

RESULTS: DRIED BLOOD SPOT TEST

Accession #: 100065929 • Patient: JANE SMITH

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Patient: JANE SMITH

Tel: (123) 456-789

Email: test@test.com

Sex: Female

Age: 29 yr

Date of Birth: 1996-06-26

Accession #: 100065929

Sample received: 2026-09-15

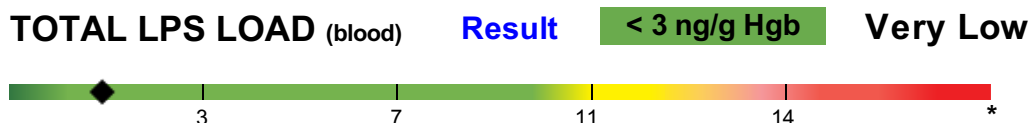
Report issued: 2026-09-26

Health Care Professional: John Smith

Address: 400 March Road #420, Ottawa, Ontario K2K 3H4

Sample collection: 2025-07-22

TOTAL LIPOPOLYSACCHARIDES LOAD



*The reference range represents a quintile distribution, each quintile being 20 % of a population determined from archival data.

These test(s) were performed by Rhein Consulting Laboratories (RCL); F.J. Nordt, Ph.D., Director, 4475 SW Scholls Ferry Road, Suite 101, Portland, OR 97225, USA. CLIA #38D0676504. The test(s) were developed and their performance characteristics were determined by RCL and have not been cleared or approved by the US FDA nor are they intended to diagnose or treat any disease(s).

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[Convert to pdf, Save or PRINT >>](#)**TOTAL LIPOPOLYSACCHARIDES LOAD****General Commentary**

Lipopolysaccharide (LPS), is a component of the outer membrane of gram-negative bacteria. When confined to the gut, LPS plays a role in gram-negative bacterial integrity. When it crosses the intestinal barrier, moves through the portal circulation to the liver, and then enters systemic circulation, it can become a potent endotoxin. Its presence in the blood, especially in its active micellar form, can trigger widespread inflammation, contribute to immune dysregulation, and ultimately lead to conditions like insulin resistance, cardiovascular disease, and even sepsis.

In blood there are only minute quantities of LPS in a free, dissociated form. The vast majority is immediately bound to proteins. By far the most important protein binder is the high-density lipoprotein (HDL). Well known as the 'good cholesterol', HDL does more than help remove excess cholesterol from the bloodstream and arterial walls and carry it back to the liver, for removal from the body. One could argue that it plays an even more important role as the principle binder of free LPS in the blood, being responsible for > 90% of its binding and transport to the liver¹. The remainder of the LPS is captured by the LPS Binding Protein (LBP), whose major function is to transport LPS to cells of the immune system, such as the macrophage^{2,3}.

Free LPS does exist in blood cells. This intracellular reservoir of LPS exists largely in micellar form, the lipid-carbohydrate clusters that represent the biologically active, inflammation-triggering configuration of LPS. When cells die, or undergo autophagy (cellular recycling), these micelles are released into circulation, often triggering a cascade of immune activation. For individuals with chronic gut dysbiosis, intestinal permeability or metabolic dysfunction, this constant trickle of LPS may help explain unresolved symptoms, such as fatigue, and other pathology related to inflammation.

The FLUIDS iQ Dysbiosis – Total LPS Load Biomarker

The FLUIDS iQ' Dysbiosis – Total LPS Load biomarker Pat Pend measures the total LPS content in a dried blood spot extract (DBSE), and is a powerful marker of chronic inflammation and endotoxemia. The use of a DBSE as the testing modality permits the capture of LPS levels stored inside blood cells, and gives practitioners a radically clearer view of the body's true inflammatory burden.

The DBSE test takes advantage of the fact that cells undergo apoptosis (self-destruction) when dried, releasing their intracellular contents. Once the contents are extracted and solubilized, it allows for the assay to measure the total LPS 'load' (quantity) stored within cells: This is something serum-based methods are unable to provide. With this biomarker, the findings show that LPS levels are 1 to 2 orders of magnitude (10x to 100x) greater than previously thought to exist and reported, fundamentally shifting our understanding of how much LPS can actually accumulate in the body.

Increased levels of total LPS lead to an increased risk of LPS, in its free state in blood, acting as an endotoxin, with all of the downstream ramifications. These include heightened inflammation, as well as the downspiral to sepsis, septic shock and even death.

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[Convert to pdf, Save or PRINT >>](#)**Reading the FLUIDS iQ' Dysbiosis – Total LPS Load Biomarker** Pat Pend**Total LPS Load:**

The values for the total amount of LPS move from 'Very Low' in Quintile Distribution (QD)-1, on the extreme left of the bar - through 'Low, Medium, High' from QD-2 to 4, and 'Very High' in QD-5, on the extreme right of the bar.

'Total LPS Load' represents the total amount of LPS, after the lab procedure that causes dissociation of LPS aggregates to the free, 'dissociated' state. This dramatically increases the total amount of detected LPS.

'Total LPS Load' doesn't just detect the levels of total LPS, but also helps in explaining its relation to inflammation.

When used alongside FLUIDS iQ's 'Intestinal iQ' Mucosal Barrier Assessment (MBA) functional lab test, practitioners can identify whether intestinal permeability is contributing to systemic endotoxemia. This clarity allows for highly targeted healing strategies, especially in individuals struggling with some, or all, of the following issues: Chronic fatigue and brain fog, histamine intolerance and high diamine oxidase, cardiometabolic concerns, persistent gastrointestinal inflammation, as well as autoimmune activity and immune dysregulation.

One of the biggest clinical advantages of pairing the LPS Marker with the other analytes in the 'Intestinal iQ' profile - Zonulin, Histamine and Diamine Oxidase - is the ability to determine whether inflammation is driven mostly by paracellular transport (via elevated Zonulin) or a combination of para and transcellular mechanisms - something that Zonulin testing alone cannot reveal. It requires the LPS marker. With this information, practitioners can prioritize gut healing protocols more effectively, reducing "trial and error," and optimizing the timing of interventions like probiotics or antimicrobials.

Over the last decade, North America has witnessed a sharp rise in obesity, metabolic syndrome, and hospital admissions tied to systemic inflammation. Much of this is rooted in gut dysbiosis and high dietary levels of ultra-processed foods, that disrupt microbial balance and compromise the gut barrier. Notably, interventions like fasting, or autophagy (cellular recycling), often thought of as anti-inflammatory, may exacerbate LPS issues if not timed correctly. During autophagy, overloaded immune cells can rupture, releasing stored LPS into the circulation and triggering an acute inflammatory response. This underscores the importance of testing and personalizing interventions, rather than just applying general protocols.

For years, LPS has been a hidden player in chronic disease. It is a toxin known to science, but difficult to measure clinically. With the FLUIDS iQ' Dysbiosis – Total LPS Load Pat Pend, that barrier has been breached. Practitioners now have a tool that not only validates the gut-inflammation connection, but also helps personalize healing protocols with new precision. One where chronic inflammation is no longer vague or invisible - but measurable, actionable, and finally addressable at its root cause.

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1. . Murch O et al. Intensive Care Med 2007; 33:13–24.; 2. Mousa H et al. Biochemistry and Biophysics Reports 2023; 34: 101445.; 3. Tobias PS, Ulevich RJ. Immunobiology 1993; 187: 227-232.;



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