LifeLabs Medical Laboratory Services

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Physicians' Newsletter
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Non-fasting Lipids

There are arguments in the literature both for and against the use of fasting samples for lipid levels. Some groups suggest non-fasting specimens may actually be better at predicting cardiac risk and making therapeutic decisions, while others have provided reasons why fasting levels are still needed. Still, the majority of clinical guidelines and intervention trials for lipids, completed with non- and diabetic populations, are based on fasting samples. Trials of statin therapies have used fasting lipids for the most part but two major studies (MRC/BHF and SEARCH) have published outcomes using non-fasting specimens.

The 2012 Canadian Cardiovascular Society (CCS) Guidelines for the Diagnosis and Treatment of Patients with Dyslipidemia classify patients and treatment goals based on risk category and lipid measurements. Treatment approach and optimal ranges are defined for each cardiovascular disease (CVD) category based on LDL-Cholesterol (LDL-C), non-HDL-Cholesterol (non HDL-C; *i.e.* TC less HDL-C) and apolipoprotein B (Apo B) concentrations. LDL-C is calculated by the Friedewald equation, which includes measured triglycerides, total cholesterol (TC) and HDL-C. Food intake influences measured triglycerides and therefore calculated LDL-C. Note that the TC/HDL-C is no longer included in the guidelines as a treatment target although it is still recognized as a significant risk indicator if >6.0.[1]

The CCS 2012 guidelines endorse non-HDL-C as a new lipid marker which reflects cholesterol transported within atherogenic particles. Non-HDL-C is unaffected by fasting status, lending support to the use of non-fasting specimens. The 2014 BC Guideline on Cardiovascular Disease Prevention uses an updated panel for follow-up of treated hypercholesterolemia that includes TC, HDL-C, and non HDL-C and does not require fasting[2]. This panel was introduced on the BC Standard Outpatient Lab Requisition in June 2014.

A large community-based population study in 2012 found differences between fasting and non-fasting measurements to be <20% for triglycerides and <10% for LDL-C. Triglycerides increased at 2-6 hours post-meal and then declined to baseline, but these differences were not statistically significant; similarly, calculated LDL-C decreased up to 3 hours post-meal but the differences did not achieve statistical significance from fasting values. The authors illustrated that the changes in TC and HDL-C for fasting *vs* non-fasting specimens were only about 2%: indeed, TC and HDL-C results obtained 1 hour post-meal were not statistically different from values at 8-12 hours[3] These data reflect an earlier study by Langsted *et al*.[4]

Epidemiological data illustrate that non-fasting data may be a more significant predictor of CVD, independent of post-prandial time[5]. Since patients spend most of their day in a post-prandial state, use of non-fasting lipids has been determined by most to be acceptable for initial assessment.

CVD Risk Calculation – since the Framingham CVD risk calculations are based on TC and HDL-C, the small change in fasting *vs* non-fasting values will have minimal impact on the risk score.

CVD prognosis – after adjustment for risk factors in a study of 2809 males, Eberly reported that non-fasting and fasting triglycerides yielded very similar hazard ratios for 25 year mortality and for 8 year fatal or non-fatal CHD.[6]

Triglycerides and LDL-C: non-fasting triglycerides may be up to 0.2 mmol/L higher than in fasting state, leading to a lower calculated LDL-C[7]. This discrepancy for increases with triglycerides >2.08 mmol/L[8].

Non-fasting Lipids (cont'd)

Diabetics: while a small study in 2011 suggested that a significant fraction of diabetic patients would be misclassified at decreased CVD risk through the use of non-fasting collections[9], a subsequent study of 1337 diabetics over 8 years found no significant effect on CVD risk assessment in these patients[10].

Continued use of fasting specimens should be considered when assessing those at high CVD risk, initiating or altering drug therapy, or when non-fasting triglycerides exceed 2.0 mmol/L.

1) Anderson TJ *et al.*, *Can. J. Cardiol.* **2013**, *29*, 151.
2) www.bcguidelines.ca/pdf/cvd.pdf
3) Sidhu D and Naugler C, *Arch. Intern. Med.* **2012**,*172(22)*,1707.
4) Langsted A *et al.*, *Circulation* **2008**, *118*, 2047.
5) Mora S *et al.*, *Circulation* **2008**, *118(10)*, 993.
6) Eberly LE *et al.*, *Arch. Int. Med.* **2003**, *163*, 1077.
7) Langsted A and Nordestgaard BG, *Clin. Chem.* **2008**, *57(3)*, 482.
8) Lund SS *et al.*, *Clin. Chem.* **2011**, *57(2)*, 298.
9) Lund SS and Jensen T, *Clin. Chem.* **2011**, *57(9),* 1336.
10) Van Dieren *et al.*, *Diabetologia* **2011**, *54*, 73.

Sheila Boss, PhD, FCACB (LifeLabs ON)
adapted for BC by *Jan Palaty, PhD, FCACB*
and *Cheryl Tomalty, PhD, FCACB*

Chemistry Specimen Retention

LifeLabs will be moving to a policy of retaining routine Chemistry specimens for 3 days after the specimen is collected. There are no changes expected for Hematology or Microbiology specimens. Retention times for specialized tests (*e.g.* infectious disease serology, urine drugs of abuse) will remain unchanged.

At present, Chemistry specimen retention varies from 4 to 7 days, depending on the facility. There are approximately 200 physician calls per day to add on tests to previously collected chemistry specimens, 95% of which occur within 4 calendar days of specimen collection. By moving to a shorter standardized storage period, we hope to achieve greater consistency between the various LifeLabs testing sites as well as reducing errors due to sample instability.

Dr. Ayesha Vawda, our new Hematopathologist

Originally from South Africa, Dr. Ayesha Vawda completed a BSc in Biochemistry and Microbiology at the University of Victoria in 2005 before obtaining her medical degree from the University of BC in 2009. She served as chief resident in Hematopathology from 2012-2013 and obtained her FRCPC certification in 2014.

Dr. Vawda has a particular interest in lymphoma, flow cytometry, lymph node morphology, and molecular diagnostics. She recently undertook research into the role of the lymph node microenvironment in lymphoma pathogenesis. Dr. Vawda's awards include the Edythe Hembroff-Schleicher Scholarship, the Rowan Scholarship in Medicine, and Research Awards from NSERC and the BC Cancer Foundation.

Editor: Jan Palaty, PhD, FCACB