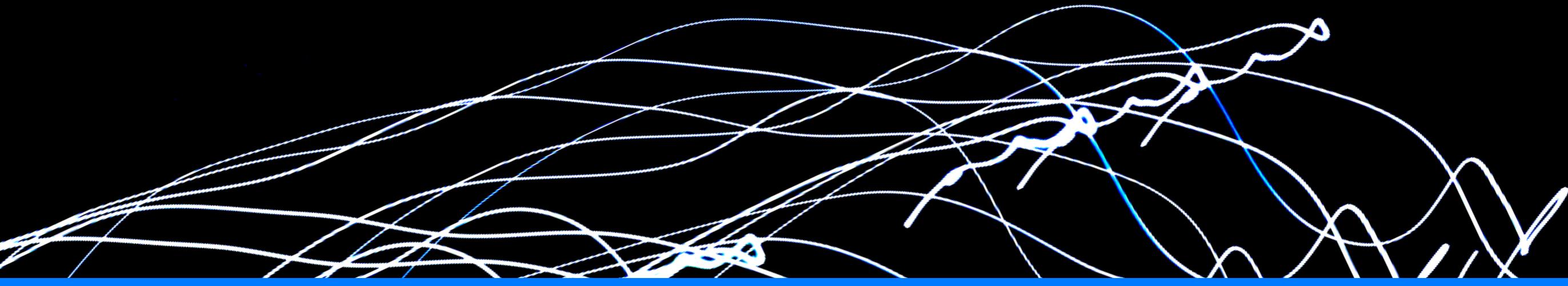


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Cell Biology, Chromosomes and Gene Expression

Chromosome mis-segregation and cytokinesis failure in trisomic human cells

Joshua M Nicholson, Joana C Macedo, Aaron J Mattingly, Darawalee Wangsa, Jordi Camps, Vera Lima, Ana M Gomes, Sofia Dória, Thomas Ried
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Virginia Tech, United States; Universidade do Porto, Portugal; National Institutes of Health, United States

Research Article · May 5, 2015

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Abstract

Cancer cells display aneuploid karyotypes and typically mis-segregate chromosomes at high rates, a phenotype referred to as *chromosomal instability* (CIN). To test the effects of aneuploidy on chromosome segregation and other mitotic phenotypes we used the colorectal cancer cell line DLD1 (2n = 46) and two variants with trisomy 7 or 13 (DLD1+7 and DLD1+13), as well as euploid and trisomy 13 amniocytes (AF and AF+13). We found that trisomic cells displayed higher rates of chromosome mis-segregation compared to their euploid counterparts. Furthermore, cells with trisomy 13 displayed a distinctive

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Chromosome mis-segregation and cytokinesis failure in trisomic human cells

Joshua M Nicholson, Joana C Macedo, Aaron J Mattingly, Darawalee Wangsa, Jordi Camps, Vera Lima, Ana M Gomes, Sofia Dória, Thomas Ried, Elsa Logarinho, Daniela Cimini

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“...Compared to the diploid parental line, the frequencies of chromosome missegregation and micronuclei formation were significantly elevated in most PTA clones (Figure 2A) but not in the tetraploid line (Figure 2A). In agreement with previous work ([Nicholson et al , 2015](#)), the trisomic clones showed similar aberrations, albeit to a lesser extent (Supplemental Figure S2B). Furthermore, we observed an increase of structural aberrations in PTA lines and, consistent with earlier work ([Kuznetsova et al , 2015](#) ; [Passerini et al , 2016](#)), also in trisomic clones (Figure 2B)...”

[Quantitative proteomic and phosphoproteomic comparison of human colon cancer DLD-1 cells differing in ploidy and chromosome stability](#) Viganó, von Schubert, Ahrné, et al. 2018 *MBoC* Section: RESULTS

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Studies carried out primarily in yeast and mammalian cell lines have shown that aneuploidy comes with a fitness cost. Aneuploid cells typically grow slower [McCoy et al., 1974; Torres et al., 2007; Williams et al., 2008; Tang et al., 2011; Siegel and Amon, 2012; Stingele et al., 2012] and suffer from replication stress that leads to DNA damage and gene mutation [Janssen et al., 2011; Crasta et al., 2012; Santaguida and Amon, 2015a; Passerini et al., 2016; Ly and Cleveland, 2017]. Also, both in vitro engineered aneuploid cells and chromosomally unstable cancer cells display gene expression patterns [Sheltzer, 2013] reminiscent of stress responses first described in yeast [Gasch, 2007]. Accordingly, aneuploid cells were found to show increased sensitivity toward compounds inducing energy stress and proteotoxic stress [Tang et al., 2011]. In nontransformed cells, chromosome missegregation generally leads to p53-dependent cell cycle arrest and, ultimately, cell death [Li et al., 2010; Thompson and Compton, 2010; Jetake and Sluder, 2010; Janssen et al., 2011; Ambrus et al., 2014]. Yet, despite this fitness cost, severe aneuploidy and CIN are hallmarks of human cancers [Hanahan and Weinberg, 2011; Holland and Cleveland, 2012; Funk et al., 2016; De Braekeleer et al., 2017]. They contribute to increased transformative potential [Paulsson and Johansson, 2007; Weaver et al., 2007] and correlate with poor prognosis [McGrath et al., 2012]. To resolve this apparent conundrum, it is generally argued that aneuploidy and CIN result in deregulated gene expression, which then confers a selective advantage during the evolution of a tumor in a changing microenvironment [Baek et al., 2009; Pavelka et al., 2010; Kwon-Chung and Chang, 2012; Yona et al., 2012]. As one example supporting this notion, DLD-1 cells engineered to carry single-chromosome aneuploidies were found to have a selective advantage over diploid control cells when cultured under non-standard conditions, such as serum starvation, drug treatment, or hypoxia [Rutledge et al., 2016]. Such observations, as well as data obtained in tumor models, strongly support the hypothesis that aneuploidy is not a by-product of cell transformation but, when present at appropriate levels, contributes to tumor development [Hanks et al., 2004; Holland and Cleveland, 2012; Davoli et al., 2013].

Aneuploidy in cancer cells may arise when diploid progenitors gain or lose individual chromosomes. However, chromosome loss is not well tolerated in diploid cells [Alvaro et al., 2006; Anders et al., 2009]. Moreover, cancer cells often carry near-tetraploid chromosome numbers, indicative of whole genome duplication events [Zack et al., 2013]. This suggests that aneuploid cancer cells often derive from tetraploid intermediates [Cowell and Wigley, 1980; Mayer and Aguilera, 1990; Storchova and Pellman, 2004; Storchova and Kuffer, 2008; Holland and Cleveland, 2012]. Considering that tetraploidization creates redundancy in chromosome content, it is expected to protect descendant aneuploid cells from the negative effects of haploinsufficiency [Shackney et al., 1989; Storchova and Pellman, 2004; Ganem and Pellman, 2007; Thompson and Compton, 2010; Dewhurst et al., 2014].

Aneuploidy has traditionally been ascribed to defects in mitotic spindle organization and/or dysfunction of the spindle assembly checkpoint [Wang et al., 2007; Kops et al., 2005]. However, although mutations in spindle checkpoint genes can indeed cause aneuploidy [Hanks et al., 2004; Yost et al., 2017], such mutations have not been commonly observed in cancers [Cahill et al., 1999; Haruki et al., 2001]. Deregulated expression of essential regulators of chromosome segregation and cell division has been observed in cancers with high degrees of aneuploidy and, accordingly, a CIN marker signature (CIN70) was proposed [Carter et al., 2006]. However,

subsequent studies argued that this CIN signature reflects altered proliferation rate rather than chromosome missegregation [Venet et al., 2011; Sheltzer, 2013; Buccitelli et al., 2017]. Thus, a specific cellular response to CIN has not yet been identified.

Here we established a set of transformed cancer cell lines of isogenic origin but differing in chromosome content and propensity to chromosome missegregation. To determine the effects of gains in chromosome mass versus CIN on protein expression and phosphorylation, we subjected the different cell lines to extensive proteomic and phosphoproteomic analyses. We found that proteomic changes in response to CIN are similar to those observed in response to tetraploidy and are more readily detectable at the level of protein phosphorylation than at the level of protein expression. Furthermore, our results indicate that large gains in chromosome number, as caused by tetraploidization, trigger widespread responses in protein expression and phosphorylation patterns, lending support to the notion that an initial genome doubling event can set the stage for survival and propagation of descendant aneuploid tumor cells.

RESULTS

Establishment of DLD-1-derived cell lines differing in ploidy and aneuploidy

Chromosome gains or losses result in massive changes in gene expression [Lyle et al., 2004; Jpender et al., 2004; Stingele et al., 2012], and protein expression patterns in cancer cell lines are known to reflect tissue origin, a priori making it difficult to identify a proteomic signature attributable to CIN. This notwithstanding, we subjected a panel of human cell lines to a proteomic quantification based on multiplexed tandem mass tag (TMT) labeling, a method of choice for achieving high proteome coverage in multiple samples and within a reasonable time frame [Thompson et al., 2003; Ahrne et al., 2016] (Supplemental Figure S1A and Supplemental Table S1). This panel included seven karyotypically stable (nonCIN) and unstable (CIN) cancer cell lines originating from different tumor tissues [Gascoigne and Taylor, 2008] and the immortalized retinal cell line hTERT. In line with previous data [Gascoigne and Taylor, 2008], we found that differences in global protein expression patterns were too profound to allow a distinction between CIN and karyotypically stable (nonCIN) cell lines through hierarchical cluster analysis (Supplemental Figure S1B). Nevertheless, this pilot study showed that our proteomics approach allowed for reliable quantification of thousands of proteins in each cell line.

To reduce interline variation due to tissue origin, we next used the diploid colon cancer cell line DLD-1 to generate descendant lines differing in karyotype. DLD-1 cells show microsatellite instability (MIN) but proliferate in a near-diploid state [Lengauer et al., 1997]. As DLD-1 cells are deficient in p53, tetraploid derivatives can readily be established through inhibition of cytokinesis [Drosopoulos et al., 2014]. This afforded a syngeneic pair of stable diploid and tetraploid cells (Figure 1A). Starting with a culture of tetraploid DLD-1 cells, we then used single cell fluorescence-activated sorting (FACS) to isolate spontaneously arising aneuploid descendants. This provided us with four different PTA clones, specifically three near-triploid lines and one near-tetraploid line (Figure 1B). Finally, we applied microcell-mediated chromosome transfer [Stingele et al., 2012] to the parental diploid DLD-1 culture and obtained two viable trisomic clones carrying three copies of chromosome 7 (Tr 7) (Figure 1B). For all cell lines, DNA content was confirmed by chromosome counting (Figure 1C) and chromosome painting (Supplemental Figure S2A). This collection of isogenic cell lines set the stage for analyzing chromosomally stable diploid,

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https://doi.org/10.1038/s41388-018-0276-2

ARTICLE

Epigenetic silencing of miR-483-3p promotes acquired gefitinib resistance and EMT in EGFR-mutant NSCLC by targeting integrin β 3

Jinnan Yue¹ · Dacheng Lv¹ · Caiyun Wang¹ · Ling Li¹ · Qingnan Zhao¹ · Hongzhuan Chen¹ · Lu Xu¹

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Abstract

All lung cancers patients with epidermal growth factor receptor (EGFR) mutation inevitably develop acquired resistance to EGFR tyrosine kinase inhibitors (TKI). In up to 30% of cases, the mechanism underlying acquired resistance remains unknown. MicroRNAs (miRNAs) is a group of small non-coding RNAs commonly dysregulated in human cancers and have been implicated in therapy resistance. The aim of this study was to understand the roles of novel miRNAs in acquired EGFR TKI resistance in EGFR-mutant non-small cell lung cancer (NSCLC). Here, we reported the evidence of miR-483-3p silencing and epithelial-to-mesenchymal transition (EMT) phenotype in both in vitro and in vivo EGFR-mutant NSCLC models with acquired resistance to gefitinib. In those tumor models, forced expression of miR-483-3p efficiently increased sensitivity of gefitinib-resistant lung cancer cells to gefitinib by inhibiting proliferation and promoting apoptosis. Moreover, miR-483-3p reversed EMT and inhibited migration, invasion, and metastasis of gefitinib-resistant lung cancer cells. Mechanistically, miR-483-3p directly targeted integrin β 3, and thus repressed downstream FAK/Erk signaling pathway. Furthermore, the silencing of miR-483-3p in gefitinib-resistant lung cancer cells was due to hypermethylation of its own promoter. Taken together, our data identify miR-483-3p as a promising target for combination therapy to overcome acquired EGFR TKI resistance in EGFR-mutant NSCLC.

INTRODUCTION

EGFR tyrosine kinase inhibitors (TKI) including gefitinib and erlotinib have demonstrated dramatic efficacy in non-small cell lung cancer (NSCLC) patients with EGFR-activating mutation [1]. In general, activating EGFR mutations are more commonly observed in non-smoking, female, Asian patients with adenocarcinoma histology, which is one of the most common histological subtypes of NSCLC. Despite impressive initial response, almost all

patients eventually have a relapse due to the occurrence of acquired resistance. Several mechanisms leading to acquired resistance have been demonstrated, including EGFR T790M mutation, MET amplification, PIK3CA mutation, AXL activation, small cell lung cancer (SCLC) transformation, or acquiring an epithelial-to-mesenchymal transition (EMT) phenotype [2–7]. To note, these mechanisms of acquired resistance can take place together in different subclones of the same tumor at the same time. However, the mechanisms remain unknown in ~ 30% of cases.

MicroRNAs (miRNA) are a class of small non-coding, endogenous RNAs of 21–25 nucleotides in length, which repress target genes expression by directly binding to the 3'-untranslated region (UTR) of target gene mRNAs and

These authors contributed equally: Jinnan Yue and Dacheng Lv

Electronic supplementary material The online version of this article (<https://doi.org/10.1038/s41388-018-0276-2>) contains supplementary material, which is available at <https://doi.org/10.1038/s41388-018-0276-2>.

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Oncogene volume 37, issue 31, P4300-4312 2018 DOI: [10.1038/s41388-018-0276-2](https://doi.org/10.1038/s41388-018-0276-2)

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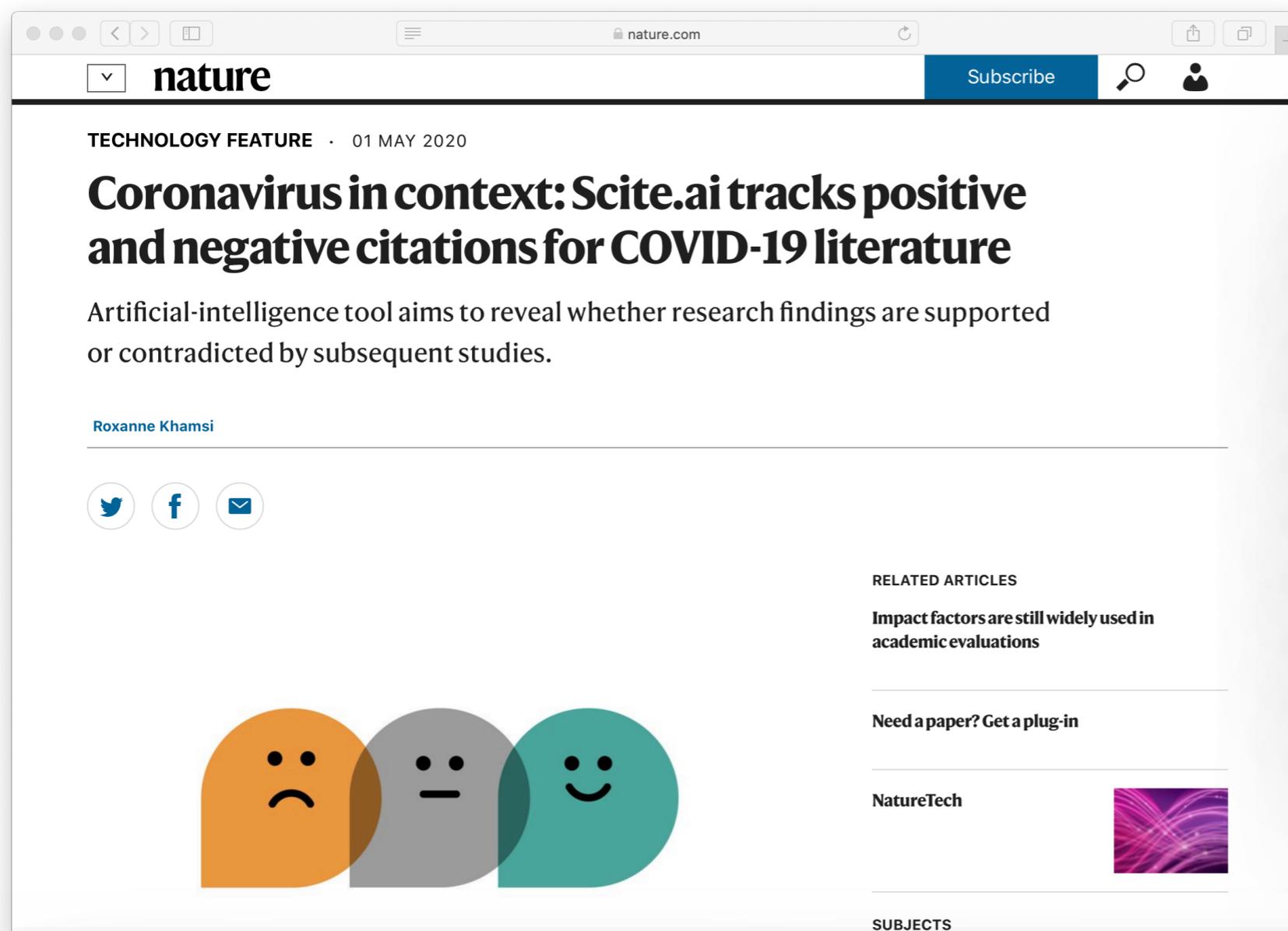
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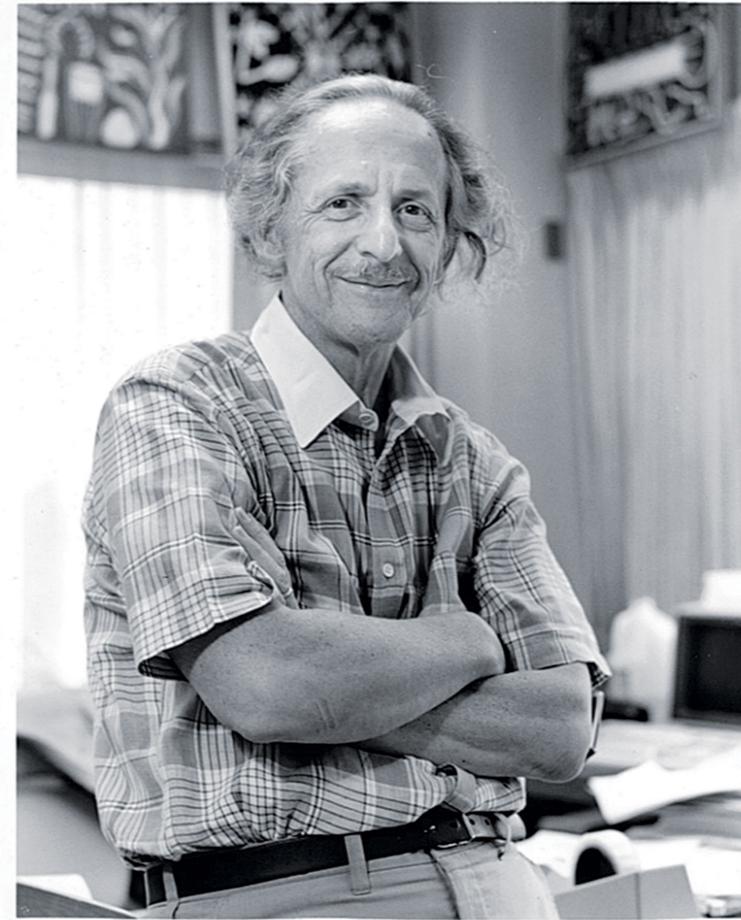
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