarum* . 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.
50	**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.*Loma salmonae* spores), or inflammatory cell origin; toxins in the water or feed are less likely.
51	**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).
52	**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.