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# WHAT DOES SBI BIND TO?

## *OVERVIEW*

The health and performance of the gut is influenced by diet, genetics, environment, stress, intestinal barrier, immune status, and the microbiome. Altering any of these can lead to enteropathy, which is an increase in intestinal permeability, impairment of gut immune function, and nutrient malabsorption[1].

Serum-derived bovine immunoglobulin/protein isolate (SBI) is a plasma fraction rich with immunoglobulins. SBI reduces inflammation in the gastrointestinal tract by immune and steric exclusion mechanisms resulting from oral immunoglobulins binding proinflammatory antigens.

Gastrointestinal tract imbalances can impact the intestinal barrier function, allowing pathogens to translocate and induce circulating immune regulators. This leads to a cyclical relationship of increased inflammation and aberrant cytokine production[2]. That loop of gut inflammation can ultimately cause chronic disease states such as IBS-D, IBD, or HIV-associated enteropathy.

Employing a modified ELISA setup, this study expanded the list of antigens to which SBI can bind and therefore the situations in which SBI may reduce inflammation.

## *GOAL*

The goal of this study was to expand the list of antigens to which SBI bind by adapting the ELISA used by Detzel et al[3] to demonstrate binding of IgG to lipopolysaccharide, lipid A, and Pam3CSK4 antigens.

## *DISCUSSION*

The IgGs contained in SBI specifically bind C. albicans lysate and Als3 protein, CDT subunits A and C, Shiga-like toxin 1 subunit B, and H. pylori CagA protein as determined by ELISA.

## *CONCLUSION*

The immunoglobulin content of SBI has previously been shown to bind to a variety of microbial antigens (e.g., LPS, flagellin, peptidoglycan, etc) associated with gastrointestinal disorders. To continue to understand the broad impact of SBI, new relevant antigens of interest were chosen to test for IgG binding. The results presented here suggest SBI can bind to antigenic components from negative-gram bacteria (C. albicans, H. pylori, S. dysenteriae, and E. coli) commonly associated with GI inflammation and disease.

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## *FIGURE 1*

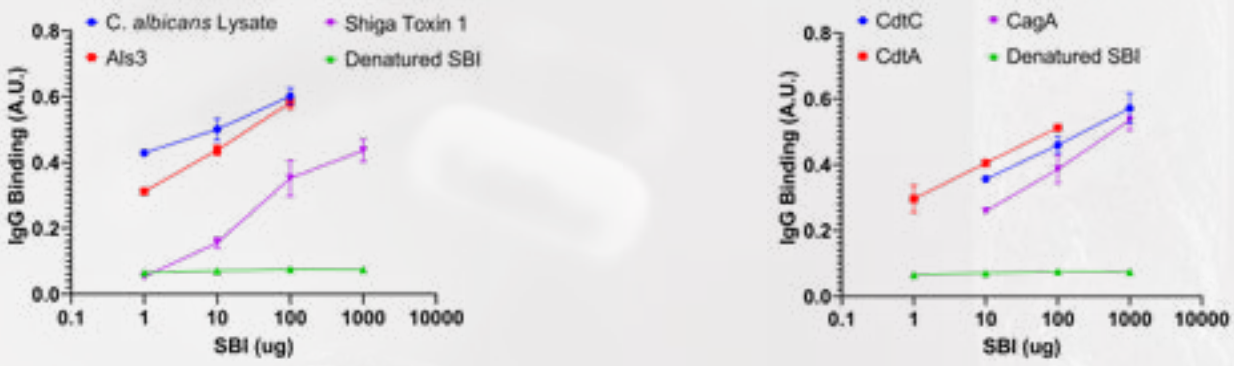


Figure 1. Binding of SBI IgGs to antigens produces significantly more absorbance than the denatured SBI control versus the same antigens. Green triangles show non-specific binding absorbance of denatured SBI to antigens. Other markers show specific binding of SBI IgG to antigens. A) SBI-IgG binds to Shiga toxin type-1 and C. albicans lysate and Als3 antigenic protein. B) SBI-IgG binds to Cytolethal Distending toxins A & B, and H. pylori CagA antigenic protein. Error bars represent ± one standard deviation from triplicate data. Absorbance units (A.U.) measured at 450 nm.

## *SBI BINDS TO THESE ANTIGENS*

JUST ADDED **AFLATOXIN B2 & G1**

- ***C. albicans*** **lysate**
- ***C. albicans*** **Als3 protein**
- ***H. pylori*** **CagA protein**
- ***H. pylori*** **lysate**
- **Shiga-like toxin type 1**
- **Lipopolysaccharide (LPS)**
- **C. difficile Toxin A & B**
- **Peptidoglycan**
- **Listeria Monocytogenes Lysate**
- **Cytolethal distending toxin subunit A & C**
- **Aflatoxin B2 & G1**
- **Lipoteichoic acid (LTA)**
- **Mycoplasma spp.**
- **Serratia Marcescens**
- **Salmonella Typhimurium**
- **Klebsiella Pneumonia**
- **C-di-AMP**
- **Poly 1:C**
- **Zymosan**
- **E. coli**
- **Gliadin**
- **Staphylococcus**
- **Pam3CSK4**
- **Flagellin**
- **NSP4**
- **MDP**
- **CpG**
- **ATP**

### *REFERENCES*


1. Zhang Y-J, Li S, Gan R-Y, Zhou T, Xu D-P, Li H-B. Impacts of Gut Bacteria on Human Health and Diseases. International journal of molecular sciences. 2015;16(4):7493-7519.
2. Ghosh SS, Wang J, Yannie PJ, Ghosh S. Intestinal Barrier Dysfunction, LPS Translocation, and Disease Development. Journal of the Endocrine Society. 2020;4(2)doi:10.1210/jendso/bvz039
3. Detzel CJ, Horgan A, Henderson AL, et al. Bovine immunoglobulin/protein isolate binds pro-inflammatory bacterial compounds and prevents immune activation in an intestinal co-culture model. PloS one. 2015;10(4):e0120278. doi:10.1371/journal.pone.0120278