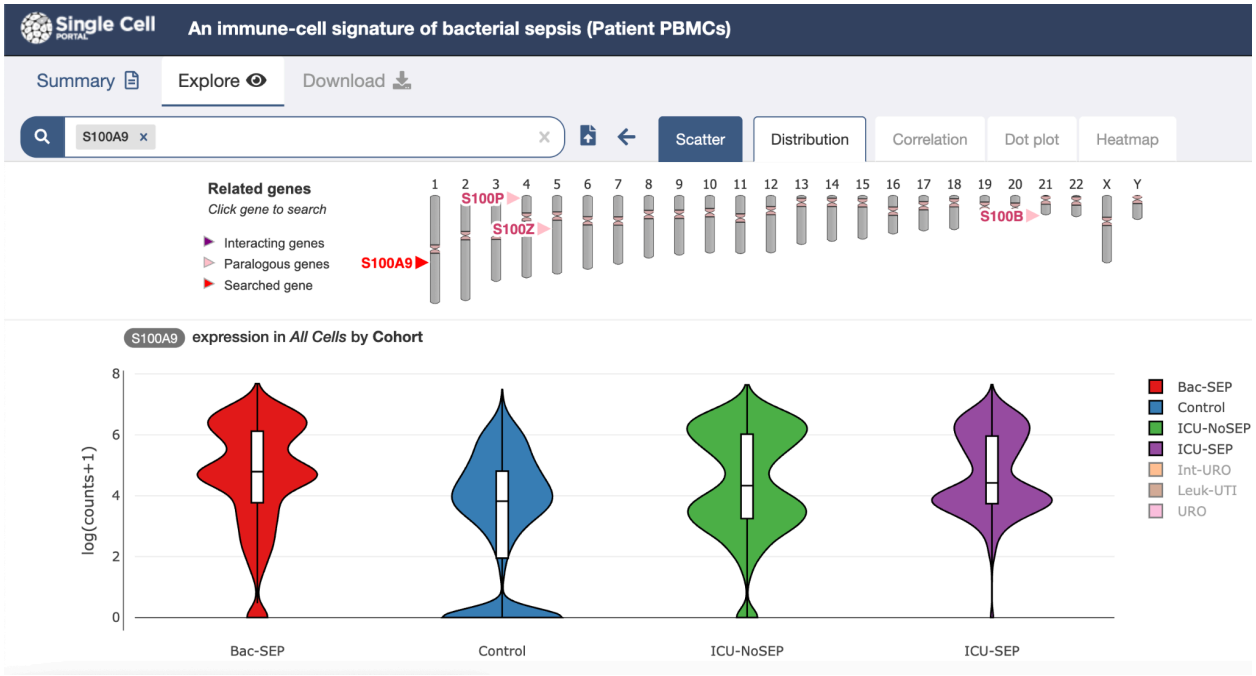
## scRNA-seq_S100A9:

This violin plot shows the log-normalized expression levels of the gene S100A9 across four cohorts of patients using single-cell RNA-seq data from peripheral blood mononuclear cells (PBMCs):




| Cohort | Violin Shape | Median Expression (Box center) | Overall Height | Interpretation |
|---|---|---|---|---|
| **Bac-SEP** (🔴 Red) | Tallest & Widest | Median around ~5.2 | Max > 7 | Very high expression across many cells. |
| **ICU-SEP** (🟣 Purple) | Slightly shorter but wide | Median ~4.5 | Max ~7 | Moderately high expression, close to Bac-SEP. |
| **Control** (🔵 Blue) | Narrow, low | Median ~2–3 | Max ~5 | Low expression. Most cells have minimal S100A9 activity. |
| **ICU-NoSEP** (🟢 Green) | Moderate-to-high | Median ~4.5 | Max < 5 | Significant expression. |

S100A9 (also known as calgranulin B or MRP14) is a calcium-binding protein predominantly expressed in neutrophils and monocytes. It often forms a heterodimer with S100A8 (together known as calprotectin).

### 🧪 Biological role:

Acts as a damage-associated molecular pattern (DAMP).
Amplifies inflammatory responses by binding to receptors like TLR4 and RAGE.
Plays a central role in sepsis, infection, autoimmune disease, and cancer.
Facilitates leukocyte recruitment and contributes to antimicrobial defense.

Overexpression of S100A9 is strongly linked to:

- Systemic inflammation
- Sepsis severity
- Innate immune activation

The violin plot demonstrates that S100A9 expression is markedly elevated in bacterial sepsis (Bac-SEP) and ICU-related sepsis (ICU-SEP), reflecting its role as a key inflammatory mediator. Interestingly, moderate upregulation in ICU-NoSEP suggests that critical illness alone, even in the absence of sepsis, can stimulate S100A9. These findings support its **potential as a biomarker for immune activation** and disease severity in sepsis-related contexts.