APPENDIX 8

Supplemental Water Quantity and Quality Information

Environmental Assessment of

Canadian Strategic Infrastructure Funded

Upgrades to the City of Winnipeg

Water Pollution Control Centres

APPENDIX 8

Supplemental

Water Quality and Quantity

Information

Prepared by: Tetr*ES* Consultants Inc.

Environmental Assessment of
Canadian Strategic Infrastructure Funded
Upgrades to the City of Winnipeg
Water Pollution Control Centres

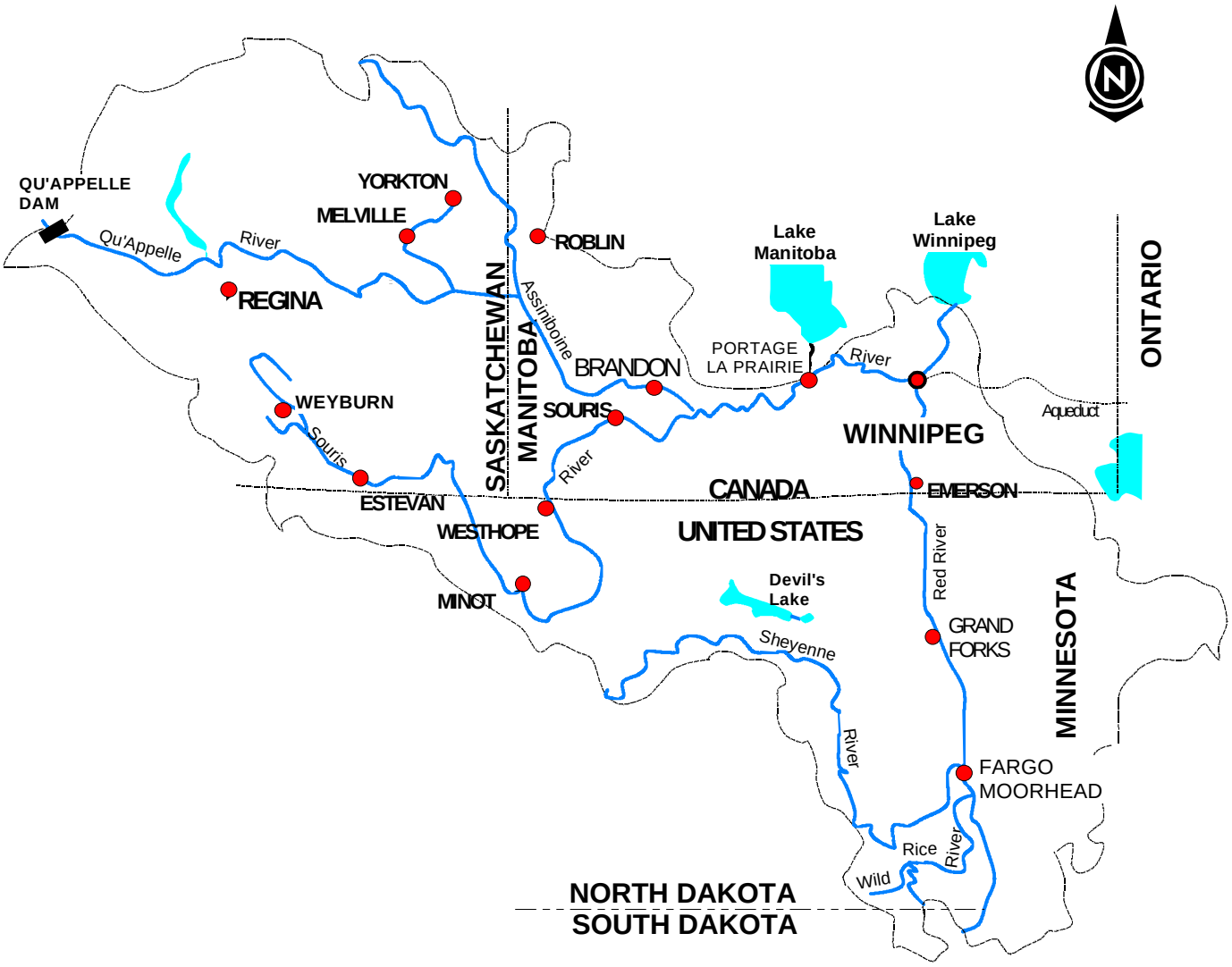
APPENDIX 8A
General Overview of
Study Area Hydrology

1.0 GENERAL AREA OVERVIEW

The City of Winnipeg operates three Water Pollution Control Centres. The outflow from the North End Water Pollution Control Centre and the South End Pollution Control Centre is released into the Red River, while the outflow from the West End Pollution Control Centre is released into the Assiniboine River. The Red River begins in the United States and flows north along the North Dakota-Minnesota border and into Canada. The Red River flows through the City of Winnipeg where it receives the flow from the Assiniboine River at The Forks in central Winnipeg. It then flows north into Lake Winnipeg. The Assiniboine River originates in Saskatchewan and flows east through southern Manitoba then into the Red River in Winnipeg. The Red and Assiniboine Rivers drain the prairie regions of southern Manitoba, southeastern Saskatchewan, North Dakota, northern South Dakota, and northwestern Minnesota (Figure 1-1). The main tributaries of the Red and Assiniboine rivers include the Ottertail, Cheyenne, Pembina, Rouseau and Souris Rivers, Red Lake, and numerous small rivers and streams. The basins of both the Red and Assiniboine rivers are almost entirely underlain by limestone bedrock covered with a thick clay deposit. The Red River valley plain is level, while the Assiniboine River passes through the Manitoba escarpment in the western portion of the province. The total drainage area exceeds 270,000 km^2 (MacLaren 1986). Much of the basin has been extensively drained for agricultural purposes.

The annual total flow in both rivers is dominated by spring runoff. The snowmelt, in combination with spring rains, has caused major floods. Following the snowmelt, flows decrease steadily throughout the summer, with minimum annual flows typically occurring in January or February. Annual average flows on the Red River upstream of Winnipeg at Ste. Agathe are 176.82 m^3/s (1958-2005 data). Annual average flows at Lockport, which include the contribution from the Assiniboine River, are 240.67 m^3/s (1962-2003 data). The average annual flows of the Assiniboine River at Headingley upstream of Winnipeg are 69.54 m^3/s (1974-2005 data).

River flows and levels are regulated throughout the drainage basin, with over 15 control structures (Wardrop/Tetr*ES* 1991). The Red River Floodway and the St. Andrew's Lock and Dam are the major hydraulic structures on the Red River in Manitoba, although many smaller ones have been built on tributaries such as the La Salle River. In the US, five major reservoirs are located on tributaries of the Red River: the Red Rock Reservoir on the Red Rock River, Orwell on the Otter Tail River, Bald Hill on the Sheyenne River, and Homme Dam on the Park River and Lake Traverse. On the Assiniboine River, important control structures include the Shellmouth Dam, which began operations in 1970, and the Portage Diversion. Five small



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The Red and Assiniboine Rivers Watershed

Figure 1-1

structures control flows on the Qu'Appelle River in Saskatchewan, which is a tributary of the Assiniboine River. The Souris River is also regulated within Saskatchewan.

Flood protection infrastructure on both the Red and Assiniboine Rivers is being upgraded. The Red River Floodway is currently being expanded to increase it's capacity to divert floodwaters from the Red River around the City of Winnipeg. The Shellmouth Dam on the Assiniboine River is also being upgraded to improve flood protection on the Assiniboine River.

Other projects that may affect the river basins include the Devil's Lake Outlet in North Dakota and plans to divert water from the Missouri River into the Souris River (tributary of the Assiniboine River) and the Red River. Devil's Lake has no natural inlet or outlet and in recent years this has resulted in flooding of the surrounding areas. The Devil's Lake project involves the diversion of 170,000 litres/minute of water from Devil's Lake into the Sheyenne River. The Devil's Lake Outlet has been under dispute by the Manitoba provincial government due to concerns about differences in water quality between Devil's Lake and the Red River basin. Foreign biota, salts, sulfates, and phosphorous have all been identified as potential water quality concerns related to the outlet (Government of Manitoba, *undated*).

1.1 REGIONAL LAND USE

Land use in the drainage basins is principally agricultural, but numerous cities and towns are located on the riverbanks. The principal urban centres are: Fargo, Moorhead, Grand Forks, Winnipeg and Selkirk on the Red River and Brandon and Portage la Prairie on the Assiniboine River. The agricultural use of the land affects water-quality through the runoff of nutrients, pesticides and sediments. Towns and cities and residential areas discharge domestic and industrial sewage that has received varying levels of treatment. Sections of the riverbank still remain in their natural state and support a variety of birds and mammals, while many aquatic species are present within the rivers.

1.2 FLOWS WITHIN THE STUDY AREA

The Water Survey of Canada maintains flow gauges at three locations near Winnipeg; these gauges are located at Ste. Agathe and Lockport on the Red River and at Headingley on the Assiniboine River. Ste. Agathe, located upstream of the City of Winnipeg on the Red River, has a record of daily flows from 1958 to 2005. The Headingley Station located just upstream of the City of Winnipeg on the Assiniboine River has a record of daily flows from 1913 to 2005. The Lockport Station, located downstream of Lockport, has a daily record from 1962 to 2003.

Frequency of flows at the St. Agathe and Lockport locations has been analyzed with data from the beginning of measurement until the end of 2003 (i.e., the last complete year at the time of analysis). Frequencies of flows at the Headingley station were analyzed from 1974 to the end of 2005 to cover the period of operation of the Shellmouth Dam.

Daily flows at Ste. Agathe were analyzed in order to determine the frequency of various flows at any time of the year. A frequency analysis was done on each date of the year for the period of record. The results are shown on Figure 1-2 and in Table 1-1. The following analysis is illustrated on Figure 1-2:

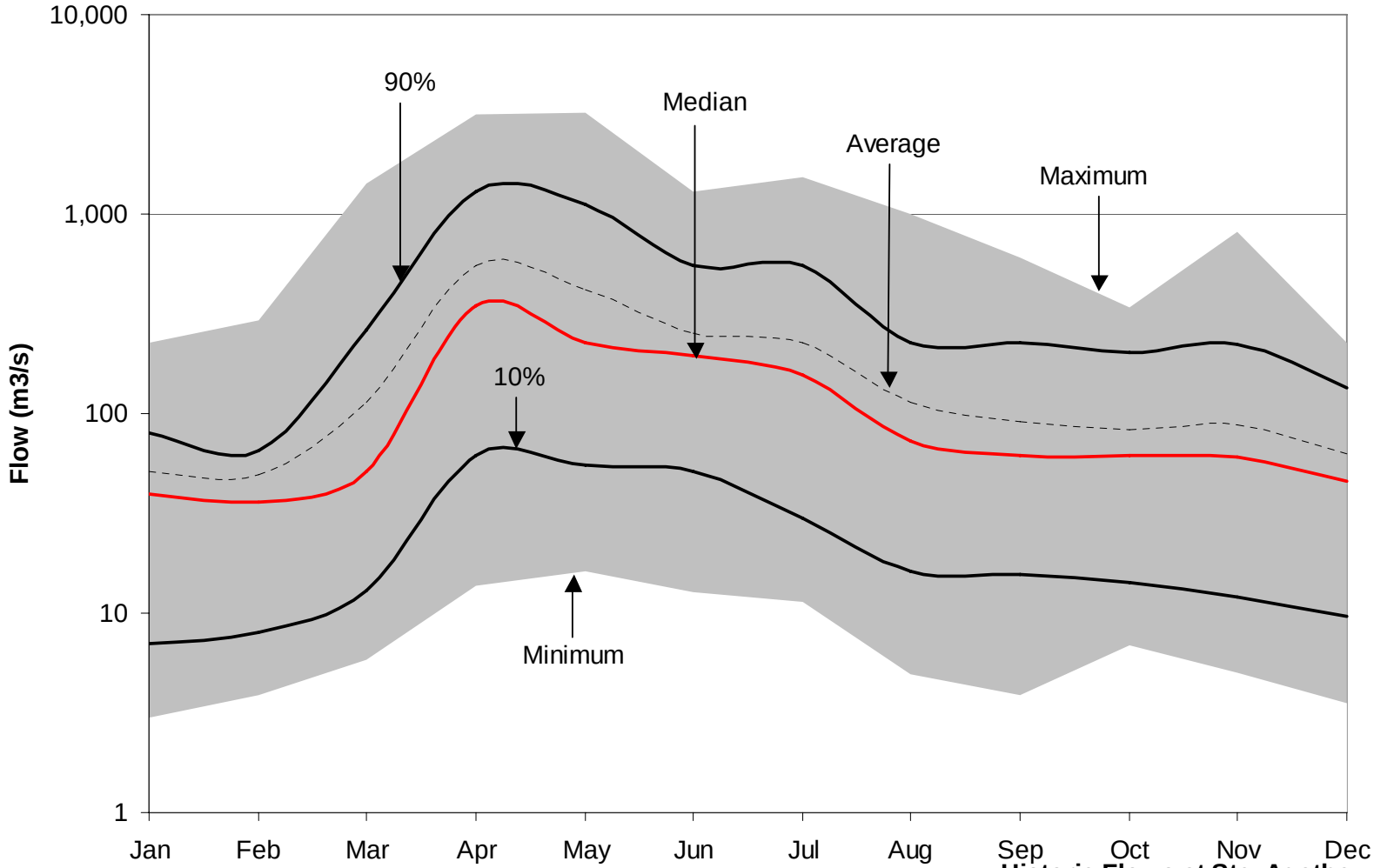
- The daily maximum flow in the historic record.
- The daily 90th percentile flow.
- The daily median flow.
- The daily mean flow.
- The daily 10th percentile.
- The daily minimum flow.

It should be noted that the scale on Figure 1-2 is a log scale, indicating the large variability in flow from year to year. This variability indicates that there is considerable change in river conditions from year to year.

Table 1-1
Summary of Daily Flow Data in the Red River at Ste. Agathe

Month	Daily Flows (m^3/s)[1]					
	Minimum	10th Percentile	Median	Mean	90th Percentile	Maximum
Jan	3	7	40	52	81	225
Feb	4	8	36	50	66	294
Mar	6	13	51	115	262	1430
Apr	14	62	348	552	1300	3170
May	16	55	226	418	1110	3230
Jun	13	51	196	251	556	1290
Jul	11	30	157	226	553	1540
Aug	5	16	73	115	226	995
Sep	45	16	61	92	226	605
Oct	7	14	62	83	203	341
Nov	5	12	60	87	224	813
Dec	4	10	46	63	134	226

Notes: [1]Based on data from 1962-2005



Historic Flows at Ste. Agathe (1958-2005)

Figure 1-2

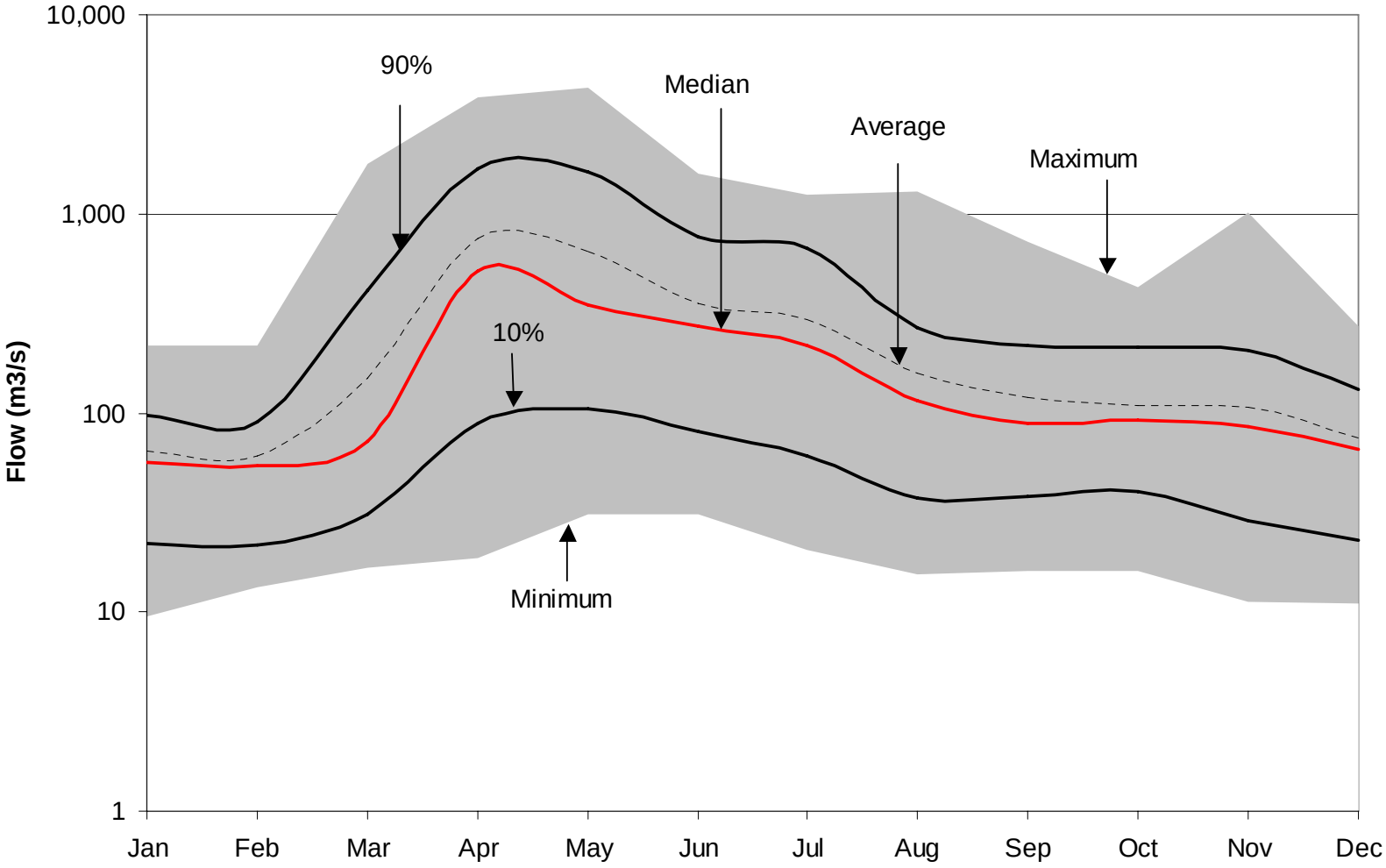
A similar analysis was done for the flows recorded at the Lockport Station (Figure 1-3 and Table 1-2). The flows at Lockport represent typical flows in the river downstream of The Forks within the City of Winnipeg. As with Ste. Agathe, the range of flows from year to year is considerable. The flows at Lockport are higher than those at Ste. Agathe due to contributions from tributaries, the most significant being the Assiniboine River. Figure 1-3 shows that the minimum flows are higher at Lockport indicating the moderating influence of the Assiniboine River flows.

Table 1-2
Summary of Daily Flow Data in the Red River at Lockport

Month	Flows (m^3/s)[1]					
	Minimum	10th Percentile	Median	Mean	90th Percentile	Maximum
Jan	9	22	57	64	98	217
Feb	13	22	54	61	90	217
Mar	17	31	71	149	410	1770
Apr	19	89	521	749	1694	3840
May	31	104	350	649	1630	4320
Jun	31	80	272	354	767	1590
Jul	20	61	217	294	675	1240
Aug	15	38	115	159	268	1300
Sep	16	38	88	119	218	723
Oct	16	41	91	109	215	427
Nov	11	29	85	106	206	1010
Dec	11	23	65	75	132	275

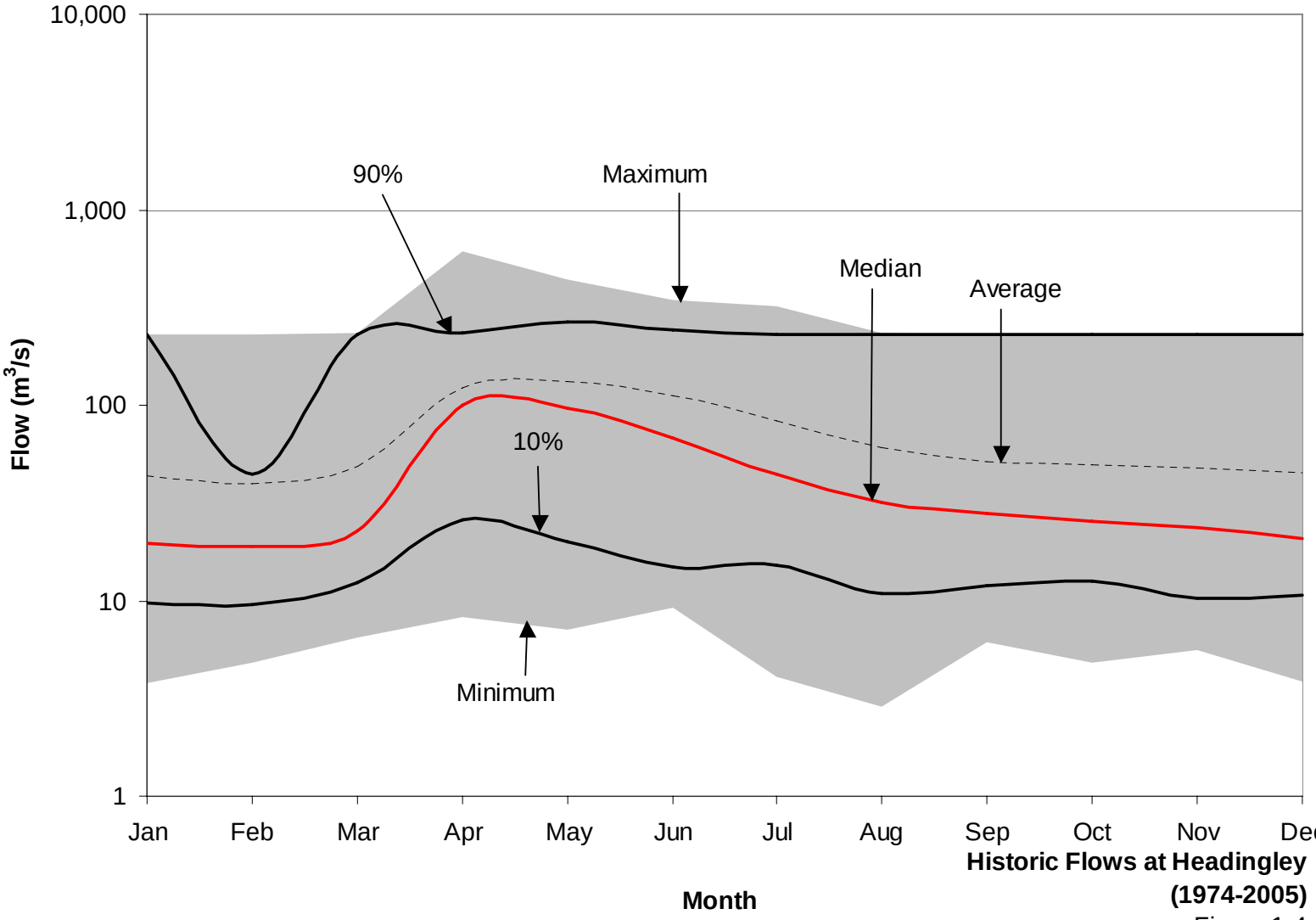
Notes:
[1]Based on data from 1962-2005

An analysis of flows on the Assiniboine at Headingley was done using the historical flows from 1974 to 2005 (Figure 1-4 and Table 1-3). Although earlier flow data is available, the 1974 start date was chosen as it is the beginning of the operation of the Shellmouth Dam. As with the Red River, the Assiniboine River flows are extremely variable from one year to the next. The operation of the Shellmouth Dam maintains the minimum flow at 5.6 m^3/s at any time of the year.



Historic Flows at Lockport (1962-2003)

Figure 1-3



Historic Flows at Headingley (1974-2005)

Figure 1-4

Table 1-3
Summary of Daily Flow Data in the Assiniboine River at Headingley

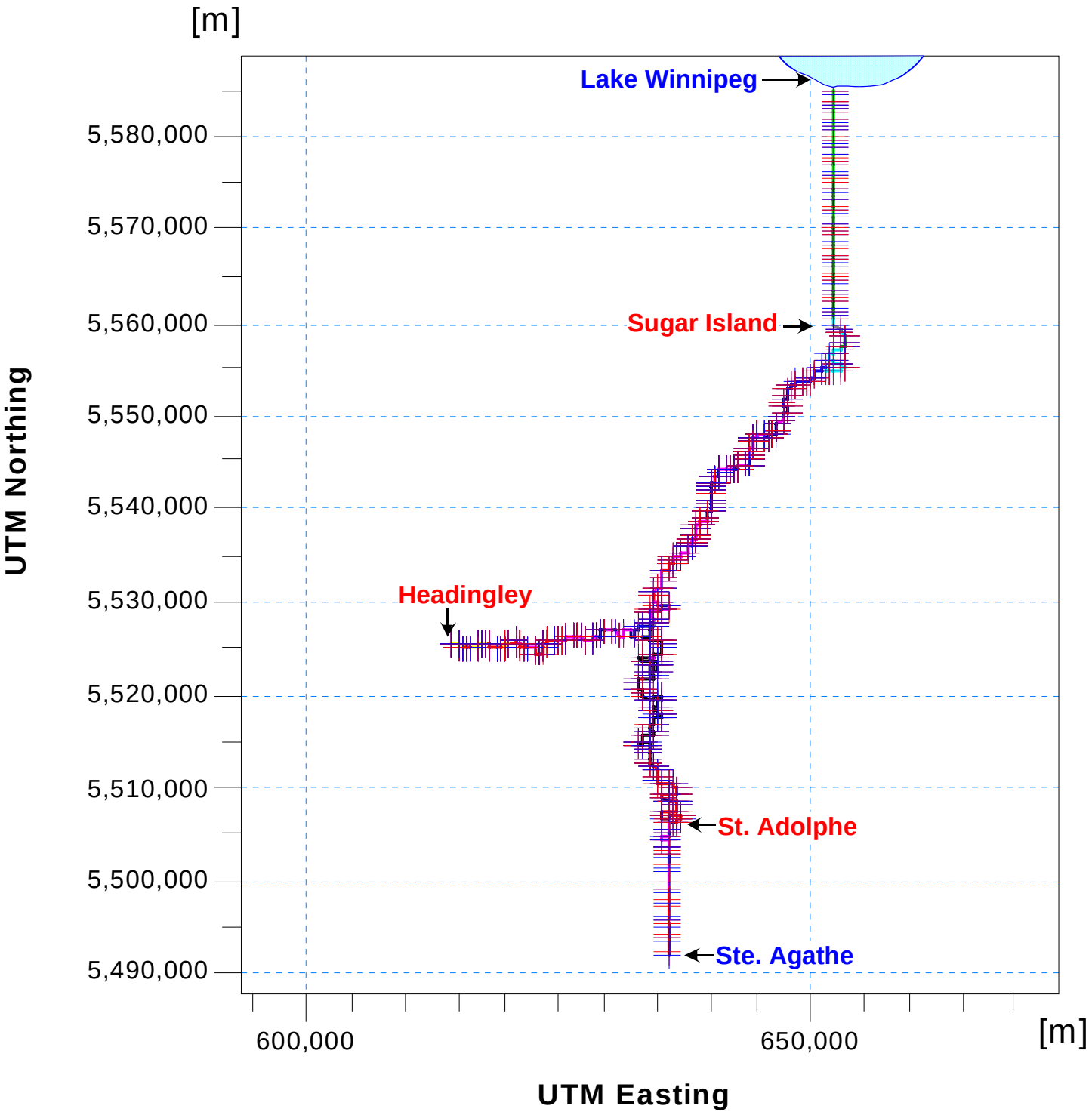
Month	Flows (m^3/s)[1]					
	Minimum	10th Percentile	Median	Mean	90th Percentile	Maximum
Jan	4	10	20	43	231	231
Feb	5	10	19	40	45	232
Mar	6	12	23	49	231	236
Apr	8	26	100	122	233	614
May	7	20	97	131	266	442
Jun	9	15	67	111	241	348
Jul	4	15	33	83	231	320
Aug	3	11	32	60	231	233
Sep	6	12	28	52	231	232
Oct	5	13	25	50	231	231
Nov	6	10	23	48	231	231
Dec	4	11	21	45	231	231

Notes:
[1]Based on data from 1974 - 2005

1.3 RIVER HYDRAULICS

A river study program (Tetr*ES* 2001) was previously conducted as part of a larger study on ammonia toxicity. In this study, a hydraulic model of the Red and Assiniboine Rivers was developed to understand how the river flows influence hydraulic parameters. The MIKE11 model, a sophisticated hydrodynamic model, was used for this analysis. The model encompassed the reach of the Red and Assiniboine rivers within the City of Winnipeg with boundaries at Headingley to the west, Ste. Agathe to the south and Lake Winnipeg to the north. The model was used to determine hydraulic parameters such as depth, water velocity, and wetted perimeter for a range of historic flows. The model was set up with 489 cross-sections, as shown on the model network schematic on Figure 1-5. Some typical cross-sections for the Red River are shown on Figure 1-6, while some typical cross-sections for the Assiniboine River are shown on Figure 1-7.

Hydrodynamic simulations were performed year-round to provide a better understanding of seasonal flow characteristics. The Lockport Dam was simulated as overflow gate position for



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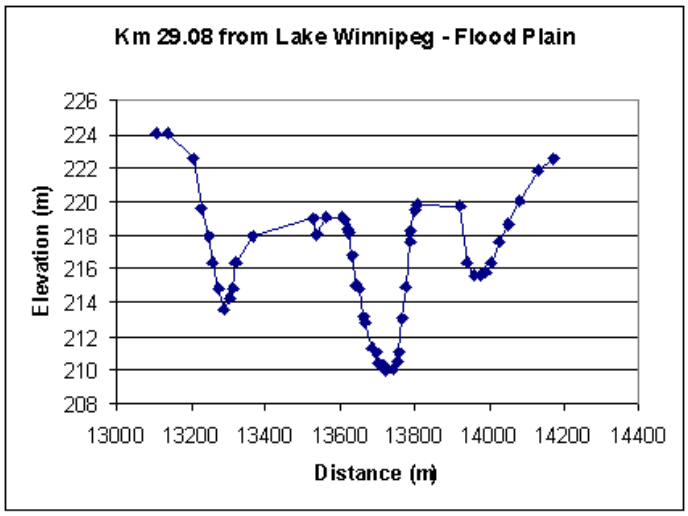
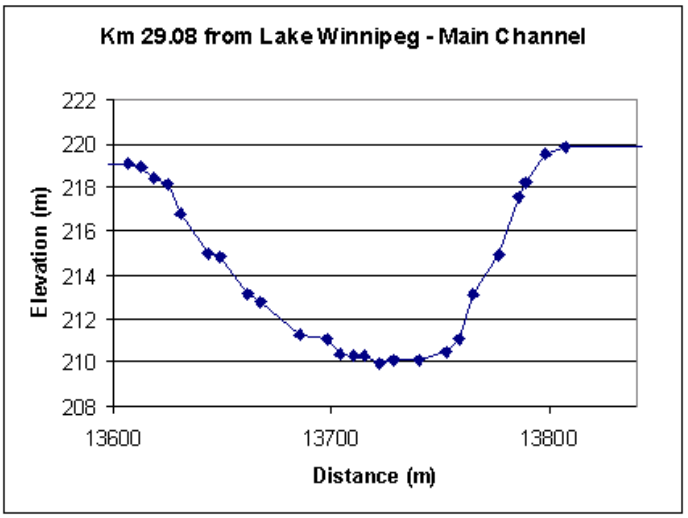
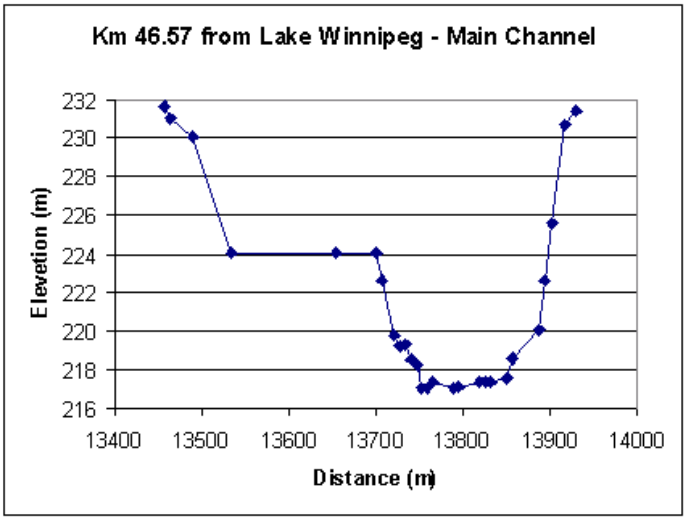
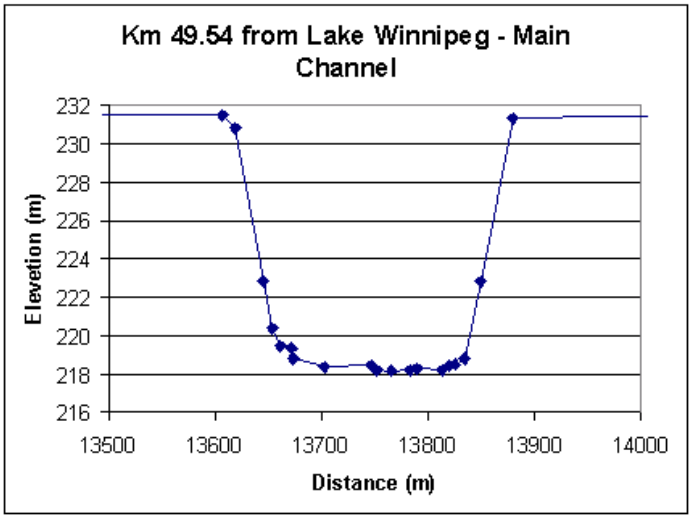
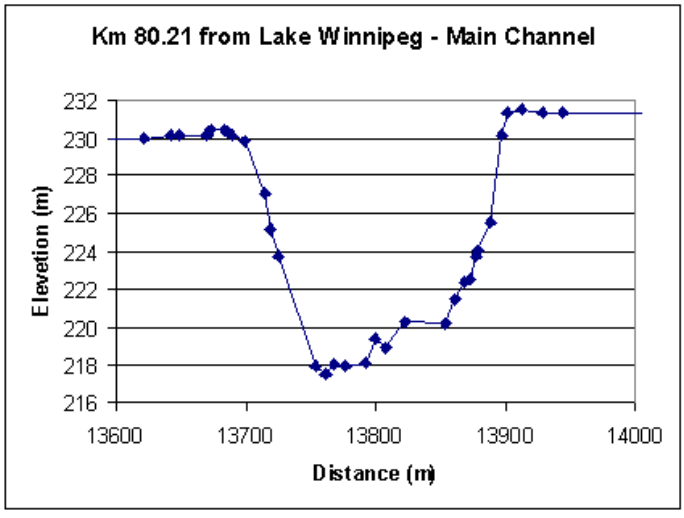
MIKE 11 Model Network
Figure 1-5

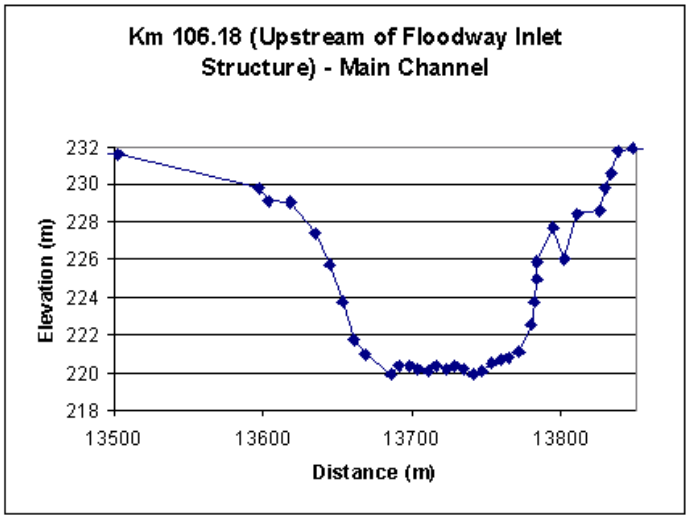












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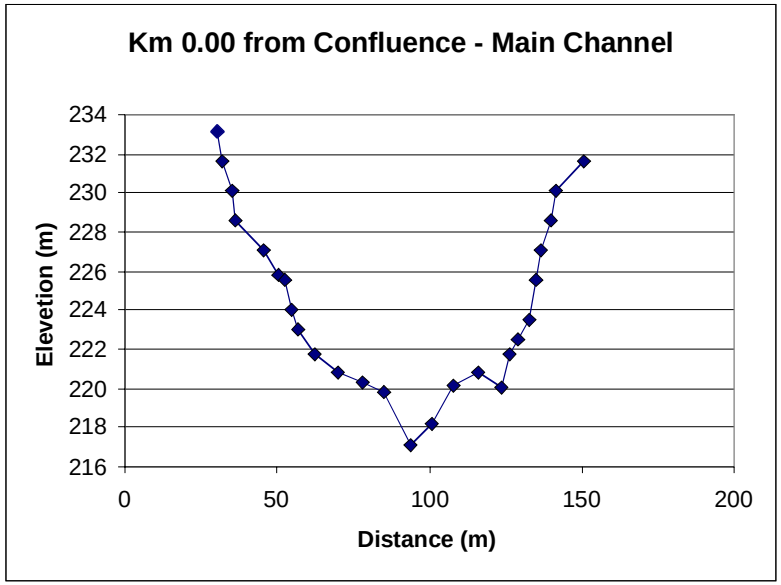
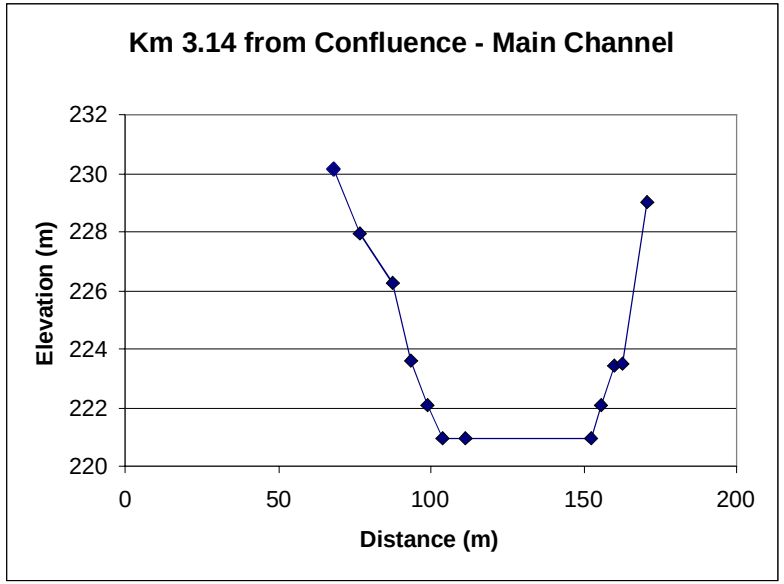
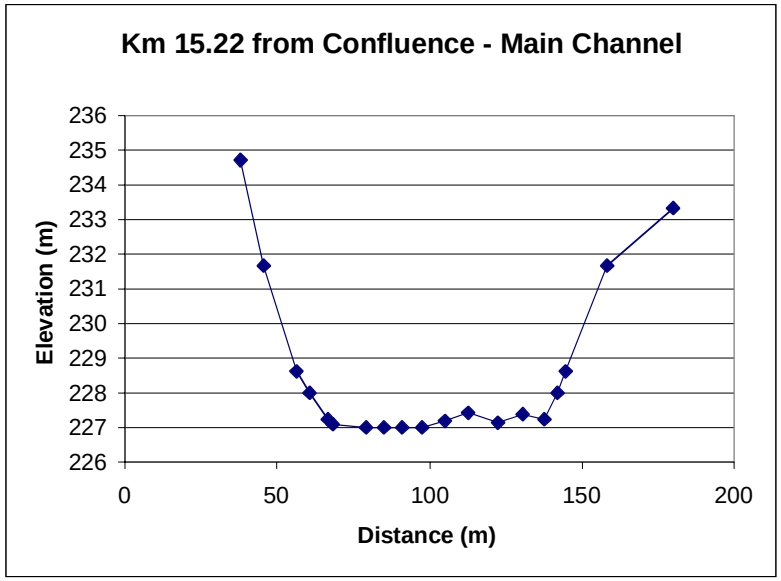
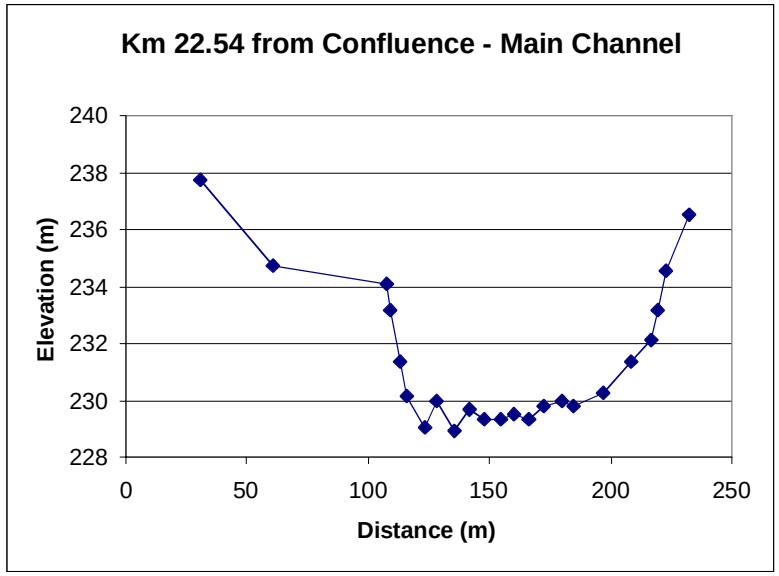
Typical Cross-Sections of the Red River
Figure 1-6









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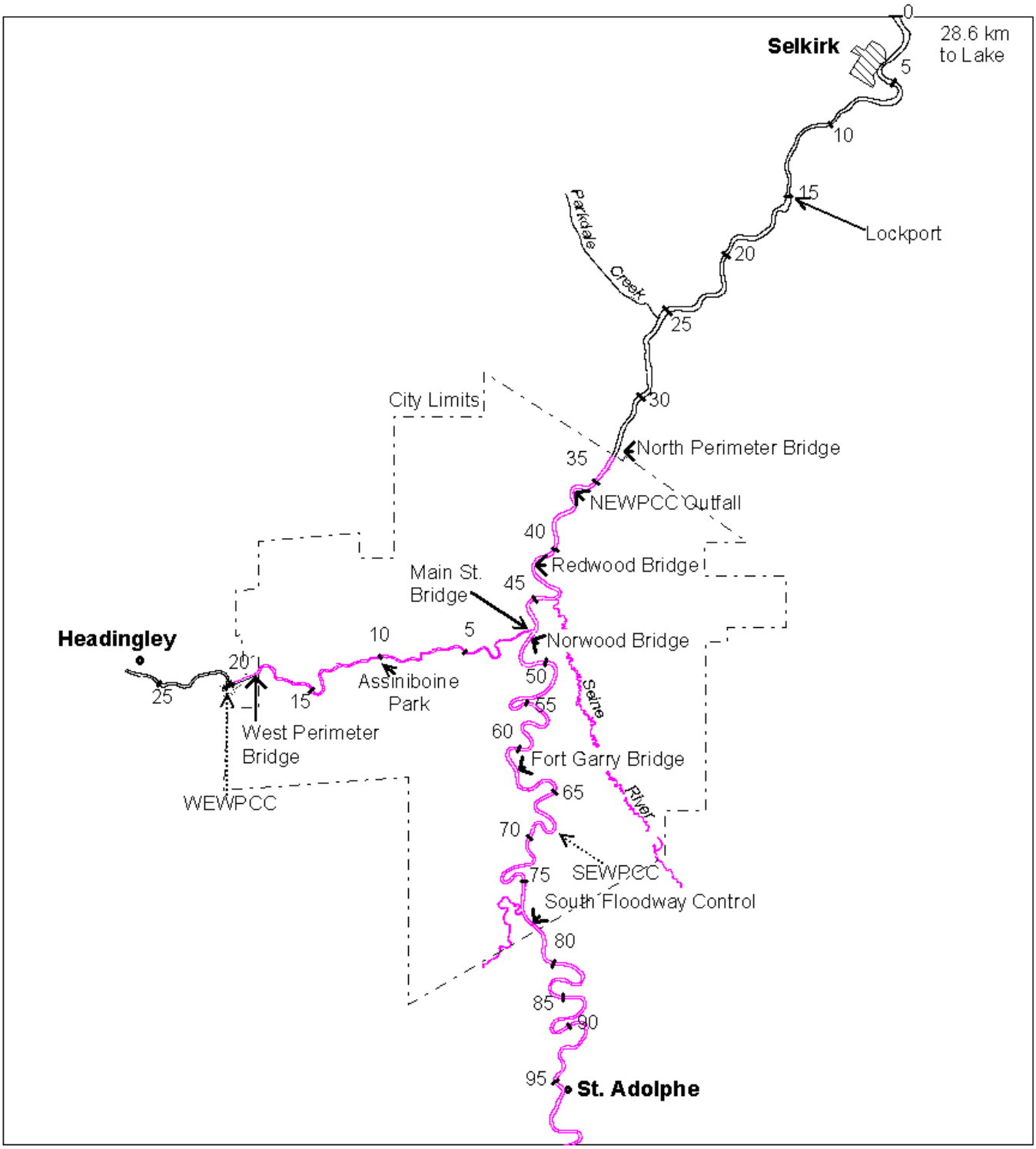
Typical Cross-Sections of the Assiniboine River

Figure 1-7



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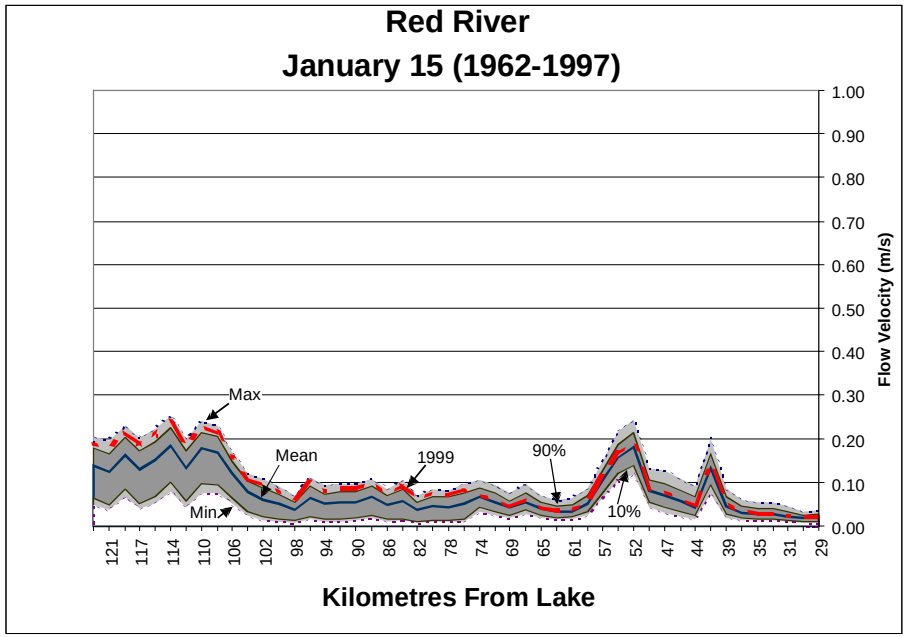
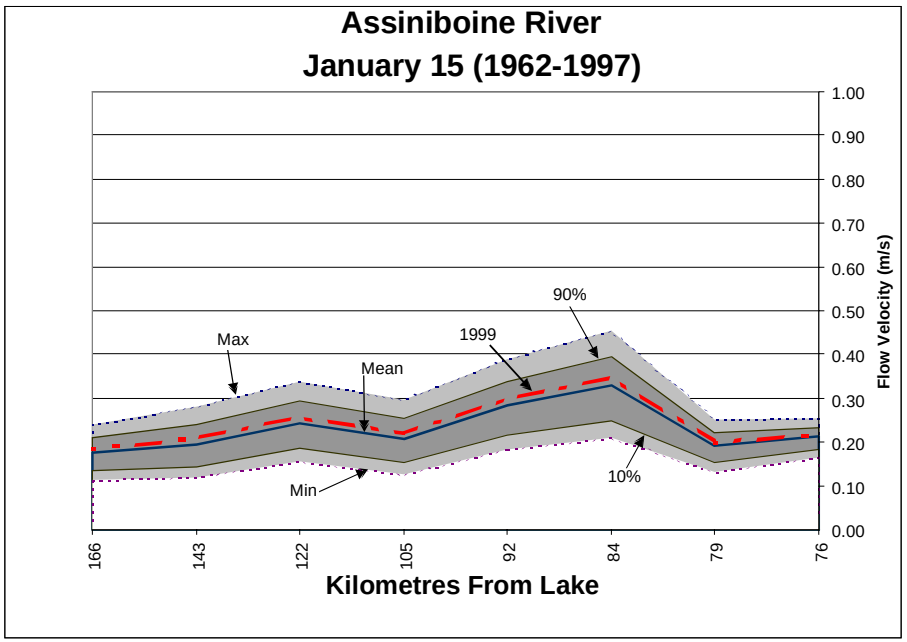
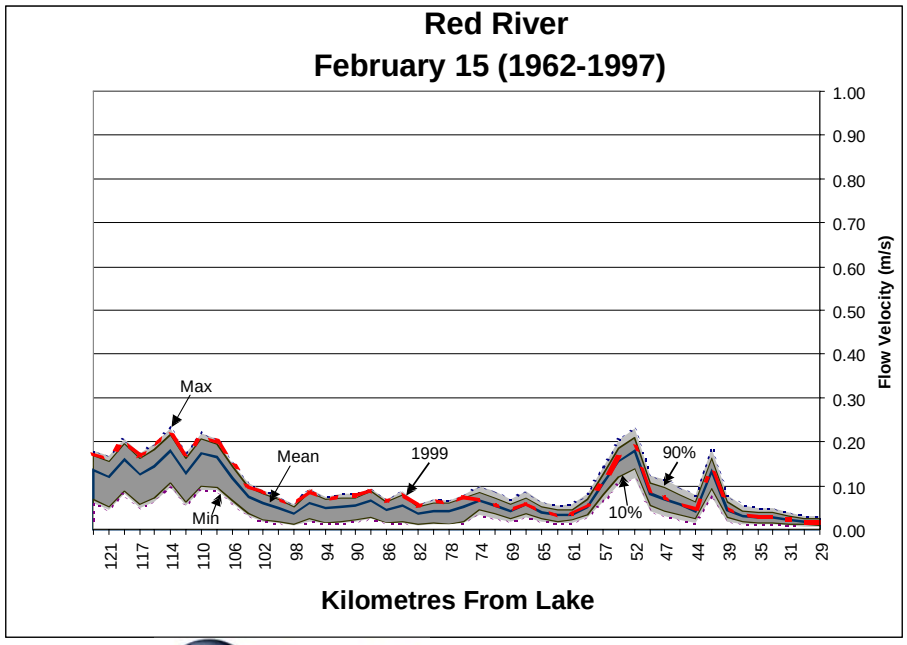
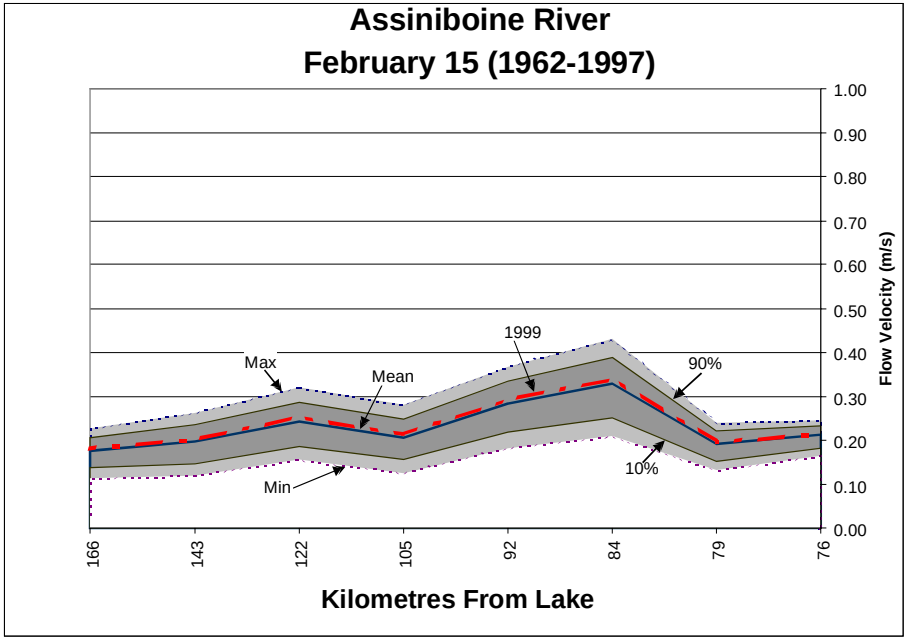
River Distances (km) in the Study Area and Locations of Key Features
Figure 1-8









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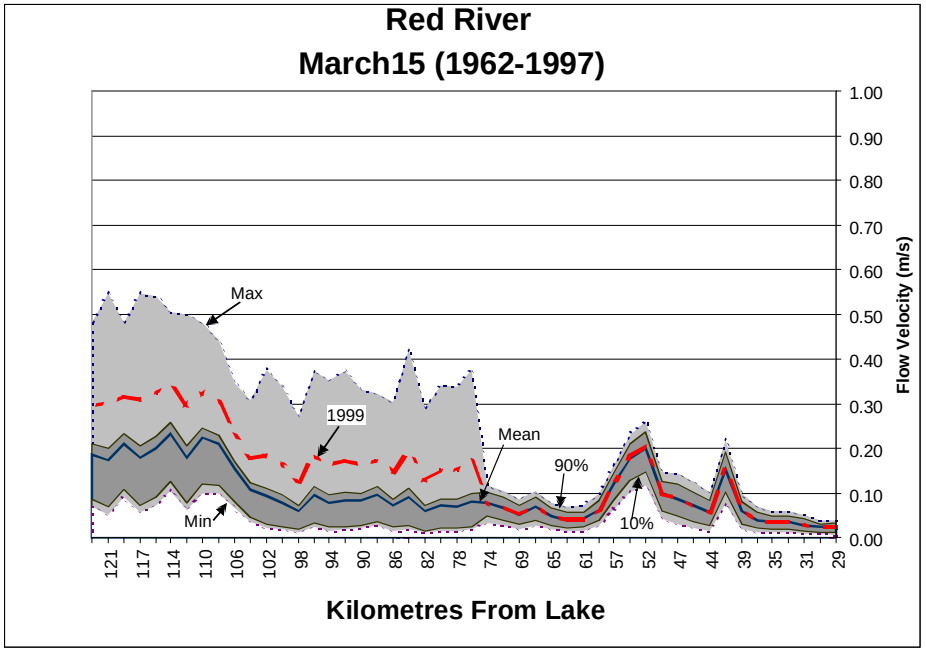
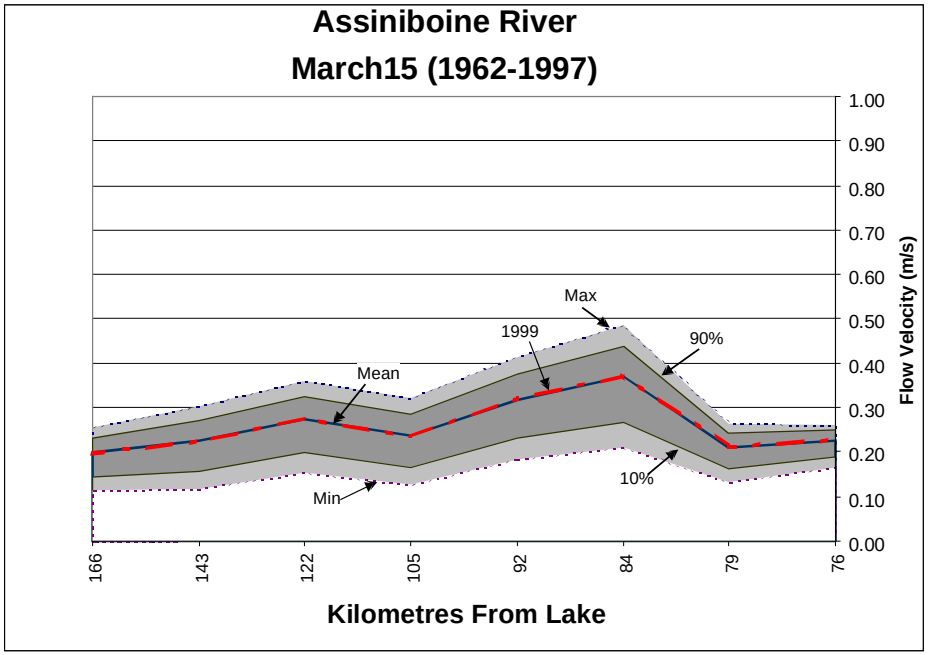
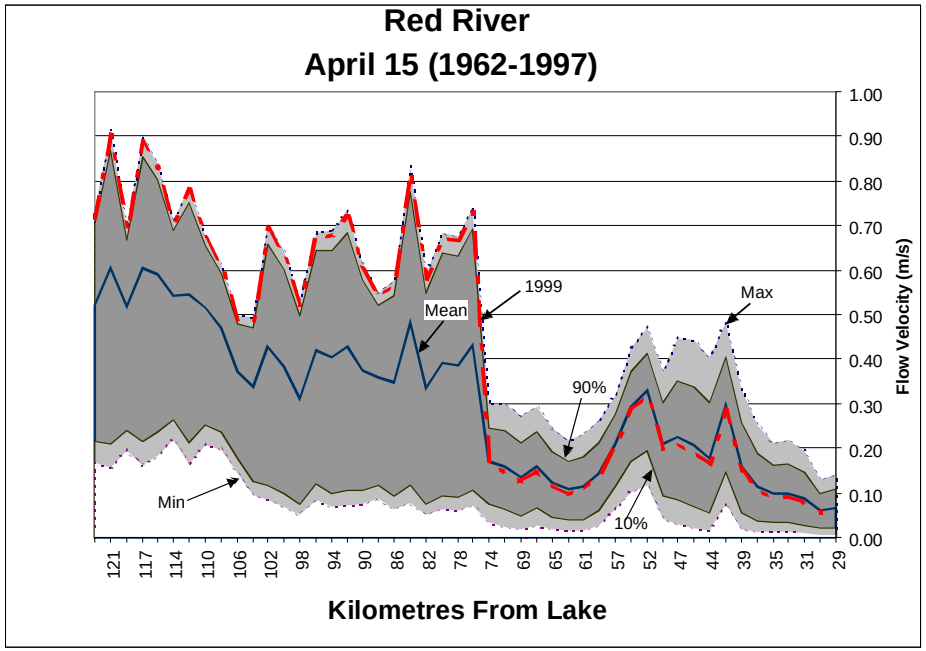
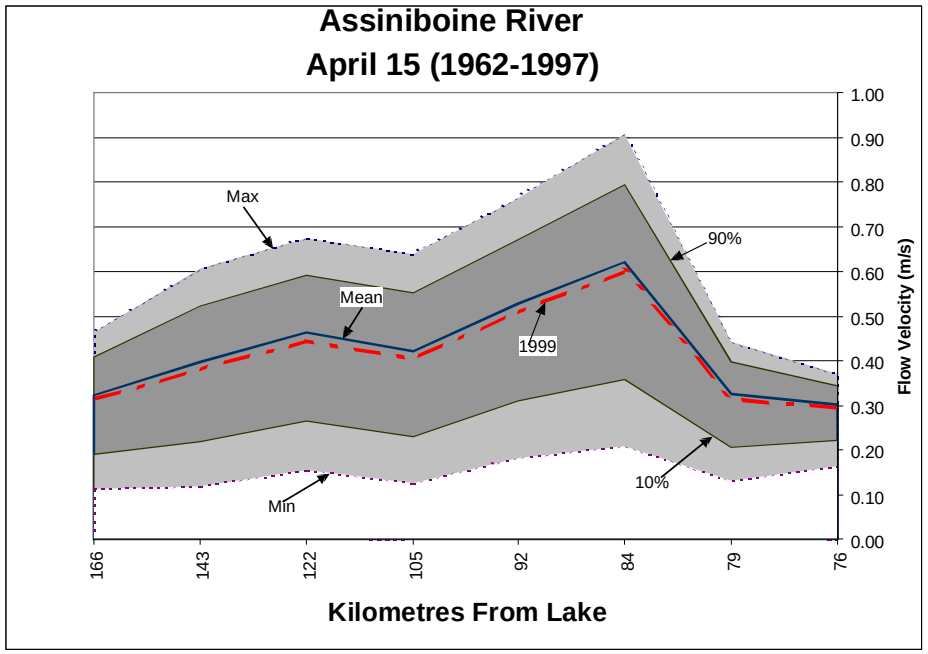
Range of Velocities for January and February

Figure 1-9









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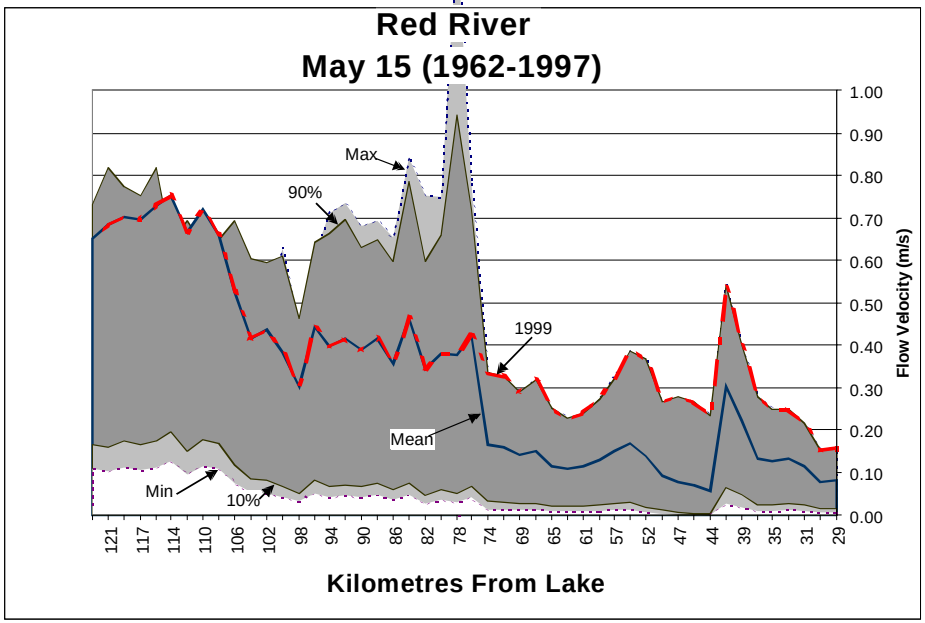
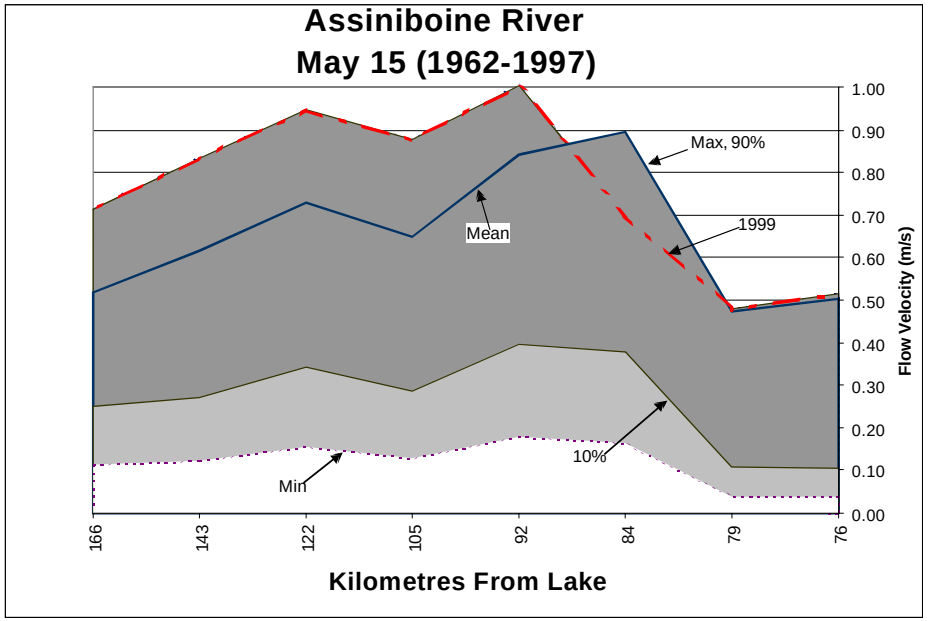
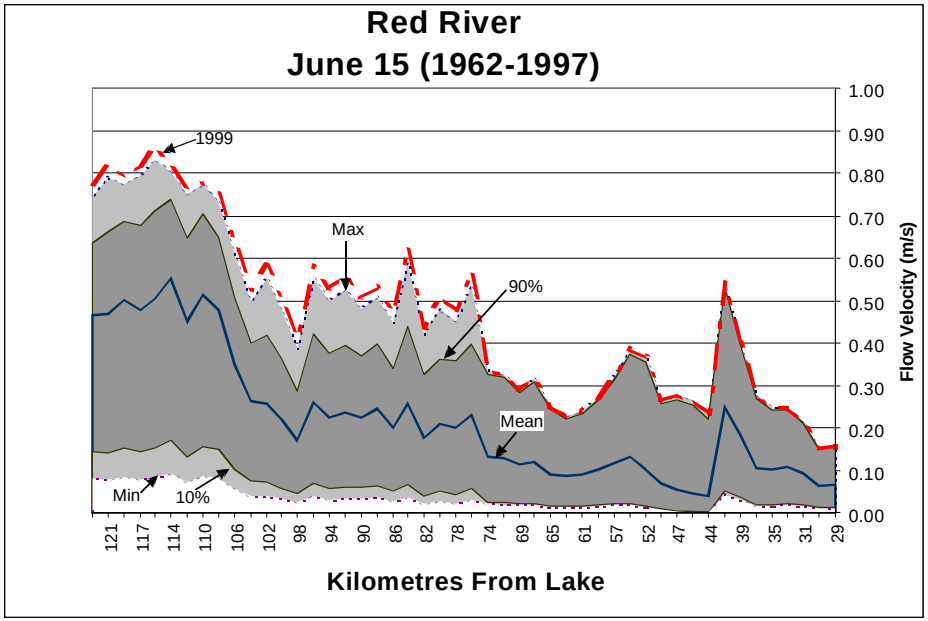
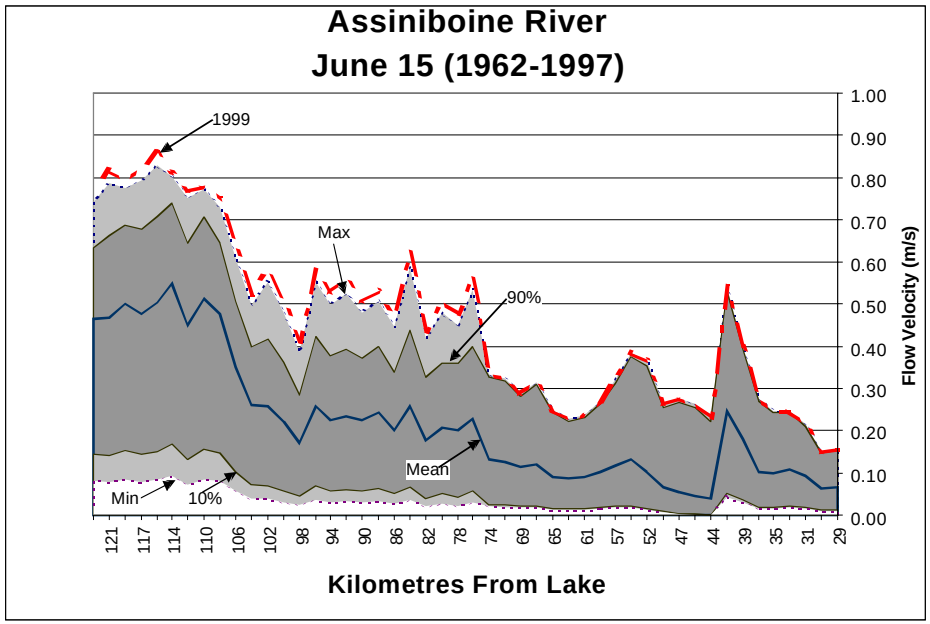
Range of Velocities for March and April

Figure 1-10









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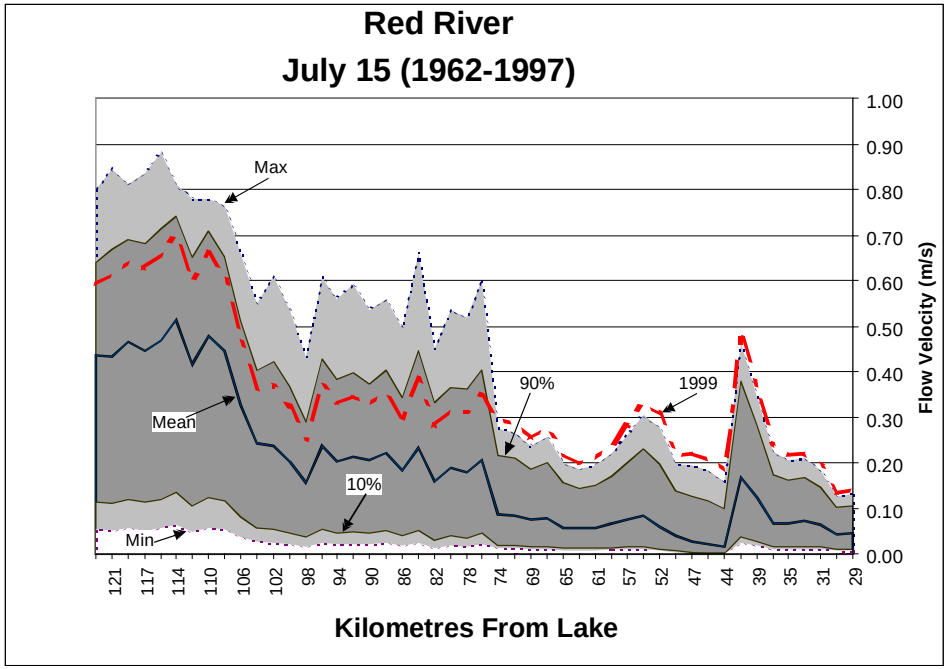
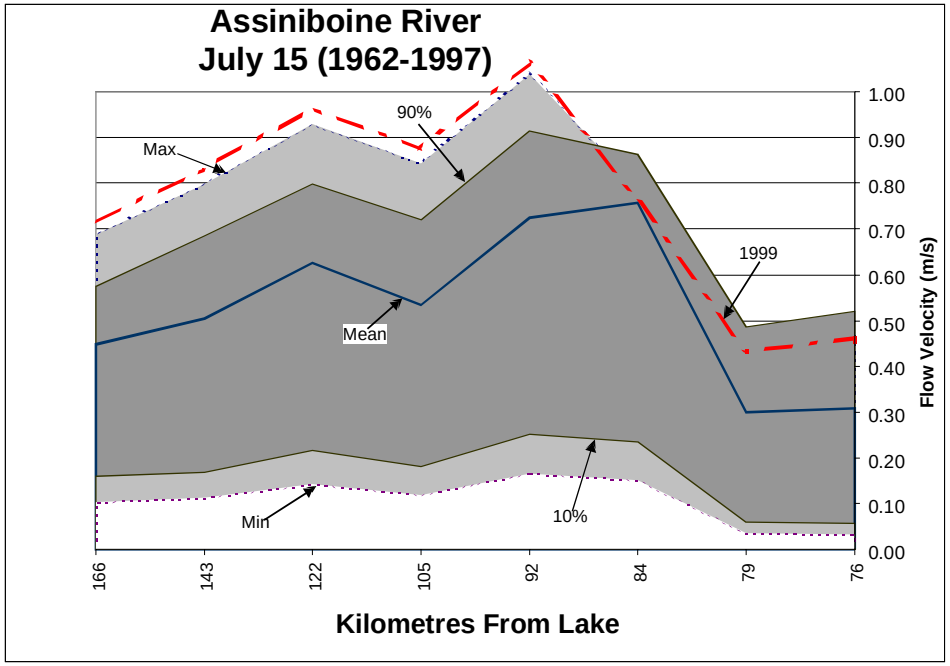
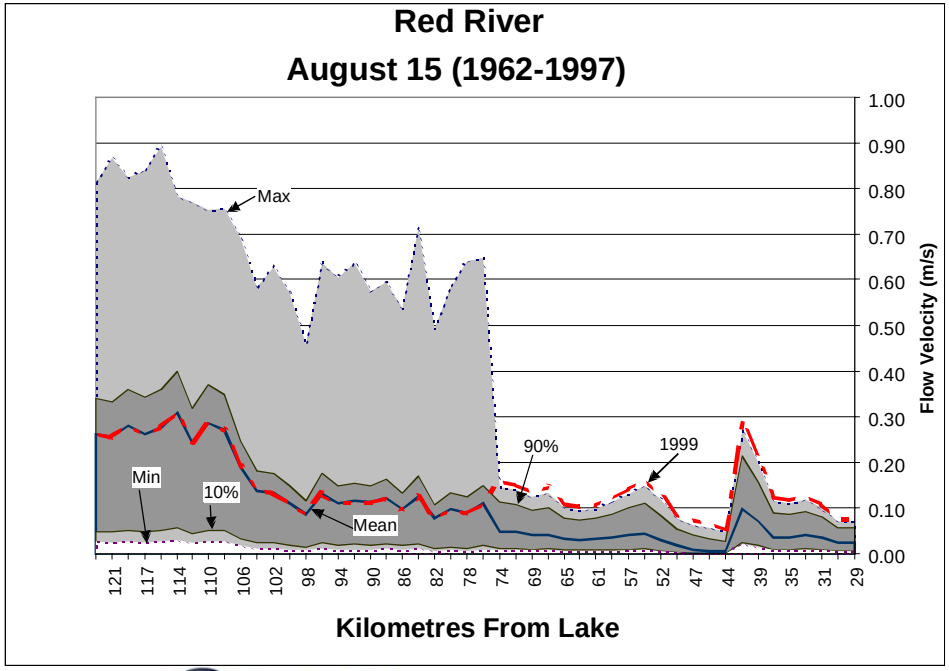
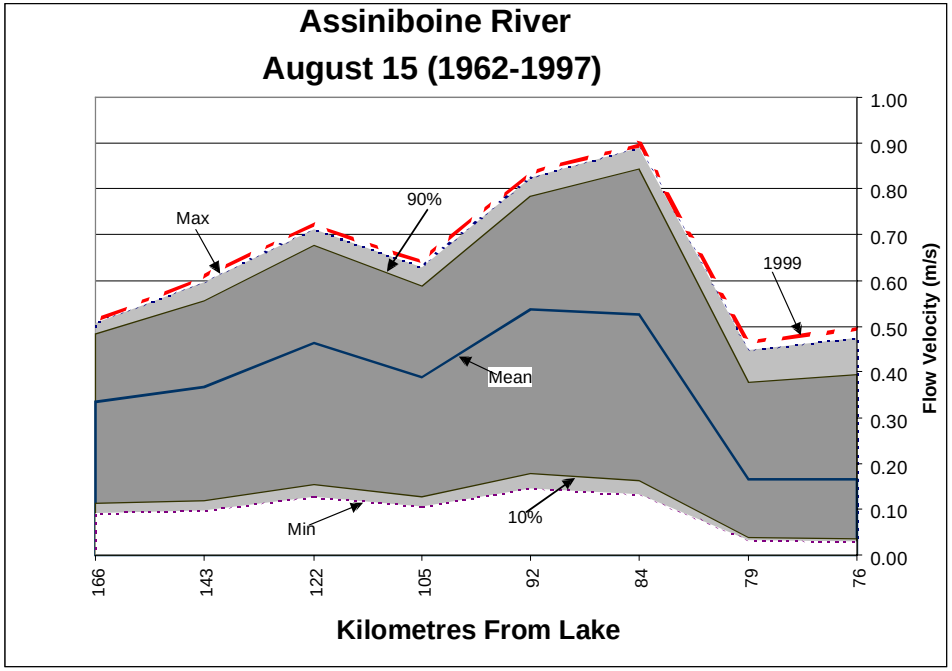
Range of Velocities for May and June

Figure 1-11









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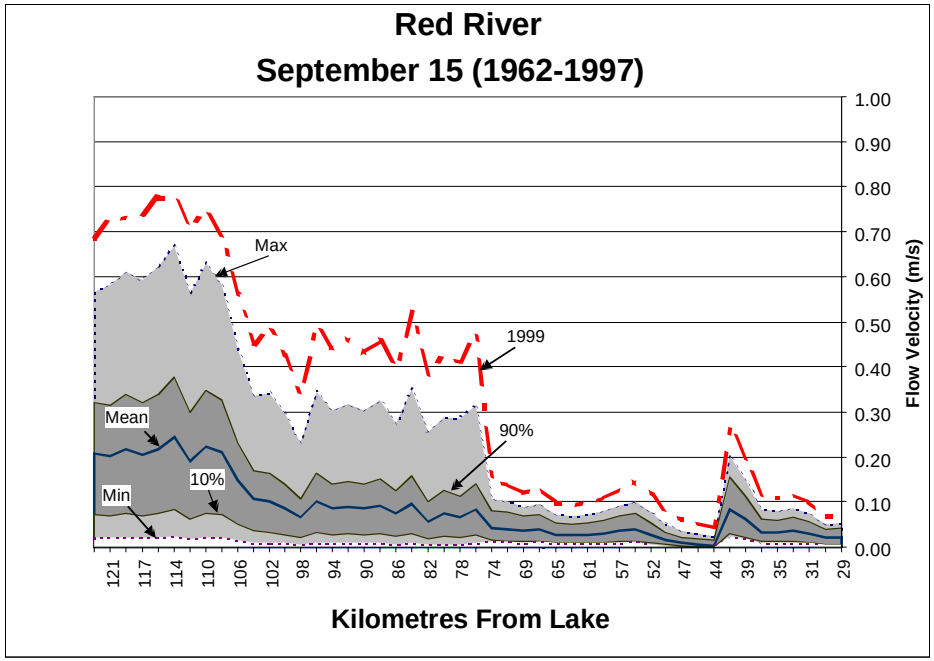
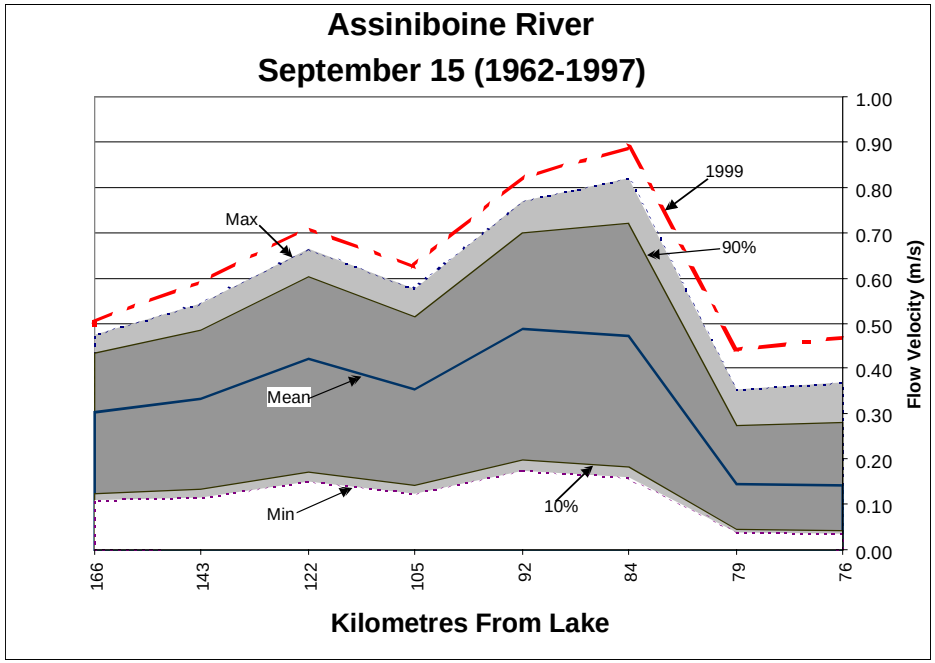
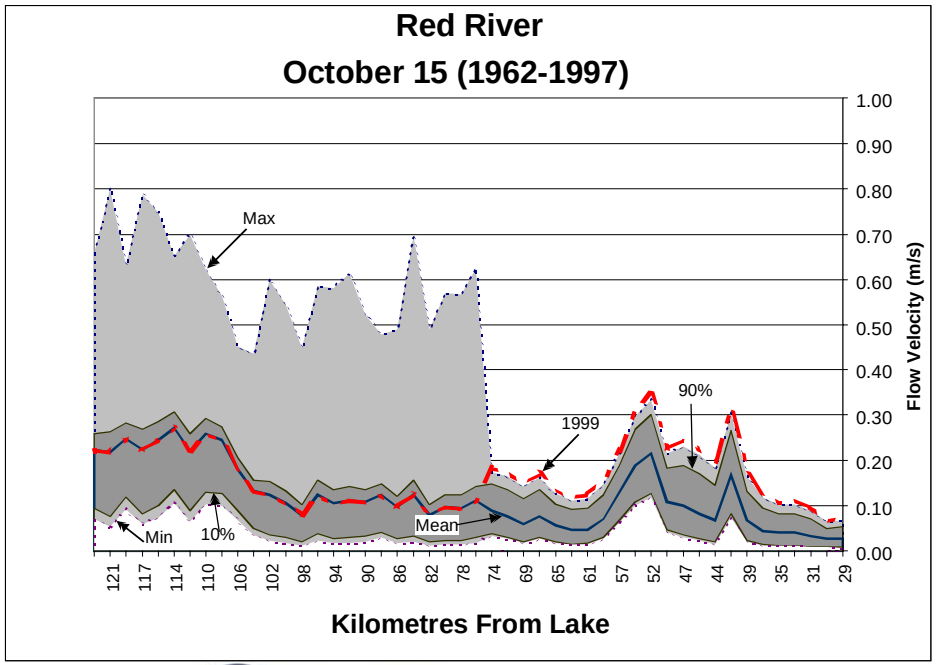
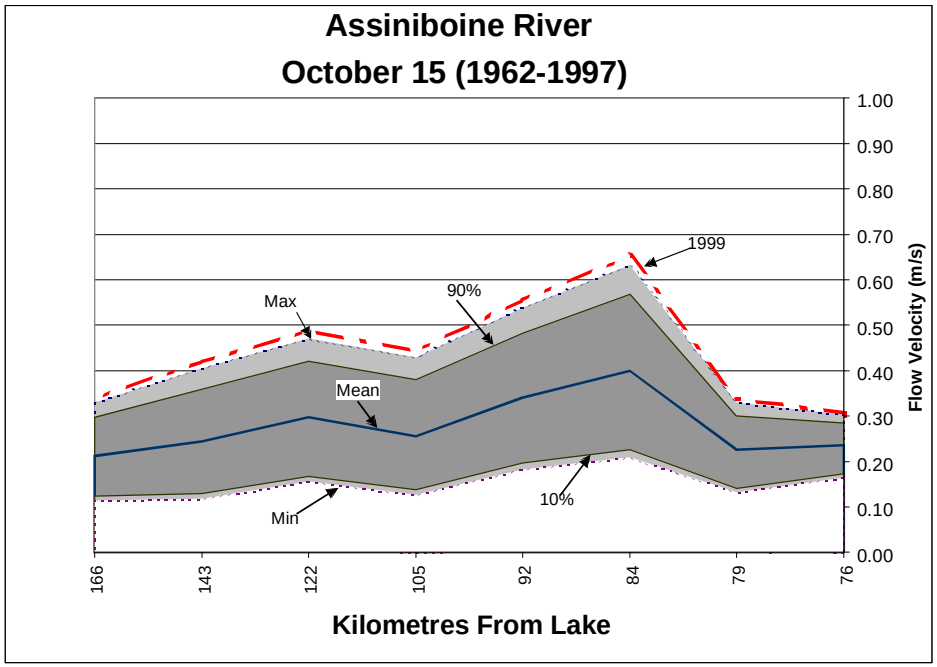
Range of Velocities for July and August

Figure 1-12









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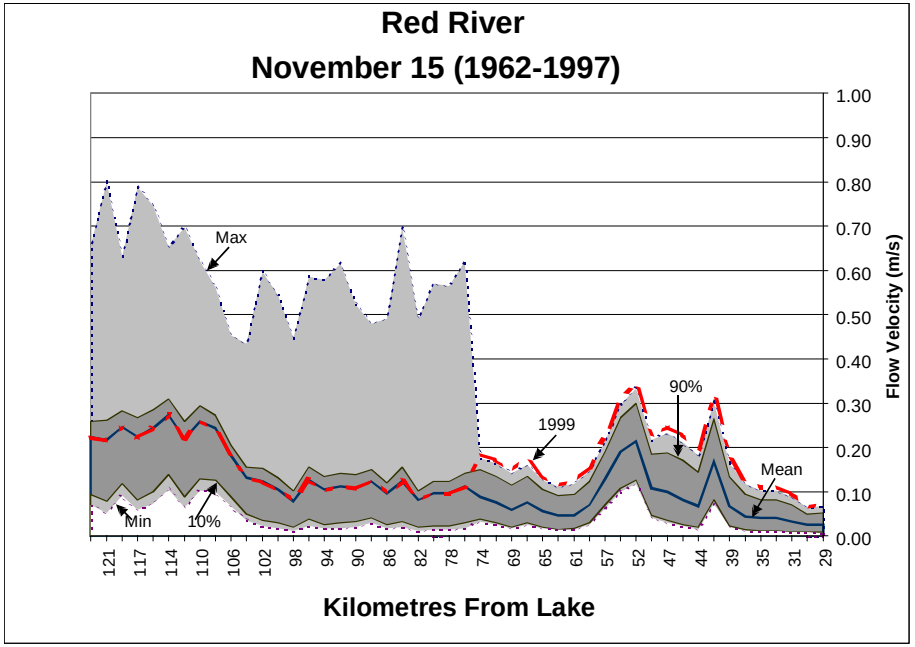
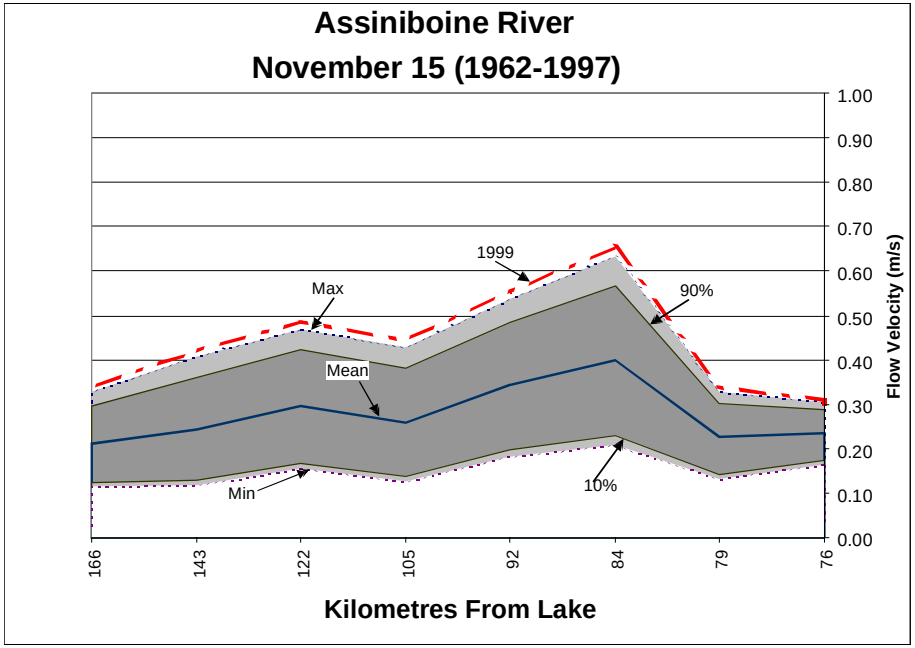
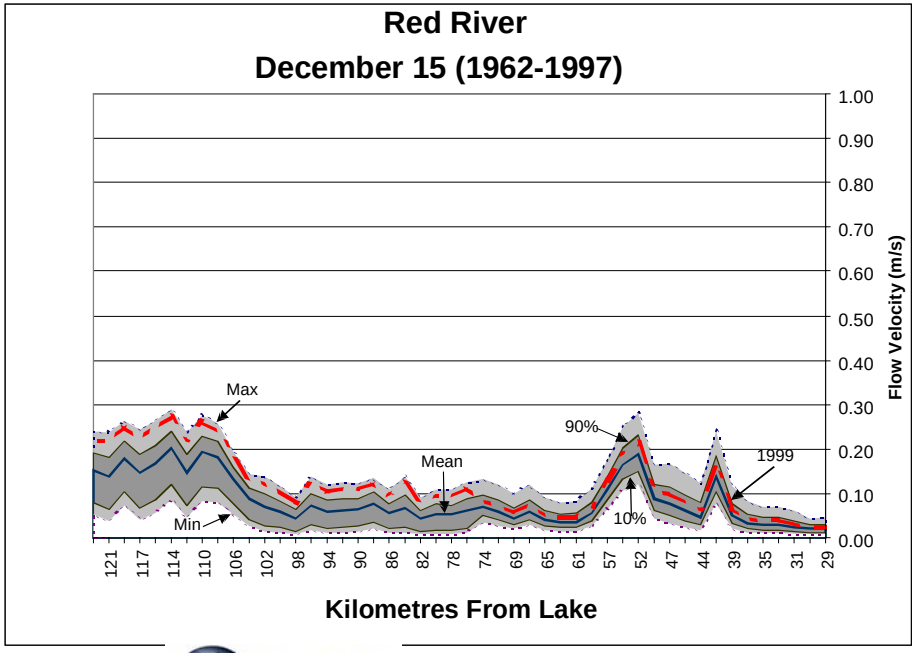
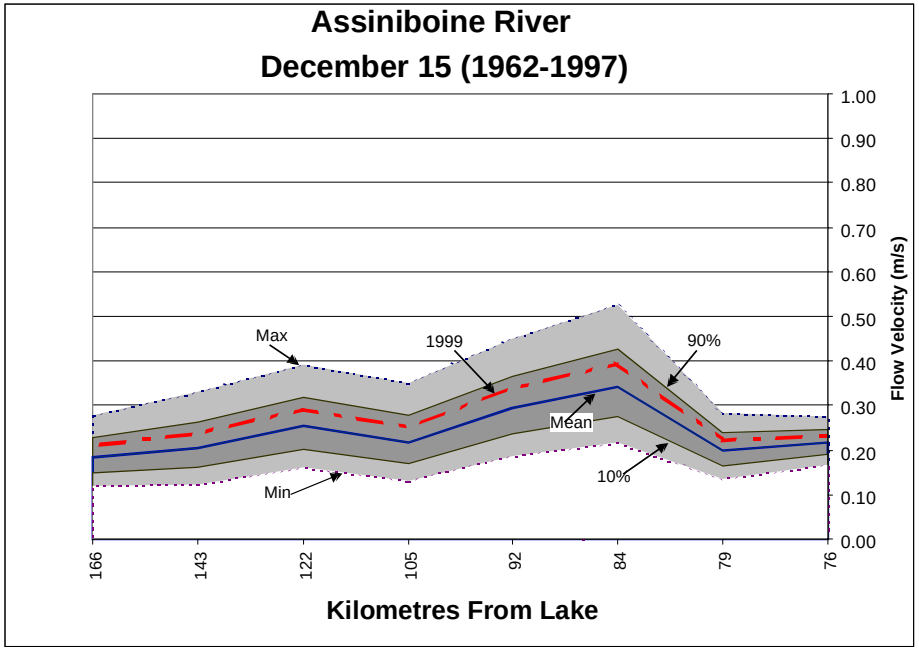
Range of Velocities for September and October

Figure 1-13









Range of Velocities for November and December

Figure 1-14

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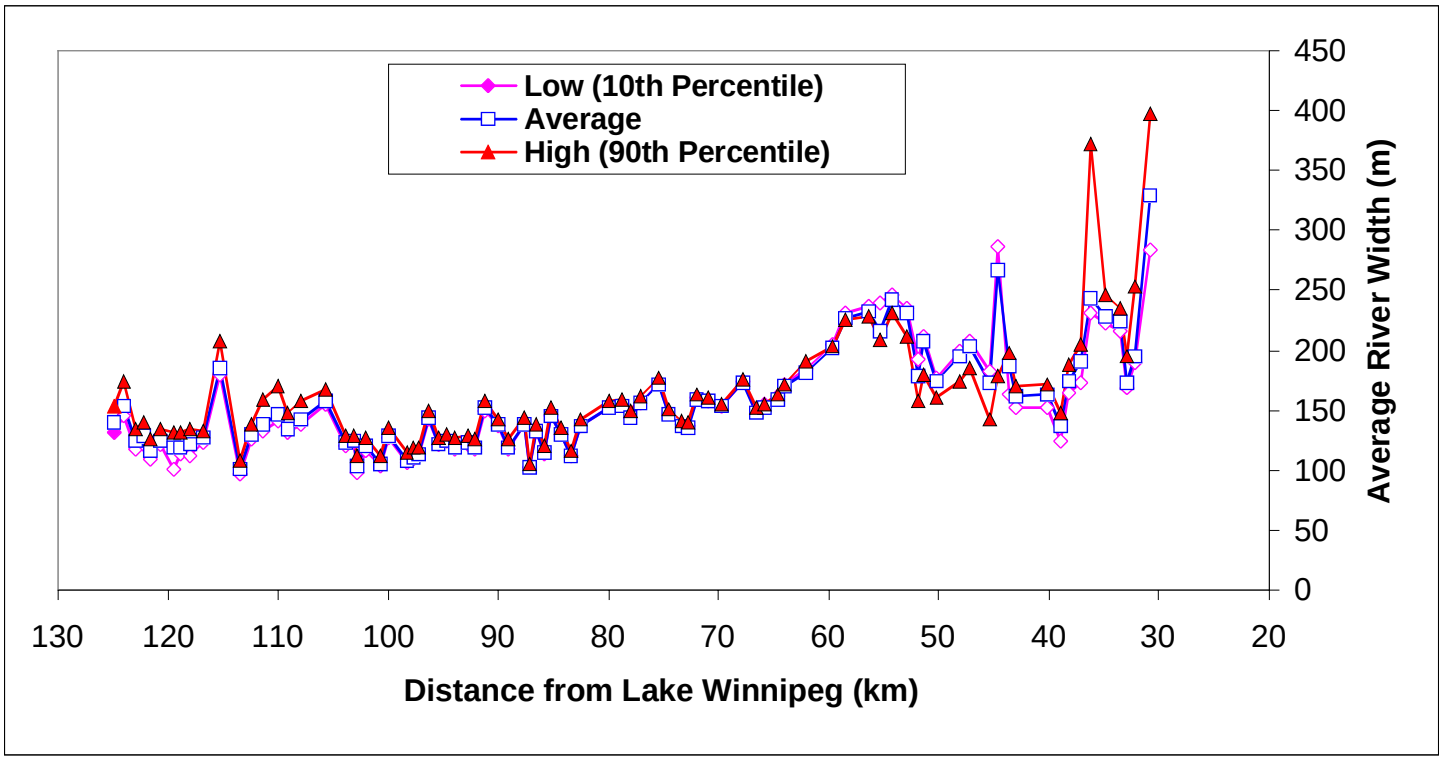
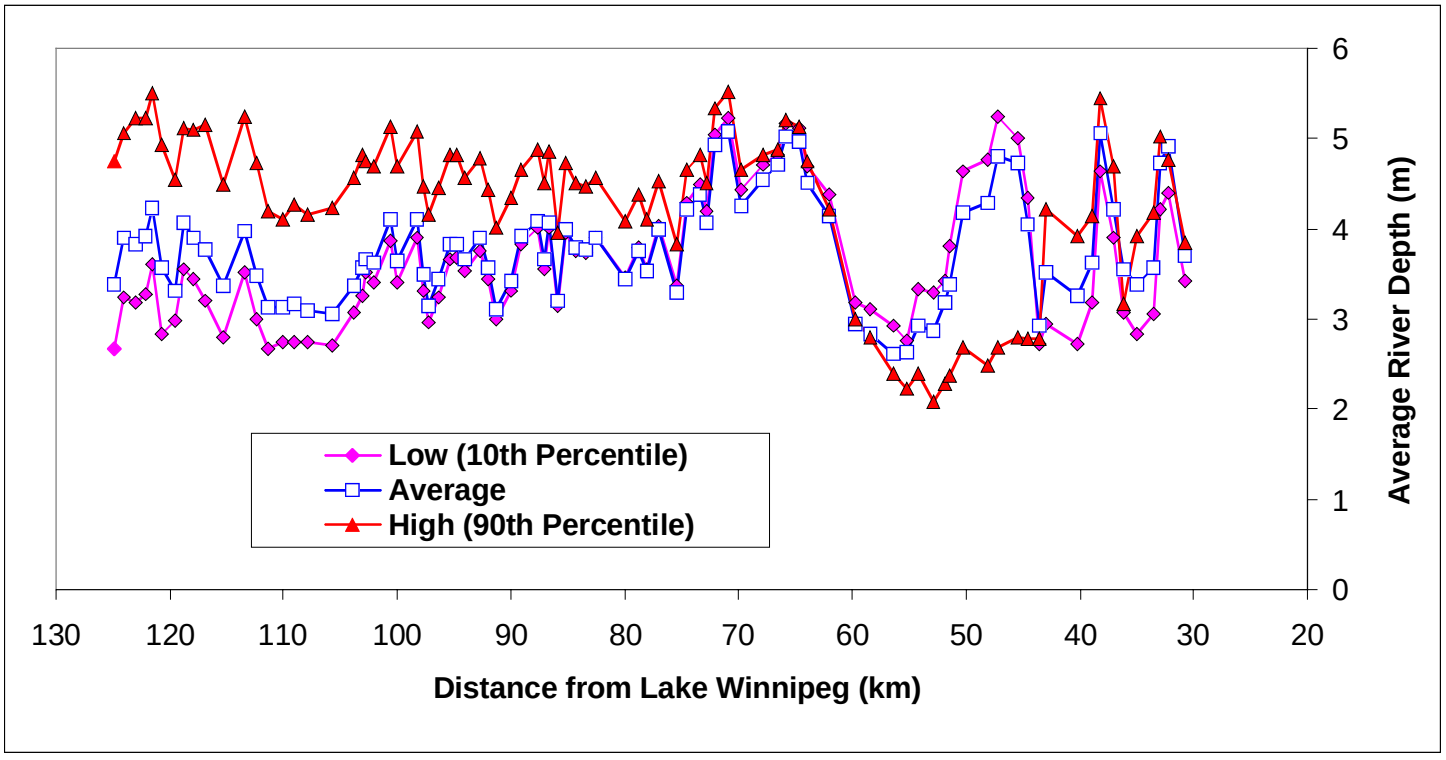
different flows to maintain 734 feet a.s.l. water elevation at James Avenue in the centre of Winnipeg. This operation of the dam maintains water levels within Winnipeg in order to provide recreation opportunities for boaters during summer.

In order to develop the simple relationship between flow and velocity needed to perform long-term water quality modelling for the 2001 study, the MIKE11 model was run through the full range of historic flow data from 1962 to 1997. The MIKE11 model was then developed to derive equations to calculate the velocity and depth for sections of the Red and Assiniboine Rivers based on flow. Leopold Maddox equations were used to calculate velocity and depth for the winter months. Due to the complicating effect of the Lockport gates, polynomial equations were used to calculate the velocities and depths during the summer. An analysis was then done to estimate the frequency of average velocities at various cross-sections through the river system as indicated on Figure 1-8. The results of this analysis are shown in Figures 1-9 to 1-14. Figures 1-9 and 1-10 show the velocities from January to April, Figures 1-11 and 1-12 show the velocities during the summer months and Figures 1-13 and 1-14 show the velocities from September to December. The analysis found the following:

- In January and February, velocities in the Red River are very low. They are almost always less than 0.2 m/s and typically much less than 0.1 m/s at many locations. In the Assiniboine River, velocities are generally in the range of 0.2 to 0.4 m/s during these two months.
- In March, the velocities in the Red River are generally less than 0.2 m/s except in years where there is an early spring melt. If there is an early spring melt, the velocities in the river upstream of The Forks range from 0.3 to 0.5 m/s and downstream of The Forks the velocities vary between 0.2 and 0.5 m/s. In the area of Lister Rapids the velocities are in the range of 0.2 m/s, while in other portions of the lower Red River, the velocities are less than 0.1 m/s. In March the Assiniboine River velocities are more consistent in the range of 0.2 to 0.4 m/s
- In April the Red River flows are extremely variable, depending on the size of the spring flood. Velocities can be as low as 0.1 m/s and as high as 0.8 m/s in the upstream reaches of the Red River. In the downstream reaches (i.e., at Lister Rapids) the velocities are generally in the range of 0.4 to 0.5 m/s. On the Assiniboine River the velocities in April are generally higher than in the Red River and range from 0.2 to 0.8 m/s.

The MIKE11 model was run at low (10^{th} percentile), average, and high (90^{th} percentile) flows to determine how the width and depth of the river respond to variations in flow. The width and depth of each reach was calculated for each of these flows. There was little variation in width for changing flows on all reaches of the Red River throughout the modelled area (Figures 1-15





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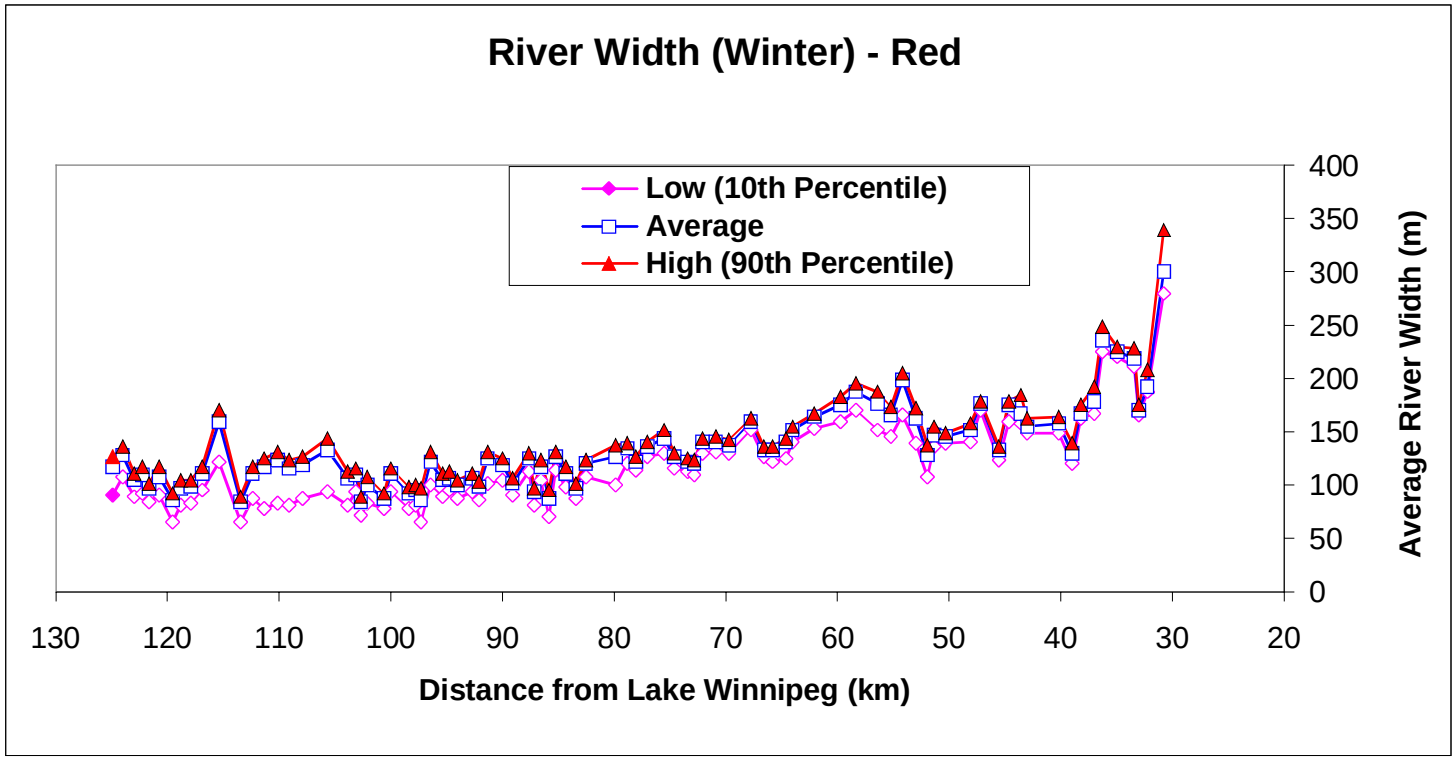
Range of Red River Width and Depth in Summer
Figure 1-15

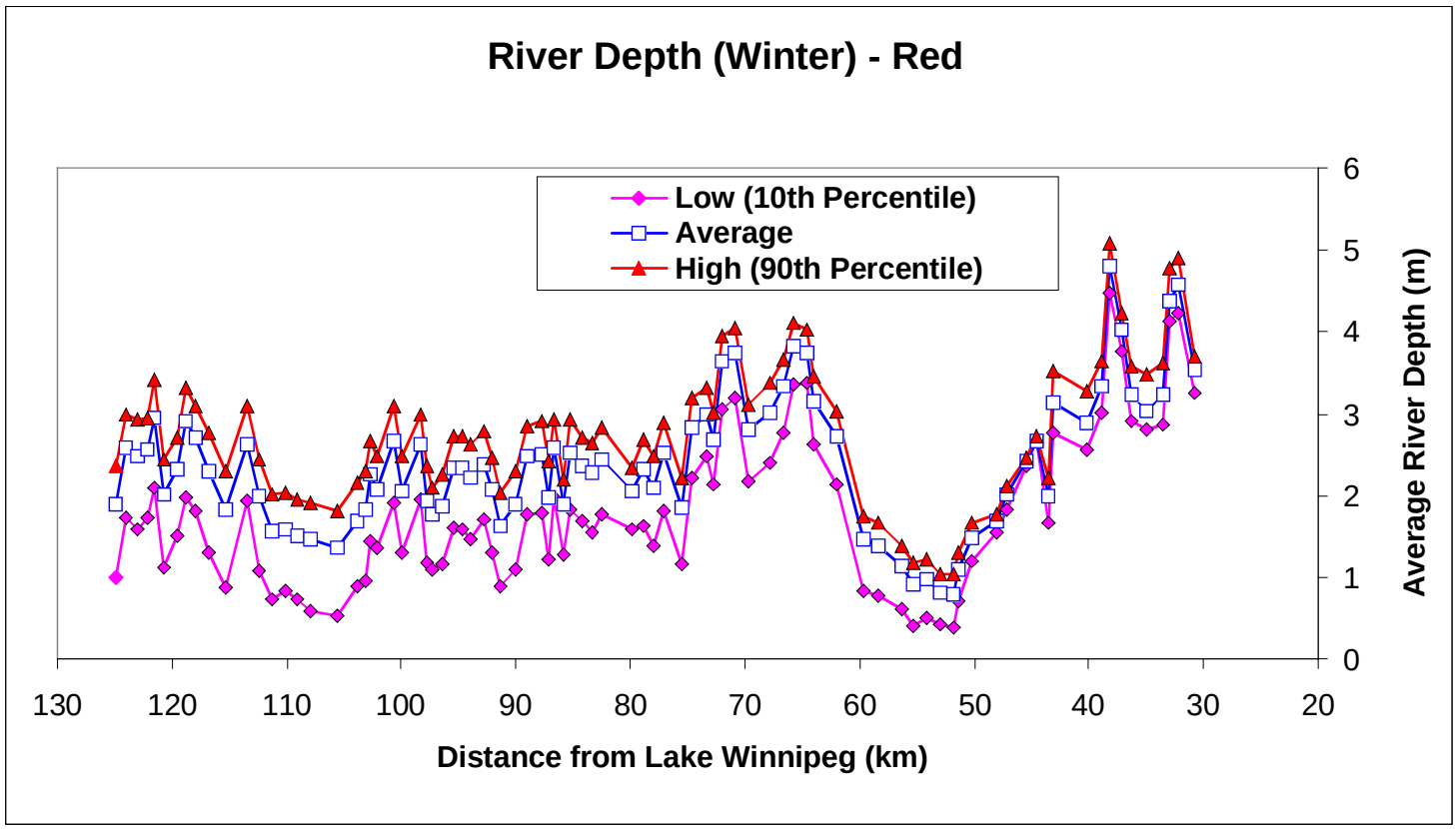
and 1-16). At the upstream part of the modelled area, the Red River is approximately 120 m wide, increasing to 150 m in width downstream of the Forks then to as much as 250 m wide in the Lister Rapids area.

During the summer months, the depth in the Red River varies with flow (Figure 1-15). The model showed the average depth in the Red River throughout the study area varies between 2 and 5 m, dependant on the location and flow in the river. Within the centre of the City of Winnipeg, the Lockport Dam maintains a depth of about 5 m for all flows between the 10th and 90th percentiles. Upstream of The Forks, the depth can vary from as little as 3 to 4 m during low and average flows to as much as 5 m during high flow conditions. Downstream of the City of Winnipeg, the depths are influenced by Lister Rapids and the operation of the Lockport Dam. The average depth downstream can decrease to approximately 2 m during high flows in the summer. When the flows are very high, the gradient between Winnipeg and Lockport is steep, causing a shallow river in the area from Lister Rapids to the Lockport Dam. In low and average flow conditions, the dam maintains high water levels in the range of 4 to 5 m at the Lockport Dam. This is done to maintain high water levels within the City of Winnipeg. During lower flows, when the dam is in operation, the depth at Lister Rapids can increase to 3 m. In winter, the widths of the Red River are similar to those in the summer, and the depth decreases significantly (Figure 1-16). Thus, the Red River is shallow in winter, especially in the Lister Rapids Region.

The width of the Assiniboine River decreases from approximately 150 m in the Headingley reaches to at little as 50 m in downtown Winnipeg. The width of the river remains fairly constant between the low and high flow conditions and in winter and summer (Figures 1-17 and 1-18). The reaches near Headingley have a tendency to become wider during high flows and can increase in some areas from 100 m during low flows to 150 m wide during high flows.

The depths within the Assiniboine River vary over most of the reaches with depths ranging from as low as 0.5 m during low flows to as high as 1.5 m during high flows for most of the reaches between Headingley and Omands Creek. Downstream of Omands Creek the depth increases as much as 3 m due to the influence of the backwater from the Red River. Depths during low and average flows are almost identical in the lower reaches of the Assiniboine River and during high flows the increase in depth is less than 0.5 m. In winter, the depth of the lower Assiniboine in downtown Winnipeg decreases due to the removal of the Lockport Dam. The depth is generally in the range of 1 to 1.5 m in winter.





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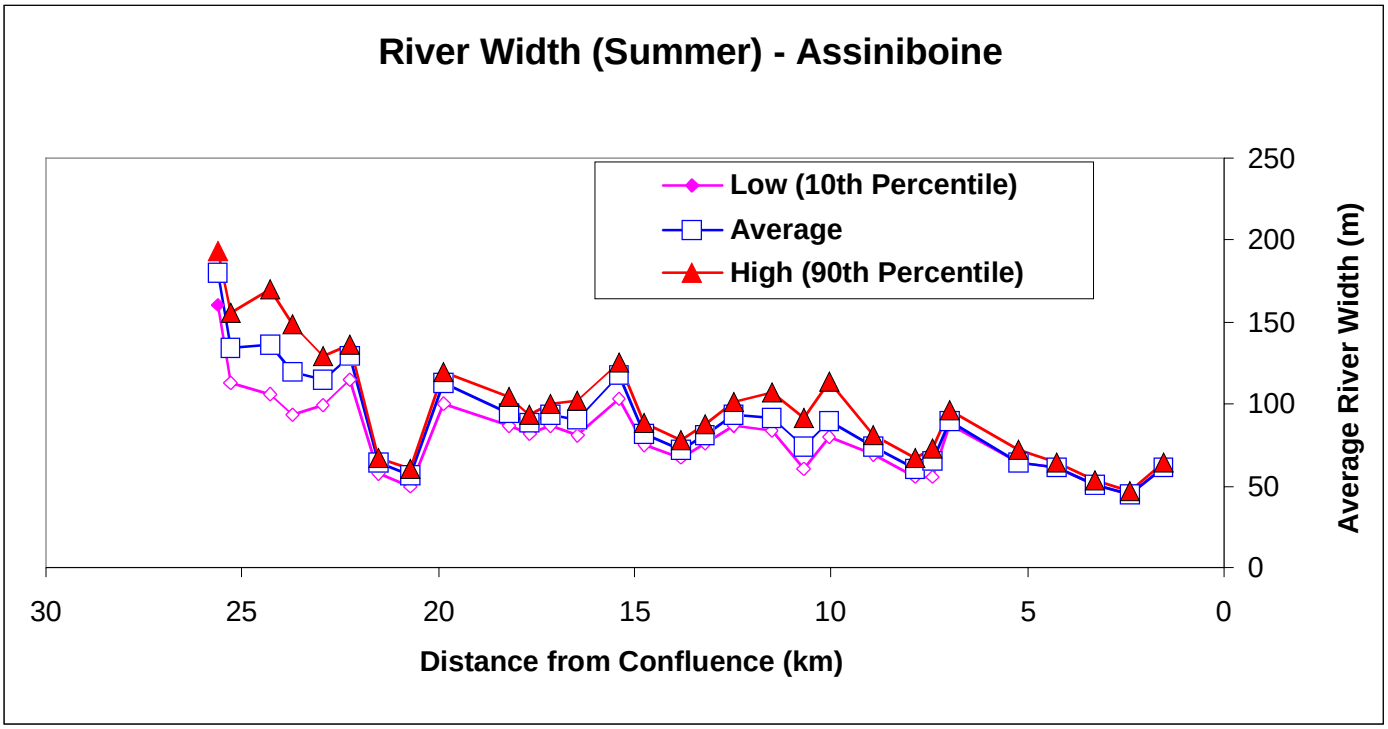
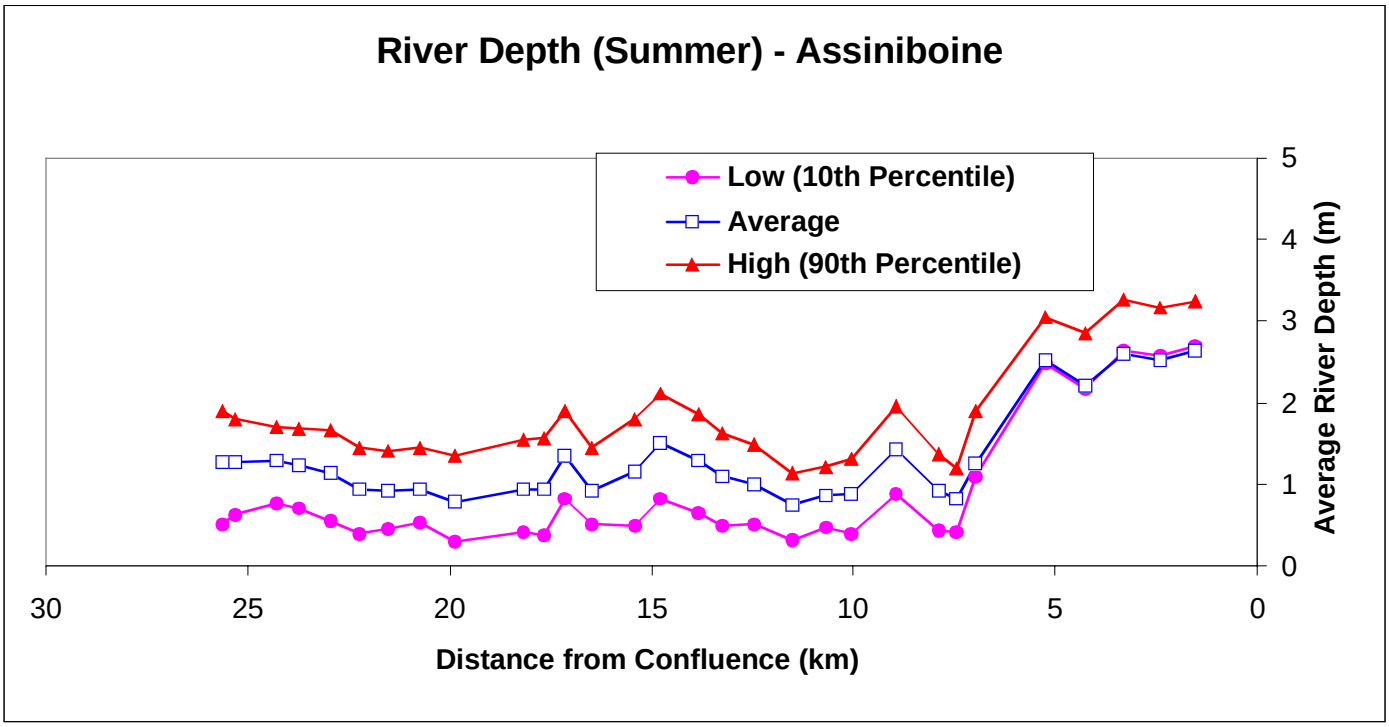
Range of Red River Width and Depth in Winter
Figure 1-16





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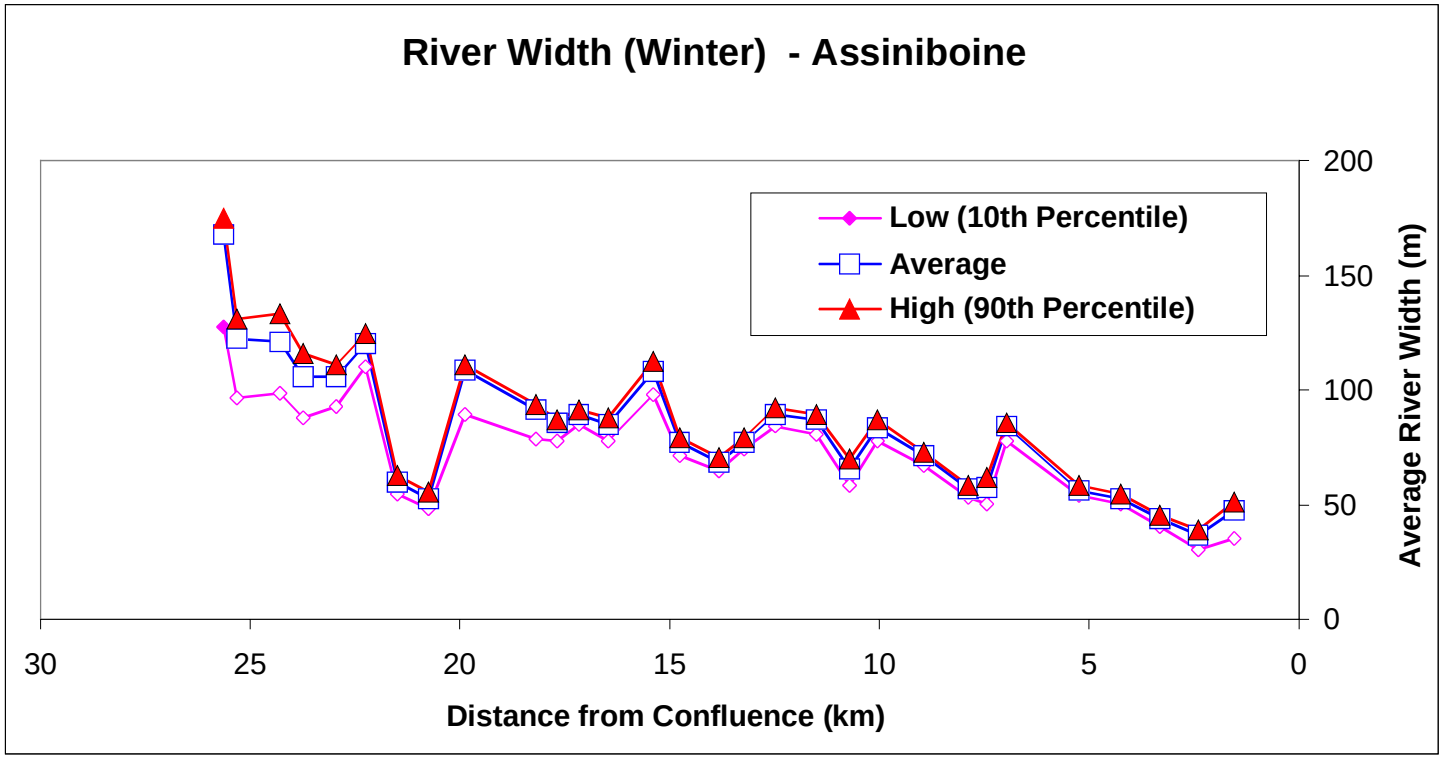
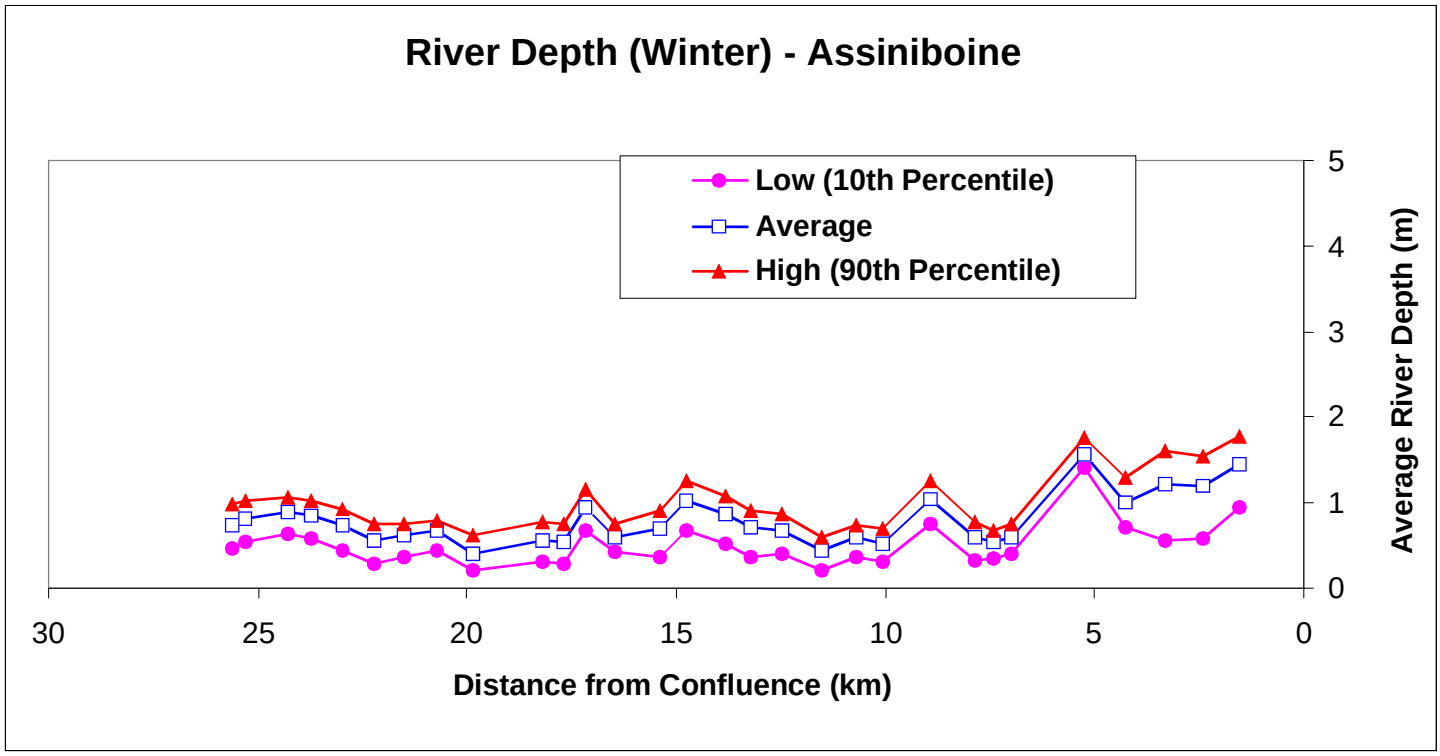
Range of Assiniboine River Width and Depth in Summer
Figure 1-17





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Range of Assiniboine River Width and Depth in Winter
Figure 1-18

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Wardrop Engineering Inc./Tetr*ES* Consultants Inc. 1991. The Red and Assiniboine Surface Water Quality Objectives. Report to City of Winnipeg Waterworks, Waste and Disposal Department. September 1991.

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Water Pollution Control Centres

APPENDIX 8B

General Overview of Study Area

Surface Water Quality

1.0 INTRODUCTION

The objective of this section is to demonstrate the current Red River water conditions. The selected key parameters are divided into four categories as follows:

1. Physical and chemical
2. Nutrients
3. Metals
4. Herbicides/pesticides

The monitoring stations near St. Norbert and Selkirk have been selected for a representative of upstream and downstream river conditions. The sampling at these locations was done on a monthly basis and data was analyzed and stored electronically on spreadsheets.

2.0 DATA SOURCES

The 1970 to 2004 water-quality data presented in this section were obtained from Manitoba Conservation.

2.1 METHODS

In this study, the data from different water-quality database was complied into a single database for efficient analysis. The monthly medians and averages for each parameter were calculated based on the long-term record from 1970 to 2004, as well as the values of statistical analyses, such as absolute maximum, absolute minimum, 90^{th} percentile and 10^{th} percentile.

The selected key parameters presented in physical and chemical section are pH, temperature, turbidity and total suspended solids, as well as ammonia, nitrite-nitrate, total nitrogen, total phosphorus, nitrogen and phosphorus ratio and dissolved oxygen for nutrient section.

The focus of metal and pesticide sections is on selected key parameters. All key parameters are assessed for acceptable contaminant levels based on the Manitoba Water Quality Standards, Objectives and Guidelines (MWQSOG 2002).

3.0 WATER QUALITY CONDITIONS

3.1 PHYSICAL AND CHEMICAL PARAMETERS

Figures 1 through 5 demonstrate the physical and chemical conditions of upstream and downstream Red River conditions (see detailed information in Attachment A). A summary of monthly variation for some parameters are discussed as follow:

- **Temperatures** in the Red River vary significantly over the year from a low of 0°C to a high of 30°C in August. No significant differences between upstream and downstream of the City of Winnipeg temperatures are observed.
- **pHs** within the Red River vary from a low of 7.0 to 7.2 in February and April to as high as 8.9 to 9.1 from August through November. pH values are also slightly different between upstream and downstream with lower pH values upstream of the City of Winnipeg and the values rising slightly downstream towards Selkirk. This is caused by a number of factors including the influence of WPCC discharges ad combined sewer overflows and land drainage.
- **Turbidity and Total Suspended Solids** show a seasonal trend which indicates higher concentrations occurring during spring freshet and gradually decreasing towards the end of summer months.

3.2 NUTRIENTS

Seasonal upstream and downstream (of the City of Winnipeg) nutrient conditions of the Red River are demonstrate in Figures 6 through 13 (see detailed information in Attachment B). The variation of key parameters is discussed as follows:

- **Average Ammonia Concentrations** vary significantly from month to month. The lowest concentrations generally occur between spring and summer months, while the highest concentrations occur in late fall and winter. Ammonia levels also vary significantly between upstream and downstream in which the downstream concentrations are much greater than those of upstream levels. This is due to influence of WPCC discharges.
- **Total Nitrogen Concentrations** are measured in the form of total Kjeldahl nitrogen (TKN) and nitrates. Nitrates are generally higher in the winter and spring months and are reduced over the summer months. Overall average total nitrogen concentration for upstream and downstream are relatively low. No significant difference of nitrogen levels between upstream and downstream is observed.
- **Total Phosphorus Concentrations** for upstream and downstream measurements are generally fairly constant between 0.25 to 0.32 mg/L with higher concentration occurring during the spring freshet (0.41 to 0.46 mg/L). No significant difference of total phosphorus levels between upstream and downstream conditions is observed.
- **Dissolved Oxygen** varies significantly throughout the year for the upstream condition. Higher concentrations occur during late fall and early winter between 9.9 and 13.0 mg/L. Only four months of record are presented for the downstream DO condition.
- **Nitrogen/Phosphorus ratio** is an indicator used to determine nutrient conditions that may be limiting for algal growth in the river water. Overall average N:P ratios for the upstream and downstream conditions are fairly constant over the year with a range between 8.26 and 8.48, respectively. It is evident that nitrogen is a limiting factor in controlling algal growth in Red river as the values are below a 16:1 ratio. Upstream concentrations appear to have higher concentrations than those of downstream levels.

3.3 METALS

Figures 14 through 36 demonstrate levels of metal concentrations for the upstream and downstream conditions of the Red River (see detailed information in Attachment C). Overall upstream and downstream metal concentrations are relatively low and well below appropriate federal/provincial water quality guidelines except manganese. Seasonal trend of manganese indicates high concentrations in the spring months, which exceed water quality guidelines for both irrigation and aesthetic drinking water, and gradually drops towards the end of fall. This is due to spring freshet in spring.

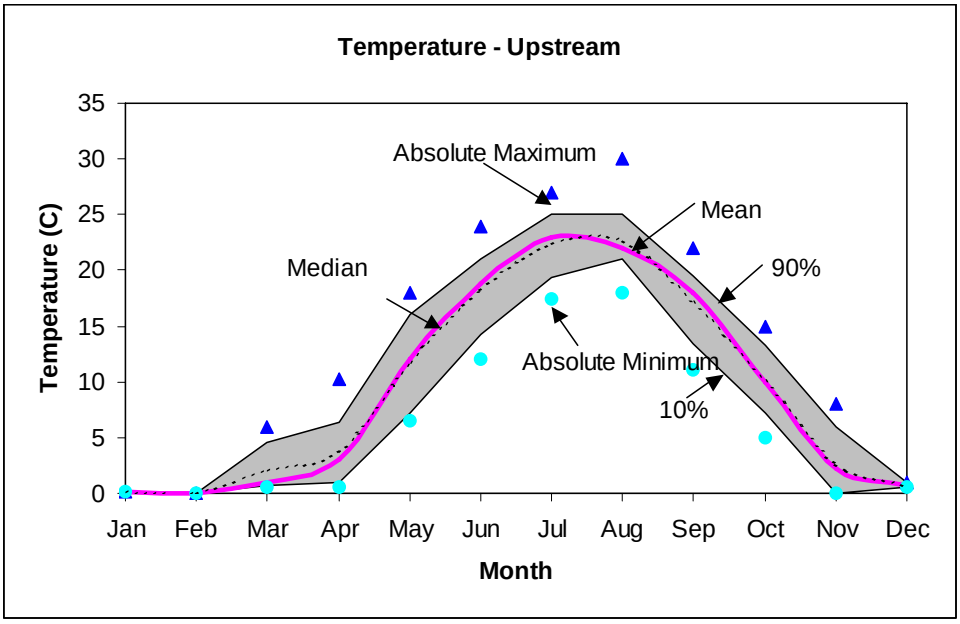
3.4 HERBICIDES/PESTICIDES

Figures 27 through 29 demonstrate herbicide and pesticide levels on the upstream and downstream of Red River (see detailed information in Attachment D). Overall upstream contaminant levels are very low in comparison with downstream levels. Peaks of downstream contaminant concentrations occur in the spring months and dramatically drop thereafter.

4.0 REFERENCES

Red/Assiniboine River. Water Quality Data – 2001 to 2005. Water Quality Management Section (2005). Water Science and Management Branch, Manitoba Water Stewardship, 160-123 Main Street, Winnipeg, Manitoba R3C 1A5.

Manitoba Conservation. 2002. Final Draft: Manitoba Water Quality Standards, Objectives, and Guidelines. Manitoba Conservation Report 2002-11.



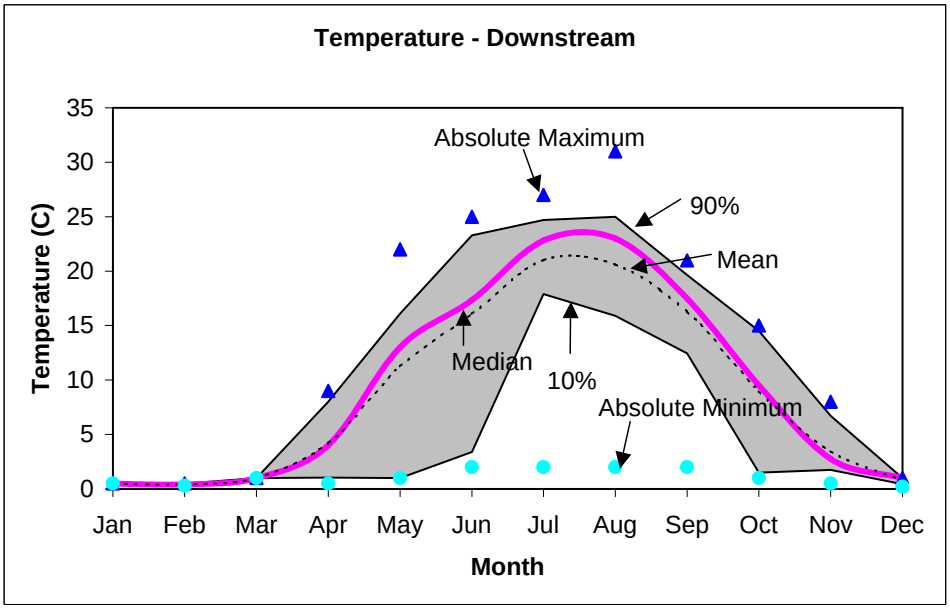


Figure 1: Seasonal Temperature on the Red River Upstream and Downstream of the City of Winnipeg

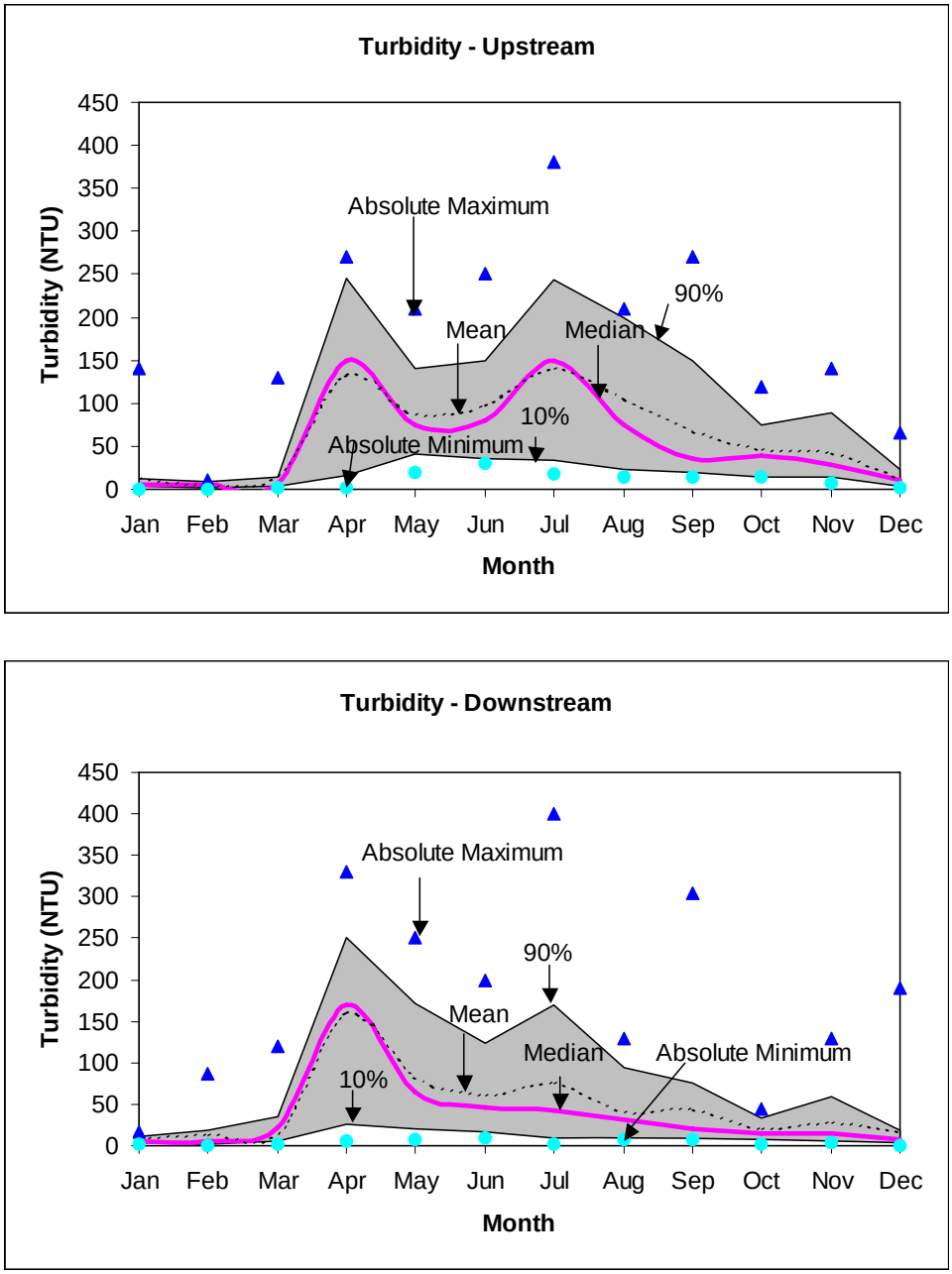


Figure 2: Seasonal Turbidity on the Red River Upstream and Downstream of the City of Winnipeg

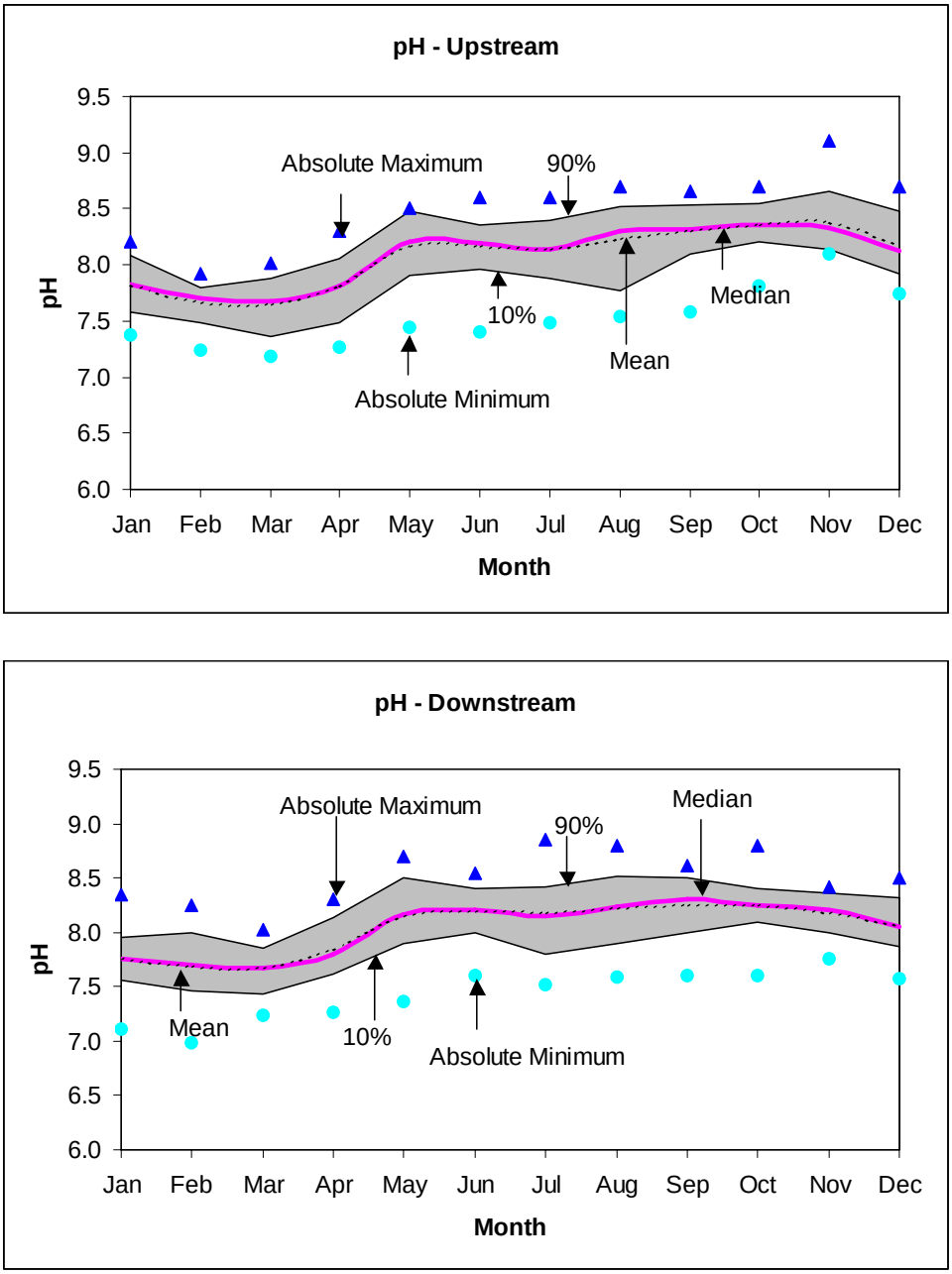


Figure 3: Seasonal pH on the Red River Upstream and Downstream of the City of Winnipeg

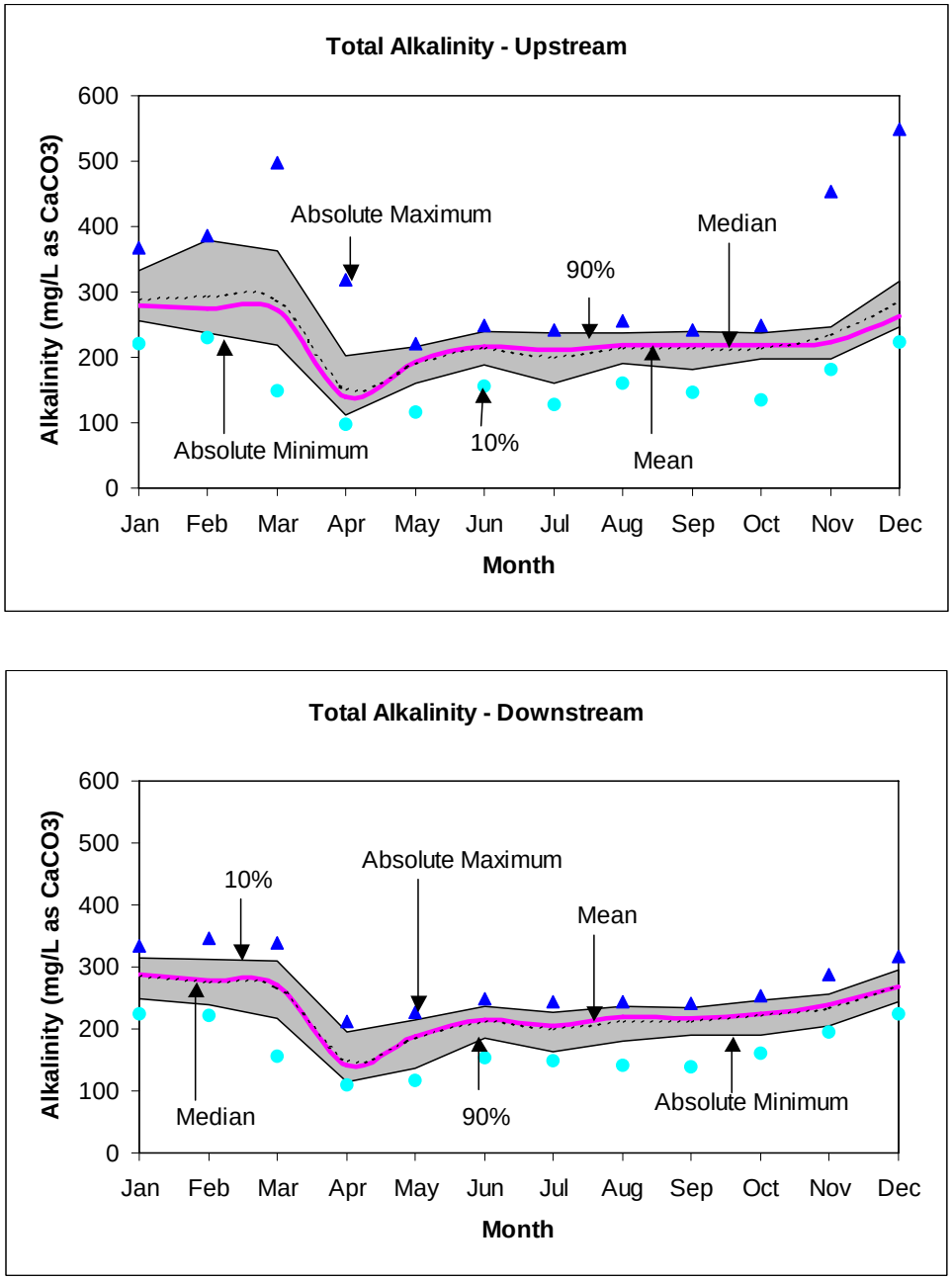


Figure 4: Seasonal Alkalinity on the Red River Upstream and Downstream of the City of Winnipeg

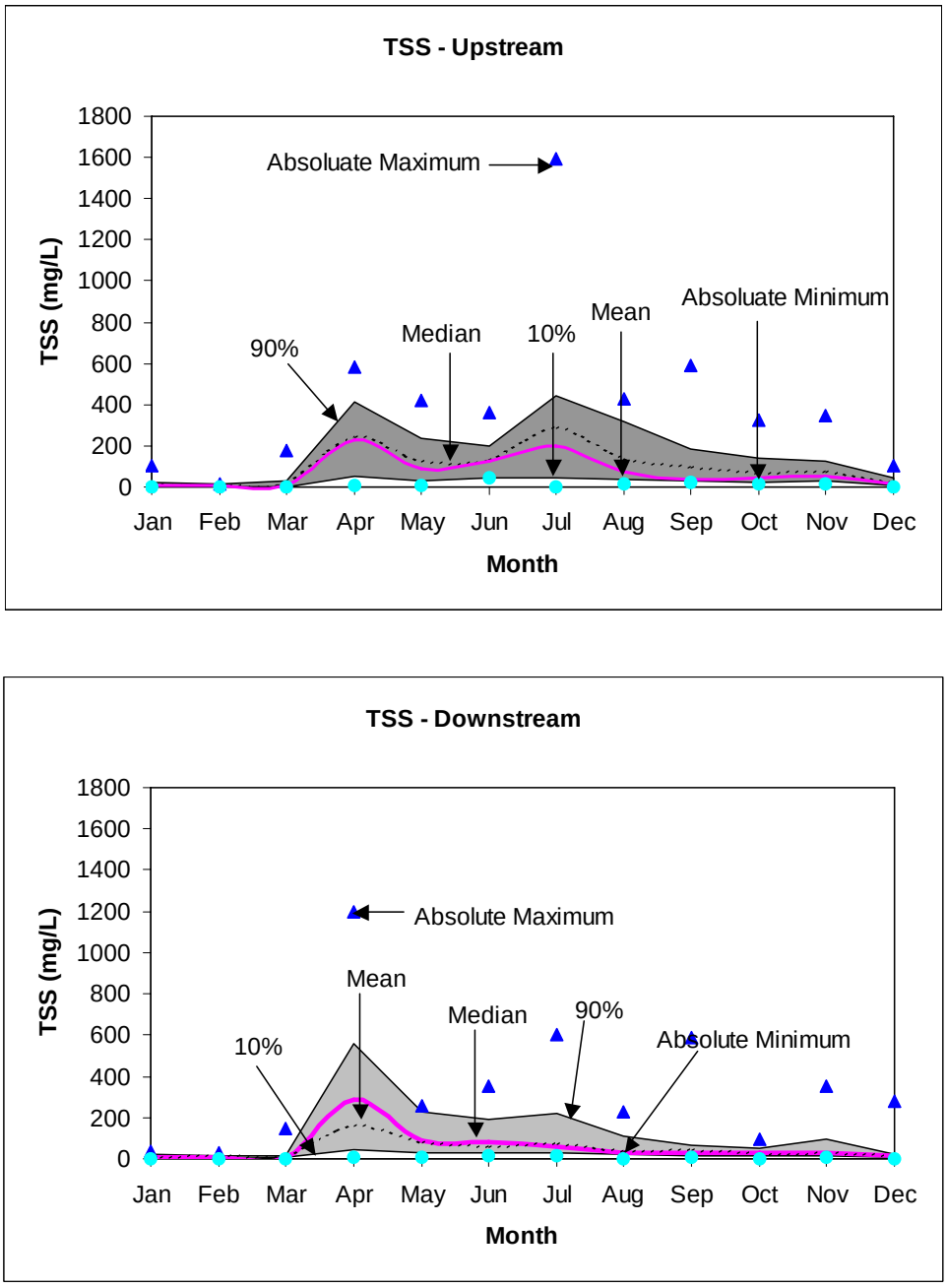


Figure 5: Seasonal TSS on the Red River Upstream and Downstream of the City of Winnipeg

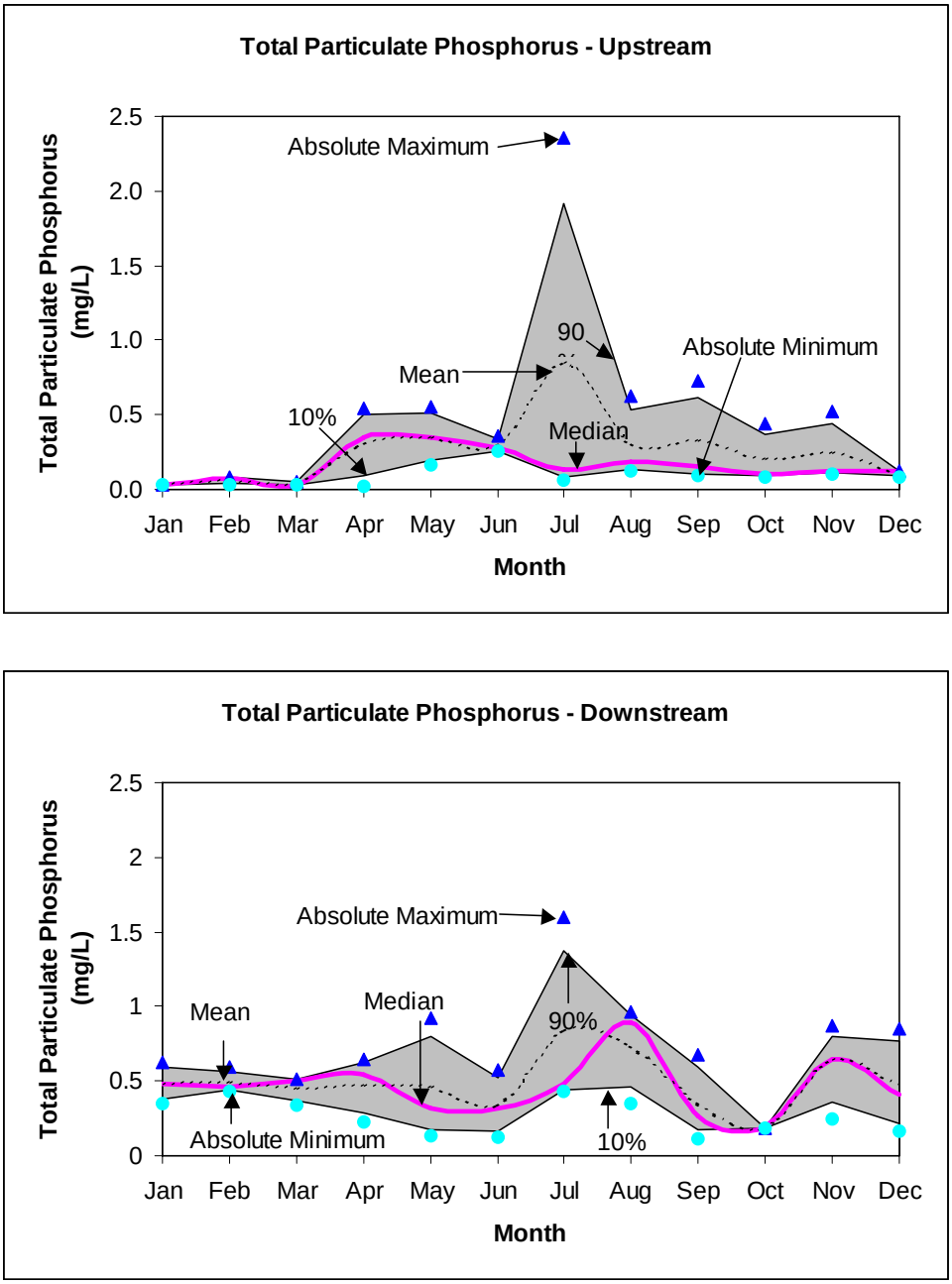
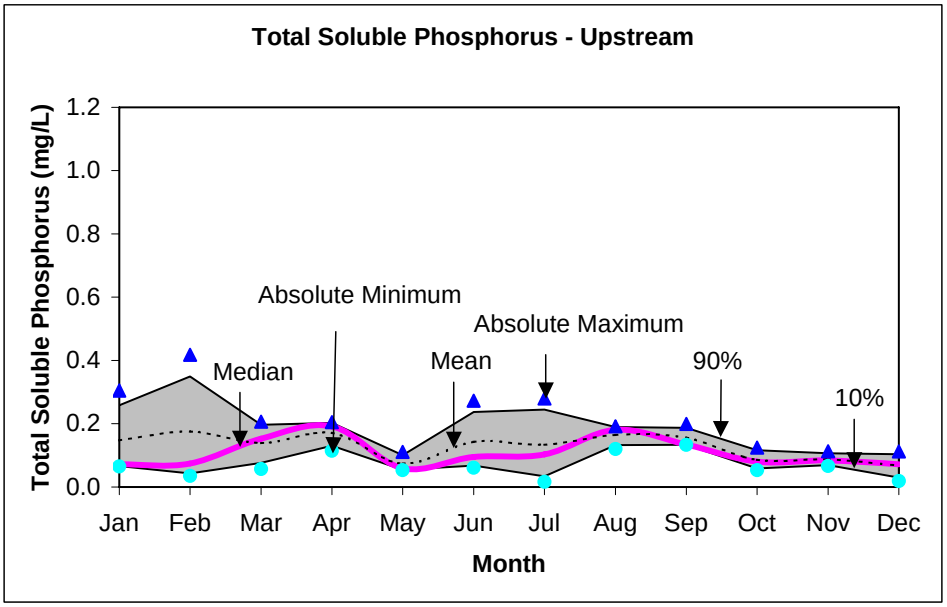


Figure 6: Seasonal Particulate Phosphorus on the Red River Upstream and Downstream of the City of Winnipeg



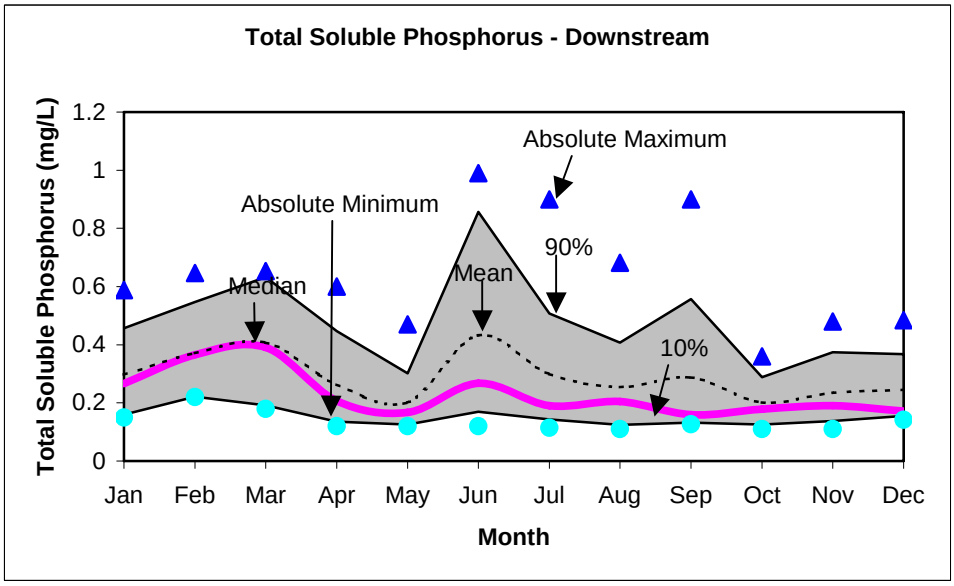
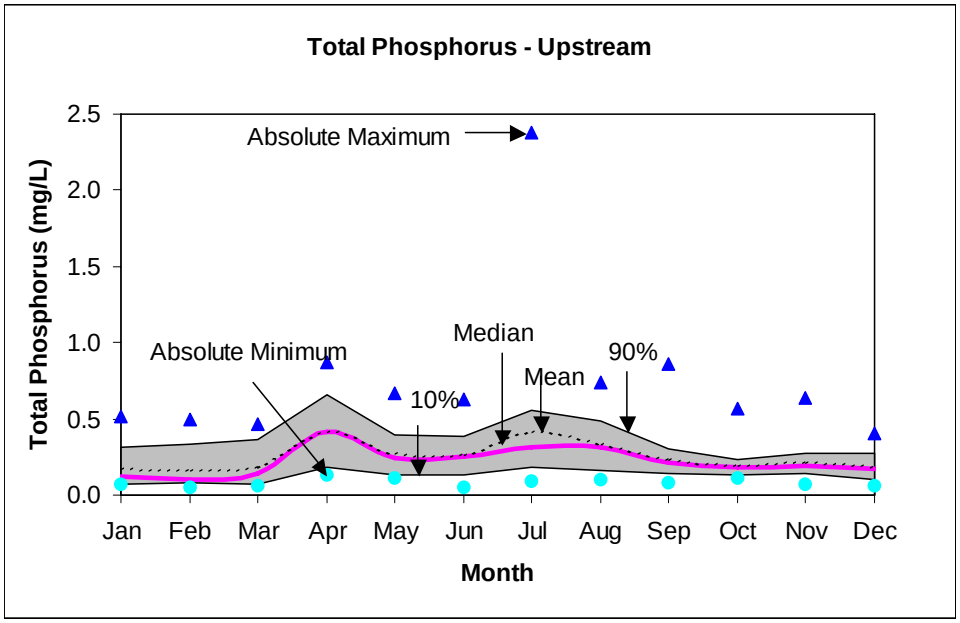


Figure 7: Seasonal Soluble Phosphorus on the Red River Upstream and Downstream of the City of Winnipeg



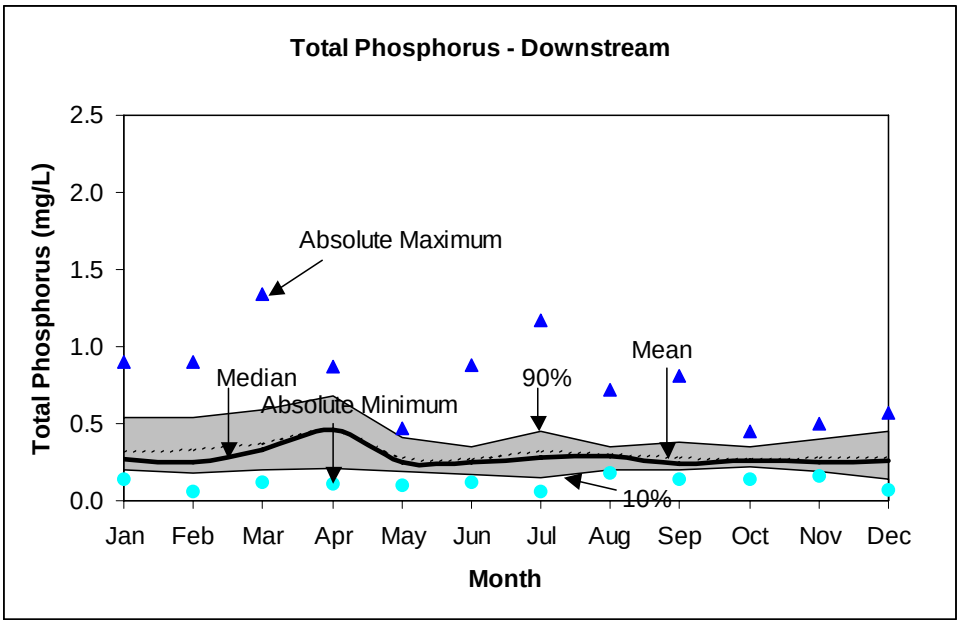


Figure 8: Seasonal Phosphorus on the Red River Upstream and Downstream of the City of Winnipeg

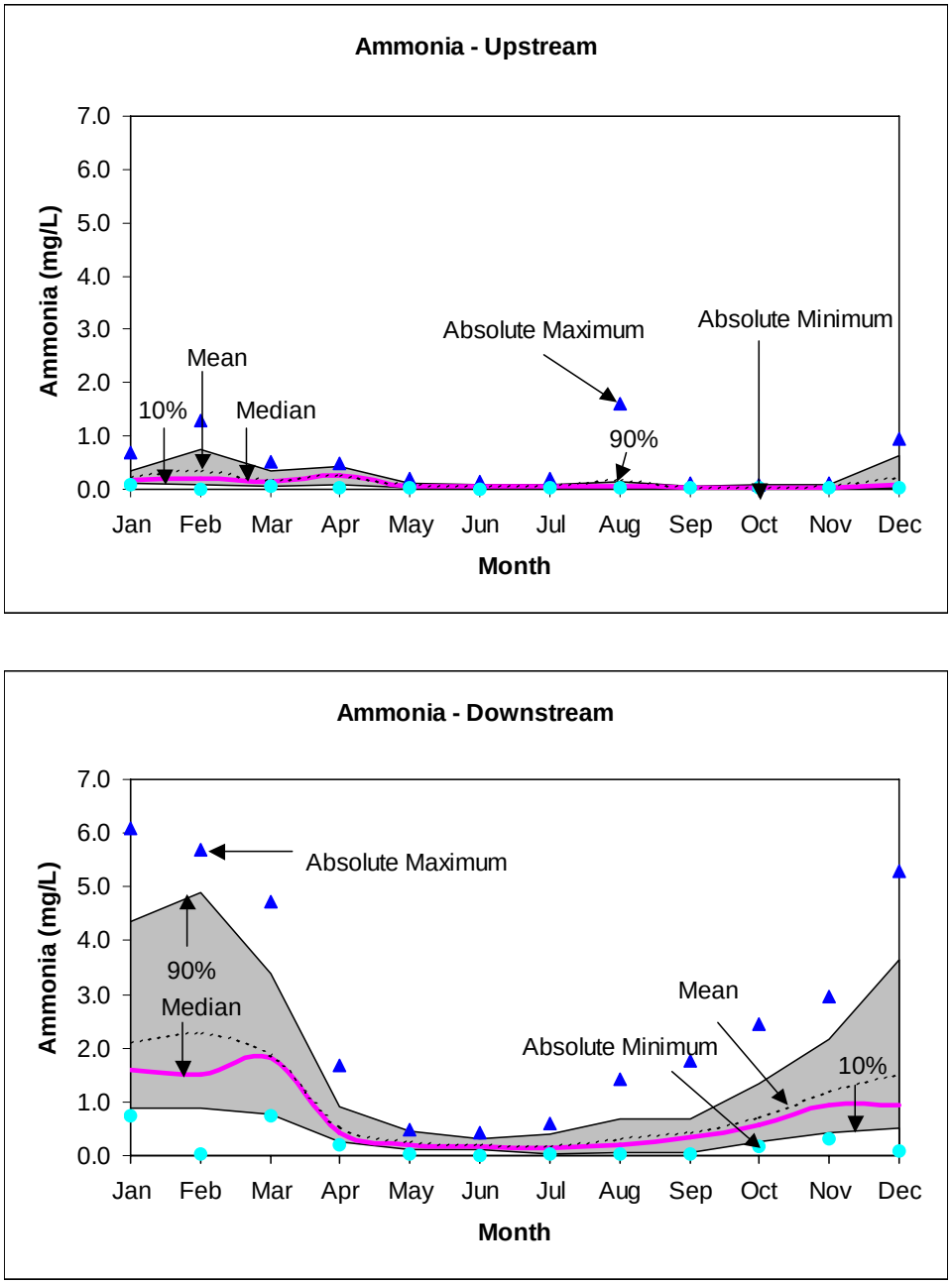
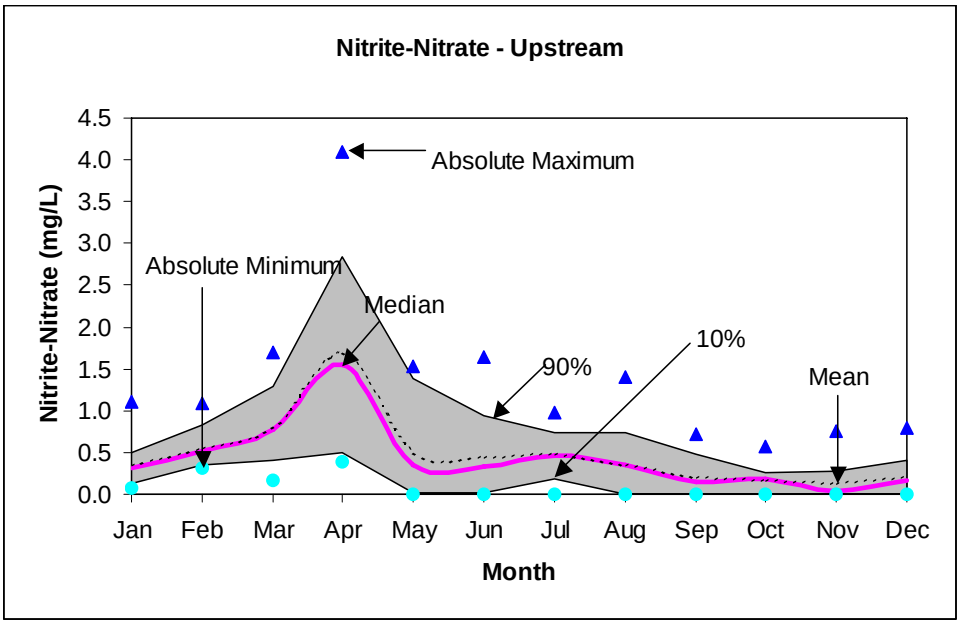


Figure 9: Seasonal Ammonia on the Red River Upstream and Downstream of the City of Winnipeg



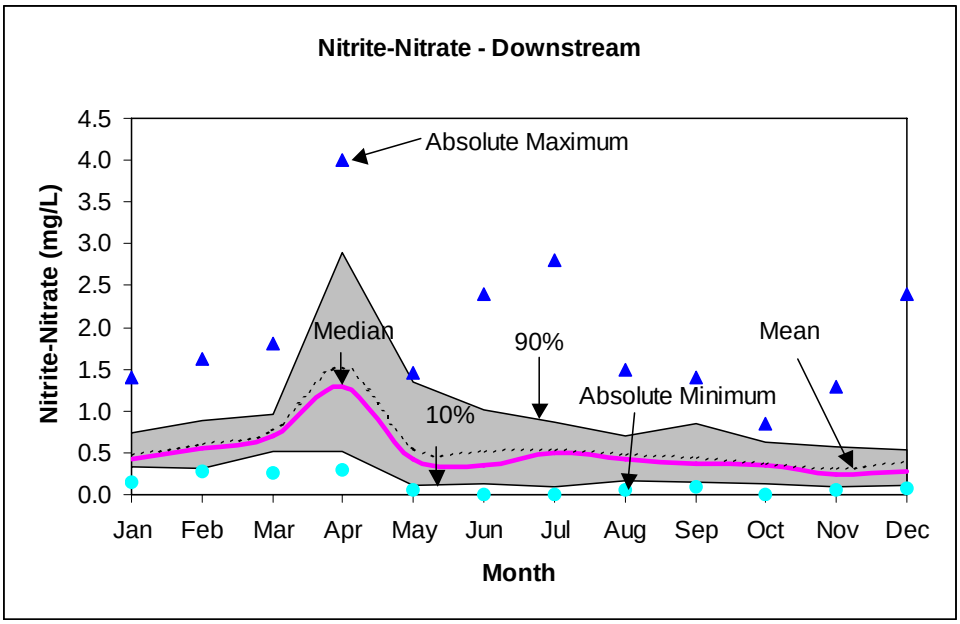
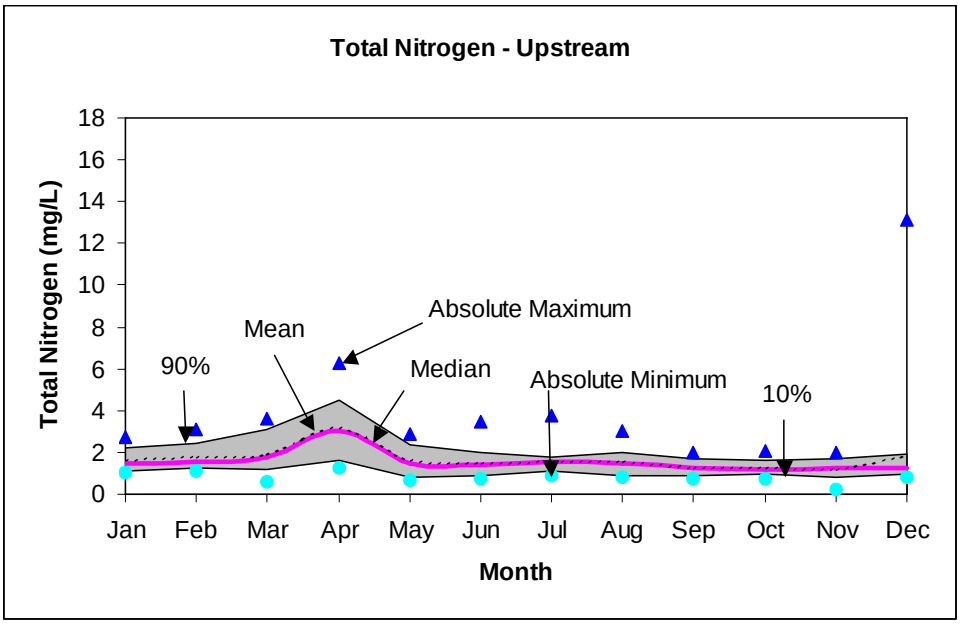


Figure 10: Seasonal Nitrite-Nitrate on the Red River Upstream and Downstream of the City of Winnipeg



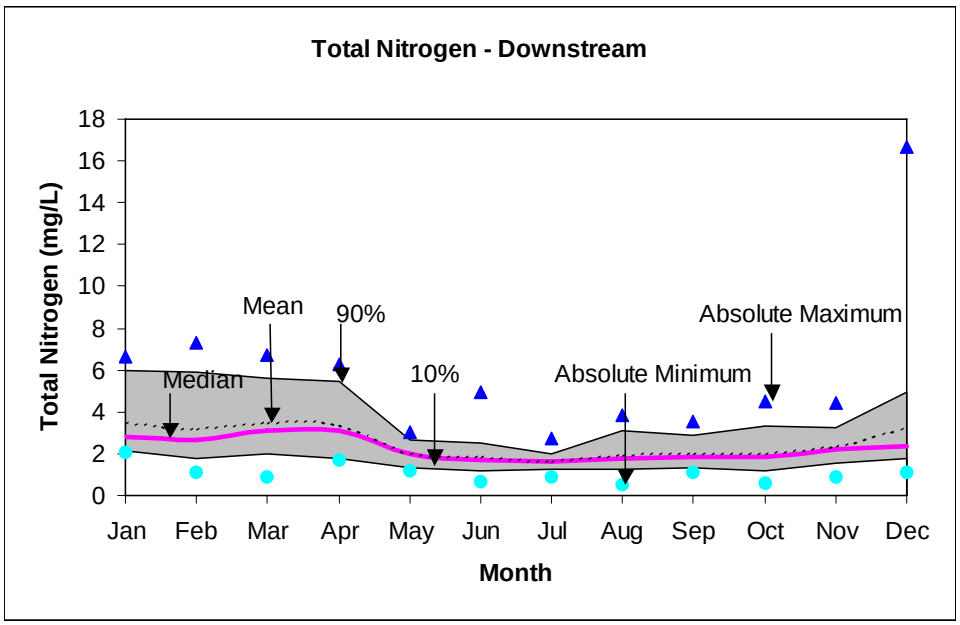
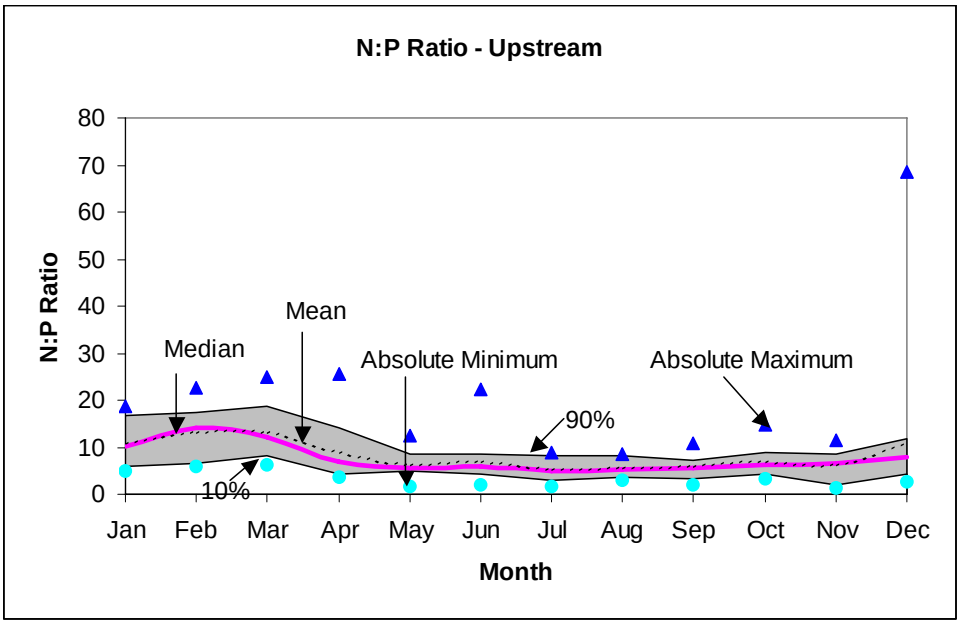


Figure 11: Seasonal Nitrogen on the Red River Upstream and Downstream of the City of Winnipeg



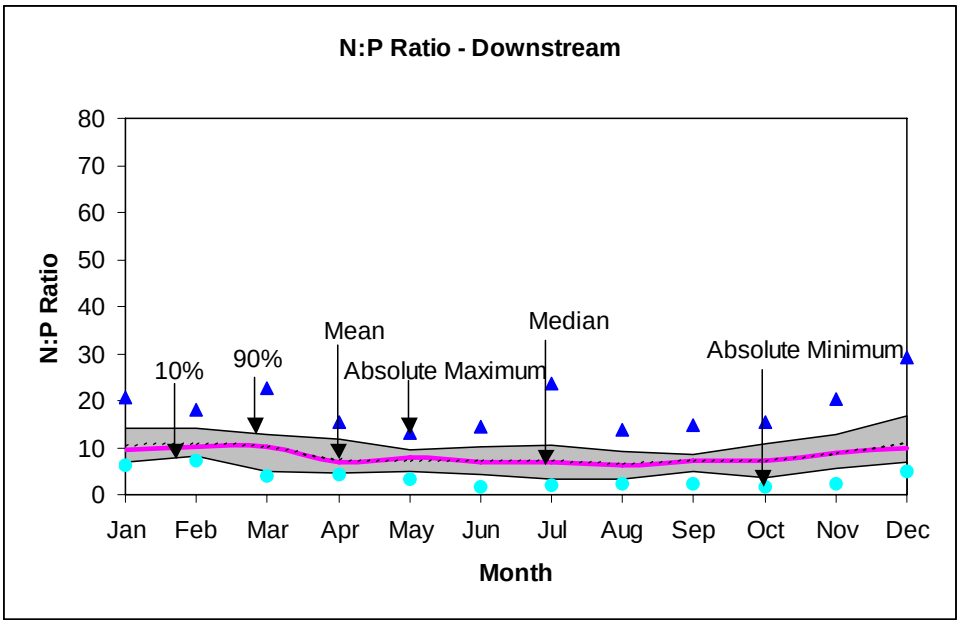


Figure 12: Seasonal N:P Ratio on the Red River Upstream and Downstream of the City of Winnipeg

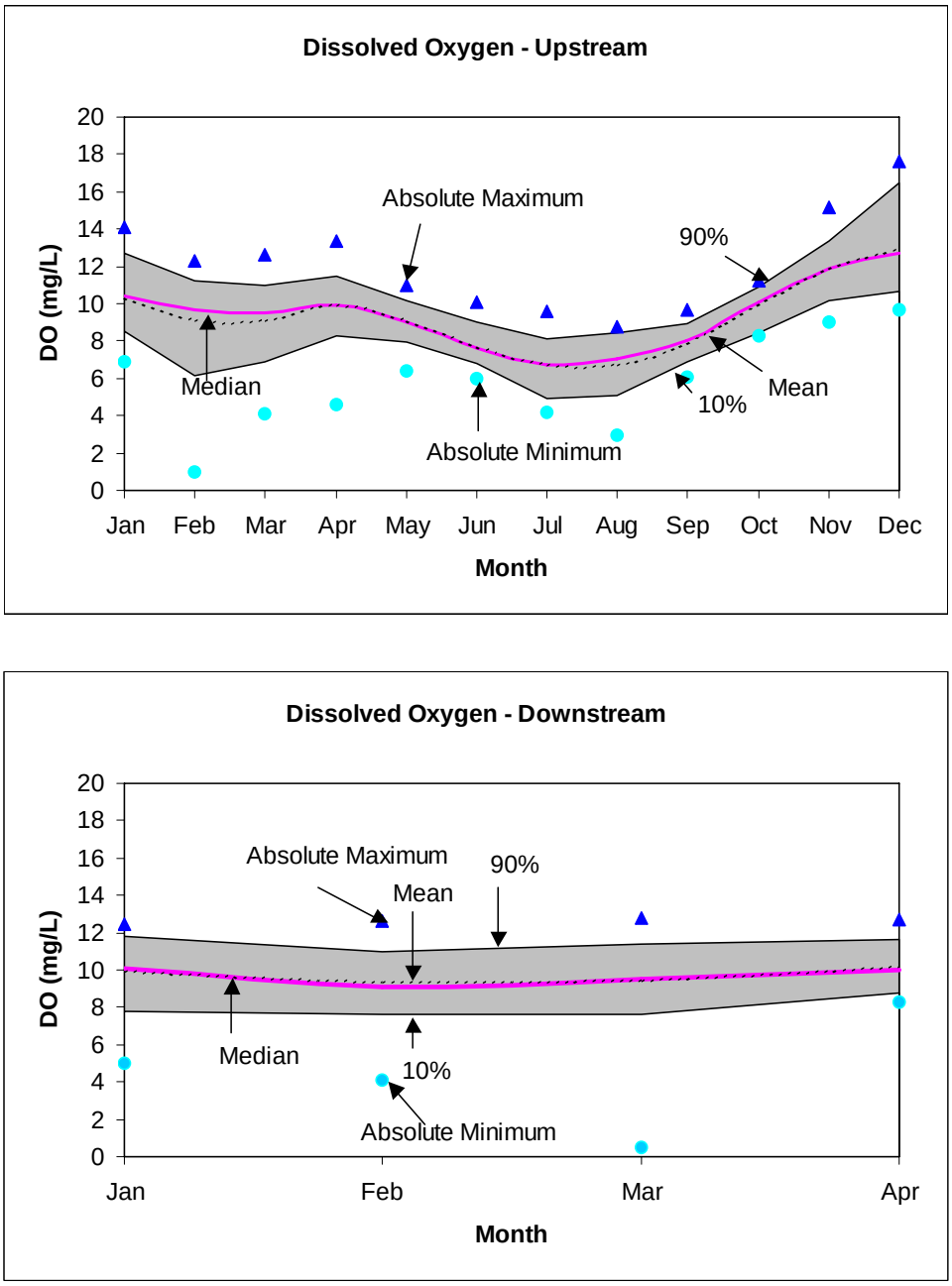


Figure 13: Seasonal Dissolved Oxygen on the Red River Upstream and Downstream of the City of Winnipeg

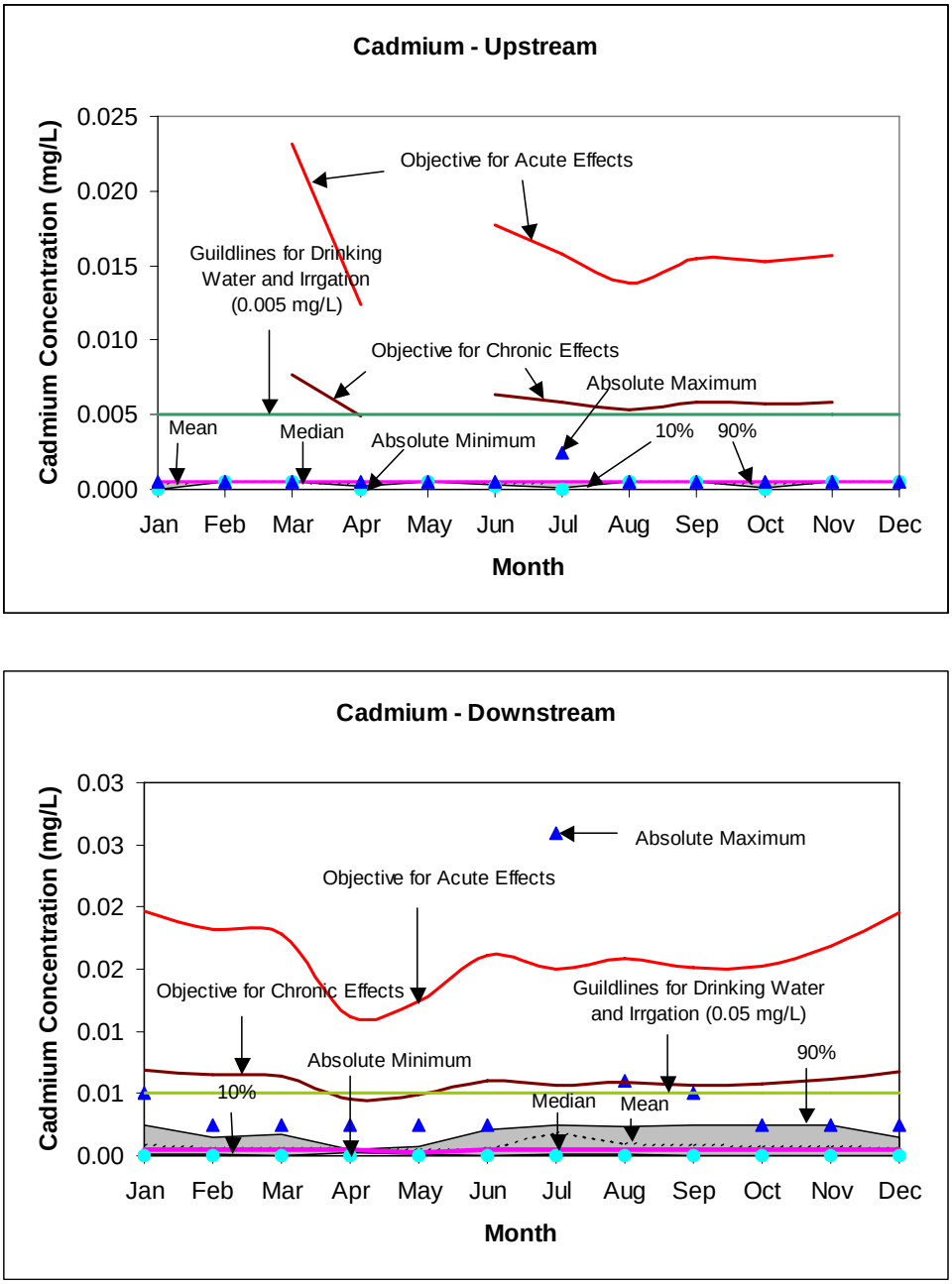


Figure 14: Seasonal Cadmium on the Red River Upstream and Downstream of the City of Winnipeg

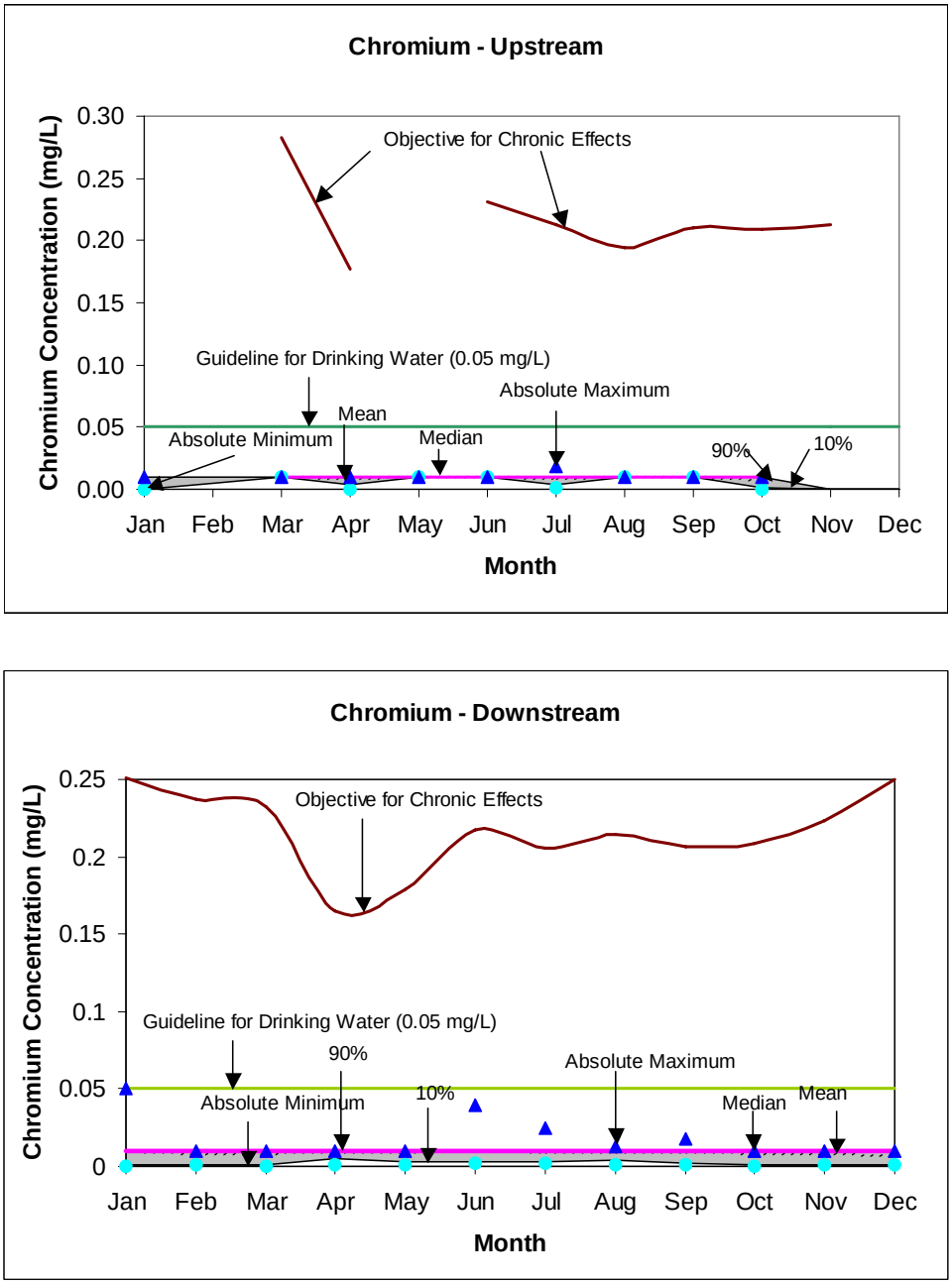


Figure 15: Seasonal Chromium on the Red River Upstream and Downstream of the City of Winnipeg

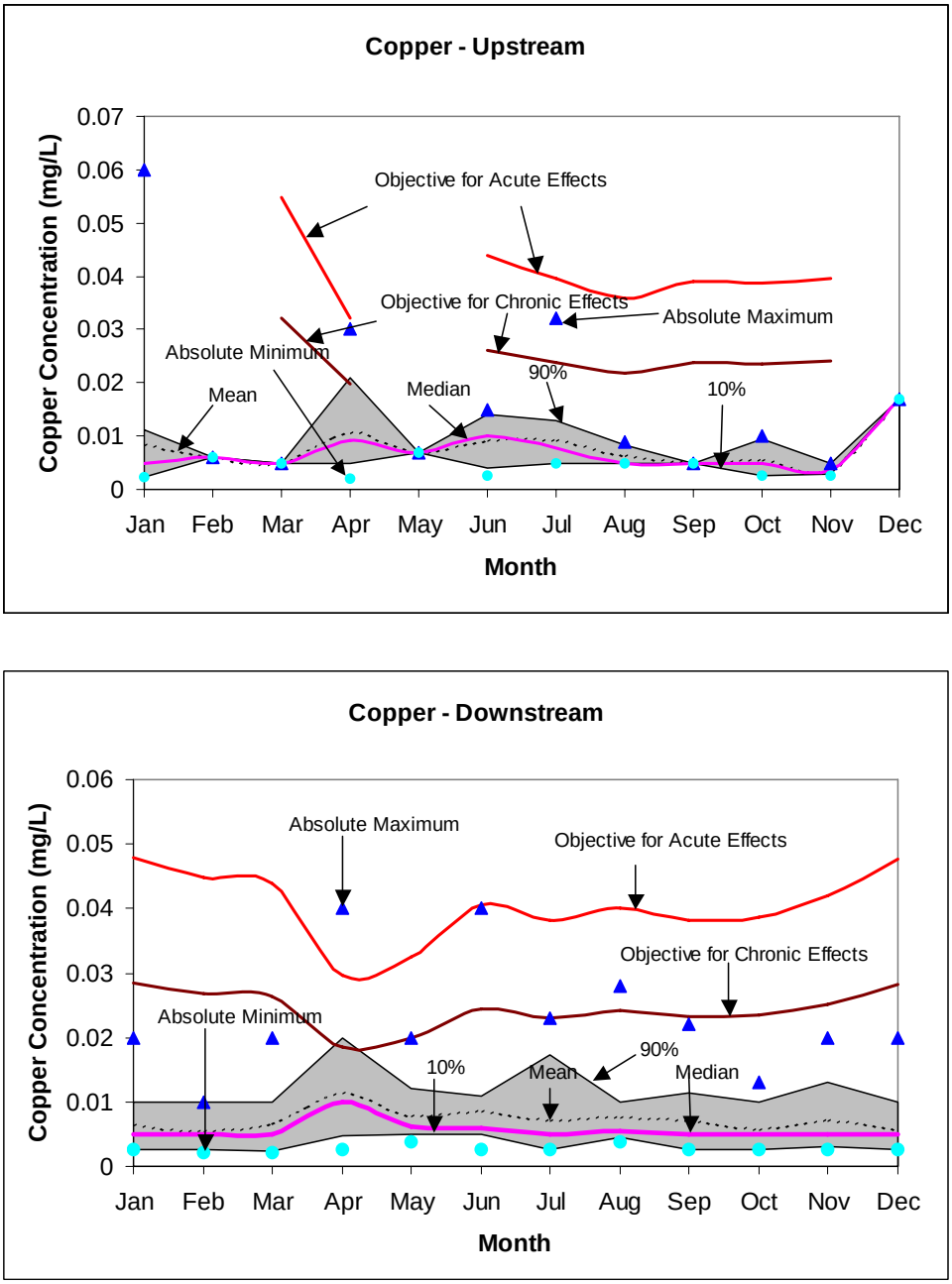


Figure 16: Seasonal Copper on the Red River Upstream and Downstream of the City of Winnipeg

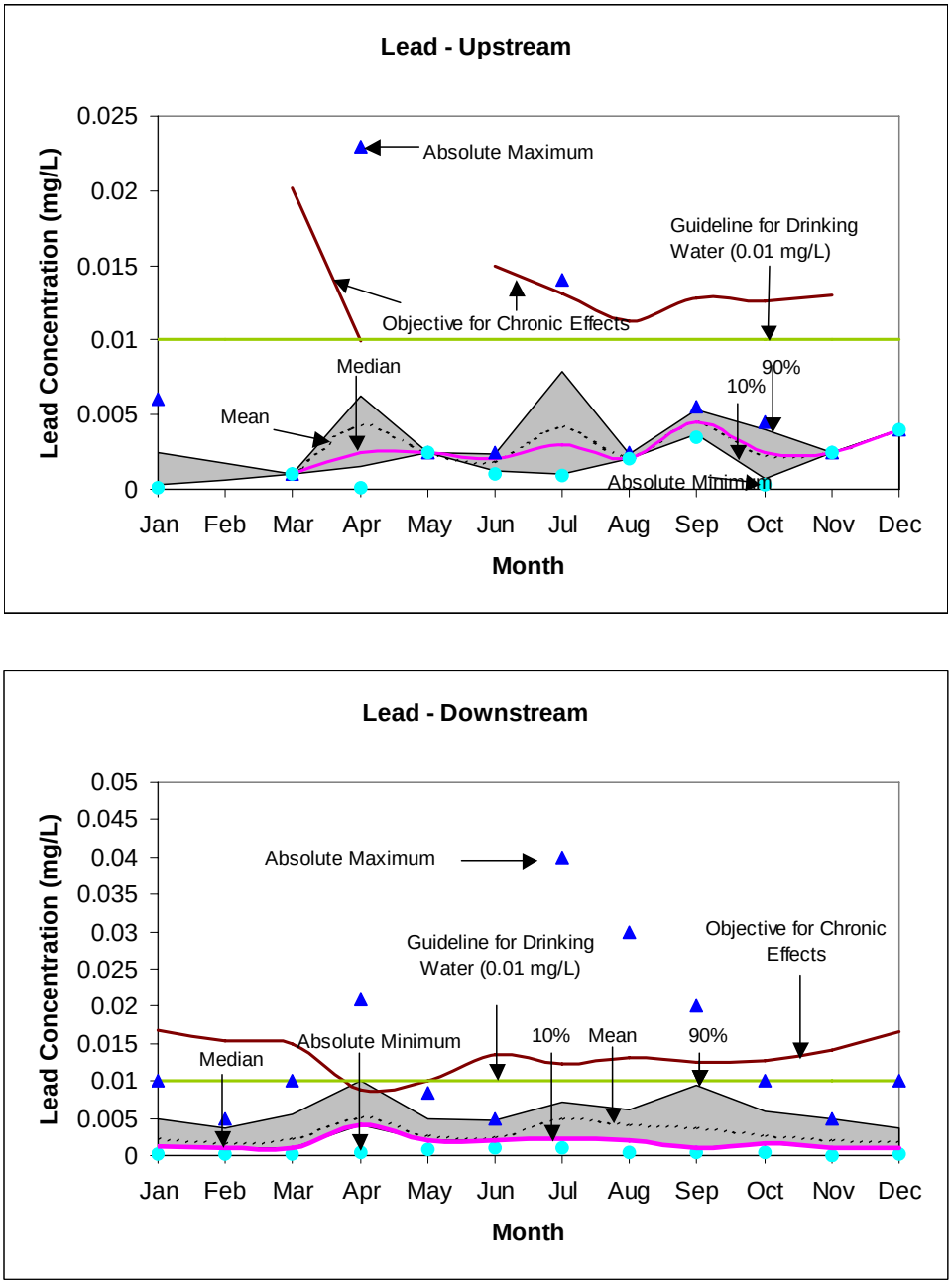
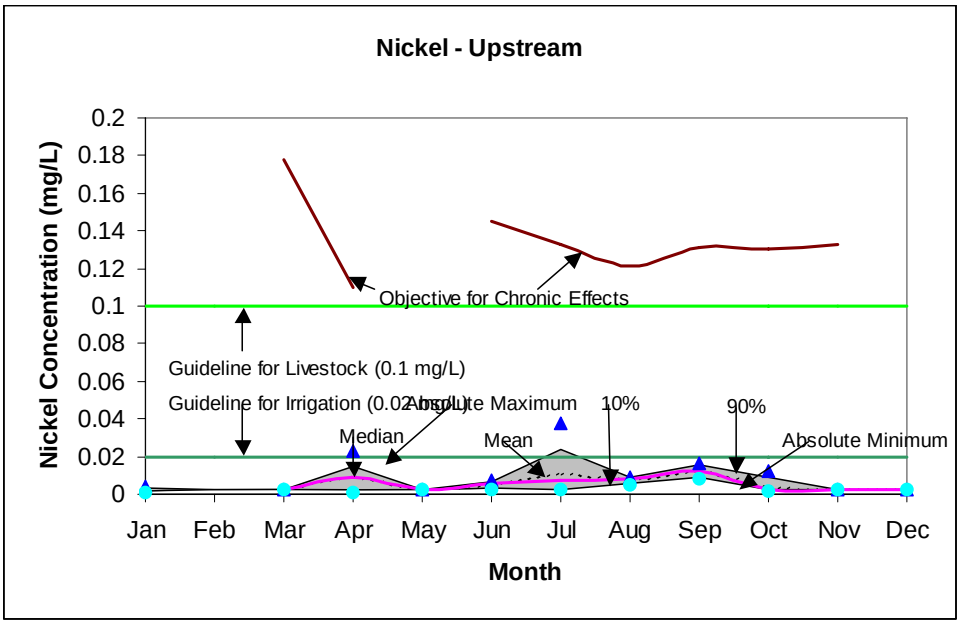


Figure 17: Seasonal Lead on the Red River Upstream and Downstream of the City of Winnipeg



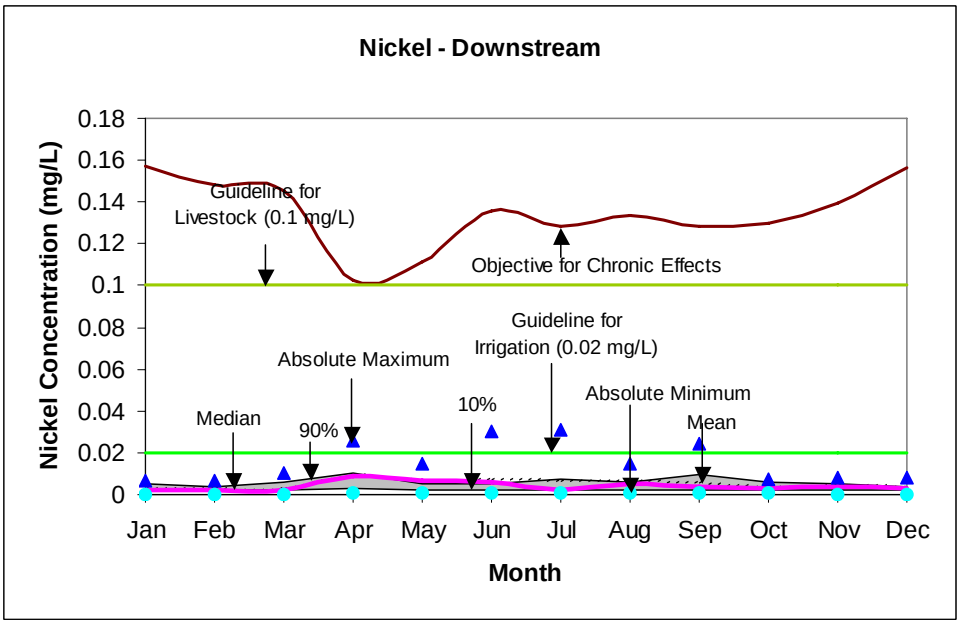


Figure 18: Seasonal Nickel on the Red River Upstream and Downstream of the City of Winnipeg

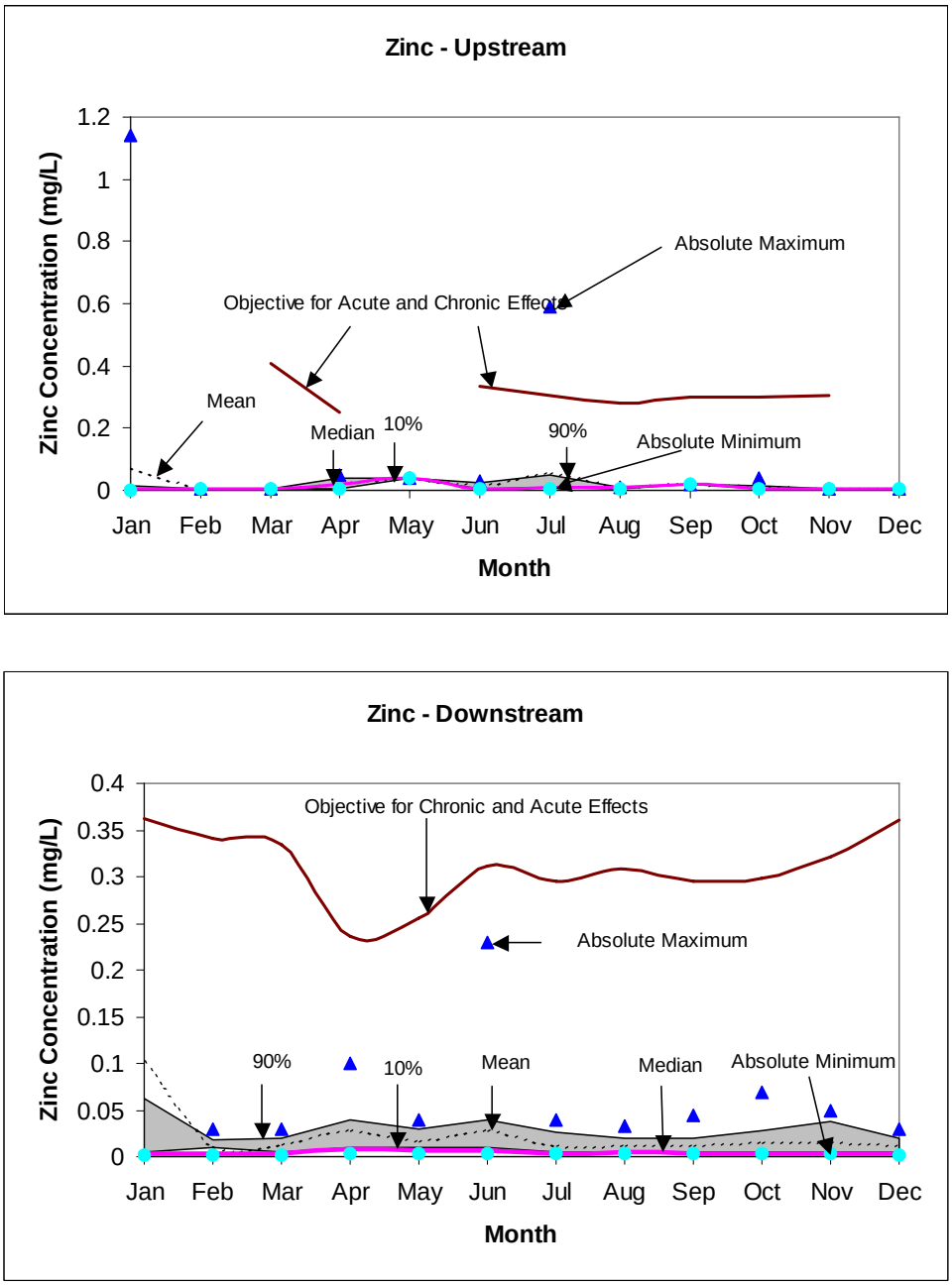
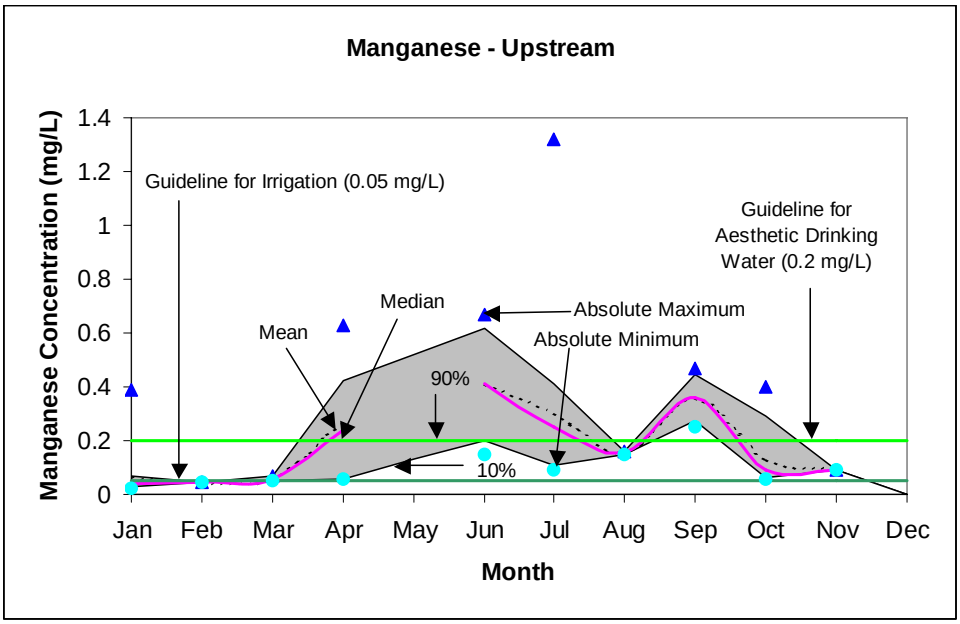


Figure 19: Seasonal Zinc on the Red River Upstream and Downstream of the City of Winnipeg



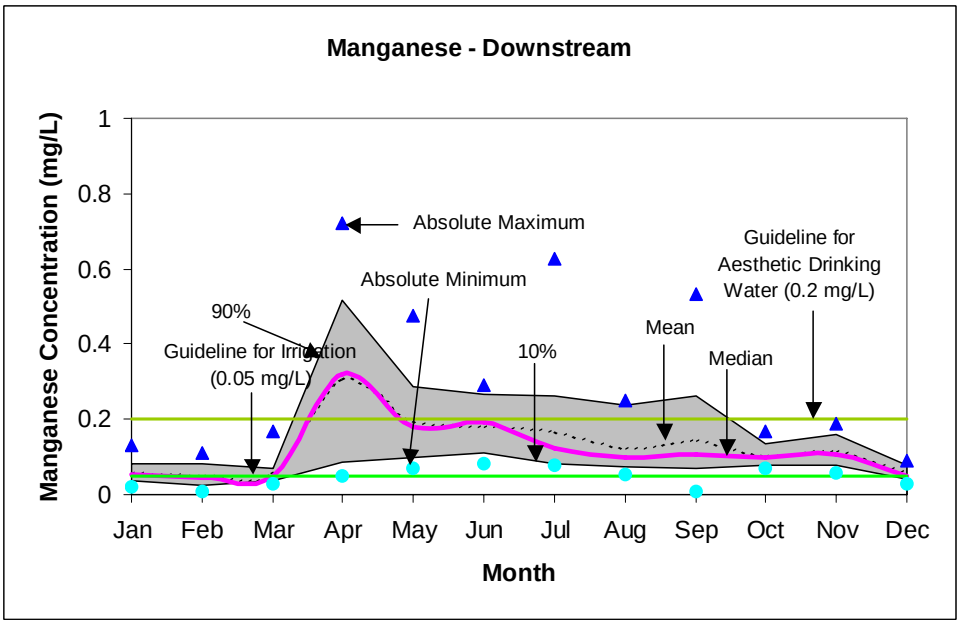
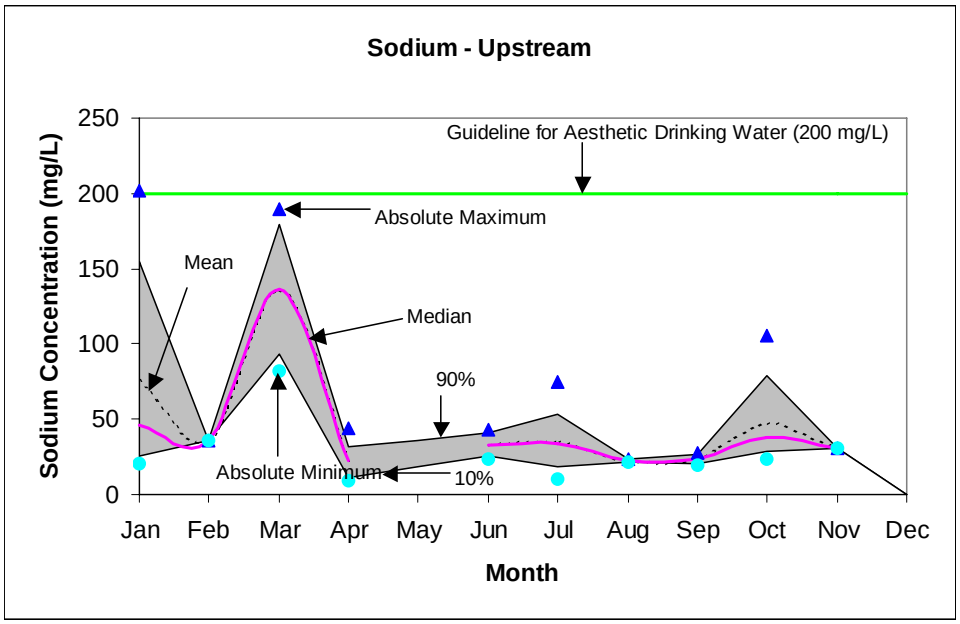


Figure 20: Seasonal Manganese on the Red River Upstream and Downstream of the City of Winnipeg



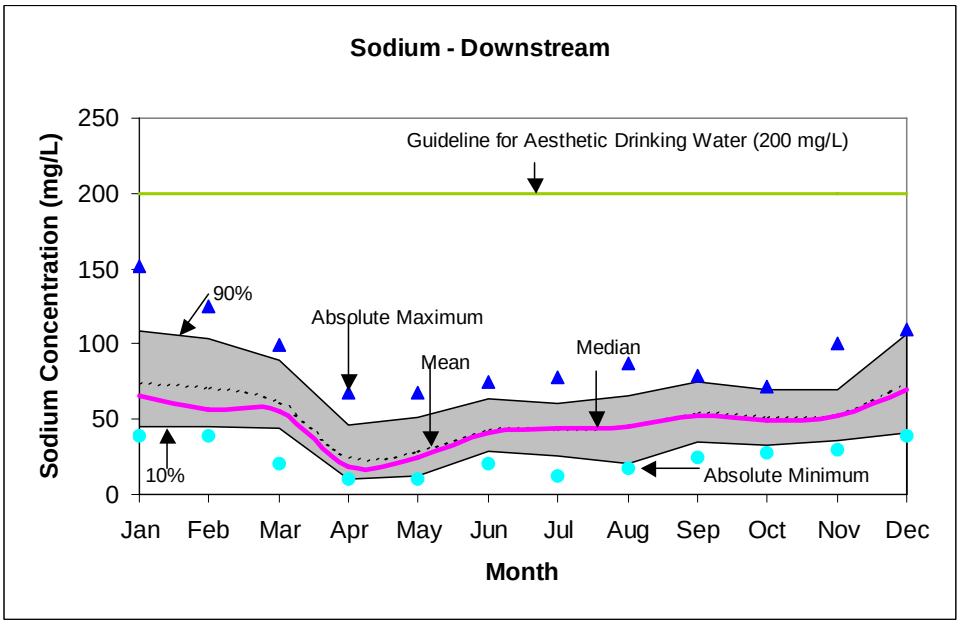


Figure 21: Seasonal Sodium on the Red River Upstream and Downstream of the City of Winnipeg

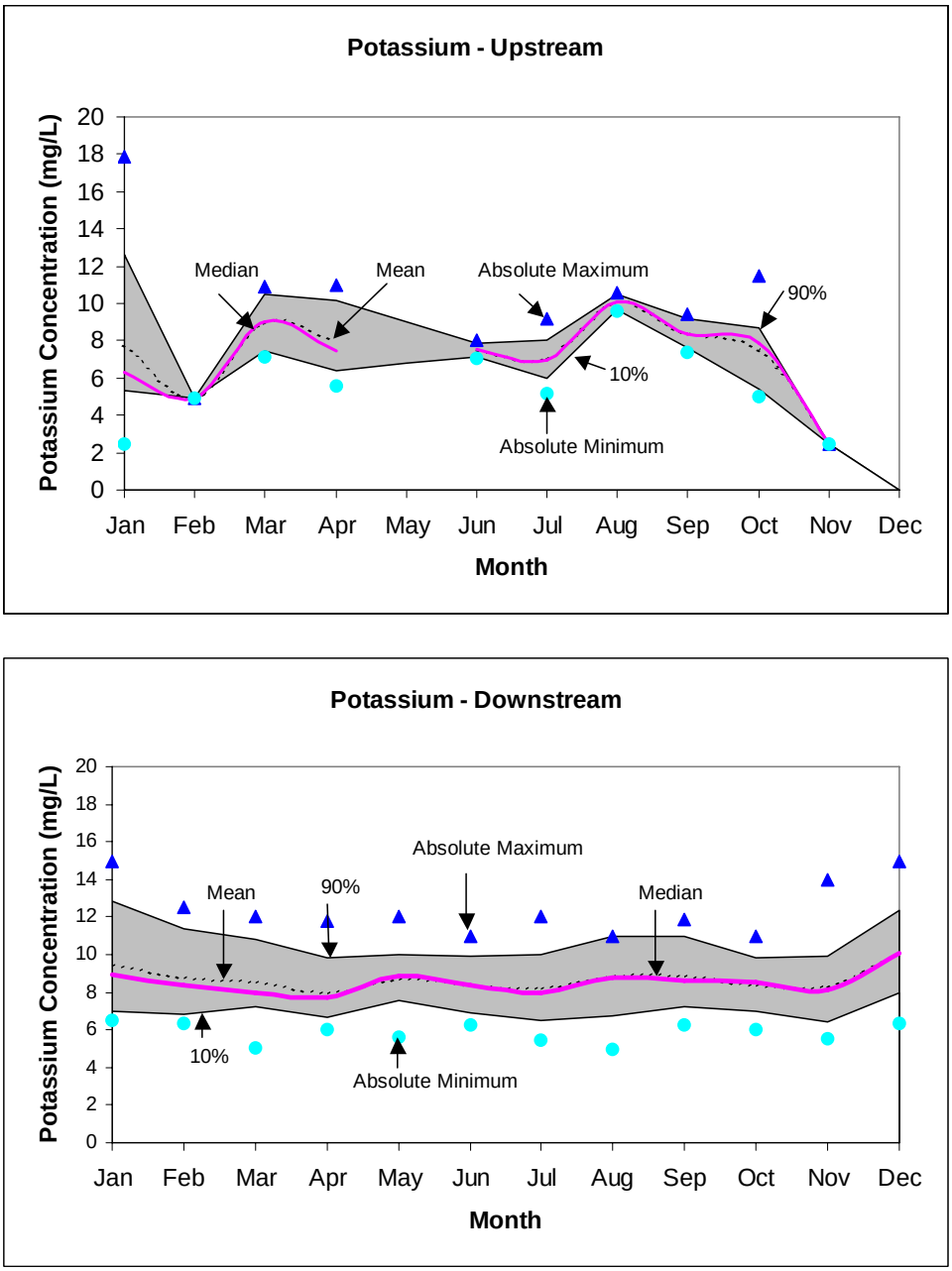


Figure 22: Seasonal Potassium on the Red River Upstream and Downstream of the City of Winnipeg

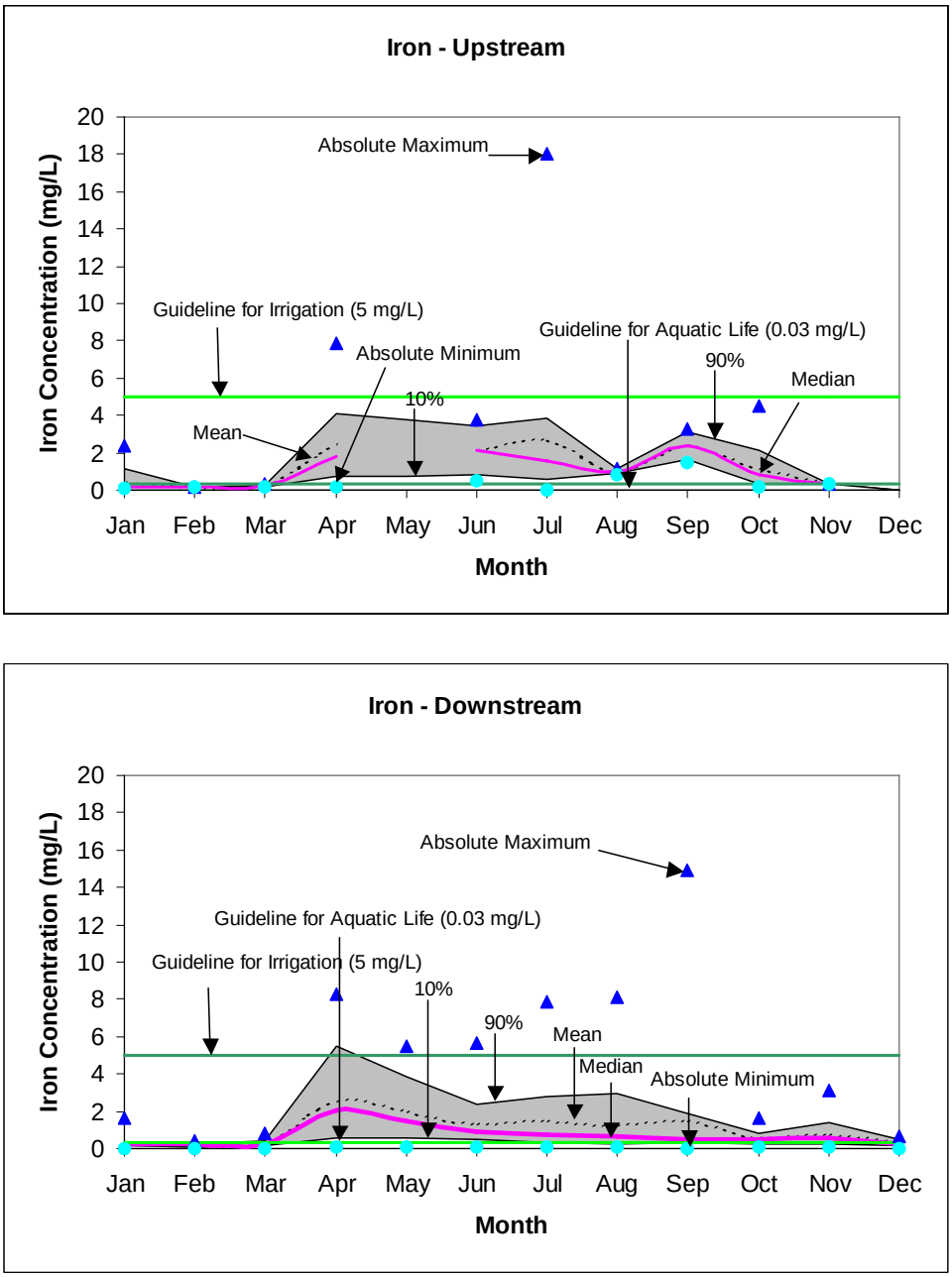
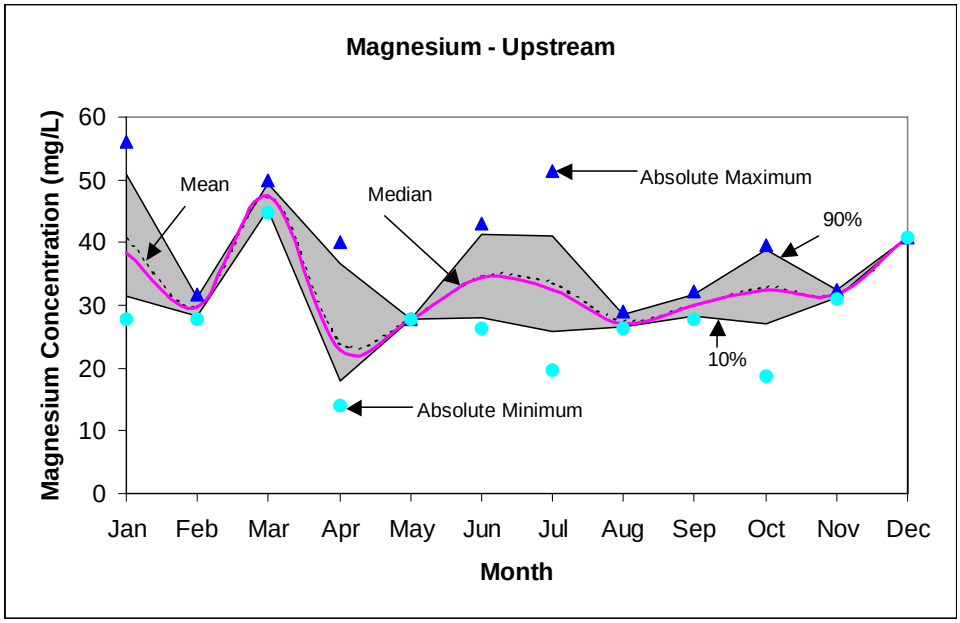


Figure 23: Seasonal Iron on the Red River Upstream and Downstream of the City of Winnipeg



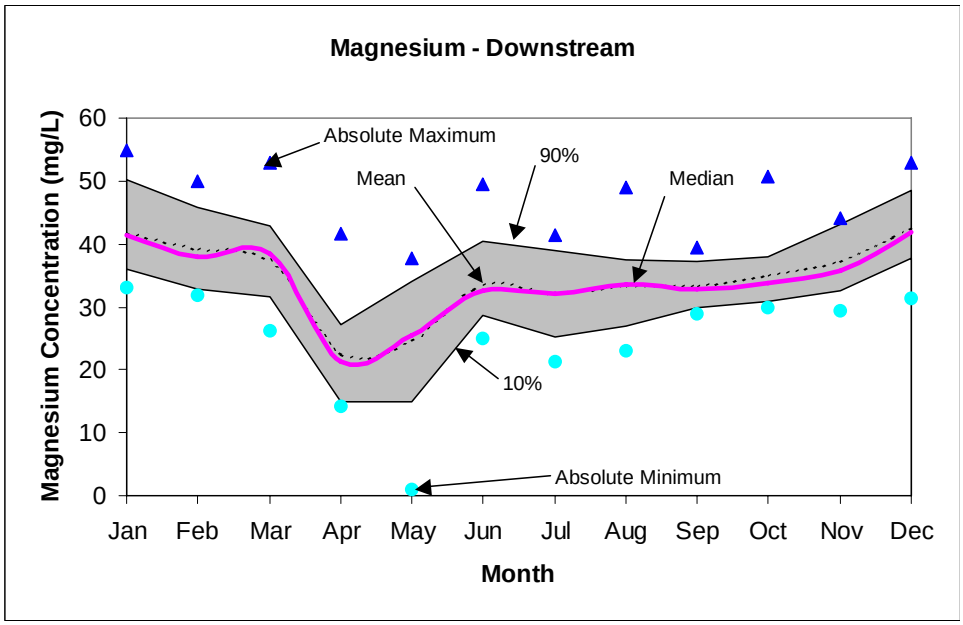
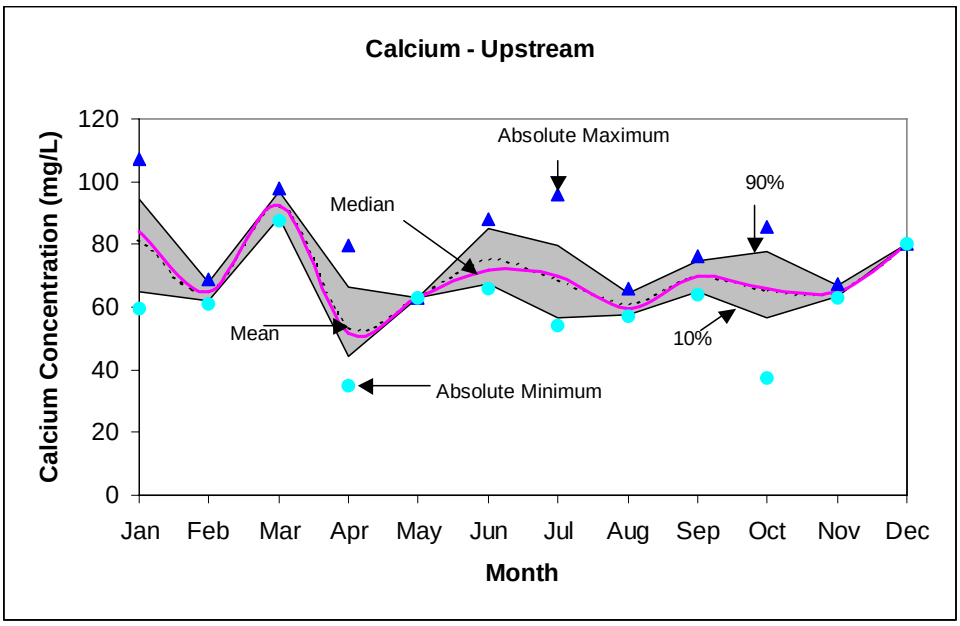


Figure 24: Seasonal Magnesium on the Red River Upstream and Downstream of the City of Winnipeg



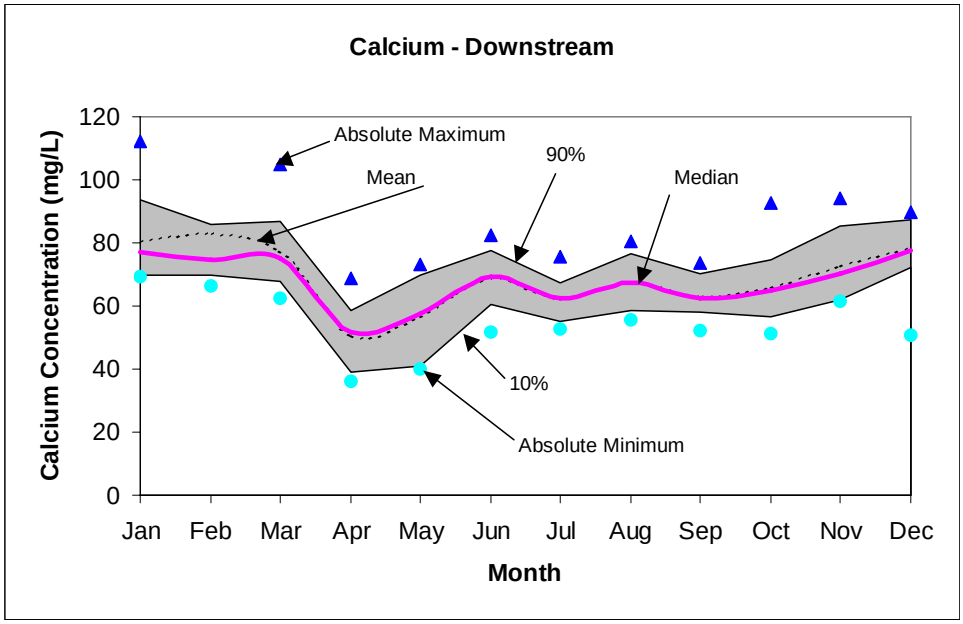
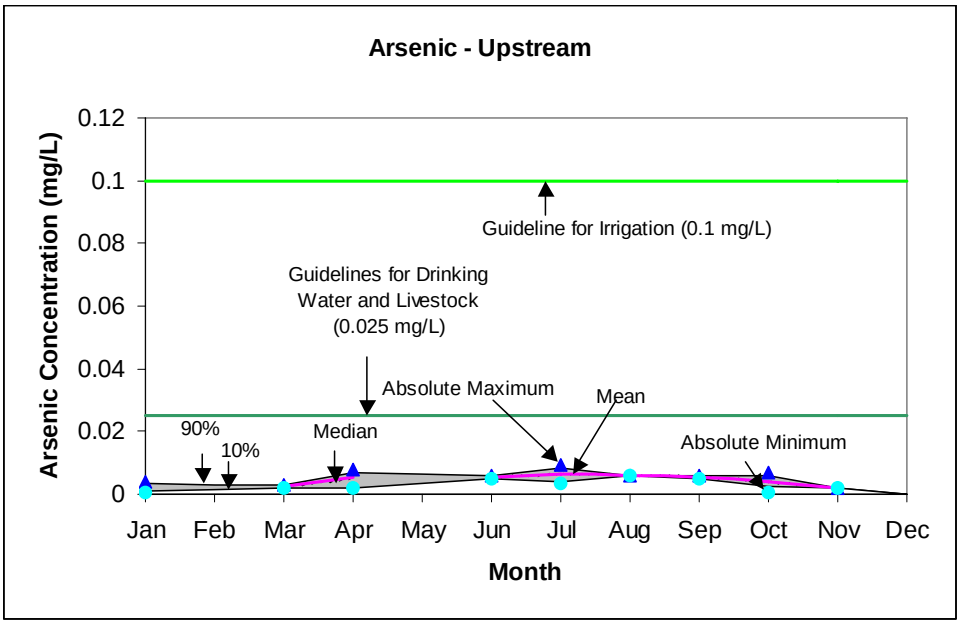


Figure 25: Seasonal Calcium on the Red River Upstream and Downstream of the City of Winnipeg



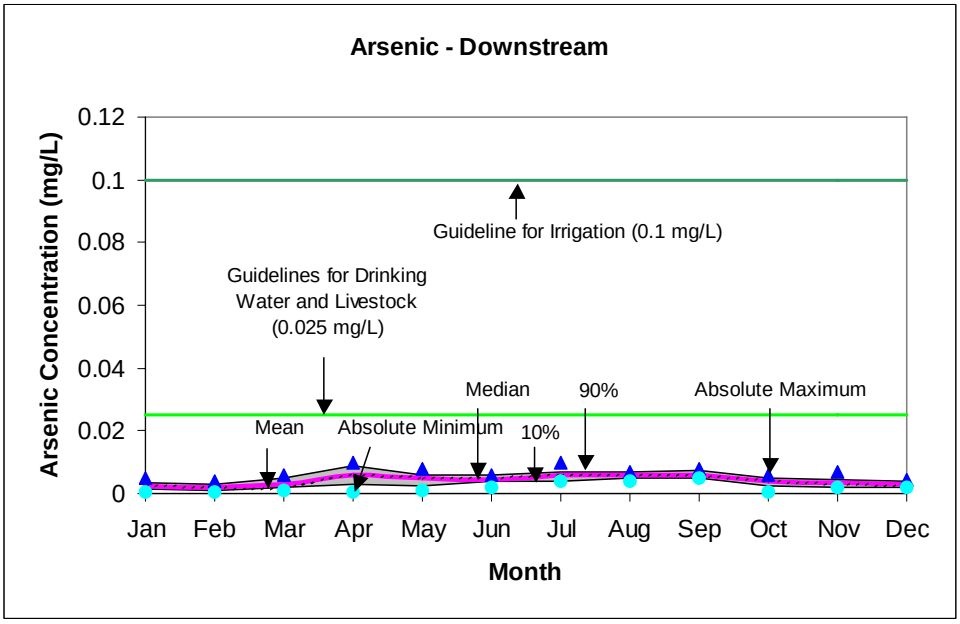
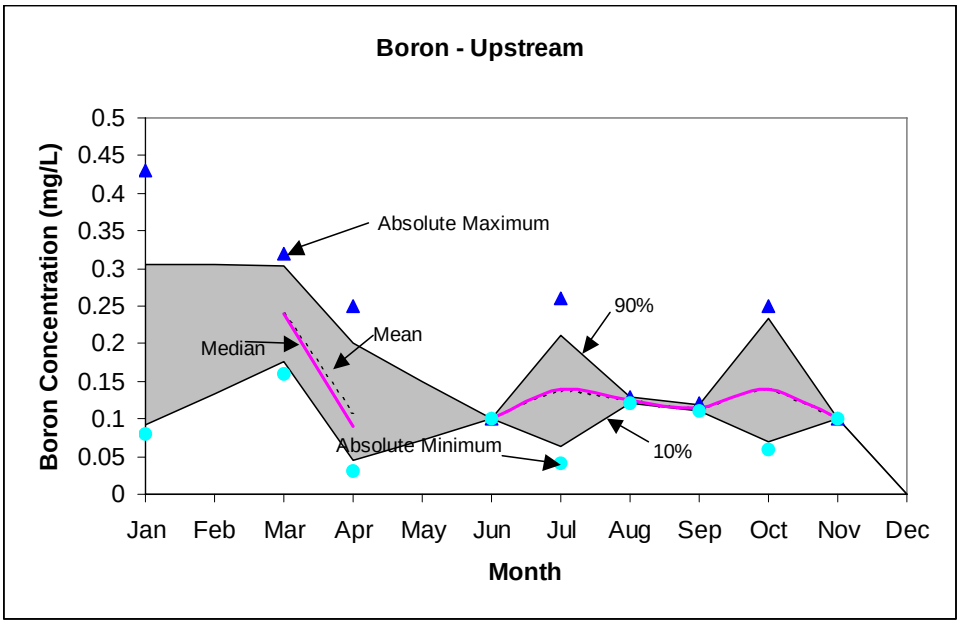


Figure 26: Seasonal Arsenic on the Red River Upstream and Downstream of the City of Winnipeg



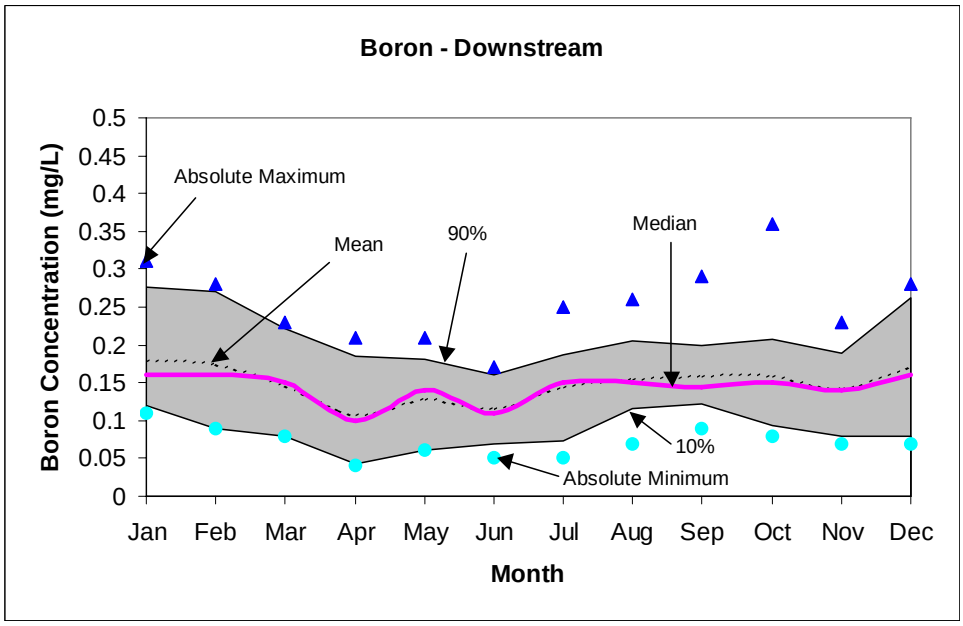
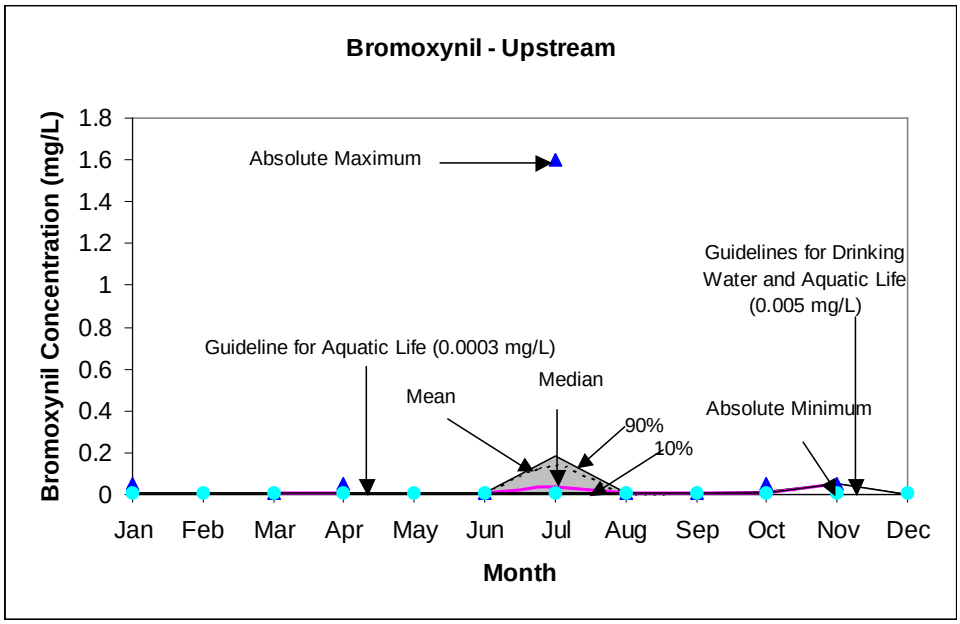


Figure 27: Seasonal Boron on the Red River Upstream and Downstream of the City of Winnipeg



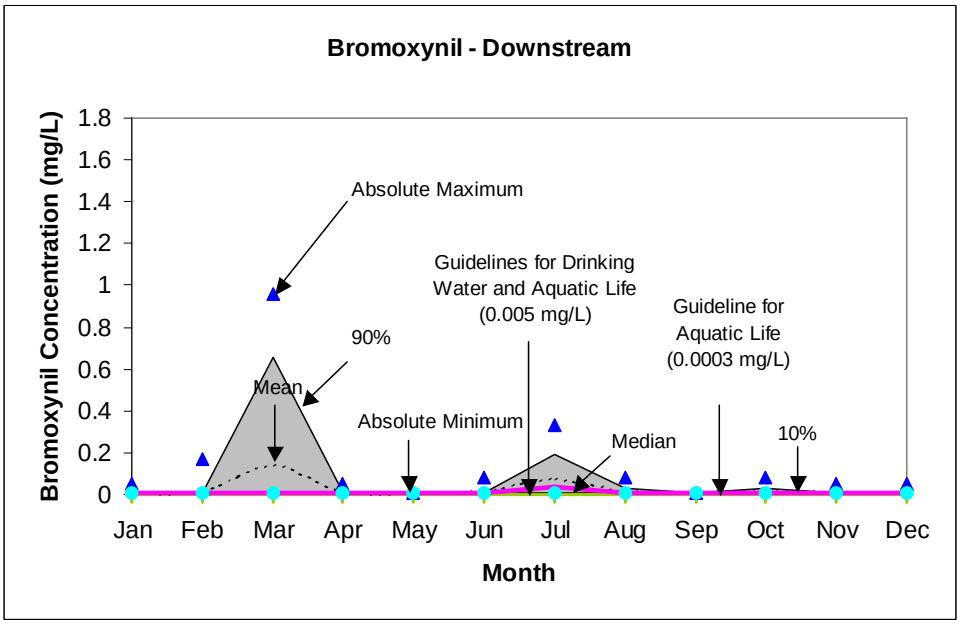


Figure 28: Seasonal Bromoxynil on the Red River Upstream and Downstream of the City of Winnipeg

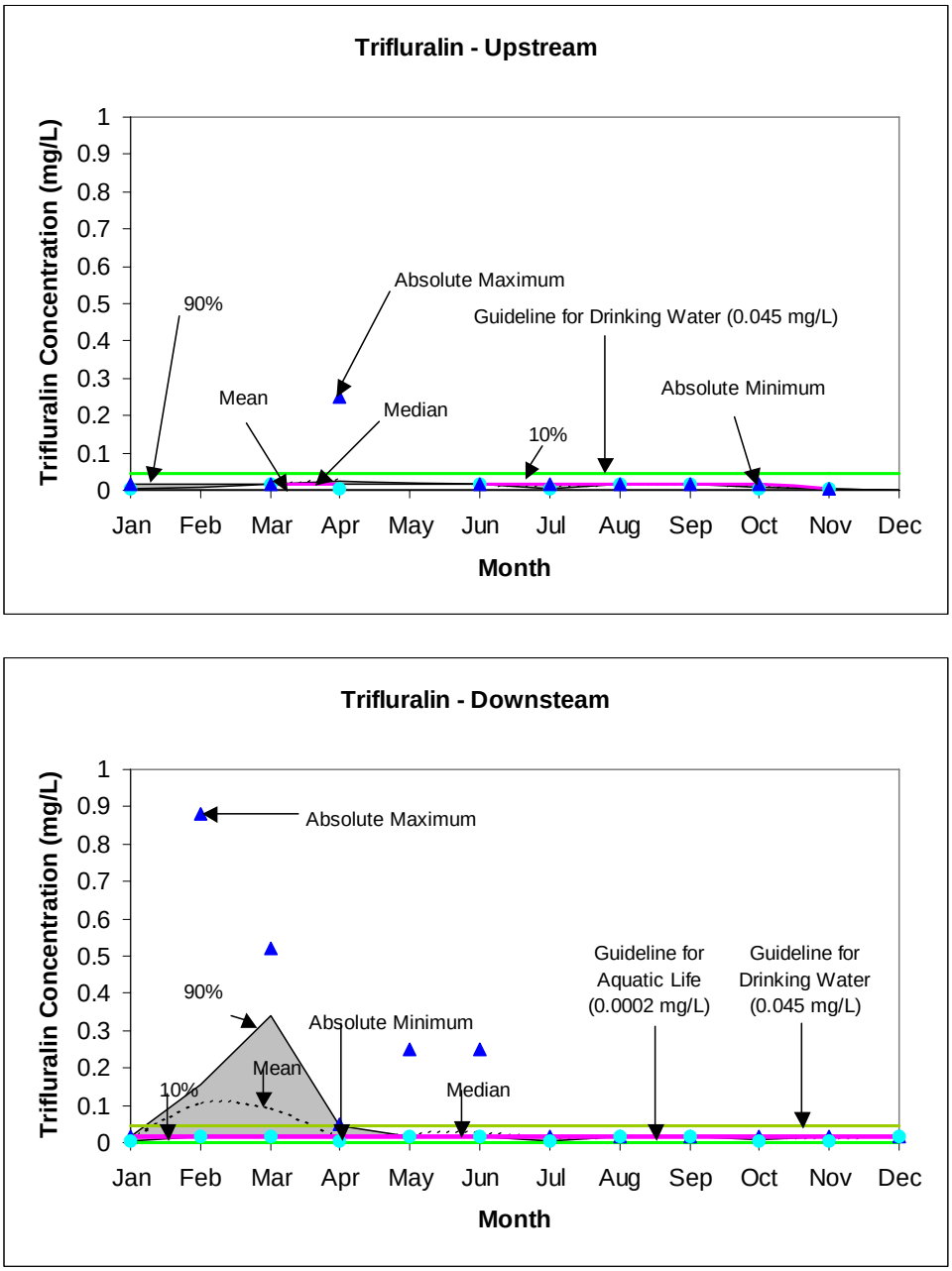
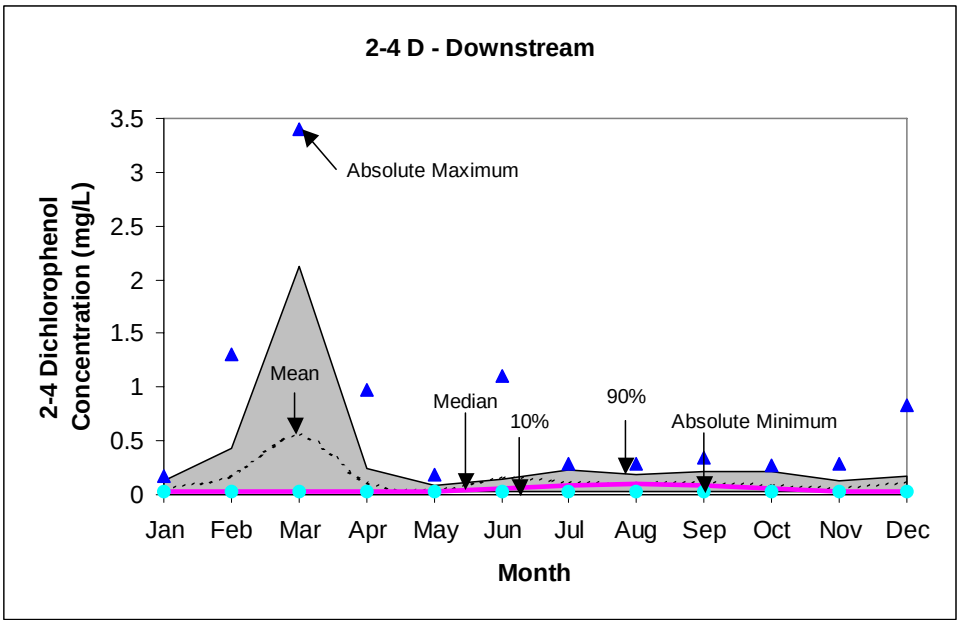


Figure 29: Seasonal Trifluralin on the Red River Upstream and Downstream of the City of Winnipeg



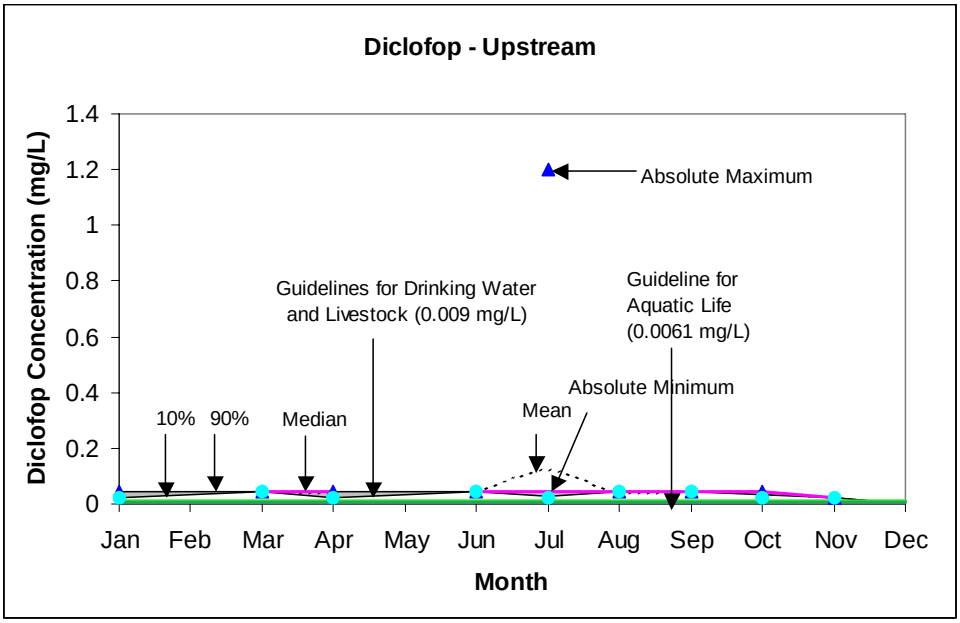


Figure 30: Seasonal 2-4D on the Red River Downstream and Diclofop Upstream of the City of Winnipeg

Attachment 8B-A

Physical and Chemical Parameters

Upstream Red River

Table A-1 Average Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Temperature (C)	0.2	-	2.1	3.8	11.8	18.2	22.4	22.7	17.2	10.1	2.7	0.8
Tatal Alkalinity (as CaCO3)	288.4	292.2	289.2	151.2	191.5	213.5	199.6	214.9	213.7	213.6	234.0	287.0
Tubudity (NTU)	12.5	5.6	15.7	133.2	87.0	97.7	139.8	105.5	67.3	45.8	43.6	12.9
TSS (mg/L)	12.3	6.9	19.8	246.1	128.5	134.7	290.6	141.9	92.5	68.4	70.3	19.1
pH	7.8	7.7	7.6	7.8	8.2	8.2	8.1	8.2	8.3	8.4	8.4	8.2

Table A-2 Median Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Temperature (C)	0.2	-	1.0	3.0	12.0	18.8	23.0	22.0	18.0	10.0	2.3	0.8
Tatal Alkalinity (as CaCO3)	278.5	275.0	273.0	140.3	194.0	215.5	212.5	219.5	219.5	217.5	224.0	262.0
Tubudity (NTU)	5.9	4.8	6.5	150.0	74.0	80.0	150.0	74.5	36.0	38.5	28.0	10.0
TSS (mg/L)	7.5	7.5	8.0	226.7	91.0	125.0	202.0	75.0	37.0	45.0	50.5	13.0
pH	7.8	7.7	7.7	7.8	8.2	8.2	8.1	8.3	8.3	8.4	8.3	8.1

Table A-3 Maximum Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Temperature (C)	0.2		6.0	10.2	18.0	24.0	27.0	30.0	22.0	15.0	8.0	1.0
Tatal Alkalinity (as CaCO3)	368.0	387.0	497.0	318.0	222.0	248.0	243.0	256.0	242.0	248.0	454.0	550.0
Tubudity (NTU)	140.0	10.0	130.0	270.0	210.0	250.0	380.0	210.0	270.0	120.0	140.0	65.0
TSS (mg/L)	100.0	15.0	180.0	580.0	420.0	360.0	1590.0	430.0	590.0	324.0	348.0	100.0
pH	8.2	7.9	8.0	8.3	8.5	8.6	8.6	8.7	8.7	8.7	9.1	8.7

Table A-4 Minimum Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Temperature (C)	0.2	-	0.5	0.5	6.5	12.0	17.5	18.0	11.0	5.0	0.0	0.5
Tatal Alkalinity (as CaCO3)	222.0	231.0	148.0	98.5	116.0	156.0	128.0	160.0	146.0	134.0	182.0	223.0
Tubudity (NTU)	0.8	0.6	2.5	2.1	20.0	30.0	18.0	14.0	15.0	14.0	7.0	2.2
TSS (mg/L)	2.5	2.0	2.0	8.0	4.0	41.0	2.5	17.0	20.0	12.0	15.0	2.5
pH	7.4	7.2	7.2	7.3	7.4	7.4	7.5	7.5	7.6	7.8	8.1	7.8

Downstream Red River

Table A-5 Average Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Temperature (C)	0.5	0.4	1.0	4.2	11.2	16.1	21.0	20.6	16.3	9.0	3.5	0.8
Tatal Alkalinity (as CaCO3)	284.1	275.4	267.1	148.8	185.1	211.1	199.9	212.8	212.6	221.7	235.4	270.0
Tubudity (NTU)	6.8	12.1	14.8	160.9	82.9	61.4	75.7	40.4	44.1	19.7	26.9	16.1
TSS (mg/L)	10.7	7.9	17.6	317.7	113.7	100.3	109.2	50.7	52.8	31.4	48.2	21.8
pH	7.8	7.7	7.7	7.8	8.2	8.2	8.2	8.2	8.3	8.2	8.2	8.1

Table A-6 Median Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Temperature (C)	0.5	0.4	1.0	4.0	13.0	17.3	22.8	23.0	17.5	9.5	2.8	1.0
Tatal Alkalinity (as CaCO3)	287.0	277.0	271.5	142.0	189.0	214.0	204.0	220.0	216.5	223.5	238.5	268.5
Tubudity (NTU)	5.5	5.4	5.1	170.0	65.0	47.0	42.0	31.8	21.0	15.0	14.0	7.5
TSS (mg/L)	9.0	7.0	8.5	290.0	90.0	83.0	58.5	32.0	29.0	30.0	26.0	15.0
pH	7.8	7.7	7.7	7.8	8.2	8.2	8.2	8.2	8.3	8.3	8.2	8.1

Table A-7 Maximum Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Temperature (C)	0.5	0.5	1.0	9.0	22.0	25.0	27.0	31.0	21.0	15.0	8.0	1.0
Tatal Alkalinity (as CaCO3)	335.0	346.0	339.0	212.0	228.0	248.0	243.0	244.0	242.0	254.0	288.0	318.0
Tubudity (NTU)	16.0	86.0	120.0	330.0	250.0	200.0	400.0	130.0	305.0	43.5	130.0	190.0
TSS (mg/L)	40.0	26.0	150.0	1200.0	260.0	352.0	605.0	225.0	587.0	93.0	350.0	280.0
pH	8.4	8.3	8.0	8.3	8.7	8.6	8.9	8.8	8.6	8.8	8.4	8.5

Table A-8 Minimum Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Temperature (C)	0.5	0.3	1.0	0.5	1.0	2.0	2.0	2.0	2.0	1.0	0.5	0.2
Tatal Alkalinity (as CaCO3)	224.0	223.0	273.0	110.0	118.0	154.0	149.0	142.0	140.0	160.0	195.0	225.0
Tubudity (NTU)	2.1	0.8	3.4	5.3	6.5	8.5	2.5	7.7	7.0	1.0	4.0	0.8
TSS (mg/L)	2.5	2.5	4.0	10.0	8.0	17.0	14.0	2.5	6.0	2.5	7.0	2.5
pH	7.1	7.0	7.2	7.3	7.4	7.6	7.5	7.6	7.6	7.6	7.8	7.6

Attachment 8B-B

Nutrients

Upstream Red River

Table B-1 Average Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DO (mg/L)	10.30	9.12	9.08	9.93	9.10	7.71	6.71	6.73	7.88	9.91	11.92	12.98
Ammonia (mg/L)	0.22	0.34	0.18	0.26	0.08	0.05	0.05	0.16	0.04	0.04	0.05	0.23
Nitrite-Nitrate (mg/L)	0.36	0.56	0.80	1.70	0.50	0.45	0.48	0.36	0.20	0.17	0.12	0.20
Total Phosphorus (mg/L)	0.18	0.16	0.18	0.42	0.27	0.26	0.41	0.33	0.23	0.19	0.21	0.18
Total Particulate Phosphorus (mg/L)	0.03	0.06	0.04	0.31	0.35	0.30	0.85	0.31	0.32	0.21	0.25	0.11
Total Soluble Phosphorus (mg/L)	0.15	0.18	0.14	0.17	0.07	0.14	0.13	0.16	0.16	0.09	0.09	0.07
Total Nitrogen (mg/L)	1.59	1.74	1.89	3.19	1.59	1.49	1.56	1.53	1.25	1.23	1.17	1.86
TN:TP	10.81	12.97	13.00	8.96	6.29	6.98	5.11	5.45	5.85	6.81	6.29	10.66

Table B-2 Median Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DO (mg/L)	10.40	9.65	9.50	9.90	9.05	7.60	6.70	7.05	8.05	10.05	11.90	12.70
Ammonia (mg/L)	0.17	0.20	0.14	0.25	0.07	0.05	0.05	0.05	0.03	0.04	0.04	0.08
Nitrite-Nitrate (mg/L)	0.32	0.52	0.77	1.55	0.35	0.34	0.46	0.35	0.15	0.18	0.04	0.18
Total Phosphorus (mg/L)	0.13	0.10	0.15	0.41	0.24	0.25	0.31	0.31	0.22	0.18	0.20	0.17
Total Particulate Phosphorus (mg/L)	0.03	0.07	0.03	0.35	0.35	0.27	0.14	0.19	0.15	0.10	0.12	0.12
Total Soluble Phosphorus (mg/L)	0.07	0.07	0.15	0.19	0.06	0.10	0.10	0.18	0.13	0.08	0.08	0.07
Total Nitrogen (mg/L)	1.46	1.58	1.77	3.05	1.50	1.42	1.54	1.46	1.25	1.16	1.23	1.24
TN:TP	10.30	14.14	12.13	7.00	5.55	6.00	4.81	5.21	5.69	6.20	6.61	7.73

Table B-3 Maximum Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DO (mg/L)	14.10	12.30	12.60	13.40	11.00	10.10	9.60	8.80	9.70	11.20	15.20	17.60
Ammonia (mg/L)	0.70	1.28	0.52	0.49	0.21	0.13	0.21	1.60	0.11	0.09	0.12	0.94
Nitrite-Nitrate (mg/L)	1.10	1.09	1.70	4.10	1.53	1.65	0.97	1.40	0.72	0.58	0.76	0.80
Total Phosphorus (mg/L)	0.52	0.50	0.47	0.87	0.66	0.63	2.38	0.74	0.86	0.57	0.64	0.40
Total Particulate Phosphorus (mg/L)	0.03	0.08	0.05	0.55	0.55	0.36	2.36	0.62	0.73	0.44	0.52	0.12
Total Soluble Phosphorus (mg/L)	0.31	0.42	0.21	0.21	0.11	0.27	0.28	0.19	0.20	0.13	0.11	0.11
Total Nitrogen (mg/L)	2.75	3.10	3.60	6.30	2.91	3.48	3.77	3.00	2.02	2.06	2.00	13.13
TN:TP	18.71	22.71	24.83	25.65	12.58	22.20	9.00	8.66	10.69	14.71	11.50	68.39

Table B-4 Minimum Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DO (mg/L)	6.90	1.00	4.10	4.60	6.40	6.00	4.20	2.94	6.10	8.30	9.00	9.70
Ammonia (mg/L)	0.10	0.01	0.05	0.02	0.02	0.01	0.02	0.02	0.02	0.02	0.02	0.02
Nitrite-Nitrate (mg/L)	0.08	0.31	0.17	0.38	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Total Phosphorus (mg/L)	0.07	0.05	0.06	0.14	0.11	0.05	0.10	0.11	0.08	0.11	0.07	0.06
Total Particulate Phosphorus (mg/L)	0.03	0.03	0.03	0.02	0.16	0.26	0.07	0.13	0.09	0.09	0.11	0.08
Total Soluble Phosphorus (mg/L)	0.07	0.04	0.06	0.12	0.05	0.06	0.02	0.12	0.13	0.05	0.07	0.02
Total Nitrogen (mg/L)	1.01	1.09	0.60	1.28	0.67	0.77	0.86	0.81	0.71	0.71	0.21	0.80
TN:TP	4.77	5.80	6.17	3.57	1.68	2.09	1.58	2.87	1.96	3.12	1.19	2.58

Downstream Red River

Table B-5 Average Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DO (mg/L)	9.94	9.31	9.40	10.14	-	-	-	-	-	-	-	-
Ammonia (mg/L)	2.10	2.27	1.89	0.55	0.25	0.19	0.18	0.31	0.41	0.72	1.20	1.50
Nitrite-Nitrate (mg/L)	0.49	0.61	0.77	1.54	0.55	0.51	0.54	0.47	0.44	0.37	0.31	0.39
Total Phosphorus (mg/L)	0.32	0.33	0.37	0.46	0.28	0.27	0.32	0.30	0.28	0.27	0.28	0.28
Total Particulate Phosphorus (mg/L)	0.48	0.49	0.45	0.47	0.46	0.34	0.84	0.73	0.35	0.18	0.64	0.48
Total Soluble Phosphorus (mg/L)	0.30	0.37	0.41	0.26	0.20	0.43	0.30	0.25	0.29	0.20	0.23	0.24
Total Nitrogen (mg/L)	3.45	3.20	3.45	3.40	2.00	1.84	1.63	1.89	1.98	2.00	2.35	3.23
TN:TP	10.53	10.71	10.03	7.70	7.37	7.33	7.10	6.46	7.23	7.33	8.99	11.02

Table B-6 Median Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DO (mg/L)	10.10	9.10	9.50	10.00	-	-	-	-	-	-	-	-
Ammonia (mg/L)	1.60	1.50	1.82	0.43	0.21	0.16	0.14	0.19	0.33	0.57	0.94	0.94
Nitrite-Nitrate (mg/L)	0.42	0.56	0.70	1.30	0.43	0.35	0.50	0.42	0.36	0.35	0.24	0.28
Total Phosphorus (mg/L)	0.27	0.25	0.33	0.46	0.25	0.25	0.28	0.29	0.24	0.26	0.25	0.26
Total Particulate Phosphorus (mg/L)	0.48	0.46	0.50	0.54	0.32	0.32	0.48	0.89	0.24	0.18	0.81	0.44
Total Soluble Phosphorus (mg/L)	0.27	0.37	0.39	0.21	0.17	0.27	0.19	0.20	0.16	0.18	0.19	0.17
Total Nitrogen (mg/L)	2.84	2.63	3.11	3.10	1.97	1.69	1.60	1.75	1.82	1.85	2.25	2.33
TN:TP	9.59	10.07	10.13	6.80	7.81	7.00	6.75	6.15	7.35	7.12	8.75	9.84

Table B-7 Maximum Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DO (mg/L)	12.50	12.60	12.80	12.70	-	-	-	-	-	-	-	-
Ammonia (mg/L)	6.10	5.70	4.72	1.68	0.50	0.42	0.61	1.41	1.75	2.44	2.95	5.28
Nitrite-Nitrate (mg/L)	1.40	1.63	1.80	4.00	1.45	2.40	2.80	1.50	1.40	0.84	1.30	2.40
Total Phosphorus (mg/L)	0.90	0.90	1.34	0.87	0.47	0.88	1.17	0.72	0.81	0.45	0.50	0.57
Total Particulate Phosphorus (mg/L)	0.62	0.59	0.51	0.65	0.92	0.57	1.60	0.96	0.68	0.19	0.87	0.85
Total Soluble Phosphorus (mg/L)	0.59	0.65	0.65	0.60	0.47	0.99	0.90	0.68	0.90	0.36	0.48	0.48
Total Nitrogen (mg/L)	6.64	7.34	6.74	6.24	3.00	4.93	2.71	3.85	3.55	4.48	4.40	16.64
TN:TP	20.73	18.00	22.56	15.45	13.08	14.43	23.61	13.61	14.79	15.35	20.30	29.24

Table B-8 Minimum Monthly

Parameter	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DO (mg/L)	5.00	4.10	6.70	8.30	-	-	-	-	-	-	-	-
Ammonia (mg/L)	0.74	0.03	0.85	0.21	0.02	0.01	0.02	0.02	0.04	0.16	0.32	0.08
Nitrite-Nitrate (mg/L)	0.15	0.28	0.75	0.29	0.05	0.01	0.01	0.05	0.10	0.01	0.06	0.08
Total Phosphorus (mg/L)	0.14	0.06	0.23	0.11	0.11	0.12	0.06	0.18	0.14	0.14	0.16	0.07
Total Particulate Phosphorus (mg/L)	0.35	0.43	0.34	0.22	0.13	0.12	0.43	0.35	0.12	0.18	0.25	0.16
Total Soluble Phosphorus (mg/L)	0.15	0.22	0.18	0.12	0.12	0.12	0.11	0.11	0.13	0.11	0.11	0.14
Total Nitrogen (mg/L)	2.03	1.08	0.90	1.70	1.17	0.65	0.92	0.49	1.10	0.61	0.89	1.08
TN:TP	6.24	7.33	3.91	4.23	3.30	1.71	2.12	2.32	2.38	1.73	2.15	5.04

Attachment 8B-C

Metals

Upstream Red River

Table C-1 Average Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
CADMIUM	0.0004	0.0005	0.0005	0.0004	0.0005	0.0004	0.0005	0.0005	0.0005	0.0004	0.0005	0.0005
CHROMIUM	0.0077	-	0.0100	0.0086	0.0100	0.0100	0.0090	0.0100	0.0100	0.0078	-	-
COPPER	0.0082	0.0060	0.0050	0.0105	0.0070	0.0092	0.0092	0.0063	0.0050	0.0054	0.0038	0.0170
LEAD	0.0015	-	0.0010	0.0043	0.0025	0.0018	0.0042	0.0022	0.0045	0.0023	0.0025	0.0040
NICKEL	0.0025	-	0.0025	0.0092	0.0025	0.0052	0.0103	0.0073	0.0120	0.0042	0.0025	0.0025
ZINC	0.073	0.005	0.005	0.021	0.040	0.013	0.052	0.008	0.020	0.009	0.005	0.005
MANGANESE	0.066	0.044	0.060	0.271	-	0.409	0.300	0.155	0.360	0.134	0.090	-
SODIUM	75.36	35.50	136.15	22.26	-	33.00	34.69	22.50	23.80	47.16	30.70	-
POTASSIUM	7.81	4.90	9.00	8.05	-	7.53	7.02	10.10	8.40	7.51	2.50	-
IRON	0.437	0.190	0.235	2.440	-	2.115	2.726	1.015	2.385	1.123	0.350	-
MAGNESIUM	40.63	29.75	47.35	24.14	27.70	34.67	33.73	27.43	29.95	32.88	31.75	40.80
CALCIUM	81.48	64.90	92.70	53.76	62.80	75.40	69.02	60.87	70.00	65.66	65.10	80.00
ARSENIC	0.0022	0.0005	-	0.0040	0.0050	0.0020	0.0055	0.0080	-	0.0033	0.0005	0.0005
MERCURY	0.0350	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100
BORON	0.1823	-	0.2400	0.1094	-	0.1000	0.1379	0.1250	0.1150	0.1385	0.1000	-

Table C-2 Median Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
CADMIUM	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005
CHROMIUM	0.0100	-	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	-	-
COPPER	0.0050	0.0060	0.0050	0.0091	0.0070	0.0100	0.0078	0.0050	0.0050	0.0050	0.0038	0.0170
LEAD	0.0010	-	0.0010	0.0025	0.0025	0.0020	0.0030	0.0020	0.0045	0.0025	0.0025	0.0040
NICKEL	0.0025	-	0.0025	0.0090	0.0025	0.0060	0.0070	0.0080	0.0120	0.0025	0.0025	0.0025
ZINC	0.0050	0.0050	0.0050	0.0200	0.0400	0.0050	0.0100	0.0100	0.0200	0.0050	0.0050	0.0050
MANGANESE	0.040	0.044	0.060	0.240	-	0.409	0.250	0.155	0.360	0.090	0.090	-
SODIUM	45.75	35.50	136.15	22.40	-	33.00	34.25	22.50	23.80	38.20	30.70	-
POTASSIUM	6.35	4.90	9.00	7.50	-	7.53	7.00	10.10	8.40	7.84	2.50	-
IRON	0.170	0.190	0.235	1.830	-	2.115	1.560	1.015	2.385	0.840	0.350	-
MAGNESIUM	38.30	29.75	47.35	22.80	27.70	34.50	32.50	27.00	29.95	32.55	31.75	40.80
CALCIUM	83.90	64.90	92.70	51.45	62.80	72.00	70.00	59.70	70.00	65.95	65.10	80.00
ARSENIC	0.0024	0.0005	-	0.0045	0.0050	0.0020	0.0050	0.0080	-	0.0031	0.0005	0.0005
MERCURY	0.0350	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100
BORON	0.1300	-	0.2400	0.0900	-	0.1000	0.1400	0.1250	0.1150	0.1400	0.1000	-

Table C-3 Maximum Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
CADMIUM	0.0005	0.0005	0.0005	0.0005	0.0005	0.0005	0.0025	0.0005	0.0005	0.0005	0.0005	0.0005
CHROMIUM	0.0100	-	0.0100	0.0100	0.0100	0.0100	0.0180	0.0100	0.0100	0.0100	-	-
COPPER	0.0600	0.0060	0.0050	0.0300	0.0070	0.0150	0.0320	0.0090	0.0050	0.0100	0.0050	0.0170
LEAD	0.0060	-	0.0010	0.0230	0.0025	0.0025	0.0140	0.0025	0.0055	0.0045	0.0025	0.0040
NICKEL	0.0040	-	0.0025	0.0230	0.0025	0.0070	0.0380	0.0090	0.0160	0.0120	0.0025	0.0025
ZINC	1.140	0.005	0.005	0.050	0.040	0.030	0.590	0.010	0.020	0.040	0.005	0.005
MANGANESE	0.386	0.044	0.070	0.630	-	0.670	1.320	0.160	0.470	0.400	0.090	-
SODIUM	202.00	35.50	190.00	44.50	-	42.60	75.10	23.80	27.80	106.00	30.70	-
POTASSIUM	17.90	4.90	10.90	11.00	-	8.00	9.20	10.60	9.40	11.50	2.50	-
IRON	2.370	0.190	0.290	7.880	-	3.770	18.000	1.170	3.270	4.490	0.350	-
MAGNESIUM	56.00	31.60	50.00	40.00	27.70	43.10	51.50	29.00	32.10	39.70	32.50	40.80
CALCIUM	107.00	68.70	98.00	79.50	62.80	88.10	95.80	65.80	76.10	85.80	67.20	80.00
ARSENIC	0.0031	0.0005	-	0.0053	0.0050	0.0020	0.0092	0.0080	-	0.0052	0.0005	0.0005
MERCURY	0.0600	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100
BORON	0.4300	-	0.3200	0.2500	-	0.1000	0.2600	0.1300	0.1200	0.2500	0.1000	-

Table C-4 Minimum Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
CADMIUM	0.00002	0.00050	0.00050	0.00002	0.00050	0.00025	0.00002	0.00050	0.00050	0.00002	0.00050	0.00050
CHROMIUM	0.0001	-	0.0100	0.0004	0.0100	0.0100	0.0016	0.0100	0.0100	0.0006	-	-
COPPER	0.0022	0.0060	0.0050	0.0021	0.0070	0.0025	0.0048	0.0050	0.0050	0.0025	0.0025	0.0170
LEAD	0.0001	-	0.0010	0.0001	0.0025	0.0010	0.0009	0.0020	0.0035	0.0003	0.0025	0.0040
NICKEL	0.0010	-	0.0025	0.0010	0.0025	0.0025	0.0025	0.0050	0.0080	0.0020	0.0025	0.0025
ZINC	0.0020	0.0050	0.0050	0.0030	0.0400	0.0050	0.0050	0.0050	0.0200	0.0030	0.0050	0.0050
MANGANESE	0.0210	0.0440	0.0500	0.0580	-	0.1480	0.0900	0.1500	0.2500	0.0580	0.0900	-
SODIUM	20.90	35.50	82.30	9.43	-	23.40	10.40	21.20	19.80	23.70	30.70	-
POTASSIUM	2.50	4.90	7.10	5.56	-	7.05	5.20	9.60	7.40	5.00	2.50	-
IRON	0.110	0.190	0.180	0.200	-	0.460	0.030	0.860	1.500	0.180	0.350	-
MAGNESIUM	27.90	27.90	44.70	14.00	27.70	26.40	19.70	26.30	27.80	18.80	31.00	40.80
CALCIUM	59.60	61.10	87.40	35.00	62.80	66.10	54.00	57.10	63.90	37.40	63.00	80.00
ARSENIC	0.0010	0.0005	-	0.0021	0.0050	0.0020	0.0034	0.0080	-	0.0020	0.0005	0.0005
MERCURY	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100
BORON	0.0800	-	0.1600	0.0300	-	0.1000	0.0400	0.1200	0.1100	0.0600	0.1000	-

Downstream Red River

Table C-5 Average Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
CADMIUM	0.0008	0.0006	0.0006	0.0005	0.0005	0.0006	0.0019	0.0010	0.0009	0.0007	0.0007	0.0006
CHROMIUM	0.0101	0.0076	0.0077	0.0088	0.0088	0.0099	0.0094	0.0088	0.0088	0.0076	0.0076	0.0073
COPPER	0.0064	0.0055	0.0066	0.0114	0.0079	0.0086	0.0071	0.0076	0.0071	0.0056	0.0071	0.0057
LEAD	0.0022	0.0016	0.0023	0.0050	0.0027	0.0024	0.0049	0.0041	0.0036	0.0027	0.0020	0.0018
NICKEL	0.0032	0.0030	0.0033	0.0098	0.0069	0.0073	0.0065	0.0066	0.0060	0.0040	0.0041	0.0036
ZINC	0.1024	0.0123	0.0133	0.0274	0.0170	0.0284	0.0119	0.0109	0.0111	0.0140	0.0150	0.0109
MANGANESE	0.059	0.051	0.058	0.306	0.196	0.179	0.168	0.123	0.142	0.103	0.114	0.055
SODIUM	73.50	70.52	61.40	24.92	29.05	43.23	43.20	45.25	53.83	50.99	53.75	73.54
POTASSIUM	9.44	8.79	8.50	7.96	8.72	8.40	8.18	8.82	8.86	8.41	8.30	10.05
IRON	0.307	0.212	0.271	2.556	1.928	1.282	1.461	1.244	1.488	0.535	0.773	0.302
MAGNESIUM	41.83	39.12	37.48	22.52	24.65	33.46	32.00	33.32	33.25	35.02	37.18	42.63
CALCIUM	80.36	82.91	77.61	50.96	56.74	68.95	62.22	67.14	62.81	66.07	72.49	78.39
ARSENIC	0.0082	0.0079	0.0083	0.0068	0.0079	0.0077	0.0085	0.0096	0.0079	0.0081	0.0071	0.0049
MERCURY	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100
BORON	0.1792	0.1740	0.1470	0.1071	0.1282	0.1164	0.1436	0.1538	0.1592	0.1592	0.1418	0.1710

Table C-6 Median Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
CADMIUM	0.0008	0.0006	0.0006	0.0005	0.0005	0.0006	0.0019	0.0010	0.0009	0.0007	0.0007	0.0006
CHROMIUM	0.0101	0.0076	0.0077	0.0088	0.0088	0.0099	0.0094	0.0088	0.0088	0.0076	0.0076	0.0073
COPPER	0.0064	0.0055	0.0066	0.0114	0.0079	0.0086	0.0071	0.0076	0.0071	0.0056	0.0071	0.0057
LEAD	0.0022	0.0016	0.0023	0.0050	0.0027	0.0024	0.0049	0.0041	0.0036	0.0027	0.0020	0.0018
NICKEL	0.0032	0.0030	0.0033	0.0098	0.0069	0.0073	0.0065	0.0066	0.0060	0.0040	0.0041	0.0036
ZINC	0.1024	0.0123	0.0133	0.0274	0.0170	0.0284	0.0119	0.0109	0.0111	0.0140	0.0150	0.0109
MANGANESE	0.059	0.051	0.058	0.306	0.196	0.179	0.168	0.123	0.142	0.103	0.114	0.055
SODIUM	73.50	70.52	61.40	24.92	29.05	43.23	43.20	45.25	53.83	50.99	53.75	73.54
POTASSIUM	9.44	8.79	8.50	7.96	8.72	8.40	8.18	8.82	8.86	8.41	8.30	10.05
IRON	0.307	0.212	0.271	2.556	1.928	1.282	1.461	1.244	1.488	0.535	0.773	0.302
MAGNESIUM	41.83	39.12	37.48	22.52	24.65	33.46	32.00	33.32	33.25	35.02	37.18	42.63
CALCIUM	80.36	82.91	77.61	50.96	56.74	68.95	62.22	67.14	62.81	66.07	72.49	78.39
MERCURY	0.0082	0.0079	0.0083	0.0068	0.0079	0.0077	0.0085	0.0096	0.0079	0.0081	0.0071	0.0049
ARSENIC	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100
BORON	0.1600	0.1600	0.1500	0.1000	0.1400	0.1100	0.1500	0.1500	0.1450	0.1500	0.1400	0.1600

Table C-7 Maximum Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
CADMIUM	0.0050	0.0025	0.0025	0.0025	0.0025	0.0025	0.0260	0.0060	0.0050	0.0025	0.0025	0.0025
CHROMIUM	0.0500	0.0100	0.0100	0.0100	0.0100	0.0400	0.0250	0.0130	0.0180	0.0100	0.0100	0.0100
COPPER	0.0200	0.0100	0.0200	0.0400	0.0200	0.0400	0.0230	0.0280	0.0220	0.0130	0.0200	0.0200
LEAD	0.0100	0.0050	0.0100	0.0210	0.0085	0.0050	0.0400	0.0300	0.0200	0.0100	0.0050	0.0100
NICKEL	0.0070	0.0070	0.0100	0.0260	0.0150	0.0300	0.0310	0.0150	0.0240	0.0072	0.0080	0.0080
ZINC	1.560	0.030	0.030	0.100	0.040	0.230	0.040	0.033	0.045	0.069	0.050	0.030
MANGANESE	0.130	0.110	0.166	0.720	0.474	0.290	0.628	0.250	0.533	0.166	0.190	0.090
SODIUM	152.00	125.00	99.80	68.00	68.00	75.00	78.00	87.00	79.10	71.70	100.00	110.00
POTASSIUM	15.00	12.50	12.00	11.80	12.00	11.00	12.00	11.00	11.90	11.00	14.00	15.00
IRON	1.680	0.440	0.860	8.280	5.460	5.670	7.880	8.080	14.900	1.670	3.120	0.680
MAGNESIUM	54.80	49.90	52.90	41.70	37.80	49.50	41.30	48.90	39.40	50.60	44.00	53.00
CALCIUM	112.00	205.00	105.00	68.80	73.20	82.40	75.50	80.60	73.80	92.70	94.00	89.80
MERCURY	0.010	0.010	0.010	0.010	0.010	0.010	0.010	0.010	0.010	0.010	0.010	0.010
ARSENIC	0.020	0.020	0.020	0.020	0.020	0.020	0.020	0.020	0.020	0.020	0.020	0.020
BORON	0.310	0.280	0.230	0.210	0.210	0.170	0.250	0.260	0.290	0.360	0.230	0.280

Table C-8 Minimum Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
CADMIUM	0.00002	0.00002	0.00002	0.00002	0.00002	0.00002	0.00002	0.00002	0.00002	0.00002	0.00002	0.00002
CHROMIUM	0.0003	0.0006	0.0001	0.0007	0.0013	0.0018	0.0016	0.0008	0.0013	0.0004	0.0008	0.0007
COPPER	0.0025	0.0022	0.0022	0.0025	0.0039	0.0025	0.0025	0.0039	0.0025	0.0025	0.0025	0.0025
LEAD	0.0002	0.0003	0.0003	0.0004	0.0009	0.0010	0.0010	0.0005	0.0005	0.0004	0.0001	0.0003
NICKEL	0.0020	0.0010	0.0010	0.0025	0.0025	0.0025	0.0025	0.0025	0.0025	0.0025	0.0025	0.0010
ZINC	0.0040	0.0100	0.0050	0.0090	0.0070	0.0090	0.0050	0.0040	0.0040	0.0030	0.0050	0.0050
MANGANESE	0.0220	0.0100	0.0270	0.0500	0.0700	0.0800	0.0770	0.0520	0.0100	0.0700	0.0560	0.0300
SODIUM	39.20	39.30	20.30	9.75	10.10	20.10	12.50	17.00	24.20	28.00	30.00	39.10
POTASSIUM	6.50	6.32	5.07	6.00	5.64	6.30	5.46	5.00	6.30	6.00	5.50	6.34
IRON	0.130	0.040	0.080	0.340	0.190	0.390	0.240	0.190	0.150	0.130	0.180	0.090
MAGNESIUM	33.10	31.80	26.10	14.30	1.00	25.00	21.40	23.00	28.80	30.00	29.50	31.30
CALCIUM	69.10	66.50	62.30	35.90	40.00	51.50	52.90	55.40	52.10	51.40	61.60	50.90
MERCURY	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100
ARSENIC	0.0010	0.0005	0.0017	0.0020	0.0024	0.0025	0.0025	0.0050	0.0030	0.0025	0.0005	0.0005
BORON	0.1100	0.0900	0.0800	0.0400	0.0600	0.0500	0.0500	0.0700	0.0900	0.0800	0.0700	0.0700

Attachment 8B-D

Herbicides/Pesticides

Upstream Red River

Table D1 Average Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DICLOFOP	0.039	-	0.045	0.041	-	0.045	0.123	0.045	0.045	0.042	0.020	-
TRIFLURALIN	0.013	-	0.015	0.029	-	0.015	0.013	0.015	0.015	0.014	0.003	-
BROMOXYNIL	0.010	-	0.005	0.011	-	0.005	0.142	0.005	0.005	0.010	0.050	-

Table D2 Median Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DICLOFOP	0.045	-	0.045	0.045	-	0.045	0.045	0.045	0.045	0.045	0.020	-
TRIFLURALIN	0.015	-	0.015	0.015	-	0.015	0.015	0.015	0.015	0.015	0.003	-
BROMOXYNIL	0.005	-	0.005	0.005	-	0.005	0.040	0.005	0.005	0.005	0.050	-

Table D3 Maximum Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DICLOFOP	0.045	-	0.045	0.045	-	0.045	1.200	0.045	0.045	0.045	0.020	-
TRIFLURALIN	0.015	-	0.015	0.250	-	0.015	0.015	0.015	0.015	0.015	0.003	-
BROMOXYNIL	0.050	-	0.005	0.050	-	0.005	1.600	0.005	0.005	0.050	0.050	-

Table D4 Minimum Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
DICLOFOP	0.020	-	0.045	0.020	-	0.045	0.020	0.045	0.045	0.020	0.020	-
TRIFLURALIN	0.003	-	0.015	0.003	-	0.015	0.003	0.015	0.015	0.003	0.003	-
BROMOXYNIL	0.005	-	0.005	0.005	-	0.005	0.005	0.005	0.005	0.005	0.050	-

Downstream Red River

Table D5 Average Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
2,4-D	0.055	0.178	0.562	0.119	0.043	0.153	0.112	0.099	0.113	0.083	0.061	0.114
TRIFLURALIN	0.013	0.106	0.093	0.020	0.025	0.029	0.013	0.015	0.015	0.014	0.014	0.015
BROMOXYNIL	0.009	0.018	0.142	0.011	0.006	0.011	0.072	0.014	0.008	0.014	0.011	0.010

Table D6 Median Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
2,4-D	0.055	0.178	0.562	0.119	0.043	0.153	0.112	0.099	0.113	0.083	0.061	0.114
TRIFLURALIN	0.100	0.100	0.100	0.100	0.425	0.367	0.100	0.100	0.100	0.100	0.100	0.100
BROMOXYNIL	0.009	0.018	0.142	0.011	0.006	0.011	0.072	0.014	0.008	0.014	0.011	0.010

Table D7 Maximum Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
2,4-D	0.170	1.300	3.400	0.970	0.180	1.100	0.280	0.290	0.350	0.270	0.290	0.830
TRIFLURALIN	0.015	0.880	0.520	0.050	0.250	0.250	0.015	0.015	0.015	0.015	0.015	0.015
BROMOXYNIL	0.050	0.170	0.960	0.050	0.010	0.080	0.330	0.080	0.010	0.080	0.050	0.050

Table D8 Minimum Monthly

Parameter (in ug/L)	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
2,4-D	0.025	0.025	0.025	0.025	0.025	0.025	0.025	0.025	0.025	0.025	0.025	0.025
TRIFLURALIN	0.003	0.015	0.015	0.003	0.015	0.015	0.003	0.015	0.015	0.003	0.003	0.015
BROMOXYNIL	0.005	0.005	0.005	0.005	0.005	0.005	0.005	0.005	0.005	0.005	0.005	0.005

Environmental Assessment of

Canadian Strategic Infrastructure Funded

Upgrades to the City of Winnipeg

Water Pollution Control Centres

APPENDIX 8C

Water Quality Model Results

1.0 INTRODUCTION

Water quality modeling was performed to support the Environmental Assessment of proposed upgrades to the City of Winnipeg Water Pollution control Centres (WPCCs). This appendix presents results obtained from a computer model that was used to simulate water quality conditions in the Red and Assiniboine Rivers based on projected WPCC discharges in the year 2031. Details of the model and results are not discussed here, but are dealt with in other sections of the assessment (e.g., Section 8 – Water Quantity and Quality). The model is briefly described below in order to provide the context for the graphics showing model results. Model results for a representative water quality parameter are discussed to demonstrate the information presented in the graphics.

2.0 BRIEF OVERVIEW OF THE WATER-QUALITY MODEL

Proposed upgrades to the City's WPCCs will change the water quality characteristics of the effluent being discharged from these plants, with nutrient reduction being a specific objective of the upgrade projects. The upgraded WPCCs will be required to discharge no more than 15 mg/L total Nitrogen and 1 mg/L total Phosphorous. Changing the effluent quality will change the effects of these discharges on the Red and Assiniboine Rivers. A computer model was used to simulate river conditions in order to qualitatively and quantitatively investigate changes in river water quality resulting from the proposed WPCC upgrades.

Water quality in the Red and Assiniboine Rivers was modeled using the WASP computer-modeling package, which is available from and supported by the U.S. Environmental Protection Agency. This program is used to model water quality along one or more river reaches over time for transient conditions wherein contaminant loads, flows and other parameters may change over the time period being modeled. The program can model and report on a variety of environmental parameters, however parameters considered most important for the present study are the nutrients Nitrogen (N) and Phosphorous (P), particularly the compounds Ammonia (NHx), Nitrate/Nitrite (NOx), and OrthoPhosphate (Ortho-P). Nutrients are significant relative to the growth of phytoplankton (algae), which is modeled and reported in terms of Chlorophyll-a (Chl-a) concentration. Algae dynamics, along with carbonaceous bio-chemical oxygen demands (CBOD) resulting from the effluent discharge, can cause impacts upon Dissolved Oxygen (DO) levels in the river. Concentrations of DO and NHx in the rivers are important since low DO levels can lead to fish mortality while elevated NHx levels can be chronically or acutely toxic to aquatic life.

Future river conditions, with respect to nutrients, phytoplankton and DO, were simulated for two plant-effluent scenarios based on the projected population in the year 2031, the design year for WPCC improvements. The first scenario is the "do-nothing" or without-Project condition, which assumes that plant discharges increase along with population but discharge quality remains consistent with recent historic performance of the WPCCs. The second scenario is the with-Project condition, which assumes that planned upgrades are completed, thereby reducing nutrient concentrations in the discharge streams to meet more stringent regulations. The with-Project scenario assumes completion of all proposed upgrades receiving CSIF funding and related parallel projects planned by the City of Winnipeg.

Effluent quality for the without-Project and with-Project scenarios is described in the Project Description section of this report. The difference between the scenarios is briefly described below for reference when considering the model results that are presented. Effluent quality in the without-Project scenario is based on the average monthly effluent quality reported for the three WPCCs during the years 2000-2003. With-Project effluent quality is based on results obtained from previous performance analyses of preliminary designs for WPCC upgrades. The NEWPCC and SEWPCC preliminary design analyses were completed before the license limits for nutrients were finalized. The preliminary designs for these two plants assumed a Total Nitrogen limit of 10 mg/L, which is more stringent than the 15 mg/L limit the plants will need to meet. The WEWPCC preliminary design however was based on the 15 mg/L limit. For this reason, the with-Project scenario assumes that future NEWPCC and SEWPCC effluent characteristics will be the same as the WEWPCC effluent characteristics since similar treatment trains will be employed at all three plants.

The WASP model was used to simulate river conditions for each effluent scenario in the April 1 - October 31 time period for the 2031 design-year. River conditions for these months were assumed to be the same as measured in 1988. This represents a critical period due to low flow levels and elevated temperatures, when reduced dilution of nutrient loads could favour excess algal growth and oxygen consumption. All other model parameters, including background river-loading conditions, are the same as those utilized in calibrating the model to the 1988 data. Modeling was also performed for both effluent scenarios using median river flows instead of the 1988 critical low-flow in order to assess the relative effect of the project at more typical flow levels.

The model considers the Assiniboine River from the Headingley Bridge downstream to the confluence with the Red River, and the Red River from Ste. Adolphe upstream of Winnipeg to Sugar Island just downstream of Selkirk. Three distinct reaches in terms of flow and water-

quality are the Assiniboine River, the Red River upstream of the confluence with the Assiniboine, and the Red downstream of the confluence. Effluent is discharged into each of these reaches, with the WEWPCC discharging to the Assiniboine, SEWPCC discharging to the Red upstream of the confluence, and NEWPCC discharging downstream of the confluence. Each reach is subdivided into smaller model segments representing river areas ranging from 0.5 to 2.25 km in length. There are a total of 112 model segments, with 96 on the Red and 16 on the Assiniboine. Segments are identified in a sequential, numerical order going upstream from Sugar Island to Ste. Adolphe and then continuing upstream along the Assiniboine from the Red to the Headingley Bridge. The general pattern for segment identification is 1, 1b, 2, 2b, ... 55, 55b, 56, 56b starting at Sugar Island (segment 1) and ending at Headingley Bridge (segment 56b). Some segments are identified by locations within the segment rather than numerically, such as segments covering Lockport, the confluence, or the West Perimeter Bridge.

3.0 UNDERSTANDING THE PRESENTATION OF MODEL RESULTS

The WASP program produces output files for each parameter of interest (N, P, etc.) identified in the model setup. The concentration (or other measure as appropriate) of the specific parameter is reported for each model segment for each day in the time frame being modeled. Given that there are 11 water-quality parameters of interest for 2 effluent scenarios affecting 112 model segments over a 7-month period, it should be apparent that the modeling effort produces a significant amount of output that must be considered in order to identify project effects. To facilitate a general comparison of without-Project and with-Project water-quality conditions across all segments for the time period modeled, the model results have been summarized in a series of coloured-area charts. Figure 1-1 (a & b) shows the coloured-area charts for Total Nitrogen (TN) concentrations in the without-Project and with-Project scenarios. These charts are representative of the charts used to summarize results obtained for other water quality parameters. The following describes how to interpret and understand the TN charts, but the description may be generalized and applied to the coloured-area charts for all other parameters.

In each coloured-area chart the three distinct reaches in the model have been separated for ease of identification and are labelled Assiniboine R., Red R. upstream of Assiniboine R., and Red R. downstream of Assiniboine R. Model-segment labels are shown on the right hand side of the each chart while model dates are shown across the bottom. Three horizontal white lines with the labels WEWPCC, SEWPCC and NEWPCC indicate the location of the segments in which these treatment plant discharges occur.

Each coloured-area chart depicts the range of TN concentrations (mg/L) predicted by the model for each of the 112 segments over the Apr-Oct modeling period. The legend in each chart shows the concentration range represented by each colour. For the TN charts, concentrations in the range 0-1 mg/L are indicated by light blue, 1-2 mg/L is coloured bright green, 2-3 mg/L is coloured yellow, etc. up to the number of ranges required to encompass the maximum and minimum values in the model results. The TN charts use ranges of 1 mg/L, but other parameters may have different ranges as appropriate to the data presented.

The overall spatial and temporal variation of TN concentration can be considered by visual inspection of the colour patterns for a particular scenario. Similarly, visual inspection of colour differences between the without-Project and with-Project charts provides an indication of the project effect. The charts may also be used to identify the approximate times and locations when maximum or minimum concentration values occurred. The coloured-area charts only show the range of concentrations that occurred, specific values cannot be determined. The following two examples illustrate the information that is shown in the coloured-area charts. The first considers temporal concentration changes for the Lockport segment and the second considers spatial concentration differences on October 21.

Moving horizontally (left to right) along line for the Lockport segment, the changes in colour represent changes in TN over time at this location. In the without-Project scenario (Figure 1-1a), the colour along the line is turquoise in the last part of April indicating a concentration of 3-4 mg/L, changing to the 2-3 mg/L range for most of May based on the yellow colour, and then changing to the 1-2 mg/L range for most of June where the colour is bright green. In October, the Lockport TN concentration is 4-5 mg/L at the beginning of the month and increases to 7-8 mg/L at the end of the month, as indicated by the colour change from dark purple to light blue.

By contrast, in the with-Project scenario (Figure 1-1b), the colouring along the Lockport segment line is yellow in the last part of April, indicating a concentration of 2-3 mg/L, changing to bright green for May and most of June, indicating a concentration of 1-2 mg/L. The concentration in October increases from 1-2 mg/L at the beginning of the month to 2-3 mg/L at the end of the month based on a colour change from bright green to yellow. For the Lockport segment, the project reduces the late April concentration from 3-4 mg/L to 2-3 mg/L and reduces May concentrations from 2-3 mg/L to 1-2 mg/L. Concentrations for most of June are 1-2 mg/L in both scenarios, but these charts do not identify which scenario has the lower concentration in this range. October concentrations are substantially reduced in the with-Project scenario.

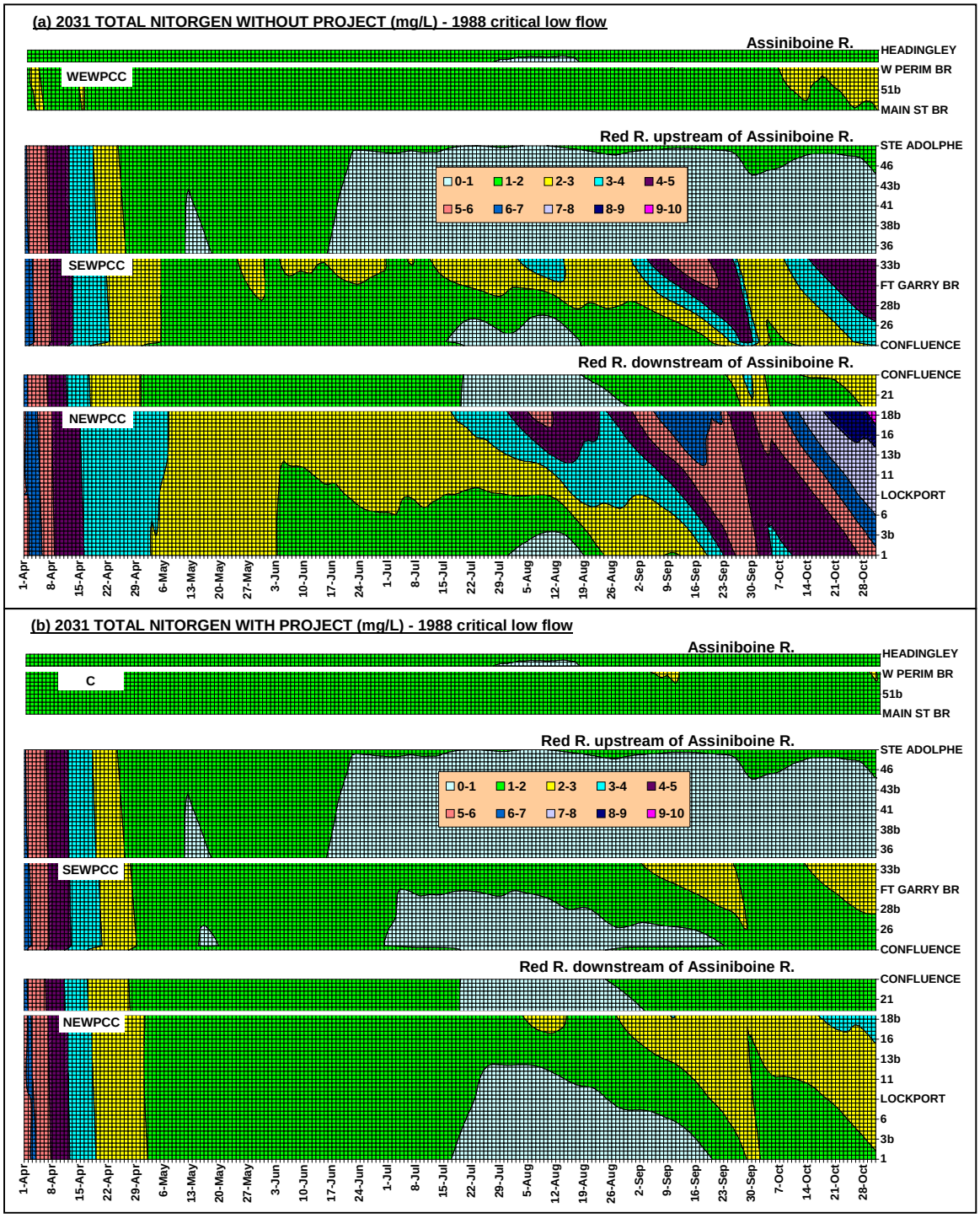


Figure 1-1: Representative Coloured-Area Charts for (a) Without-Project and (b) With-Project

In the without-Project scenario (Figure 1-1a), colours along the vertical line for October 21 show that TN concentrations are in the 1-2 mg/L range at Headingley, the 2-3 mg/L range downstream of the WEWPCC outfall, and in the 1-2 mg/L range at the Main St. Bridge immediately upstream of the confluence. Similarly, TN concentrations along the Red R. are in the 0-1 mg/L range for segments between Ste. Adolphe and the SEWPCC outfall, but jumps to the 4-5 mg/L range immediately downstream of SEWPCC. Moving downstream from SEWPCC the TN level declines until it is in the 1-2 mg/L range just upstream of the NEWPCC outfall. The concentration jumps to the 8-9 mg/L range immediately downstream of the NEWPCC but decreases in the downstream direction and is in the 4-5 mg/L range at segment 1, the downstream end of the modeled area. The October 21 results for the with-Project scenario (Figure 1-1b) show that the project significantly reduces TN concentrations immediately downstream of the WPCC's. Concentrations are reduced from 2-3 mg/L to 1-2 mg/L at WEWPCC, from 4-5 mg/L to 2-3 mg/L at SEWPCC, and from 8-9 mg/L to 3-4 mg/L at NEWPCC. Concentrations in segments downstream of the WPCCs are likewise reduced. Both sets of coloured-area charts show abrupt changes in concentration (i.e., colour) between the segments immediately upstream and downstream of each WPCC discharge point, which shows the effect of effluent loading on river water-quality at these locations.

The information presented in the coloured-area charts for TN has been highlighted through the consideration of a specific segment (Lockport) and a specific day (October 21). The examples also show how model results for the without-Project and with-Project scenarios may be compared to get an indication of project effects. While specific segments or dates can be considered, the coloured-area charts are most useful for investigating overall patterns of concentration changes in space and time, both within and between scenarios.

For example, colour variations in both scenarios show that TN concentrations, in general, are high in early April, low from about May to September, and increase from September to October. Additionally, peak TN concentrations occur immediately downstream of the WPCCs and decline downstream of the WPCCs. Comparing the charts for the two scenarios, it is apparent that the with-Project scenario has much more area covered by the colours light turquoise, bright green, and yellow (i.e., concentration ranges of 0-1, 1-2 and 2-3 mg/L). This is particularly evident downstream of NEWPCC (segment 18b downstream to segment 1) from about August through October. The differences in colouring between the two scenarios readily show the effect of the project on these segments in this time frame. The coloured-area charts quickly show concentration variations without having to consider results in a cumbersome segment-by-segment or day-by-day manner.

What the coloured-area charts do not show, however, is the specific values of model results, either for a single segment over time, or across all segments on a specific day. In order to consider the actual results obtained from the model, the temporal variation of TN for a specific segment and spatial variation of TN on a specific day can be plotted using typical line-charts. Model results for the Lockport segment and the October 21st date are presented in line-charts in Figure 1-2 (a & b). Separate lines show the model results obtained in the without-Project and with-Project scenarios.

Recall from consideration of the Lockport segment using the coloured-area charts that TN concentrations were in the 1-2 mg/L range for much of June in both scenarios, but the scenario with the higher or lower value could not be determined. The line-chart for the Lockport segment (Figure 1-2a) shows that the with-Project results are lower by about 0.75 mg/L in this period. Additionally, this line-chart clearly shows the significant effect of the project in reducing TN concentrations from about August through October. This general effect was evident from the overall comparison of colour differences between the without-Project and with-Project coloured-area charts.

Figure 1-2b shows the TN concentrations at each model segment on the October 21 modeling date. The three distinct river reaches are indicated and segment labels along the bottom of the chart indicate a downstream progression along the river reaches from left to right. Segments in which the WPCCs discharge are also indicated. This line-chart clearly shows the jump in TN concentration between model segments immediately upstream and downstream of the WPCC discharges, as was noted based on colour changes in the coloured-area charts. The line-chart also shows that the project reduces TN concentrations downstream of the WPCCs, with the biggest reduction occurring downstream of NEWPCC.

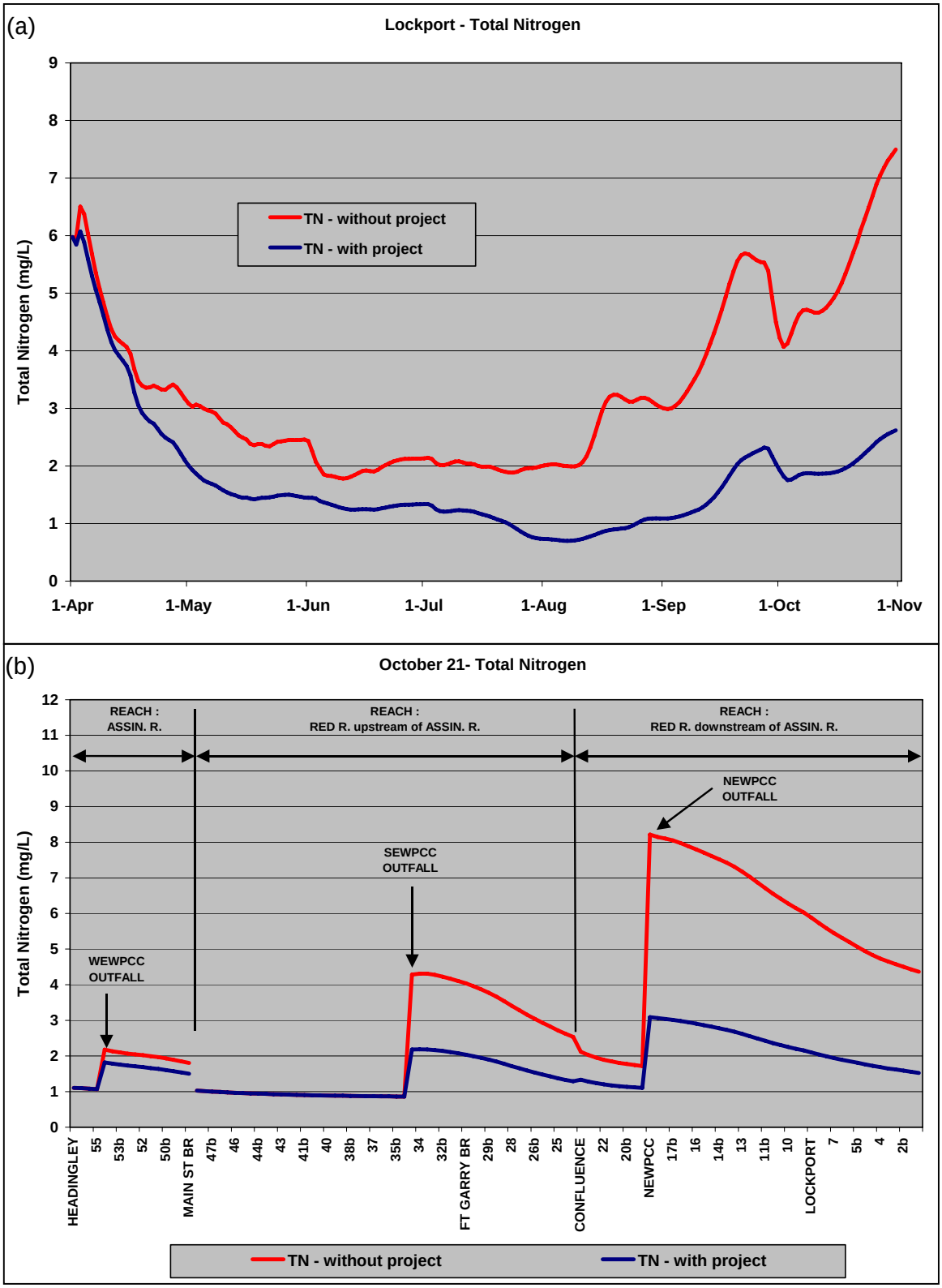


Figure 1-2: Representative Line Charts for (a) Specific Segment and (b) Specific Day

The preceding discussion has described two methods used to summarize and display results obtained from the water-quality for the Red and Assiniboine rivers. Coloured-area charts are used to present overall water-quality conditions across all segments in the time period modeled. By comparing differences between the without-Project and with-Project charts, the general, overall effects of the project can be identified. While coloured-area charts conveniently show overall conditions, line-charts are used to show specific differences between the without-Project and with-Project model results for a specific segment or date. These two types of charts are complementary and are used together for a more complete assessment of project effects. For example, locations or dates of interest due to high concentrations may be identified using coloured-area charts, while specific results for the location or date of interest may be considered using line-charts. Both types of charts are used in the assessment of project effects because each has particular strengths in conveying information about water-quality changes.

4.0 LIST OF MODEL RESULTS PRESENTED

While the WASP model produces output for a number of water-quality parameters, the graphic results presented in this appendix are limited to those considered to be of greatest significance relative to the effects of proposed WPCC upgrades. Figures showing compliance with in stream ammonia criteria and percent saturation for minimum DO levels are also provided. These results are not reported by WASP but are calculated based on model output. Charts for the without-Project and with-Project model results under critical low-flow conditions (i.e., 1988 flows) are presented first, followed by model results for both effluent scenarios under the more typical median flow conditions.

Without-Project and with-Project coloured-area charts are provided in Attachment 8C-A for the following water quality parameters (in order of presentation):

- Total Phosphorous (TP, mg/L)
- Ortho-Phosphate (Ortho-P, mg/L)
- Total Nitrogen (TN, mg/L)
- Ammonia (NHx, mg/L)
- Ammonia Compliance
- Nitrate/Nitrite (NOx, mg/L)
- Ammonia + Nitrate/Nitrite (NHx+NOx, , mg/L)
- Chlorophyll-a (Chl-a, ug/L)
- Carbonaceous Biochemical Oxygen Demand – ultimate (CBOD, mg/L)
- Dissolved Oxygen – minimum (DOmin, mg/L)

- DOmin Percent Saturation (%DOmin, %)

Line charts are also presented to show the time series results for three segments downstream of the three treatment plants. These show specific conditions from the without-Project and with-Project scenarios for segments affected by the different points of effluent discharge. Results are shown for the Fort Garry Bridge segment downstream of SEWPCC, segment 52 (just downstream of Assiniboine Park) downstream of WEWPCC, and the Lockport segment downstream of NEWPCC and the other two plants. Time series data for the following parameters are presented:

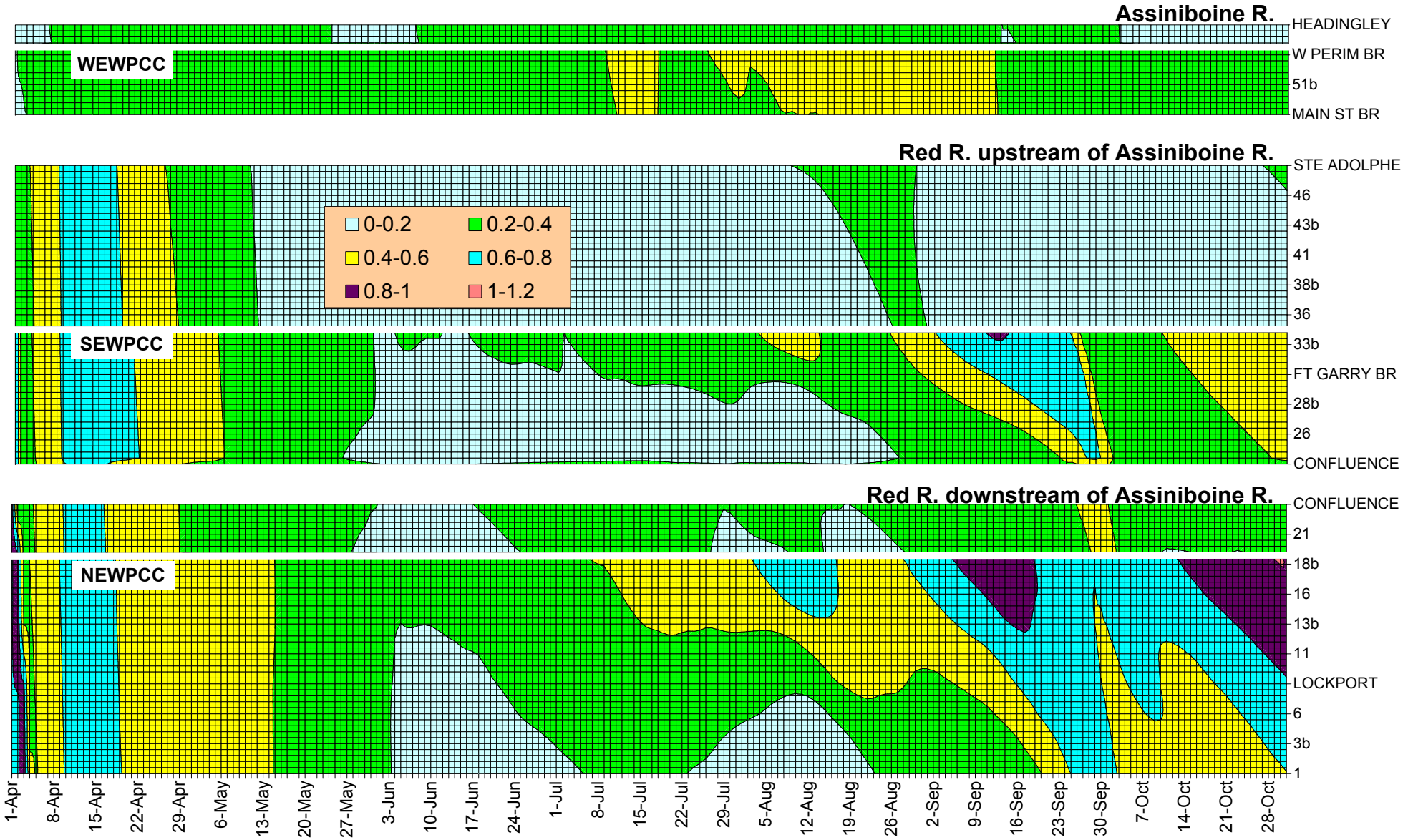
- Total Phosphorous (TP, mg/L)
- Total Nitrogen (TN, mg/L)
- Ortho-Phosphate (Ortho-P, mg/L)
- Ammonia + Nitrate/Nitrite (NHx+NOx, mg/L)
- Ammonia (NHx, mg/L)
- Chlorophyll-a (Chl-a, ug/L)
- Carbonaceous Biochemical Oxygen Demand – ultimate (CBOD, mg/L)
- Dissolved Oxygen – minimum (DOmin, mg/L)

Lastly, time series data are also presented showing the without-Project and with-Project Ammonia (NHx) concentrations for the three model segments in which the WPCC's discharge. These are the segments with the highest NHx concentrations, representing to locations with the greatest likelihood of exceeding NHx compliance limits. These charts include a line showing the maximum allowable NHx concentration in each segment.

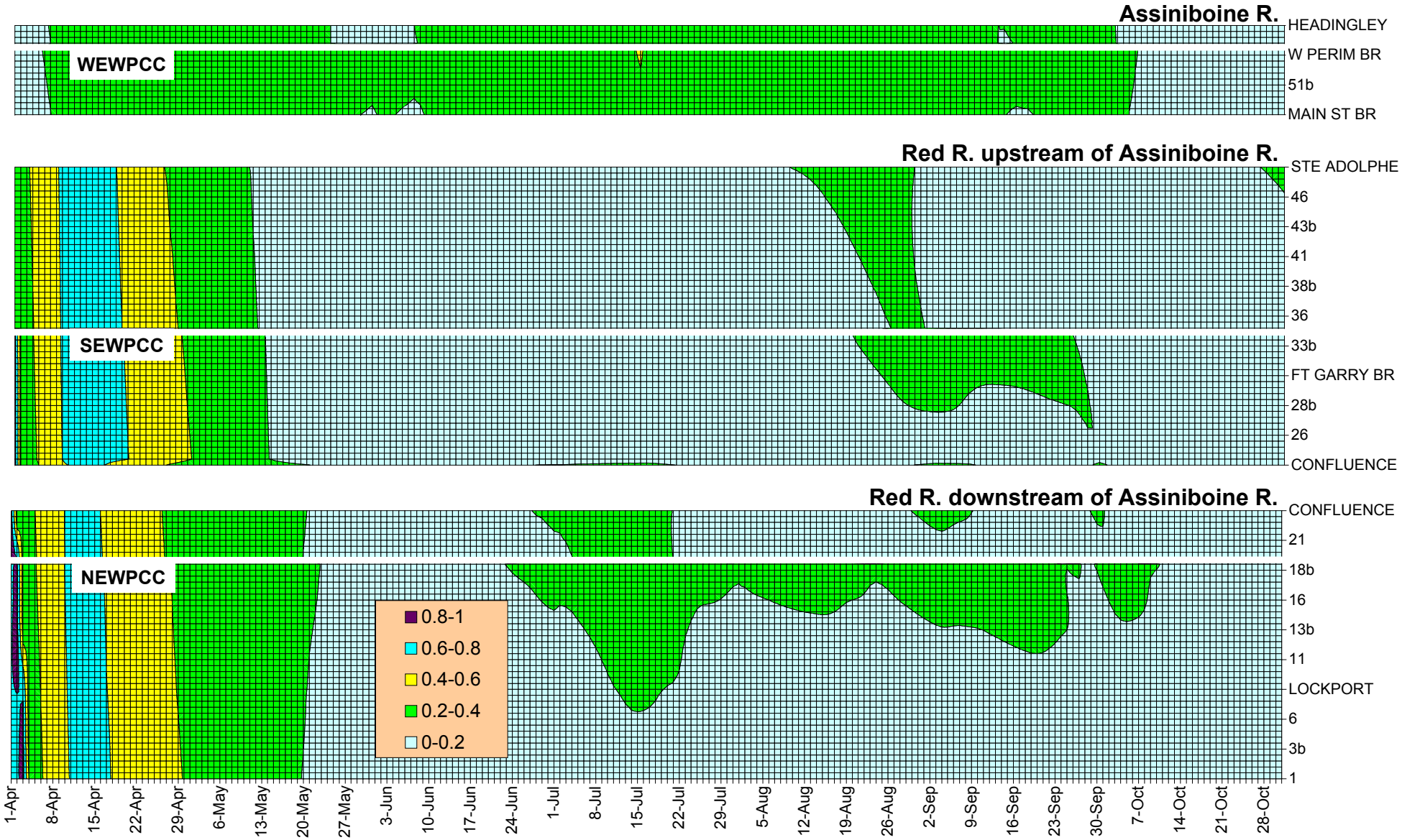
Attachment 8C-A

Without-Project and With-Project Coloured Area Charts

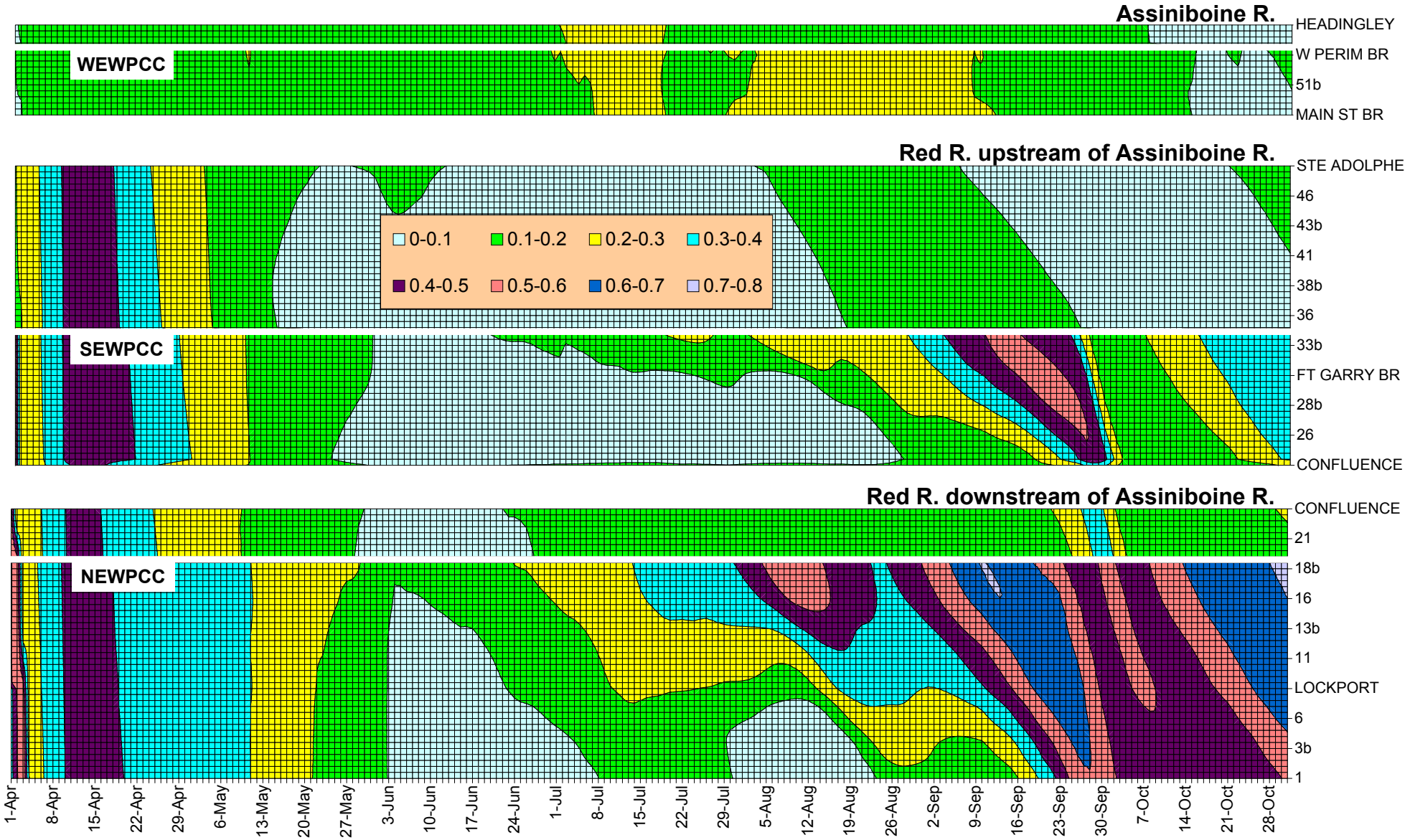
2031 TOTAL PHOSPHOROUS WITHOUT PROJECT (mg/L) - 1988 critical low flow



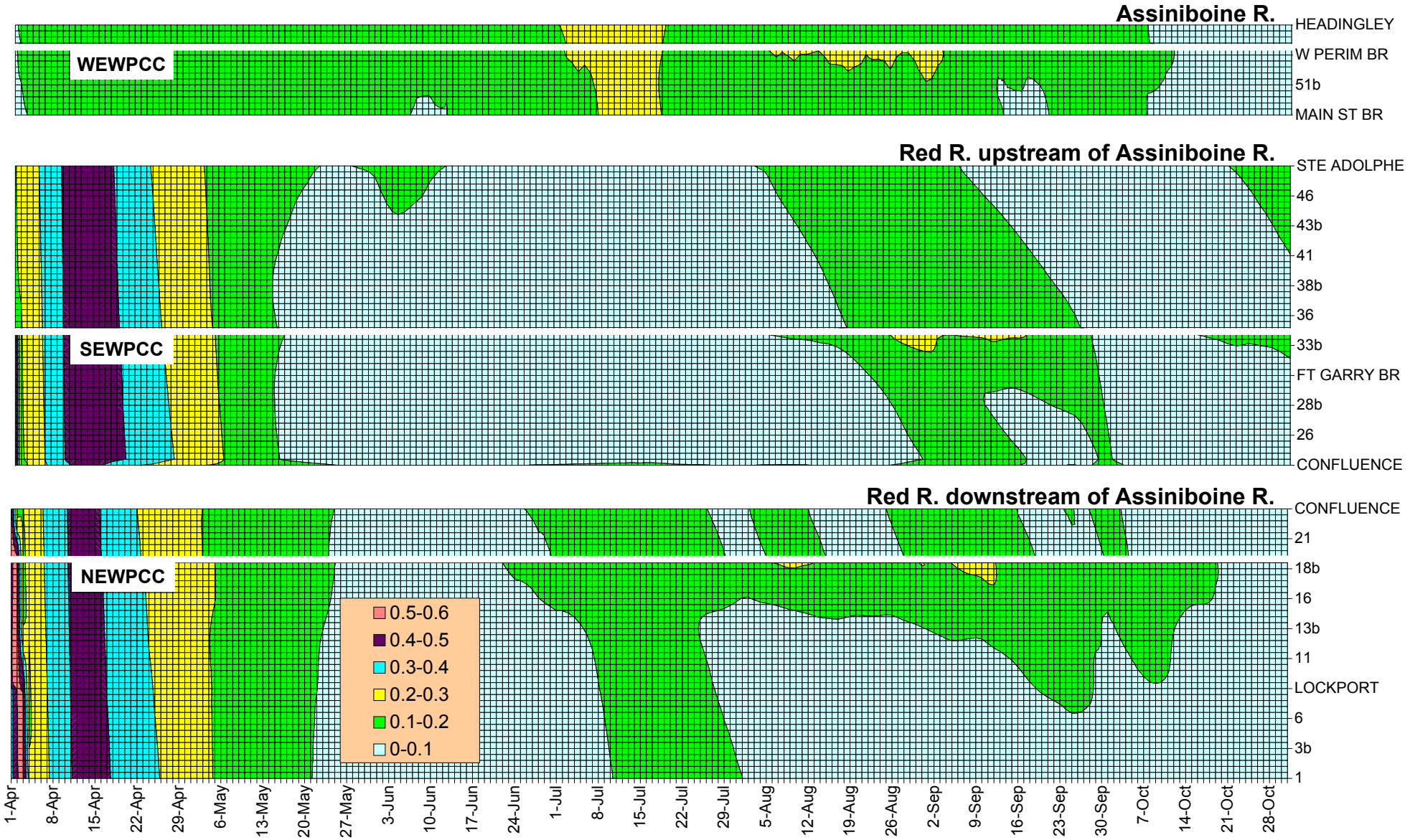
2031 TOTAL PHOSPHOROUS WITH PROJECT (mg/L) - 1988 critical low flow



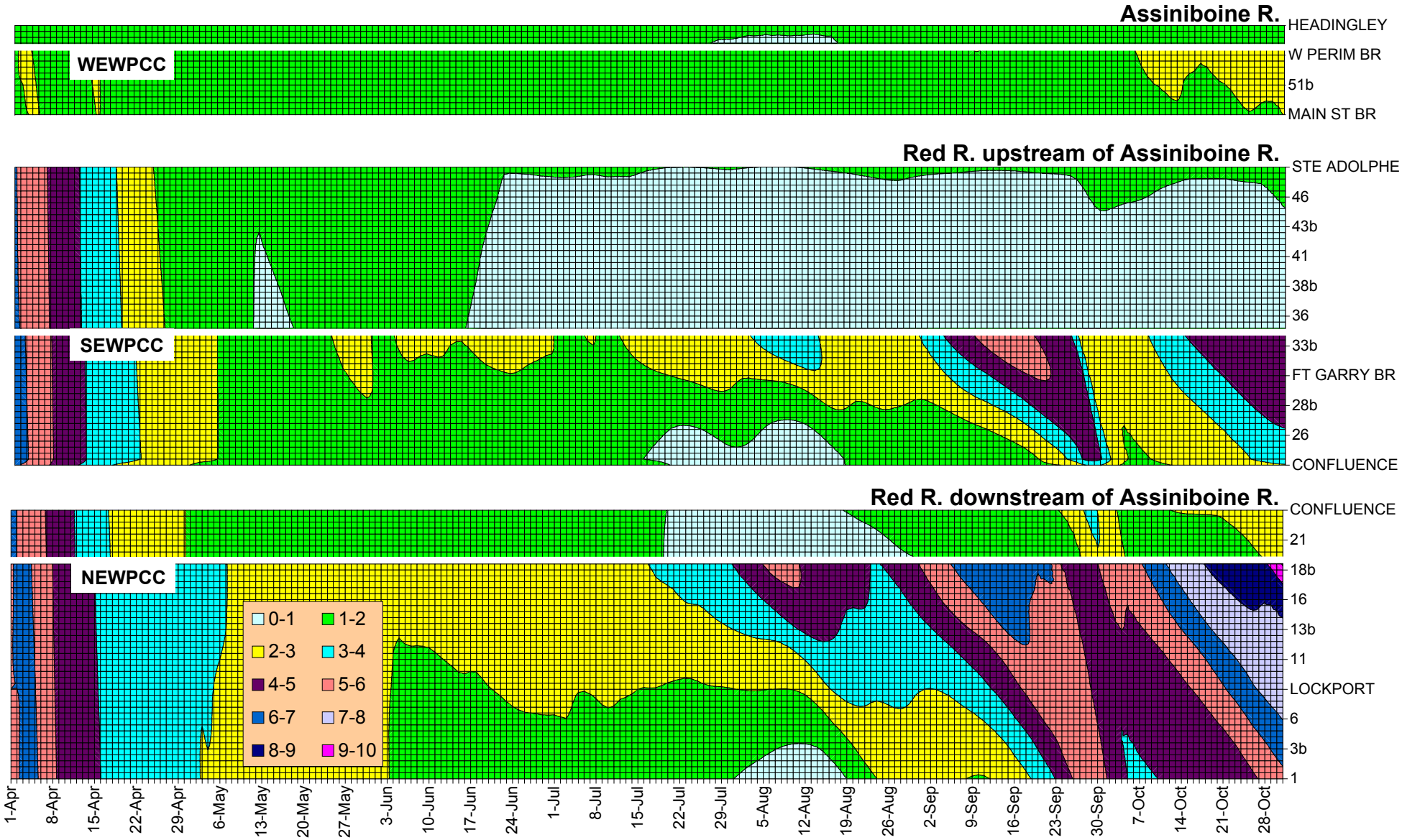
2031 ORTHOPHOSPHATE WITHOUT PROJECT (mg/L) - 1988 critical low flow



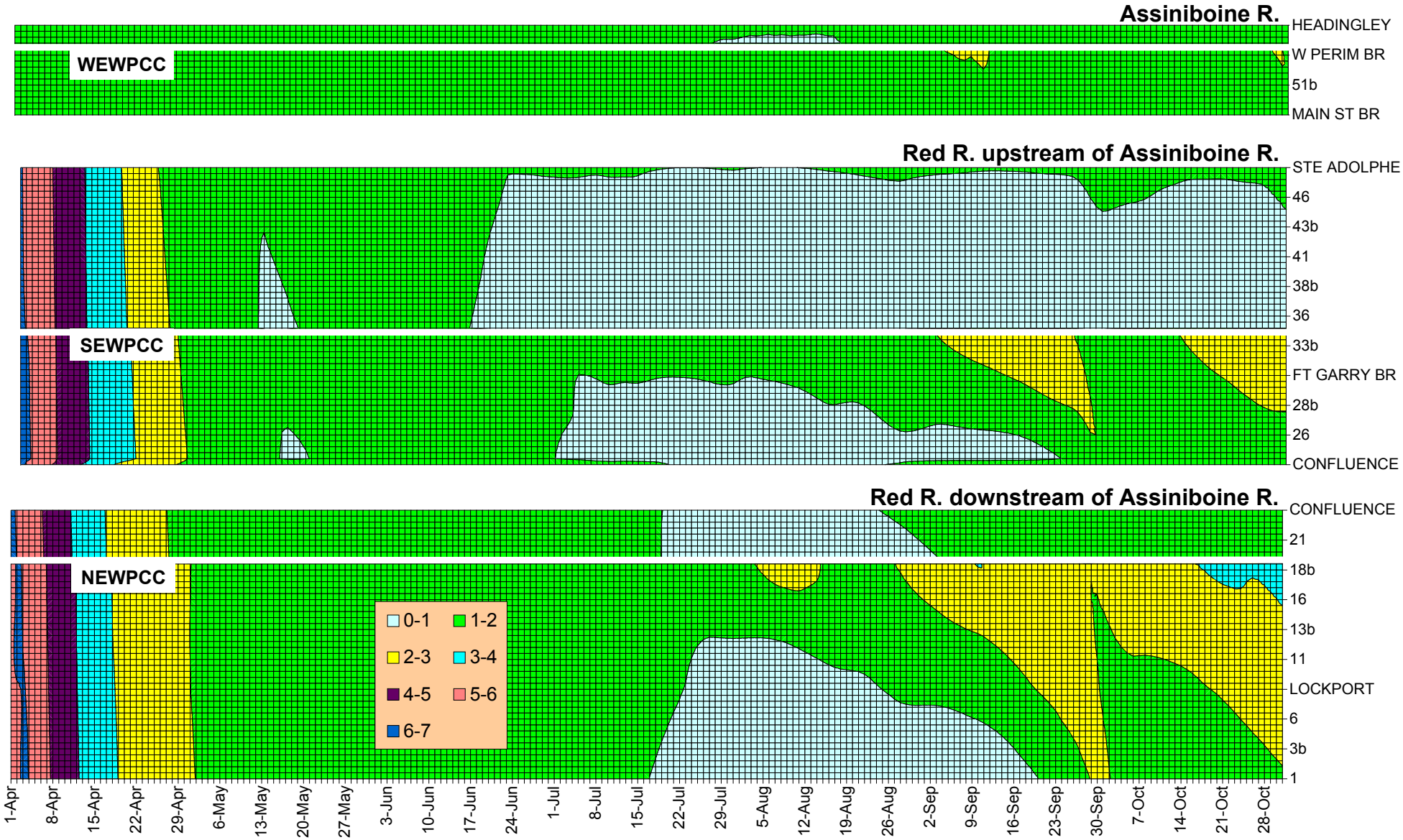
2031 ORTHOPHOSPHATE WITH PROJECT (mg/L) - 1988 critical low flow



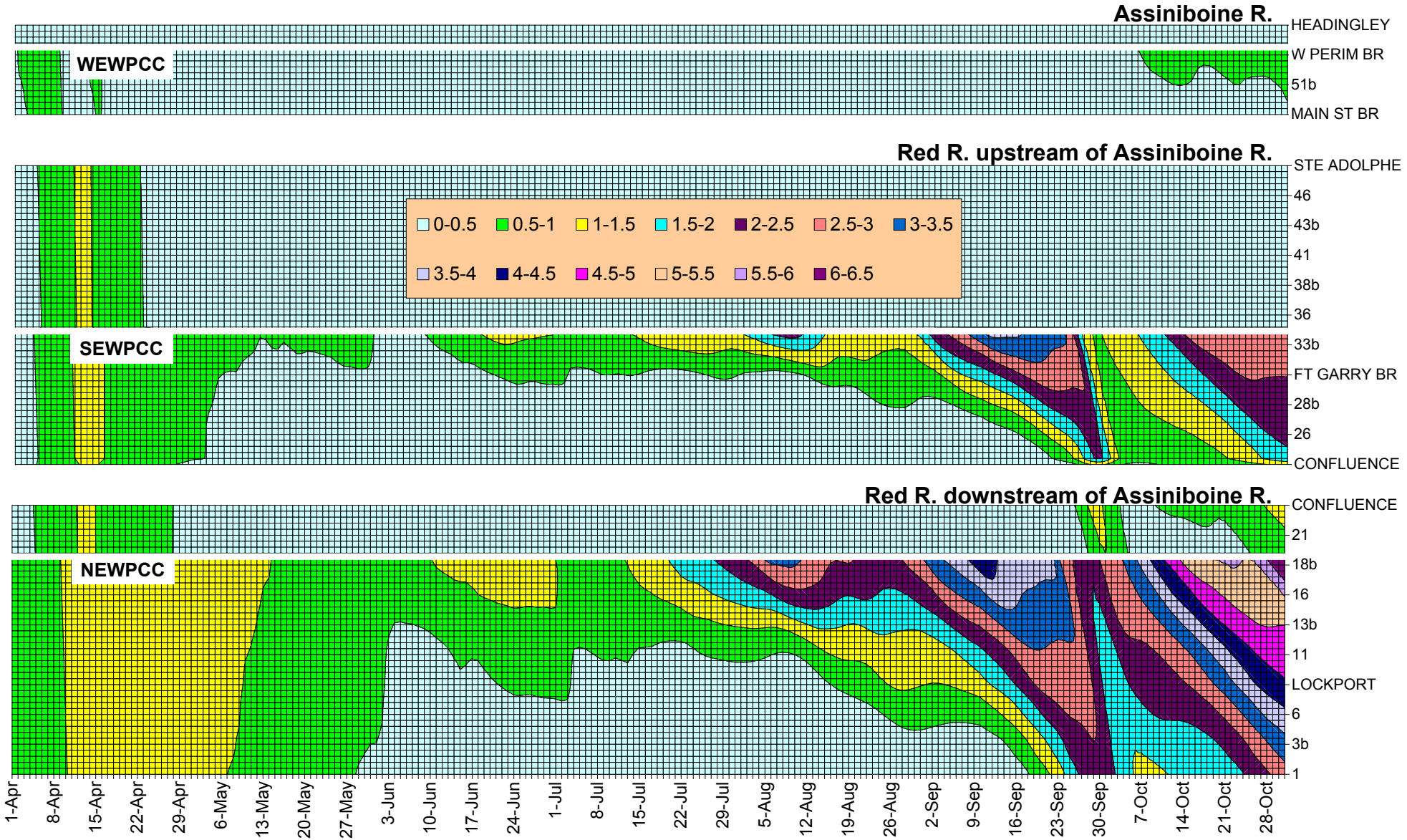
2031 TOTAL NITORGEN WITHOUT PROJECT (mg/L) - 1988 critical low flow



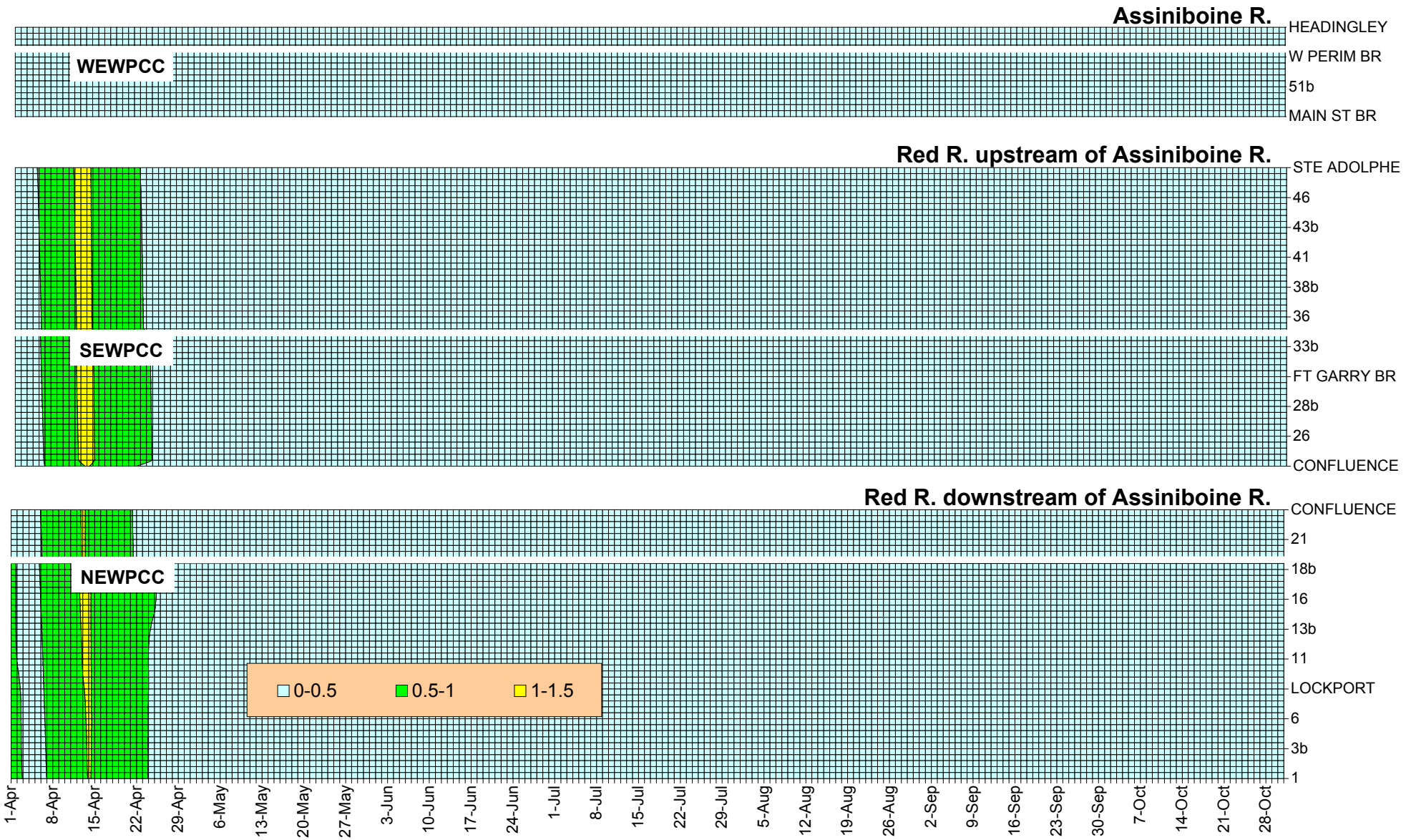
2031 TOTAL NITROGEN WITH PROJECT (mg/L) - 1988 critical low flow



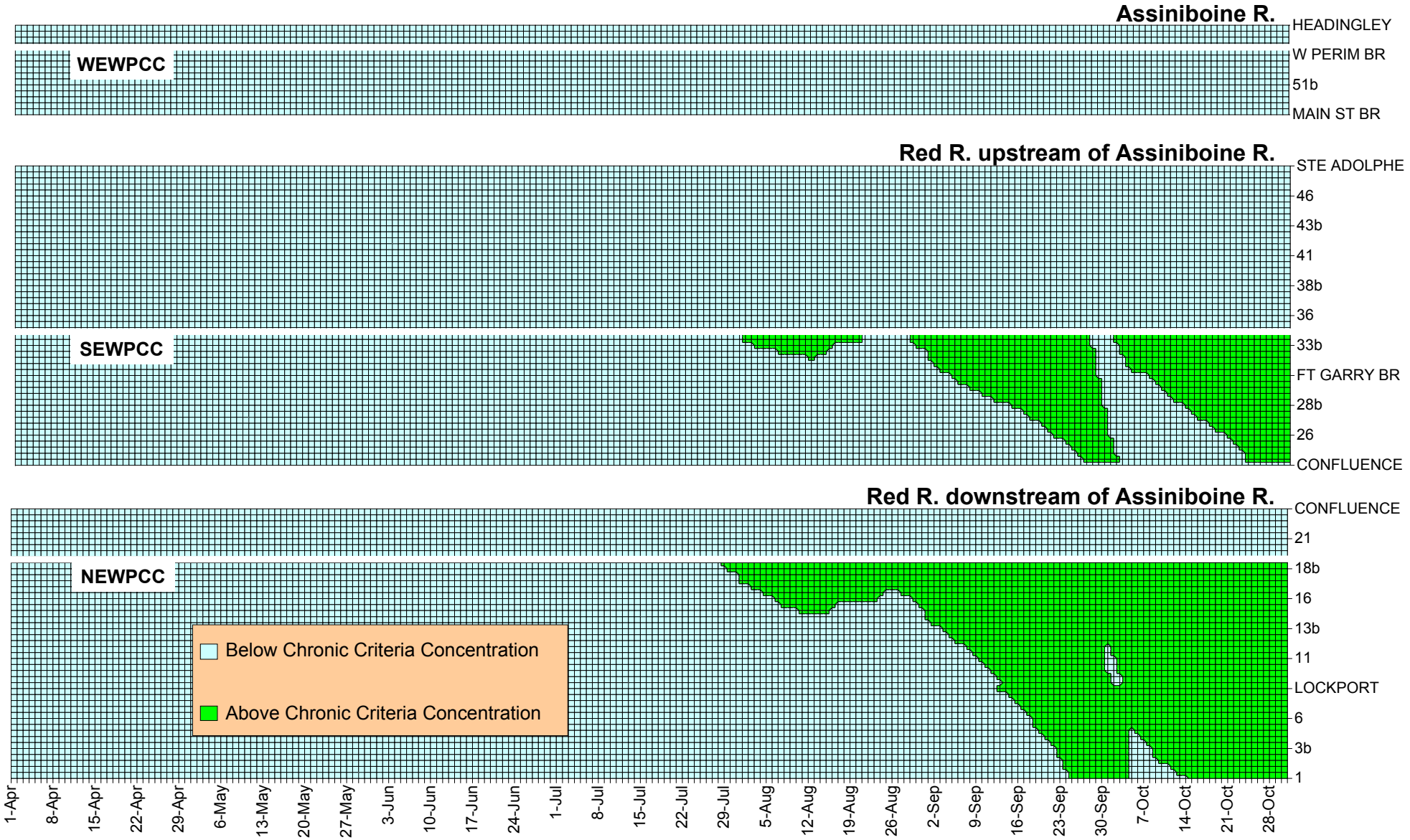
2031 AMMONIA WITHOUT PROJECT (mg/L) - 1988 critical low flow



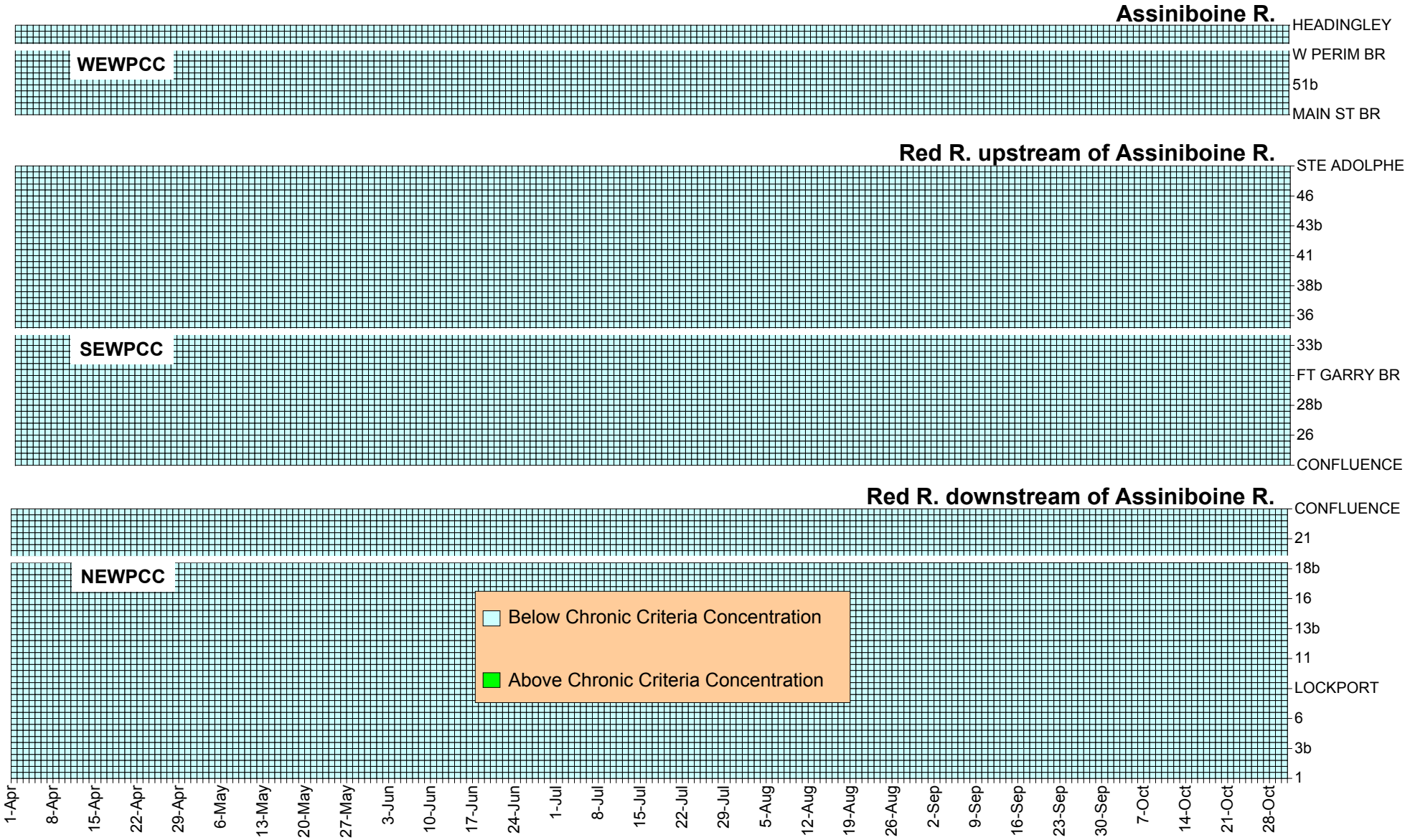
2031 AMMONIA WITH PROJECT (mg/L) - 1988 critical low flow



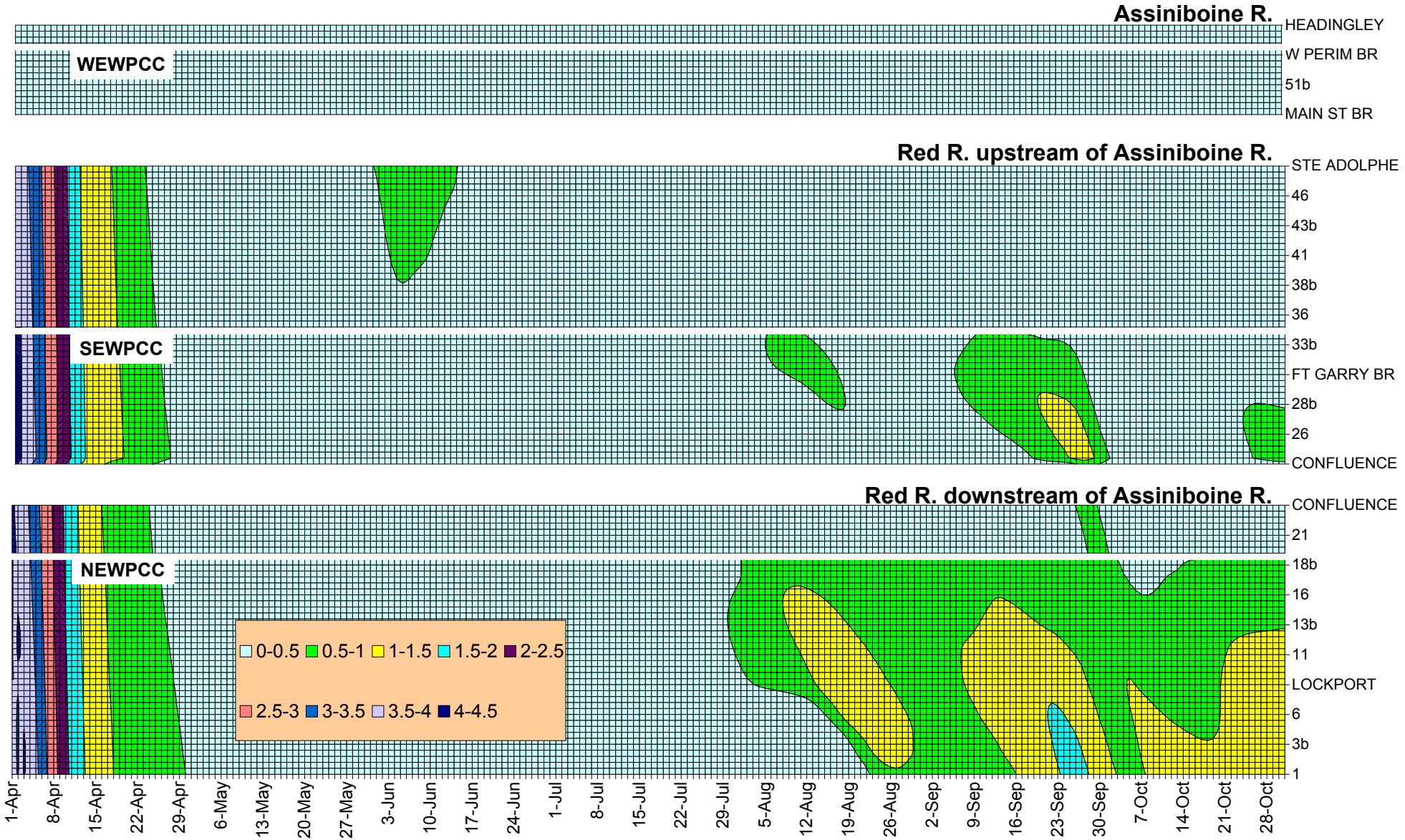
2031 AMMONIA CRITERIA COMPLIANCE WITHOUT PROJECT - 1988 critical low flow



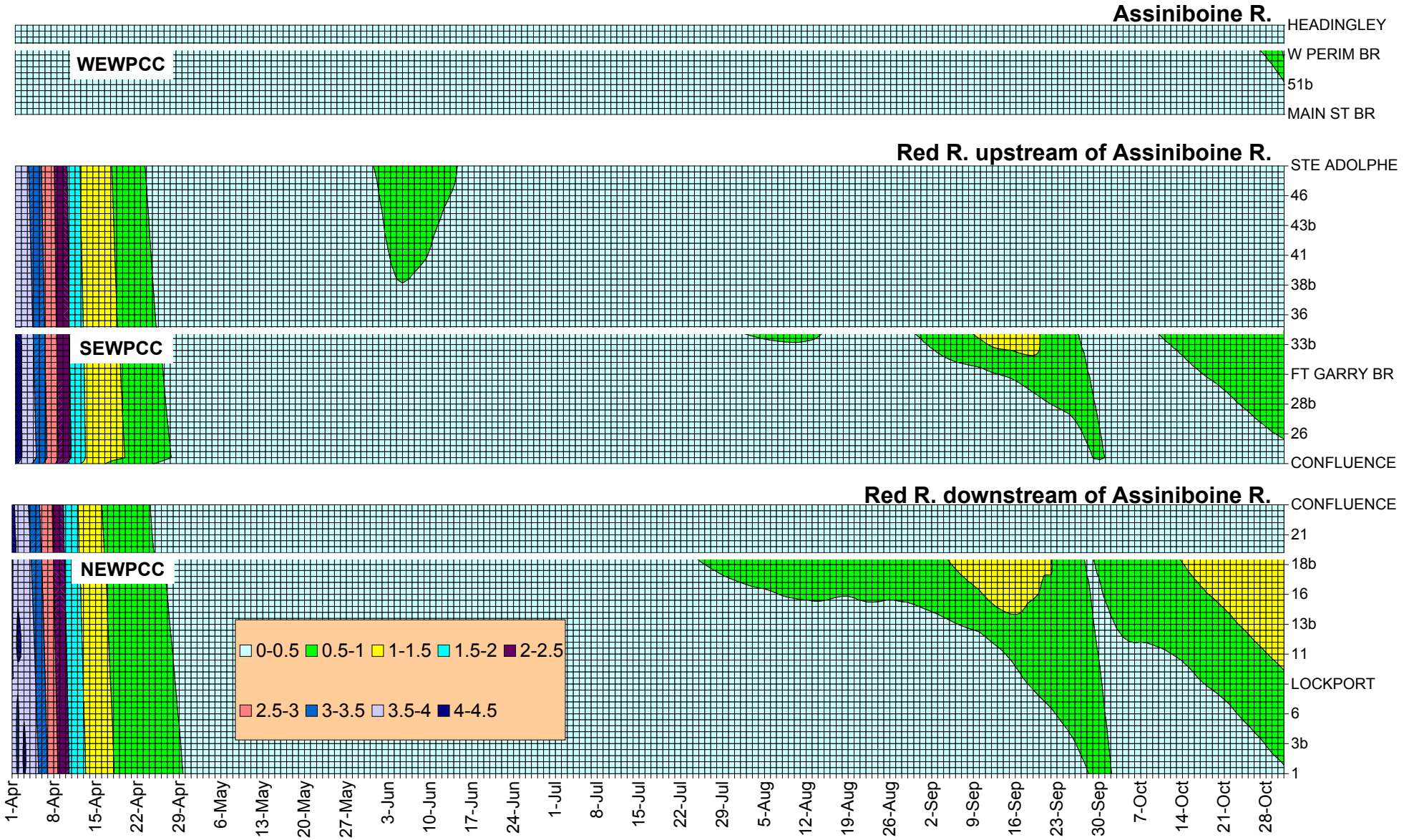
2031 AMMONIA CRITERIA COMPLIANCE WITH PROJECT - 1988 critical low flow



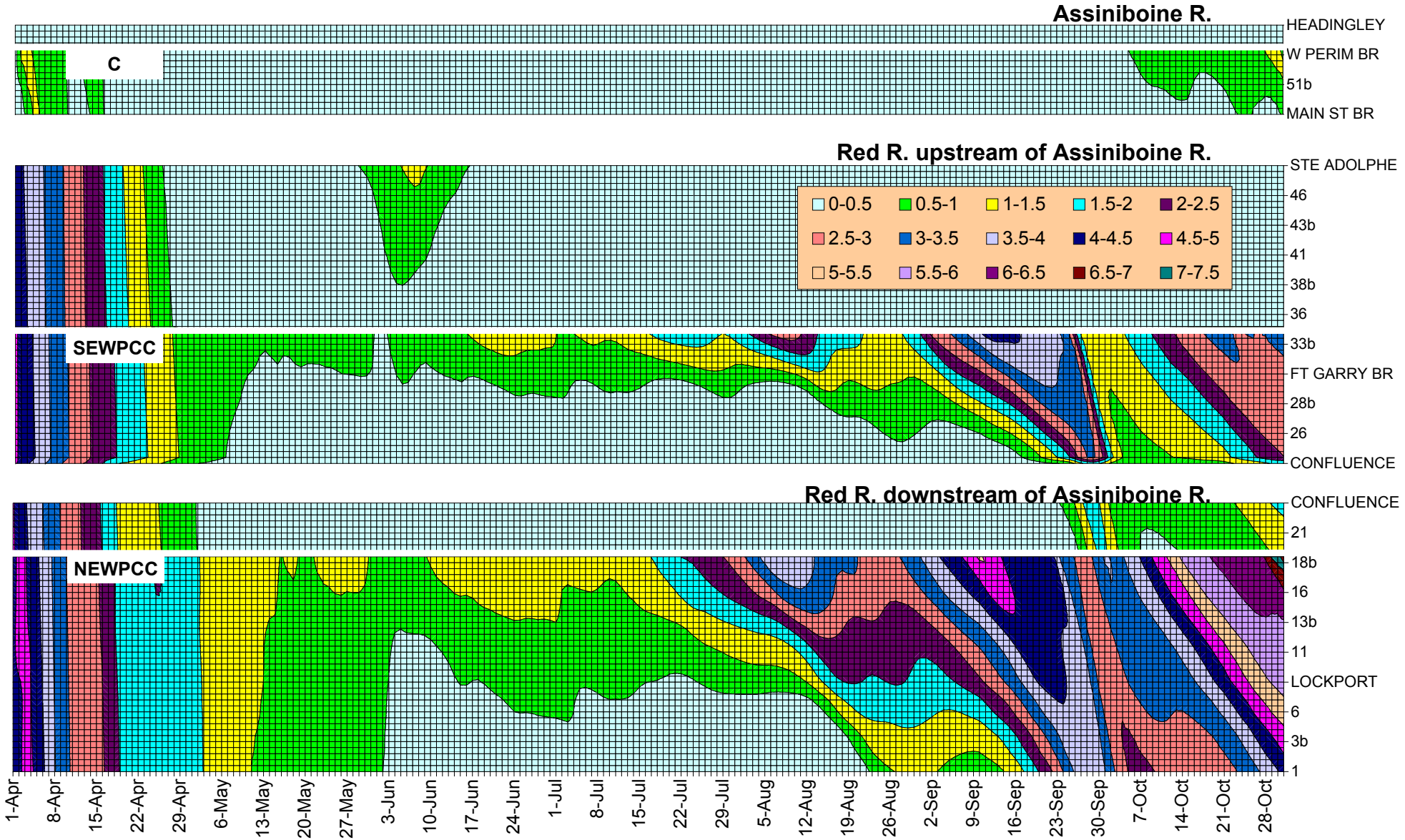
2031 NITRATE/NITRITE WITHOUT PROJECT (mg/L) - 1988 critical low flow



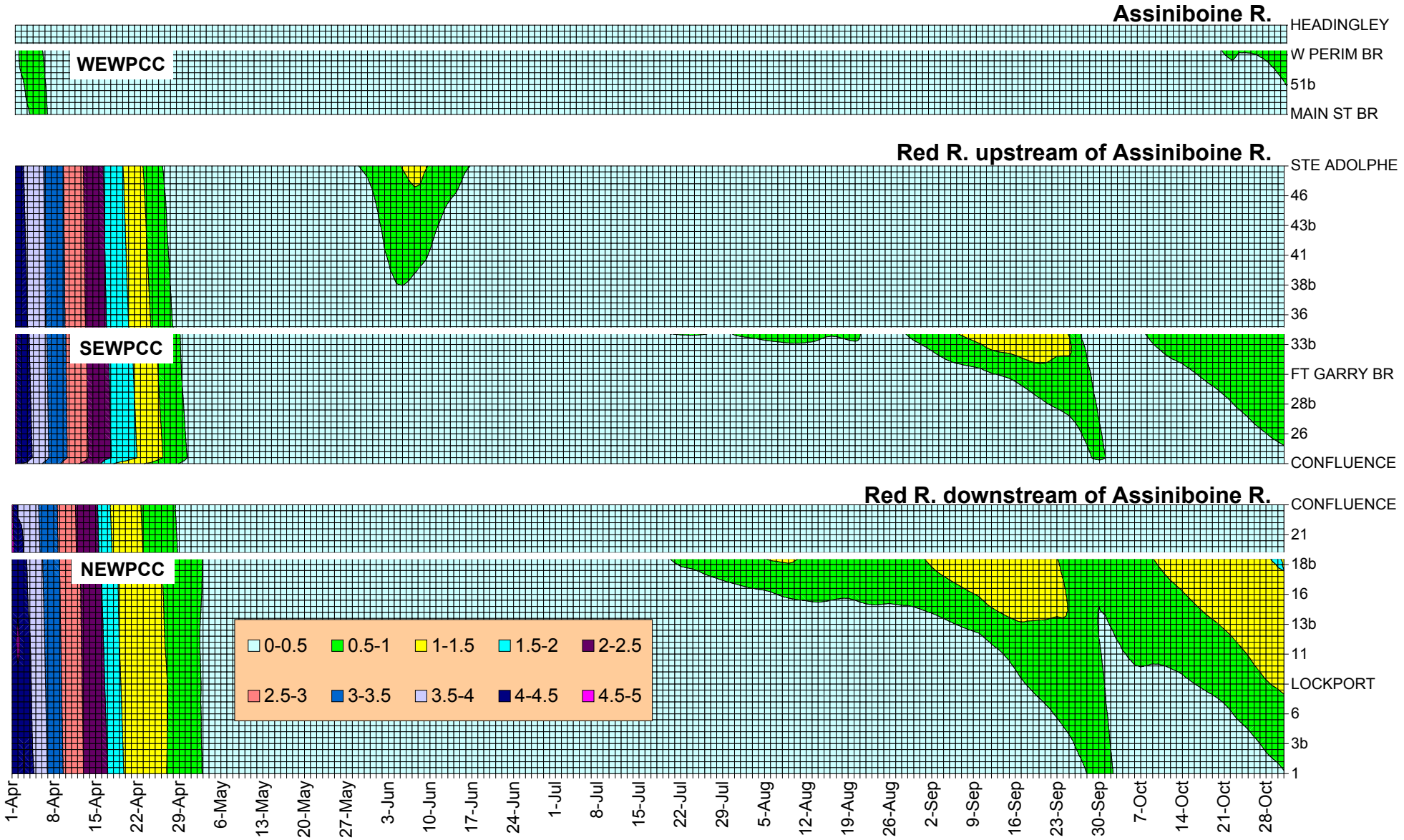
2031 NITRATE/NITRITE WITH PROJECT (mg/L) - 1988 critical low flow



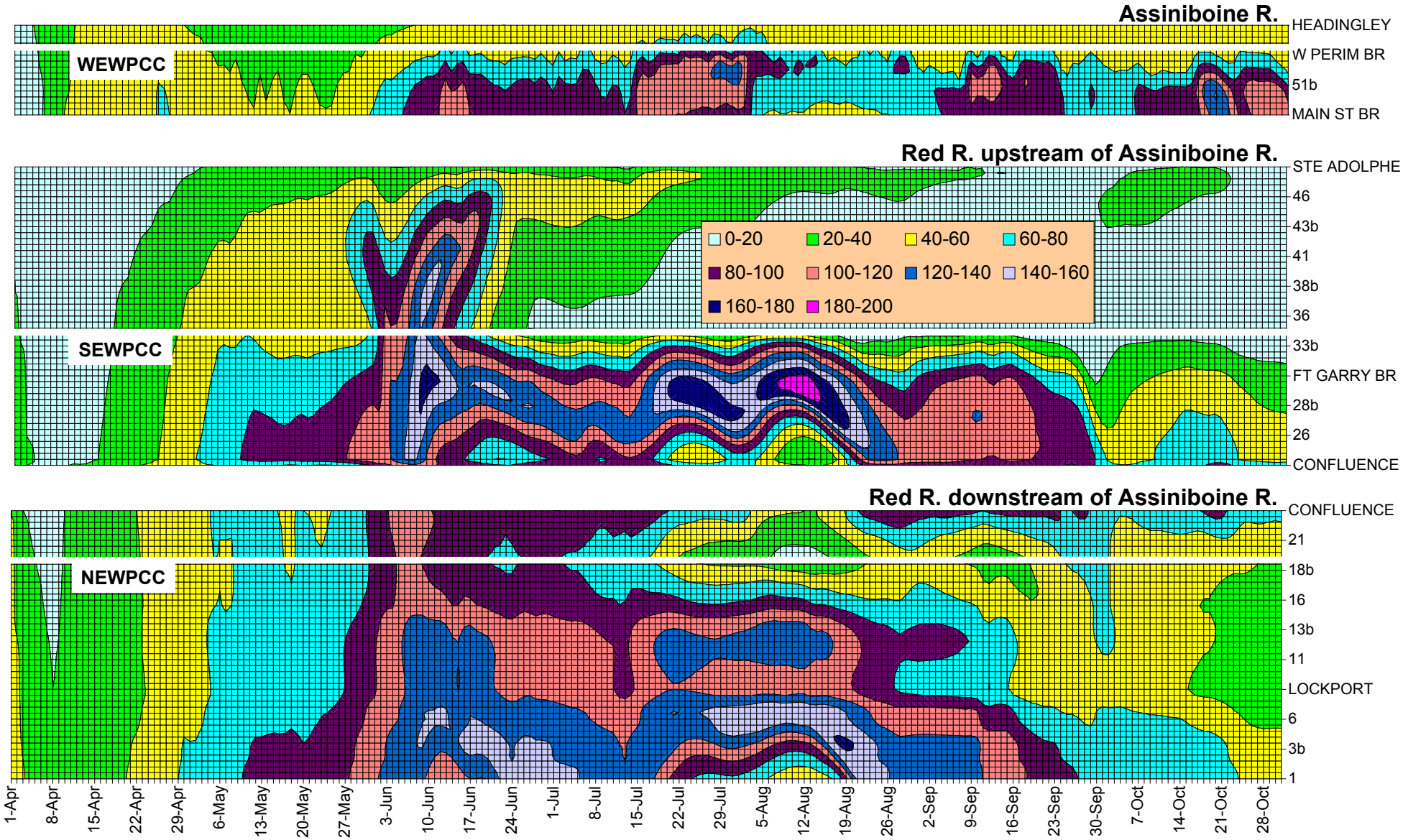
2031 AMMONIA + NITRATE/NITRITE WITHOUT PROJECT (mg/L) - 1988 critical low flow



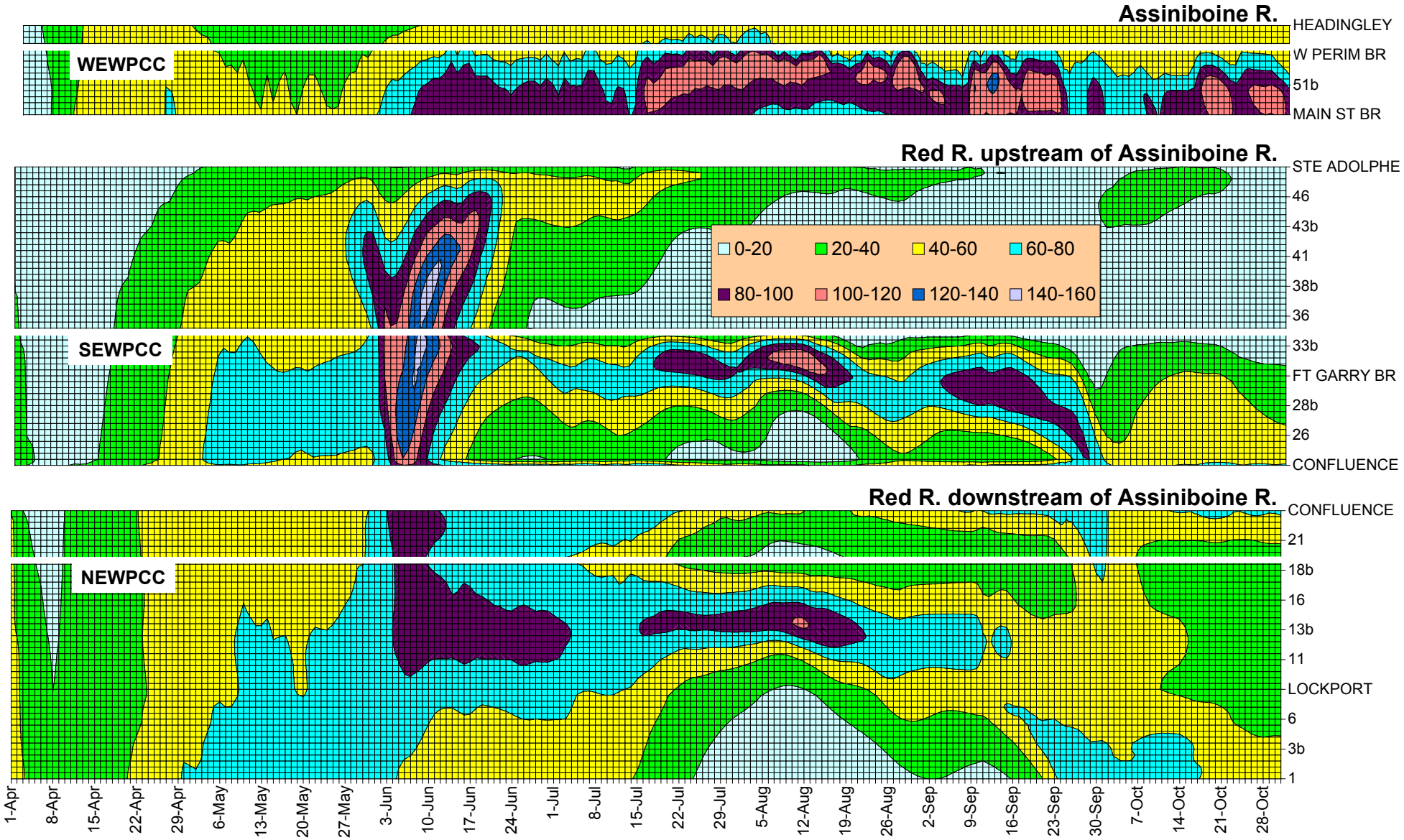
2031 AMMONIA + NITRATE/NITRITE WITH PROJECT (mg/L) - 1988 critical low flow



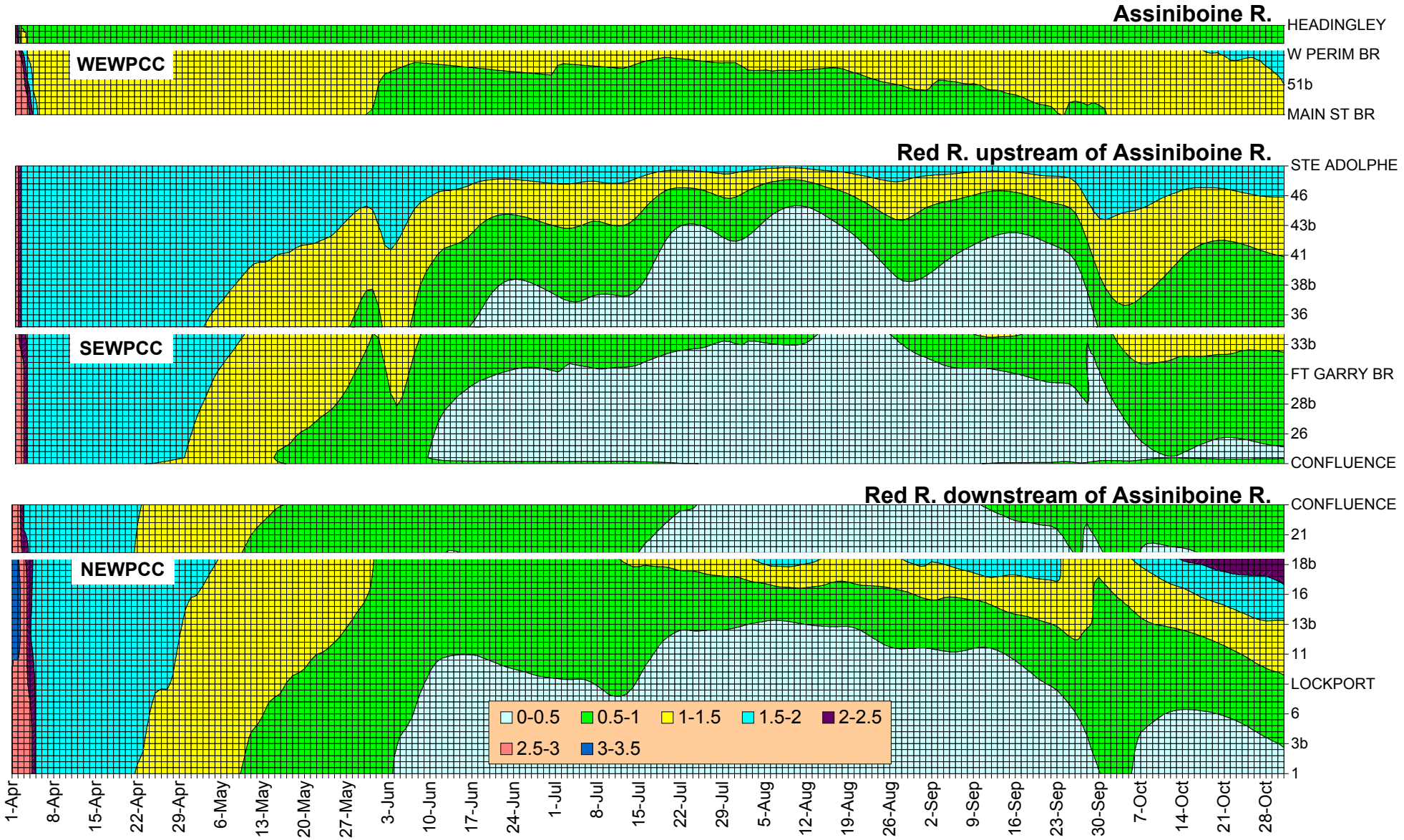
2031 CHLOROPHYLL-a WITHOUT PROJECT (ug/L) - 1988 critical low flow



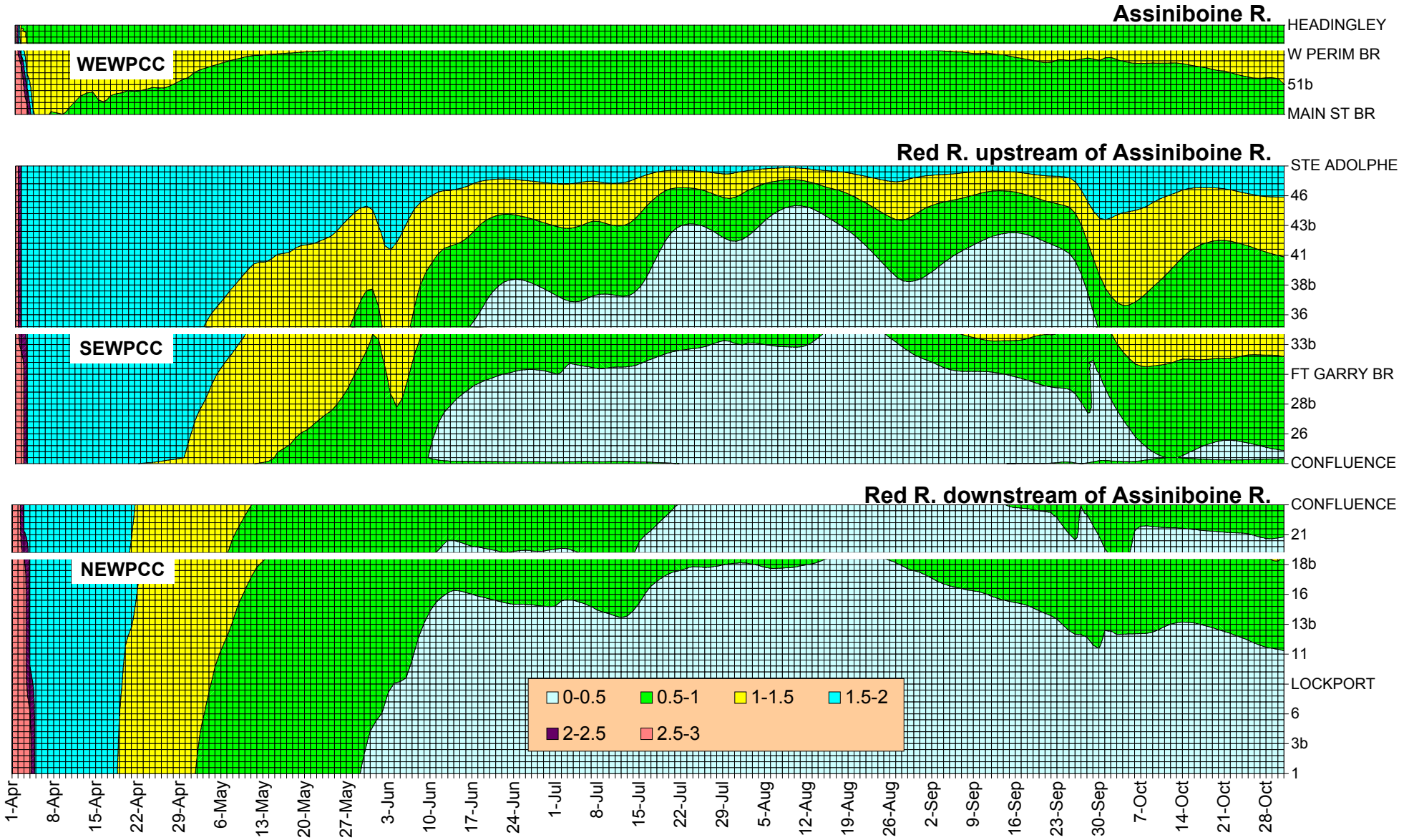
2031 CHLOROPHYLL-a WITH PROJECT (ug/L) - 1988 critical low flow



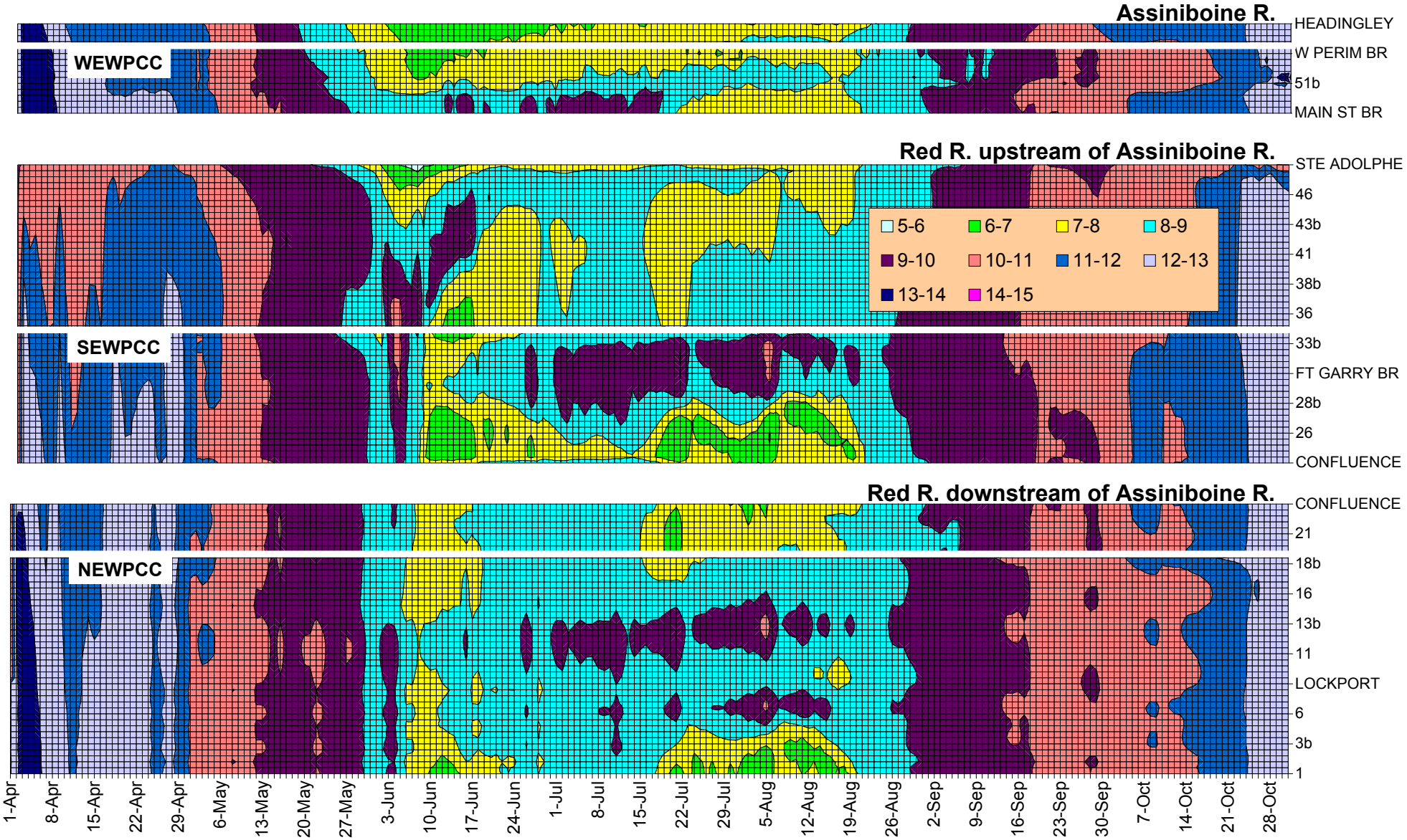
2031 CBOD WITHOUT PROJECT (mg/L) - 1988 critical low flow



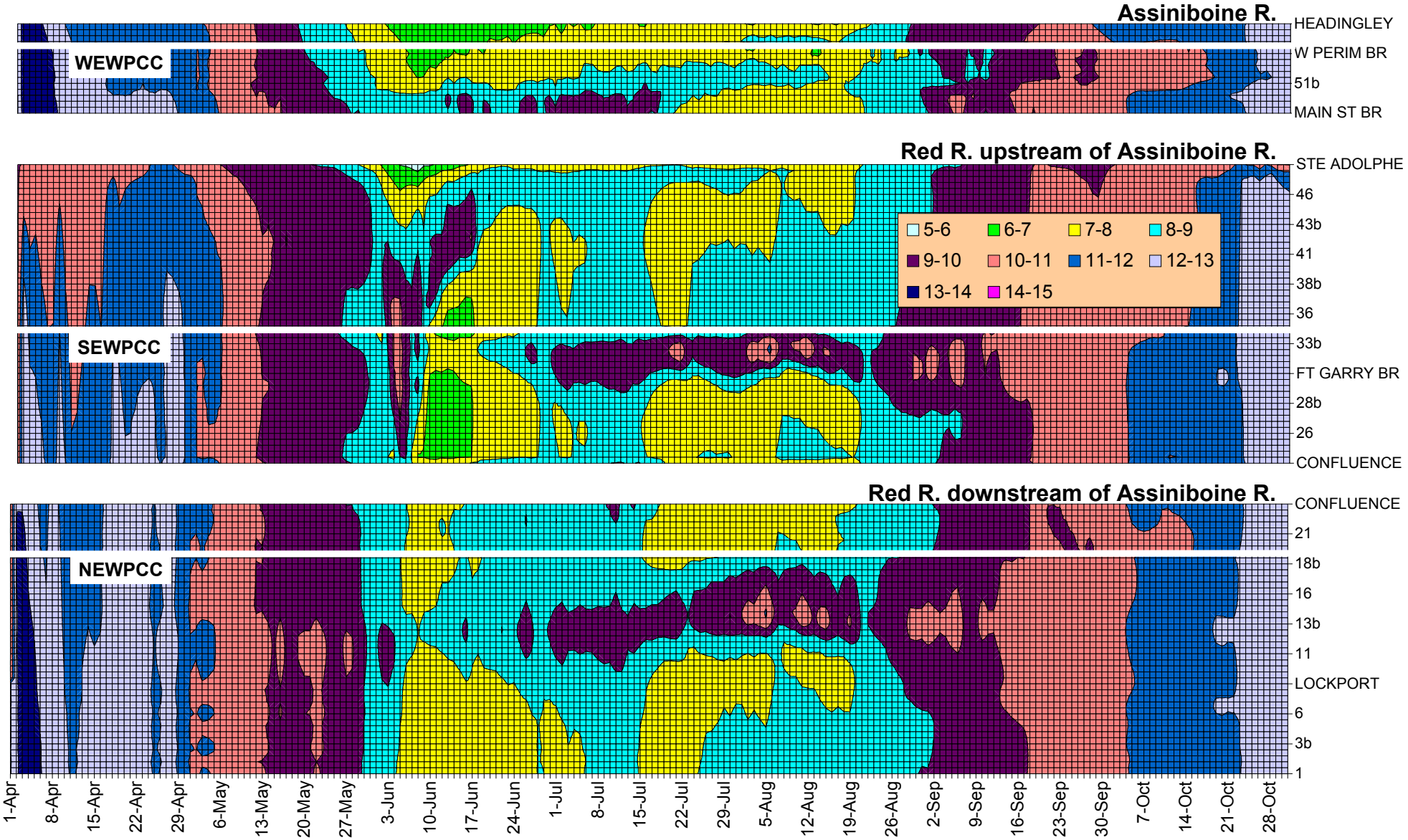
2031 CBOD WITH PROJECT (mg/L) - 1988 critical low flow



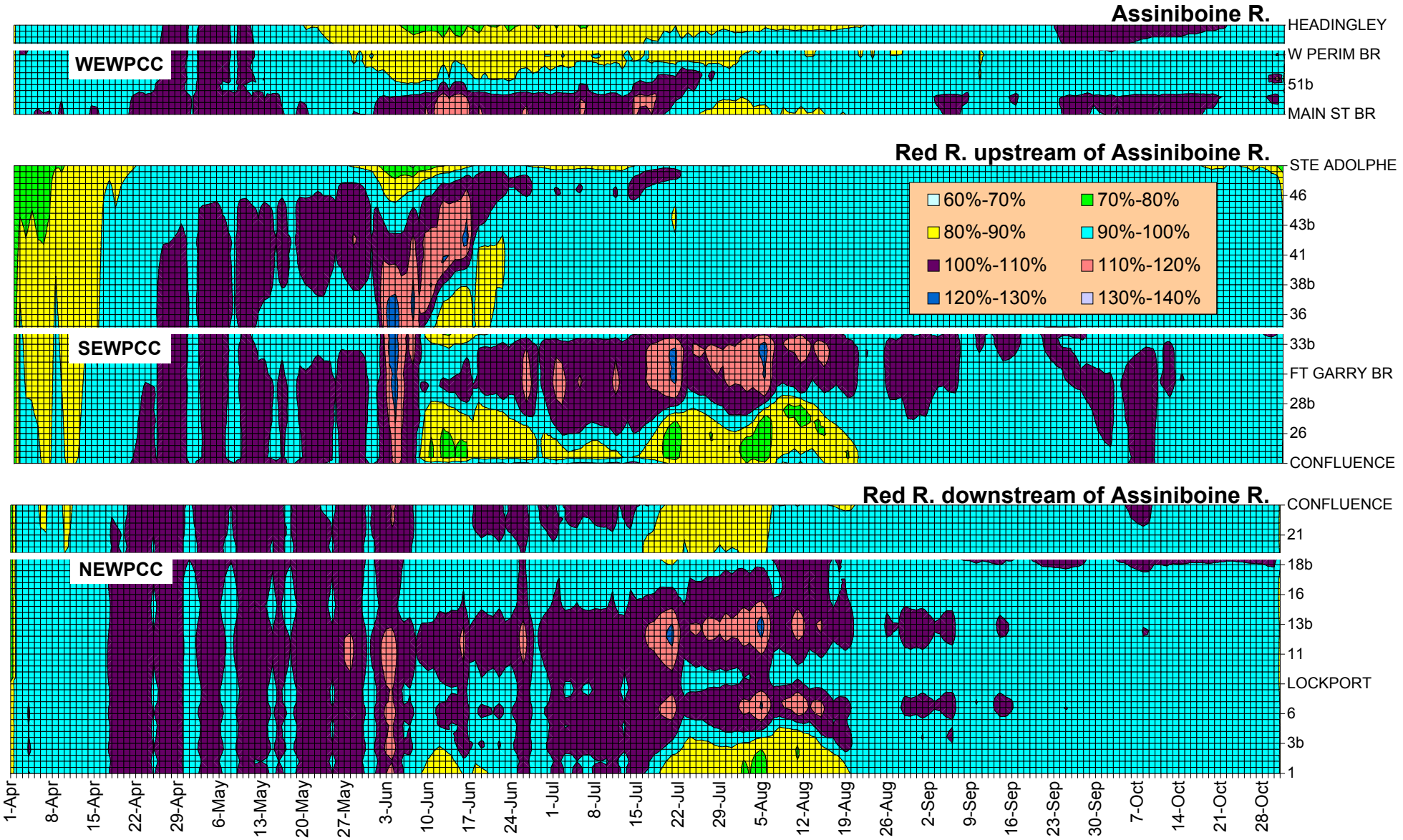
2031 DISSOLVED OXYGEN WITHOUT PROJECT (mg/L) - 1988 critical low flow



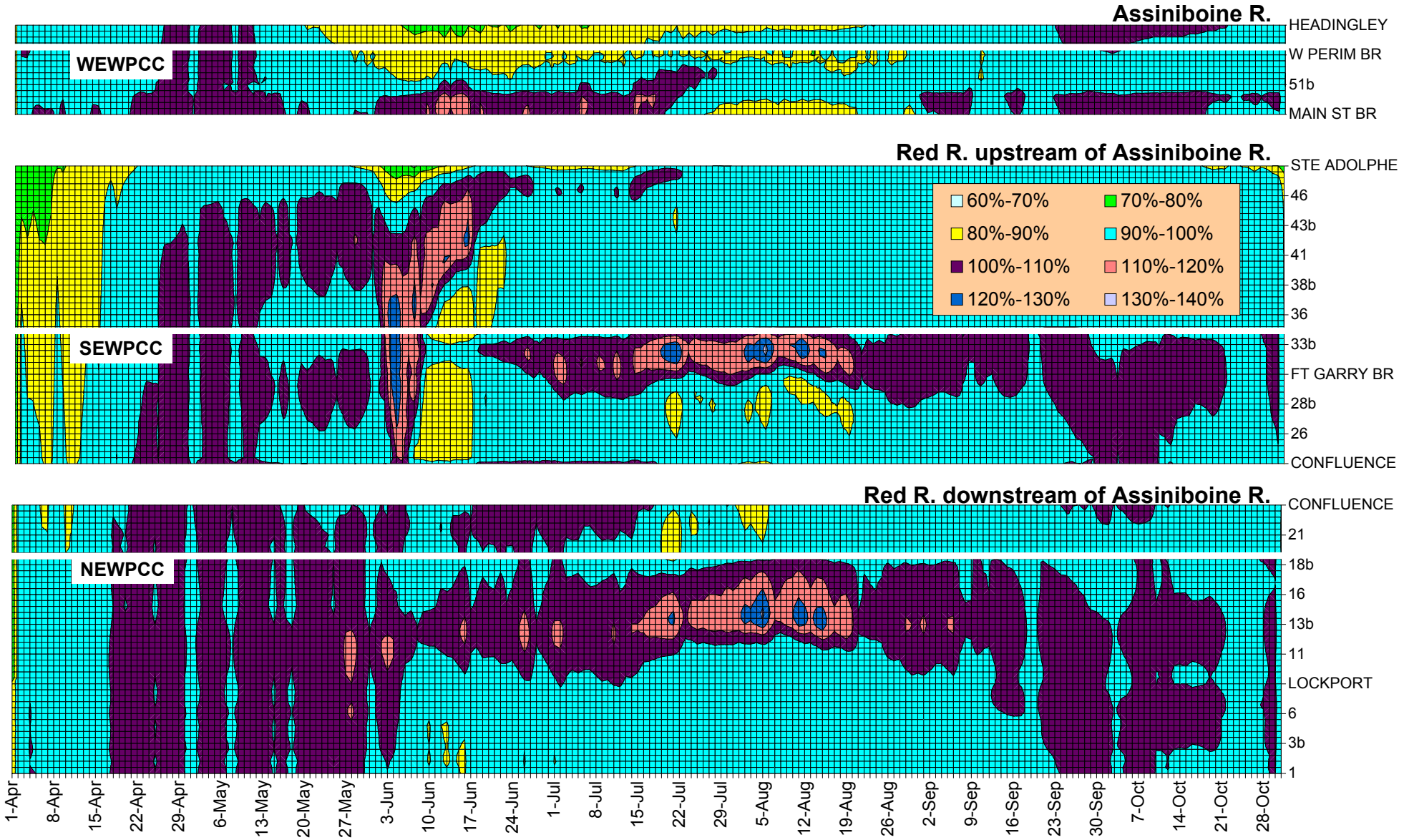
2031 DISSOLVED OXYGEN WITH PROJECT (mg/L) - 1988 critical low flow

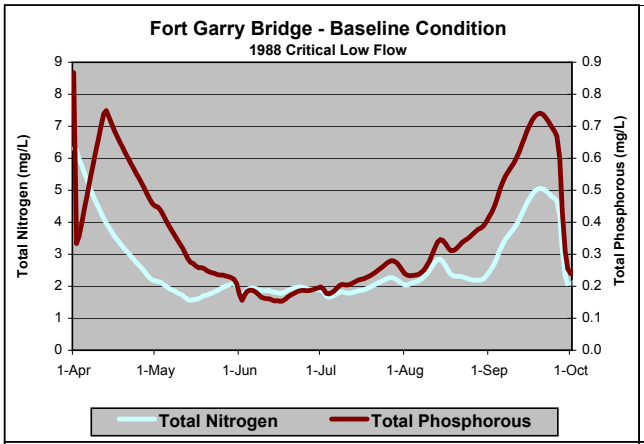


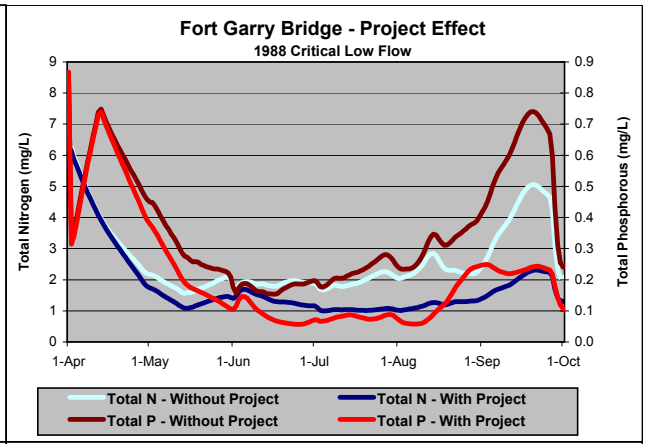
2031 MINIMUM DO SATURATION WITHOUT PROJECT - 1988 critical low flow

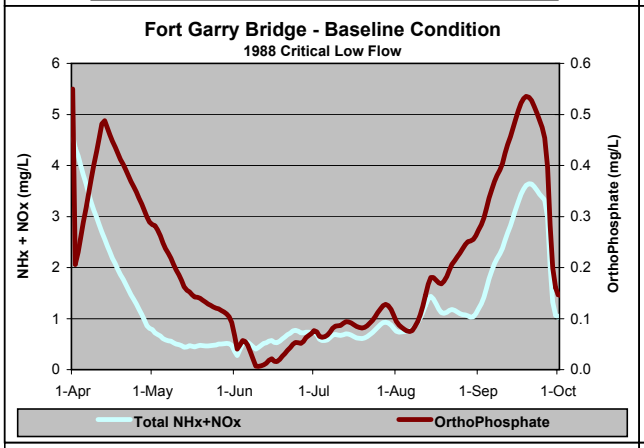


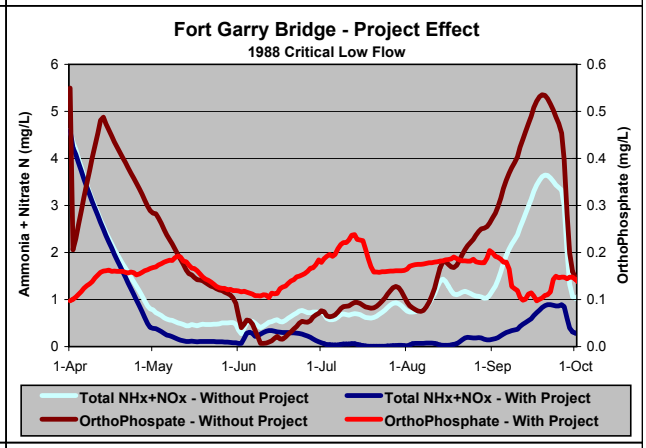
2031 MINIMUM DO SATURATION WITH PROJECT (mg/L) - 1988 critical low flow

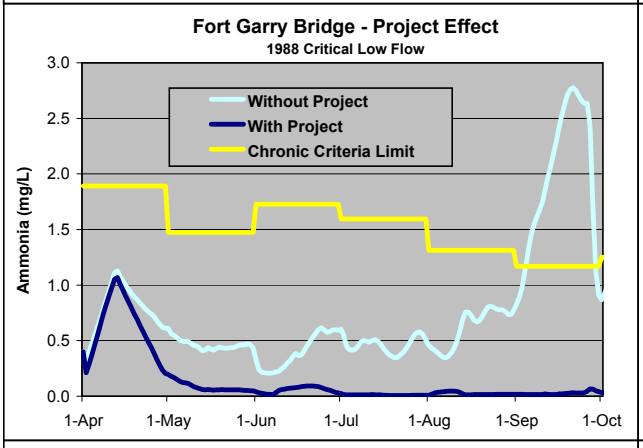


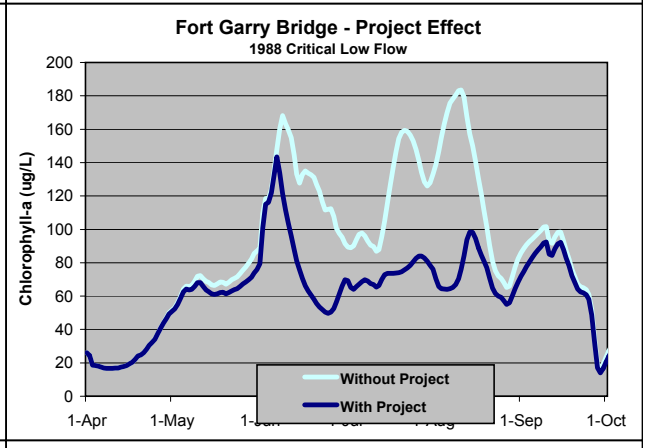

Fort Garry Bridge - Baseline Condition
1988 Critical Low Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total Nitrogen
Total Phosphorous

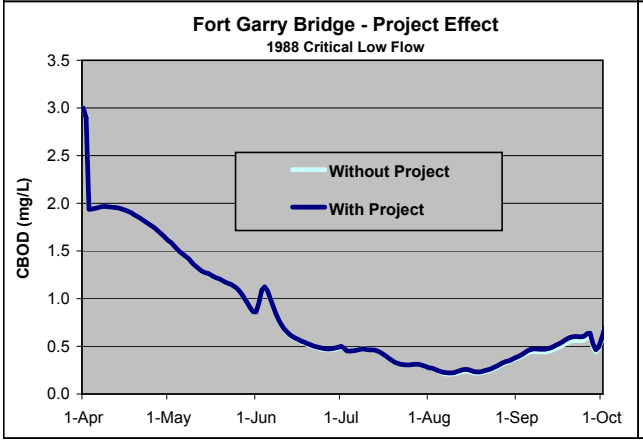

Fort Garry Bridge - Project Effect
1988 Critical Low Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total N - Without Project
Total N - With Project
Total P - Without Project
Total P - With Project

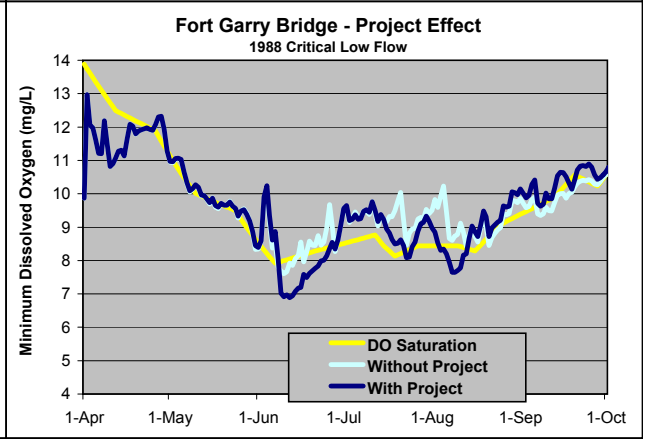

Fort Garry Bridge - Baseline Condition
1988 Critical Low Flow
NHx + NOx (mg/L)
OrthoPhosphate (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx
OrthoPhosphate

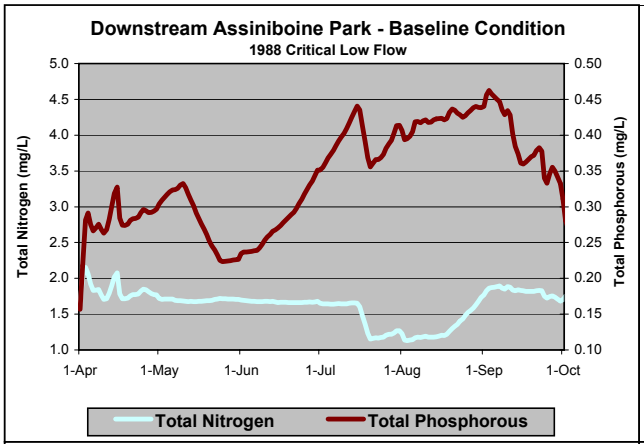

Fort Garry Bridge - Project Effect
1988 Critical Low Flow
Ammonia + Nitrate N (mg/L)
OrthoPhosphate (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx - Without Project
Total NHx+NOx - With Project
OrthoPhospate - Without Project
OrthoPhosphate - With Project

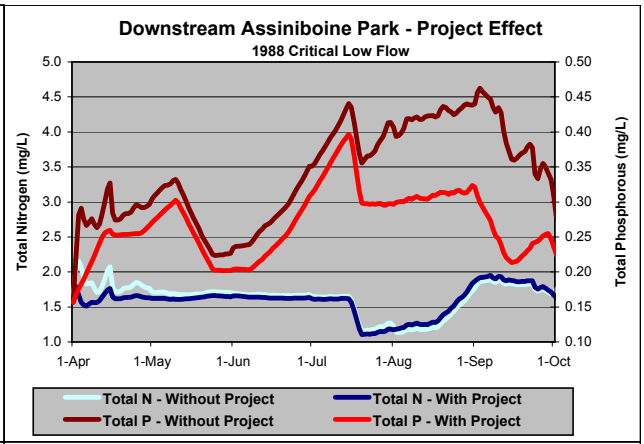

Fort Garry Bridge - Project Effect
1988 Critical Low Flow
Ammonia (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Without Project
With Project
Chronic Criteria Limit

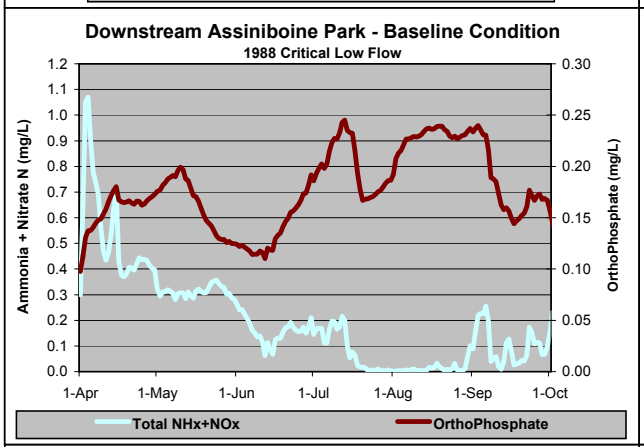

Fort Garry Bridge - Project Effect
1988 Critical Low Flow
Chlorophyll-a (ug/L)
1-Apr
1-May
1-Sep
1-Oct
Without Project
With Project

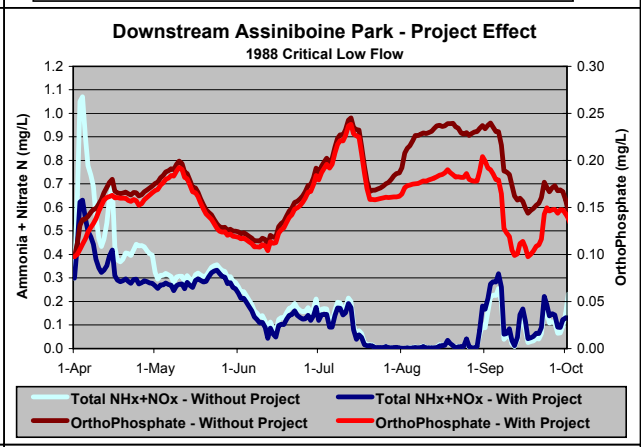

Fort Garry Bridge - Project Effect
1988 Critical Low Flow
CBOD (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Without Project
With Project

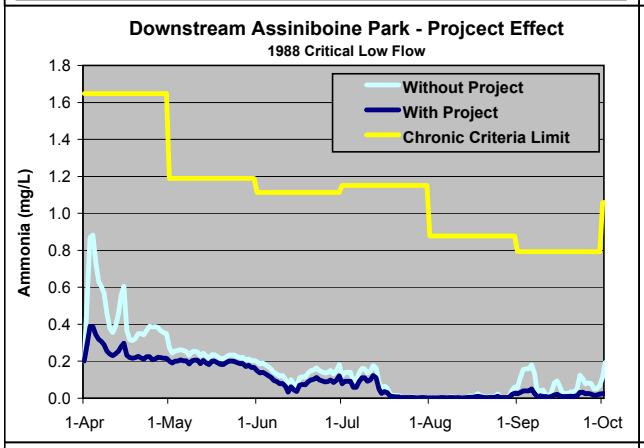

Fort Garry Bridge - Project Effect
1988 Critical Low Flow
Minimum Dissolved Oxygen (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
DO Saturation
Without Project
With Project

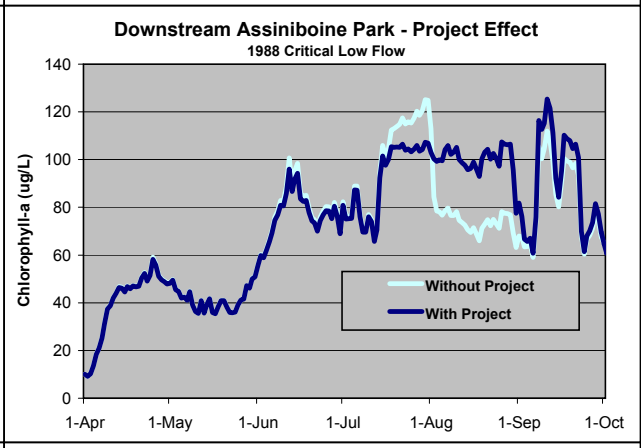

Downstream Assiniboine Park - Baseline Condition
1988 Critical Low Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total Nitrogen
Total Phosphorous

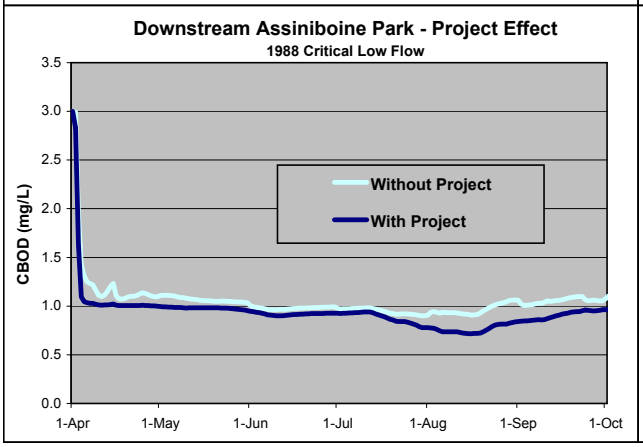

Downstream Assiniboine Park - Project Effect
1988 Critical Low Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total N - Without Project
Total N - With Project
Total P - Without Project
Total P - With Project

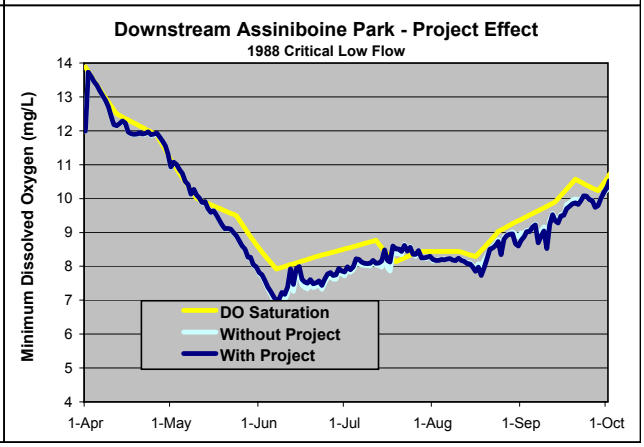

Downstream Assiniboine Park - Baseline Condition
1988 Critical Low Flow
Ammonia + Nitrate N (mg/L)
OrthoPhosphate (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx
OrthoPhosphate

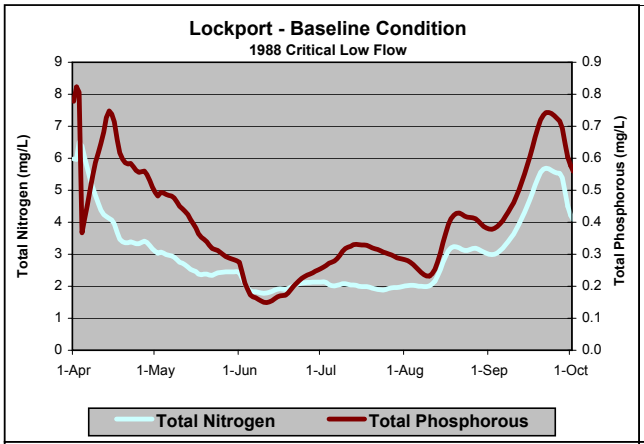

Downstream Assiniboine Park - Project Effect
1988 Critical Low Flow
Ammonia + Nitrate N (mg/L)
OrthoPhosphate (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx - Without Project
Total NHx+NOx - With Project
OrthoPhosphate - Without Project
OrthoPhosphate - With Project

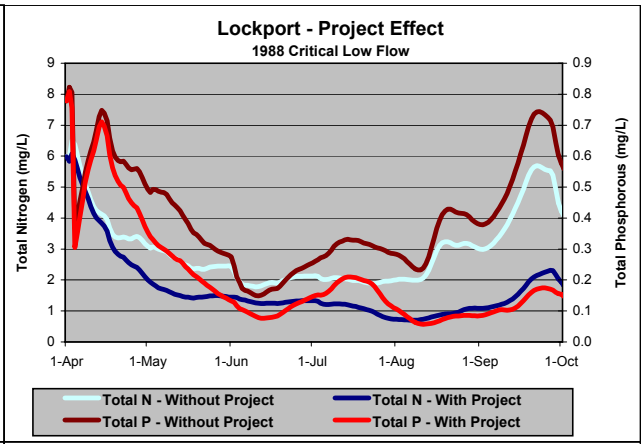

Downstream Assiniboine Park - Projcect Effect
1988 Critical Low Flow
Ammonia (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Without Project
With Project
Chronic Criteria Limit

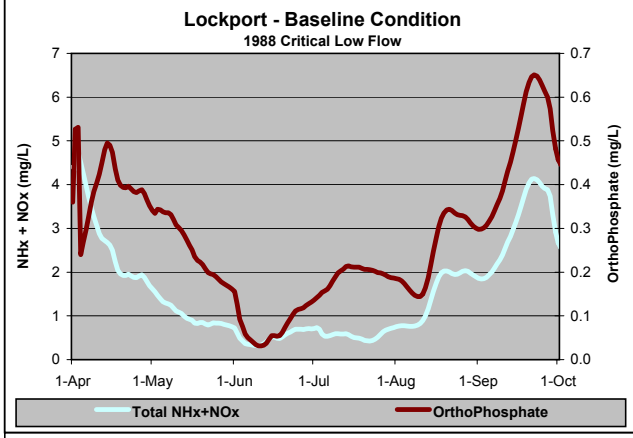

Downstream Assiniboine Park - Project Effect
1988 Critical Low Flow
Chlorophyll-a (ug/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Without Project
With Project

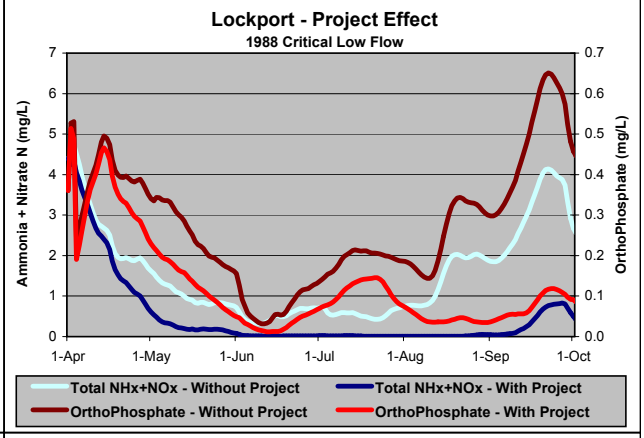

Downstream Assiniboine Park - Project Effect
1988 Critical Low Flow
CBOD (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Without Project
With Project

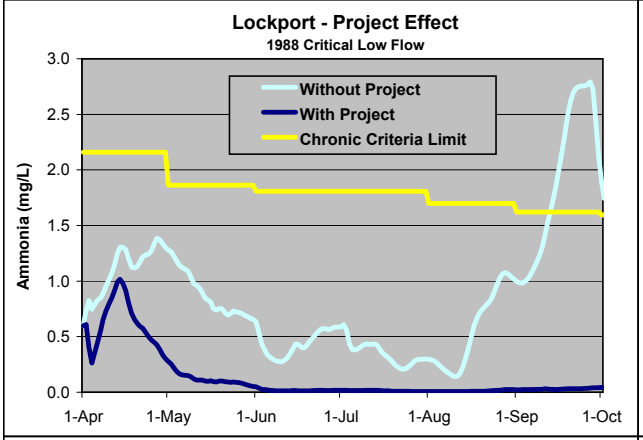

Downstream Assiniboine Park - Project Effect
1988 Critical Low Flow
Minimum Dissolved Oxygen (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
DO Saturation
Without Project
With Project

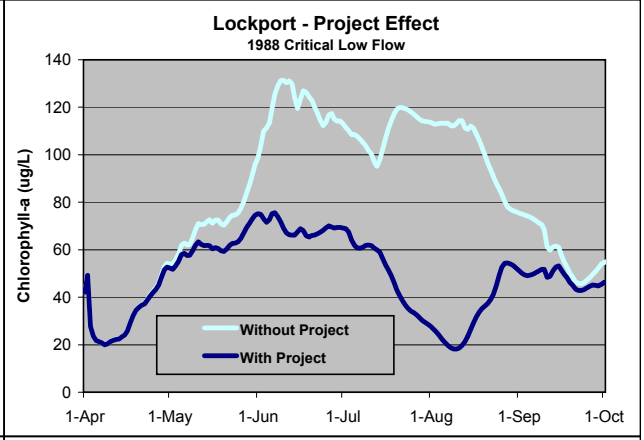

Lockport - Baseline Condition
1988 Critical Low Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total Nitrogen
Total Phosphorous

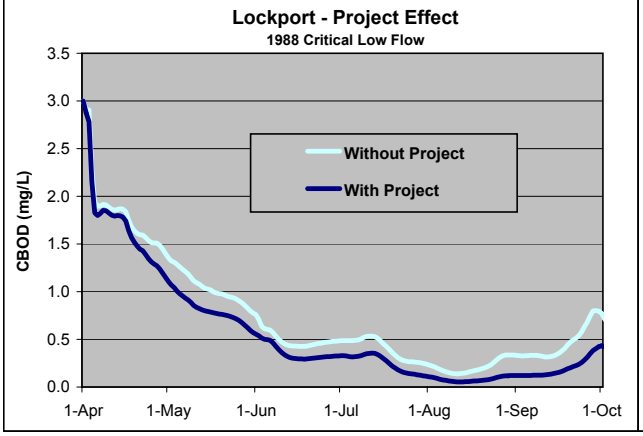

Lockport - Project Effect
1988 Critical Low Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total N - Without Project
Total N - With Project
Total P - Without Project
Total P - With Project

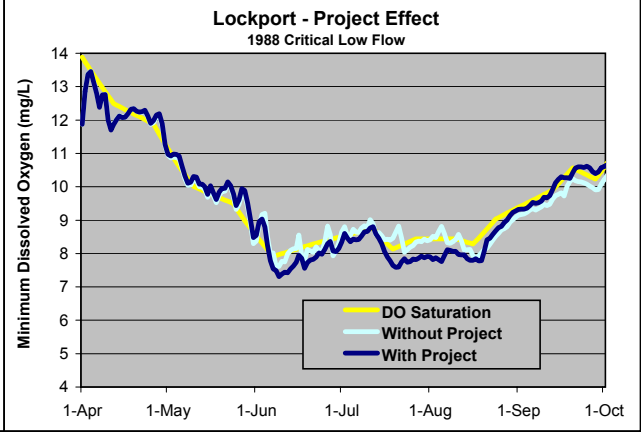

Lockport - Baseline Condition
1988 Critical Low Flow
NHx + NOx (mg/L)
OrthoPhosphate (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx
OrthoPhosphate

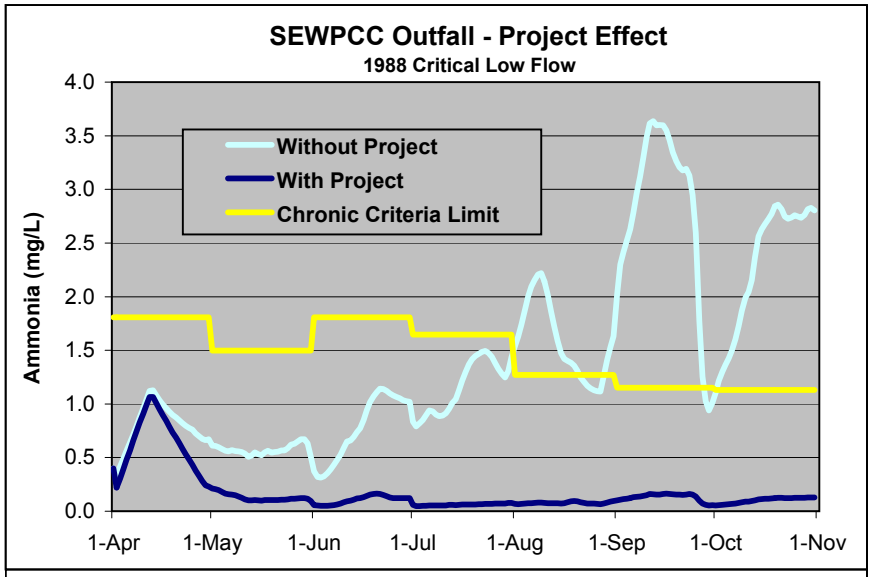

Lockport - Project Effect
1988 Critical Low Flow
Ammonia + Nitrate N (mg/L)
OrthoPhosphate (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx - Without Project
Total NHx+NOx - With Project
OrthoPhosphate - Without Project
OrthoPhosphate - With Project

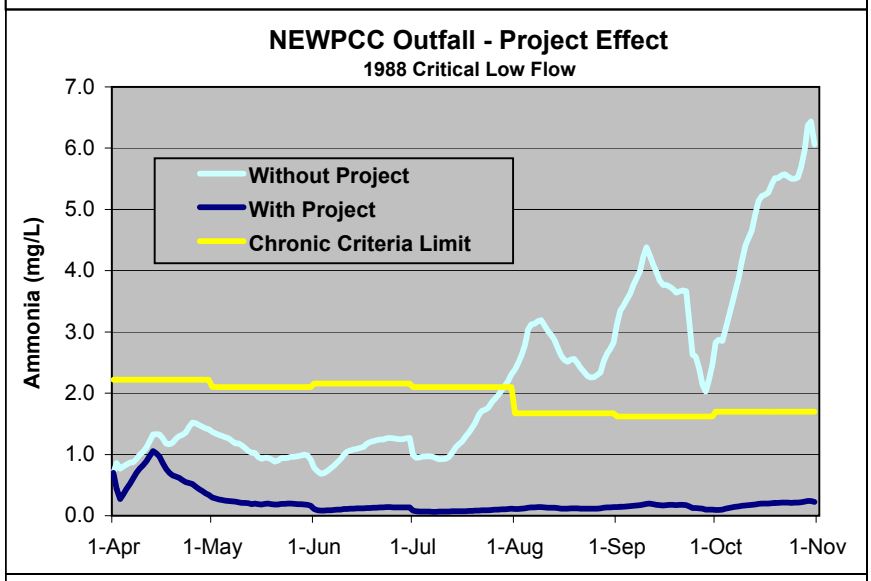

Lockport - Project Effect
1988 Critical Low Flow
Ammonia (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Without Project
With Project
Chronic Criteria Limit

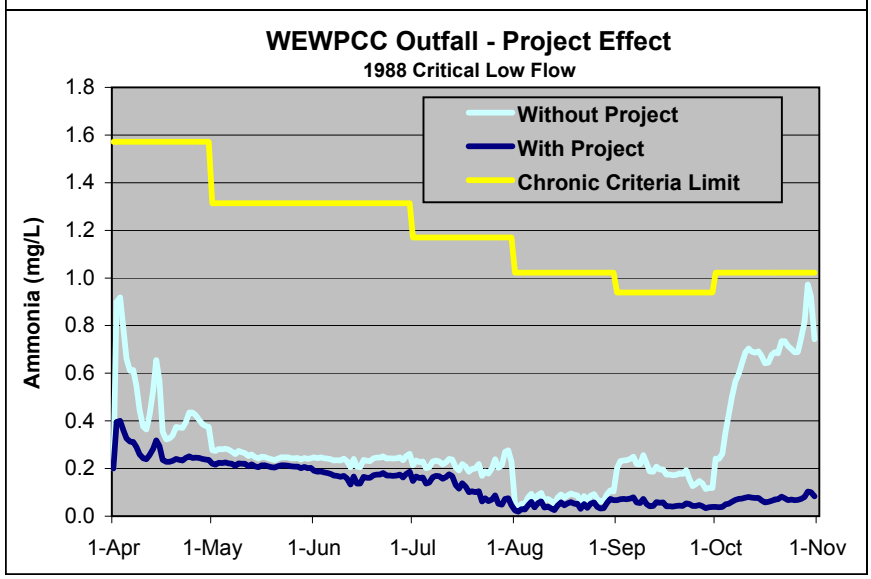

Lockport - Project Effect
1988 Critical Low Flow
Chlorophyll-a (ug/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Without Project
With Project


Lockport - Project Effect
1988 Critical Low Flow
CBOD (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Without Project
With Project

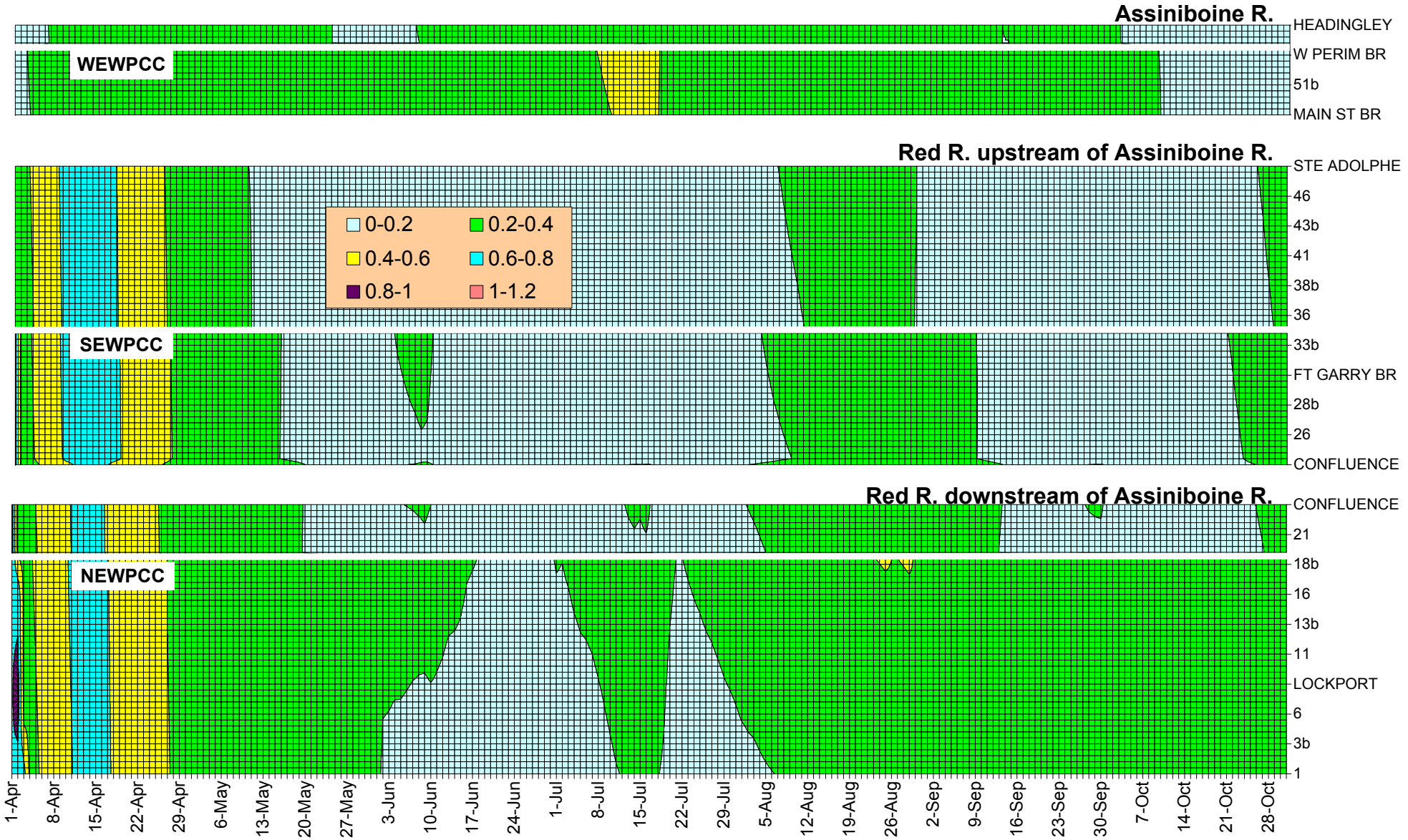

Lockport - Project Effect
1988 Critical Low Flow
Minimum Dissolved Oxygen (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
DO Saturation
Without Project
With Project


SEWPCC Outfall - Project Effect
1988 Critical Low Flow
Ammonia (mg/L)
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
Without Project
With Project
Chronic Criteria Limit
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
1-Nov

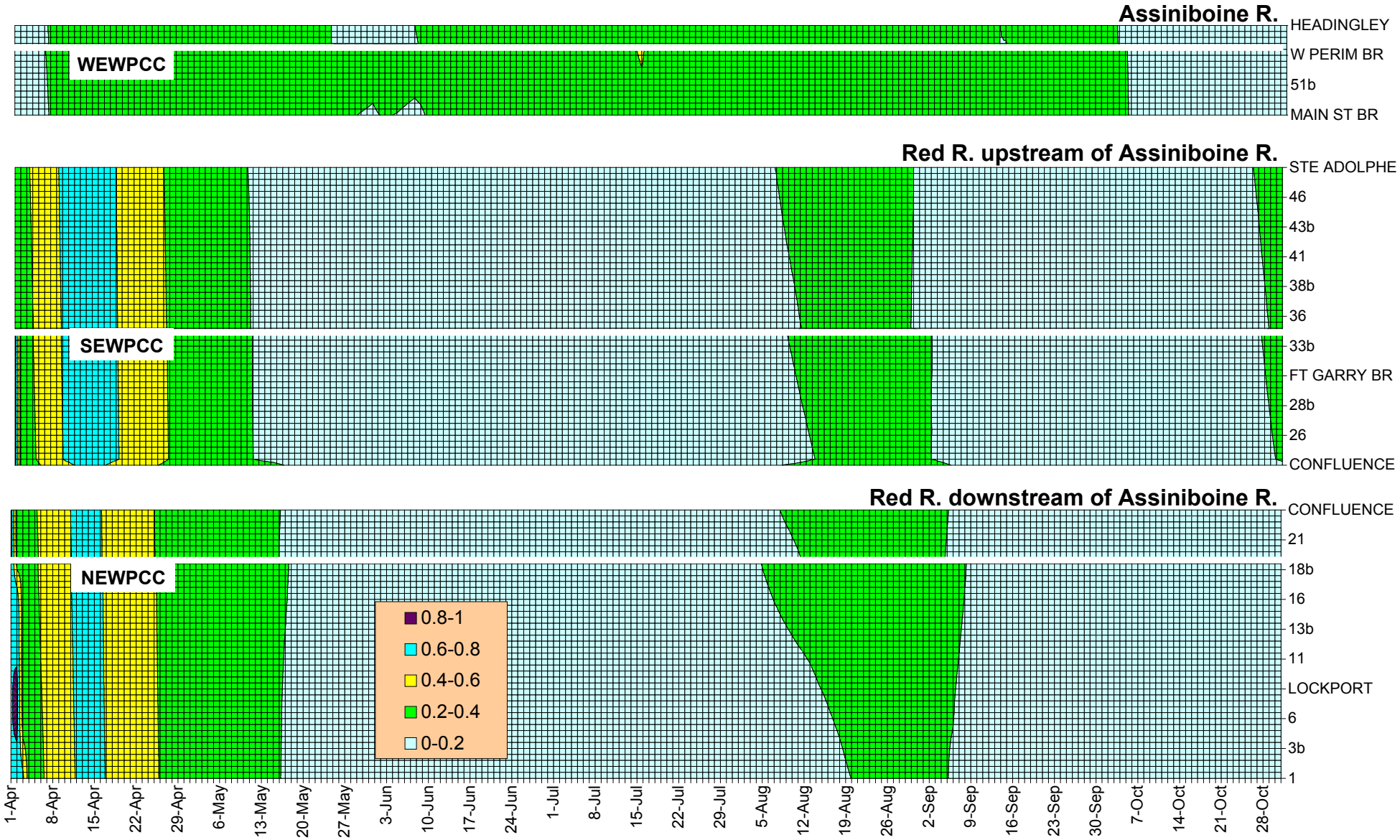

NEWPCC Outfall - Project Effect
1988 Critical Low Flow
Ammonia (mg/L)
7.0
6.0
5.0
4.0
3.0
2.0
1.0
0.0
Without Project
With Project
Chronic Criteria Limit
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
1-Nov


WEWPCC Outfall - Project Effect
1988 Critical Low Flow
Ammonia (mg/L)
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0.0
Without Project
With Project
Chronic Criteria Limit
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
1-Nov

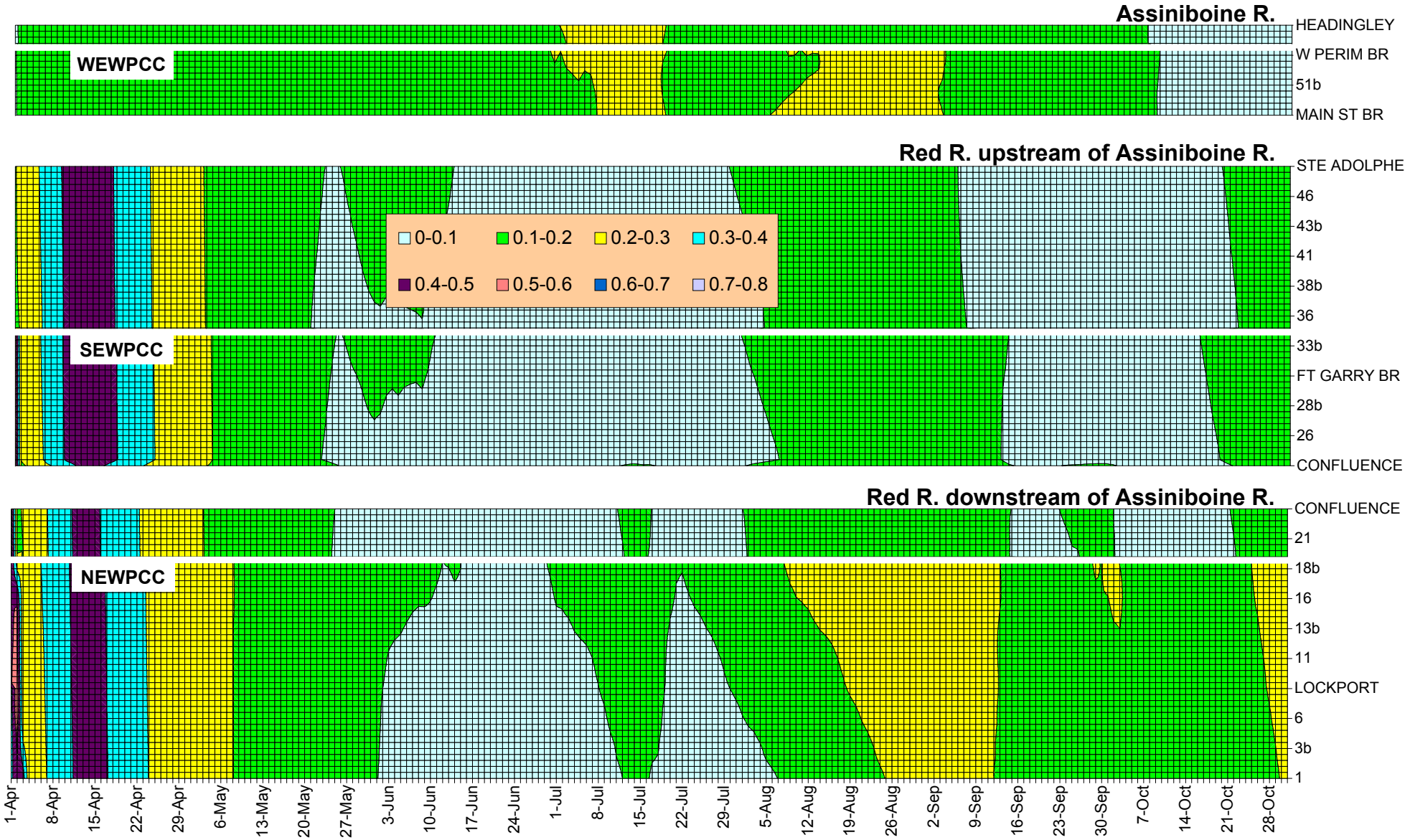
2031 TOTAL PHOSPHOROUS WITHOUT PROJECT (mg/L) - median flow



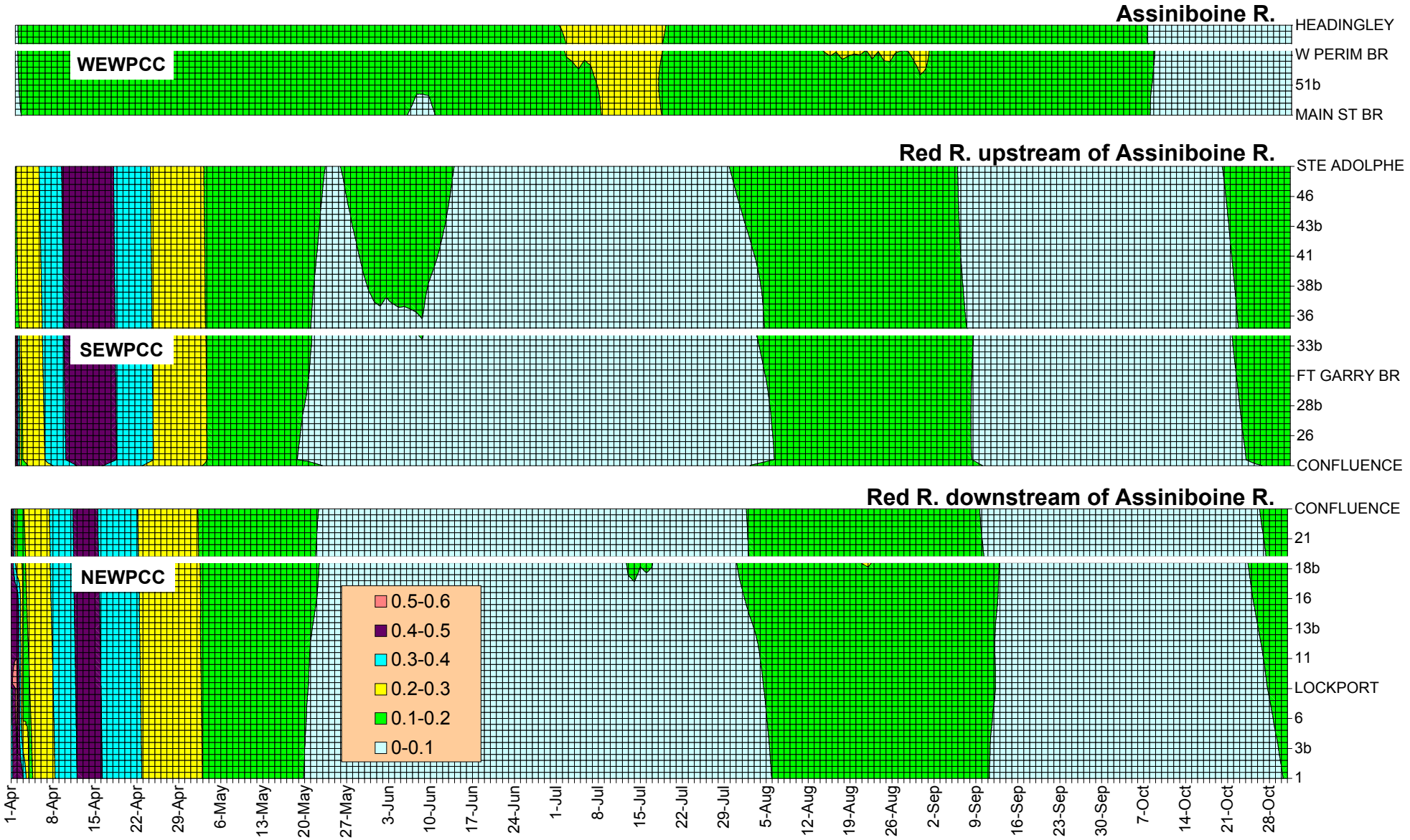
2031 TOTAL PHOSPHOROUS WITH PROJECT (mg/L) - median flow



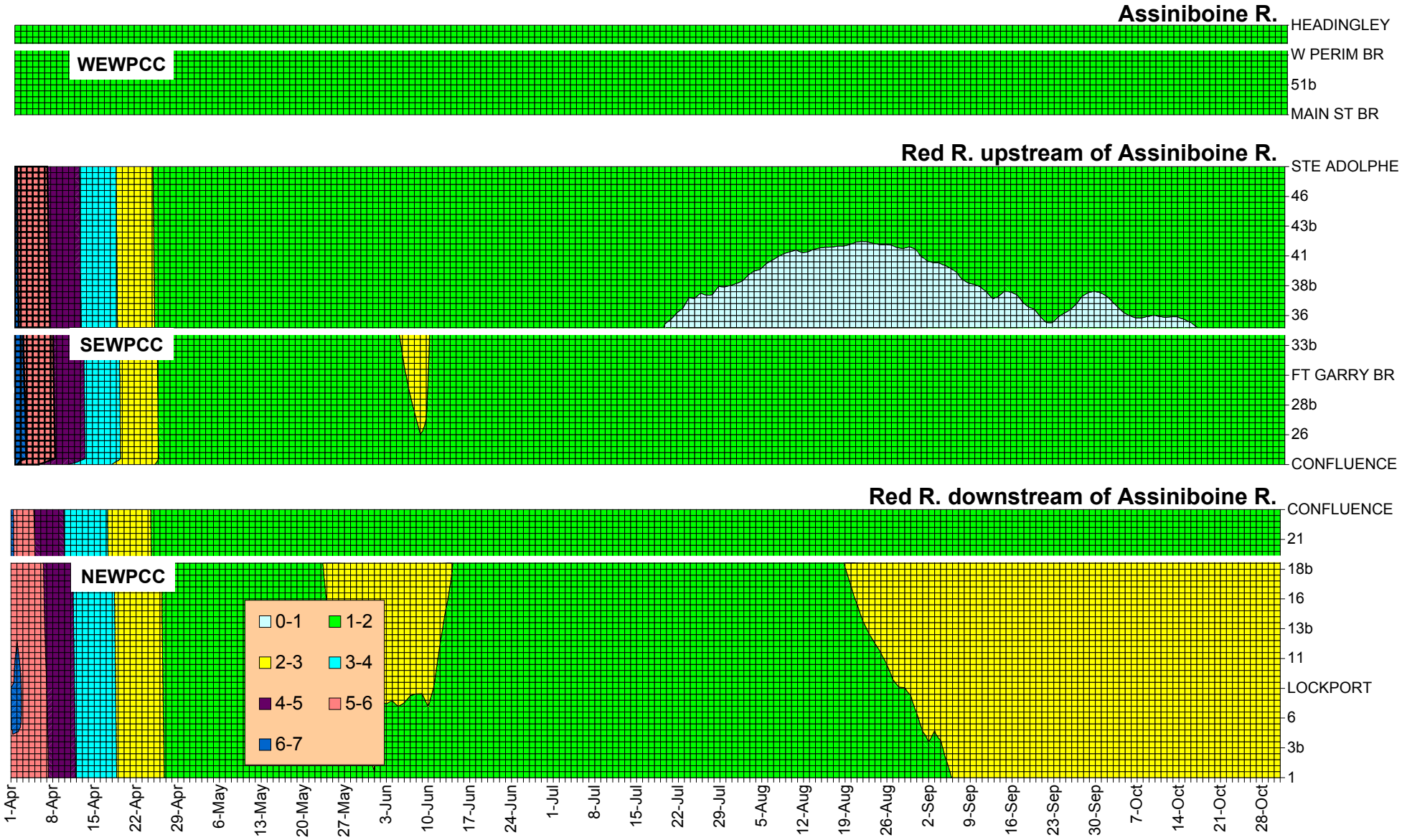
2031 ORTHOPHOSPHATE WITHOUT PROJECT (mg/L) - median flow



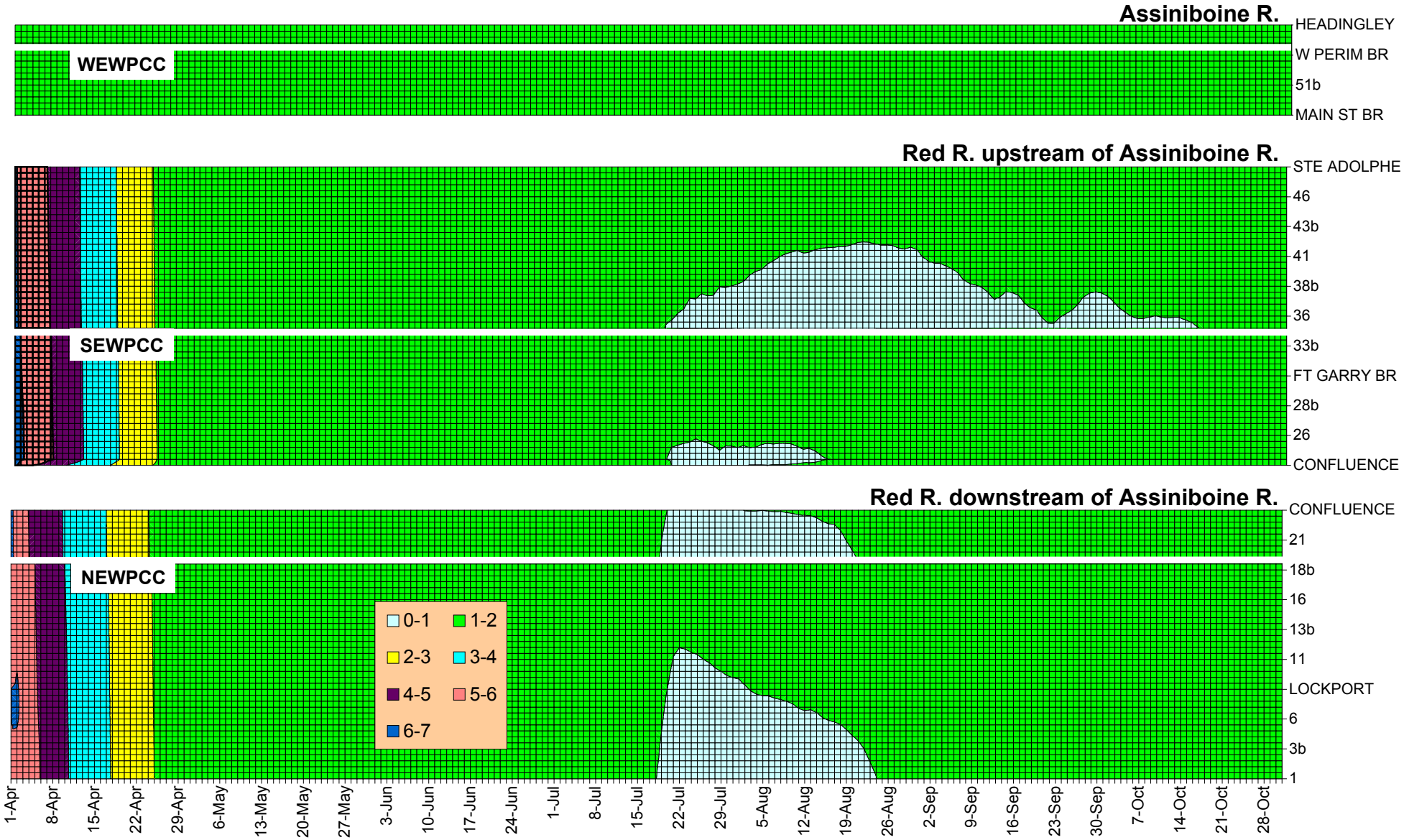
2031 ORTHOPHOSPHATE WITH PROJECT (mg/L) - median flow



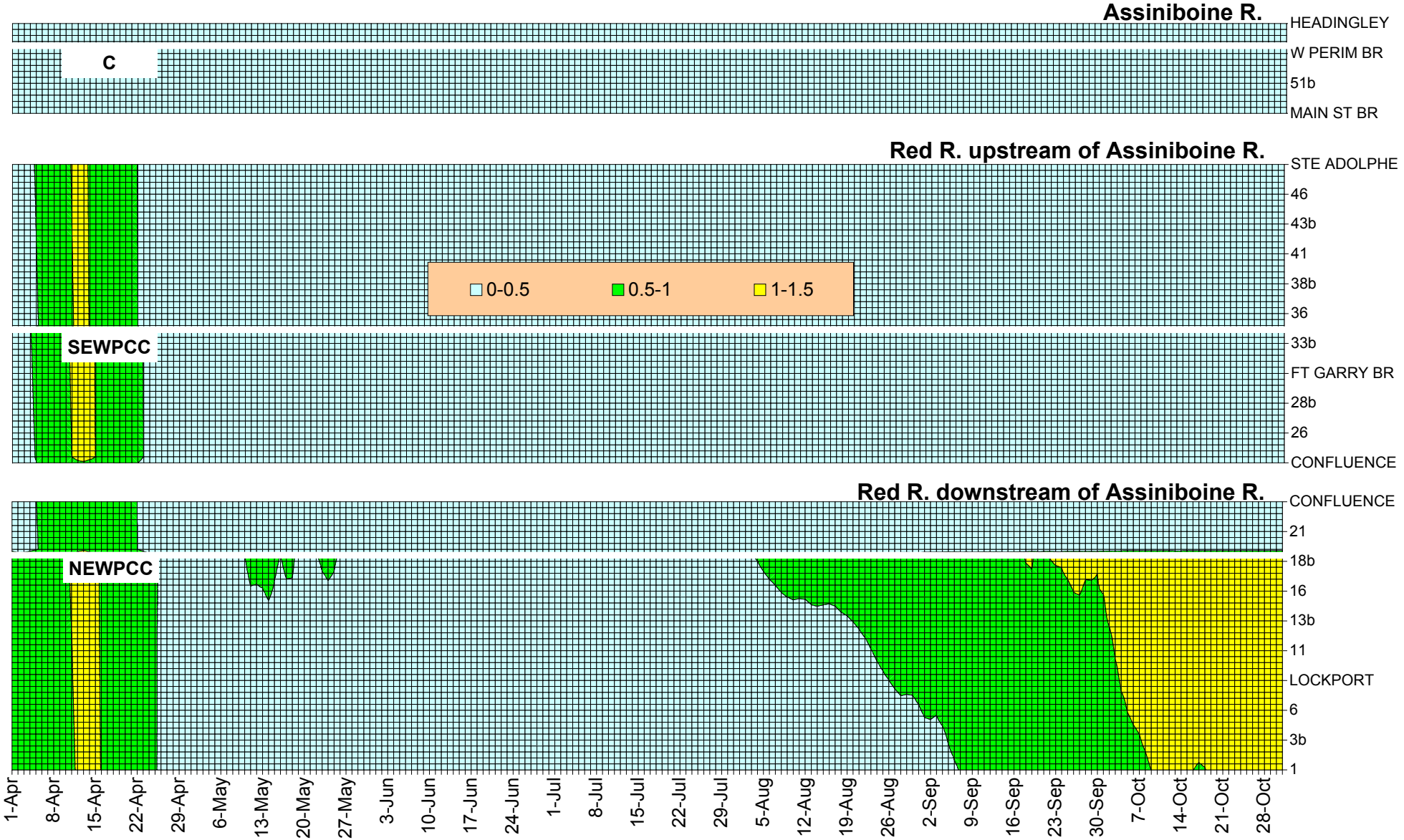
2031 TOTAL NITORGEN WITHOUT PROJECT (mg/L) - median flow



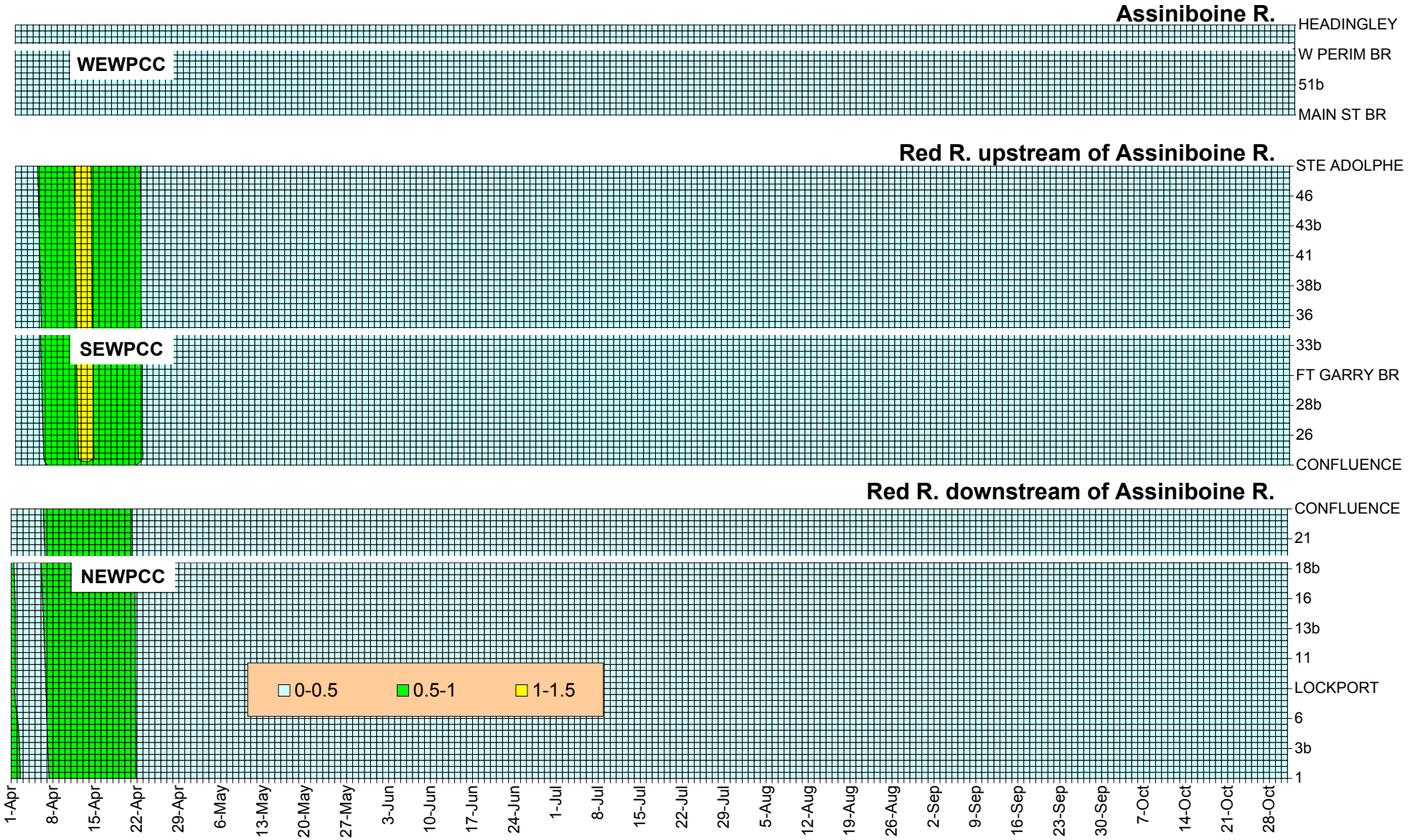
2031 TOTAL NITROGEN WITH PROJECT (mg/L) - median flow



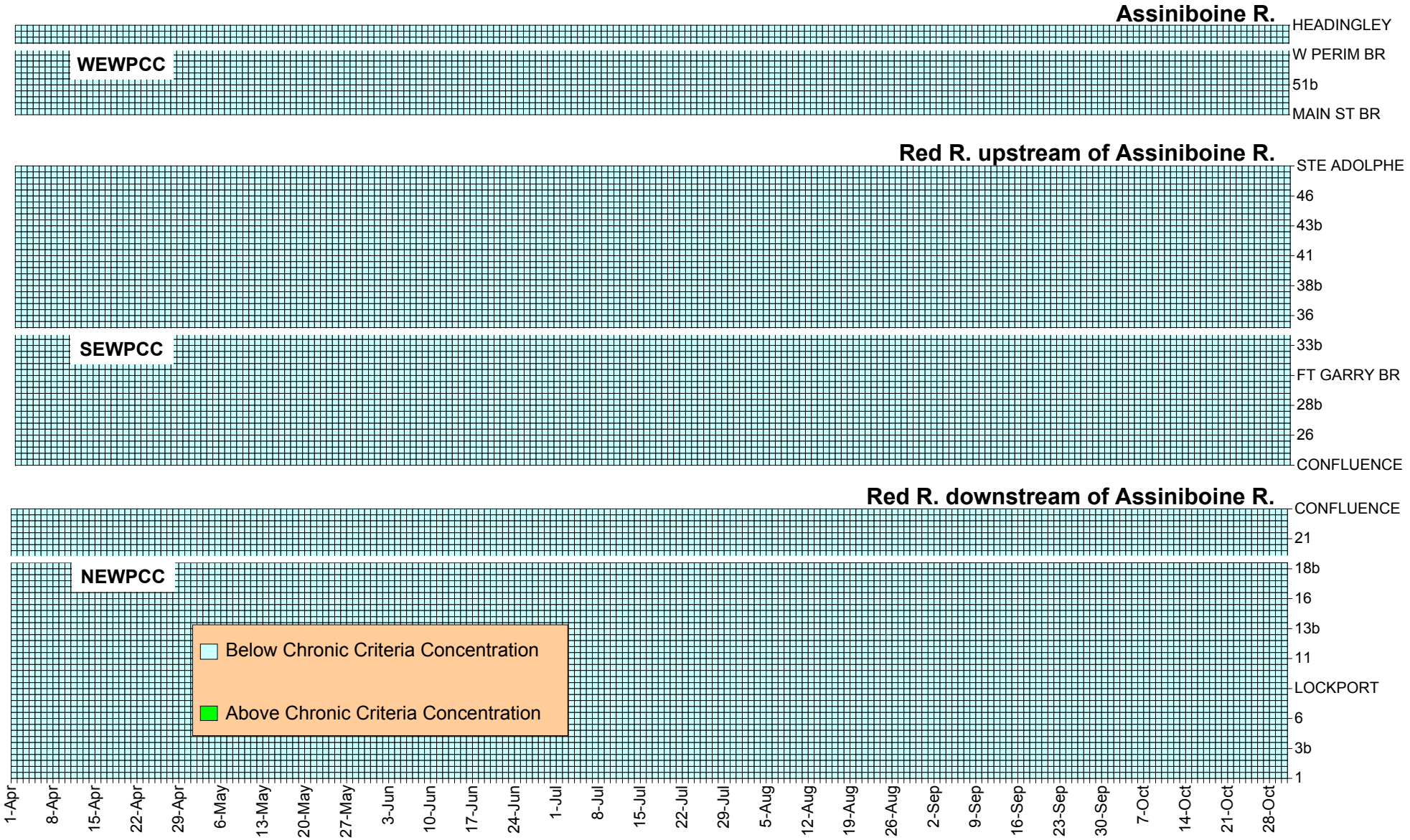
2031 AMMONIA WITHOUT PROJECT (mg/L) - median flow



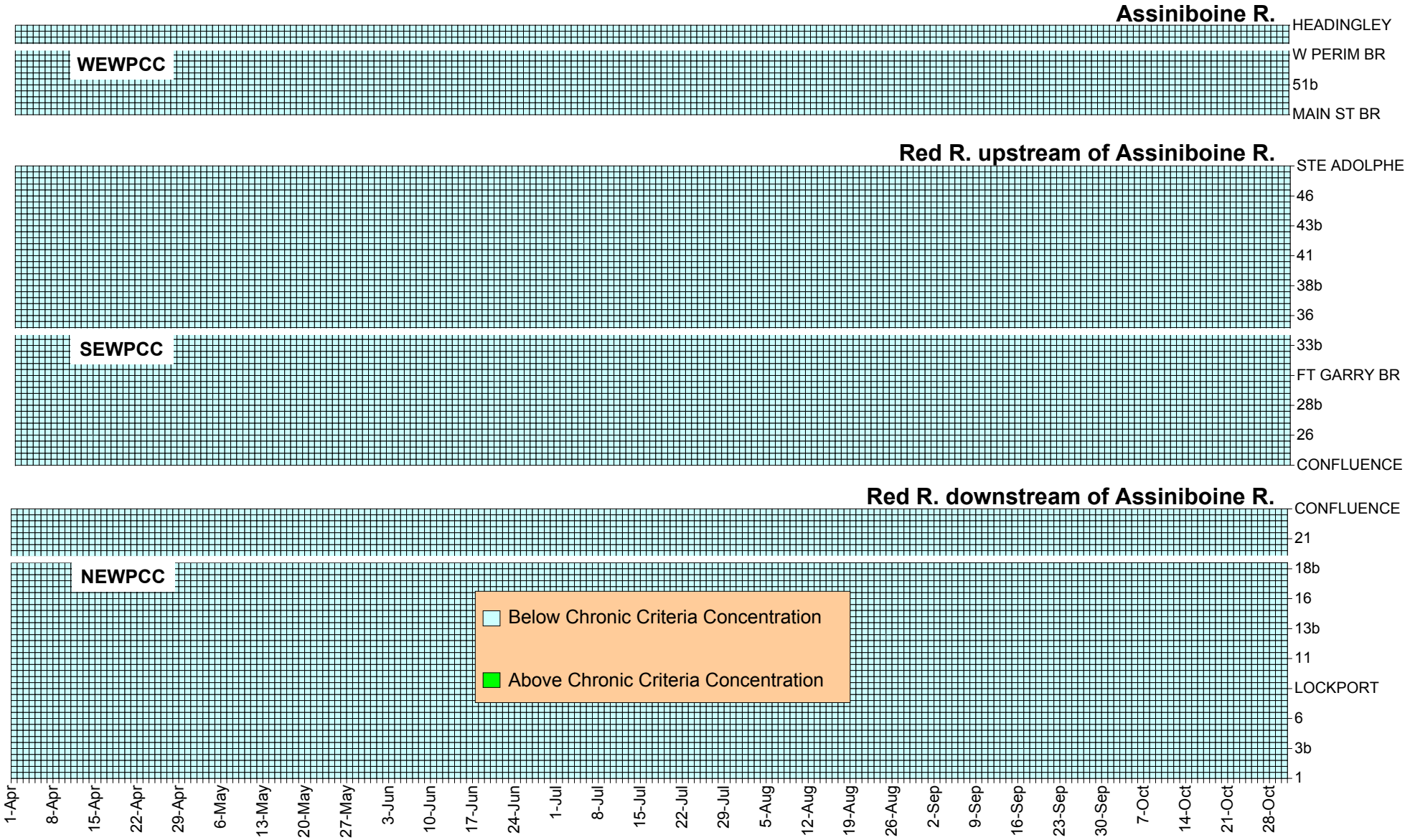
2031 AMMONIA WITH PROJECT (mg/L) - median flow



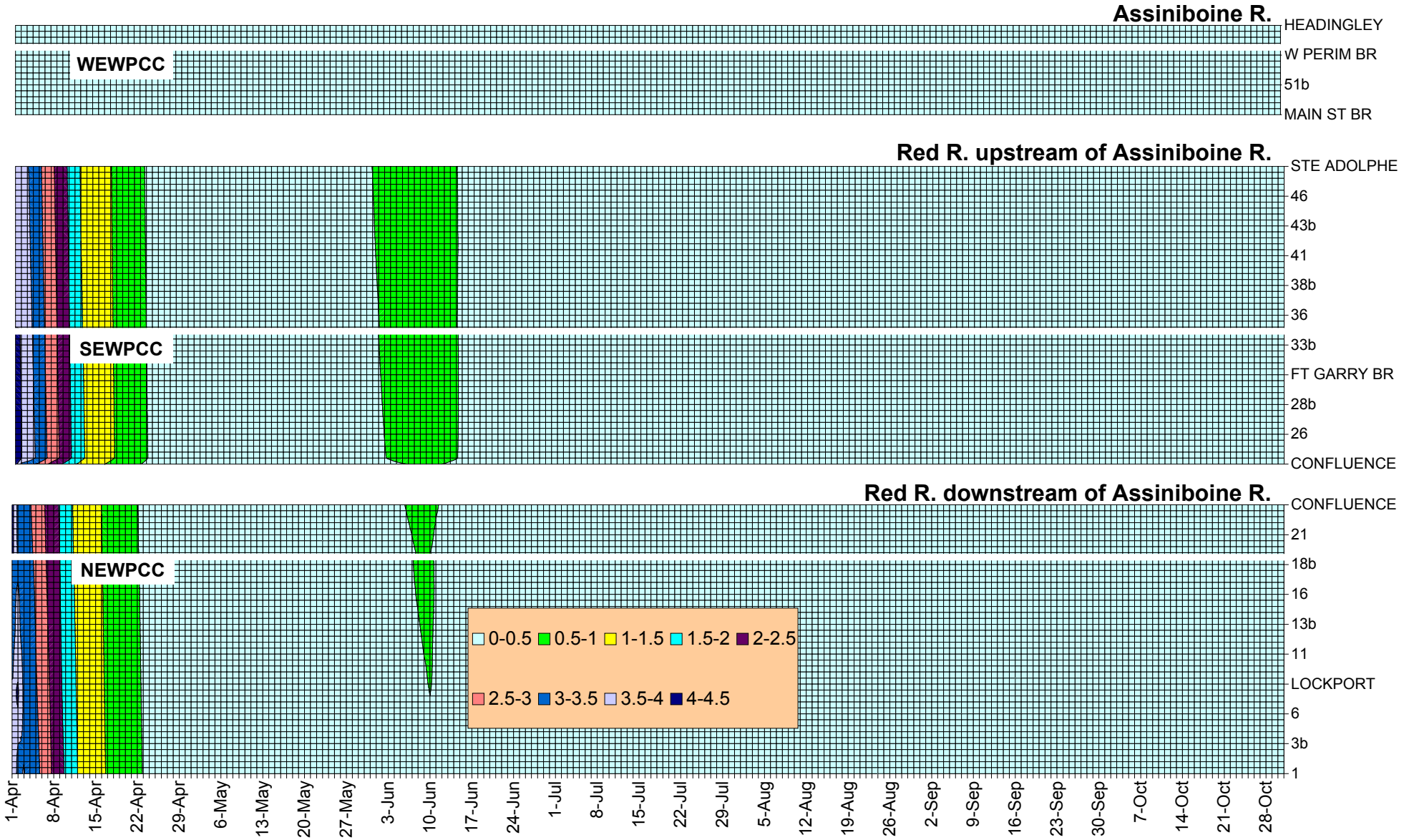
2031 AMMONIA CRITERIA COMPLIANCE WITHOUT PROJECT - median flow



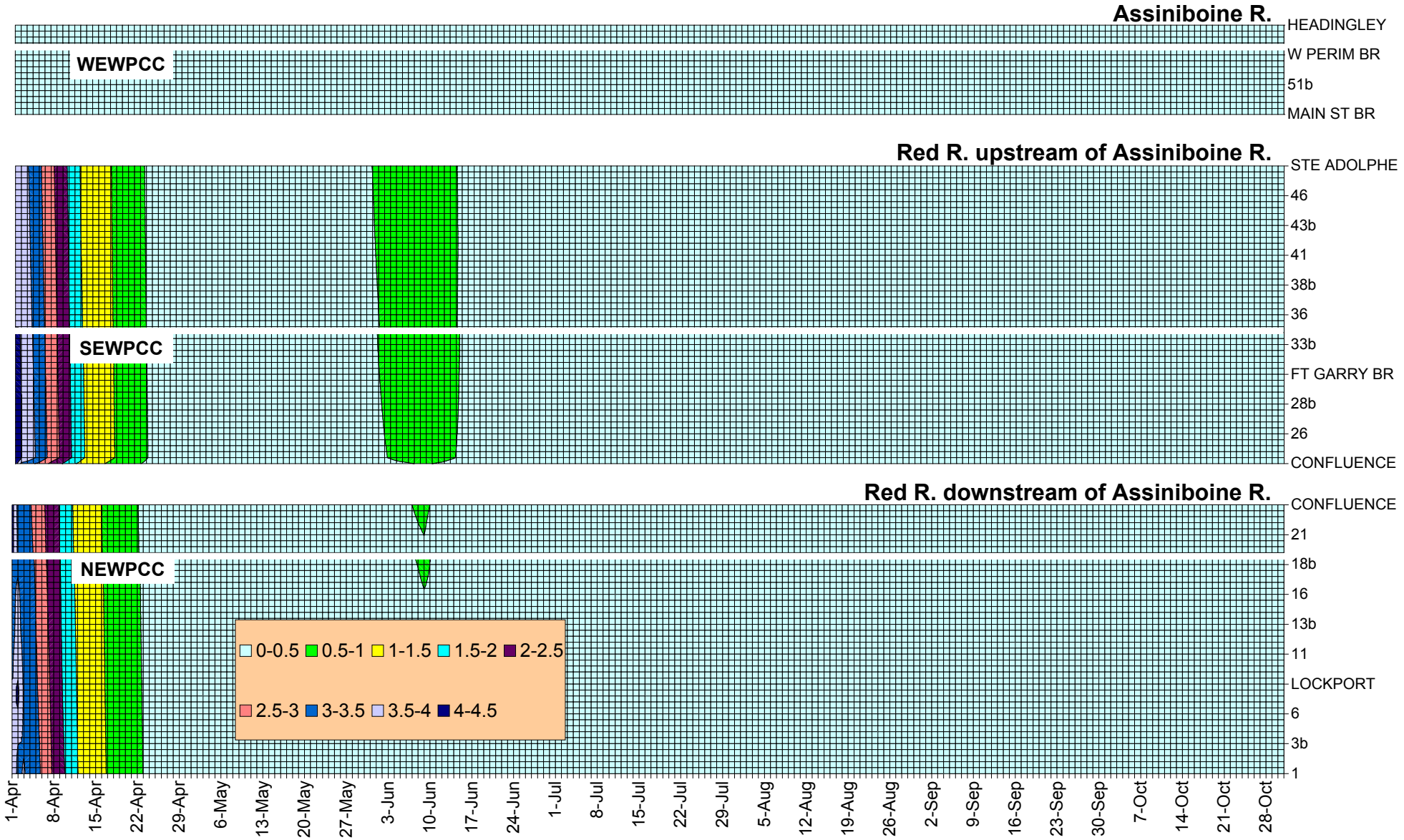
2031 AMMONIA CRITERIA COMPLIANCE WITH PROJECT - median flow



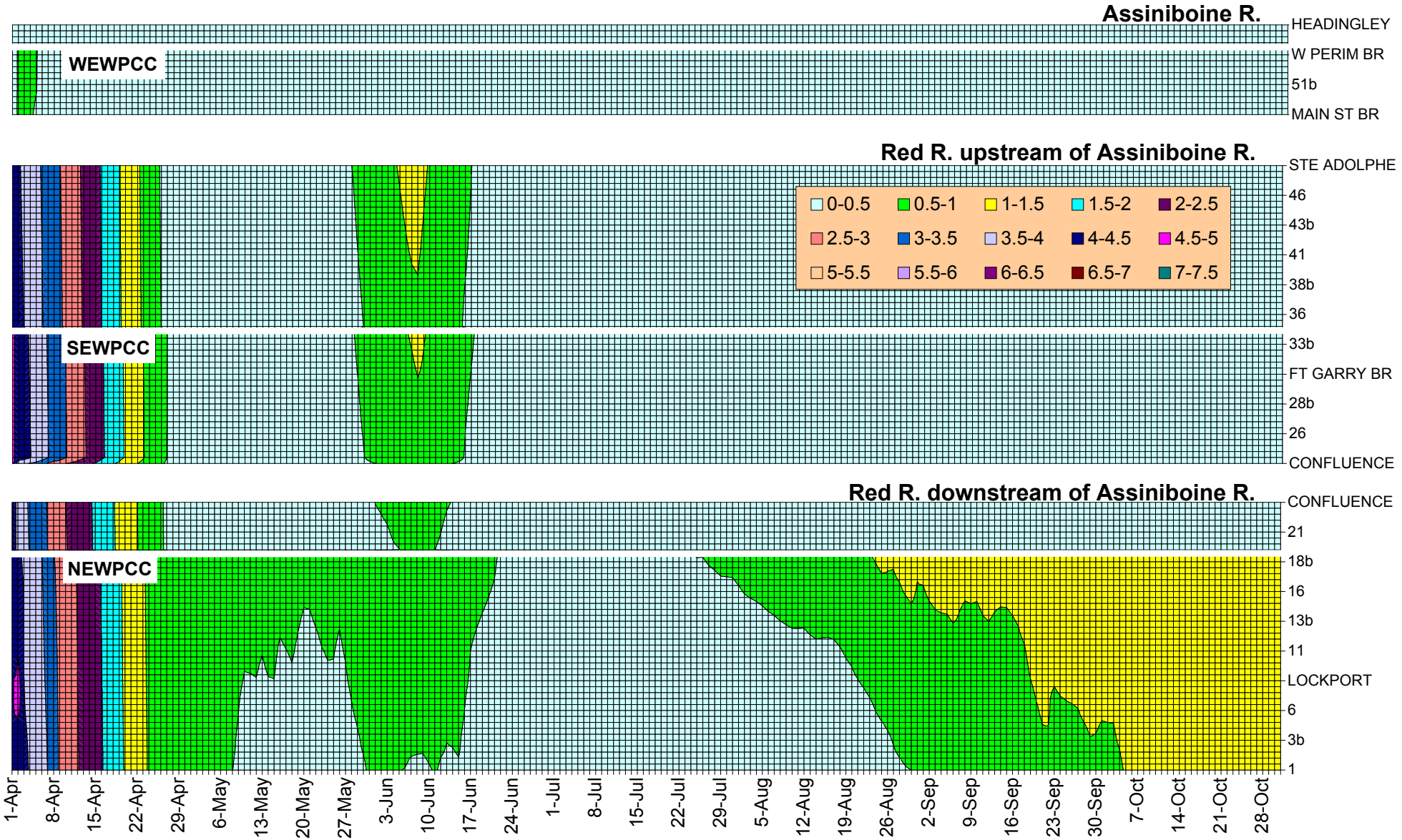
2031 NITRATE/NITRITE WITHOUT PROJECT (mg/L) - median flow



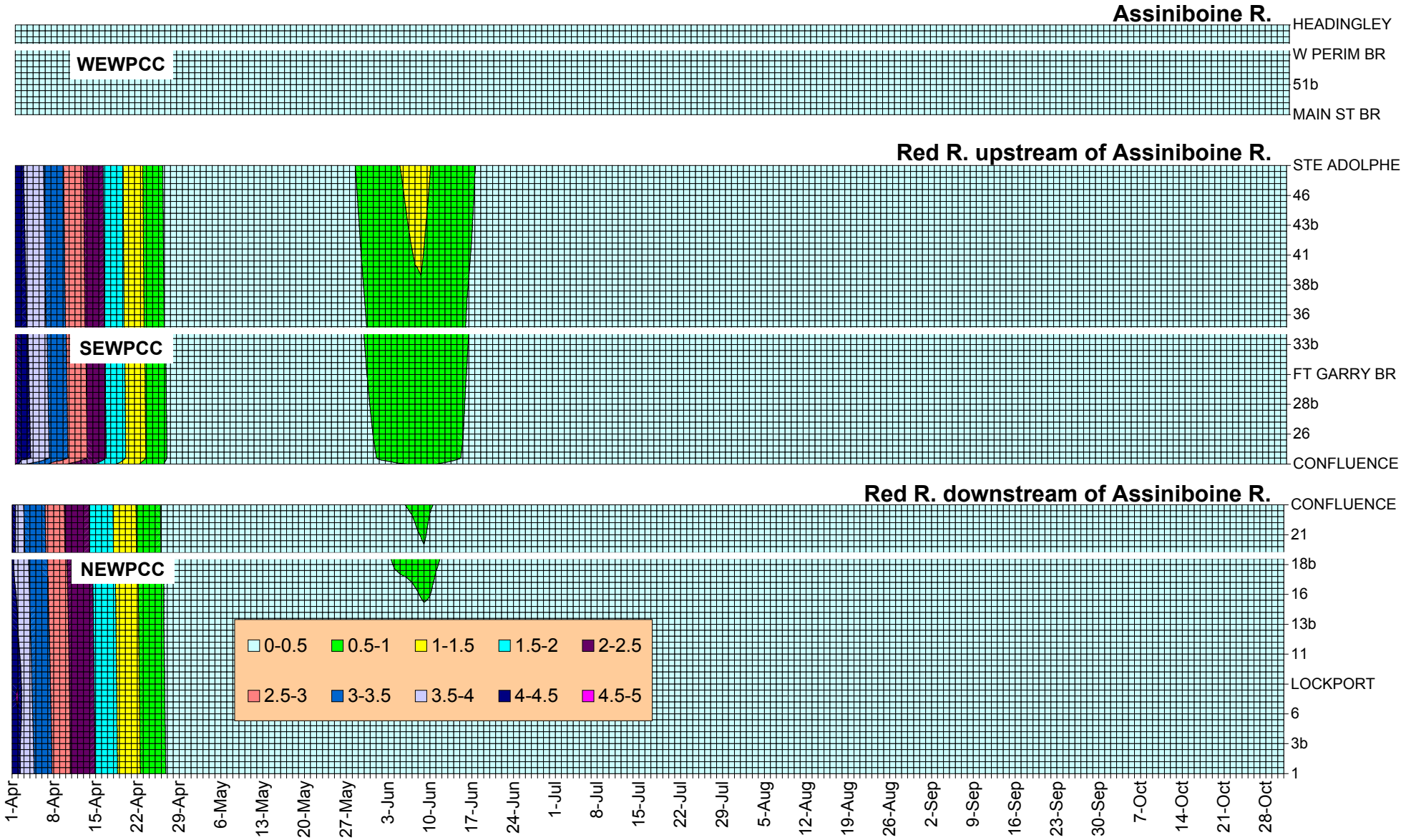
2031 NITRATE/NITRITE WITH PROJECT (mg/L) - median flow

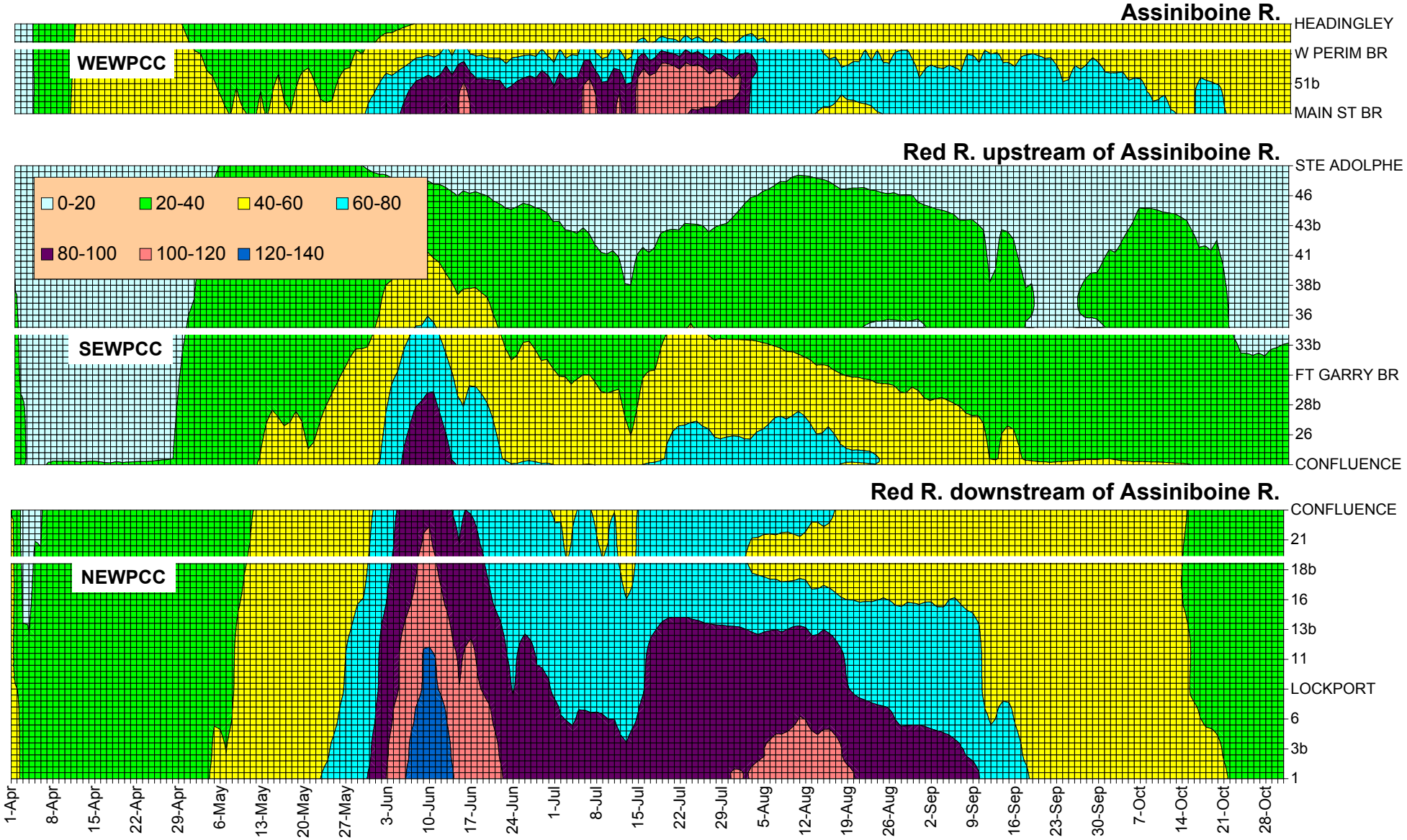


2031 AMMONIA + NITRATE/NITRITE WITHOUT PROJECT (mg/L) - median flow

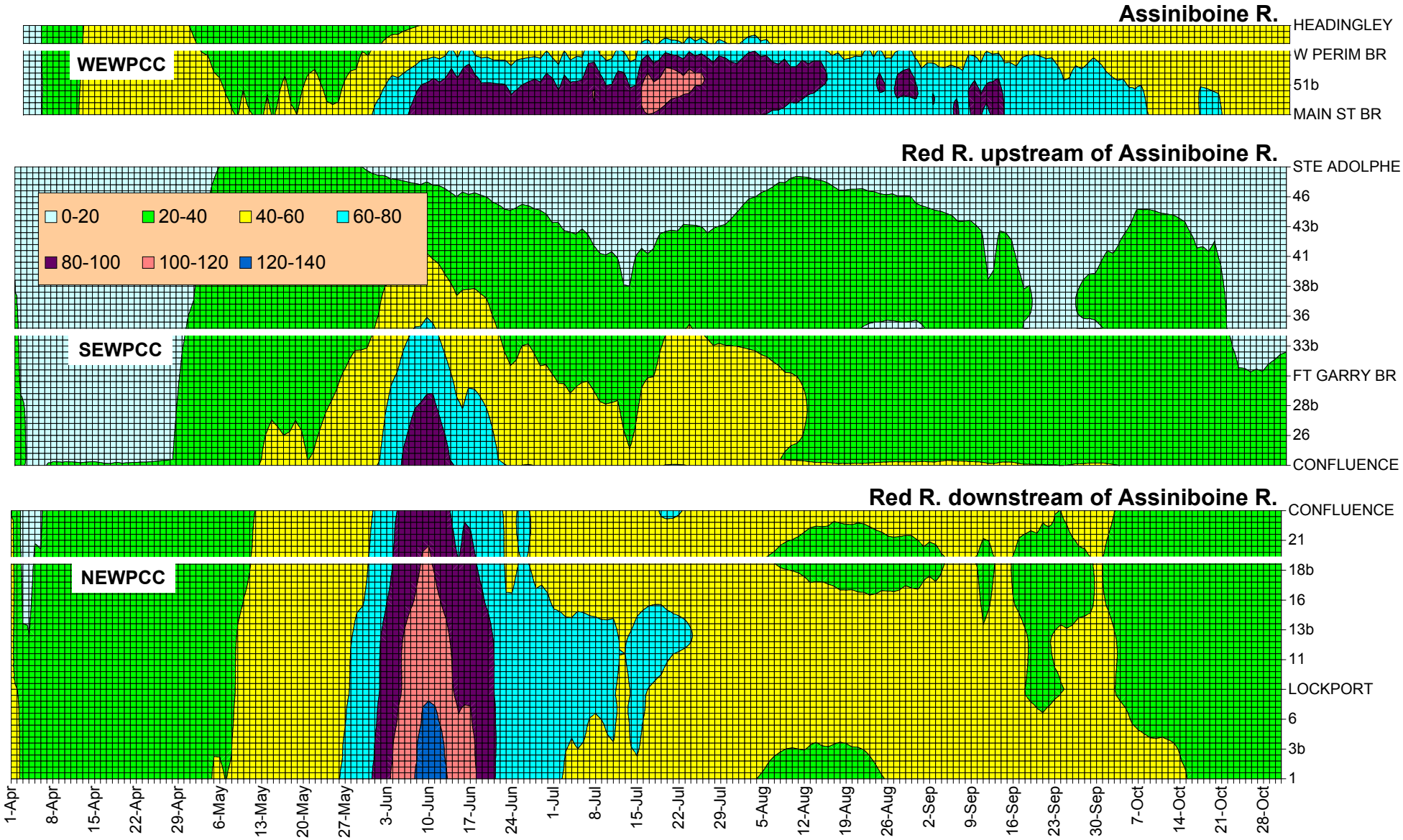


2031 AMMONIA + NITRATE/NITRITE WITH PROJECT (mg/L) - median flow

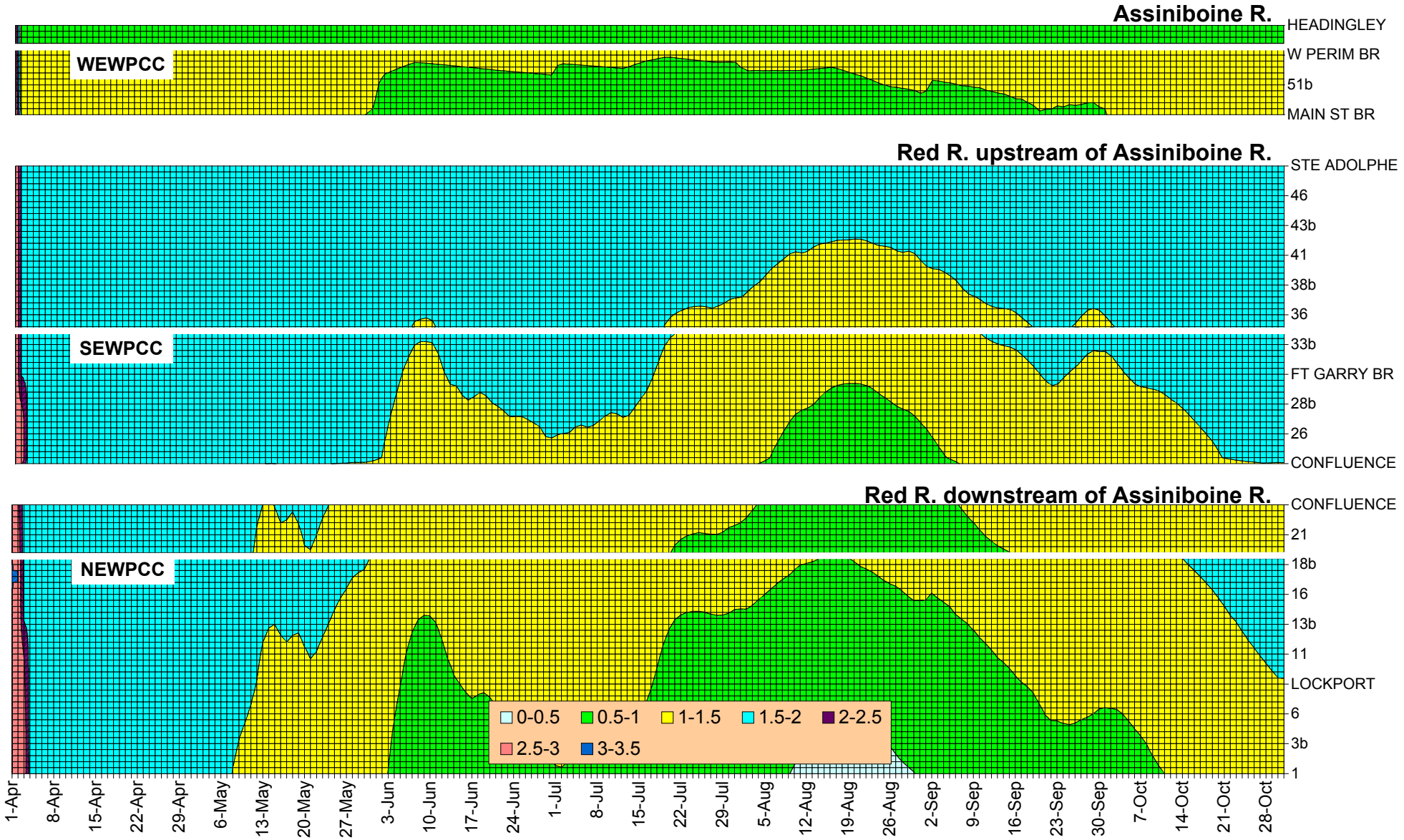



2031 CHLOROPHYLL-a WITHOUT PROJECT (ug/L) - median flow
Assiniboine R.
HEADINGLEY
W PERIM BR
51b
MAIN ST BR
WEWPCC
Red R. upstream of Assiniboine R.
STE ADOLPHE
46
43b
41
38b
36
33b
FT GARRY BR
28b
26
CONFLUENCE
0-20
20-40
40-60
60-80
80-100
100-120
120-140
SEWPCC
Red R. downstream of Assiniboine R.
CONFLUENCE
21
18b
16
13b
11
LOCKPORT
6
3b
1
NEWPCC
1-Apr
8-Apr
15-Apr
22-Apr
29-Apr
6-May
13-May
20-May
27-May
3-Jun
10-Jun
17-Jun
24-Jun
1-Jul
8-Jul
15-Jul
22-Jul
29-Jul
5-Aug
12-Aug
19-Aug
26-Aug
2-Sep
9-Sep
16-Sep
23-Sep
30-Sep
7-Oct
14-Oct
21-Oct
28-Oct

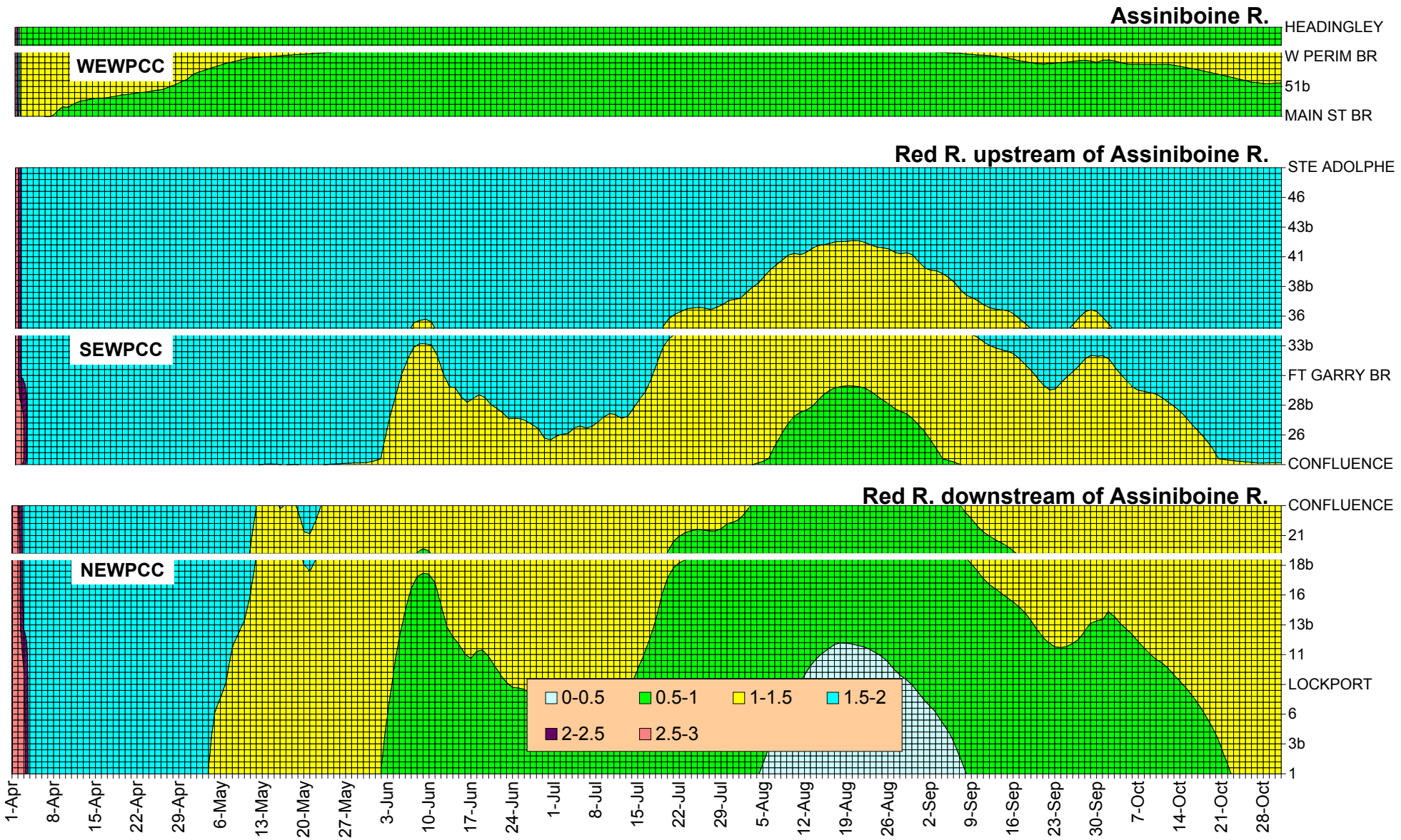
2031 CHLOROPHYLL-a WITH PROJECT (ug/L) - median flow



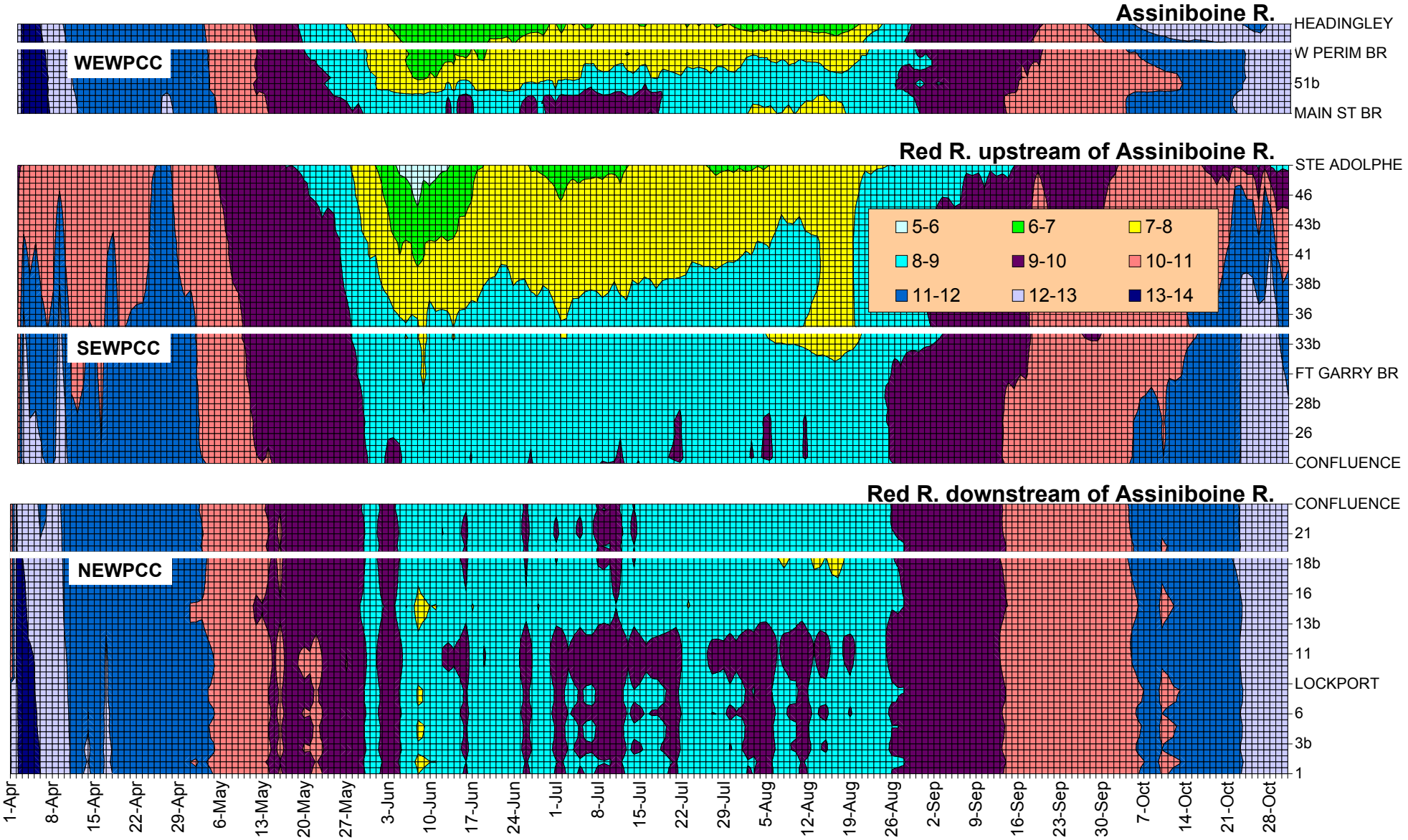
2031 CBOD WITHOUT PROJECT (mg/L) - median flow



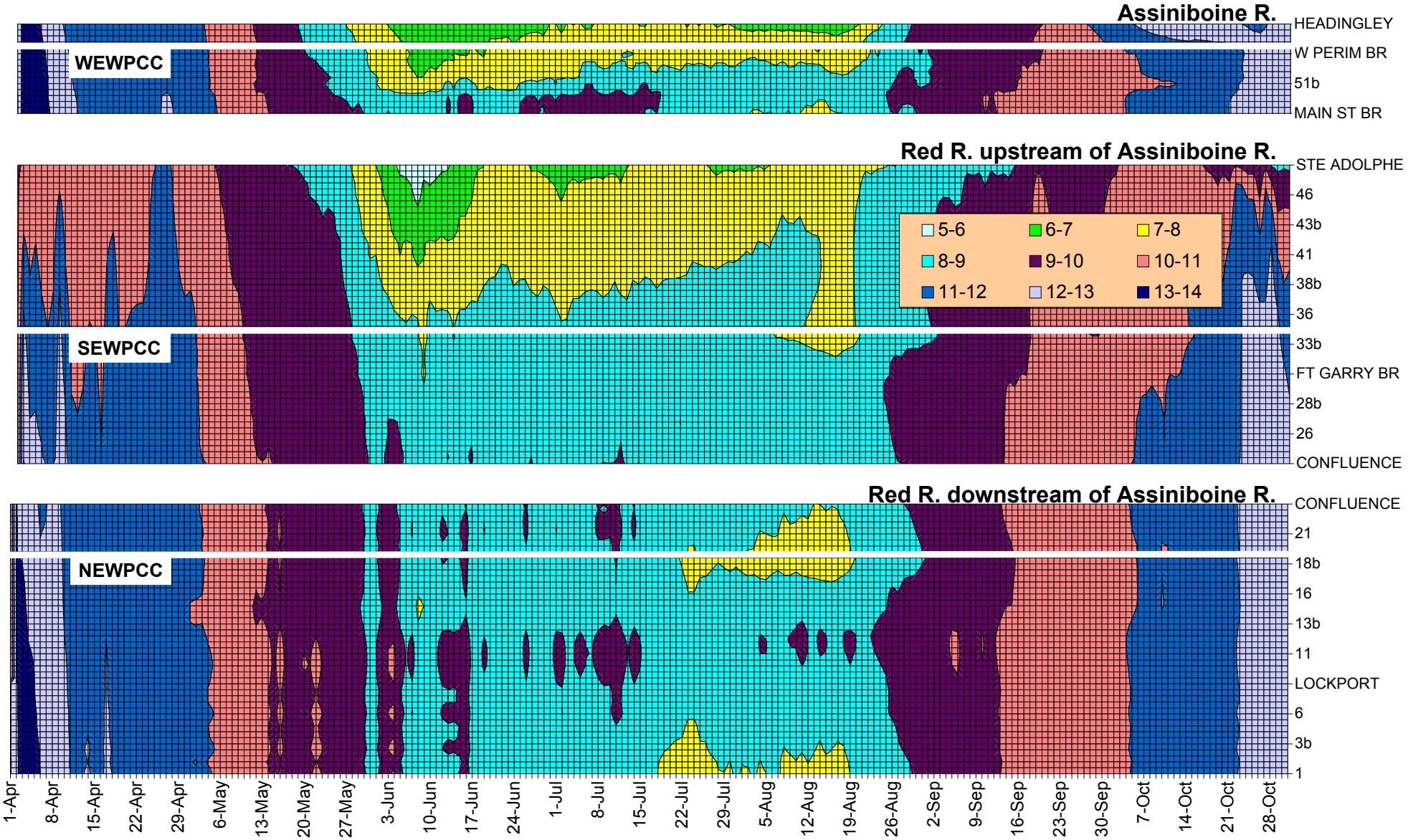
2031 CBOD WITH PROJECT (mg/L) - median flow



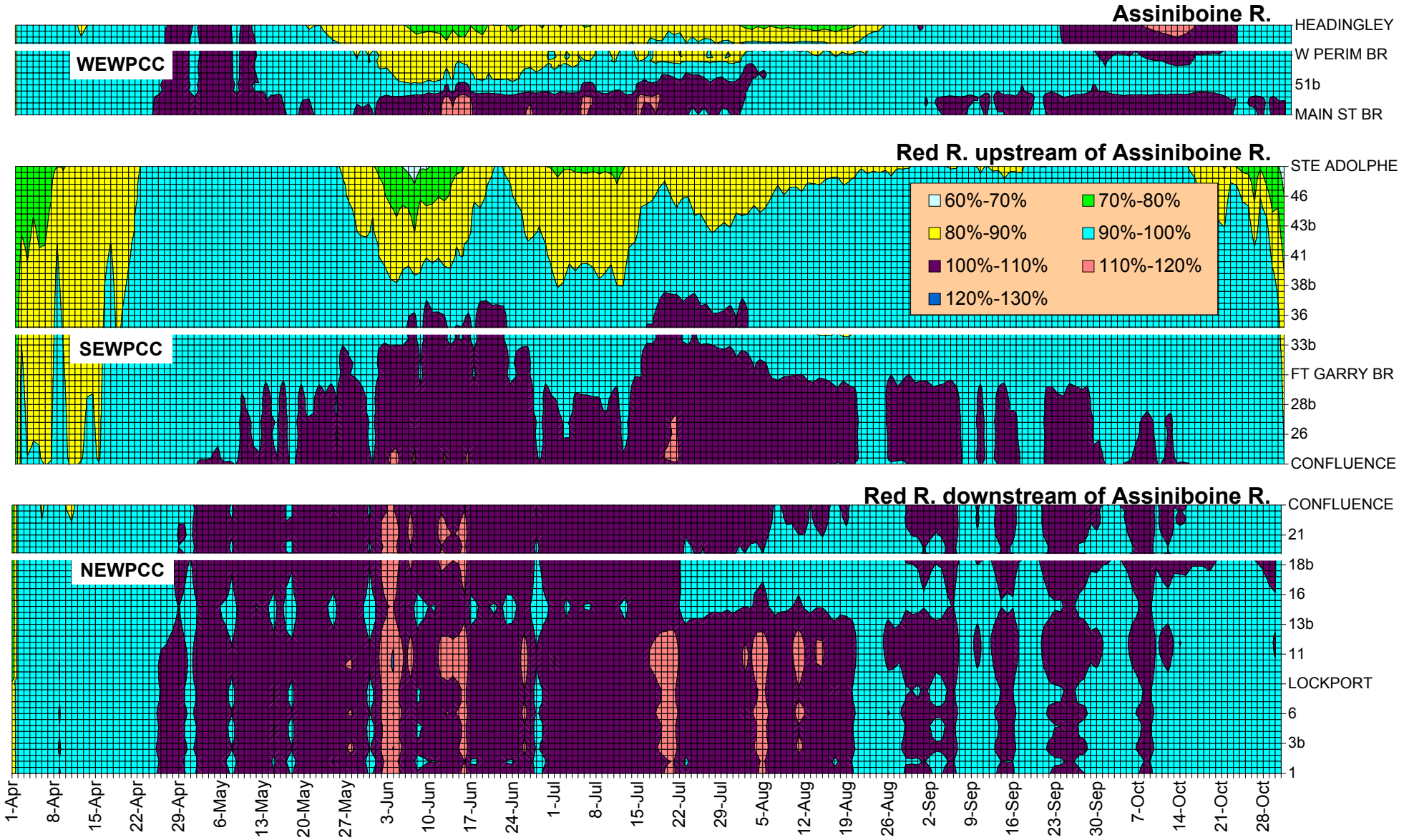
2031 DISSOLVED OXYGEN WITHOUT PROJECT (mg/L) - median flow



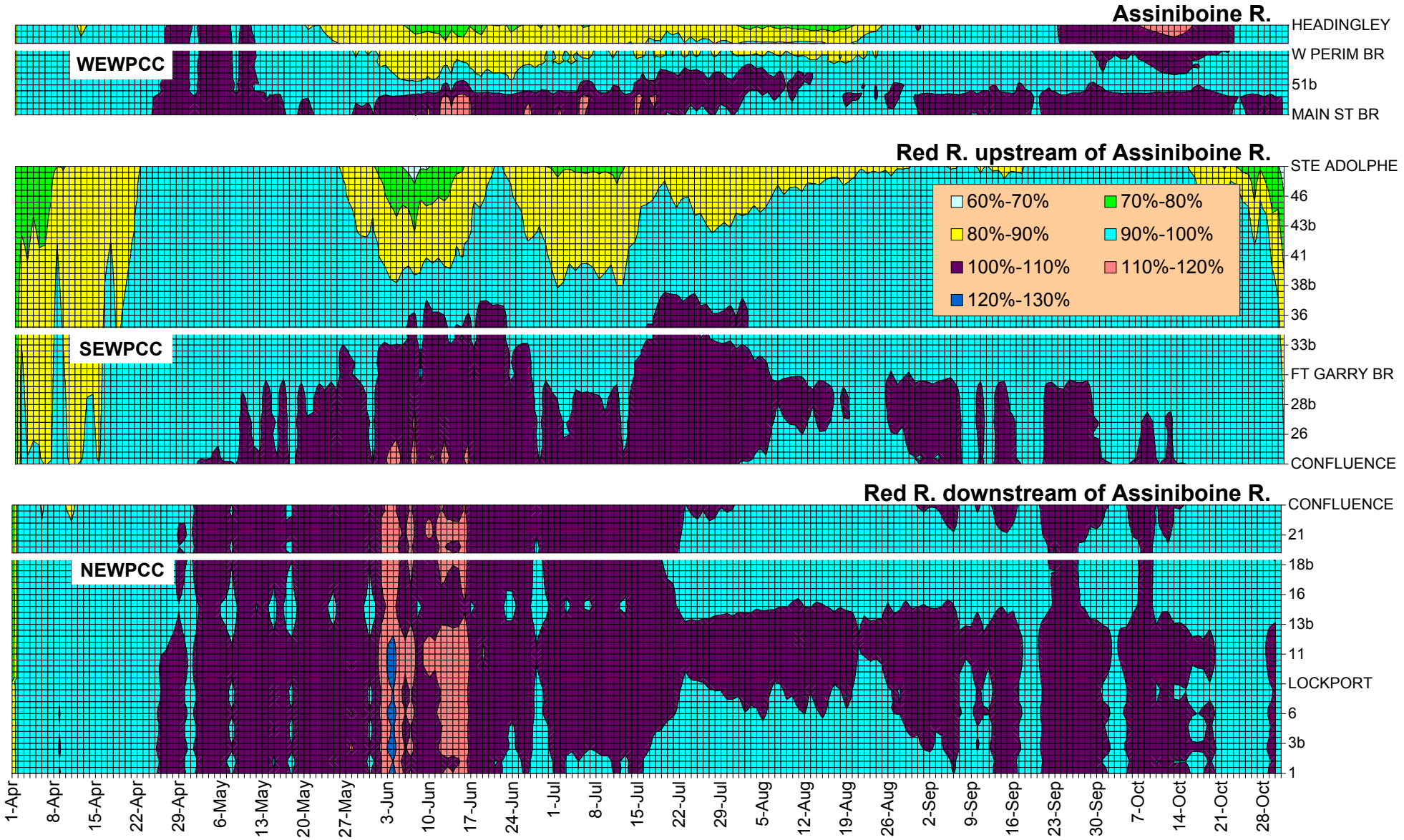
2031 DISSOLVED OXYGEN WITH PROJECT (mg/L) - median flow

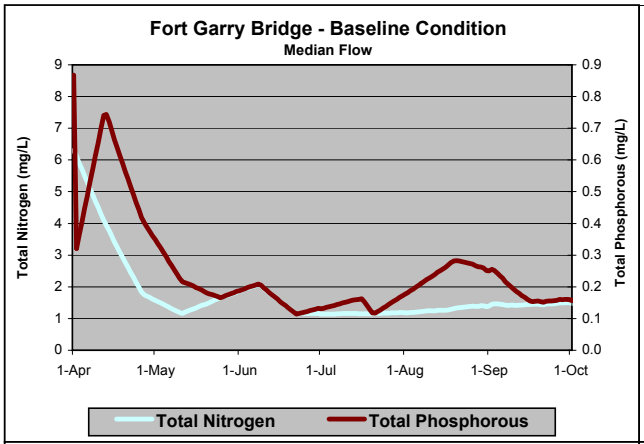


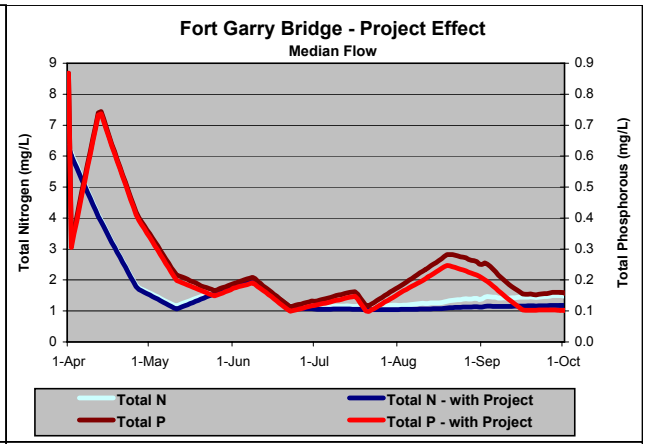
2031 MINIMUM DO SATURATION WITHOUT PROJECT - median flow

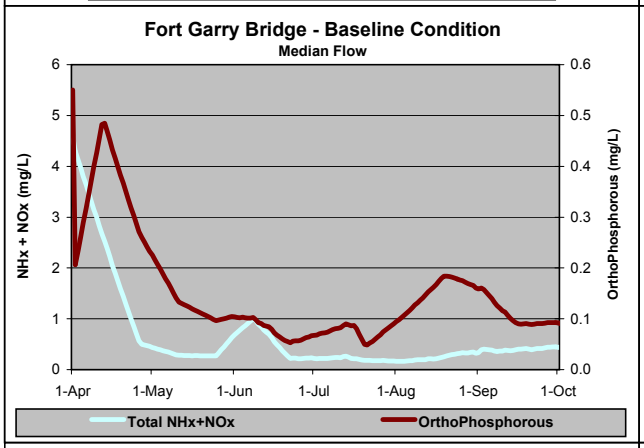


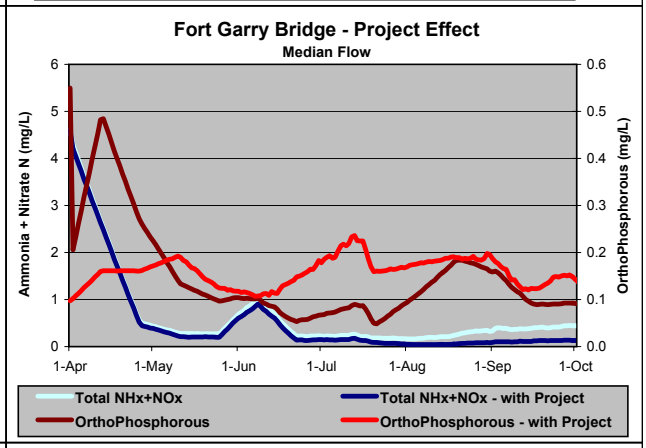
2031 MINIMUM DO SATURATION WITH PROJECT (mg/L) - median flow

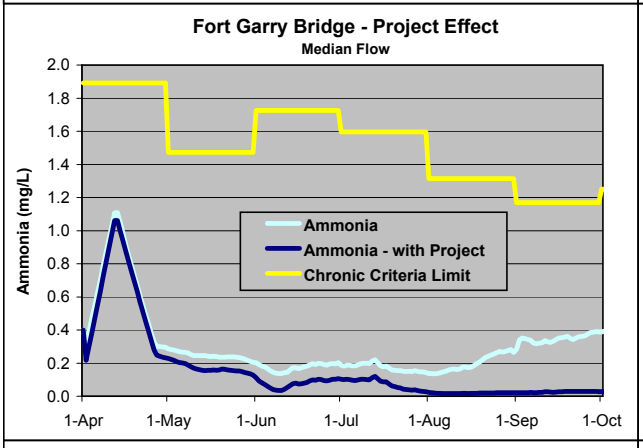


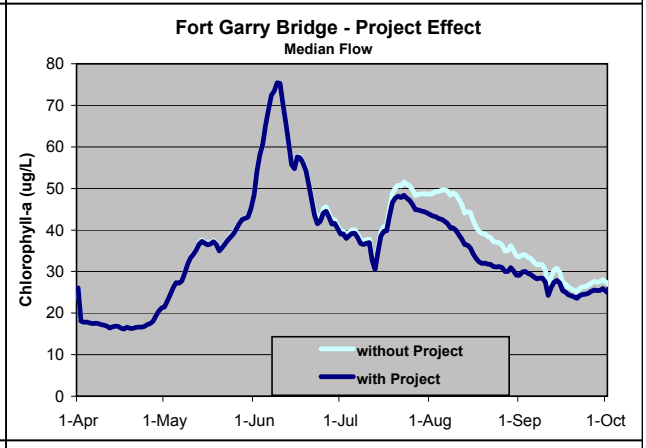

Fort Garry Bridge - Baseline Condition
Median Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total Nitrogen
Total Phosphorous

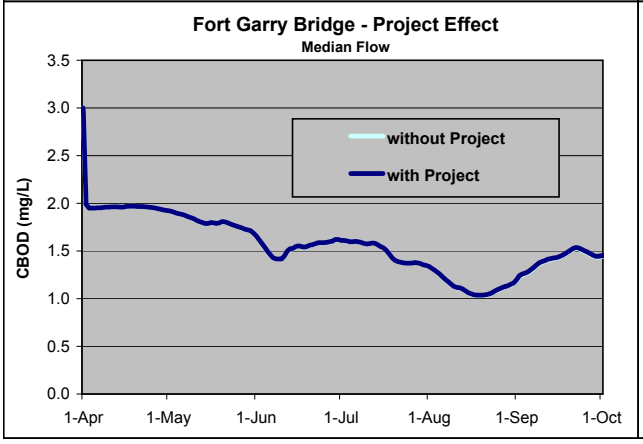

Fort Garry Bridge - Project Effect
Median Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total N
Total N - with Project
Total P
Total P - with Project

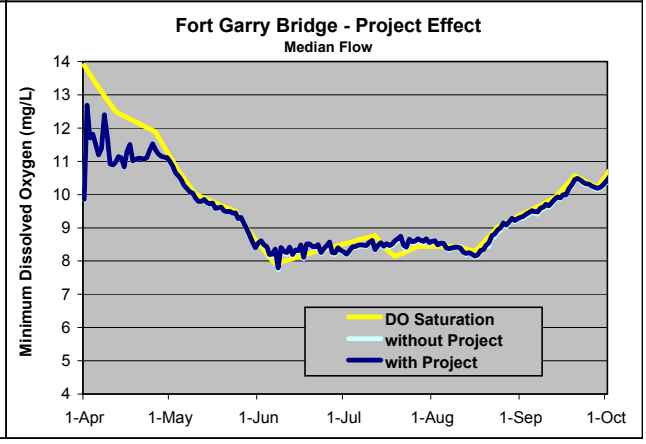

Fort Garry Bridge - Baseline Condition
Median Flow
NHx + NOx (mg/L)
OrthoPhosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx
OrthoPhosphorous

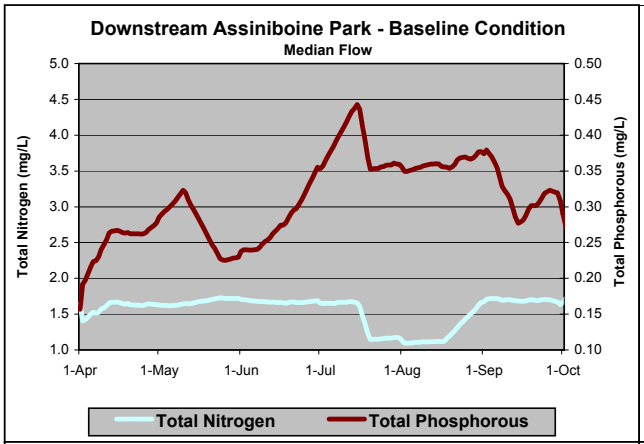

Fort Garry Bridge - Project Effect
Median Flow
Ammonia + Nitrate N (mg/L)
OrthoPhosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx
Total NHx+NOx - with Project
OrthoPhosphorous
OrthoPhosphorous - with Project

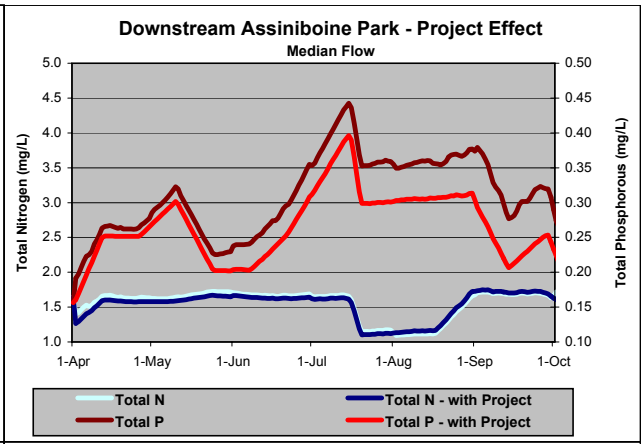

Fort Garry Bridge - Project Effect
Median Flow
Ammonia (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Ammonia
Ammonia - with Project
Chronic Criteria Limit

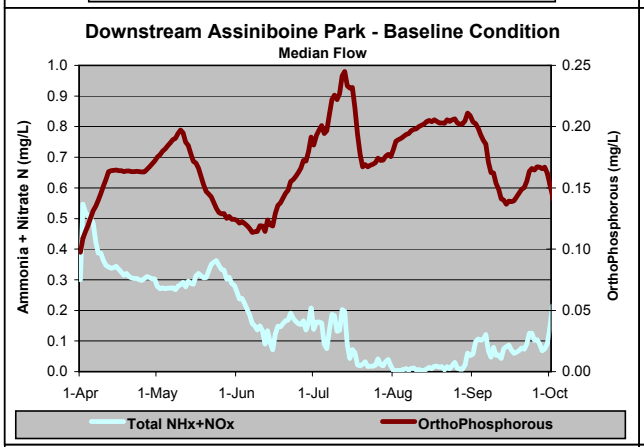

Fort Garry Bridge - Project Effect
Median Flow
Chlorophyll-a (ug/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
without Project
with Project

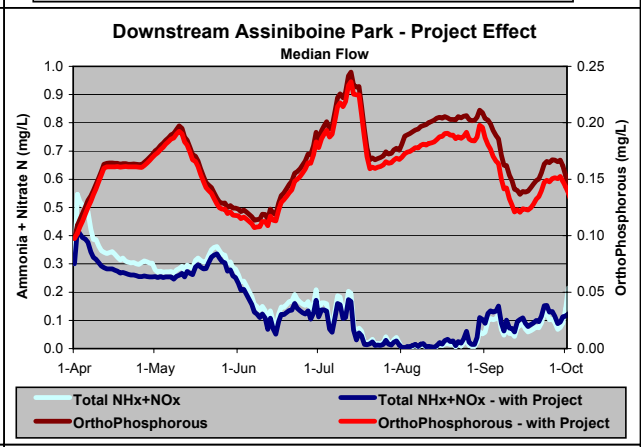

Fort Garry Bridge - Project Effect
Median Flow
CBOD (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
without Project
with Project


Fort Garry Bridge - Project Effect
Median Flow
Minimum Dissolved Oxygen (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
DO Saturation
without Project
with Project


Downstream Assiniboine Park - Baseline Condition
Median Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total Nitrogen
Total Phosphorous


Downstream Assiniboine Park - Project Effect
Median Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total N
Total N - with Project
Total P
Total P - with Project


Downstream Assiniboine Park - Baseline Condition
Median Flow
Ammonia + Nitrate N (mg/L)
OrthoPhosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx
OrthoPhosphorous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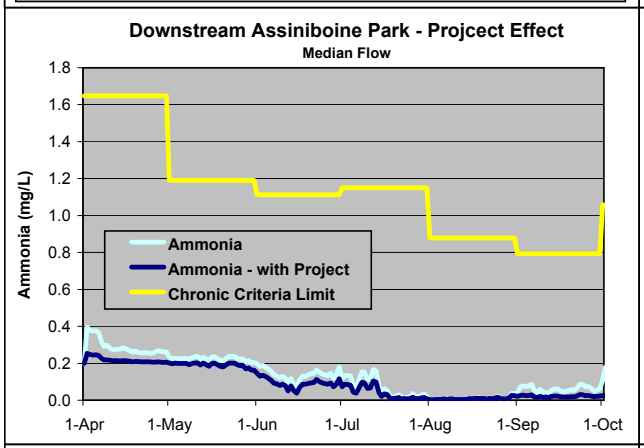
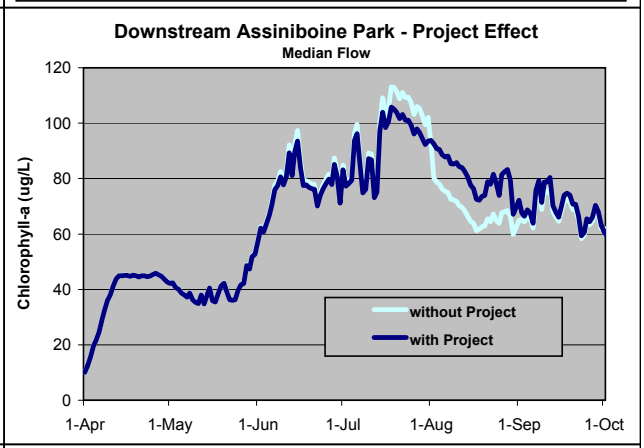
Median Flow
Ammonia + Nitrate N (mg/L)
OrthoPhosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx
Total NHx+NOx - with Project
OrthoPhosphorous
OrthoPhosphorous - with Project


Downstream Assiniboine Park - Projcect Effect
Median Flow
Ammonia (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Ammonia
Ammonia - with Project
Chronic Criteria Limit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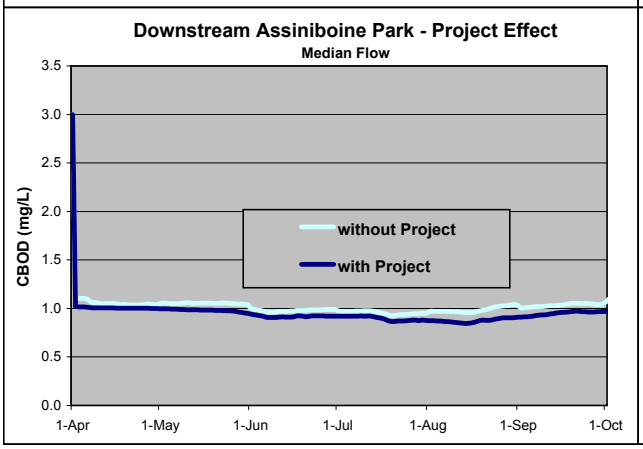
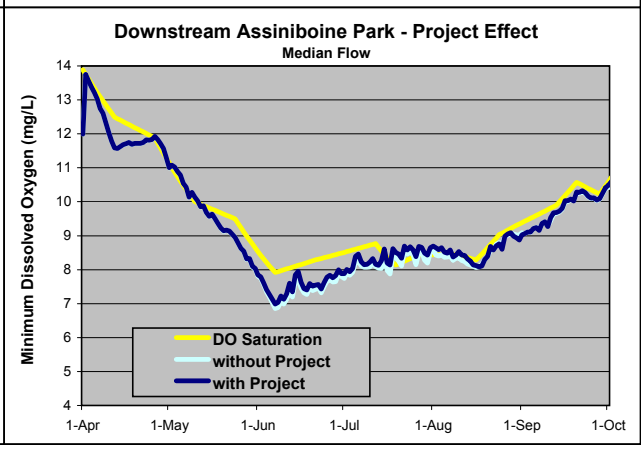
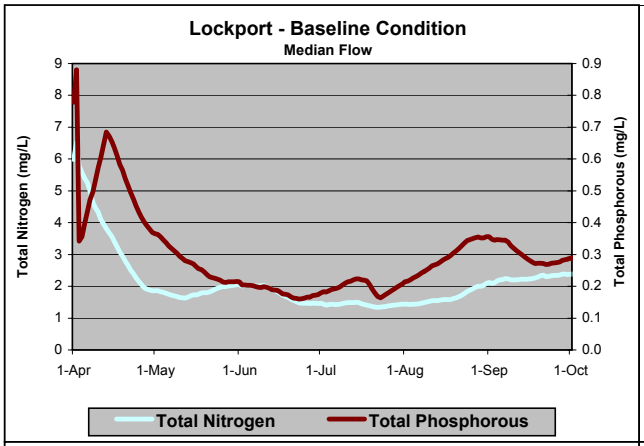
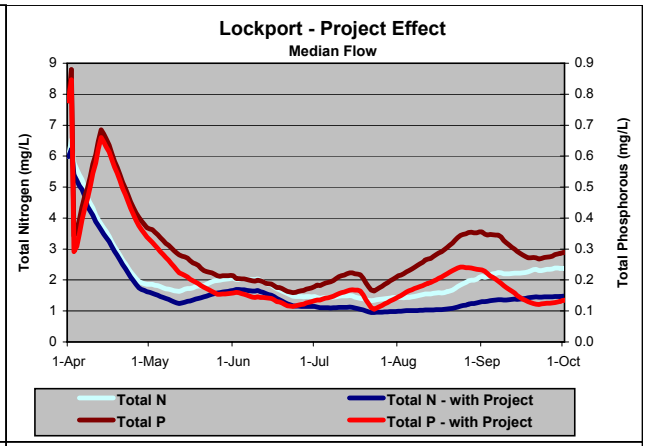
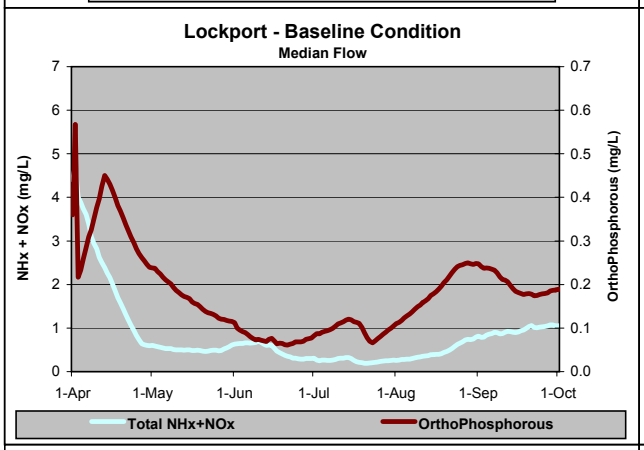
Median Flow
Chlorophyll-a (ug/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
without Project
with Project

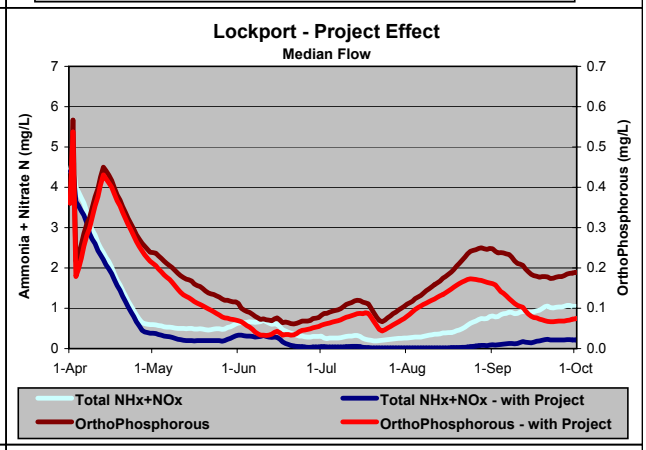

Downstream Assiniboine Park - Project Effect
Median Flow
CBOD (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
without Project
with Project

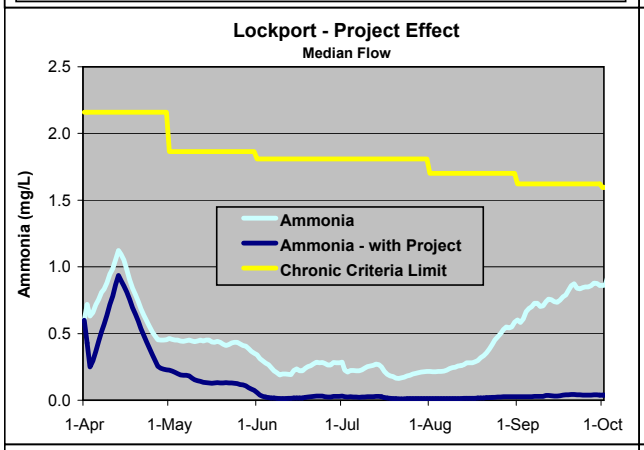

Downstream Assiniboine Park - Project Effect
Median Flow
Minimum Dissolved Oxygen (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
DO Saturation
without Project
with Project

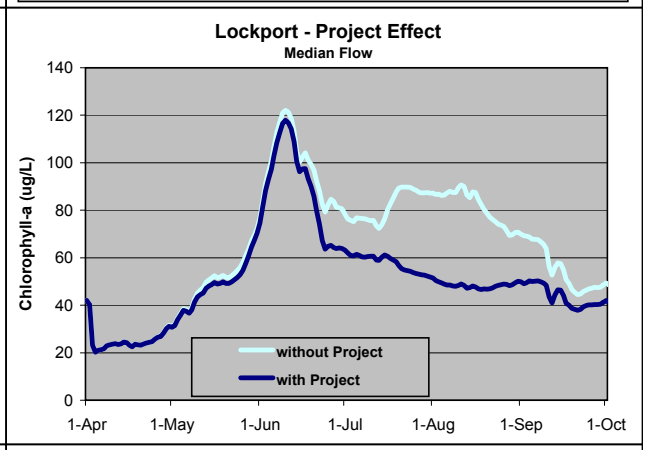

Lockport - Baseline Condition
Median Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total Nitrogen
Total Phosphorous

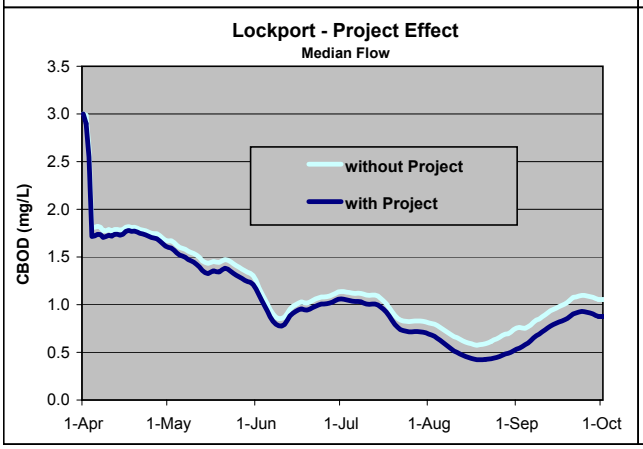

Lockport - Project Effect
Median Flow
Total Nitrogen (mg/L)
Total Phosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total N
Total N - with Project
Total P
Total P - with Project

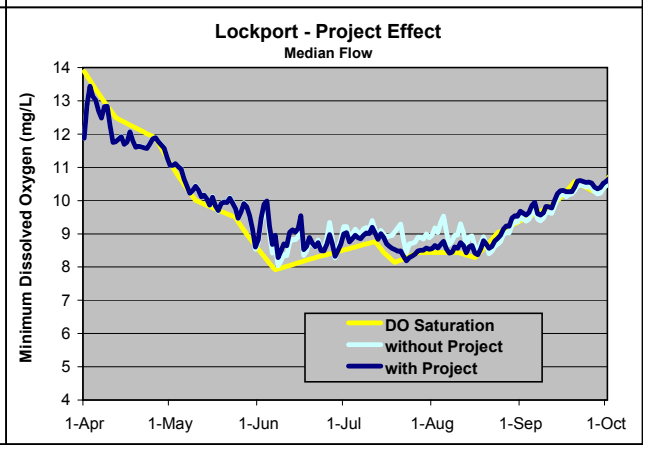

Lockport - Baseline Condition
Median Flow
NHx + NOx (mg/L)
OrthoPhosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx
OrthoPhosphorous

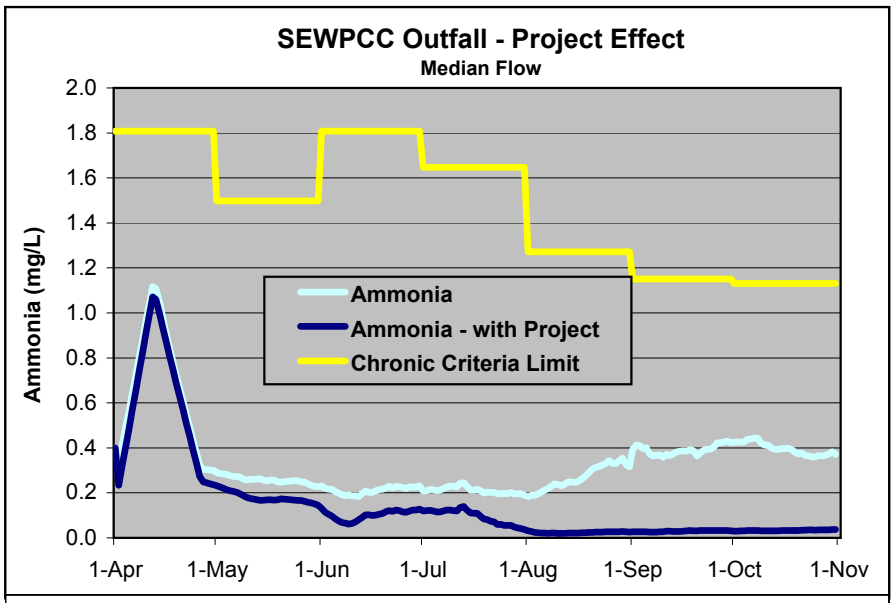

Lockport - Project Effect
Median Flow
Ammonia + Nitrate N (mg/L)
OrthoPhosphorous (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Total NHx+NOx
Total NHx+NOx - with Project
OrthoPhosphorous
OrthoPhosphorous - with Project

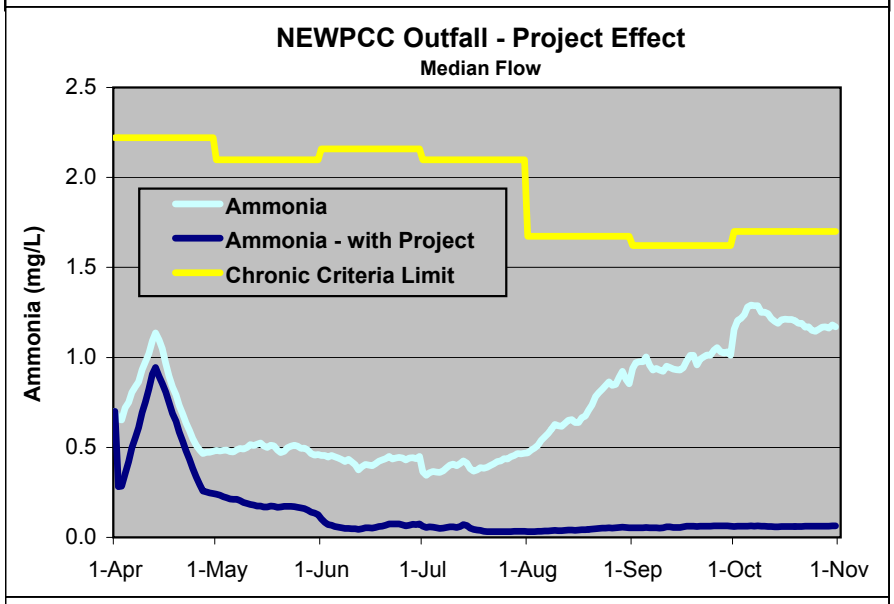

Lockport - Project Effect
Median Flow
Ammonia (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
Ammonia
Ammonia - with Project
Chronic Criteria Limit

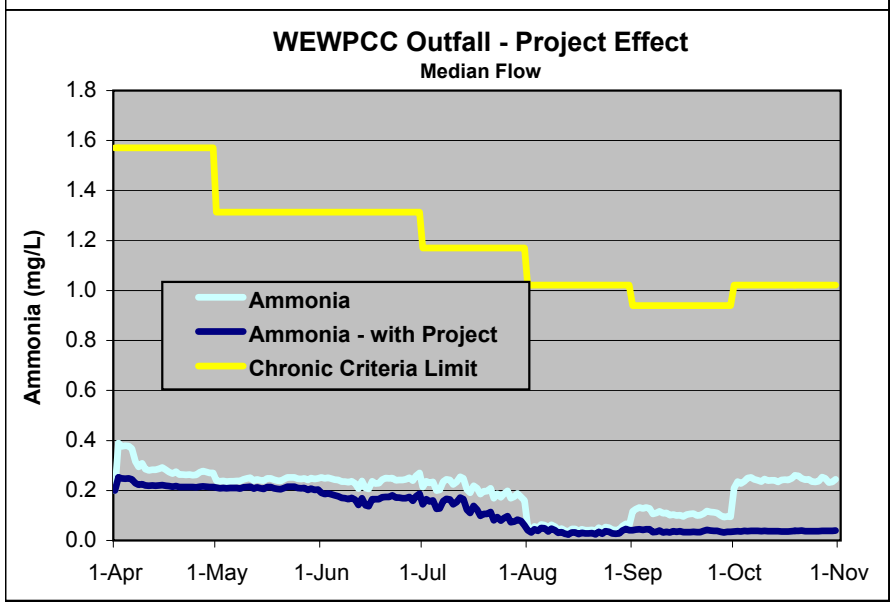

Lockport - Project Effect
Median Flow
Chlorophyll-a (ug/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
without Project
with Project


Lockport - Project Effect
Median Flow
CBOD (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
without Project
with Project


Lockport - Project Effect
Median Flow
Minimum Dissolved Oxygen (mg/L)
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
DO Saturation
without Project
with Project


SEWPCC Outfall - Project Effect
Median Flow
Ammonia (mg/L)
2.0
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0.0
Ammonia
Ammonia - with Project
Chronic Criteria Limit
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
1-Nov


NEWPCC Outfall - Project Effect
Median Flow
Ammonia (mg/L)
2.5
2.0
1.5
1.0
0.5
0.0
Ammonia
Ammonia - with Project
Chronic Criteria Limit
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
1-Nov


WEWPCC Outfall - Project Effect
Median Flow
Ammonia (mg/L)
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0.0
Ammonia
Ammonia - with Project
Chronic Criteria Limit
1-Apr
1-May
1-Jun
1-Jul
1-Aug
1-Sep
1-Oct
1-Nov

Environmental Assessment of

Canadian Strategic Infrastructure Funded

Upgrades to the City of Winnipeg

Water Pollution Control Centres

APPENDIX 8D

Literature Review of

Endocrine Disrupting Compounds

1.0 INTRODUCTION

Endocrine-disrupting substances are chemicals that interfere with the normal functioning of the endocrine system of complex organisms (primarily animals). Endocrine-disrupting compounds (EDCs) can interfere with the endocrine systems in a variety of ways, including (US EPA 2004):

- Mimicking a natural hormone, causing an over response to the stimulus.
- Responding at inappropriate times (i.e., creating hormones when not required).
- Blocking the effects of a hormone from certain receptors.
- Stimulating the endocrine system and cause overproduction of hormones.
- Inhibiting the endocrine system and causing underproduction of hormones.

A literature review on the effects of EDCs in the aquatic environment was conducted to support the assessment. The purpose of this literature review was to:

- Identify the types and concentrations of EDCs typically found in municipal wastewater effluent.
- Identify the likely concentrations at which these EDCs are expected to cause effects to the aquatic environment.
- Identify the potential effects of these compounds on the aquatic environment.
- Identify the potential effects of EDCs on aquatic species.
- Examine the effectiveness of various wastewater treatment processes in reducing EDCs.

This literature review focused particularly on aquatic effects from wastewater treatment plant (WWTP) effluent, although effects from other sources were also considered.

2.0 SOURCES OF EDCs IN THE AQUATIC ENVIRONMENT

The presence of these compounds in the aquatic environment has been shown to lead to detrimental effects in aquatic species. Even low levels of these compounds in the environment can affect the growth, reproduction, and development of organisms (Environment Canada 2002).

Many substances are considered to be EDCs (Environment Canada 2002). These include pesticides, industrial chemicals, surfactants, natural and synthetic hormones, and pharmaceuticals. These compounds are found in industrial, municipal and agricultural wastes, and are released to the environment from both point and non-point sources, including wastewater treatment plant (WWTP) effluent (Environment Canada 2002).

McMaster (2001) reviewed of the evidence of endocrine disruption in Canadian aquatic ecosystems. In this review, the following sites and/or sectors were cited as being of potential concern for endocrine disruption in fish:

- Municipal effluents:
  - detectable levels of estradiol, estrone, and ethynylestradiol have been found in effluent from Canadian sewage treatment plants.
- Intensive agriculture, including pesticides and livestock production.
- Textile mill effluents.
- Pulp and paper sector.
- Mining and metals.
- Historically contaminated sites.
- Identified Areas of Concern, e.g., Great Lakes AOCs.
- Contaminants in the Arctic, including aboriginal foods.

This review focuses primarily on EDC effects from municipal effluent, although other sources of EDC may be relevant for the assessment of cumulative effects.

3.0 AQUATIC ENVIRONMENT EFFECTS

3.1 WWTP EFFLUENT

A database search was conducted to obtain scientific studies on the types and concentrations of EDCs found in WWTP effluent and the EDC effects of WWTP effluent on aquatic life. Many studies were available and these studies are discussed in this section.

3.1.1 Concentrations of EDCs in WWTP effluent

Endocrine-disrupting compounds, particularly natural and synthetic estrogens along with nonylphenol ethoxylates, have been detected in WWTP effluent in various locations throughout the world. The concentrations of EDCs in WWTP effluent have been measured at ng/L in the case of estrogens and µg/L in the case of nonylphenol ethoxylates. The types of EDCs and maximum detected concentrations of these compounds measured in WWTP effluent are shown in Table 3-1.

Other studies have also shown that WWTP effluent can be estrogenic in nature. Burnison *et al.* (2002) measured the estrogenicity and androgenicity of WWTP effluent and pulp and paper mill discharges into the Miramichi River, NB, and found the WWTP effluent was the main source of

estrogenic compounds in the river; however, they did not identify the types and concentrations of estrogens in this effluent. Svenson and Allard (2004) demonstrated there was androgenic activity in domestic WWTP effluent in Sweden, indicating the presence of androgenic EDCs in WWTP effluent. The type and concentrations of androgen EDCs present in this WWTP effluent were not measured (Svenson and Allard 2004). Only one study was found on androgenic EDC effects in aquatic species but many were available on estrogenic effects. The effects on the aquatic environment of concentrations of EDCs typically found in WWTP effluent are discussed below.

Table 3-1
Concentrations of EDCs Measured in WWTP Effluent

EDC	Maximum Concentration detected	Location	Source
17β-estradiol (E2)	up to 3.66 ng/L	South central Michigan	Snyder *et al.* (1999)
	48 ng/L	United Kingdom	Desbrow *et al.* (1998)
	3.5 ng/L maximum ng/L median	Italy	Baronti *et al*. (2000)
	1.1 ng/L	Sweden	Larsson *et al*. (1999)
	4.6 ng/L (average concentration)	Japan	Nakada *et al.* (2004)
	4 ng/L	United States	Kolodziej *et al.* (2003)
	2 ng/L (90^{th} percentile) 3 ng/L maximum	Germany	Ternes *et al.* (1999)
	6 ng/L (median) 14 ng/L (90^{th} percentile) 64 ng/L (maximum)	Ontario, Canada	Ternes *et al.* (1999)
Estrone (E1)	76 ng/L	United Kingdom	Desbrow *et al.* (1998)
	82.1 ng/L (maximum) 9.3 ng/L (median)	Italy	Baronti *et al.* (2000)
	5.8 ng/L	Sweden	Larsson *et al.* (1999)
	47 ng/L	The Netherlands	Belfroid *et al* (1999)
	33 ng/L	Japan	Nakada *et al.* (2004)
	12 ng/L	United States	Kolodziej *et al.* (2003)
	2.56 ng/L	Calgary	Chen *et al.* (2005)
	9 ng/L (median) 22 ng/L (90^{th} precentile) 70 ng/L (maximum)	Germany	Ternes *et al.* (1999)
	3 ng/L (median) 10 ng/L (90^{th} percentile) 48 ng/L (maximum)	Ontario, Canada	Ternes *et al.* (1999)
Ethinyl estradiol (EE2)	up to 759 pg/L	Michigan	Snyder *et al.* (1999)
	7,000 pg/L	Britain	Desbrow *et al.* (1998)

Table 3-1
Concentrations of EDCs Measured in WWTP Effluent

EDC	Maximum Concentration detected	Location	Source
Ethinyl estradiol (EE2) (cont'd.)	1,700 pg/L (maximum) 450 pg/L (minimum)	Italy	Baronti *et al.* (2000)
	1 ng/L (median) 4 ng/L (90th percentile) 15 ng/L (ng/L)	Germany	Ternes *et al.* (1999)
	9 ng/L (median) 29 ng/L (90th percentile) 42 ng/L (maximum)	Ontario, Canada	Ternes *et al.* (1999)
Nonylphenol	3 µg/L	United Kingdom	Lye *et al.* (1999)
	up to 37 µg/L	Michigan, USA	Snyder *et al.* (1999)
	0.84 µg/L	Sweden	Larsson *et al.* (1999)
	3 µg/L	United Kingdom	Lye *et al.* (1999)
	0.564 µg/L	Japan	Nakada *et al*. (2004)
Nonylphenol polyethoxylate	up to 332 µg/L	Michigan	Snyder *et al.* (1999)
Nonylphenol monoethoxylate	45 µg/L	United Kingdom	Lye *et al.* (1999)
Alkylphenol polyethoxylates	30 µg/L	Massachusetts, USA	Rudel *et al.* (1998)
Octylphenol	up to 0.7 µg/L	Michigan	Snyder *et al.* (1999)
Bisphenol A	490 ng/L	Sweden	Larsson *et al.* (1999)
	258 ng/L	Germany	Hansen *et al.* (1998)
	8-33 ng/L	Germany	Hansen *et al.* (1998)
	27 ng/L	Japan	Nakada *et al.* (2004)
	2 – 5.5 ng/L	Massachusetts, USA	Rudel *et al.* (1998)

3.1.2 Potential EDC Effects on the Aquatic Environment from WWTP Effluent

Studies of the effects of EDCs in WWTP effluent on the aquatic environment fall into three broad categories:

- Studies that involve in-situ exposure of fish in cages to WWTP.
- Exposure of fish to WWTP effluent in laboratory studies.
- Assessment of wild fish near WWTP effluent outflows.

The results that have been obtained by each of these categories are discussed in this section.

3.1.2.1 In-Situ Exposure Studies

A number of studies on the effects of EDCs in WWTP effluent on aquatic life have been conducted via in-situ research. Generally, these studies have focused on the estrogenic effects of WWTP effluent.

Svenson *et al.* (2002) exposed juvenile rainbow trout (*Oncorhynchus mykiss*) to wastewater from WWTP outflows in Sweden and found that only moderate increases in vitellogenin (an indication of exposure to estrogenic materials [Sumpter and Jobling 1995]) occurred in the fish. The measured estrogenicity of the WWTP effluent outflows in this study ranged up to 15 ng/L 17-b-estradiol equivalents. The same study also included exposure of fish to undiluted domestic WWTP effluent and the results showed the effluent was estrogenic. This study did not include an evaluation of other effects associated with the exposures.

Another study of WWTP effluent effects on rainbow trout conducted in Sweden by Larsson *et al.* (1999) involved exposure of juvenile rainbow trout contained in cages downstream of a WWTP outflow. Larsson *et al.* (1999) found that the WWTP effluent contained measurable levels of EDCs including estrone, 17β-estradiol, 17α-ethinyl estradiol, nonylphenol and bisphenol A. Fish exposed downstream of the outflow had bile containing concentrations of EDCs that were 1,000 to 1,000,000 times greater than concentrations measured in the water. Larsson *et al.* (1999) also noted that a physiological disturbance caused by the presence of estrogenic EDCs in the water was demonstrated by the induction of vitellogenesis in exposed fish. A similar study, using adult male rainbow trout, was conducted in the United Kingdom by Harries *et al.* (1996). Harries *et al.* (1996) found that fish held up to 4.5 km downstream of WWTP effluent outflows showed statistically significant increases in plasma vitellogenin levels. rainbow trout exposure to WWTP effluent in Sweden demonstrated that bile estrogenicity in exposed fish was elevated compared to the control exposures (Allard *et al.* 2004). While these studies demonstrate that exposure of rainbow trout to WWTP effluent does produce an EDC-related effect on this fish species (i.e., increased vitellogenin levels), it is uncertain from these studies what type of long-term ecological effects could result from EDC exposure.

Other studies have demonstrated a link between exposure to WWTP effluent and effects on aquatic species. For example, Gagne *et al.* (2002) exposed *Elliptio complanata* mussels to a WWTP effluent plume and found prolonged exposure to the plume skewed the sex ratio in favour of females. Tilton *et al.* (2002) exposed male channel catfish (*Ictalurus punctatus*) to WWTP effluent at a location in Mississippi. Fish were kept in cages and exposed to the effluent for a 21-day period with each experiment being repeated during the spring and fall for three years. Increases in vitellogenin levels were observed during some of the exposure periods,

indicating that the WWTP did affect the fish. Significant increases in vitellogenin-like proteins were also found in Zebra Mussels (*Dreissena polymorpha*) exposed to WWTP effluent in Ireland (Quinn *et al.* 2004) potentially indicating endocrine disruption-related effects.

Other scientific research has not demonstrated a link between exposure to WWTP effluent and EDC-related effects. Nichols *et al.* (1999) conducted an experiment involving the in-situ exposure of fathead minnow (*Pimephales promelas*) to WWTP effluent in central Michigan. In this study, caged male and female fish were exposed to WWTP effluent at seven different outflows and compared to control fish at riverine and lacustrine sites. The authors did not find any trends in hormone concentrations, male secondary sex characteristics or gonad histology in the exposed fish that could be attributed to the WWTP effluent. These results were similar to the results obtained by Geisy *et al.* (2003) in a similar study involving goldfish (*Carassius auratus*). In both studies the authors note the results obtained did not agree with results obtained by researchers in the United Kingdom, UK and speculated that reasons for this discrepancy may include:

- Trickling filter treatment technologies are more common in the UK.
- There are greater volumes of flow from WWTP.
- There is less dilution in receiving streams as compared to the U.S (Nichols *et al.* 1999, Geisy *et al.* 2003) causing increased EDC concentrations to be discharged to aquatic receiving environments.

Another study conducted in the United States did, however, find evidence of EDC exposure in fathead minnow exposed to WWTP effluent. The study by Hemming *et al.* (2001) involved the examination of the estrogenicity of a WWTP effluent flowing through a constructed wetland. Vitellogenin levels in fathead minnow were found to be significantly elevated in fish exposed to WWTP effluent at the inflow site. Furthermore, fish exposed at this site showed reduced gonadosomatic index (GSI) and increased hepatosomatic index (HSI) indicative of probable estrogenic effects from the effluent. Neither Nichols *et al.* (1999) or Geisy *et al.* (2003) measured EDC levels in the streams where the studies were conducted so it is not certain if the concentrations of any EDCs present were lower than would be expected to cause effects. In contrast, the Hemming *et al.* (2001) study showed a consistent presence of nonylphenol congeners (1.0 – 2.2 µg/L) and ethinyl estradiol (1.0 – 2.2 µg/L).

3.1.2.2 Laboratory Studies

Many of the laboratory studies available on EDC-related effects of exposure to WWTP effluent have been conducted with rainbow trout and all of these studies have shown WWTP effluent to

be estrogenic. Allard *et al.* (2004) found rainbow trout exposed to WWTP effluent in a laboratory setting had a bile estrogenicity that was higher than both the control and in-situ exposed fish. Gibson *et al.* (2005) also exposed rainbow trout to WWTP effluent in laboratory experiments and obtained results that agreed with the Allard *et al.* (2004) findings. Both sets of results indicated that fish exposed to WWTP effluent had considerably higher levels of estrogenic chemicals in their bile as compared to fish exposed to river water or tap water. Both studies indicate that WWTP effluent is estrogenic. Jobling *et al.* (2003) exposed rainbow trout and common carp (*Cyprinus carpio*) to WWTP effluent in a laboratory setting. Vitellogenin levels in the fish increased substantially within 3 days indicating that WWTP effluent was potentially estrogenic. Tyler *et al.* (2005) exposed rainbow trout to two different WWTP effluents, both of which had been shown to be estrogenic, in a flow-through system. They found that the more estrogenic effluent with an estrogenic activity of between 24.3 and 104.1 ng/L 17β-estradiol equivalents (E2 eq) resulted in a 700-fold induction of vitellogenin production in male fish and a 240-fold induction in female fish. The less potent effluent with an estrogenic activity of between 4.1 and 6.8 ng/L E2 eq induced vitellogenin production in fish with only a 4-fold induction in males and an 18-fold induction in females. These studies demonstrated that exposure to WWTP effluent resulted in EDC type (i.e., increased vitellogenin production) effects in rainbow trout.

Tyler *et al.* (2005) and Gibson *et al.* (2004) exposed roach (*Rutilus rutilus*) to WWTP effluent. Gibson *et al.* (2004) found the estrogens 17β-estradiol (E2), estrone (E1) and ethinyl estradiol (EE2) were all detected in the bile of effluent exposed roach and EE2 was found in the ovaries and testes of these fish. Tyler *et al.* (2005) found male roach exposed to more estrogenic effluent had elevated vitellogenin levels, while there was no change in fish exposed to less estrogenic effluent. Gagne and Blaise (2002) exposed brine shrimp (*Artemia franciscana*) to extracts from WWTP effluent and found these resulted in increases in vitellogenin levels. In general, laboratory studies have demonstrated that WWTP effluent does produce EDC-related effects and these were typically concentration-dependent and estrogenic in nature.

3.1.2.3 Aquatic Wildlife Studies

A few other studies have involved the measurement of EDC-related effects in wildlife populations downstream of WWTP outflows. McMaster *et al.* (2002) examined longnose sucker (*Catostomus catostomus*) exposed to WWTP and pulp and paper mill effluent in a northern Alberta river. Fish were found to show signs of altered reproductive fitness including altered circulating steroid and vitellogenin levels and increased levels of hepatic oxidative stress (McMaster *et al.* 2002). McMaster *et al.* (2002) noted it was difficult to separate the effects caused by the WWTP effluent from those caused by the pulp and paper mill effluent.

Other authors have also observed EDC-related effects in wild fish downstream of WWTP outflows. Hegrenes (1999) observed spawning female channel catfish on the Red River collected approximately 5 km downstream of the Fargo-Moorhead, North Dakota area with secondary sex characteristics normally attributed to male fish, an indication of androgenic effects. Hegrenes (1999) noted the effects could potentially be attributed to a number of point and/or non-point sources in the area including agricultural runoff, WWTP effluent or effluent from a sugar beet processing plant.

Bacigalupi (2004) collected walleye (*Sander vitreus*) downstream of a WWTP outflow located in St. Paul, Minnesota. Low concentrations of estrogenic compounds including estradiol, estrone and nonylphenol were detected in the effluent water. Male walleye collected from the outflow had elevated vitellogenin and estradiol levels, and decreased gonad size and testosterone levels compared to fish obtained from a control site. Male fish at the outflow did not have any expressible milt. Bacigalupi (2004) noted that the presence of estrogen in the WWTP effluent likely played a role in inhibiting expression of milt in the male walleye.

Petrovic *et al.* (2002) collected common carp downstream of WWTP outflows in Spain. Although vitellogenin levels varied between sites and sampling periods, increased levels of vitellogenin were observed in fish downstream of the main WWTP. Petrovic *et al.* (2002) found a correlation between measured EDC concentrations in water and sediment and plasma vitellogenin concentrations in male common carp.

Jobling *et al.* (1998) studied the occurrence of intersex species ("perceived males" that were found upon histological examination to have the presence of both male and female gonadal characteristics) in roach in eight rivers in the UK receiving discharge from WWTP and found the incidence of intersex males was much higher at sites that received WWTP effluent than at the control sites. The incidences of intersexuality were statistically significant in fish populations located downstream of WWTP effluent outflows (Jobling *et al.* 1998).

One study found WWTP outflow did not appear to have any effect on the fish population studied. Angus *et al.* (2002) studied reproductive characteristics of male mosquitofish (*Gambusia affinis*) at the outflow of a domestic WWTP effluent and found that there were no detectable levels of vitellogenin in either the exposed fish or the control population and that there were no histological changes in the exposed fish to indicate exposure to estrogens.

3.2 STUDIES OF SPECIFIC ENDOCRINE-DISRUPTING COMPOUNDS

Most of the studies on EDC related effects from WWTP effluent are limited to examining the estrogenicity of WWTP effluent that is typically measured through the induction of vitellogenin in fish. None of the studies found consider the implications these effects might have on fish reproduction or population and community dynamics. There are, however, studies of specific EDC chemicals found in WWTP effluents that examine other EDC effects. This section considers the results of studies that have focused on specific EDCs.

3.2.1 Estrogens

Estrogens are released into the environment from human and livestock waste and they include natural estrogens as well as synthetic estrogens such as those found in birth control pills. They include 17β-estradiol (E2), estrone (E1), and ethynylestradiol (EE2). Several of the studies on EDCs in WWTP effluent have focused on the effects of estrogens in general, while many other studies have focused on the effects of specific estrogens. Both EE2 and E2 have been shown to cause toxic effects at concentrations that have been measured in WWTP effluent (Mills and Chichester 2005) while E1 concentrations have been shown to cause toxic effects at concentrations lower than those measured in WWTP effluent (Mills and Chichester 2005). The observed toxic effects of EE2 and E2 are discussed below.

3.2.1.1 Ethinyl estradiol (EE2)

As shown in Table 3-1, EE2 has been found in WWTP effluent in concentrations ranging from 0.45 – 42 ng/L. In a study of fathead minnow exposure to concentrations of 0 – 32 ng/L EE2, Parrott *et al.* (2000) found fish exposed to >10 ng/L EE2 showed changes in secondary sex characteristics (most male fish had ovipositors) at 60 days. At higher concentrations (32 ng/L) fish were smaller and stunted with enlarged livers and ovipositors. Lange *et al.* (2001) also conducted a study on exposure of fathead minnow to EE2. In this study, fish were exposed to 0.2 – 64 ng/L concentrations of EE2 in a flow-through system. The no observed effects concentration for growth survival and reproduction in adult fish and the F1 embryo hatching success and larval survival were all found to be ≥ 1.0 ng/L. Male fish exposed to 4.0 ng/L failed to develop secondary sex characteristics. Fish exposed to 4.0 ng/L EE2 at 56 days post-hatch had a female:male sex ratio that was skewed to females and 11% of the fish had ova-testes. Pawlowski *et al.* (2004) found that fathead minnow exposed to between 10 ng/L – 100 ng/L EE2 showed a significant decrease in gonadosomatic index, condition factor, number of batches of eggs produced and their fertilization rate. The lowest observed effective concentration for plasma vitellogenin induction in both sexes and for ultrastructural changes in the testes and

livers was 1 ng/L. This appears to agree with the results obtained by Lange *et al.* (2001) for the no observed effects concentration in fathead minnow.

Ethinyl estradiol has been shown to have EDC related effects on other fish species. In a study of rainbow trout exposed to EE2, Jobling *et al.* (1996) found that a 2 ng/L concentration of EE2 resulted in elevated vitellogenin levels and a higher gonadosomatic index when compared to control fish. Schultz *et al.* (2003) exposed rainbow trout to concentrations of EE2 of between 10 ng/L – 1,000 ng/L and found that EE2 exposure concentrations between 10 ng/L and 100 ng/L caused an increase in sperm density (sperm concentration and spermatocrit) while exposure to the 100 ng/L concentration resulted in a significant reduction in testis mass. Exposure to 1,000 ng/L caused complete mortality of the treatment group within 57 days. Schultz *et al.* (2003) also harvested semen from the fish exposed to concentrations of 10 ng/L and 100 ng/L and used it to fertilize eggs from females that had not been exposed to EE2. The viability of embryos from EE2 exposed fish was significantly decreased when compared to control fish. Trudeau *et al.* (2003) found that exposure to elevated EE2 concentrations resulted in feminization of gonadal development in Japanese medaka (*Oryzias latipes*) including complete sex reversal or gonadal intersex depending on the dose.

Ethinyl estradiol has also been shown to have EDC-related effects on amphibians. In a study of the effects of estrogenic contaminants on amphibian sex differentiation (MacKenzie *et al.* 2000) Leopard Frog (*Rana pipiens*) exposed to low concentrations of EE2 produced 100% females or ova-testes. Park and Kidd (2001) studied a lake that was dosed with EE2 and found the hatch rate for Green Frog (*R. clamitans melanota*) eggs were lower in the treated lake than in reference lakes, although this difference was not statistically significant. This data suggests that low-level exposure to EE2 in the wild has toxic effects on Green Frog embryos (Park and Kidd, 2001). Park *et al.* (2002) studied the same treated lake and detected low frequencies of intersex in Mink Frog (*R. septentrionalis*) tadpoles and Green Frog tadpoles on the treated lake, while no occurrences of intersex were found in tadpoles in a reference lake. Ruby *et al.* (2003) showed that short-term exposure of *Xenopus laevis* tadpoles to EE2 during sex differentiation can significantly effect normal development of the testes for the life of the organism.

In addition to species-level effects, estrogens have also been shown to have an effect on community dynamics. In a multi-year study of EDC related effects on a lake with EE2 added, changes in abundance and diversity were found in certain species (Kidd *et al.* 2003). The results showed that in the treated lake abundance of fathead minnow decreased, diversity (but not abundance) of the algae decreased and the production of eggs by several species of zooplankton also decreased.

3.2.1.2 17β-estradiol (E2)

17β-estradiol (E2) has been measured in WWTP effluent in concentrations as high as 64 ng/L, although most measured concentrations are reported between 1.0 – 4.6 ng/L (Table 3-1). In general, most studies of E2 exposure have been conducted with concentrations that are higher than 4.6 ng/L. Kramer *et al.* (1998) exposed fathead minnow to concentrations of E2 ranging from 27.24 ng/L to 2,724 ng/L. Effects concentrations (EC50) of 120 ng/L for the inhibition of egg production and of 251 ng/L for vitellogenin production in male fish were observed. The primary effect of E2 exposure in females was found to be inhibition of egg production.

Routledge *et al.* (1998) exposed rainbow trout and roach to E2 and found a dose-related significant increase in vitellogenin production in rainbow trout at nominal E2 exposure concentrations of 100 ng/L. Roach exposed to 100 ng/L E2 also produced significantly elevated vitellogenin levels, although the response was lower than that in the rainbow trout (Routledge *et al.* 1998).

Other studies have shown EDC-related effects of E2 on the reproduction of Japanese medaka. Kang *et al.* (2002) exposed Japanese medaka to E2 concentrations of between 29.3 ng/L and 463 ng/L and found that males in all treatment groups developed ova-testes although there was no concentration related effect on the occurrence of ova-testis. Additionally, elevated vitellogenin levels were present in all treatment groups and VTG levels in males correlated with the E2 concentrations. Males treated with 55.7 ng/L E2 had large concentrations of vitellogenin but no other effects on reproduction were observed. In contrast, at the highest concentration of 463 ng/L significantly less eggs were collected and the gonadosomatic index of males were significantly lower compared to the control. Another study on Japanese medaka conducted by Nimrod and Benson (1998) involved the exposure of the fish to between 10 and 1,660 ng/L. All concentrations of E2 used were found to produce entirely female populations. The highest concentration of E2 resulted in lower fecundity in female fish. Bjerselius *et al.* (2001) studied the reproductive behaviour and physiology of male goldfish exposed to E2 through ingestion (100 ng/L – 10,000 ng/L) and water exposure (96 – 339 ng/L). Exposure to elevated levels of E2 both by ingestion and in water severely affected male Goldfish reproductive behaviour and physiology. The results included:

- Significantly decreased gonadosomatic index values in the groups exposed to 96 ng/L and 339 ng/L through water exposure and 1,000 ng/L and 10,000 ng/L by ingestion.
- Significantly less males with milt in the food exposure groups but no significant differences in water production in the water exposure groups.

- Significantly less males with tubercles in both exposure groups, with none of the males in the water exposure groups having tubercles by the end of the exposure period.
- Elevated concentrations of E2 in blood plasma, with the ratios of E2 in blood of exposed fish:E2 in blood of control fish as follows:
  - water exposure of 96 ng/L, ratio of 4:1
  - water exposure of 339 ng/L, ratio of 8:1
  - ingestion of 1,000 ng/L, ratio of 2:1
  - ingestion of 10,000 ng/L, ratio of 4:1
- Sexual activity was dramatically decreased in both exposure groups.

Bjerselius *et al.* concluded the observed physiological effects of E2 exposure could affect the reproductive capacity of Goldfish.

3.2.2 Nonylphenol ethoxylates

Nonylphenol ethoxylates are a group of chemicals used in a range of manufacturing processes as well as in cleaners and consumer products whose main source of entry into the environment is through industrial and municipal wastewater effluents (Environment Canada, Health Canada 2001). Nonylphenol ethoxylates released into WWTP systems are typically biodegraded into more toxic and peristent metabolites such as nonylphenol (NP), nonylphenol ethoxylate (NP1EO), nonylphenol diethoxylate (NP2EO), nonylphenoxyacetic acid (NP1EC) and nonylphenoxyethoxyacetic acid (NP2EC; Environment Canada, Health Canada 2001). Nonylphenols have been found to cause estrogenic responses, however, their potency is much lower than that of estrogens (Environment Canada, Health Canada 2001). Studies were available on the effects of nonylphenol and octylphenol on aquatic life and are discussed below.

Nonylphenol (NP)

Nonylphenol has also been shown to be estrogenic in nature, although, concentrations at which it produces EDC-related effects are much higher than concentrations of estrogens known to have EDC-related effects. Nonylphenols have been found in WWTP effluent in concentrations ranging from 0.564 µg/L – 37 µg/L, but measured concentrations are generally below 3 µg/L (Table 3-1).

Harries *et al.* (2000) studied EDC related effects of nonylphenol (NP) on fathead minnow using doses between 1 µg/L and 100 µg/L. Exposure to 100 µg/L of NP showed dramatically reduced egg production in female fish. This study also found that exposure to NP resulted in a significant reduction in the number of times spawnings occurred in a dose dependent manner and egg

batch size. NP exposure also resulted in a dose-related induction of vitellogenin levels in fish, with the levels in male fish being substantially higher (4,000 – 45,000X) than levels in the controls and in similarly exposed female fish (1-10x control). It was also found to effect secondary-sex characteristics in male fish with the highest dose resulting in a reduction in the number of tubercles in male fish and a dose-dependent reduction in the thickness of the fat pad.

Schwaiger *et al.* (2002) evaluated EDC related effects of NP concentrations of 1 µg/L and 10 µg/L on sexually mature rainbow trout and their offspring. Adult fish exposed to 10 µg/L exhibited significantly decreased hatching rates. Female offspring of the exposed fish exhibited significantly elevated vitellogenin levels compared to the control (Schwaiger *et al.* 2002). An analysis of offspring sex steroid levels found a two-fold increase of estradiol in males and a 13-fold increase in testosterone in females, indicating that NP exposure in adult fish can produce EDC related effects in offspring. Jobling *et al.* (1996) also demonstrated that NP has an estrogenic effect on rainbow trout by determining that exposure to 36.81 µg/L NP resulted in elevated vitellogenin levels in the plasma of exposed fish.

Octylphenol

Few studies are available on EDC effects of octylphenol (OP) and only one study was found that provided a measurement of the concentration of octylphenol in WWTP effluent (0.7 µg/L; Snyder *et al.* 1999). Jobling *et al.* (2003) exposed New Zealand Mud Snail (*Potamopyrgus antipodarum*) to OP and found higher concentrations of OP caused an inhibition of embryo production. Gray *et al.* (1999) studied the reproductive and transgenerational effects of exposure of Japanese Medaka to OP. Male fish exposed to concentrations of 25 µg/L and 50 µg/L OP showed a reduction in overall reproductive success. Eggs produced by matings of male and female fish exposed to OP had various developmental problems including circulatory system difficulties, incomplete eye development and failure to inflate swim bladders upon hatch. Jobling *et al.* (1996) also conducted experiments exposing rainbow trout to OP and found significant elevations in vitellogenin production at exposure concentrations of 38.52 µg/L. Routledge *et al.* (1998) exposed rainbow trout and roach to OP and found exposure concentrations of 10 µg/L and 100 µg/L produced significantly elevated dose-dependent vitellogenin levels in male rainbow trout while exposure of male roach to 100 µg/L produced elevated vitellogenin levels in these fish. Exposure of female roach to 100 µg/L OP also resulted in significantly elevated vitellogenin levels in these fish (Routledge *et al.* 1998).

3.3 PHARMACEUTICALS

Pharmaceuticals have been detected in surface water in Canada. Detectable levels of various pharmaceuticals have been measured in WWTP effluent. The types and concentrations of pharmaceuticals that have been found in WWTP effluent are given in Table 3-2. Currently, it is not clear, if pharmaceuticals other than synthetic estrogens are considered EDCs. Two recent reviews of the aquatic toxicity of EDCs (McMaster 2001 and Mills and Chichester 2005) do not include a discussion of EDC effects of pharmaceuticals. Nonetheless, a summary of the types and concentrations of (non-estrogen) pharmaceuticals found in WWTP effluent was compiled (Table 3-2). Limited studies on EDC related effects of these pharmaceuticals are available in the public domain.

A study of acidic pharmaceuticals in surface waters in southern Ontario found five acidic pharmaceuticals at concentrations above analytical detection limits including, ibuprofen, gemfibrizol, naproxen, triclosan, and salicylic acid (Bennie and Struger 2005). At least one of these drugs, gemfibrizol, has been found to be an EDC. Mimeault *et al.* (2002) found that Goldfish injected with gemfibrozil had a two-fold increase in serum glucose level and a 54% triglyceride reduction indicating the drug could affect non-target species and that chronic-environmental exposure to this drug might have biological effects. Woodhouse *et al.* 2003 found Goldfish exposure to gemfibrozil resulted in decreased plasma sex steroid levels and dose-dependent decreases in testoterone levels and 17β-estradiol levels, indicating this drug does have an endocrine-disrupting effect.

Solomon *et al.* (2003) conducted experiments of chronic exposure of aquatic organisms to mixtures of pharmaceuticals. The exposure to mixtures was expected to provide a realistic imitation of the natural environment. In these experiments, plants showed severe growth inhibition at higher concentrations of antibiotics, while the effects of other pharmaceuticals on zooplankton and phytoplankton resulted in changes in diversity and structure of plant communities. It is not clear if these effects are considered to be the result of endocrine disruption.

Table 3-2
Concentrations of Pharmaceuticals Measured in WWTP Effluent

Pharmaceutical	Detected concentrations	Location	Source
Acetominophen	0.11 µg/L (median) 10 µg/L (maximum)	United States	Kolpin *et. al*. (2002)
Acetylsalicylic acid	50 ng/L (median) ~1,200 ng/L (maximum)	Rio de Janeiro, Brazil	Stumpf *et al.* (1999)
Bezafibrate	0.144 µg/L (maximum)	Calgary	Chen *et. al*. (2005)
	~1,100 ng/L (median) ~1,200 ng/L (maximum)	Rio de Janeiro, Brazil	Stumpf *et al.* (1999)
	0.48 µg/L (March) 0.06 µg/L (May) 0.04 µg/L (August)	Finland	Vieno *et al.* (2005)
Caffeine	0.405 µg/L (maximum)	Calgary	Chen *et al.* (2005)
	0.081-0.1 µg/L (median) 5.7-6.0 µg/L (maximum)	United States	Kolpin *et. al*. (2002)
Carbamazepine	0.925 µg/L (maximum)	Calgary	Chen *et al.* (2005)
Chloramphenicol	0.56 µg/L (maximum)	Germany	Hirsch *et al.* (1999)
Cimetidine	0.074 µg/L (median) 0.58 µg/L (maximum)	United States	Kolpin *et al.* (2002)
Clarithromycin	0.24 µg/L (maximum)	Germany	Hirsch *et al.* (1999)
Clofibric acid	~120 ng/L (median) ~1,500 ng/L (maximum)	Rio de Janeiro, Brazil	Stumpf *et al.* (1999)
Codeine	0.012-0.2 µg/L (median) 0.019-1.0 µg/L	United States	Kolpin *et al.* (2002)
Cotinine	0.165 µg/L (maximum)	Calgary	Chen *et. al*. (2005)
Cyclophosphamide	0.055 µg/L (maximum)	Calgary	Chen *et al.* (2005)
Dehydronifedipine	0.012 µg/L (median) 0.03 µg/L (maximum)	United States	Kolpin *et al.* (2002)
Diclofenac	0.359 µg/L (maximum)	Calgary	Chen *et al.* (2005)
	400 ng/L (median) ~1,500 (maximum)	Rio de Janeiro, Brazil	Stumpf *et al.* (1999)
Diclofenac (cont'd.)	0.36 µg/L (September) 0.46 µg/L (March) 0.24 µg/L (May) 0.34 µg/L (August)	Finland	Vieno *et. al*. (2005)
Diltiazem	0.021 µg/L (median) 0.049 µg/L (maximum)	United States	Kolpin *et al.* (2002)
Enalaprilat	0.046 µg/L (maximum)	United States	Kolpin *et al.* (2002)
Erythromycin-H_2O	2.50 µg/L (median) 5.10 (90^{th} percentile) 6.00 (maximum)	Germany	Hirsch *et al.* (1999)
Fenofibric acid	~50 ng/L (median) ~750 ng/L (maximum)	Rio de Janeiro, Brazil	Stumpf *et al.* (1999)

Table 3-2
Concentrations of Pharmaceuticals Measured in WWTP Effluent

Pharmaceutical	Detected concentrations	Location	Source
Fenoprofen	0.336 µg/L (maximum)	Calgary	Chen *et. al*. (2005)
Fluoxetine	0.509 µg/L (maximum)	Calgary	Chen *et. al*. (2005)
	0.012 µg/L (maximum)	United States	Kolpin *et. al*. (2002)
Gemfibrozil	0.799 µg/L (maximum)	Calgary	Chen *et. al*. (2005)
	300 ng/L (median) ~1,800 ng/L (maximum)	Rio de Janeiro, Brazil	Stumpf *et. al*. (1999)
	0.048 µg/L (median) 0.79 µg/L (maximum)	United States	Kolpin *et. al*. (2002)
Ibuprofen	0.383 µg/L (maximum)	Calgary	Chen *et. al*. (2005)
	600 ng/L (median) ~3,700 (maximum)	Rio de Janeiro, Brazil	Stumpf *et al.* (1999)
	0.24 µg/L (March) 0.04 µg/L (August)	Finland	Vieno *et al.* (2005)
	0.20 µg/L (median) 1.0 µg/L (maximum)	United States	Kolpin *et al.* (2002)
Indomethacin	0.105 µg/L (maximum)	Calgary	Chen *et al.* (2005)
	50 ng/L (minimum) 1,000 ng/L (maximum)	Rio de Janeiro, Brazil	Stumpf *et. al*. (1999)
Ketoprofen	~180 ng/L (median) ~680 ng/L (maximum)	Rio de Janeiro, Brazil	Stumpf *et al.* (1999)
Metformin	0.11 µg/L (median) 0.15 µg/L (maximum)	United States	Kolpin *et. al*. (2002)
Naproxen	1.785 µg/L (maximum)	Calgary	Chen *et. al*. (2005)
	600 ng/L (median) 3,000 ng/L (maximum)	Rio de Janeiro, Brazil	Stumpf *et. al*. (1999)
	0.16 µg/L (September) 0.8 µg/L (March) 0.2 µg/L (May) 0.52 µg/L (August)	Finland	Vieno *et. al*. (2005)
Pentoxifylline	0.099 µg/L (maximum)	Calgary	Chen *et. al*. (2005)
Roxithromycin	0.68 µg/L (median) 0.80 µg/L (90^{th} percentile) 1.00 µg/L (maximum)	Germany	Hirsch *et al.* (1999)
Sulfamethoxazole	0.40 µg/L (median) 0.90 µg/L (90^{th} percentile) 2.00 µg/L (maximum)	Germany	Hirsch *et al.* (1999)
Tolfenamic acid	~1,600 ng/L (maximum)	Rio de Janeiro, Brazil	Stumpf *et. al*. (1999)

Table 3-2 Concentrations of Pharmaceuticals Measured in WWTP Effluent			
Pharmaceutical	**Detected concentrations**	**Location**	**Source**
Trimethoprim	0.907 µg/L (maximum)	Calgary	Chen *et al.* (2005)
	0.32 µg/L (median) 0.62 µg/L (90^{th} percentile) 0.66 µg/L (maximum)	Germany	Hirsch *et al.* (1999)

3.4 EDC-RELATED EFFECTS FROM NON-WWTP SOURCES

3.4.1 Pulp and Paper Mill Effluent

Pulp and paper mill effluent has been shown to result in EDC-related effects. A number of studies have been conducted that examine EDC-related effects from pulp and paper mill effluent. MacLatchey *et al.* (2000) exposed common mummichog (*Fundulus heteroclitus*) to 1% concentrations of primary and secondary effluents from a bleached kraft pulp mill. Exposed male and female fish were found to have significantly decreased levels of plasma testosterone. This effect was greater in the concentration composed of secondary effluent. Van den Heuval *et al.* (2001) conducted a three-year multi-species study of the EDC-related effects of mixed bleached kraft / thermomechanical pulp and paper mill effluent. Results indicated that pulp and paper mill effluent elicited EDC-related effects that appeared to be androgenic in nature including reduced gonad size in rainbow trout correlating with levels of circulating sex steroid hormones prior to sexual maturation and the induction of male secondary sex characteristics in female mosquitofish. Further investigations showed the effluent contained compounds that bind to goldfish androgen receptors in vitro (van den Heuval *et al.* 2001).

Parrot *et al.* (2001) conducted a study of bleached sulfide mill effluent on fathead minnow and found the effluent caused a significant increase in the fish growth, changes in external sex characteristics in 32% and 48% effluent at 60 days post hatch, and ovipositors in male fish in concentrations of 3% effluent at 125 days post hatch. The only fish in the study that produced eggs were those exposed to 1% concentrations of effluent. In a study of bioactive substances present in pulp and paper mill effluent, Hewitt *et al.* (2003) found that fish at the outflow of a bleached kraft mill and a bleached sulfite/groundwood mill accumulated compounds from final effluents that interacted with sex steroid hormone receptors.

3.5 SUMMARY OF POTENTIAL EDC EFFECTS ON AQUATIC SPECIES

The effects of EDCs on aquatic life that have been found in the literature and are discussed in detail in the previous sections are summarized in Tables 3-3 and 3-4. EDC related effects shown to occur as a result of exposure to WWTP effluents are summarized in Table 3-3 while EDC-related effects resulting from exposure to specific EDCs are summarized in Table 3-4. The EDC-related effect that is most frequently recorded as a result of exposure to WWTP effluent is increases in vitellogenin levels in exposed fish. In general, studies conclude that the increase in vitellogenin levels in fish exposed to WWTP effluent indicate the presence of estrogenic chemicals in the effluent. Currently, there is limited information regarding long-term EDC-related effects resulting from exposure to WWTP effluent on aquatic life. Information is available on the EDC-related effects of specific EDCs found in WWTP effluent on aquatic life including examination of longer-term effects to aquatic biota (Table 3-4). For comparison purposes Table 3-4 includes data on EDC concentrations that have been detected in WWTP effluent and indicates the concentrations of specific EDCs shown to have effects on the reproduction of aquatic life.

Table 3-3
Aquatic Effects of EDC at Concentrations Found in WWTP Effluent

Species	EDC Type and Concentration	Aquatic Effects
Rainbow Trout	Up to 15 ng/L 17-β-estradiol equivalents	• Moderate increases in vitellogenin production (Svenson *et. al.* 2002) indicating that WWTP effluent is estrogenic • Elevated estrogenicity in the bile of WWTP exposed fish compared to controls (Allard et. al. 2004)
	5.8 ng/L estrone, 1.1 ng/L 17-β-estradiol, 4.5 ng/L 17-α-ethinyl estradiol, 850 ng/L nonylphenol, 490 ng/L bisphenol A	• Bile of fish downstream of outflow contained EDC concentration of 1,000 to 1,000,000 times greater than that in the water and induction of vitellogenesis in exposed fish (Larsson *et. al.* 1999)
	Not measured	• Increases in plasma vitellogenin levels (Harries *et al.* 1996)
	195 ng/L estrone 38.9 ng/L 17-β-estradiol 7.9 ng/L 17-α-ethinyl estradiol	• Elevated estrogenicity in the bile of WWTP exposed rainbow trout (Gibson *et al.* 2004)
	4 – 56 ng/L Natural steroid estrogens (i.e., estrone, 17-β-estradiol) concentrations of alkylphenolic chemicals in the µg/L range up to 2 ng/L 17-α-ethinyl estradiol	• Increase in plasma vitellogenin concentrations (Jobling *et al.* 2003)
	24.3-104.1 ng/L 17-β-estradiol equivalents	• 700-fold induction of vitellogenin production in male fish and a 240-fold induction in female fish (Tyler *et al.* 2005)
	4.1-6.8 ng/L 17-β-estradiol equivalents	• 4-fold induction of vitellogenin production in male fish and an 18-fold induction in female fish (Tyler *et. al.* 2005)
Eastern Elliptio	Not measured	• Sex ratio skewed in favour of females (Gagne *et. al.* 2002)
Channel Catfish	21 – 147 ng/L E2 equivalents	• Increases in vitellogenin levels (Tilton *et. al.* 2002)
	Not stated, effects are attributed to several potential causes including WWTP effluent as well as agricultural runoff or effluent from a beet processing plant	• Masculinization of female fish (Hegrenes 1999)
Common Carp	4 – 56 ng/L Natural steroid estrogens (i.e., estrone, 17-β-estradiol concentrations of alkylphenolic chemicals in the microgram/litre range up to 2 ng/L 17-α-ethinyl estradiol	• Increase in plasma vitellogenin concentrations (Jobling *et. al.* 2003)

Table 3-3
Aquatic Effects of EDC at Concentrations Found in WWTP Effluent

Species	EDC Type and Concentration	Aquatic Effects
Common Carp (cont'd)	31 µg/L nonylphenol ethoxylates 35 µg/L nonylphenoxy carboxylate up to 25 ng/L estrone	• Increased vitellogenin levels (Petrovic *et. al.* 2002)
Zebra Mussel	Estrone, 17-β-estradiol, 17-α-ethinyl estradiol, bisphenol A, Butylbenzyl phthalate, nonylphenol, octylphenol, concentrations not measured	• Increases in vitellogenin-like proteins (Quinn *et. al.* 2004)
Fathead minnow	1.0-2.2 µg/L nonylphenol congeners 1.0-2.2 µg/L 17-α-ethinyl estradiol	• Elevated vitellogenin levels, reduced GSI index, increased HSI (Hemming *et al.* 2001)
Roach	195 ng/L estrone 38.9 ng/L 17-β-estradiol 7.9 ng/L 17-α-ethinyl estradiol	• Detectable estrone, 17-β-estradiol, and 17-α-ethinyl estradiol in the bile of these fish and 17-α-ethinyl estradiol was detected in the ovaries and testes of the fish (Gibson *et al.* 2005)
	24.3-104.1 ng/L 17-β-estradiol equivalents	• Elevated vitellogenin levels in male fish (Tyler *et. al.* 2005)
	Not measured	• Significant incidence of male intersex fish (Jobling et. al. 1998)
Brine Shrimp	Not measured	• Increases in vitellogenin levels (Gagne and Blaise 2002)
Longnose Sucker	Not measured	• Altered circulating steroid hormone levels and vitellogenin levels, increased levels of hepatic oxidative stress (McMaster *et. al.* 2002)
Walleye	<0.2 ng/L estradiol <0.2 ng/L estrone <0.3 ng/L nonylphenol	• Elevated vitellogenin and estradiol levels, decreased gonad and testosterone size, lack of expressible milt (Bacigalupi 2004)

Table 3-4
Effects of EDC at Concentrations Found in WWTP Effluent

EDC	Conc. in WWTP Effluent	Species	Conc. of EDC	Aquatic Effects
EE2	0.45 – 42 ng/L	Fathead Minnow	> 10 ng/L	• Most male fish had ovipositors (Parrott *et. al.* 2000)
			32 ng/L	• Smaller stunted fish with enlarged livers and huge ovipositors (Parrott *et al.* 2000)
			≥1.0 ng/L	• No observed effects concentration (NOEC) for growth survival and reproduction in adult fish and F_1 embryo hatching success and larval survival (Lange *et. al.* 2001)
			4.0 ng/L	• Male fish failed to develop secondary sex characteristics (Lange *et. al.* 2001) • Sex ratio skewed to females, ova-testes detected in some fish (Lange *et. al.* 2001)
			10-100 ng/L	• Decreases in gonadosomatic index, number of batches of eggs, and fertilization rate (Pawlowski *et. al.* 2004)
			1 ng/L	• Lowest observed effects for plasma vitellogenin induction in both sexes and for ultrastructural changes in the testes and liver (Pawlowski *et. al.* 2004)
			3 ng/L	• Reduction in extent of parenchymatic areas in ovaries and ultrastructural changes in the livers of females (Pawlowski *et. al.* 2004)
			5-6 ng/L	• Decrease in abundance (Kidd *et. al.* 2003)
		Rainbow Trout	1.79 ng/L	• Elevated vitellogenin levels and a higher gonadosomatic index (Jobling *et. al.* 1996)
			10-100 ng/L	• Increase in sperm density (sperm concentration and spermatocrit), semen harvested from exposed fish and used to fertilize eggs from non-exposed fish resulting in the viability of the embryos produced from the sperm of exposed fish being significantly reduced from the control (Schultz *et. al.* 2003)
			100 ng/L	• Significant reduction in testis mass (Shultz *et. al.* 2003)
		Japanese Medaka	Not stated	• Feminization of gonadal development including complete sex reversal or gonadal intersex (Trudeau *et. al.* 2003)
		Leopard Frog	10-100 ng/L	• Produced 100% females or ova-testes (Mackenzie *et. al.* 2000)
		Mink Frog tadpoles	6 ng/L	• Low frequencies of intersex (Park *et al.* 2002)
		Green Frog tadpoles	6 ng/L	• Low frequencies of intersex (Park *et. al.* 2002)
		African Clawed frog	5 ng/L	• Significant decreases in primary spermatogonia and increased testicular degeneration (Ruby *et. al.* 2003)

Table 3-4
Effects of EDC at Concentrations Found in WWTP Effluent

EDC	Conc. in WWTP Effluent	Species	Conc. of EDC	Aquatic Effects
E2	1 – 64 ng/L	Fathead Minnow	27.24 – 1,724 ng/L	• Inhibition of egg production in females (Kramer *et. al*. 1998) • EC_{50} of 120 ng/L for inhibition of egg production (Kramer *et. al*. 1998) • EC_{50} of 251 ng/L for induction of vitellogenin in male fish (Kramer *et al.* 1998)
	1 – 64 ng/L	Japanese Medaka	29.3 – 463 ng/L	• Males developed ova-testes (Kang *et al.* 2002) • Males treated with 55.7 ng/L had greater concentrations of hepatic vitellogenin (Kang *et. al*. 2002) • Number of eggs produced and fertility of fish exposed to 463 ng/L was significantly less than the controls (Kang *et. al*. 2002)
			10 – 1,660 ng/L	• All concentrations produced entirely female populations (Nimrod and Benson 1998) • Female fish had lower fecundity at the highest concentration (Nimrod and Benson 1998)
		Goldfish	96 – 339 ng/L	• Concentrations in water of 10 ng/L and 100 ng/L resulted in a significantly decreased GSI in male fish (Bjerselius *et. al*. 2001) • Significantly less males with tubercles compared to control fish (Bjerselius *et al.* 2001) • Elevated E2 levels in blood plasma in male fish (Bjerselius *et. al*. 2001) • Change in reproductive behaviour (Bjerselius *et. al*. 2001)
		Rainbow Trout	100 ng/L	• Significantly elevated vitellogenin levels (Routledge *et al.* 1998)
NP	0.56 – 37 µg/L	Fathead Minnow	0.65 – 100 µg/L	• Measured concentrations of above 48 µg/L inhibited reproduction completely (Harries *et al.* 2000) • Exposure to nominal NP concentrations of 100 µg/L (mean measured concentration of 75.5 µg/L) reduced egg production (Harries *et. al*. 2000) • Dose dependent reduction in the number of spawnings occurring (Harries *et. al*. 2000) • Statistically significant reduction in GSI for female fish exposed to 8.1 µg/L (Harries *et. al*. 2000) • Dose-related induction of vitellogenin at concentrations between 0.65 – 8.1 µg/L in male fish and 8.1 – 57.7 µg/L in females (Harries *et. al*. 2000); • Reduction in the number of tubercles in males exposed to the highest dose (Harries *et. al*. 2000); • Dose-dependent reduction in thickness of the fat pad (Harries *et. al*. 2000)
		Rainbow Trout	1-10 µg/L	• 10 µg/L resulted in significantly decreased hatching rate (Schwaiger *et. al*. 2002) • vitellogenin levels were significantly elevated in female offspring of exposed fish (Schwaiger *et. al*. 2002) • 2-fold increase in estradiol in males offspring of exposed fish (Schwaiger *et. al*. 2002) • 13-fold increase in testosterone levels in female offspring of exposed fish (Schwaiger et. al. 2002)
NP (cont'd)	0.56 – 37 µg/L	Rainbow Trout	0.5 – 65 µg/L	• Exposure to 36.81 µg/L resulted in elevations in vitellogenin levels (Jobling *et. al*. 1996)

Table 3-4
Effects of EDC at Concentrations Found in WWTP Effluent

EDC	Conc. in WWTP Effluent	Species	Conc. of EDC	Aquatic Effects
OP	Up to 0.7 µg/L	Rainbow Trout	0.5 – 65 µg/L	• Exposure to 38.52 µg/L resulted in elevations in vitellogenin levels (Jobling *et al.* 1996)
			10 – 100 µg/L	• Significantly elevated vitellogenin levels in male fish (Routledge *et al.* 1998)
		Japanese Medaka	10 – 100 µg/L	• Male fish exposed to concentrations of 25 – 50 µg/L showed a reduction on overall reproductive success (Gray *et al.* 1999) • Eggs produced by exposed fish has various development problems including circulatory system difficulties, incomplete eye development and failure to inflate swim bladders upon hatch (Gray *et. al.* 1999)
		New Zealand Mud Snail	1 – 100 µg/L	• Inhibition of embryo production at 100 µg/L (Jobling *et al.* 2003)
		Roach	100 µg/L	• Significantly elevated vitellogenin levels in male and female fish (Routledge *et al.* 1998)

4.0 EFFECTIVENESS OF EDC REMOVAL

The scientific literature was examined to provide an indication of the effectiveness of various treatment methods in removing EDCs from WWTP. Table 4-1 provides a summary of EDC concentrations that have been measured post-treatment for varying treatment types and flows. Few of the studies available measured the end concentrations of nonylphenol ethoxylates so no conclusions could be drawn regarding the effectiveness of removal of these compounds. There were several studies that provided information on both treatment processes and estrogen concentrations measured in the effluent including E1, E2 and EE2.

Treatment systems found to have the highest E1 concentrations located in the UK, included primary settlement and screening, primary settlement, biological filtration and secondary humus settlement. Most of the other treatment systems appeared to lead to concentrations of estrone of <20 ng/L, although higher concentrations were recorded; in two treatment plants in Italy using activated sludge treatment; while maximum concentrations of estrone of 70 ng/L were observed in a German plant using preliminary clarification and aeration followed by Fe(II) addition and 48 ng/l in Canadian plants using preliminary and final clarification followed by aeration, aluminum sulfate addition and final disinfection.

From this data it appears that activated sludge treatment and multi-step treatment processes appear to be more effective in removing estrone than settlement. The measured EE2 levels were typically less than 2 ng/L in all treatment systems. The only exceptions were in the plant in the UK using primary settlement (7 ng/L) and in WWTPs in Canada with the treatment system consisting of preliminary and final clarification, aeration, aluminum sulfate addition and final disinfection (9 ng/L median). E2 levels were typically less than 15 ng/L with the exceptions again being in the UK primary settlement treatment system (29-48 ng/L). This data appears to support the observation that activated sludge and multi-step treatment processes are more effective than primary settlement processes in removing estrogens.

Svenson and Allard (2004) measured the removal of androgenicity from domestic WWTP effluents in Sweden and found removals of 26% - 42% for WWTP without secondary treatment and 96 - >99% for processes with secondary and tertriary treatment. Svenson and Allard (2004) noted WWTP using biological processes such as activated sludge were more effective in reducing androgenic activity in effluent.

Table 4-1
Summary of Wastewater Amount and Treatment Type on EDC Effluent Concentration

Location	Type of Wastewater Treatment	Amount of Water Treated (m^3/day)	Population Equivalent	EDC Concentrations Measured at Outflow of Effluent								
				NP (ng/L)	OP (ng/L)	NP1EO (ng/L)	NPE (ng/L)	Estrone (E1; ng/L)	17β-estradiol (E2; ng/L)	17α-estradiol (ng/L)	17α-ethinyl estradiol (EE2; ng/L)	Bi-phenol A (ng/L)
Lake Mead, Nevada[1]	Tertriary treatment	~460 million L/day	not stated	1,140	43		8,990		2.67		0.48	
United Kingdom (Southend STW)[2]	Primary settlement treatment (Vitox treatment from Apr to Oct)	45,000	197,749					32-48	29-48		7	
United Kingdom (Harpenden STW)[2]	Percolating filters and sand filters	8,250	not stated					5.2-8.9	3.7-7.1		nd	
United Kingdom (Rye Meads STW)[2]	Diffused air-activate sludge, final settlement and tertriary lagoons	88,500	357,000					1.8-3.6	2.7-6.3		nd	
United Kingdom (Deephams STW)[2]	Diffused-air activated sludge	160,000	796,000					2.0-13.0	4.3-12.0		nd	
United Kingdom (Naburn STW)[2]	Screening, primary settlement, biological filtration, secondary humus settlement	20,000	388,000					15-76	6.5-10.0		0.6-4.3	
United Kingdom (Horsham STW)[2]	Biological filtration and settlement lagoons	18,000	107,250					6.1-12.0	4.0-5.7		0.2-0.8	
United Kingdom (Billing STW)[2]	Extended aeration	60,000	285,959					1.4-9.9	6.1-7.4		nd	
Sweden[3]	chemical and biological treatment steps, no anaerobic denitrification	mean 881	3,500	800				5	1		4	500
Italy(Cobis)[4]	Activated sludge	10,000	not stated					5.4-17	0.55-2.9		nd-1.0	
Italy (Fregene)[4]	Activated sludge	120,000	not stated					2.5-6.5	0.35-2.1		nd - 1.7	
Italy (Ostia)[4]	Activated sludge	350,000	not stated					13-82.1	0.72-3.5		nd-1.1	
Italy (Roma sud)[4]	Activated sludge	1,200,000	not stated					8.7-51	0.53-3.1		nd-1.2	
Italy (Roma est)[4]	Activated sludge	800,000	not stated					3.7-10L	0.62-0.82		nd-0.73	
Italy (Roma nord)[4]	Activated sludge	800,000	not stated					6.4-40	0.44-1.9		nd-0.56	

Table 4-1
Summary of Wastewater Amount and Treatment Type on EDC Effluent Concentration

Location	Type of Wastewater Treatment	Amount of Water Treated (m^3/day)	Population Equivalent	EDC Concentrations Measured at Outflow of Effluent								
				NP (ng/L)	OP (ng/L)	NP1EO (ng/L)	NPE (ng/L)	Estrone (E1; ng/L)	17β-estradiol (E2; ng/L)	17α-estradiol (ng/L)	17α-ethinyl estradiol (EE2; ng/L)	Bi-phenol A (ng/L)
The Netherlands (domestic) [5]	Activated sludge	not stated	not stated					2.7-15	1.1	<0.2-<1.4	<0.2-<1.4	
The Netherlands (domestic) [5]	Activated sludge	not stated	not stated					<0.4-6.3	0.7	<0.2-<1.7	<0.2-<1.8	
The Netherlands (domestic) [5]	Activated sludge	not stated	not stated					2.1-47	< 0.6 - 12	<0.1-5.0	<0.3-7.5	
The Netherlands (industrial)[5]	Activated sludge	not stated	not stated					0.7-11	< 0.6 - 1.8	<0.5-2.1	<1.8-2.6	
The Netherlands (industrial)[5]	Activated sludge	not stated	not stated					<0.1 - <0.4	<0.4 - <0.7	<0.1	<0.2 - <0.3	
United Kingdom[6]	Not stated	not stated	not stated	3,000	nd	45,000						
Germany[7]	preliminary clarification, followed by an aerator tank with the addition of Fe(II)chloride for phosphate elimination and end point clarification	41,200 (ave low)	312,000					9 –med. 22 -90th percent 70 -max	n.d. -med 2 -90th percent 3 -max		1 -med 4 -90th percent 15 -max	
Ontario, Canada[7]	preliminary and final clarification and an aerator tank, aluminum sulfate and a final disinfection step	not stated	not stated					3 -med 10 -90th percent 48 -max	6 -med 14 -90th percent 64 -max		9 -med 29 -90th percent 42 -max	

Notes:
[1] Snyder *et al.* 1999
[2] Desbrow *et al.* 1998
[3] Larsson *et al.* 1999
[4] Baronti *et al.* 2000
[5] Belfroid *et al.* 1999
[6] Lye *et al.* 1999
[7] Ternes *et al.* 1999
nd = not detected

5.0 GLOSSARY OF TERMS

Androgenicity – the quality of exerting a masculinizing effect.

Condition factor – ratio of body weight:total length.

EC_{50} – Effective concentration, concentration at which there is demonstrated effects in 50% of the population.

Endocrine system – the system of glands that produce endocrine secretions that helps to control bodily metabolic activity. The endocrine system chemically controls the various functions of cells, tissues, and organs through the secretion of hormones.

Endocrine-Disrupting Compounds (EDC) - compounds that interfere with the normal functioning of the endocrine system of complex organisms.

17β-estradiol (E2) - a natural estrogenic hormone that is a phenolic alcohol $C_{18}H_{24}O_2$ secreted chiefly by the ovaries. Estradiol exists in two isomeric forms, 17β-estradiol is the active isomer.

17β-estradiol equivalents – measure of estrogenicity.

Estrogenic – pertaining to, having the effects of, or similar to an estrogen.

Estrogenicity - the quality of exerting or the ability to exert an estrogenic effect.

Estrone (E1) - a natural estrogenic hormone that is a ketone $C_{18}H_{22}O_2$ found in the body chiefly as a metabolite of estradiol , that is also secreted especially by the ovaries. It is a metabolite of 17β-estradiol and is considerably less active than 17β-estradiol.

Ethinyl estradiol (EE2) - a very potent synthetic estrogen, $C_{20}H_{24}O_2$.

Gonadosomatic index (GSI) – the percentage of gonad tissue mass to the total mass of the fish.

Hepatosomatic index – the percentage of liver tissue mass to the total mass of the fish.

Lacustrine – of or pertaining to a lake.

Nonylphenol – a chemical intermediate composed of a phenol ring attached to a lipophilic straight or branched nonyl group.

Nonylphenol ethoxylates – part of the group of compounds known as alkylphenol ethoxylate and having the general formula $C_{15}H_{24}O+(CH_2CH_2O)_n$.

Intersex – the simultaneous presence of both male and female gonadal characteristics.

Ova-testes – co-occurrence of testicular and ovarian tissue within the same gonad.

Ovipositor – a tubular organ at the end of the abdomen of some female fish that is used to deposit eggs.

Riverine – relating to or resembling a river.

Secondary sex characteristics - traits that distinguish the two sexes of a species, but that are not directly part of the reproductive system.

Steroid – any hormone affecting the growth and development of sex hormones.

Tubercles – a small rounded projection or protuberance on a bone or on the surface of an animal or plant.

Vitellogenin - A protein, precursor of several yolk proteins, especially phosvitin and lipovitellin in the eggs of various vertebrates, synthesized in the liver cells after oestrogen stimulation.

Vitellogenesis – formation of the yolk of the egg.

WWTP – wastewater treatment plant.

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Environmental Assessment of
Canadian Strategic Infrastructure Funded
Upgrades to the City of Winnipeg
Water Pollution Control Centres

APPENDIX 8E

Illness Risk Assessment Update

1.0 ILLNESS RISK ASSESSMENT UPDATE

1.1 WATER-QUALITY REGULATION AND ILLNESS RISK

Fecal and total coliforms are indicator organisms measured to assess water quality for recreational uses. The Manitoba Water Quality Standards, Objectives and Guidelines (MWQSOG 2002) state the maximum acceptable concentration levels of fecal coliform bacteria at 200 (fc) per 100 millilitres (mL) in surface water for primary recreation activities (e.g. swimming, diving). This objective is intended to protect human health during direct-contact outdoor recreational activities.

It should be noted that no secondary recreation objective is indicated in MWQSOG (2002); however the previous Manitoba Surface Water Quality Objectives (Williamson 1988) defined a maximum acceptable fecal coliform concentration for secondary recreation (e.g., boating, fishing) of 1,000 fc/100 mL. This objective was intended to protect human health where immersion is partial, accidental and unrepeated.

Other indicator organisms are also used in evaluating microbial water quality for recreational uses. In particular, *Escherichia coli* and *Enteroccoci* are extensively used as pathogen-presence indicators (Health and Welfare Canada 1992). The maximum acceptable concentrations of these organisms for protection of direct-contact recreation are demonstrated as follows (HWC 1992):

- Escherichia coli (E. coli) 200/100 mL
- *Enterococci* 35/100 mL

Further information regarding specific water quality objectives for various jurisdictions, historical water-quality regulation, evolution in regulation and public-illness risk inherent in water quality can be found in the Health-Risk Assessment relating to uses of the Red and Assiniboine rivers in Winnipeg and downstream (Tetr*ES*/Wardrop 1997).

2.0 KEY PARAMETERS IN ILLNESS-RISK ASSESSMENT

2.1 WATERBORNE PATHOGENS

Waterborne pathogens are microorganisms that cause disease in humans. Microoganisms that typically create human health threats include viruses, bacteria, protozoa and helmiths. These pathogens enter the environment from different sources (i.e., combined sewer overflows, surface runoff, sanitary sewer overflows and wastewater effluent) and subsequently enter the human body through ingestion of fecal matter that is contained in surface water.

Viruses and bacteria are among the most important and predominant pathogens present in wastewater. Viruses and bacteria can cause a wide range of diseases, such as gastrointestinal infection. Viruses are generally more infectious than bacteria because they require a smaller dose to cause infection and are very resistant to wastewater treatment processes. Protozoa and helmiths are parasites more prevalent in wastewater than in other environmental sources. Infection by these parasites more commonly occurs after consumption of water contaminated with fecal material. Severe infection cases are mostly found in the elderly and small children.

2.2 INDICATOR ORGANISM CONCEPT

The indicator organisms are used for monitoring water quality for recreational uses. A commonly used indicator is the coliform group of bacteria. This group includes a wide variety of organisms, mostly of intestinal origin (Tetr*ES*/Wardrop 1997). In particular, *Escherichia coli* has been used as a bacterial indicator of fecal contamination of water sources. The main reasons for using *E. Coli* are that its growth characteristics and behaviour in the environment are relatively well known and they are harmless for use in laboratory analysis.

2.3 PATHOGEN PERSISTENCE AND VIABILITY

A study of microorganism decay, growth, antagonism and/or parasitism dynamics is very complex and little has been reported about direct measurement of pathogen persistence and

viability in the illness-risk assessment (Tetr*ES*/Wardrop 1997). The following are examples of what has been reported pertaining to pathogen persistence and viability:

- Some of the factors affecting pathogen viability in surface water are nutrient availability, turbidity and temperature.
- Low temperatures, sediment adsorption, or anoxic conditions can prolong bacterial survival in aquatic ecosystems.
- Acellular viruses can last longer in surface waters than bacteria (Marzouk *et. al.* 1980; Gerba *et. al.* 1979), while protozoa can extend their survival time by encystations (USEPA 1993).

Detailed pathogen persistence and viability information can be found in Sections 2.4 and 2.5 of the 1997 Health-Risk Assessment (HRA) relating to uses of the Red and Assiniboine rivers in Winnipeg and downstream (Tetr*ES*/Wardrop 1997).

2.4 EXPOSURE TO PATHOGENS

Direct exposures to pathogens are mainly from ingesting contaminated water. The groups of people that are most susceptible to disease from exposure to waterborne pathogens are children, the elderly, pregnant women, immuno-compromised (e.g., AIDS patients, cancer-treatment patients), chemically-dependent, and diabetics (Tetr*ES*/Wardrop 1997).

In recent years, water sports, such as surfing and water skiing, have become very popular in Manitoba. People who conduct these activities are considered to be exposed as sensitive organs, such as eyes and nose, may come in contact directly with surface waters. However, most severe waterborne pathogen infection cases are reported from ingesting contaminated drinking water, rather than from recreation.

3.0 QUANTIFYING HUMAN-ILLNESS RISKS FROM RIVER USES

3.1 ILLNESS-RISK MODELING

The fundamental basis for quantification of illness risks from river uses is the science of epidemiology, which attempts to define, usually by "hindcasting" statistical techniques, the relationship among:

- Pathogen densities at the point of human contact.
- The extent of exposure (usually the infective dose[s] and the number of doses ingested).
- The disease(s) and disease severity attributed to the exposure(s).

These relationships are usually expressed in the form of regression equations or "models" of the dose-response (D-R) relationship. Quantitative illness-risk assessment (QRA), therefore, depends on the state of the epidemiological literature, and on whether pathogens (or indicators) of interest in a specific situation have been the subject of prior epidemiological research. To predict the societal disease caseload attributable to each organism, dose-response models or epidemiological relationships must exist which model the infectivity of the organism-host relationship for the pathogen or indicator of interest.

3.1.1 Requirements of Models

Methods to provide quantitative perspective on the human-illness risks associated with uses of river water must first be able to predict the risk rate for symptomatic diseases (as distinct from symptomatic or asymptomatic infections; Ward *et al.* 1986) linked to each pathogenic organism. In this way, and in consideration of the extent of river uses, the relative importance of each pathogenic organism to societal health can be evaluated on a standardized basis (e.g., number of disease cases caused [or attributed] to each pathogenic vector). Such comparison allows the

relative risks of each vector to be seen in a way that facilitates understanding of how pathogen-reduction plans can be evaluated.

For use in the IRA study, the models were required to be expressed in terms of risk per unit amount of recreation (e.g., cases of gastrointestinal illness [GI]/1,000 immersions) such that river-use estimates could be applied for ready calculation of total recreation-related illnesses. If models, by contrast, were expressed in terms of probabilities of infection per unit dose of micro-organisms, they could not be applied within the scope of the study (i.e., without undertaking significant epidemiological research to elucidate such necessary facts for application of these models as:

- Probability that 2 L of river water will contain the infective dose(s) of the pathogens in question:
  - 2 L is the volume typically used in the experiments to elucidate the infection probability in these models.
- Percentage of infected individuals displaying the associated illness(es):
  - Not all infected persons are symptomatic; some are “carriers”).

3.1.2 Limitations in Models

Gale (1996) outlines a number of problems associated with developing a risk assessment model for exposure to pathogens in contaminated drinking water supplies. Many of the problems cited for a drinking-water exposure are relevant to consideration of assessment models for river-use risks as well. These problems include:

- Deciding which pathogen to model:
  - If the assessment is to be utilized to evaluate different CSO control options, then every waterborne pathogen implicated by CSO discharges should be modelled. However, information on many of the pathogens in CSOs is not available, especially for pathogens of emerging public priority (e.g., rotaviruses, hepatitis F virus, *E. coli* 0157).

- Inability of many current infection-probability models to take into account or to accommodate:
  - Natural variation in pathogen densities in wastewater.
  - Natural variation in micro-organism viability in the river.
  - Variation in the amount of water consumed during an immersion event, and the natural variation in the fraction of the infective dose ingested during each immersion.
  - Variation in individual susceptibility to certain pathogens (e.g., elderly, immuno-compromised, etc.; Ward *et al.* 1986).
  - Variation in societal susceptibility to certain pathogens.

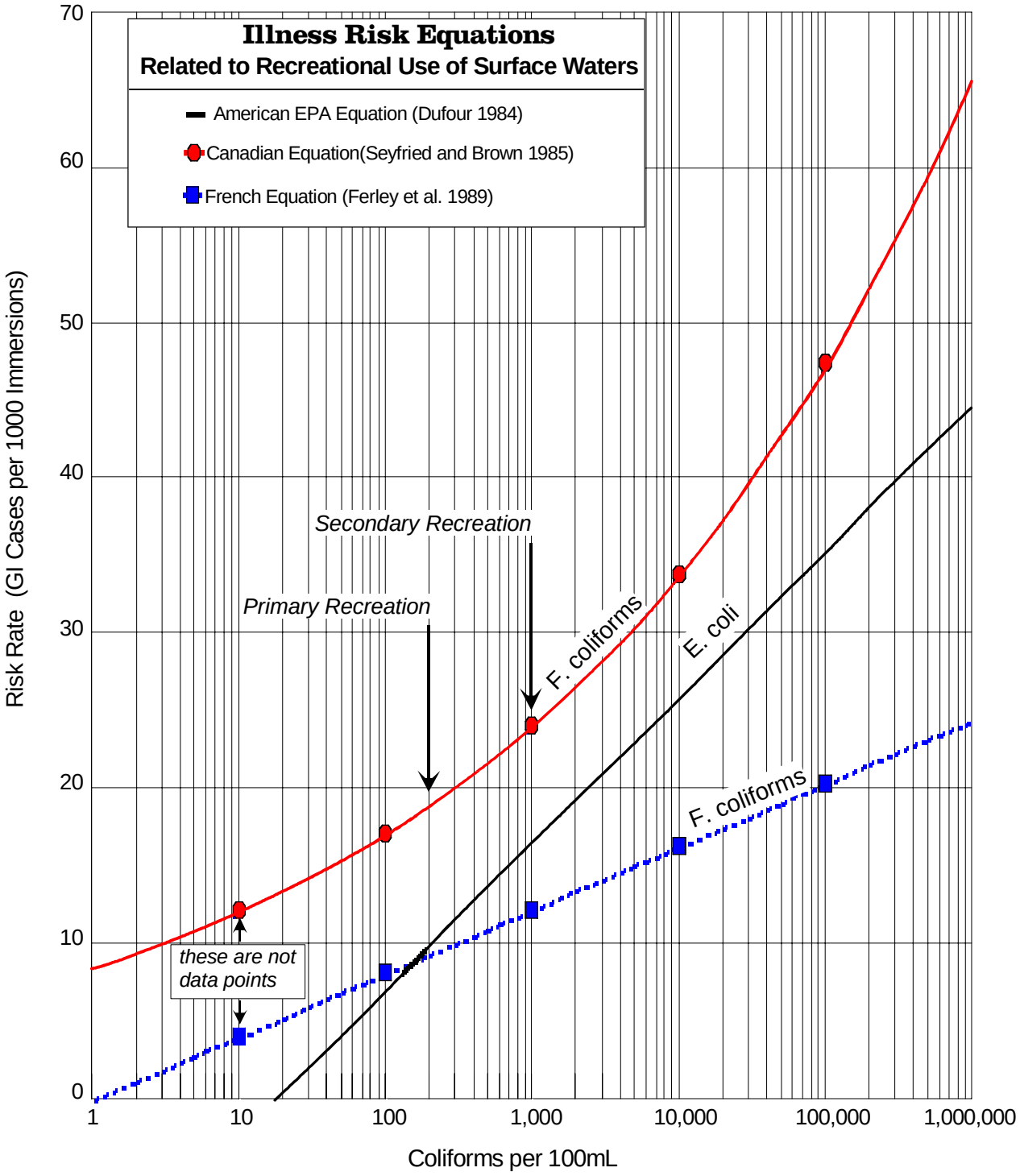
The last two points include the reasons that infection-probability models (as distinct from illness probability models) could not be applied in this study.

3.1.3 Models Known from Previous Illness-Risk Assessments

Epidemiological research on the question of recreational use of surface waters, both inland rivers (and lake beaches) and marine beaches, has resulted in publication of some practical D-R models relevant to the 1997 study.

3.1.3.1 For Indicator Organisms

Some epidemiology studies have been published which have explored recreation risk using illness-risk modelling techniques. Similar to the models used to assess risks from ingesting contaminated drinking water, the D-R models used in most of the recreation-illness risk modelling completed to date have been for indicator organisms (Figure 3-1). These models predict illness-risk rates (i.e., GI cases/1,000 immersions) for various densities of indicator bacteria (usually fecal coliform or *E. coli*). These models were relied upon in performing the QRAs documented in Tetr*ES* previous HRAs (e.g., Wardrop/Tetr*ES* 1991, 1994).



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Estimated Cases of Gastrointestinal (GI) Illness per 1000 Immersions for Coliform Concentrations in Recreational Waters

Figure 3-1

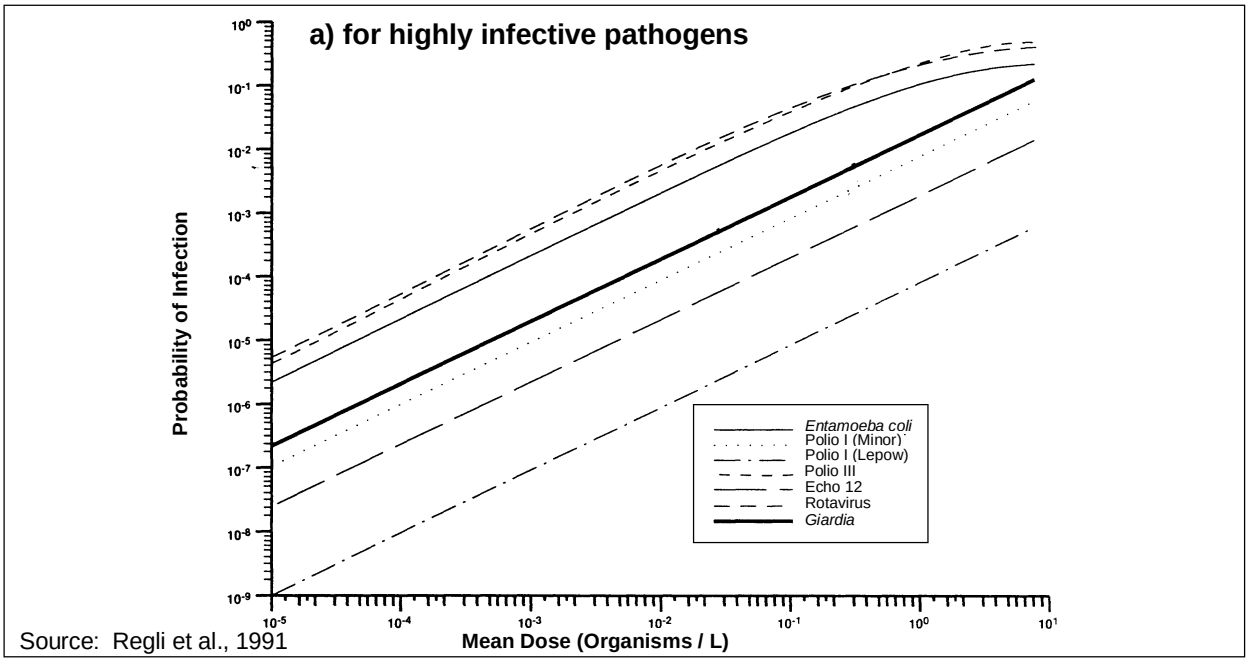
3.1.3.2 For Specific Pathogens

Some D-R models have been developed for specific pathogens, e.g., Figure 3-2. Their use, however, is usually constrained by lack of river-monitoring data for such specific pathogens, and by the need for additional information listed in Section 3.1.3.1 above. Quantifiable D-R relationships for the array of specific pathogens of current interest, expressed in terms of unit rates of recreation, are generally lacking. This has made it difficult to quantify risk from discrete pathogens known or suspected to be present in discharges to rivers.

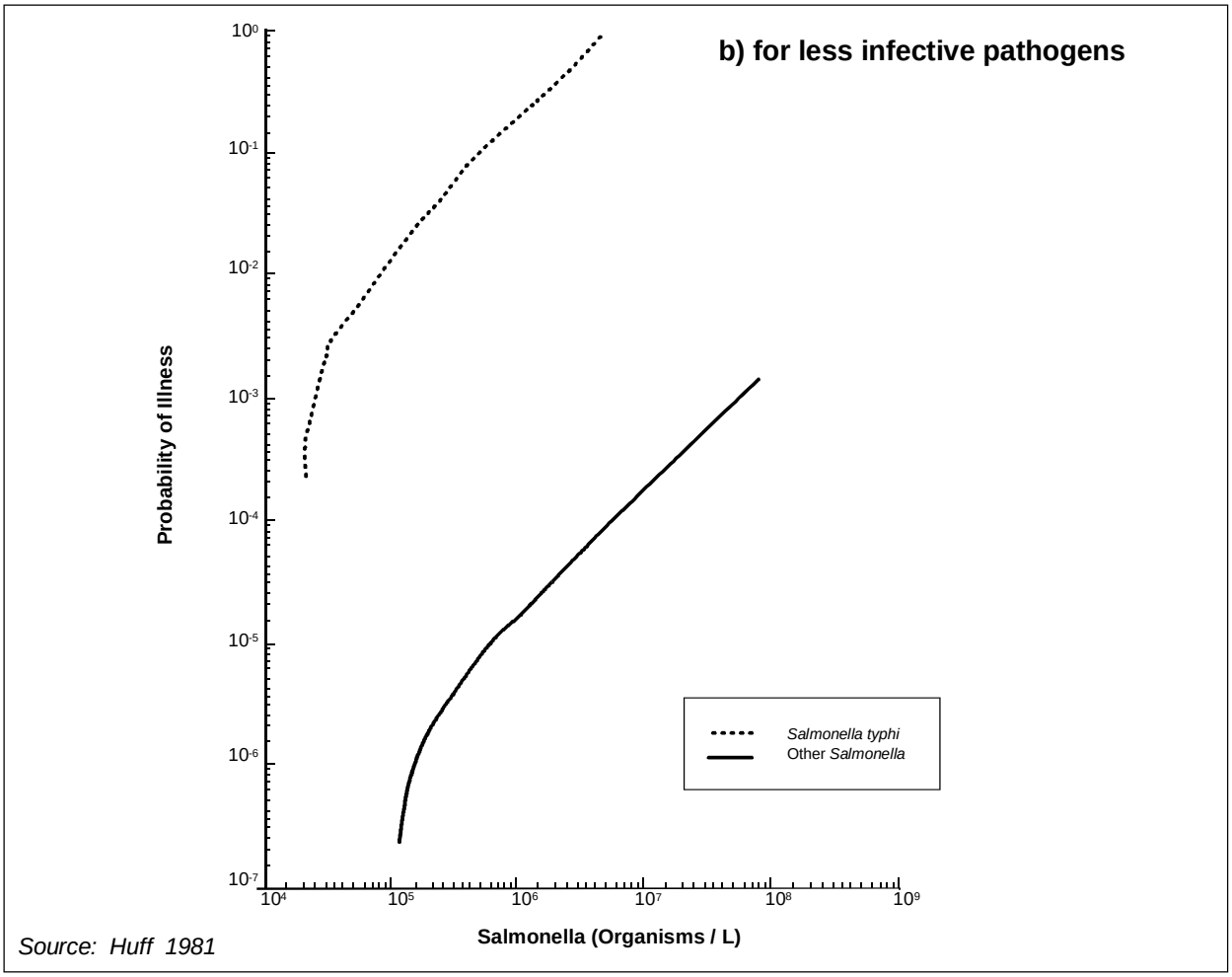
As previously noted, most D-R models have been created by hindcasting. This means they have been constructed from limited available data, because disease-caseload data related to recreational use of surface water are scarce, as has been noted in many epidemiological reports ("...*hospitalization was not reported by any of the [6,000+] subjects....*"; Cabelli 1982). The disease is usually relatively mild and of short duration and, hence, rarely reported. The lack of reported cases accounts, in substantial part, for the paucity of pathogen-specific D-R models.

3.1.4 Types of Illness-Risk Assessment Models

Three basic types of predictive models have been developed to fit the growing experimental database. Consideration of the D-R relationships reported to 1991 indicates that epidemiological relationships for indicator bacteria have evolved from simple linear relationships (as noted in the 1980s literature, e.g., Cabelli *et al.* 1982) to the "β-distributed" models, which are now more normative. The best fit with epidemiological datasets has been found for "β-Poisson-distributed" models (e.g., Regli *et al.* 1991; Haas 1983). The kinds of D-R relationships currently reported are illustrated in Figures 3-3 and 3-4. Dominating the current array of applicable models, they continue to be regarded as modeling's "best fit" tools. Their use is becoming more acceptable to regulators and public-health policy makers in predicting disease incidence because such models tend to over predict societal risk at low indicator (or pathogen) densities. This means that, to regulators and policy-makers, their use in formulating the technical basis for public decisions or public policy is better ("*safer*").



Source: Regli et al., 1991



Source: Huff 1981

Dose-response Relationships of Specific Pathogens

Figure 3-2

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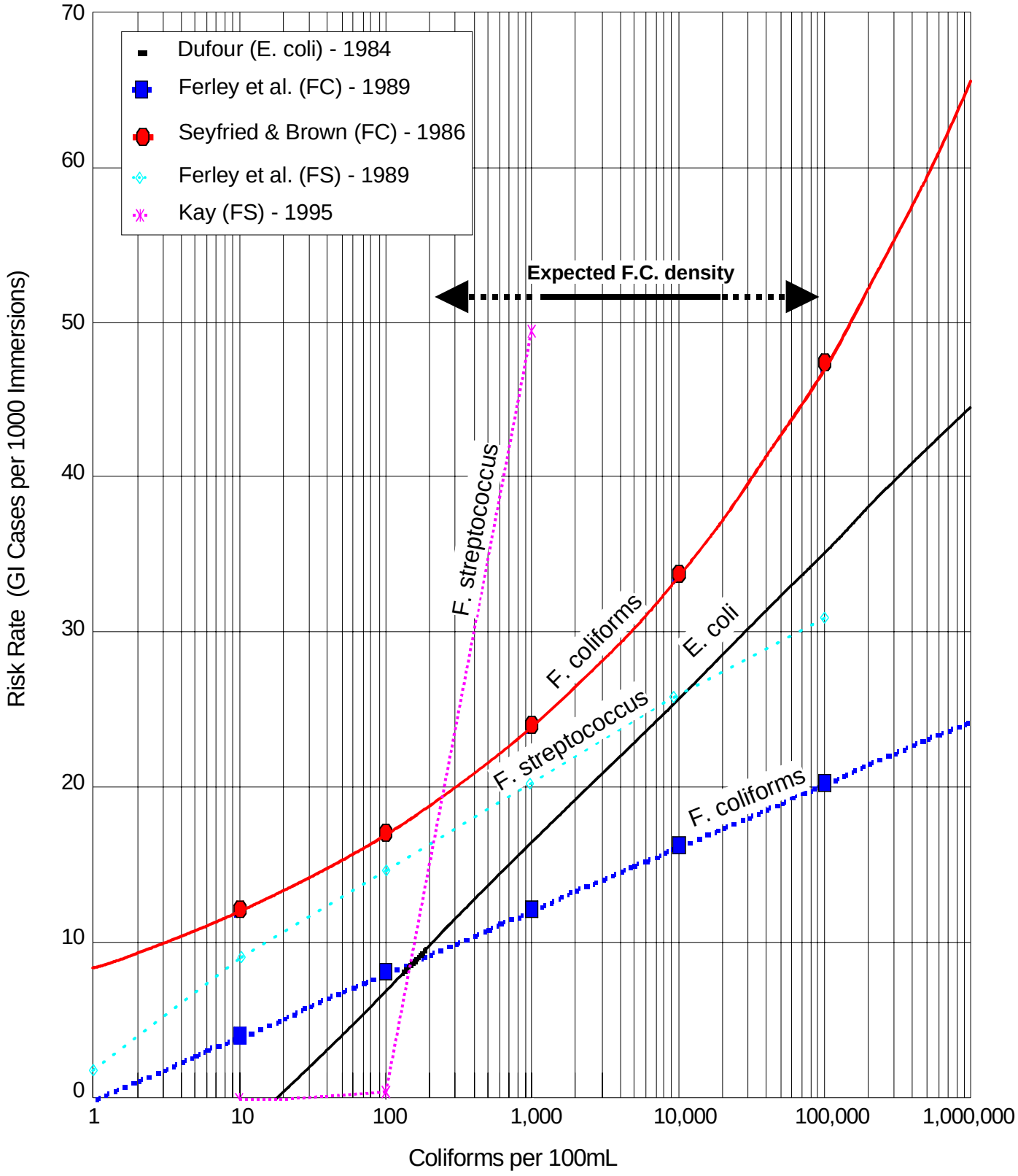

Published "Indicator Species" Dose-Response Models for Estimating Risk of Gastrointestinal Illness from Recreation in Surface Waters

Figure 3-3

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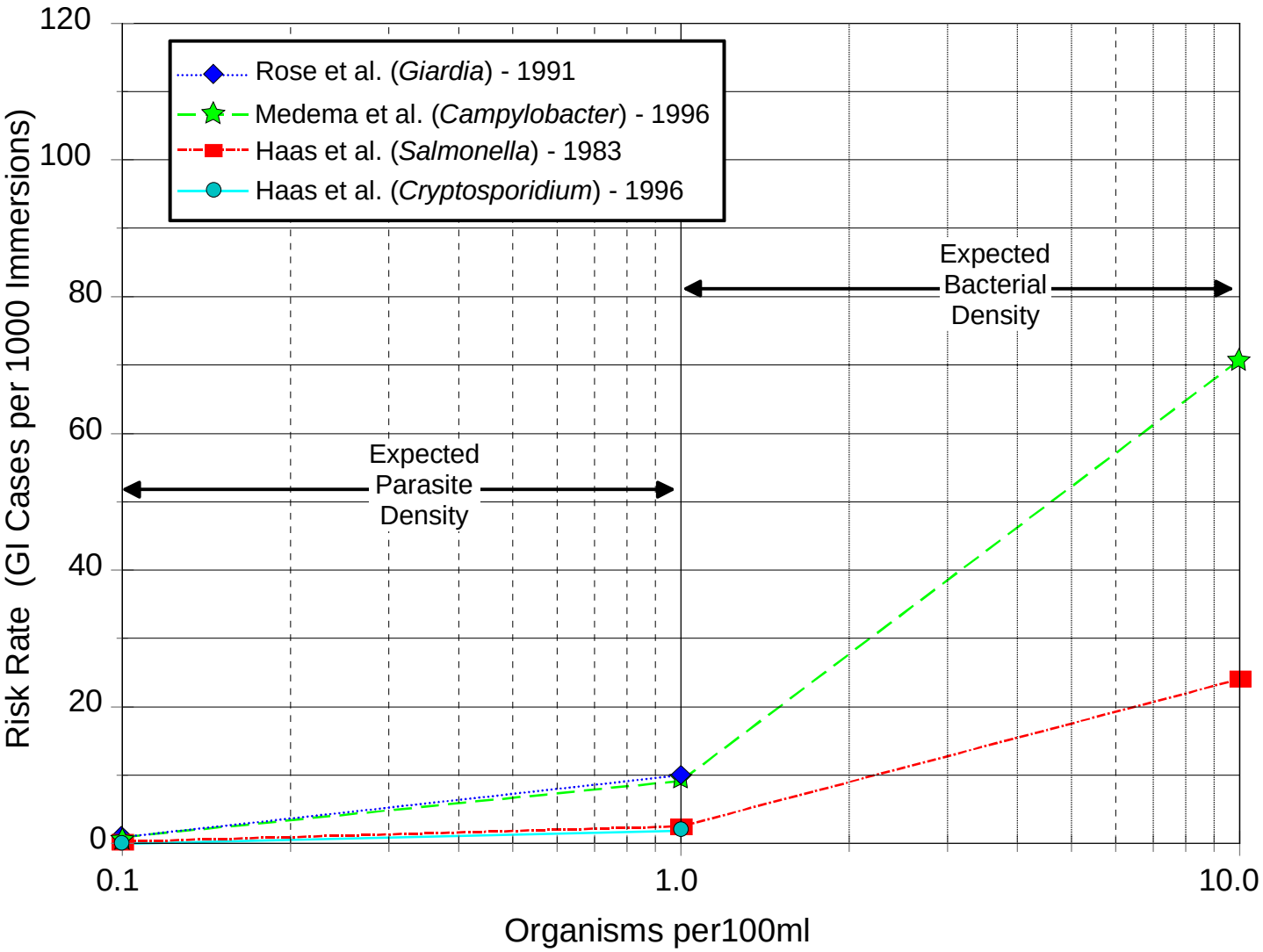

Published Pathogen-Specific Dose-Response Models for Estimating Risk of Gastrointestinal Illness from Recreation in Surface Waters

Figure 3-4

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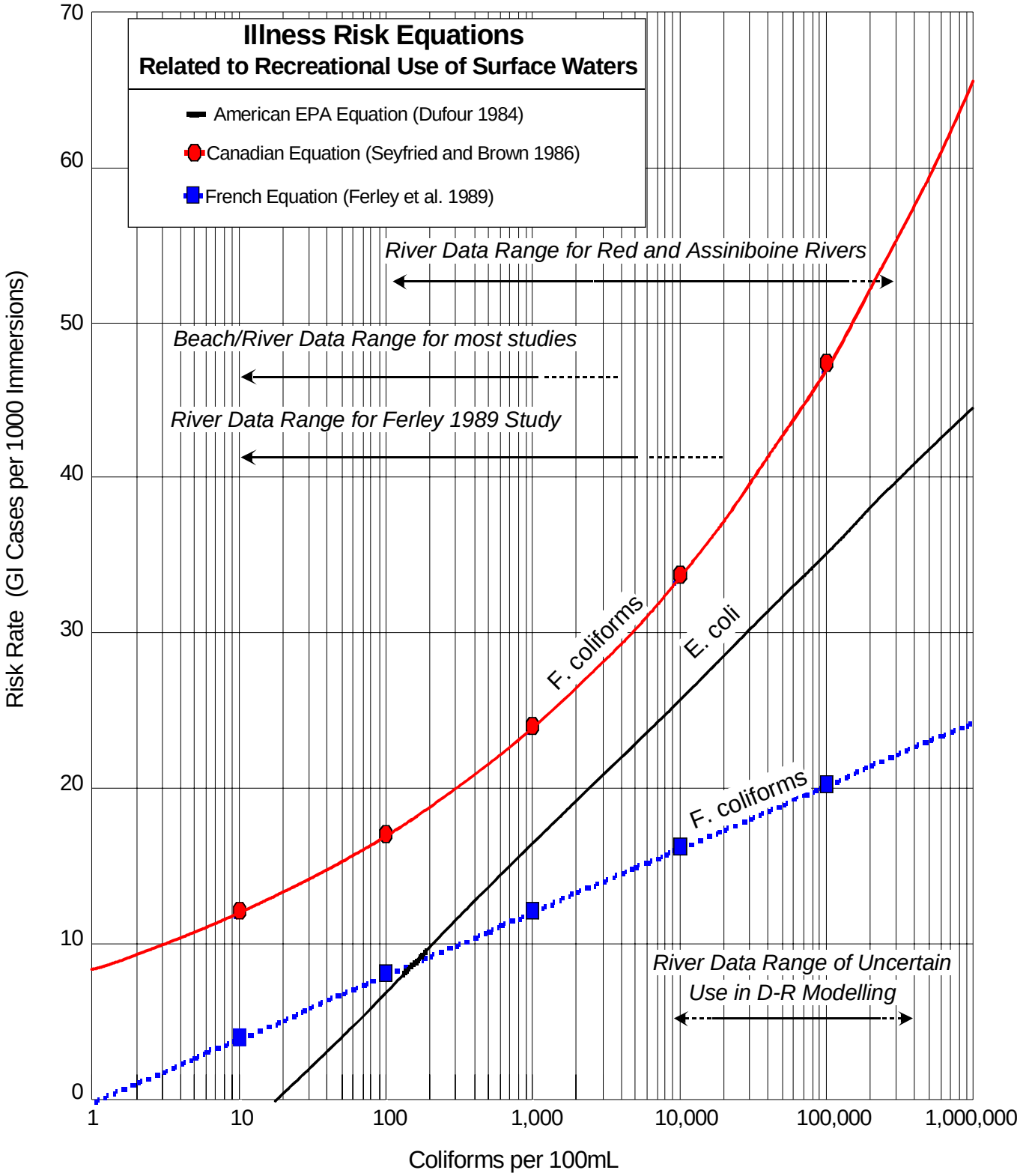
3.1.5 Application of HRA Models to Urban Prairie River Circumstances

The application of published epidemiological relationships to illness-risk modelling has been constrained by uncertainty in the use of some D-R models. Typically, uncertainty arises if the indicator-organism concentration distributions from the original research situation (e.g., bathing beach in Toronto harbour) is significantly dissimilar from the distribution recorded for the area of intended application (e.g., urban reaches of the Red and Assiniboine Rivers). This has meant that extrapolation is often required to estimate the specific river-use risk for organisms where the range of the local dataset differs from the range of the original-research dataset. In the case of the Winnipeg reaches of the Red and Assiniboine Rivers, extrapolation would be required in predicting GI caseloads from exposures to indicator organisms which exceed 1,000/100 mL (Figure 3-5). Clinicians and regulators often emphasize this uncertainty when considering the appropriateness of using D-R models in estimating illness-risk rates from certain types of activity, or the associated disease cases.

Tetr*ES* confidence in applying D-R models (i.e., Dufour, Seyfried and Ferley) relates to the reasons outlined in Section 3.3.3 and the experience gained from previous HRAs conducted for the City of Winnipeg. In the 1991 CoW HRA study, river-use illness (qua indicator bacteria) was found to be driven more by river use than by microbial densities. In the 1991 CoW HRA study, the predicted GI caseload for Winnipeg urban recreation was low because river use was low, i.e., Winnipeg's riverbank areas are not conducive for widespread public access and the turbid character of the river precludes extensive use of the urban river reaches as natural bathing areas.

3.2 CURRENT ARRAY OF PREDICTIVE MODELS

A key component of the 1997 HRA for the City of Winnipeg was a literature review intended to identify the then-current technical ability for Quantitative Risk Assessment (QRA). Over 300 abstracts and scientific papers were reviewed. From this literature, 36 organisms were identified as having potential to be causative agents of disease from recreational (or irrigation) use of inland (i.e., freshwater) waterbodies. On the basis of the abstracts and papers discussing the



Appicability of D-R Models viz. Indicator Organism Data Distribution for Reaches of the Red and Asiniboine Rivers affected by Urban Discharges from Winnipeg

Figure 3-5

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illness-risk significance of these 36 organisms, the list of 36 organisms was screened down to those for which an existing, specific D-R model would allow QRA.

On the basis of this screening process, QRA was found to be possible for 14 of the 36 pathogens or indicators listed initially (Table 3-1). The 14 organisms for which D-R models have now been reported are:

- *Campylobacter* species (spp.) - bacteria
- *Cryptosporidium parvum* - protozoan
- Echovirus (12) - virus
- *Entamoeba coli* - bacterium
- *Escheriscia coli* - bacterium
- fecal coliforms - bacteria
- *Giardia lamblia* - protozoan
- Poliovirus (I,III) - virus
- Rotavirus - virus
- *Salmonella* spp. - bacteria
- *Shigella* spp. - bacteria
- *Staphyloccus* spp. - bacteria
- *Streptococcus* spp. - bacteria

A review of the currently reported models (Table 3-1) also indicates that more D-R models are emerging for specific pathogens, including parasites, as research focuses more on actual causal agents of disease (than on indicators of disease vectors) and on diseases of high public profile (e.g., giardiasis).

It is also clear from a review of these models, especially the newer ones for specific pathogens, that most of the new D-R models have been derived by assessing disease expressions following exposure to (i.e., ingestion of) inoculated drinking water. Their applicability to QRA for ingesting river water is, accordingly, somewhat uncertain.

The 14 reported models were considered for their direct applicability to estimating public recreation (and irrigation) river-use risk within the cities of Edmonton, Alberta and Selkirk, Manitoba under a variety of river-use and control-strategy scenarios.

TABLE 3-1
DOSE-RESPONSE MODELS AVAILABLE FOR QRA
AS DETERMINED FROM 1997 LITERATURE SEARCH

Pathogen	Source	D-R Model
Campylobacter	inoculated in milk, Medema *et al*. 1996	**$P_{inf} = 1-(1 + N/B)^{-a}$** P_{inf} - probability of infection N - ingested number of organisms a and B - D-R parameters determined by infectivity (maximum estimate a = 0.145 and B = 7.59
Cryptosporidium Parvum	freshly prepared single doses, Haas *et al*. 1985	**P= 1-exp(-N/k)** p - predictive portion of affected subjects N - average dose k - average number of organisms that must be ingested Best fit k = 238.6
Echovirus 12	prepared dosage in drinking water, Regli *et al*. 1991	**$Pi = 1-(1+\mu v/\beta)^{-\alpha}$** Pi = probability of infection v = single volume μ = average organisms per unit volume Best fit α = 0.374 β = 186.69
Entamoeba coli	prepared dosage in drinking water, Regli *et al*. 1991	**$Pi = 1-(1+\mu v/\beta)^{-\alpha}$** Pi = probability of infection v = single volume μ = average organisms per unit volume Best fit α = 0.128 β = 0.581
Escheriscia coli	swimming/freshwater, Dufour 1984	**P(1000) = 9.4 log [e.coli] - 11.74**
Fecal coliform	bathers/marine water contaminated with domestic sewage, Fleisher *et al*. 1996b	"**Ln (prob.) of non-enteric illness among bathers= Log(FC-100)**"

TABLE 3-1
DOSE-RESPONSE MODELS AVAILABLE FOR QRA
AS DETERMINED FROM 1997 LITERATURE SEARCH

Pathogen	Source	D-R Model
Fecal coliform (cont'd)	swimmers/river water, Ferley *et al*. 1989	**Prob.inf.(per 1000 person days)= 4.08 log[FC]-0.68**
	swimming/freshwater, Seyfried and Brown 1986 (from 1991 HRA update)	**Log (p/1-p)= 0.35 log[FC]-4.752**
	swimming/freshwater, Seyfried *et al*. 1985a	**Log (p/1-p) = -1.441 + 0.18177 log (fc per 100 ml)**
Giardia lamblia	cyst doses were fed to volunteers, Rose *et al*. 1991	**P = 1- exp (-rN)** r = fraction of microorganisms that are ingested which survive to initiate infection (-0.01982) N = average number of organisms in single volume of water ingested
	prepared dosage in drinking water, Regli *et al.* 1991	**Pi - 1-exp(-rμV)** Pi = probability of infection v = single volume μ = average organisms per unit volume r = 0.02
Poliovirus I	prepared dosage in drinking water, Regli *et al.* 1991	**Pi - 1-exp(-rμv)** Pi = probability of infection v = single volume μ = average organisms per unit volume r = 0.009102
	prepared dosage in drinking water, Regli *et al.* 1991	**$Pi = 1-(1+\mu v/\beta)^{-\alpha}$** Pi = probability of infection v = single volume μ = average organisms per unit volume Best fit α = 0.1097 β = 1524
Poliovirus III	prepared dosage in drinking water, Regli *et al.* 1991	**$Pi = 1-(1+\mu v/\beta)^{-\alpha}$** Pi = probability of infection v = single volume μ = average organisms per unit volume Best fit α = 0.409 β = 0.788
Rotavirus	prepared dosage in drinking water, Regli *et al.* 1991	**$Pi = 1-(1+\mu v/\beta)^{-\alpha}$** Pi = probability of infection v = single volume μ = average organisms per unit volume Best fit α = 0.26 β = 0.42

TABLE 3-1
DOSE-RESPONSE MODELS AVAILABLE FOR QRA
AS DETERMINED FROM 1997 LITERATURE SEARCH

Pathogen	Source	D-R Model
Salmonella	administered orally, Haas, 1983	**$P = 1-(1-(N/B))^{-a}$** P - probability of infection N - dose of organism a, b, are parameters of the Beta-Poisson distribution a= 0.5 B = 100
Shigella	(not clear how dose was administered), Crockett *et al.* 1996	**$P=1-[1+d(2^{(1/a)} - 1)/N50]^{-a}$** P - probability of infection d - dose of organism a, measure of the model's closeness to the Poisson model N50 - median infective dose required to infect half of the exposed population Best fit a - 0.209 N50 – 1120
Staphylococci	swimmers/freshwater, Seyfried *et al.* 1985b	**Log (p/1-p)= -2.65+0.696 log(staph per 100 ml)**
Streptococci	bathers/marine water, Fleisher *et al.* 1996b	**Ln odds of illness among bathers = Log(FS -59)**
	bathing/coastal seawater, Kay *et al.* 1994	**Log(n) odds (of GI)=-0.20102 (sq rt(FS-32))-2.3561**
	swimming/river water, Ferley *et al.* 1989	**Prob. Inf. (per 1000 person days) = 5.97log(fs)+2.26**
	swimming/freshwater, Seyfried *et al.* 1985b	**Log(p/1-P)= -1/302+0.11753 log(FS per 100 ml)**

3.3 RECREATIONAL-USE ILLNESS-RISK RATE ALONG THE RED RIVER

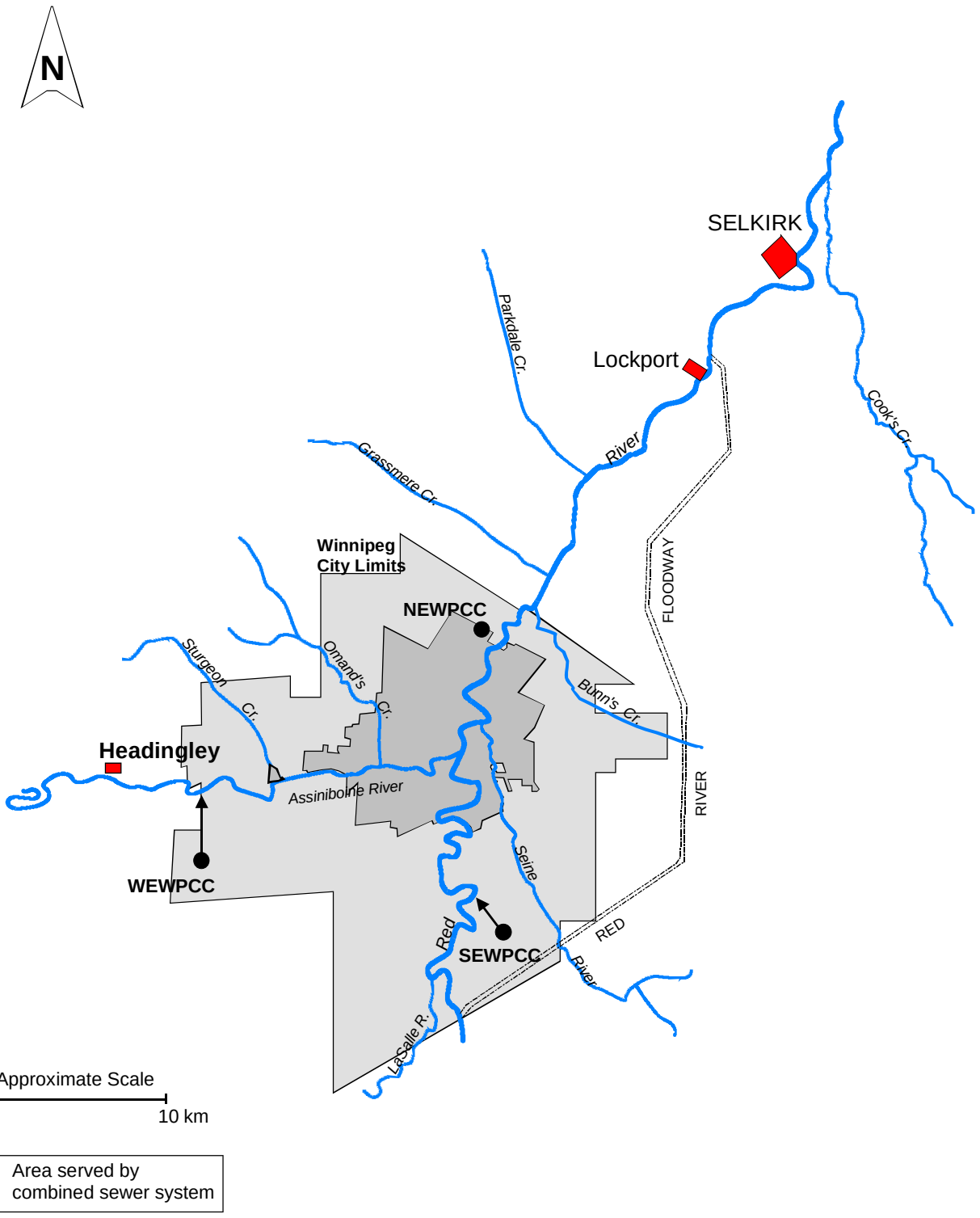
This section will estimate the quantifiable illness risks associated with recreational use of the Red River and the benefits associated with various pollution control technologies, as determined in the 1997 HRA. The analysis focuses on the Red River and not the Assiniboine River because the Red River was classified by the Clean Environment Commission (CEC) as appropriate for primary recreation (i.e., intentional water contact with the likelihood of full-body immersion). The Assiniboine River was classified as suitable for secondary (indirect-contact) recreation. A further factor limiting the use of the Assiniboine River, except in the vicinity of The Forks

Development (confluence of the Red and Assiniboine Rivers), is its shallowness that greatly limits boating activity (i.e., secondary recreation). Accordingly, all river-use statistics and illness risk analyses used in the 1997 study were applied to the Red River.

3.3.1 Study Area

In Winnipeg, dry and wet weather flows discharge into two rivers, the Red and Assiniboine Rivers. The core of the city contains the older combined sewer system while newer areas of development outside of this core contain separate land drainage and sanitary sewer systems. Three conventional secondary treatment plants are located just outside of these newer developments, as shown in Figure 3-6. It is important to note that the rivers enter the urban boundaries of the city where they first encounter continuous discharges from two of Winnipeg Water Pollution Control Centres (WPCC), i.e., the South End WPCC (SEWPCC) on the Red River, and the West End (WEWPCC) on the Assiniboine River. As the rivers flow past these continuous discharges, they pass through areas affected by land drainage discharges only during rainfalls. As both rivers flow past these areas, they enter into areas affected by combined sewer overflows. The rivers continue to flow through the combined sewer areas until they reach the North End WPCC (NEWPCC). The NEWPCC is the last discharge into the Red River from Winnipeg. The next major settlement is the City of Selkirk some 30 km downstream. Several minor discharges exist in this stretch of river and the impacts of their discharges on the water quality prior to Selkirk are uncertain. Accordingly, urban reaches of the Red and Assiniboine Rivers will benefit differently from proposed water pollution control technologies and therefore must be assessed on a reach-by-reach basis to place the accumulative benefits of control actions into proper context, as illustrated in Figure 3-6.

An important consideration in the evaluation of illness risk associated with our local rivers is their recreational use and extent of downstream impacts attributable to urban discharges from Winnipeg. A major hydraulic structure, the St. Andrews Lock and Dam, located downstream of the City of Winnipeg and upstream of the City of Selkirk, significantly influences the hydraulic character of the Red and Assiniboine Rivers in the urban reaches of Winnipeg. This structure



Study Area
Figure 3-6

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artificially increases water depth in the Red River and provides a suitable condition for recreational boating. Water levels are targeted to remain relatively constant at the confluence of the two rivers for a wide range of flows. The Assiniboine River has a steeper gradient and is only affected by backwater from the St. Andrews Dam for about 6 km upstream of the confluence under normal flows. As indicated earlier, the Assiniboine River was deemed by the CEC to be unsuitable for a primary recreational river use designation, and its shallow depth greatly limits its use for boating except in the near vicinity of the confluence (i.e., Forks Development). Accordingly, the extent of the 1997 study area was limited to the Red River from Winnipeg's upstream urban boundary to Selkirk.

3.3.2 River Use

A detailed river use survey was conducted in the Red and Assiniboine Surface Water Quality Objectives Study (Wardrop/Tetr*ES* 1991). The survey consisted of five sources of information, as listed below:

- Anecdotal information collected in 1986 by MacLaren Engineers Inc. as part of the report titled "Disinfection Evaluation: City of Winnipeg Wastewater Treatment Plant Effluents."
- Actual counts of river activity from 5 aerial surveys by the City of Winnipeg in June, July and August 1990.
- Anecdotal information collected in 1990 by the Province on club activities.
- Anecdotal information collected by the City of Winnipeg in 1990 from the Harbour Master.
- Results gathered as part of a telephone survey conducted by the City of Winnipeg in 1990.

Since this survey, it is believed that only minor differences existed in 1997 and current recreational river use. As such, the 1990 survey information is considered sufficiently accurate for subsequent estimates of illness risk associated with ingestion of raw river water. The survey found the following conclusions:

A) Immersion from primary recreation

- Winnipeg area = 3,800 events
- Selkirk area = 400 events

Total = 4,200 events

B) Immersion from secondary recreation

- Winnipeg area = 1,900 events
- Selkirk area = 400 events

Total = 2,300 events

for a 100-day recreation season (Victoria Day [May long weekend] to Labour Day [September long weekend]). In order to be consistent with the full extent of the recreation season, May 1 to September 30 (inclusive), the total recreation days needs to be increased to 153 days. A simple scaling factor of 153/100 was applied to the immersion numbers to calculate the full recreation season immersion estimates of:

A) Primary Recreation

- Winnipeg area 3,800 x 1.53 = 5,814 events
- Selkirk area 400 x 1.53 = 612 events

Total = 6,426 events

B) Secondary Recreation

- Winnipeg area 1,900 x 1.53 = 2,907 events
- Selkirk area 400 x 1.53 = 612 events

Total = 3,519 events

Accordingly, the total estimated number of immersions resulting in ingestion of raw river water from Winnipeg to Selkirk (inclusive) for the full recreation season is 9,945 unique events.

3.3.3 Dose-Response (D-R) Model Selection

Three published D-R equations were considered most appropriate for quantitative risk estimation:

- Ferley *et al.* 1989 fecal coliform model
- Seyfried & Brown 1986 fecal coliform model
- Dufour 1984 *E. coli* model

These 3 D-R equations were considered the most applicable because they are expressed in terms of unit rates of recreational use and because they focus on fecal coliforms or *E. coli*. Water-quality monitoring and modelling of discharges to the Red and Assiniboine Rivers have focussed to date on the fecal coliform indicator organism. Fecal coliform concentrations are often considered a reasonable surrogate for concentrations of *E. coli*. Other models having potential relevance (Regli *et al.* 1991; Seyfried *et al.* 1985b) could not be applied because of absence of relevant river-monitoring data (e.g., for rotavirus, *Giardia, Entamoeba, Staphylococcus, Streptococcus*) and because the epidemiological work needed to apply them (Sections 3.1.1 and 3.1.2) was beyond the scope of the 1997 study.

All three models listed above and discussed earlier in the report will be used to estimate illness-risk rates and their reduction by various pollution control options.

The potential reduction in recreational-use illness-risk rates along the Red River were determined by estimating the reduction in fecal coliform levels in the river in response to specific wastewater control programs. The current risk of acquiring gastrointestinal (GI) from full body immersion was estimated by applying estimates of organism densities from calibrated and verified river water quality models for Winnipeg urban reaches into these three D-R equations.

3.3.4 In-stream Microbial Densities

In part of this assessment, one-dimensional water quality modelling was conducted to estimate the potential effects of UV disinfection projects on the Red River water quality. The model simulation for the representative year 2004 was calibrated and verified with the actual river data to provide a high degree of confidence in the model predictions. In this model, many key factors increasing the fecal coliform levels were considered: wastewater discharge from WPCCs, combined sewer overflow; and land drainage sewer loadings.

The input 2004 data used in this assessment were obtained from the City of Winnipeg. The fecal coliform parameter along the Red and Assiniboine Rivers was measured monthly (from April to November), while the WPCC effluent quality was measured daily (year-round).

The water quality modelling simulated the spatial variation of fecal coliform densities along the Red river within the study area. This method is consistent with the method used in the 1997 Winnipeg illness risk assessment study (Tetr*ES*/Wardrop 1997). Figure 3.7 illustrates result of the existing fecal coliform levels on the Red river for full recreation season.

As noted earlier, the 2004 analysis focuses on the Red River. This is more realistic in terms of where river use occurs. Fecal coliform concentrations along the Red River were used in the D-R models. This model will result in a slightly higher estimate of an area-wide geometric mean fecal coliform concentration than if the Assiniboine River values were included and, consequently, may overestimate the actual number of illness cases.

3.3.5 Pollution Control Scenarios

To assess the improvement in water quality, as measured by a reduction in illness risk, the following three scenarios were considered:

1. Baseline Conditions:
    - Baseline for 2004: This represents current situation without project upgrade.

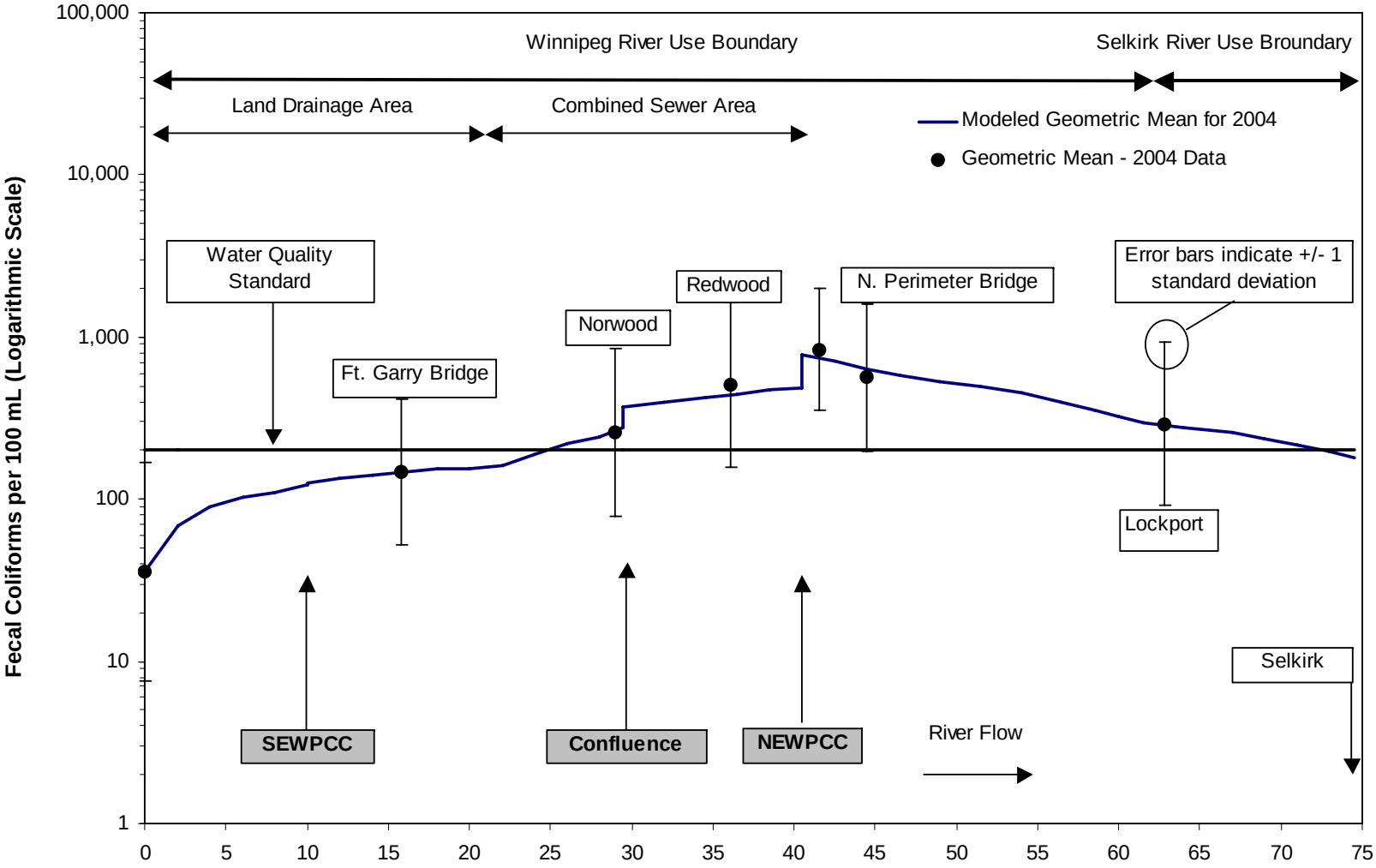


Figure 3-7 Fecal Coliform Concentrations in Red River (representative year, 2004)

- Baseline for 2031: This represents the situation by the end of the design period for the WPCCs.

2. Effluent Disinfection: This condition represents the disinfection of all dry weather effluent discharges from Winnipeg's three wastewater treatment plants.

It should be noted that effects of separate sewer systems are not considered in this assessment. Figure 3-8 illustrates the fecal coliform profile along the Red River for the two scenarios discussed above.

3.4 ASSESSMENT OF POLLUTION CONTROL ALTERNATIVES

The following section will examine the GI illness-risk rates associated with the three dose-response (D-R) equations identified in Section 3.3.3, and estimate the reduction in illness-risk rates associated with specific pollution control scenarios identified in Section 3.3.5 as they relate to reduced fecal coliform levels.

3.4.1 Fecal Coliform Concentrations

With the preceding information, it was possible to quantitatively assess the benefits of reduced illness risk that could be achieved through additional pollution control of discharges to the river. Figure 3-9 illustrates the predicted benefit of specific pollution control scenarios and the resulting geometric mean fecal coliform densities along the Red River for the full recreation season. Specifically:

1. The baseline is represented by the top line of the predicted fecal coliforms.

2. The benefit of dry-weather effluent disinfection is indicated by the grey shaded area. The bottom edge of the grey shaded area represents the remaining fecal coliform density after dry-weather effluent disinfection. It is noteworthy that this action alone has the capacity to reduce existing levels at most locations to below the Manitoba Water Quality Objective of

Predicted Geometric Mean Fecal Coliform Levels Along Red River

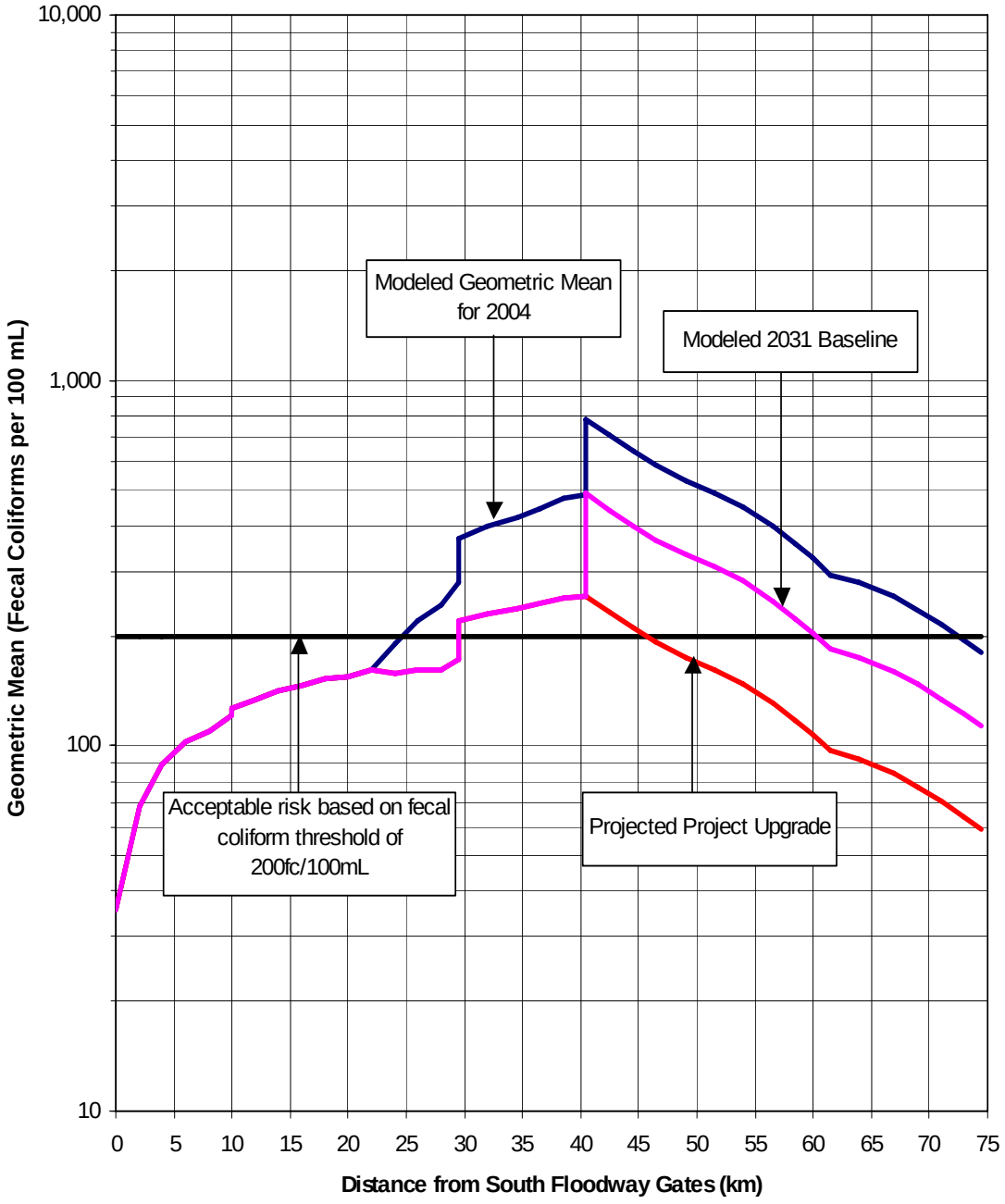


Figure 3-8

Note: Modeled baseline and projected upgrade values are geometric means.

Reduction in Fecal Coliform Levels Along Red River

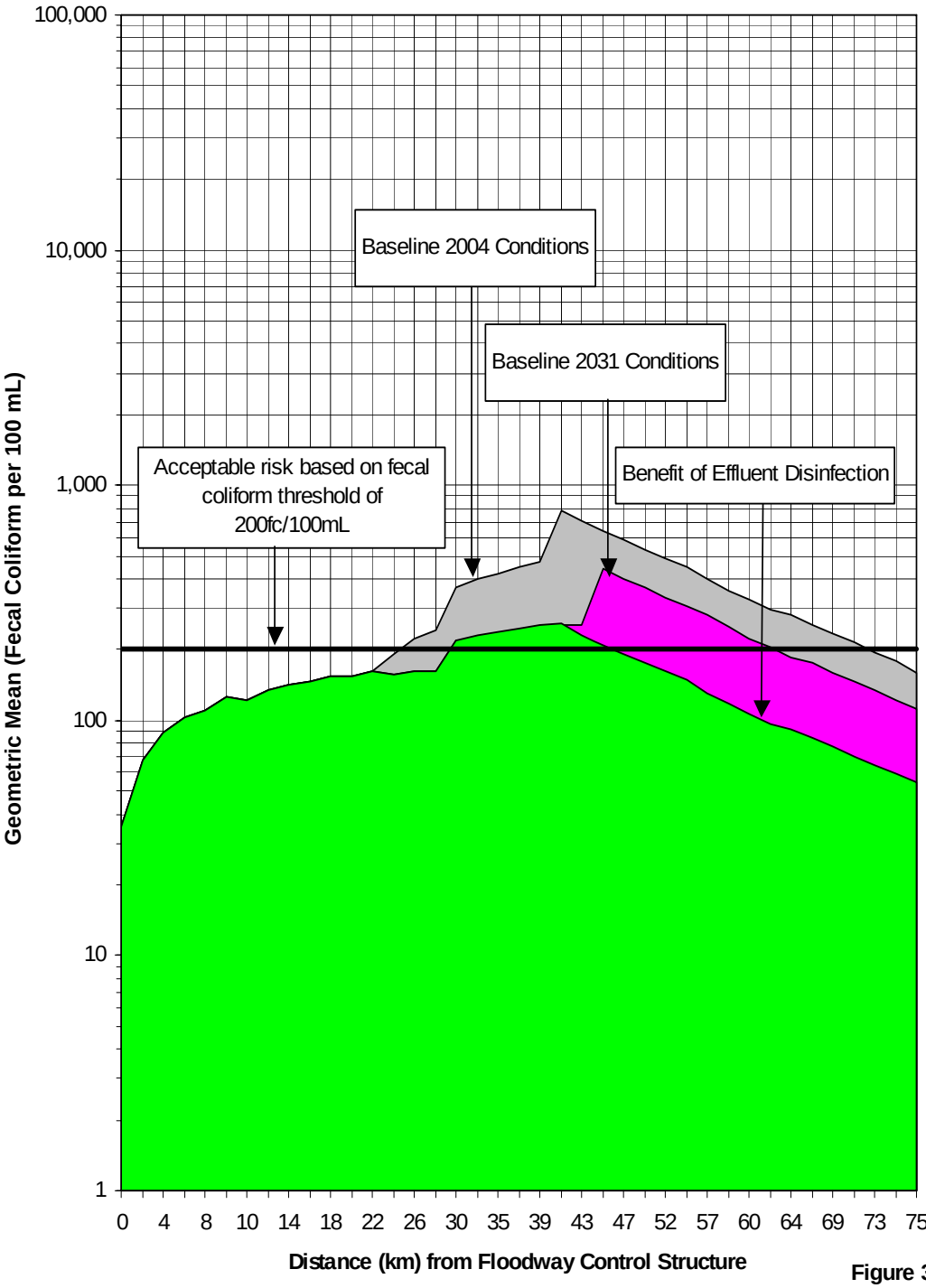


Figure 3-9

200 fecal coliform/100 mL. Simply put, disinfection of effluent discharges can achieve compliance at most locations, with the 200 fc/100 mL objective for a representative year, on the basis of geometric means.

3.4.2 Illness-Risk Rate Reduction

Using the geometric mean of fecal coliforms and applying the three D-R equations (discussed in Section 3.3.3) to the three scenarios noted above, it is possible to calculate the corresponding health risks along the Red River for the full recreation season. Figures 3-10a, b, and c display the estimated gastrointestinal (GI) illness cases for the predicted fecal coliform densities associated with the pollution control scenarios. The acceptable risk levels for primary recreation, based on the MWQO of 200 fc/100 mL, are noted on each graph for the corresponding D-R equation. The predicted GI cases vary according to each D-R equation but all have the same shape and indicate similar results relative to the acceptable levels of risk at 200 fc/100 mL.

It is important to understand these key features in relation to the health-risk rates when viewing Figures 3-10a, b and c:

- the river flow is from left to right (i.e., from kilometer 0 to kilometer 75)
- kilometer zero is considered the upstream urban boundary of Winnipeg on the Red River
- SEWPCC discharges treated effluent (non-disinfected) into the Red River at kilometer 9
- land drainage discharges occur between kilometer 0 and kilometer 20
- the combined sewer area begins at kilometer 20 and extends through to kilometer 41
- the Assiniboine River flows into the Red River at kilometer 30
- NEWPCC discharges treated effluent (non-disinfected) into the Red River at kilometer 42 and represents the last discharge to the Red River from Winnipeg
- kilometer 61 is where St. Andrews Lock and Dam is situated
- kilometer 75 is the upstream urban boundary of Selkirk

Illness Risk Along Red River using Canadian Equation (Seyfried and Brown 1986)

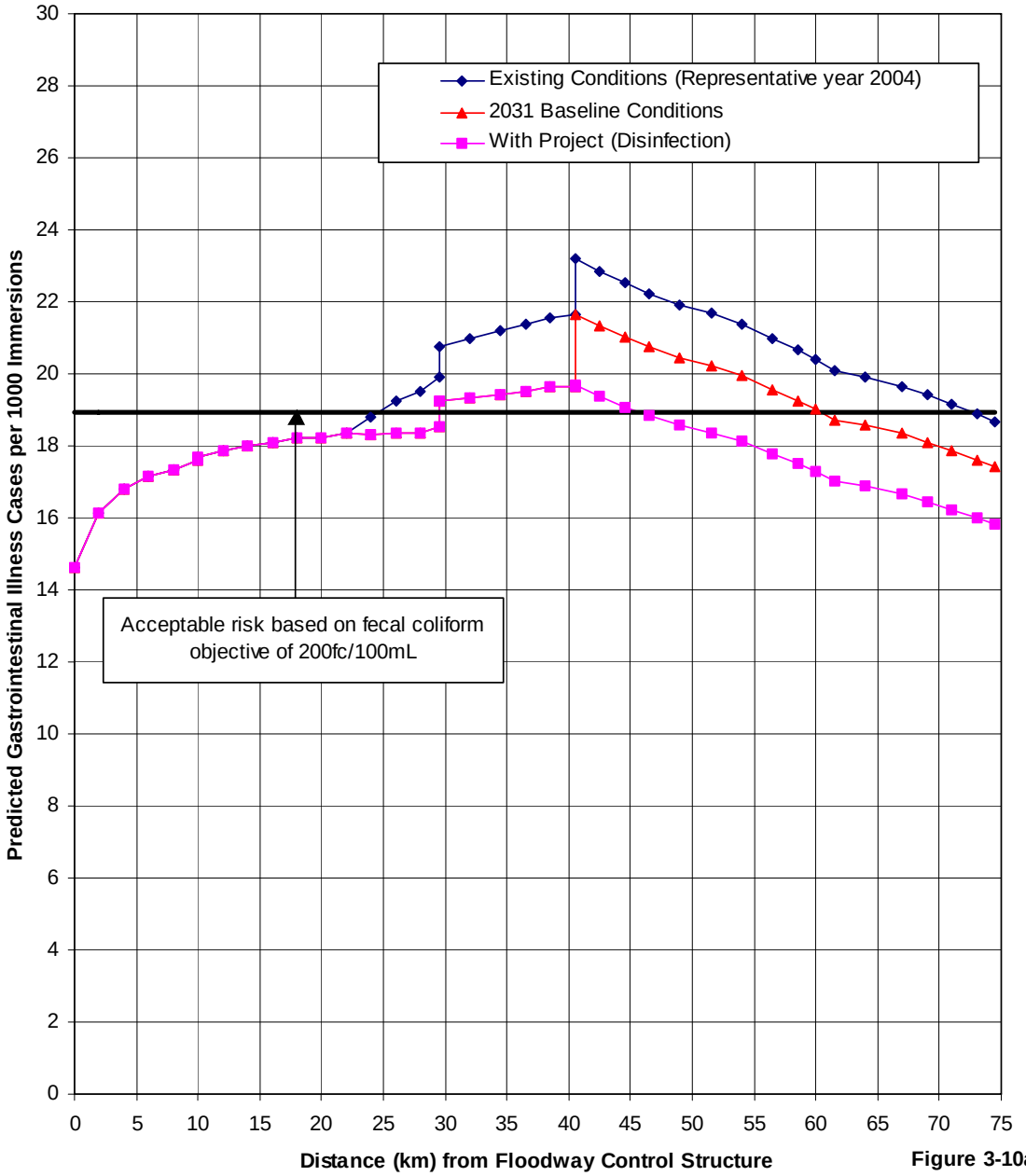


Figure 3-10a

Illness Risk Along Red River using American Equation (Dufour 1984)

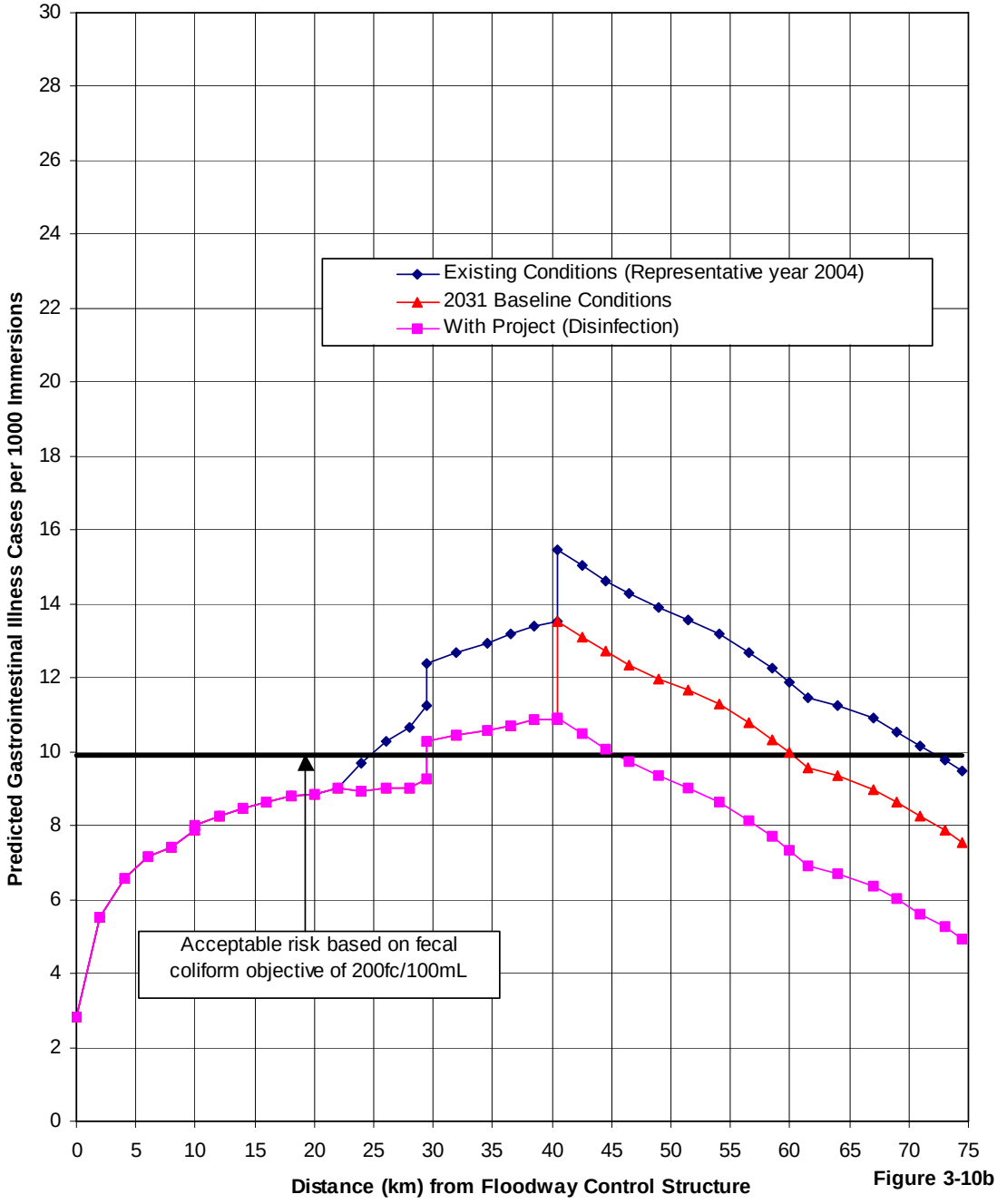


Figure 3-10b

Illness Risk Along Red River using French Equation (Ferley *et al.* 1989)

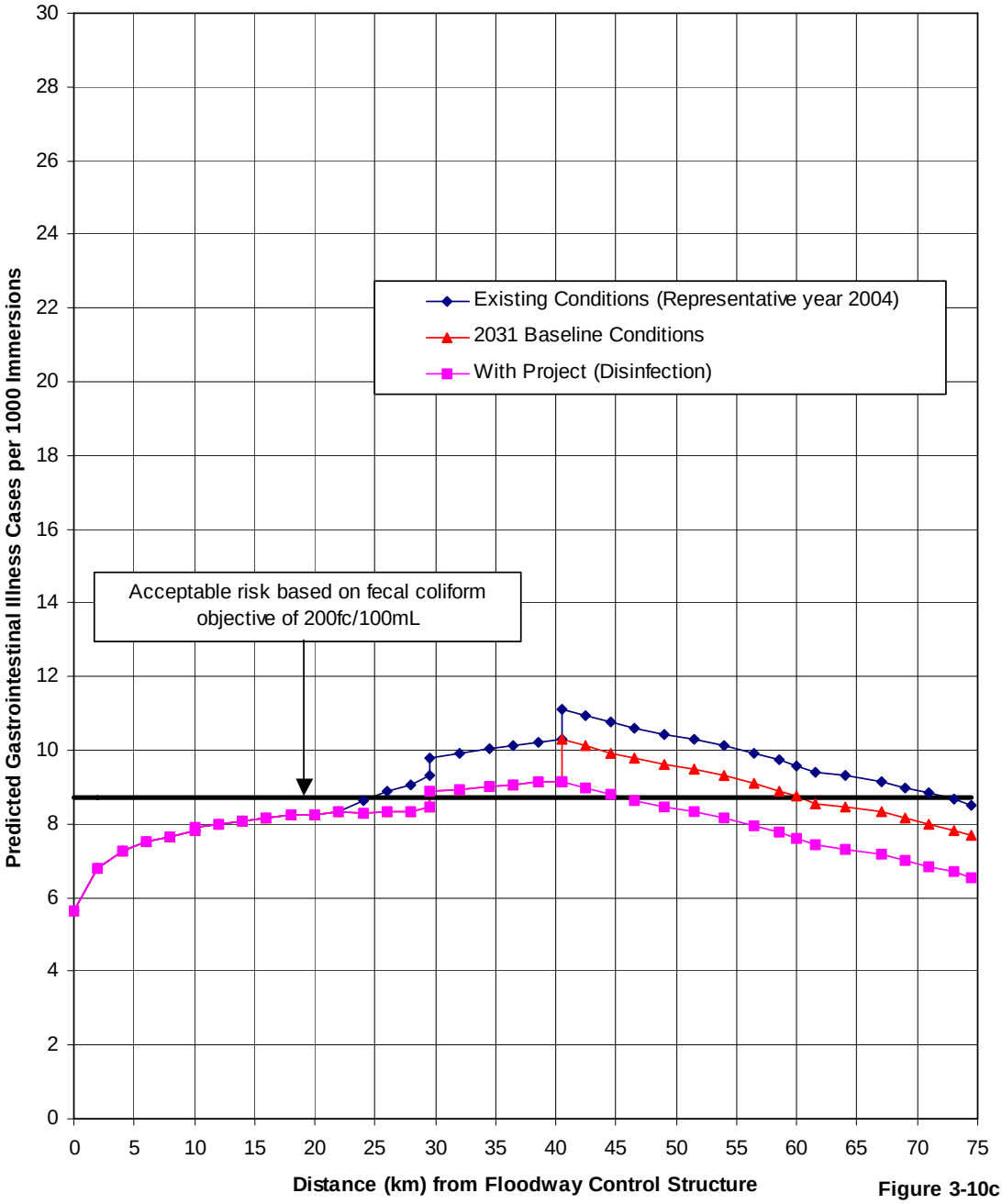
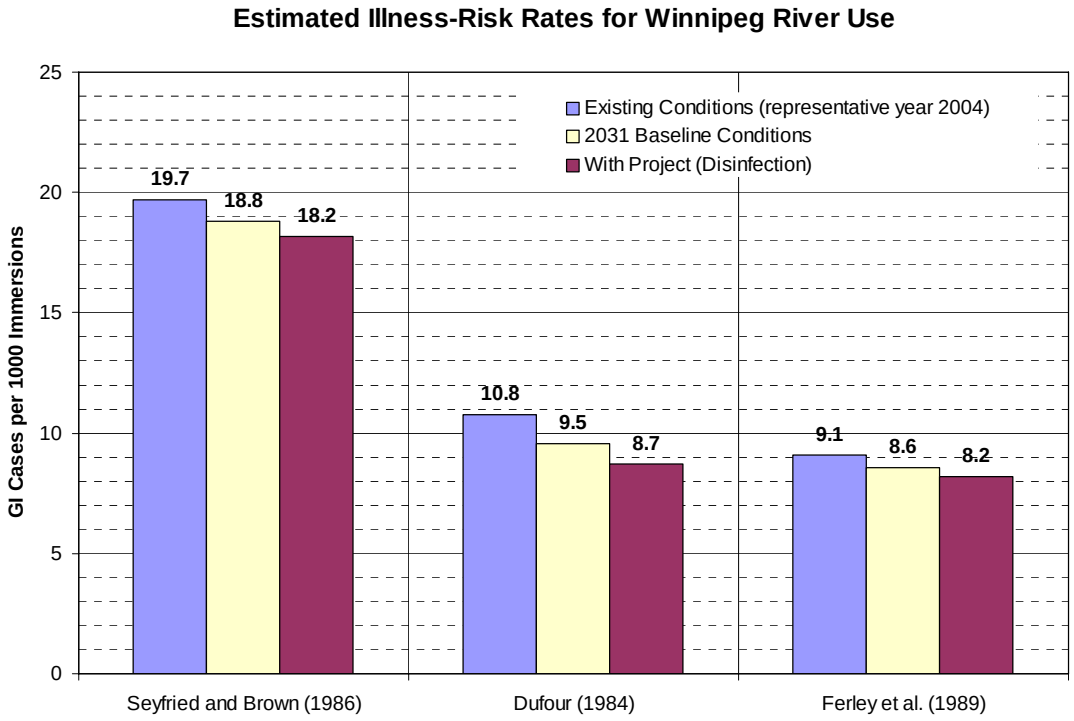


Figure 3-10c

The following sections discuss the health-risk rate profiles associated with the three D-R equations and pollution control scenarios shown on Figures 3-11a, b and c.

A) Seyfried and Brown, 1986 (Figure 3-10a)

- this D-R equation yields consistently higher predicted GI cases then the other two D-R equations.
- the "acceptable risk rate at 200 fc/100 mL", primary recreation objective, corresponds to about 19 GI cases/1,000 immersions.
- **under 2004 baseline conditions**
  - the primary recreation objective is not exceeded immediately downstream of the SEWPCC outfall and is exceeded for 18 kilometers downstream of the NEWPCC outfall. The objective is exceeded from kilometer 25 onward, likely due to CSO flows.
    - maximum risk rates at SEWPCC and NEWPCC outfalls are 21.1 and 23.2 GI cases/1,000 immersions, respectively.
  - the health risk rate at Selkirk (river kilometre 75) is slightly below the acceptable health-risk rate for primary recreation and is normally in compliance.
- **under 2031 baseline conditions**
  - the primary recreation objective is exceeded for 31.5 kilometres. The distances of exceedance are illustrated between downstream of SEWPCC and NEWPCC outfalls.
    - maximum risk rate for the distances of exceedance is 21.7 GI cases/1,000 immersions at the outfall of NEWPCC.
- **with UV disinfection upgrade**
  - the overall risk rate is well below the primary recreation objective in comparison with the baseline conditions. A small distance of exceedance (15 kilometres) is illustrated between downstream of SEWPCC and NEWPCC outfalls.
    - maximum risk rate for the distances of exceedance is 19.7 cases/1,000 immersions at the outfall of NEWPCC.


Estimated Illness-Risk Rates for Winnipeg River Use
GI Cases per 1000 Immersions
25
20
15
10
5
0
Existing Conditions (representative year 2004)
2031 Baseline Conditions
With Project (Disinfection)
19.7
18.8
18.2
10.8
9.5
8.7
9.1
8.6
8.2
Seyfried and Brown (1986)
Dufour (1984)
Ferley et al. (1989)

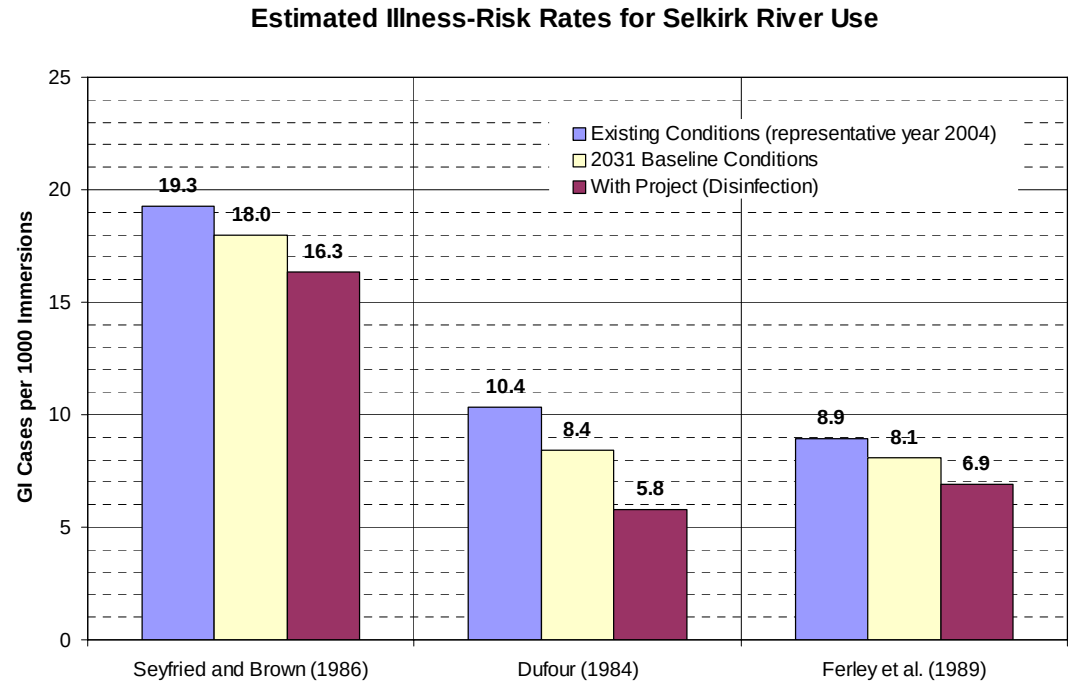

Estimated Illness-Risk Rates for Selkirk River Use
GI Cases per 1000 Immersions
25
20
15
10
5
0
Existing Conditions (representative year 2004)
2031 Baseline Conditions
With Project (Disinfection)
19.3
18.0
16.3
10.4
8.4
5.8
8.9
8.1
6.9
Seyfried and Brown (1986)
Dufour (1984)
Ferley et al. (1989)

Figure 3-11

B) Dufour, 1984 (Figure 3-10b)

- this D-R equation predicts lower GI cases than the Seyfried and Brown D-R equation for all fecal coliform levels.
- the Dufour D-R equation brackets the predicted rates from the Ferley D-R equation, i.e., this D-R equation predicts higher GI risk rates for higher fecal coliform levels than the Ferley D-R equation, and predicts lower GI case rates for lower fecal coliform levels than the Ferley D-R equation.
- the "acceptable risk rate" at 200 fc/100 mL (primary recreation objective) corresponds to about 10 GI cases/1,000 immersions.
- **under 2004 baseline conditions**
  - same distances of exceedance as for Seyfried and Brown D-R equation except lower GI health-risk rates, i.e.,
    - maximum risk rate of 15.5 GI cases/1,000 immersions at the outfall of NEWPCC
    - maximum risk rate of 13.5 GI cases/1,000 immersions downstream of the SEWPCC
- **under 2031 baseline conditions**
  - same distances of exceedance as for Seyfried and Brown D-R equation
    - maximum risk rate for the distances of exceedance is 13.5 GI cases/1,000 immersions at the outfall of NEWPCC.
- **with UV disinfection upgrade**
  - same distances of exceedance as for Seyfried and Brown D-R equation
    - maximum risk rate for the distances of exceedance is 10.9 GI cases/1,000 immersions at the outfall of NEWPCC.

C) Ferley *et al.* 1989 (Figure 3-10c)

- considered the most appropriate D-R equation because of circumstances used to develop the relationship (i.e., in a river with range of fc densities closer to local conditions).
- the "acceptable risk rate" at 200 fc/100 mL (primary recreation objective) corresponds to about 8.7 GI cases/1,000 immersions.

- **under 2004 baseline conditions**
  - same distances of exceedance as Seyfried and Brown D-R equation except lower GI health-risk rates
    - maximum risk rate of 11.1 GI cases/1,000 immersions at the NEWPCC outfall
    - maximum risk rate of 10.3 GI cases/1,000 immersions at the SEWPCC outfall
- **under 2031 baseline conditions**,
  - same distances of exceedance as for Seyfried and Brown D-R equation
    - maximum risk rate for the distances of exceedance is 10.3 GI cases/1,000 immersions at the outfall of NEWPCC
- **with UV disinfection upgrade**,
  - same distances of exceedance as for Seyfried and Brown D-R equation except lower GI health-risk rates, i.e.,
    - maximum risk rate of 9.2 GI cases/1,000 immersions.

It was noted earlier that St. Andrews Lock and Dam is a major hydraulic control structure located about 19 kilometres downstream of the City of Winnipeg and 9 kilometres upstream of the City of Selkirk. Although boaters do take advantage of the Lock, this structure tends to divide river use recreation to either upstream or downstream of this structure.

Accordingly, illness-risk assessments were performed for Winnipeg to Lockport (deemed Winnipeg river use) and Lockport to Selkirk (deemed Selkirk river use). It is assumed that river use occurs uniformly within these stretches of the Red River for river use estimates noted in Section 3.3.2. On this basis, it was possible to calculate area-wide health-risk rates for Winnipeg and Selkirk for the 3 D-R equations noted earlier. Figure 3-11 illustrates the health-risk rates for Winnipeg and Selkirk for the 3 D-R equations. It is interesting to note that there was an increasing trend at the confluence point for both baseline condition and UV disinfection upgrade scenarios. This indicates that there are numerous activities, such as land drainage, that affect fecal coliform concentrations other than the UV disinfection upgrade.

3.4.3 Caseload Reduction

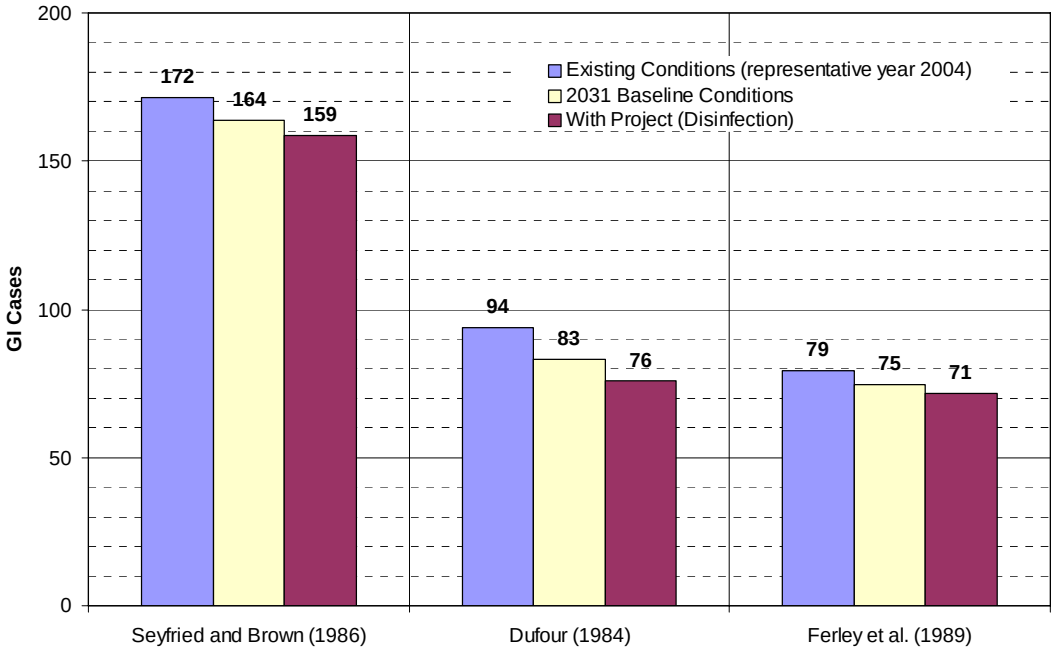
To help place the results into perspective, the area-wide illness-risk rates for Winnipeg and Selkirk control scenarios were used in the D-R equations and multiplied by the appropriate number of immersions and estimated GI caseload, as shown in Figure 3-12. It was found that the estimated number of GI cases predicted by Seyfried and Brown, Dufour and Ferley models were 195, 106 and 90 for existing conditions (see Figure 3-13), respectively. Accordingly, the implementation of UV disinfection technology is expected to result in a benefit of 17, 24 and 10 avoided cases of GI, depending on the D-R model used.

4.0 OTHER CONSIDERATIONS

As discussed earlier, wide temporal and spatial variations in fecal coliform densities exist along the Red River over the course of the recreation season. Disinfection of WPCC effluents during dry weather conditions will not eliminate wet weather discharges and the associated peak fecal coliform densities in the rivers, i.e., combined sewer overflows cause a significant rise in fecal coliform densities for a short period of time. The resultant area-wide geometric mean fecal coliform densities reflect this condition to some extent, since there are significantly more dry days than rainfall days. Fecal coliform levels in the rivers rise sharply in response to rainfall and then die off quickly, returning to background conditions within 3 to 4 days as they travel with the flow of the river. These periodic events are accounted for in the illness risk analysis by assuming recreation will occur equally during wet or dry periods, but are not specifically addressed in terms of their actual short-lived effects, i.e., if primary recreation occurred during or shortly after the wet weather event.

It is believed that complete separation of the combined sewer system into sanitary and land drainage systems could greatly diminish the area-wide geometric mean fecal coliform density (i.e., no wet weather overflows). However, land drainage discharges also contain fecal coliforms (urban wash-off from green spaces and residential areas) and will have a lesser impact on receiving stream fecal coliform concentrations during rainfall events. Land drainage discharges

Estimated GI Cases for Winnipeg River Use



Estimated GI Cases for Selkirk River Use

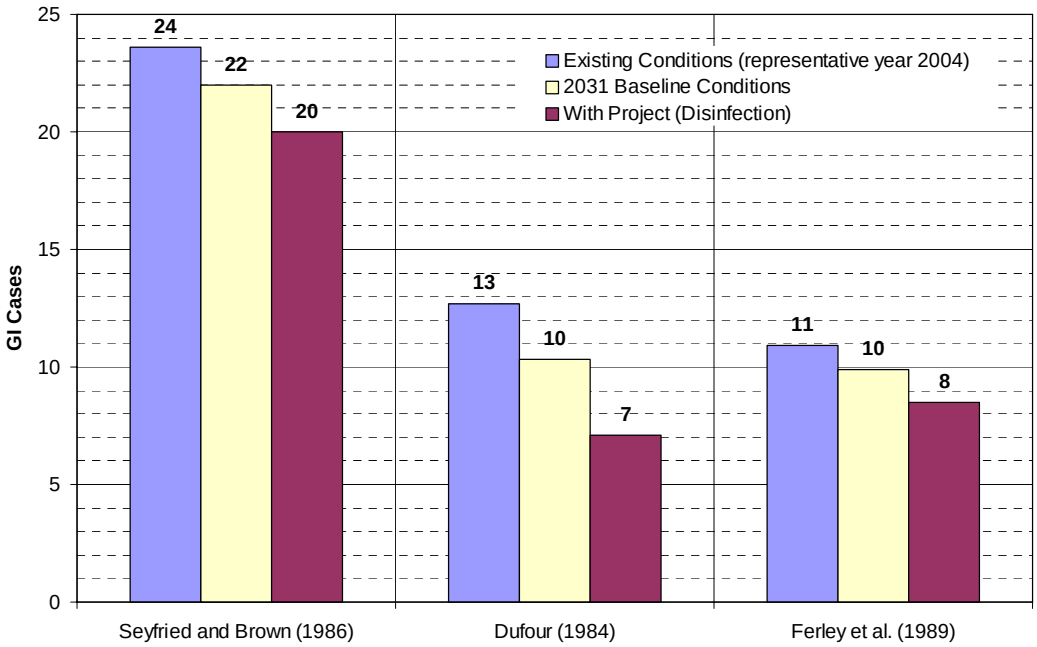


Figure 3-12

Total Estimated Gastrointestinal Case (GI) Load for Winnipeg and Selkirk

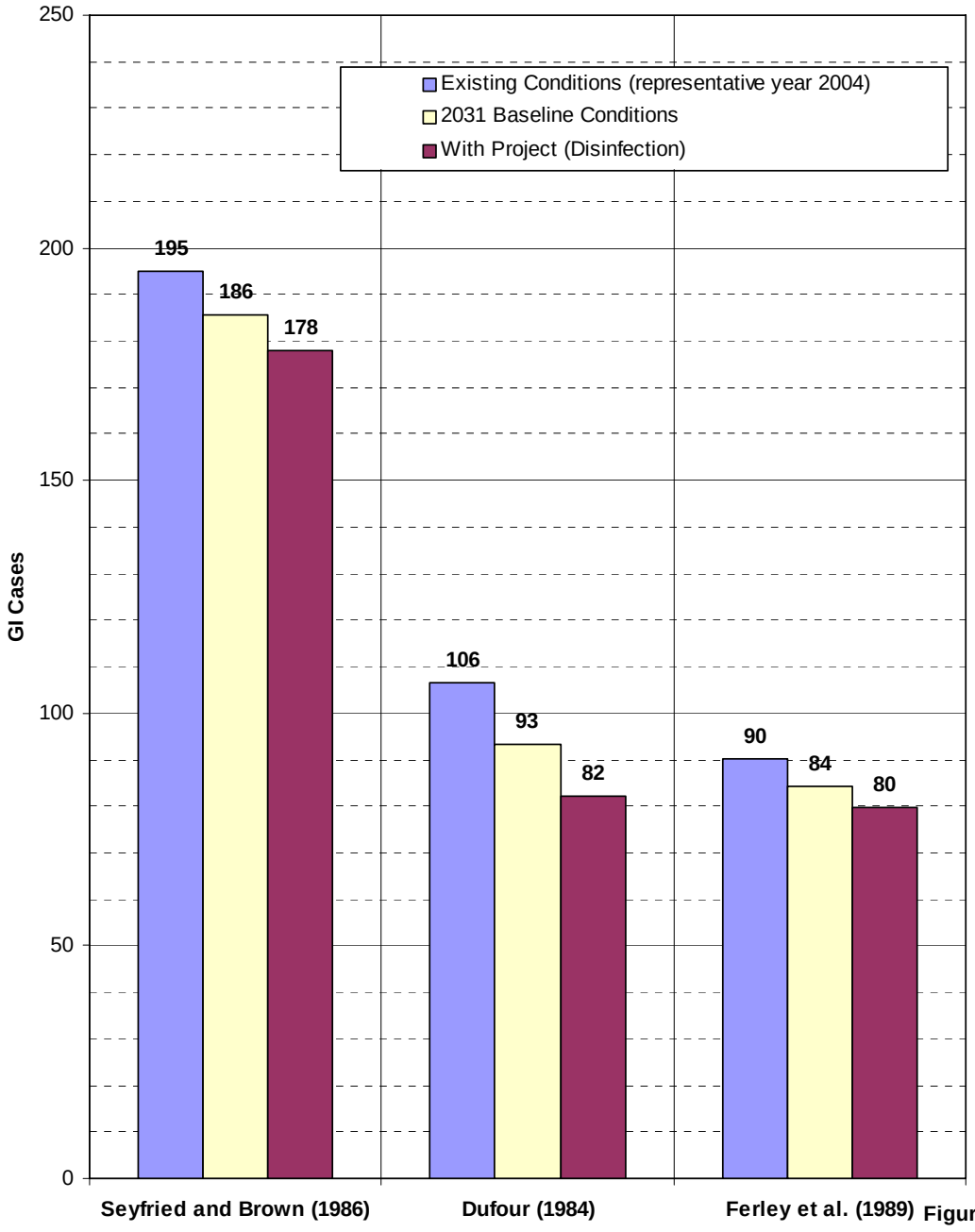


Figure 3-13

can cause localized wet weather spikes of fecal coliform concentrations that exceed MSWQO for 200 fc/100 mL. Accordingly, it may not be possible to achieve 100% compliance at all times within the study area.

An emerging control strategy as part of the CSO Management Study finds that the use of available storage in combined sewer districts that have been hydraulically relieved (to prevent basement flooding) has the potential to significantly reduce the frequency and volume of CSOs. This control option is called in-line storage and is currently under investigation. The currently available in-line storage was found to have the capacity to reduce the average number of overflows from about 20 to 10 overflows per recreation season and would also provide a commensurate benefit in reduced coliform levels and illness risk. The preliminary cost for this control option ranges from $60 to $100 million.

The addition of selective storage at strategic sites with pumping between districts along with WPCC upgrades could effectively reduce the number of overflows to about 4 per year at a cost of about $200 million. The cost to eliminate the remaining 4 overflows using this same approach escalates to from $500 to $800 million. Clearly, improvements to reduce these wet weather excursions are possible to varying degrees and associated costs. These value judgements and cost-benefit implications need to be carefully considered by the public in addressing the CSO issue.

5.0 ASSESSMENT OF RISK-REDUCTION STRATEGIES

The illness-risk rates associated with recreational use of the Red and Assiniboine Rivers are relatively low. Risk rates do rise as a result of urban discharges, but the increment in risk rate is relatively low. Of the urban discharges, the largest contribution to the increased risk rate is the treated effluent (un-disinfected) discharges from Winnipeg's three wastewater treatment plants.

Of the illness-risk-reduction strategies considered, the most effective is disinfection of treated effluent. Disinfection of NEWPCC final effluent has a significant impact only at Selkirk, with a

reduction in risk rate ranging between 3 to 6 GI cases/1,000 immersions. CSOs are the next biggest contributor. The changes in risk rate from urban discharges after disinfection of the WPCC effluents and elimination of CSOs (i.e., complete separation) could bring about a slight reduction in GI caseload. Complete separation is expected to have modest reduction at most locations in Winnipeg and negligible reduction in GI cases in Selkirk.

6.0 OBSERVATIONS ON PREDICTED ILLNESS RISK

A variety of observations flow from this exercise. They pertain to the current state of modelling for HRA, the character of the D-R models, the predicted magnitude of current risks and risk-avoidance benefits, the accuracy of model-based risk predictions, uncertainties about the comprehensiveness of the risks predicted from D-R models and, most importantly, whether engineered controls over bacterial and viral discharges to the Red and Assiniboine Rivers will have a measurable public-health benefit. These observations are as follows:

- Most D-R models useful for QRA continue to focus on GI:
  - however. a new basis for QRA for non-GI diseases (i.e., nose, ear, throat) has been established (e.g., Fleisher *et al.* 1996b).

- The river-use recreational disease caseload is driven more by the log-linear character of current D-R models and the relatively low rates of river use, than by pathogen densities in the river:
  - the measurable (i.e., reported) current GI caseload is estimated to be <1/year (Tetr*ES*/Wardrop 1997).

- There are more tools to apply for QRA than there are data to allow use of the tools:
  - few civic administrations monitor for the array of organisms for which D-R models have now been developed.

- Some potential public-illness risk exists even at low indicator (and pathogen) densities (i.e., at the lowest levels of pollution):
  - the estimated risk rate at the Manitoba objective of 200 fc/100 mL ranges from 10 (Dufour 1984) to 19 (Seyfried and Brown 1986) GI cases/1,000 immersions.
  - the risk rate upstream of Winnipeg ranges between 0.5 (Dufour 1984) to 13.5 (Seyfried and Brown 1986) GI cases/1,000 immersions.
  - this implies that risk reduction below these respective levels is likely impractical.

- Risks from specific pathogens may or may not be additive to each other, or to risks predicted by indicator D-R models:
  - insufficient knowledge exists about the independence of microbial behavior.

- Significant reductions in river-pathogen density as a result of engineered controls over wastewater and effluent discharges will cause only slight improvements in the theoretical risk rate:
  - the improvements in public health (i.e., reductions in GI caseload by means of avoided cases) from introduction of discharge-control measures will likely prove to be immeasurable.
  - recall that there are few reported GI cases attributable to recreational use of rivers, even where discharge-control measures are modest.

7.0 ILLNESS-RISK ASSESSMENT ACCURACY

The observation that investment of public capital (i.e., taxation dollars) in engineered controls over discharges to rivers will not likely result in measurable improvement in the health of river users may surprise some members of the public. The accuracy of the HRA exercise is therefore worthy of consideration, recalling the indications of what constitute “ideal” indicators of fecal pollution and what would be required of an “ideal” dose-response model (Section 3.1).

Consideration of the current state of HRA provides perspective on several key factors affecting model predictive accuracy:

- random distribution of pathogens in water
- ingestion volume
- uniform infectivity of ingested water
- water-quality sampling replication

A key fact emerging in the literature is that pathogens are not all randomly dispersed in water. Some micro-organisms, including pathogens, are clustered to some degree, even within small volumes. The "patchy distribution" that microbes display naturally in water is significant to determination of modelling accuracy. Because river-borne microbes clump together, or sorb to particles which clump together, or are embedded in (or clumped with) higher organisms (e.g., algae, rotifers, worms) (USEPA 1993), modelling assumptions about randomly distributed (uniform) dispersion in water can be invalid.

The ingestion rates assumed in most previous HRAs (e.g., 100 mL/immersion) have since been found to overstate the volumes observed empirically by the USEPA (1993) and others, i.e., only about 10-50 mL/"outing" (Phillipp *et al.* 1985). Accordingly, previous assumptions about ingestion volume likely contributed to overestimation of risk in prior HRAs.

The ingestion of small volumes of water (e.g., <50 mL/immersion) does not preclude ingestion of what constitutes an “infective dose”. The typical modelling assumption about uniform infectivity of ingested water can clearly be invalid, leading to predictive inaccuracy. Ingestion of even small doses during immersion in recreational water may result in a much higher proportion of an infective dose than originally thought. Gale (1996) points out that, by assuming pathogens are randomly dispersed, current models underestimate the risk from the less infectious agents (e.g., *C. parvum*), but overestimate the risk from more infectious pathogens (e.g., rotavirus). Indeed, some D-R models for bacteria are now regarded as less appropriate for estimating risks

from ingestion of highly infectious agents (e.g., virus) because of their tendency to overstate the risk from such organisms.

A key factor in predictive accuracy is the extent to which the epidemiological studies seeking to relate illness to recreational water quality and from which D-R models were derived (e.g., Cabelli *et al.* 1982; Seyfried *et al.* 1985b; Ferley *et al.* 1989), adequately controlled for measurement error in estimates of exposure. Failure to control for measurement error was found in a retrospective analysis by Fleisher (1990) who also statistically estimated the magnitude of the resulting bias. Fleisher found underestimation of the "true" relationships (i.e., risk rates) between indicator organism densities and swimmer morbidity varied from a minimum of 14% to a maximum of 57%.

On the basis of the foregoing, it can be seen that quantitative risk-assessment modelling can both ***over- and under-estimate*** risk. The net effect of individual sources of over- and under-estimation cannot be determined at present, but clearly offsetting occurs in each process of QRA and some balancing or 'cancelling out' must occur. Nevertheless, use of QRA must be cautious when formulating policy or regulation, or when being considered for their contributions to decision-making about capital investment.

This suggests the prudence of ensuring that non-quantifiable perspectives on illness risk also be considered in policy or regulation formulation, and in capital-project planning.

8.0 OBSERVATIONS AND CONCLUSIONS

The study has resulted in a number of observations and conclusions, which are presented in the categories of regulation of pathogens in surface water, sources of pathogens, the estimated risk from recreational use of surface waters, and the implications for control of urban discharges, specifically, CSOs in Winnipeg.

8.1 REGULATION

Most jurisdictions have objectives or guidelines for surface water quality parameters for the intent of protecting beneficial uses of the water. Manitoba Conservation has defined an objective of 200 fecal coliforms/100 mL for protecting primary recreation, consistent with most other jurisdictions.

It was confirmed that guidelines for protecting human health from recreational use of surface waters have a largely arbitrary origin. Their origin is based on protecting "natural" bathing beaches (not turbid rivers). The current standard of 200 fc/100 mL for protecting primary recreation has been rationalized by regulatory agencies:

- The criterion is relatively widely utilized and is considered "*adequate*", or "*practical.*"
- Current rationalizations of such use reflect the original U.S. PHS (i.e., 1960) doctrine of "*attainability*" (Tetr*ES*/Wardrop 1997).
- While some epidemiological studies support this numerical guideline, there is growing recognition of the weaknesses of such quality indicators and numerical values among regulators.
- Primary recreation in water meeting the fecal coliform objective does not imply a risk-free condition (the illness risk at 200 fc/100 mL is estimated to be about 9 to 19 GI illness cases/1,000 immersions, depending on the dose-response model used).

Like some other jurisdictions, Manitoba Conservation adopted an objective of 1,000 fecal coliforms/100 mL for secondary recreation in the previous MSWQO. No fecal coliform objectives are present in the current MWQSOG. There are no epidemiological studies that relate illness risk to secondary recreational use.

A transition to indicators other than fecal coliform (i.e., *E. coli*, enterococci) and involving other numerical objectives is now occurring on the basis that epidemiology shows them to be *better risk predictors*.

8.2 SOURCES OF PATHOGENS

Surface waters typically receive pathogens from a wide variety of sources, such as rural drainage, urban storm drainage, treated effluents from wastewater plants, CSOs, etc. Urban discharges typically increase concentrations of pathogens and indicator organisms in the surface waters. In the case of the Red and Assiniboine rivers, the wastewater plant effluents are the largest sources of indicator bacteria to the rivers. With disinfection of the plant effluents, indicator bacteria will be reduced but the resistant parasites, such as *Cryptosporidium* and, possibly, *Yersinia*, will likely still be present in the effluents. Upstream and zoonotic sources will continue to be important, thus urban-source control cannot preclude some residual degree of risk due to background levels from both rural and urban non-point sources.

8.3 RIVER USE AND EXPOSURE

The Red and Assiniboine rivers are very popular for passive enjoyment, active use of riverwalks, and secondary (non-contact) recreation (boating, fishing). The use of the shoreline and surface waters for primary recreation is limited (about 6,400 people/year electing to undertake these activities), in part due to flow and clarity constraints. Ingestion of river water during these activities is likely. Therefore, individuals choosing to engage in primary recreation have implicitly accepted any associated risks from exposure.

Secondary recreation is very popular (about 3,500 people/year), however, river quality is not a significant risk factor for such uses.

8.4 ILLNESS RISK FROM RIVER RECREATION

While it is known that some illness risk exists from the recreational use of surface waters, there is little reporting of such disease (<4% of total caseload). The principal documented risk remains GI illness, which is usually relatively mild and of short duration and not reported to the medical community, and diseases of the eye, ear, respiratory and skin.

For recreational use risk estimation, QRA is based almost exclusively on dose-response models for indicator organisms (e.g., fecal coliform, *E. coli*, Streptococcus) used to predict incidence of GI (or non-GI) infection. Indicator-based QRAs will not necessarily be predictive of risks from organism exposure to other pathogenic bacteria, viruses, protozoa or parasites.

Urban recreational risk remains of relatively low scientific interest. Pathogens in contaminated drinking water (e.g., *Giardia lambia*) and foodborne pathogens (e.g., *E. coli* 0157:H7) are more topical. Current research is not well suited for application to recreational risk assessment, given the available data.

For the Red and Assiniboine rivers, the estimated illness-risk rates for current ("baseline") conditions are shown below:

- upstream of the urban reaches of the rivers 1-13 cases GI/1,000 immersions (geometric-mean fecal coliform concentration of 20 cfu/100 mL)
- within the urban reaches 8-26 cases GI/1,000 immersions (geometric-mean fecal coliform concentrations of 10-1,100 cfu/100 mL)

For comparison, the "acceptable" risk rate, i.e., corresponding to the Manitoba Water Quality Objective for primary recreation of 200 fc/100 mL, is estimated to be about 9-19 cases GI/1,000 immersions (the estimates vary depending on dose-response model used).

The above risk rates translate to a predicted GI caseload arising from recreational use of the Red and Assiniboine rivers of about 100-200 cases/year for Winnipeg and Selkirk combined. For perspective, the background GI caseload for the Winnipeg population is in the range of 600,000-800,000 cases per year (Tetr*ES*/Wardrop 1997).

8.5 EFFECTS OF CONTROL OF URBAN DISCHARGES

The City is examining options to further control the effects of urban discharges on the water quality of the Red and Assiniboine rivers. Some of these options will reduce the levels of indicator bacteria and pathogens in the rivers and should provide some reductions in illness risk:

- Disinfection of the 3 Water Pollution Control Centre treated effluents provides some reduction in the recreation-risk rate in the river reaches immediately downstream of these facilities:
  - the benefit is estimated between 10 to 24 avoided cases in Winnipeg and Selkirk combined.

- Assuming disinfection of WPCC effluents, the subsequent separation of combined sewers in Winnipeg is expected to have little effect on the Winnipeg urban river recreational caseload, an estimate of three to seven cases.

The reduction in risk rate and overall gastroenteritis caseload from the UV disinfection option is clearly modest. The extent of river use influences the magnitude of the predicted caseload more than the concentration of the indicator bacteria, according to the typical dose-response models. If more extensive primary recreation use of the Red and Assiniboine rivers was to occur, the disease caseload arising from the additional exposures would likely increase, even with the better quality of the water.

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