Тренды анализа текстовых корпусов:

какие данные можно извлекать из текстов

Маргарита Лангенбах

к. филол.н., компьютерный лингвист

UNICHECK

turnitin

Анализ текстовых данных

Поиск фактов

- Основная тема/идея (суммаризация, поиск ключевых слов)
- NER (Named Entities Recognition, распознавание именованных сущностей)
- утверждения (и проверка их истинности)
- определение происхождения текста (написан человеком или машиной) и проверка его авторства

Поиск связей

- Источники данных, представленных в тексте, определение заимствованных фрагментов
- Поиск/группировка похожих документов (по тематике, цитируемым источникам, рисункам и т.д.)

Library

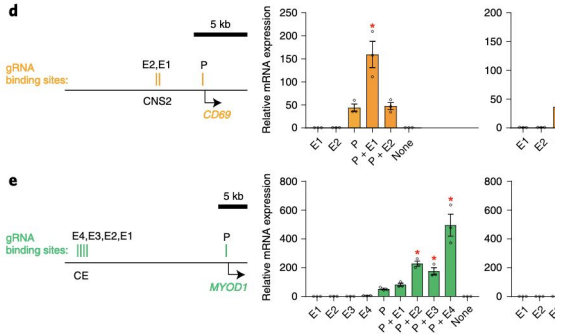

Fig. 1 | Heterotopic activation of enhancer sequences by dCas9-based aTFs ir
that activates promoter Y in cell type A, (ii) lack of enhancer X activity on pron
Y in cell type B when an aTF is recruited only to enhancer X, and (iv) robust en
both enhancer X and promoter Y. **b**, Architectures of dCas9-based bipartite an
the endogenous *IL2RA*, *CD69* and *MYOD1* genes in human cell lines in the pres
(E1, E2, E3 or E4) or promoter (P) sequences. Relative expression of each gene
normalized to *HPRT1* levels and calculated relative to that of a control sample (
replicates ($n = 3$), bars represent the mean of replicates, and error bars show th
the sample targeting only the promoter, $^*P < 0.05$ (two-tailed Student's *t*-test,

T cells[16] and present in closed chromatin in these three cell lines (Fig. 1d and Extended Data Fig. 1b). Additionally, we tested the bipartite p65 aTF with four single gRNAs targeted to a core enhancer (CE) located ~20 kb upstream of the *MYOD1* TSS (Fig. 1e), previously shown to be active in myoblasts[17], but that resides in inactive, closed chromatin in human HEK293, U2OS, HepG2 and K562 cell lines (Extended Data Fig. 1c). These experiments revealed only modest activation of *MYOD1* (sixfold) with just one of the four gRNAs (E4) in HEK293 and U2OS cells and no significant activation with any of the four gRNAs in HepG2 and K562 cells (Fig. 1e).

Concurrent artificial transcription factor promoter targeting unlocks enhancer activity. We speculated that the inability to consistently and efficiently induce gene activation from distal enhancer elements with an aTF might be due to the inactive, closed state of the target gene promoter in these heterotopic cell settings (Fig. 1a) and therefore further envisioned that concurrent targeting of an aTF

Image

originality check

Library

find and check source

citation

Find documents citing the same source

terms keywords

Find similar documents

originality check

statement

document idea