Background The pathogenesis of Systemic Lupus Erythematosus (SLE) is multifactorial with genetic make-up and environmental triggers considered major players. Among the environmental triggers, infections are a major cause of morbidity and mortality in SLE patients. Bacteremia is often overlooked in SLE patients and soft tissue abscesses, bloodstream infections, and sepsis are more common in SLE patients, suggesting that frequent exposures to microbial products may trigger flares in lupus. We have recently shown that a bacterial amyloid termed curli, expressed in the multicellular communities (biofilms) by many bacteria including *E. coli*, plays a major role in triggering lupus autoimmunity during infection. Curli bind bacterial or eukaryotic DNA and form curli/DNA complexes that strongly activate innate immunity. When given systemically, curli/DNA complexes and infections with curli-expressing *E. coli* trigger production of anti-dsDNA and anti-chromatin autoAbs in lupus prone mice and in wild type mice. This effect was diminished in TLR2 or TLR9 deficient mice.

Methods Young wild type C57BL/6 mice, lupus prone Sle1,2,3 mice and 3 H9 mice were used. Splenic B cells were stained by flow cytometry for *in vivo* experiments. For *in vitro* experiments, B cells were sorted by positive selection with CD45R (B220) Miltenyi Biotec micro bead or Easysep B cell isolation kit (Stem cell) supplemented with anti-CD43-Biotin. B cell purity (>98%), proliferation, viability, activation markers, surface antibodies and signaling were measured by CFSE dilution (Promokine), 7AAD and antibodies by Flow cytometry, *Western Blot analysis* and qRT-PCR.

Results We found that curli/DNA complexes polyclonally activate B cells *in vivo* in wildtype mice, lupus-prone mice and 3 H9 mice, the latter expressing an anti-DNA lg heavy chain and whose B cells are normally tolerized. Curli/DNA complexes can also activate B cells *in vitro* in the absence of T cell help. The induction of non-canonical NFκB in the absence of T cell help suggests that the fibrillar structure of curli/DNA complexes can cross-link BCRs, some recognizing DNA. Interestingly, curli/DNA complexes also induce isotype switching and aicda, the master regulator of class switch recombination, in the absence of T cells help in *vivo*.

Conclusions Our results suggest that curli/DNA complexes may induce anti-DNA antibody production by simultaneous BCR/TLR signaling, which leads to B cell antibody production in the absence of T cell help.

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