FLUIDS iQ®

RESULTS: DRIED BLOOD SPOT TEST

Accession #: 100035180 • Patient: Jane Smith

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Patient: Jane Smith
Sex: Female **Age:** 20 yr **Date of Birth:** 1998-07-14
Health Care Professional: John Smith
Address: 646 Petrolia Rd., Toronto, Ontario M3G 2W3

Accession #: 100035180
Sample received: 2019-03-07
Report issued: 2019-03-07
Sample collection: 2019-03-05

CANDIDA SUITE (IgM, IgG, IgA)

Analyte	Result (U/ml)	Reaction	Reference Range* (U/ml) Non Reactive	Indeterminant	Reactive
Candida IgM	15	Reactive	< 9	9 - 11	> 11
Candida IgG	8.13	Non Reactive	< 9	9 - 11	> 11
Candida IgA	12	Reactive	< 9	9 - 11	> 11

*Reference range derived from a normal distribution of results, encompassing 95% of a randomly selected population

The comments provided here are for educational and research purposes only. These analytical results, on their own, should not be interpreted as being diagnostic or treatment recommendations. They must be correlated to clinical observations and diagnostic tests. Decisions are the responsibility of the health care professional.

Definitions

Non Reactive: Values are considered Non Reactive when no significant level of the Candida albicans antibody has been detected.
Indeterminant: Values are considered Indeterminant, or borderline, due to cross reactivity with other Candida species that may have pathological potential. The level of the antibody does not permit a clear description or classification of the reactivity within the range of the assay. A follow-up test within 2 to 4 weeks may be helpful in that determination[8].
Reactive: Values within the Reactive Range show that Candida albicans is detected, and may indicate a past, active or prolonged infection, depending on the level of the antibody. Significantly elevated levels of antibody have been observed in patients with active infection.

IN THE PRESENT TEST

Reactive IgM antibody levels appear early in the course of an infection overgrowth[5] and will usually reappear with renewed exposure, but to a lesser extent.
Reactive IgA antibody levels may suggest that there is mucosal overgrowth. This overgrowth is most often situated in the digestive tract, but can also occur in the oral, nasal, urinary and respiratory tracts[8].

Dr. Aron Gonshor PhD, DDS, FRCD(C), FAO • Laboratory Director

GENERAL COMMENTARY

Candida albicans is a yeast that is normally found in small amounts in the body, and is the only fungal species belonging to the 'normal' microflora. Candidiasis is a fungal infection caused by yeasts that belong to the genus Candida, with Candida albicans found in over 80% of fungal isolates[1]. Due to colonization of mucous membranes with Candida albicans, and its passage into the host's blood stream, the humoral immune system is stimulated, which results in the production of antibodies against Candida albicans[2].

In Candidiasis, Candida albicans can infect the skin and the mucous membranes of the genitals, mouth, respiratory tract and digestive tract. These skin and mucous membrane infections may be exacerbated by factors such as pregnancy, diabetes mellitus, immune deficiency and therapies with cytostatic drugs or antibiotics. Infections of organs like the lungs may cause death in immune suppressed patients with a cellular immune deficiency[1].

This FLUIDS iQ® Candida Screening Test looks for 3 antibody types; IgM, IgG and IgA.

a. IgM antibodies are the first isotypes formed after a primary exposure to the Candida antigen, reflecting a present infection. Typically, these antibodies develop as the predominant immunoglobulins early in the course of an infection and then decrease in number over a short period of time, measured in days. Infections of the bloodstream have serious consequences unless controlled quickly and the rapid production of IgM, together with its efficient activation of the complement system, are important initiators of that control. This complement activation assists the phagocytic system to eliminate antigens from the intravascular space[9]. Upon reinfection, IgM antibody levels may often not be as elevated as in the earlier infections.

b. IgG antibodies are the most predominant isotype formed from secondary exposure to the Candida antigen, and reflect a past or more prolonged ongoing infection[2]. They are produced in increasing numbers as the IgM antibody levels decrease after the primary exposure. IgG antibodies are smaller in size than IgM and diffuse easily out of the blood into the tissues. They activate complement, and assist the phagocytic system in eliminating the antigen from the extravascular spaces[9].The IgG antibodies represent the largest class of human immunoglobulins and are evenly distributed throughout both intra and extravascular fluids. Specific IgG antibodies may remain for many years after an infection has been eliminated.

c. IgA antibodies represent only 15-20% of human serum immunoglobulins. However, they are, by far, the most predominant antibody class found in seromucous secretions[3], playing an important role in the local mucosal immune responses that occur in the digestive, respiratory and vaginal tracts, as well as in saliva and tears[4]. High levels of serum IgA antibodies are thought to be associated with mucosal, epithelial, tracheobronchial, and genito-urinary candida infections.

IgA is less potent than IgG as an opsonin; that is, it is less able to bind to foreign microorganisms, so as to make them more susceptible to phagocytosis. Also, unlike IgG, IgA is a weak activator of complement. This distinction is not surprising, since IgG functions mainly in the body tissues, where accessory cells and molecules are available, whereas IgA functions mainly on epithelial and mucosal surface environments, where complement and phagocytes are not normally present. IgA therefore functions chiefly as a neutralizing antibody[9].

Candida References: **1.** Gunther LS et al. 2014 *Sao Paulo Med J*; 132:116–120; **2.** Duggan S et al. *Virulence* 2015; 6: 316-326; **3.** Macpherson AJ, Slack E. *Current Opinion in Gastroenterology* 2007; 23: 673–678; **4.** Burns CA et al. *Infection and Immunity* 1982; 36: 1019-1022; **5.** Bassetti M et al. *J J Antimicrob Chemother* 2018; 73, Suppl 1: i14–i25; **6.** Stanley PJ et al. *Clin. exp. Immunol* 1984; 58: 703-708; **7.** Gonzalez-Quintela A et al. *Clinical and Experimental Immunology* 2007; 151: 42–50; **8.** Fagarasan S, Honjo T. *Nature Reviews Immunology* 2003; 3; 63–72. **9.** Janeway CA. *Immunobiology: The Immune System in Health and Disease*. 5th edition - Garland Science, 2001.