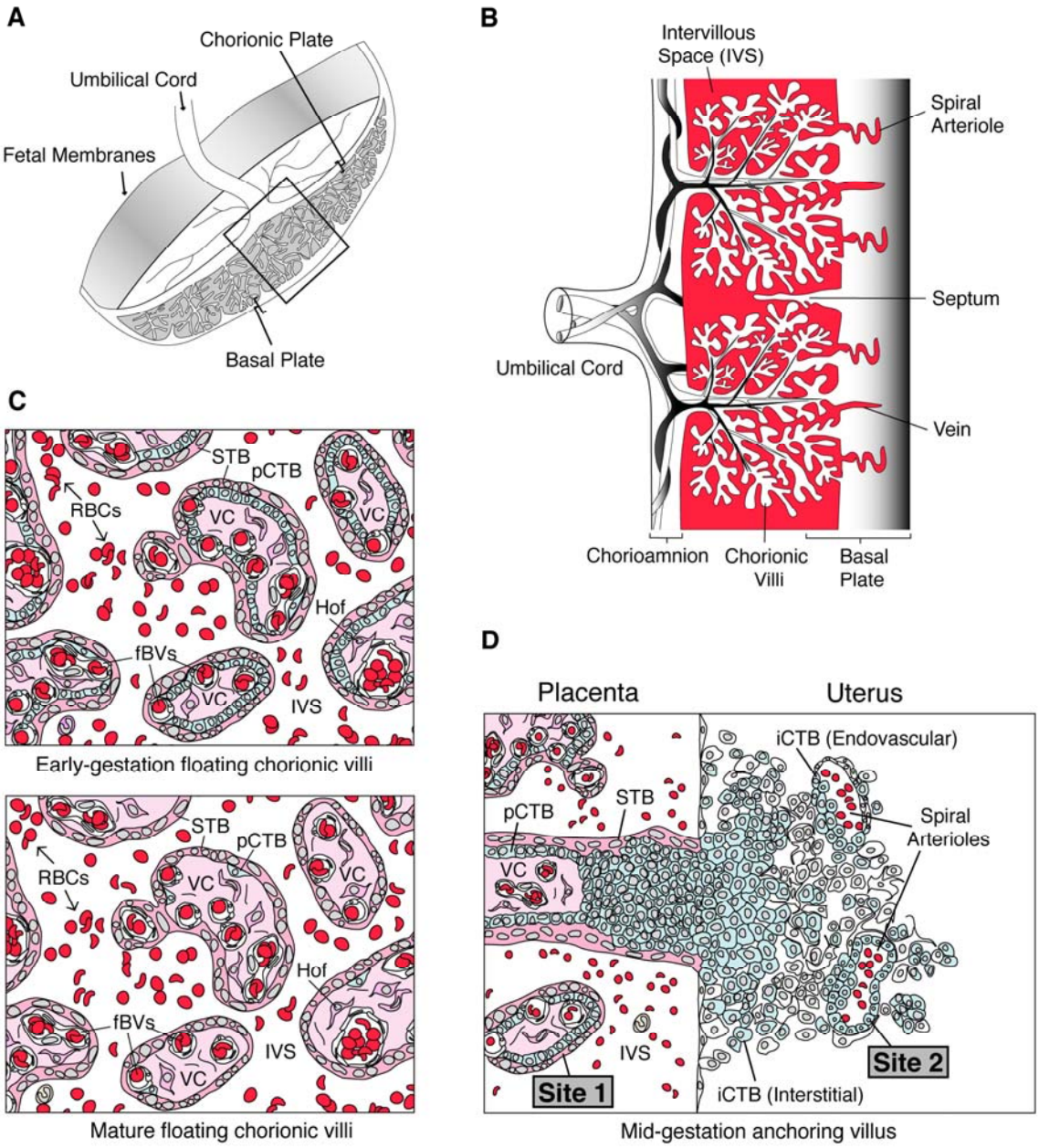

Supplemental Figure S1. The human maternal-fetal interface. (A) The human placenta at delivery. The placental surface that was adjacent to the uterine wall is termed the basal plate (bracket). The boxed area is diagrammed in panel B. (B) The placenta contains tree-like chorionic villi. Uterine spiral arterioles deliver maternal blood to the intervillous space (IVS), thus perfusing the placenta. Folds of the basal plate that project into the intervillous space are termed septa. (C) Cross-section of floating chorionic villi. During early gestation (top panel) progenitors (pCTBs, grey) form a polarized epithelium that is attached to the basement membrane surrounding the stromal villous core (VC). Progenitor CTBs detach from this membrane and fuse to form syncytiotrophoblasts (STB, pink). At term (bottom panel), the pCTB layer is largely depleted. The VC contains the intrinsic placental vascular (fetal blood vessels; fBVs), mesenchymal cells and macrophage-like Hofbauer cells (Hof). The intervillous space contains maternal blood, primarily red blood cells (RBCs). (D) Cross-section of an anchoring chorionic villus. In the basal plate region, invasive CTBs (iCTBs, grey) form a bridge between the placenta and the uterus. Interstitial iCTBs comingle with uterine cells. Endovascular iCTBs invade and subsequently line uterine spiral arterioles. Based on the physical relationship between maternal and embryonic/fetal cells, sequestration is most likely to occur via STB on the surface of floating and anchoring villi (site 1) and endovascular iCTBs (site 2). In both locations, placental cells are in direct contact with maternal blood. [Fig. 1A, D were adopted from Winn et al., [79] with permission].

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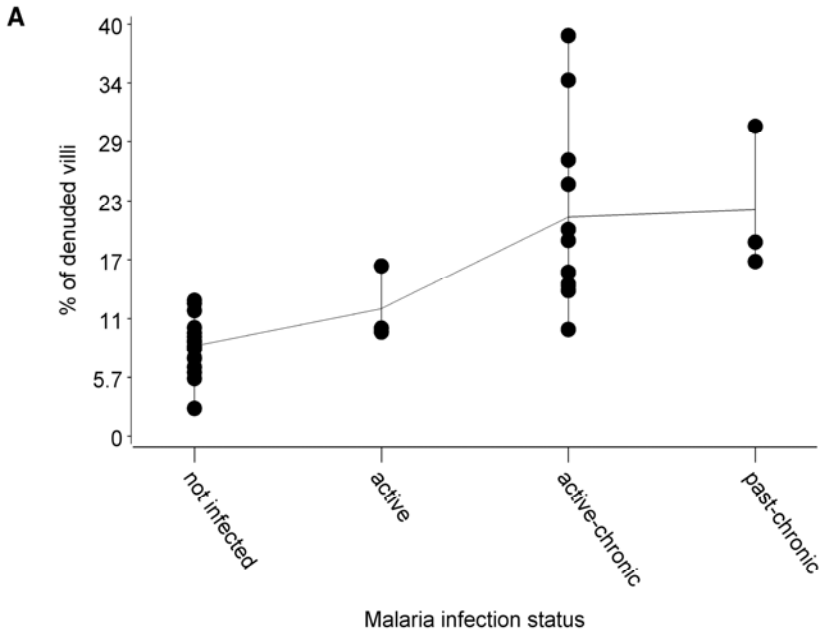

Supplemental Figure S2. STB denudation is a feature of chronic placental malaria. Cases and controls (n=17/group) were categorized according to a grading scheme by Bulmer et al. [38] and plotted as a function of STB denudation. 50% of the samples were not infected (n=17), 9% displayed active infection (n=3), ~32% displayed active-chronic infection (n=11), and ~9% displayed past-chronic infection (n=3).

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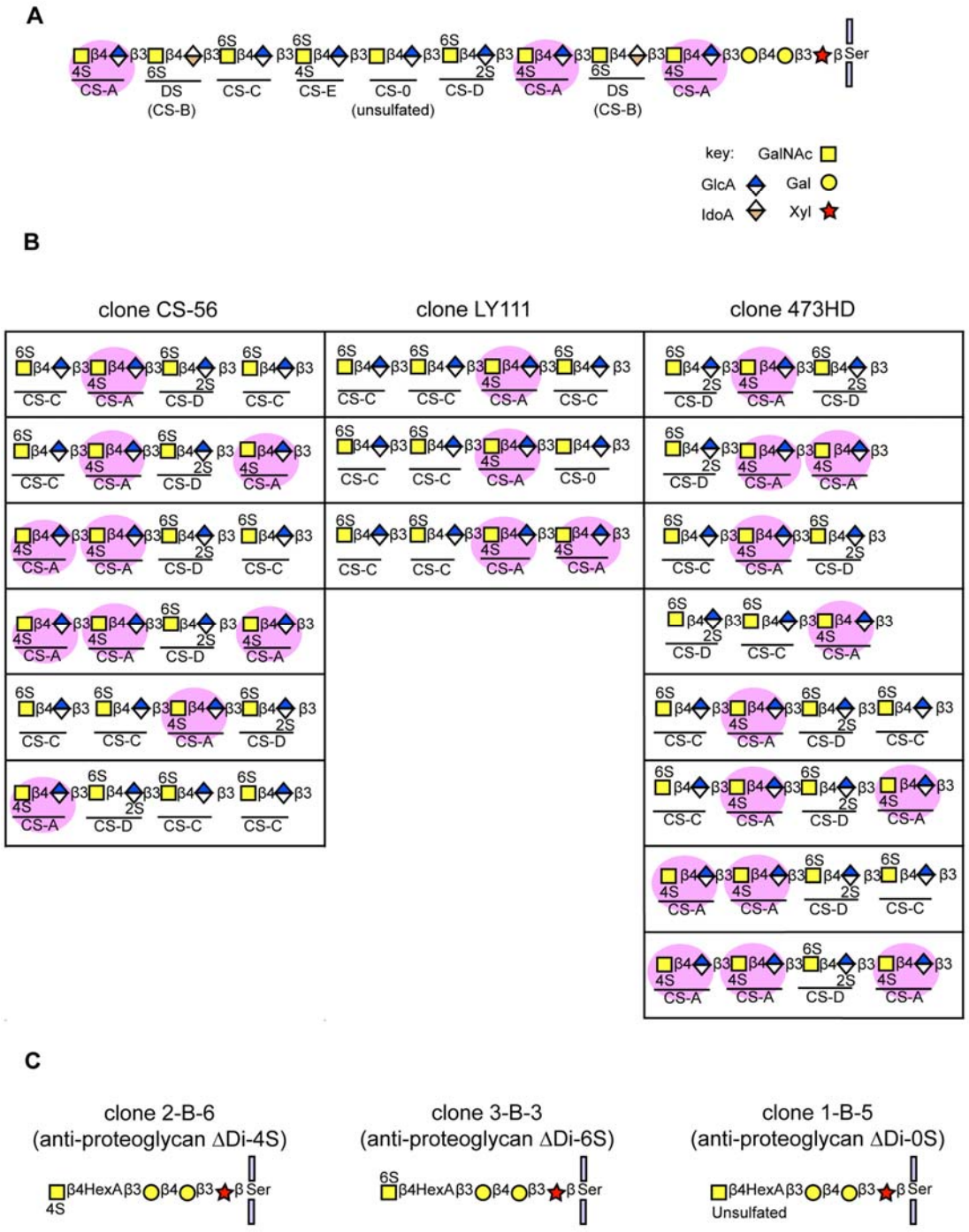

Supplemental Figure S3. Specificities of anti-chondroitin sulfate mAbs. (A) CS-GAGs are unbranched polysaccharides of variable length consisting of repeating disaccharide units of D-glucuronic acid (GlcA) and *N*-acetyl-D-galactosamine (GalNAc). CS-GAGs contain a heterogeneous mixture of CS and dermatan sulfate (DS) disaccharides. CS disaccharides are named according to the position of sulfation: C-4 of GalNAc (*i.e.*, chondroitin-4-sulfate/CS-A), C-6 of GalNAc (*i.e.*, chondroitin-6-sulfate/CS-C), both substituents of GalNAc (*i.e.*, chondroitin-4,6-sulfate/CS-E) and C-6 of GalNAc plus C-2 of GlcA (*i.e.*, CS-D). (B) Oligosaccharides recognized by CS-56, LY111, and 473HD mAbs. CS-56 and 473HD recognize epitopes that contain the A-D core tetrasaccharide [49, 51, 52]; LY111 reacts with chains that contain a CS-A motif [51, 52]. CS-A disaccharides are marked with pink circles. (C) Stub epitopes, which are created by chondroitinase ABC digestion, are recognized by mAbs 2-B-6 (left), 3-B-3 (middle) and 1-B-5 (right) [53,54 *the specificity of 2-B-6 is the same as 9-A-2]. Hexuronic acid (HexA) represents glucuronic acid if it was a CS chain, or iduronic acid if it was a DS chain. Additional information regarding these mAbs can be found on the Seikagaku website: http://www.seikagaku.co.jp/english/.

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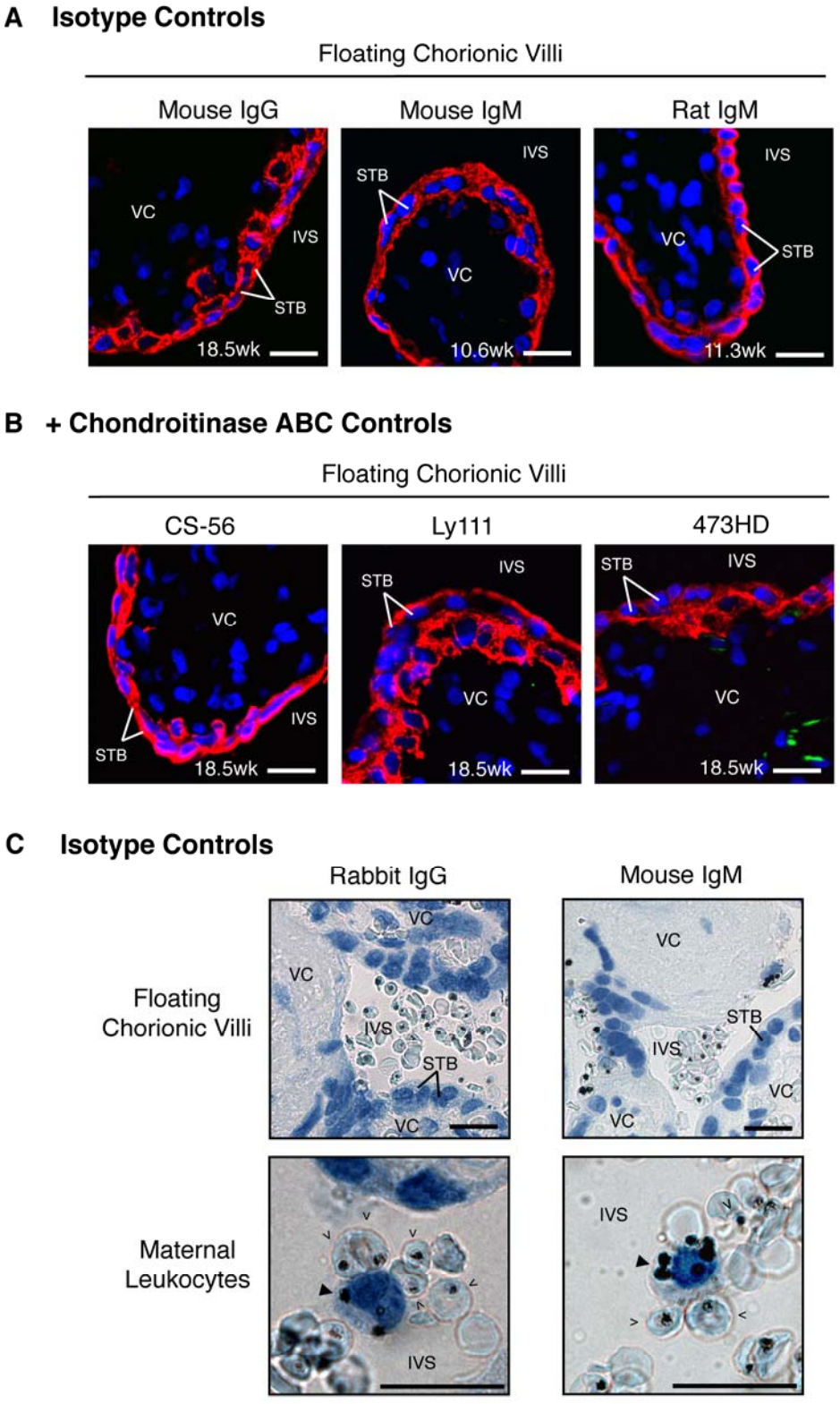

Supplemental Figure S4. Staining controls. (A) In frozen sections, mouse IgG (left), mouse IgM (middle) and rat IgM (right) isotype control antibodies were used to monitor binding via fetal Fc receptors. (B) Antibody specificity for CS-GAGs was monitored by digesting sections with chondroitinase ABC prior to staining with CS-56 (left), LY111 (middle) and 473HD (right). (C) In paraffin-embedded sections, rabbit IgG (left) and mouse IgM (right) isotype control antibodies were used to monitor binding via fetal Fc receptors in the chorionic villi (top) or on maternal leukocytes (bottom). Scale bars, 40μm.

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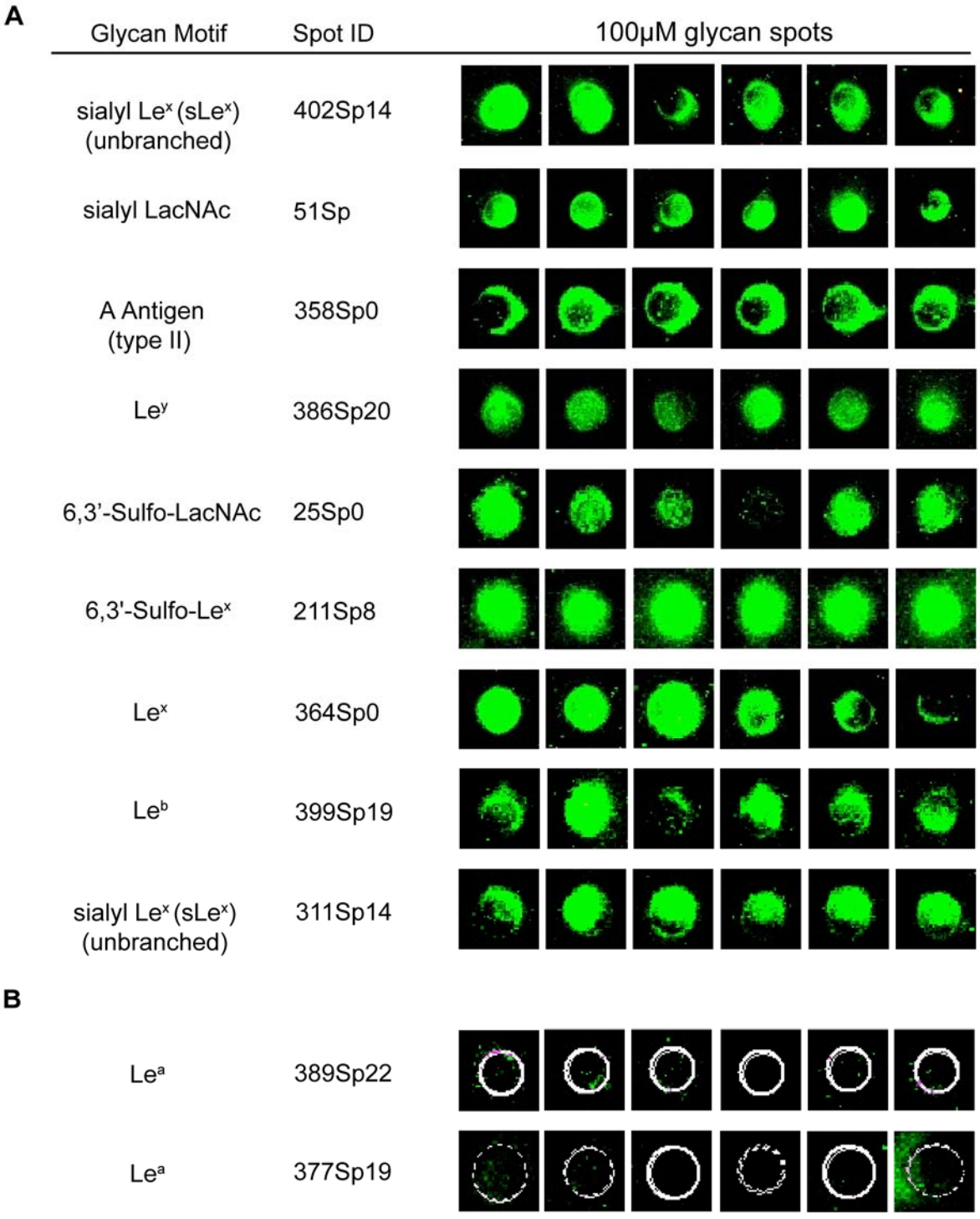

Supplemental Figure S5. Infected RBCs bound reproducibly to glycan spots. (A). The same oligosaccharide was spotted in six locations on each slide. Data are representative of two independent binding experiments. In general, iRBCs bound in a reproducible manner to the glycans shown. (B) Infected RBCs failed to bind two spots that contained Le^a, which was not expressed by STB (see Fig. 6).

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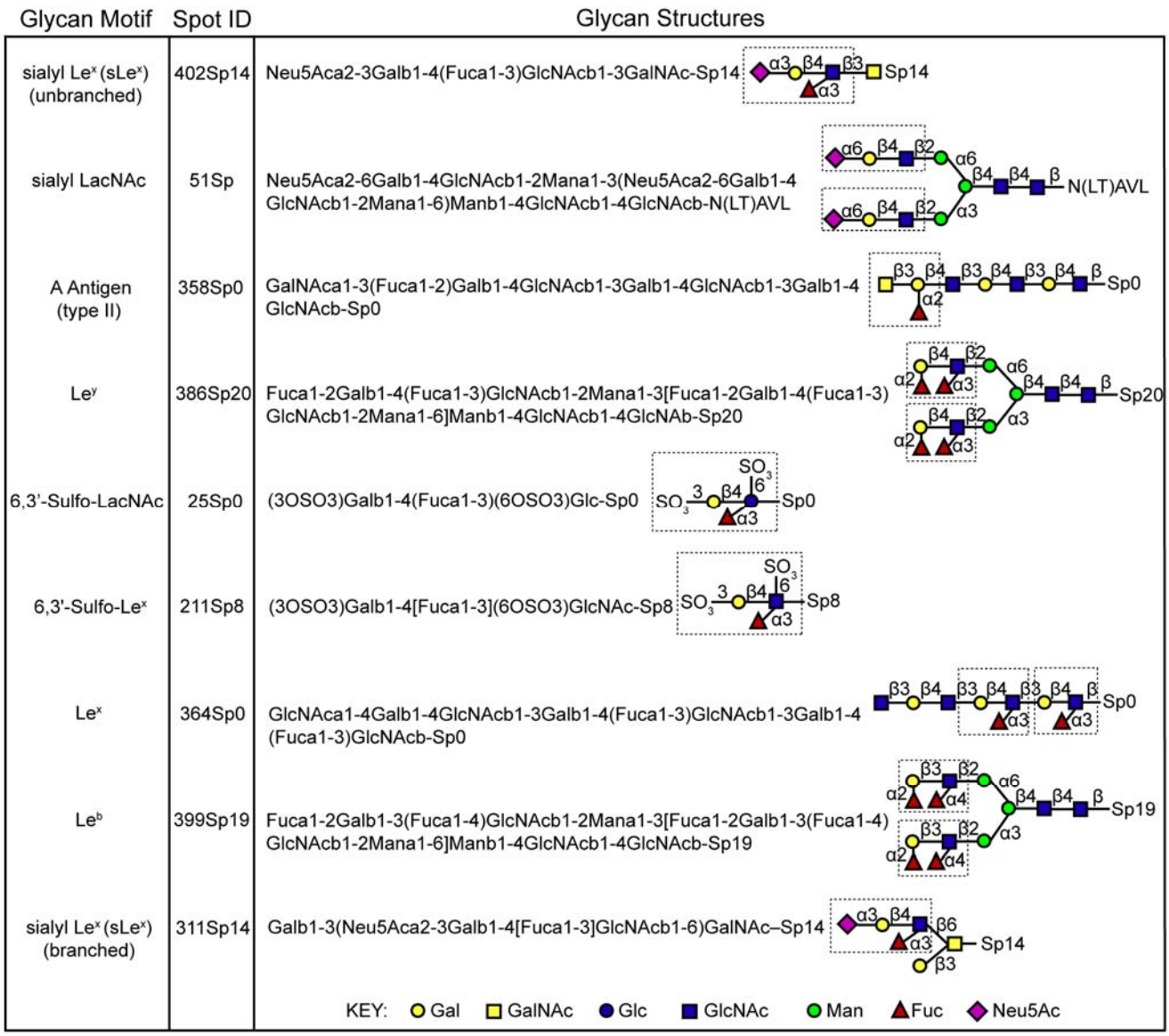

Supplemental Figure S6. Infected RBC glycan binding partners. Infected RBCs bound oligosaccharides that contained Lewis (Le) antigens and related lactosamine (LacNAc) motifs. Spot identities (spot ID column) were assigned by the Consortium for Functional Glycomics. The structures that were spotted on the array are listed in the third column; boxes outline specific carbohydrate motifs. Galactose, Gal. N-acetyl galactosamine, GalNAc. Glucose, Glc. N-acetyl glucosamine, GlcNAc. Mannose, Man. Fucose, Fuc. Sialic acid, Neu5Ac.

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