**The dataset represents data from the study by Perez-Protto et al. “*Deceased Donor Hyperglycemia and Liver Graft Dysfunction*”. *Prog Trans* 2014; 24(1): 106-112.**

**Dataset: Donor Hyperglycemia**

Optimal donor management is thought to improve the quality of transplanted organs. Deceased donor management is complicated by physiological changes that potentially compromise organ function and survival after transplant. Hyperglycemia is one of the most common derangements found in organ donors; however, no evidence-based guidelines have been established for glucose management in organ donors.

In critically ill patients, hyperglycemia increases risk for multiple adverse outcomes, including sepsis, acute renal failure, hyperbilirubinemia, and mortality. Tight glycemic control appears to improve outcomes in critical care patients under some circumstances. The benefit of the tight glycemic control may have been due to a decrease in glucose variability. For example, rapid fluctuations in blood glucose levels increase oxidative stress, provoke endothelial dysfunction and vascular damage, and augment apoptosis.

Whether donors’ glucose level or variability contributes to liver graft function remains unknown. A putative association between donor hyperglycemia and/or donor glucose variability and liver graft dysfunction would be of considerable clinical importance because—unlike so many other factors related to donors—glucose level could be tightly managed if doing so improved outcomes. The

primary aim of this study was thus to determine whether hyperglycemia in deceased liver donors, as defined by the time-weighted average (TWA) of donor glucose measurements, is associated with graft dysfunction after deceased orthotopic liver transplant. Secondarily, we assessed whether variability in donors’ glucose level, defined as glucose measurement range and standard deviation, is associated with graft dysfunction.

Data on donors, grafts, and recipients were collected for 591 liver transplants between January 2005 and October 2010 at the Cleveland Clinic. Excluded were grafts from living donors, donors after cardiac death, and transplants for which the donor’s glucose level was measured fewer than 2 times. The primary feature of interest was the TWA of donor glucose measurements; secondary features were the range and the standard deviation of donors’ glucose measurements. Graft dysfunction, the outcome, was defined as (1) primary nonfunction as indicated by death or retransplant during the first postoperative week or (2) liver graft dysfunction as indicated by an aspartate aminotransferase level greater than 2000 U/L any time between postoperative days 2 and 7 or a prothrombin time greater than 16 seconds any time between postoperative days 2 and 7.

To assess any relationships between characteristics of donors, grafts, and recipients and the TWAs of donor glucose measurements, TWA of donor glucose measurements was partitioned into 4 groups on the basis of the observed quartiles of their overall distribution. To estimate the relationship between the TWA of donor glucose measurements and the probability of liver graft dysfunction, TWA was log2 transformed. This parameterization of the model means that a relative increase of 1 on the log2 scale corresponds to a doubling on the original scale. Hence these results are reported as “relative doubling”. This was similarly conducted for the glucose range and standard deviation.