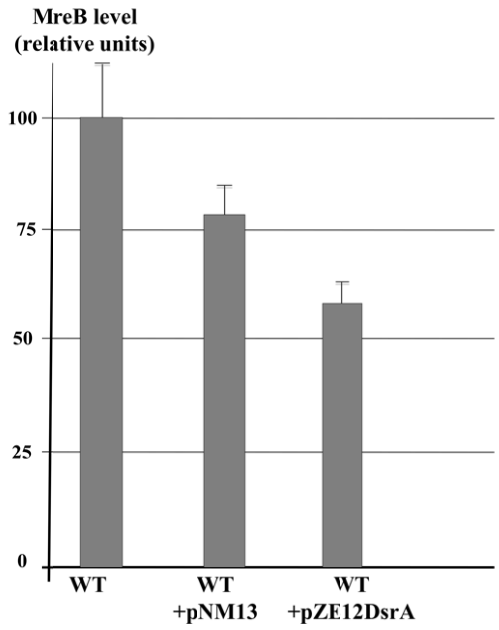
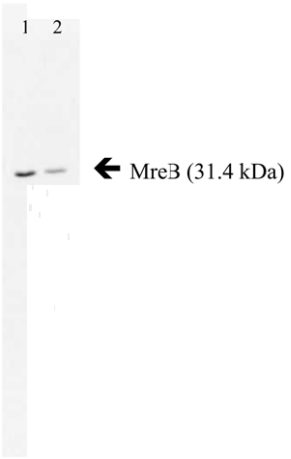
Supplementary figure S1: DNA sequence of *mreB* region. The *mreBCD* region is located at ~ 73 min in the *E. coli* chromosome.[1] Three σ^D-dependent promoters contribute to *mreB* transcription (P^{mreB}_1, P^{mreB}_2 and P^{mreB}_3, -10 and -35 boxes) and are indicated as black boxes. Probable transcription initiation sites (-269, -106 and -42) are indicated in bold.[2] Note that -106 initiation site is atypical and could in fact result from a post-transcriptional cleavage by an RNAse.[2] Shine Dalgarno (SD) and translation initiation codon of *mreB* are indicated. The region of *mreB* that anneals with DsrA is underlined with a waved-line. The coding sequence of *mreB* is indicated in light grey, whereas the beginning of *mreC* coding sequence appears in dark grey. Stop codon of *mreB* and *yhdA* upstream gene (located nearby P^{mreB}_3, *-35*)[3] are italicized.

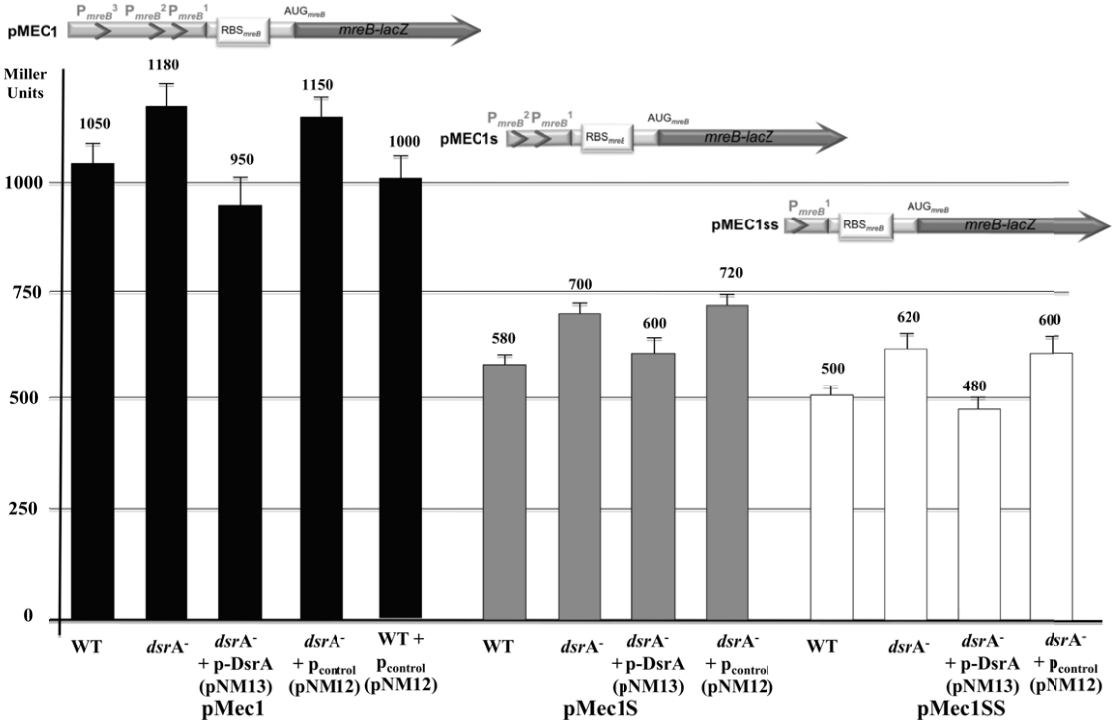
1. M. Doi, M. Wachi, F. Ishino, S. Tomioka, M. Ito, Y. Sakagami, A. Suzuki and M. Matsuhashi, *J Bacteriol*, 1988, 170, 4619-4624.
2. M. Wachi, K. Osaka, T. Kohama, K. Sasaki, I. Ohtsu, N. Iwai, A. Takada and K. Nagai, *Biosci Biotechnol Biochem*, 2006, 70, 2712-2719.
3. N. Sommerfeldt, A. Possling, G. Becker, C. Pesavento, N. Tschowri and R. Hengge, *Microbiology*, 2009, 155, 1318-1331.

Supplementary figure S2: Influence of DsrA on MreB concentration. *Left panel:* MreB quantifications were made by Western Blot as described in methods and Fig.1. Two plasmids allowing the expression of DsrA under the control of P_{BAD} (pNM13 + 0.01% arabinose) and constitutive $P_{LlacO-1}$ (pZE12) promoters were used. Both plasmids resulted in a significant diminution of MreB levels. *Right panel:* an example of Western Blot using a cell extract in the presence *(lane 2)* or not *(lane 1)* of a plasmid that express DsrA (constitutive expression). As shown, anti-MreB antibody is specific.





Supplementary figure S3: DsrA-mediated riboregulation on *mreB–lacZ* translational and transcriptional reporter fusion. β-galactosidase activities were assayed in cell extracts of the indicated strains grown at 16°C (exponential phase). Drawing is a schematic representation of the *mreB-lacZ* reporter fusion. We observe that DsrA affects *mreB* expression and that DsrA expression from pNM13 (+ 0.001% L-arabinose) in a Δ*dsrA* background complements Δ*dsrA* phenotype, while the empty pNM12 vector does not. Note that the effect observed for Δ*dsrA* strain is slightly less than that seen for the protein level (Fig. 1). Nevertheless, fusion reflects *mreB-lacZ* RNA and MreB-lacZ protein stabilities, which is different from *mreB* mRNA and MreB protein stabilities.



Supplementary figure S4: The secondary structure model of *mreB*:DsrA complexes predicted using RNAfold server (http://rna.tbi.univie.ac.at/cgi-bin/RNAfold.cgi). The *mreB* AUG start codon is highlighted. Both WT and mutated *mreB* and DsrA pairings are shown.