Fig. S1 *msrA* expression patterns under the treatments of UV radiation, heat (48°C) and cold (4°C) in *D. radiodurans*.

A-C: Relative expression levels of *msrA* in response to the treatments of heat (48°C) (A), cold (4°C) (B) and UV radiation (C) in *D. radiodurans*. Asterisks indicate statistically significant difference of the value compared to that of untreated cells (one-way Anova, Dunnett’s multiple comparisons test; ‘***’ means P≤0.001, ‘*’ means P≤0.05). Experiments were performed at least three times, and data were presented as means SEM.
FIG S2 The knockout of msrA gene in *D. radiodurans* has no effect on the tolerance to UV radiation, heat (48°C) and cold (4°C) treatments in *D. radiodurans*.

A-C: Phenotype of different *D. radiodurans* strains under the treatments of UV radiation, heat (48°C) (A), cold (4°C) (B) and UV radiation (C).

Left images: untreated control; Right images: different abiotic stress treatments. *WT*: wild type strain, Δ*msrA*: *msrA* deleted mutant; *msrA*-pRADZ3: the *msrA* mutant transformed with pRADZ3 empty plasmid; *msrA*-com: *msrA* mutant supplemented with *msrA* gene.
FIG S3 The knockout of *DsrO* gene in *D. radiodurans* has no effect on the tolerance to UV radiation, heat (48°C) and cold (4°C) treatments in *D. radiodurans*.

A-C: Phenotype of different *D. radiodurans* strains under the treatments of heat (48°C) (A), cold (4°C) (B) and UV radiation (C).

Left images: untreated control; Right images: different abiotic stresses. *WT*: wild type strain, Δ *DsrO*: *DsrO* deleted mutant, *DsrO*-pRADZ3: the *DsrO* mutant transformed with *pRADZ3* empty plasmid; *DsrO*-com: *DsrO* mutant supplemented with *DsrO* gene.
FIG S4 The characterization of DsrO and its gene locus.

A: The amplification result of DsrO gene using 5’ rapid amplification of cDNA end (5’-RACE). M: 100bp DNA ladder; B: Physical map and nucleotide sequence of DsrO. Promoter elements (-35 and -10 box) are underlined; Transcription start site mapped by 5’-RACE is emphasized with arrows; DsrO nucleotide sequence is underlined from the start point.
FIG S5 MicroScale Thermophoresis (MST) analysis the nucleotides affecting the affinity between msrA and DsrO.

A. The sketch map describing the continuous base mutation in msrA or DsrO. B. The mutation of non-complementary bases enhanced the affinity between msrA and DsrO. C-D. The completed and partial mutations of complementary sequences resulted from the lose of interacting between msrA and DsrO.

The K_d coefficients were determined employing the standard data analysis of MST with affinity analysis software. The red curve is the fitted combination curve, and the K_d (dissociation equilibrium constant) value is the binding constant of sRNAs and their target mRNAs. The graphs display the data from 4 independent measurements. Green dots, msrA versus DsrO.
FIG S6 Monitoring binding events between msrA and DsrO.

A-F. The site-directed mutations of the DsrO sequence for determining the interaction ability between msrA and DsrO. The $K_d$ coefficients were determined for the molecules interaction employing the standard data analysis of MST with affinity analysis software. The red curve was the fitted combination curve, and the $K_d$ (dissociation equilibrium constant) value was the binding constant of sRNAs and their targets. The graphs displayed the data from 4 independent measurements. Green dots, msrA versus DsrO.
FIG S7 Monitoring binding events between msrA and DsrO.

A-F. The site-directed mutations of the msrA sequence for determining the interaction ability between msrA and DsrO. The K_d coefficients were determined for the molecules’ interaction employing the standard data analysis of MST with affinity analysis software. The red curve was the fitted combination curve, and the K_d (dissociation equilibrium constant) value was the binding constant of sRNAs and their targets. The graphs displayed the data from 4 independent measurements. Green dots, msrA versus DsrO.