

Bipartite *NUP98::RARA-E412** fusion with a *cis*-aligned ligand binding domain truncation mutation in atypical acute promyelocytic leukemia

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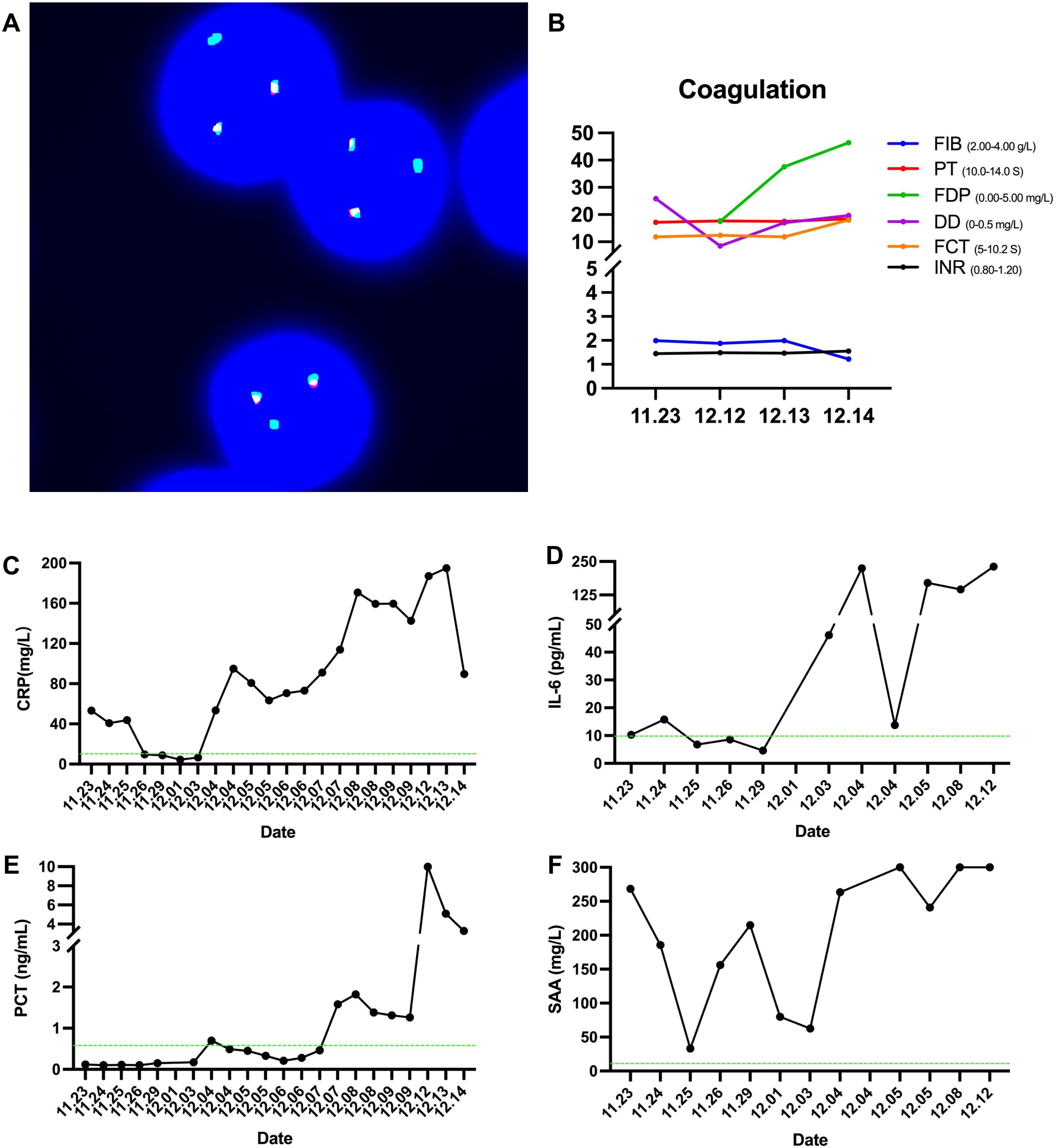
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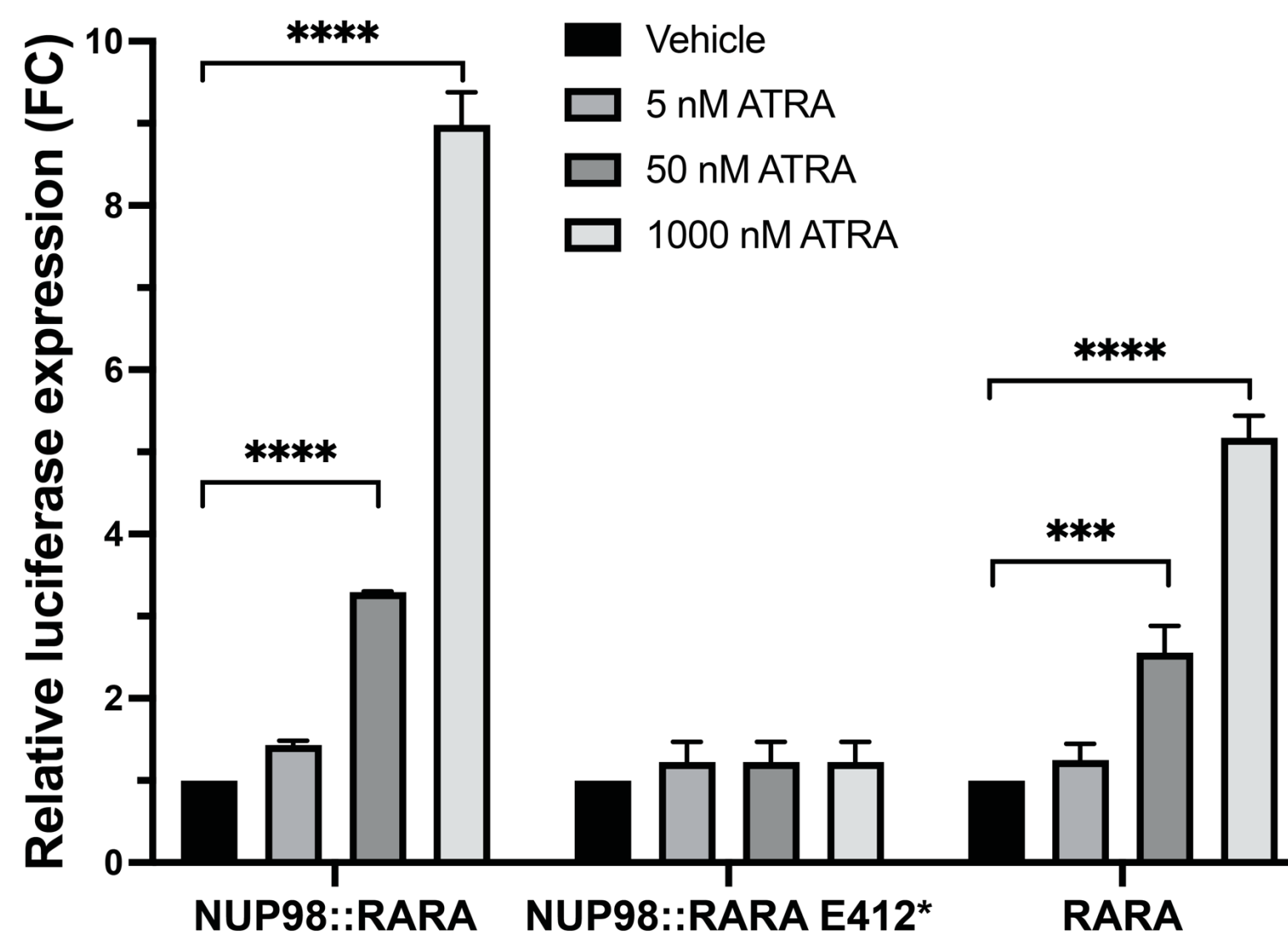
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Supplementary Figure 1. FISH testing and monitoring of inflammatory markers and coagulation in therapeutic process.

A Break-apart FISH analysis showed a gain of the green signal corresponding to the 3' *RARA* region. **B** Coagulation markers. **C** C-reactive protein. **D** Interleukin-6. **E** Procalcitonin. **F** Serum amyloid A.

FISH, fluorescence in situ hybridization; FIB, fibrinogen; PT, prothrombin time; FDP, Fibrin Degradation Products; DD, D-dimer; FCT, fibrinogen clotting time; INR, international normalized ratio.



Supplementary Figure 2. Assessment of ATRA responsiveness using a modified GAL4-UAS luciferase reporter assay.

Constructs encoding wild-type RARA, NUP98::RARA with an intact LBD, and the truncated NUP98::RARA-E412* mutant were cloned into the pBIND vector containing Renilla luciferase. NUP98::RARA showed dose-dependent ATRA responsiveness, while the E412* mutant was completely unresponsive. Luciferase activity (fold change relative to vehicle [DMSO]-treated cells) was normalized to Renilla luciferase expression. Data are presented as mean $\pm$ SD from three biological replicates; one representative experiment is shown (of three with comparable results). **** $P < 0.0001$, *** $P < 0.001$ (one-way ANOVA with Bonferroni correction).

ATRA, all-trans retinoic acid.