7 September 2010

TO: Interested parties

FROM: HCDOP IAM marine science team
Jan Newton contact (newton@apl.washington.edu)

RE: Summer 2010 hypoxic conditions in Hood Canal

Following on the uncharacteristically low oxygen concentrations during winter 2009-early spring 2010, oxygen values in Hood Canal during summer 2010 continue to be among the lowest observed based on the data sets available. Of note, deep oxygen concentrations have been sustained below hypoxia (2 mg/L) at the Twanoh HCDOP-NANOOS buoy since June 2010 (Fig 1). This buoy has been collecting data since 2005, maintained by the University of Washington. Values are similar to those seen in 2005, with only 2006 lower. At the Hoodsport HCDOP-NANOOS buoy, oxygen has been low both at mid-depth (20-30 m) and especially at depth (80-100 m) (Fig 2). The deep low oxygen signal appears to be from relict waters that did not flush over last winter. These are the lowest concentrations seen at this site since the buoy's installation in 2005.

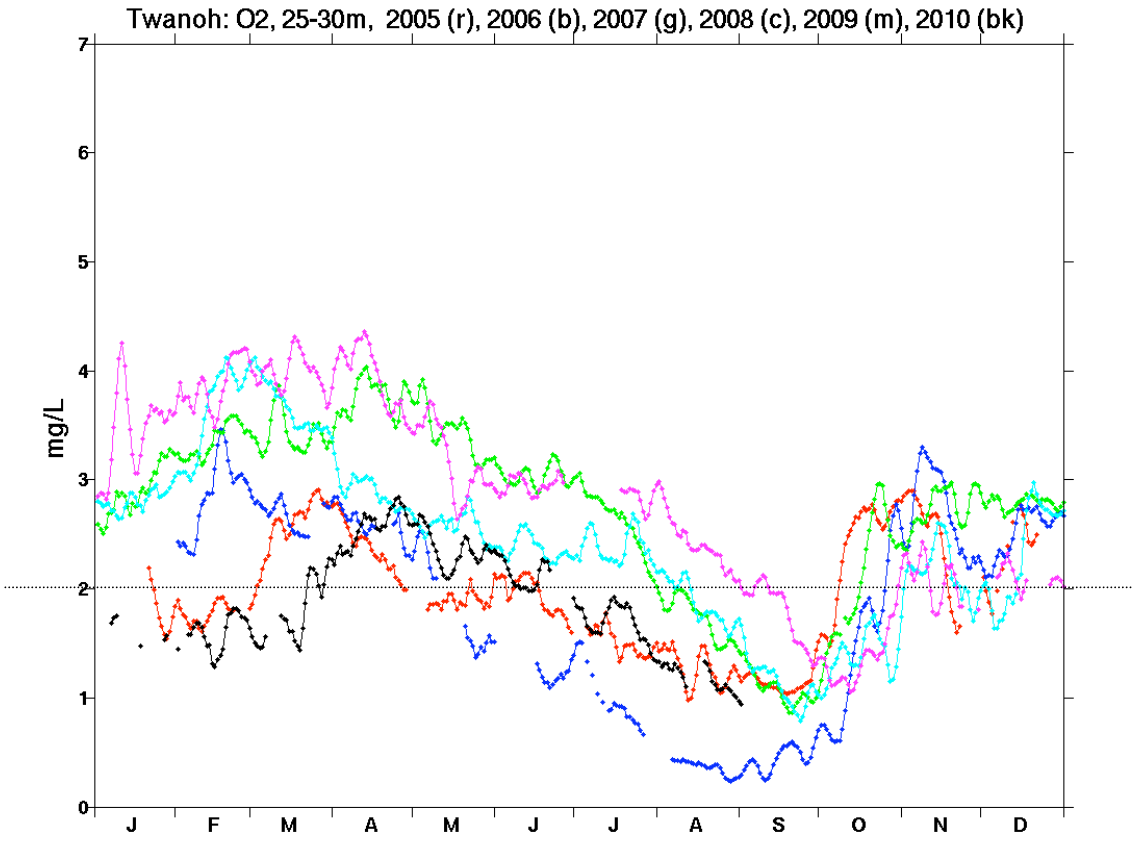

Fig. 1. Oxygen concentration at the HCDOP-NANOOS buoy at Twanoh in lower Hood Canal at 25-30 m depth for 2005 (red), 2006 (blue), 2007 (green), 2008 (cyan), 2009 (magenta), and 2010 (black). Data from the UW ORCA buoy through the HCDOP IAM science study and NANOOS.

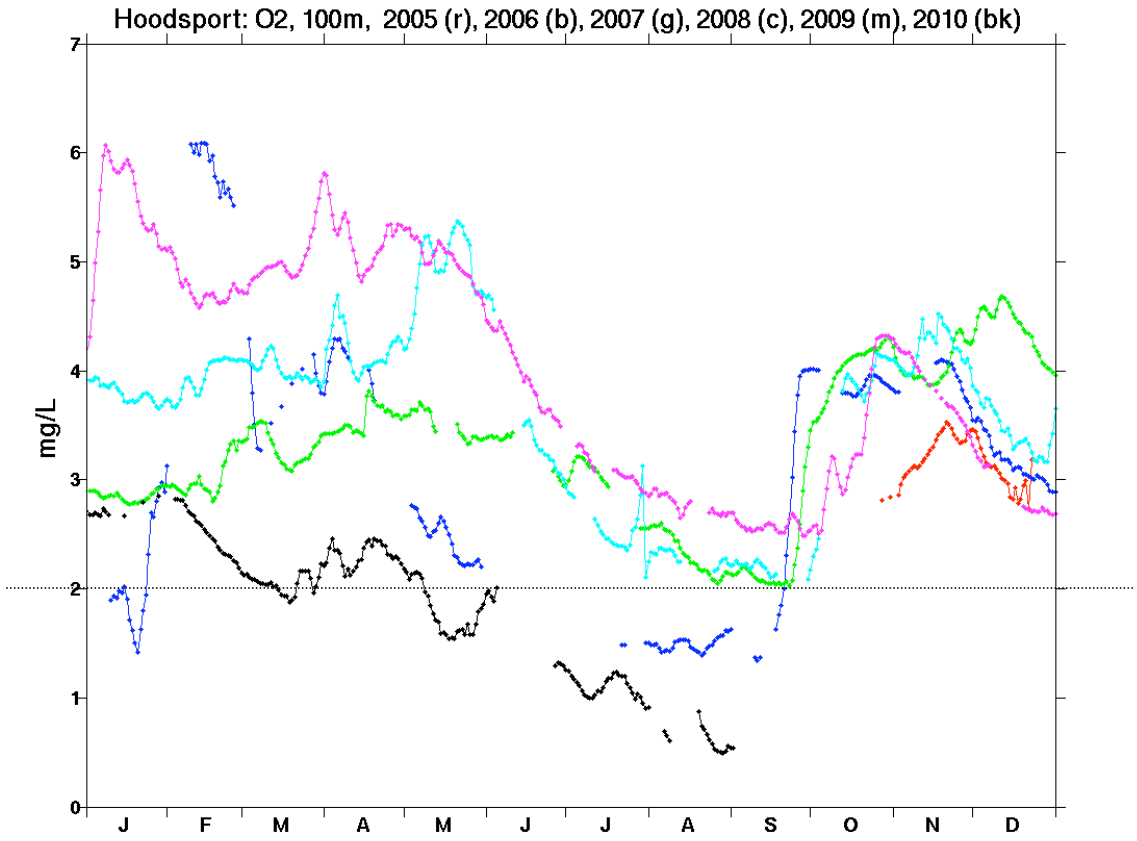

Fig. 2. Oxygen concentration at the HCDOP-NANOOS buoy at Hoodsport in Hood Canal at 100 m depth for 2005 (red), 2006 (blue), 2007 (green), 2008 (cyan), 2009 (magenta), and 2010 (black). Data from the UW ORCA buoy through the HCDOP IAM science study and NANOOS.

As stated in our previous posted note re winter-spring 2010 hypoxia (http://www.hoodcanal.washington.edu/documents/document.jsp?id=2454), the causative factors for the low winter oxygen concentrations in 2010 are likely related to mild winter weather and a weaker landward density gradient between the Pacific Ocean and Hood Canal.

As shown by the historical record, throughout the summer period (after June) typically oxygen levels decrease, due to respiration, until the fall intrusion (around September) which brings in higher oxygenated waters. While this process results in an increase of dissolved oxygen in the deep waters of Hood Canal, the HCDOP-IAM study has established that this also initiates two processes - both of which increase the risk of fish or biota mortality to low oxygen stress. First, the incoming denser oceanic waters displace deeper Hood Canal waters upwards. Currently these deeper Hood Canal waters are hypoxic. Second, the oceanic intrusion causes a sub-surface jet of low oxygen waters from the Lower Hood Canal (e.g., Great Bend to Belfair) region to "squirt" back outwards (seawards) as the incoming waters flush Lower Hood Canal. These hypoxic waters travel towards the Hoodsport region and the Sund Rock Marine Protected Area, key habitat for rockfish and other organisms.

The genesis of the 2006 fish kill involved both of these factors, which were stimulated by strong southerly winds that advected the surface waters near Hoodsport northwards, causing the subsurface hypoxic waters to rise. As previously reported by the HCDOP IAM study (see

http://www.hoodcanal.washington.edu/documents/document.jsp?id=1946), the dynamics of a fish kill are the combination of the severity of the subsurface oxygen minimum, its upward migration due to incoming denser seawater, and strong southerly winds. A widespread fish kill occurred in 2006 under these conditions; however, 2004 was a year with comparable low inventory oxygen values (pre-dates the buoys) that did not have a major fish kill nor all three of these factors.

Whether these same factors will occur this year cannot be predicted. We will monitor and report on progression of the oceanic intrusion, but winds cannot be predicted that far in advance. Currently, subsurface oxygen concentrations are low enough (<2mg/L), but are deep, around 10 m (Fig 3). Both southerly winds and strong oceanic intrusion can cause this horizon where oxygen rapidly declines (the oxy-cline) to rise. As little as one week ago this feature was around 20 m.

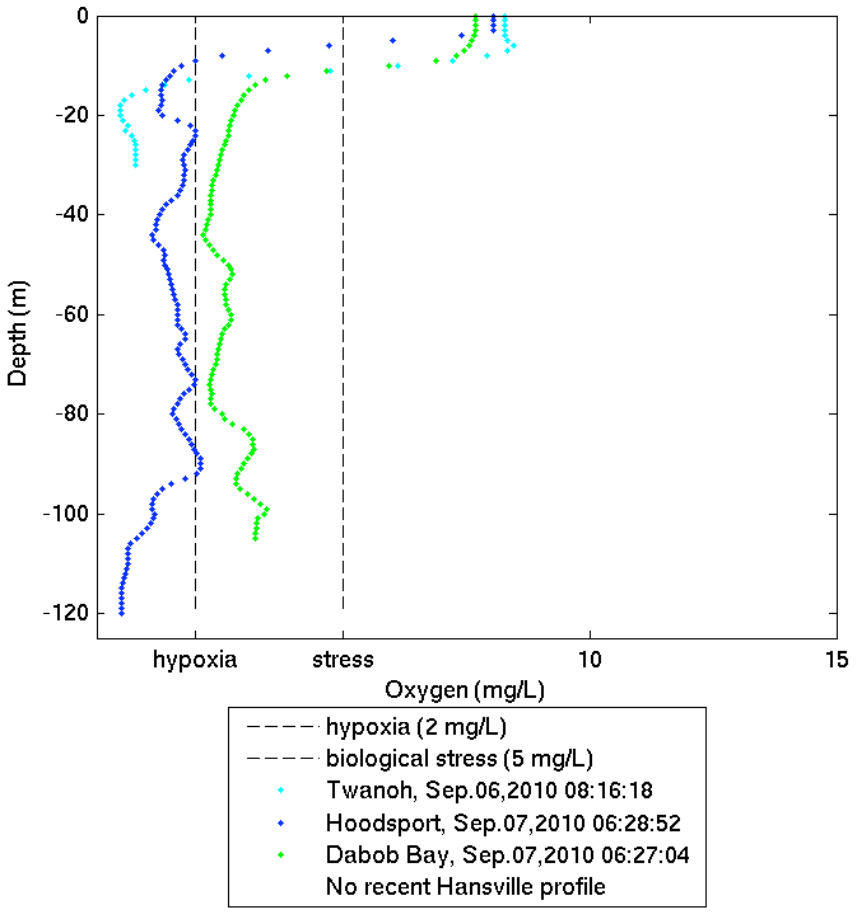

Fig. 3. Latest depth profiles of oxygen from the ORCA buoys in Hood Canal.

What is clearly evident is that 2010 is a particularly low oxygen year, even compared with historical records. Data collected at historical UW stations that are currently routinely occupied by the HCDOP Citizen Monitoring Program, run by the Hood Canal Salmon Enhancement Group and UW-PRISM, show that the average oxygen concentration below 20 m in Hood Canal

from the entrance to Dabob Bay to the Great Bend has reached values below 3 mg/L (Fig 4). The summer 2010 oxygen values are the lowest or among the lowest seen in the records available for the months of June and July for years spanning 1952-1966 and 1998-2010, with the August 2010 data point the lowest on record.

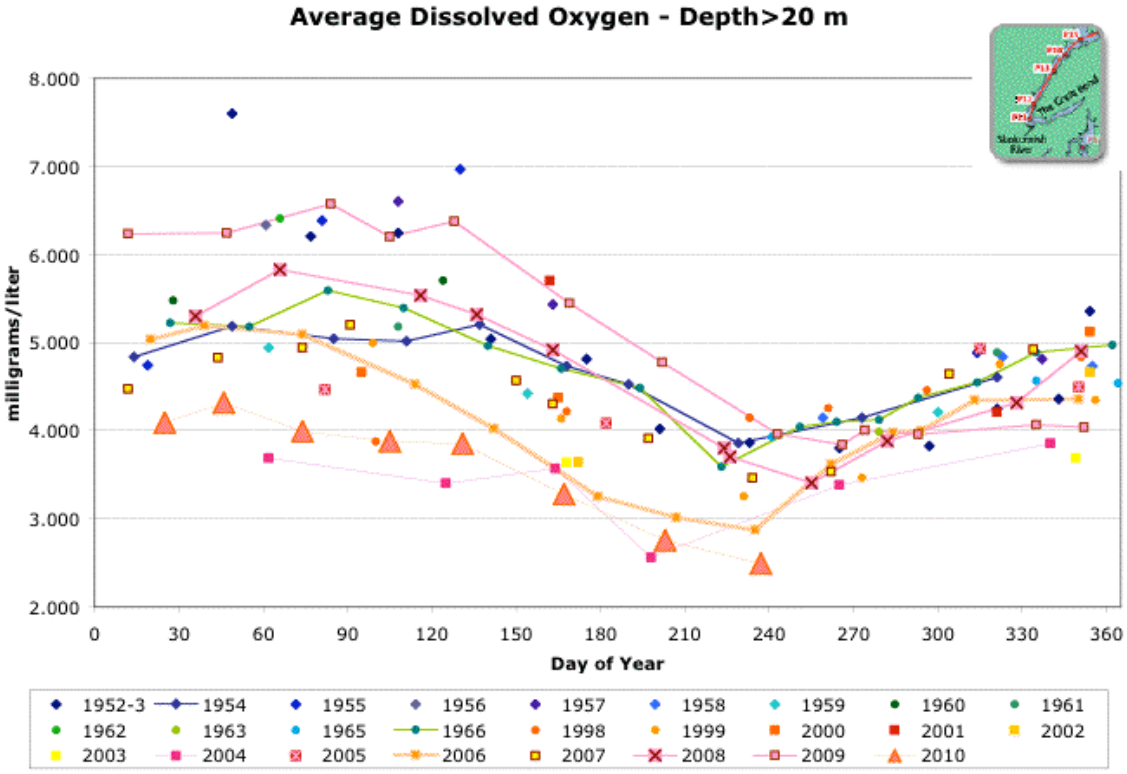

Fig. 4. Average dissolved oxygen concentration in the water below 20 meters depth in the region between Dabob Bay and the Great Bend plotted versus the day of the year. Data from UW (historical) and the HCDOP IAM science study Citizen Monitoring Program (current data).

Cross-section plots of oxygen data collected by the HCDOP IAM Citizen Monitoring Program show the regional extent of this condition; widespread low oxygen concentrations persist in the bottom 50 m throughout spring-summer 2010 (Fig. 5). Concentrations at depth near Hoodsport are particularly low, approaching anoxia.

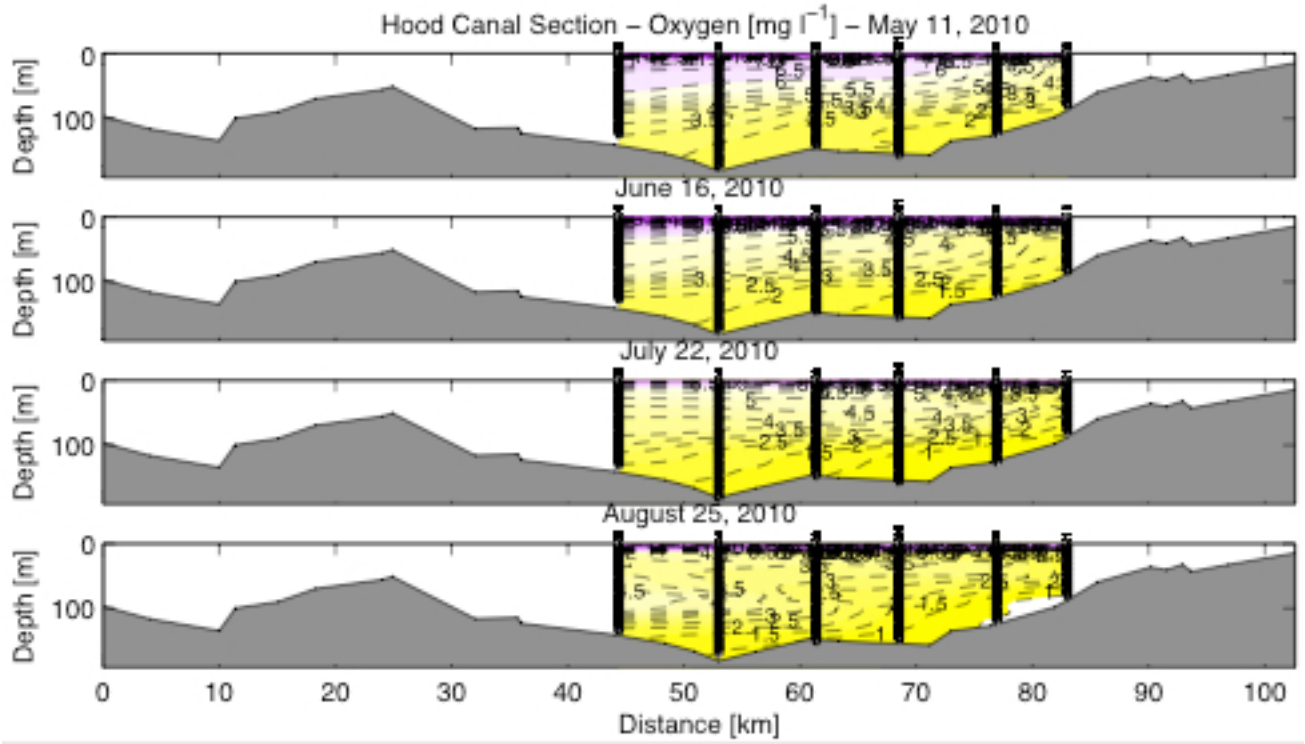

Fig. 5. Sections of oxygen concentration (mg/L) with depth versus horizontal distance between Dabob Bay and the Great Bend for May through August 2010. Data from the HCDOP Citizen Monitoring Program of the HCDOP IAM science study.

To access these and other data from Hood Canal, please see:
HCDOP website: http://www.hoodcanal.washington.edu/ or
NANOOS website: http://www.nanoos.org/nvs/nvs.php?path=NVS-Assets

The Hood Canal ORCA buoy and Citizen Monitoring data are part of and served by the Northwest Association of Networked Ocean Observing Systems (NANOOS). The are available for visualization and download there.

For more information, contact Dr. Jan Newton at newton@apl.washington.edu