bio-techne® | R&D SYSTEMS

Designed For Hyperactivity

AI Modified Proteins for Cell Culture

Activin A Hyperactive Protein

Experience the future of protein design with our AI modified proteins, engineered to solve cell culture challenges. Although similar in biological function to its wild-type counterpart, through a single point mutation (F368A), Activin A Hyperactive Protein offers superior performance providing:

- **Enhanced Activity:** Cost-effective protein provides 4-fold higher activity than wild-type Activin A, allowing less protein to be used to achieve the same potency.
- **Consistent, Reliable Performance:** Supports efficient endoderm differentiation in human iPSC lines with consistent results.
- **Confidence in Dependable Outcomes:** Facilitates the effective generation of PDX1⁺/NKX6.1⁺ pancreatic progenitors, delivering results comparable to the wild-type protein.

Optimized for Higher Bioactivity

Higher ED_{50} Value With Activin A Hyperactive

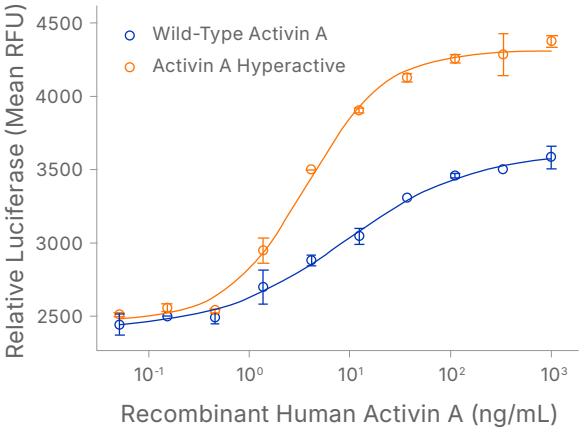

Figure 1. Activin A Hyperactive Protein Bioactivity is Greater Than Wild-Type Activin A.
Human Activin A Hyperactive Protein (Catalog # BT-ACTAH) and wild-type Activin A Protein (Catalog # 11348-AC) were assessed for their ability to induce SBE (SMAD-binding element) reporter activity as a readout of phosphorylated SMAD2/3 signaling in HEK293 human embryonic kidney cells. Activin A Hyperactive protein has greater bioactivity than wild-type Activin A.

Reliable Performance

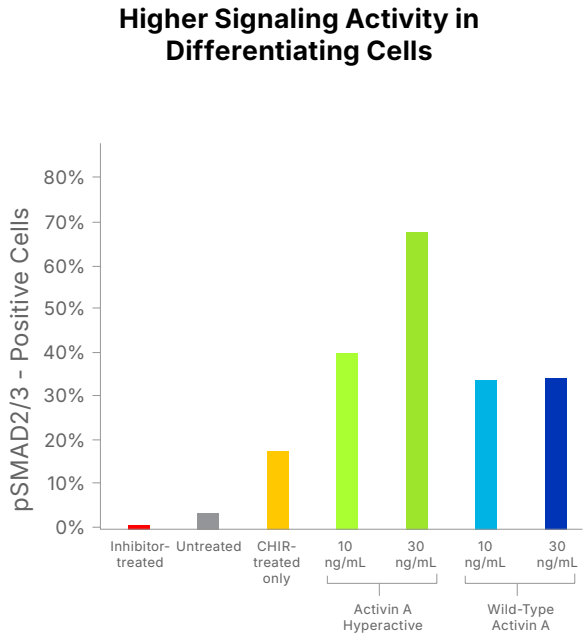

Figure 2. Activin A Hyperactive Promotes Higher Levels of pSMAD2/3 Signaling in Differentiating iPSCs than Wild-Type Activin A.
iPSCs were differentiated for 24h using CHIR 99021 (Catalog # 4423) and Activin A to examine pSMAD2/3 as a readout of signaling activity. Activin A Hyperactive (Catalog # BT-ACTAH) was compared to wild-type Activin A (Catalog # 11348-AC) at two concentrations, alongside untreated and CHIR-treated only controls. A negative control condition with TGF-β Type I Receptor inhibitor SB-431542 (Catalog # 1614), which prevents phosphorylation of SMAD2/3, was included. Cells were assessed by flow cytometry for pSMAD2/3 activity. At 24h, Activin A Hyperactive showed higher pSMAD2/3 levels compared to wild-type.

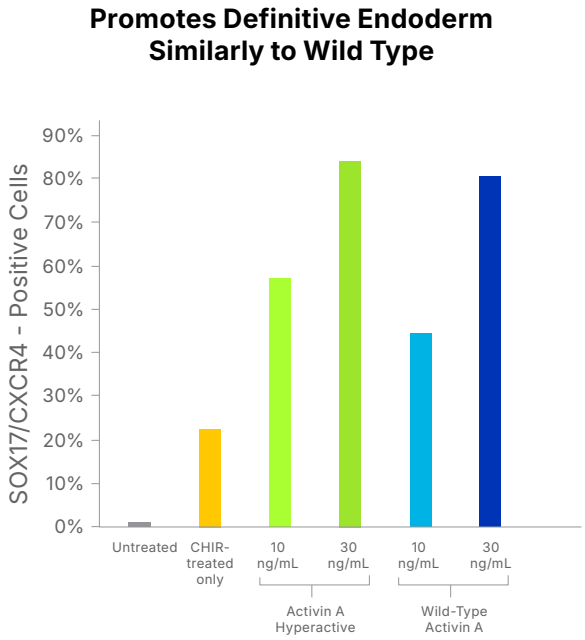

Figure 3. Activin A Hyperactive Promotes Higher Levels of pSMAD2/3 Signaling in Differentiating iPSCs than Wild-Type Activin A. iPSCs were differentiated for 72h into definitive endoderm using Activin A Hyperactive (Catalog # BT-ACTAH) or wild-type Activin A (Catalog # 11348-AC) at two concentrations, alongside untreated and CHIR-treated only controls. At 72h of differentiation, flow cytometry was performed to quantify the percentage of cells expressing both markers of endoderm, SOX17 and CXCR4. The results indicate Activin A Hyperactive promotes efficient endoderm differentiation in iPSC cells.

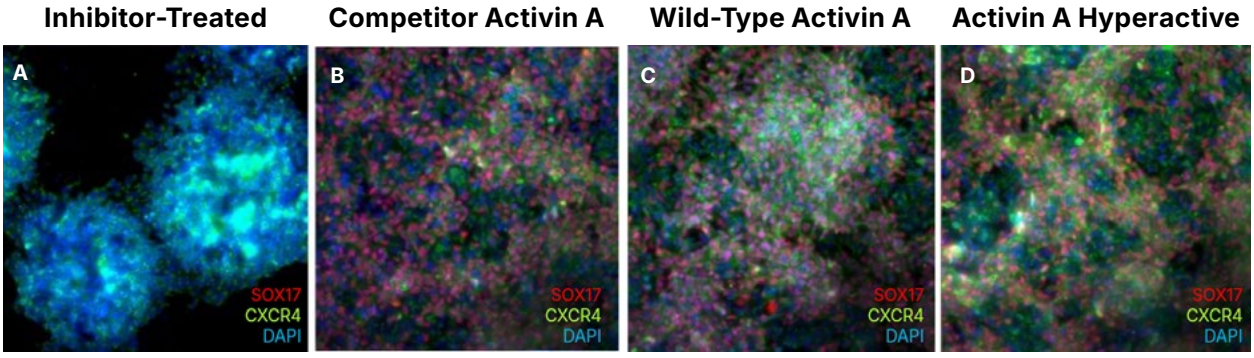

Figure 4: Efficient Derivation of SOX17⁺/CXCR4⁺ Definitive Endoderm With Activin A Hyperactive.
iPSCs were differentiated into definitive endoderm for 72h using (A) an inhibitor-treated negative control, (B) wild-type competitor Activin A, (C) wild-type Activin A (Catalog # BT-ACTAH) and (D) Activin A Hyperactive (Catalog # BT-ACTAH). To assess endoderm differentiation, cells were stained with Goat Anti-Human SOX17 (red; Catalog # AF1924), Rat Anti-Mouse CXCR4 (green; Catalog # MAB21651) and DAPI (blue; Catalog # 5748). The images show efficient derivation of SOX17⁺/CXCR4⁺ definitive endoderm using Activin A Hyperactive.

Confidence In Results

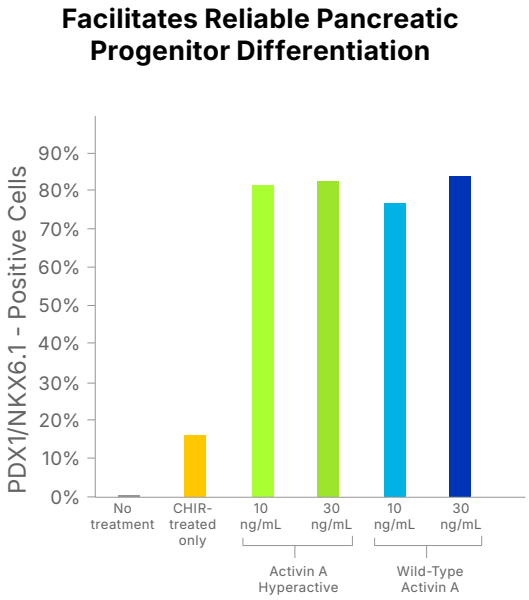

Figure 5: Activin A Hyperactive Produces High Percentage of PDX1⁺/NKX6.1⁺ Pancreatic Progenitor Cells
iPSCs were differentiated into pancreatic progenitors with Activin A Hyperactive or wild-type Activin A (Catalog # 11348-AC) for 13 days. Other supplemental proteins and small molecules included Recombinant Human FGF-2 (Catalog # 3718-FB), FGF-10 (Catalog # 345-FG), Wnt-3a (Catalog # 5036-WN), EGF (Catalog # 236-EG), SANT-1 (Catalog # 1974), Dorsomorphin (Catalog # 3093), Retinoic Acid (Catalog # 0695), and LDN-193189 (Catalog # 6053). On day 13, the cells were fixed and stained for flow cytometric analysis. Successful differentiation into pancreatic progenitors was assessed by measuring PDX1 and NKX6.1 co-expression. Results indicate efficient derivation of pancreatic progenitors (>80% PDX1⁺/ NKX6.1⁺) with Activin A Hyperactive.

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