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Novel entropy based hierarchical clustering framework for ultrafast protein structure search and comparison

Baris Ekim[1, 2]
[1]Armand Hammer United World College of the American West, USA
[2]Massachusetts Institute of Technology, USA

Identification and alignment of three-dimensional folding of proteins may yield useful information about relationships too remote to be detected by conventional methods, such as sequence comparison and may potentially lead to prediction of patterns and motifs in mutual structural fragments. With the exponential increase of structural proteomics data, the methods that scale with the rate of increase of data lose efficiency. Hence, new methods that reduce the computational expense of this problem should be developed. We present a novel framework through which we are able to find and align protein structure neighbors via hierarchical clustering and entropy based query search and present a web based protein database search and alignment tool to demonstrate the applicability of our approach. The resulting method replicates the results of the current gold standard with a minimal loss in sensitivity in a significantly shorter amount of time, while ameliorating the existing web workspace of protein structure comparison with a customized and dynamic web-based environment.

baris.ekim@uwc-usa.org

Genome-wide iRNA screening identifies novel aneuploidy inducing gene knockdowns that transform telomerase positive human cells

Cagatay Gunes[1, 2], **Jitendra K Meena**[1], **Johann M Kraus**[2], **Sarah-Fee Katz**[2], **Andre Lechel**[2], **Hans A Kestler**[1, 2] and **Karl L Rudolph**[1]
[1]Leibniz Institute of Age Research-Fritz Lipmann Institute e.V., Germany
[2]Ulm University, Germany

Aneuploidy represents a hallmark feature of aging associated carcinogenesis in humans but mutations in the classical ploidy control genes are rare in human tumors as these mutations reduce cell fitness. Recent data indicate that aneuploidy can inhibit or instigate tumorigenesis dependent on the genetic background. These observations imply that aneuploidy induction involves mechanisms that allow survival of aneuploid cells. Here, genome-wide, stable iRNA screening identified a large set of gene knockdowns with a novel role in aneuploidy induction in human cells. These gene knockdowns strongly associate with recurrent gene mutations in human cancer. Anchorage-independent growth and xenotransplantation experiments indicate that aneuploidy inducing gene knockdowns replace the requirement of iatrogenic introduction of oncogenic Ras for the malignant transformation of telomerase-positive primary human cells. Together, this study identifies cancer associated gene deletions that induce aneuploidy and malignant transformation of human cells in the presence of telomerase.

cagatay.guenes@leibniz-fli.de
cagatay.guenes@uniklinik-ulm.de