REGIS UNIVERSITY

Department of Biology Presentation in Honor of Dr. Joan L. Betz

Monday, April 14th at 5:00pm

Pomponio Science 212

Childhood

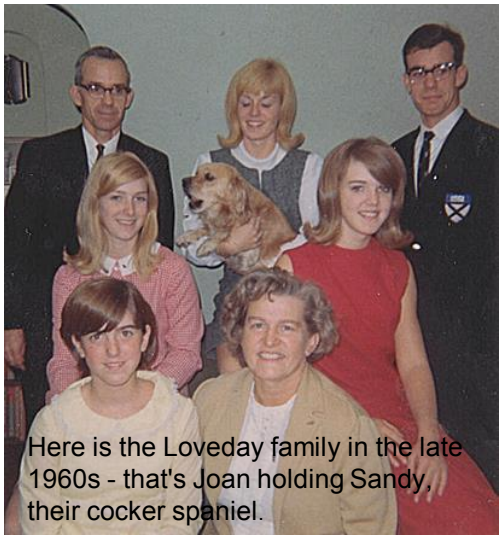
Joan grew up in Murrysville, Pennsylvania, the oldest of five children. Here is a picture of the entire family. Ken, Sharon, and Laurie are here today. Glenn, Marian and sister Wendy are surely here in spirit.
Their father Glenn was a metallurgist with U.S. Steel. Their Mother Marian was a high school math teacher.
Education was paramount, and the parents scrimped and saved for private liberal arts colleges - to which all five children attended. They collectively hold 7 advanced degrees.
Joan's love of biology nurtured on many family camping trips

High School


EXAMINES SPECIMENS — William Waskoskie, head of the science department at Franklin Area High School, examines specimens of hammerhead sharks which were part of a collection given by Joan Loveday [illegible], Miss Loveday, a senior, made the collection [illegible] study course last summer at Gulf Coast Resea[illegible] Sprin[illegible] Mi[illegible]

During high school, it was the Sputnik era, and Joan took accelerated math and science classes, and participated in a Saturday science enrichment program. Here she is with two other science nerds.
She was awarded a National Science Foundation summer Marine Biology fellowship to study near Biloxi, Miss. She gave her collection of marine specimens to her school. Here she is with her biology teacher, Mr. Waskoski.
Did I mention that she was Valedictorian of her high school class, and winner of the band award? (she played the clarinet).

Oberlin


Here is the Loveday family in the late 1960s - that's Joan holding Sandy, their cocker spaniel.

Joan attended Oberlin college. Founded in 1833, it was the first coeducational college in the United States, and Joan retains close connections to the school.
She majored in Biology - nearly half of the biology majors were women, but only one in 30 science and mat faculty members was a woman.
She also found time to play bridge - she was a highly skilled player, and taught me how to play later in the sixties.
Did I mention that she graduated Magna cum Laude and Phi Beta Kappa?

Yale

Joan entered graduate school in 1967 in the Department of Microbiology at Yale. Her brother was there as an undergraduate. Joan could not have applied for undergraduate study at Yale, because it did not admit women until 1969. Not that Yale is academically better than Oberlin, but I think Joan drew some satisfaction seeing both of her daughters doing what she was not allowed to do - they graduated from Yale in 1995 and 1999.

At her graduate school interview at Yale, Joan was asked (by one of nine male faculty members in the department of 10), “Will you get a PhD or get married and quit?”
She was also told to "dress professionally," which meant no jeans.

While at Yale, she met this young man, and they married in 1968.
Here she is in June of 1969, supported by her brother Ken and husband Bill. We all three graduated from Yale on the same day, with 3 different degrees - bachelors, masters, and doctorate.

University College London

Joan transferred to University College London finish her PhD, having been accepted to work with Dr. Patricia Clarke. For British women, things weren't much better than in the U.S. - a graduate student’s stipend would be cut in half if she married.
Pat Clarke, her advisor, was only the 30th woman to be elected to the Royal Society (there have been more than 8000 fellows since its founding in 1660). Pat said, "I'm one of the fellows now.“
Here they are in the 1980s during one of Joan's return visits.
Joan's thesis - awarded in 1972 - dealt with the concept of evolution in action. Joan made and characterized mutants of the bacterium, pseudomonas aeruginosa (an important pathogen). It made an enzyme that cleaved a molecule into two parts that it could then metabolize. Joan isolated mutant bacteria that could cleave much bigger molecules, and they actually lost the ability to cleave the original - truly seeing "evolution in action."

Presentation by Bill Betz

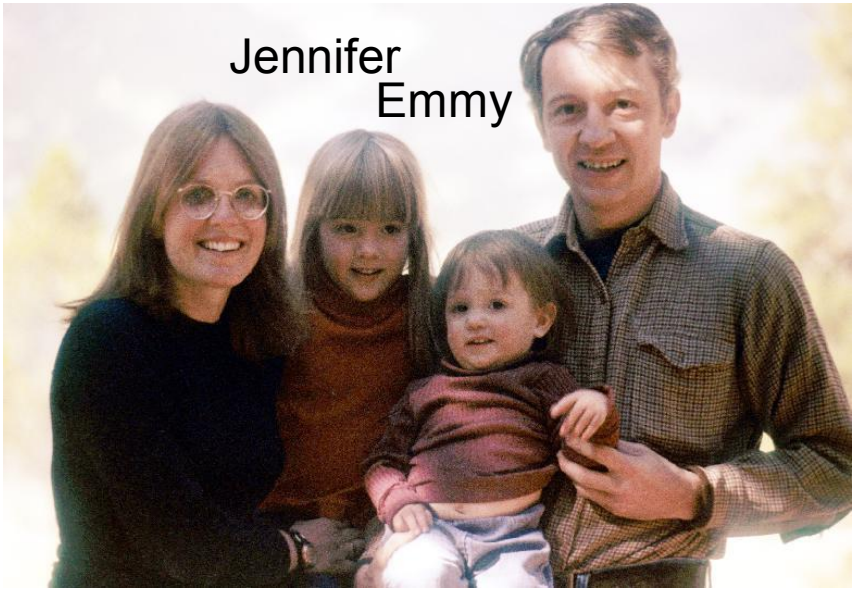
1972 Denver, Colorado

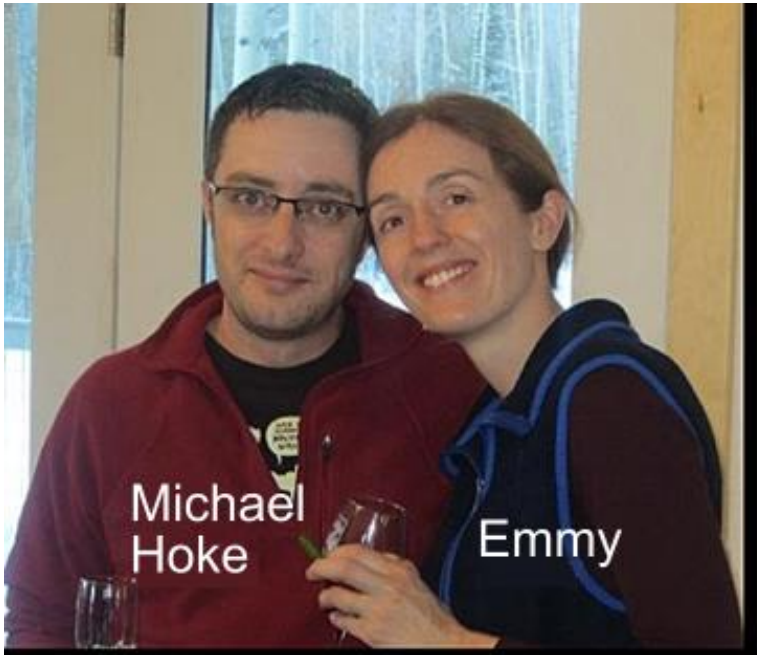
In an interview in U.S. for a postdoctoral position, she was asked again, "how will you manage children and a scientific career?" At that time, only about ten percent of attendees at scientific meetings were women.

In 1972 we moved to Denver and began work at C.U. Medical School. After several months, Joan flew back briefly to London for her thesis defense. The touch of nausea she felt the morning of her defense might have been due to the jitters - a thesis defense in England was no rubber stamp, like it typically is here. It also might have been due to her being three months pregnant with Jennifer.

Joan worked at C.U. Medical School with Jack Sadler and Lew Pizer, and later with Judith Jaehning and Bob Sclafani. Judith and Bob will describe those years.

Before turning over to Bob, I want to jump ahead and describe a few bits from Joan's personal life in Colorado. For us, one thing led to another, and our two daughters - Jennifer and Emmy - appeared on the scene. As I mentioned, they attended Yale College, and are now practicing physicians. They met and married two wonderful men - Dan Liptzin and Michael Hoke - and Joan and I are indescribably happy that they have chosen to live and work in Denver.





For them, like us, one thing led to another, and our four grandchildren - Matilda and Thalia Hoke, and Zachary and Clara Liptzin - push our indescribable happiness to even higher levels.

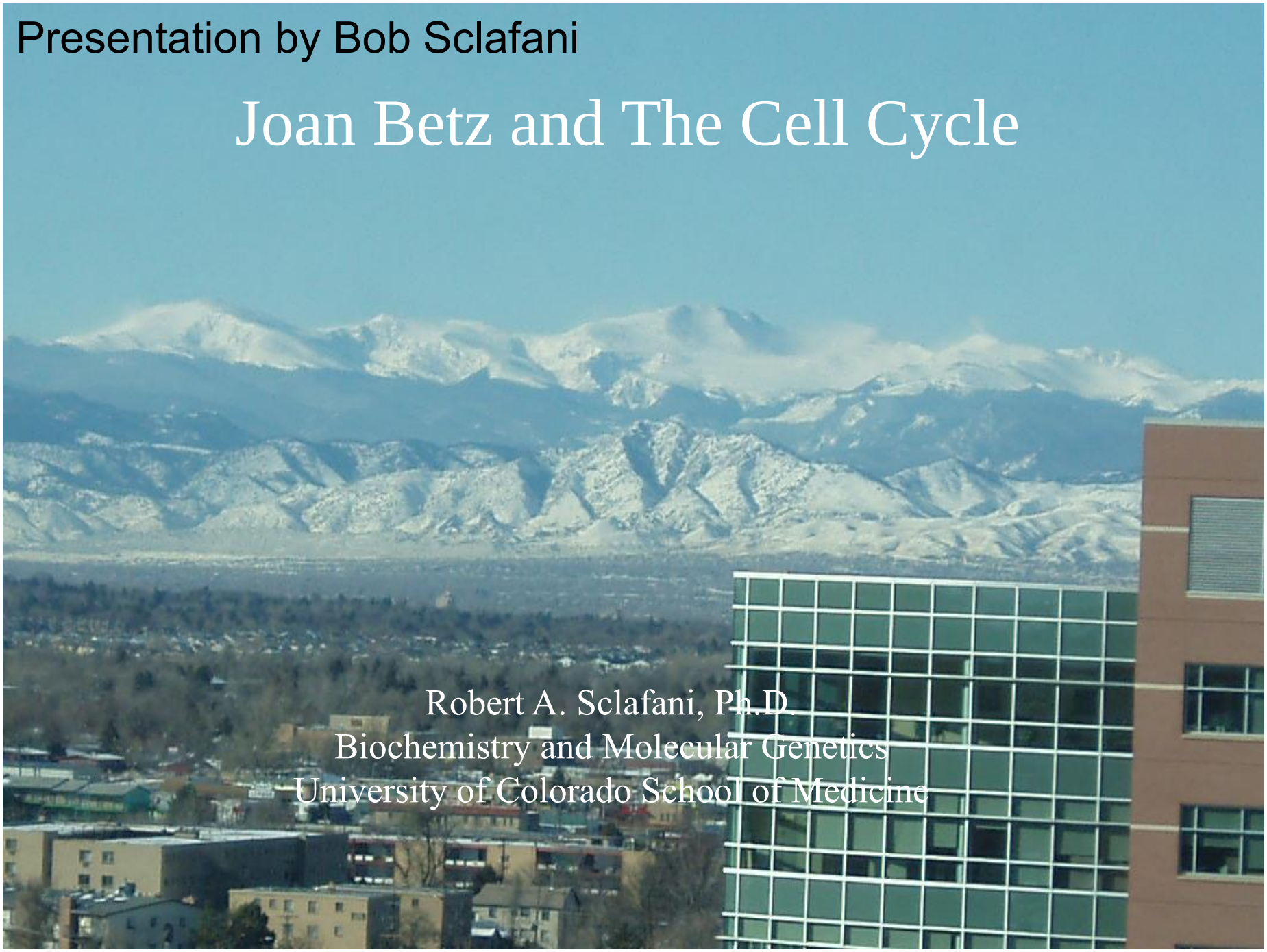
And finally, while all her family are glad that we will be able to see more of Joan in her retirement, there is one who will be most happy of all...

Presentation by Bill Betz

Zippy

Presentation by Bob Sclafani

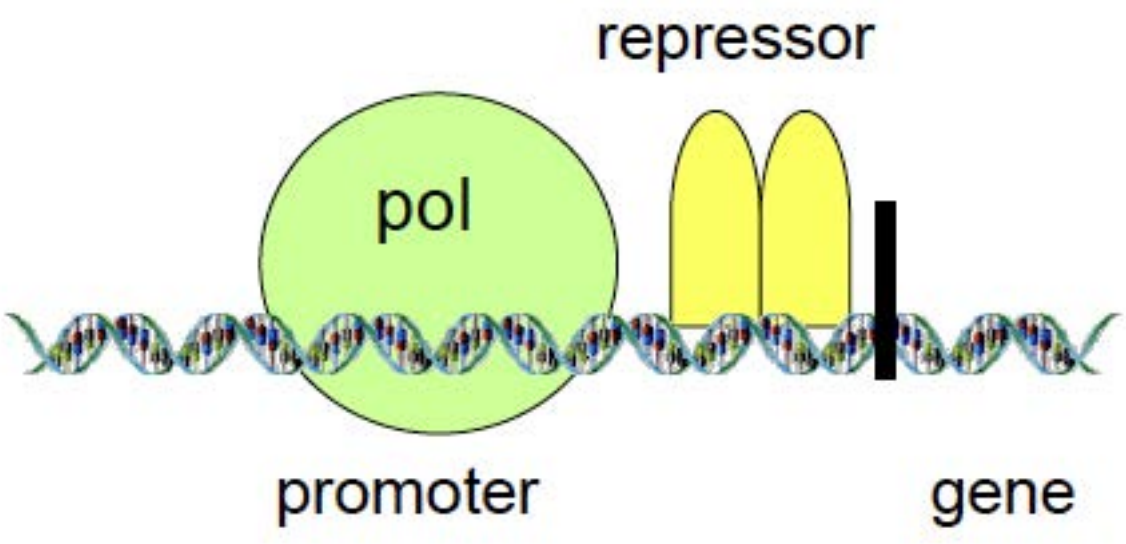
Joan Betz and The Cell Cycle

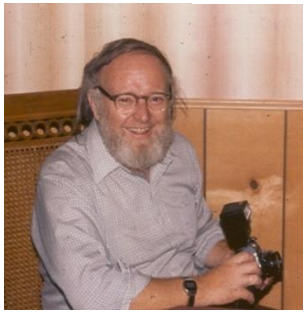


Robert A. Sclafani, Ph.D.
Biochemistry and Molecular Genetics
University of Colorado School of Medicine

Presentation by Bob Sclafani

Gene Transcription Studies on Classic Lac Operon of E. Coli





Postdoc with Jack Sadler-Dept of Biophysics at UCHSC; 1972-1977

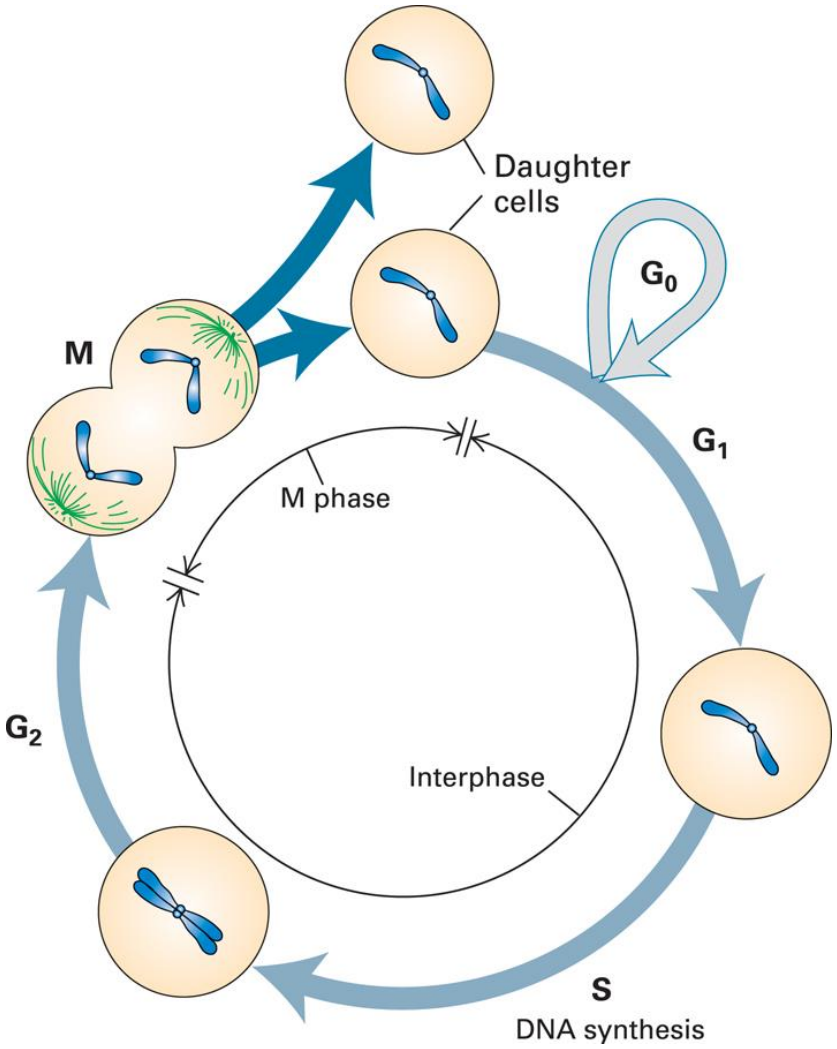
Joan Continues Focus on Gene Transcriptional Regulation

Lew Pizer at UCHSC Herpes Virus Regulation; 1980-1981

EMBL Heidelberg, Germany 1993-1994; 1997-1998 EMBL KUN (Katholieke Univ Nijmegen) in Holland; with Henk Stunnenberg; RAR receptors in Leukemia

Presentation by Bob Sclafani

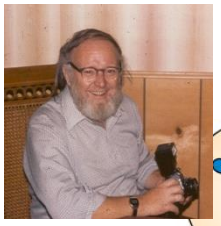
The Cell Cycle



Lodish et al., *Molecular Cell Biology* 7th Edition (2013)

Joan Betz-Bob Sclafani Cycle

Joan's Postdoc Advisor Jack Sadler dies in 1983



Bob and Joan Meet in Biochemistry, Biophysics and Genetics 1985
Establish Grad Course

Joan to Regis 1990

Joan Returns to Work with Bob on Yeast DNA Replication 2009

Joan Teams up with Judith Jaehning in 1998

Joan Studies Yeast Transcription

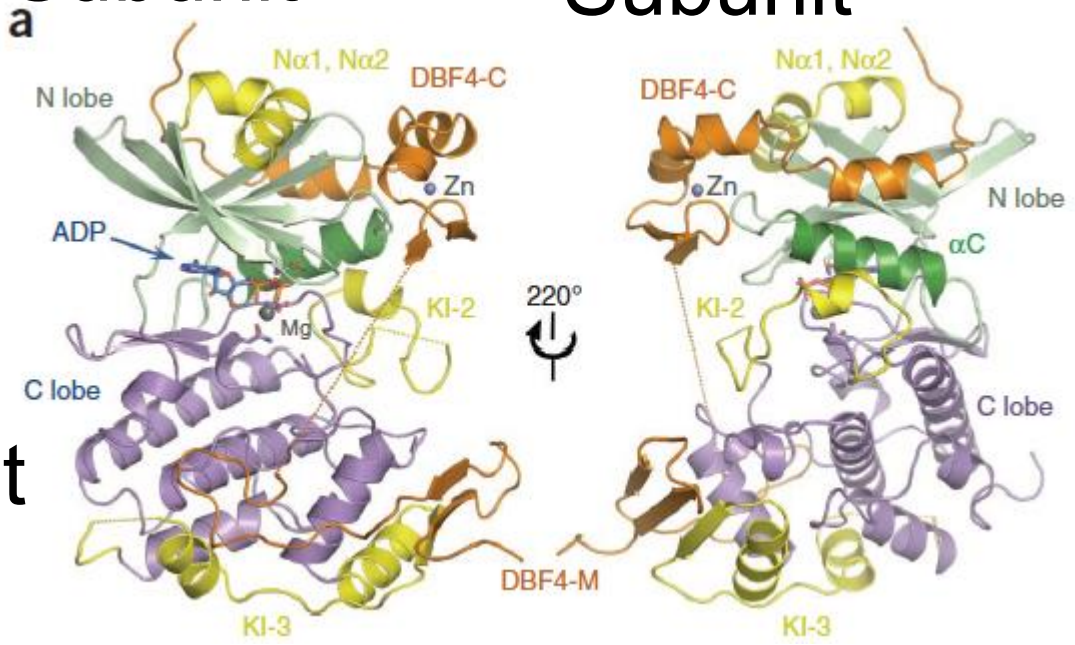
DDK=Cdc7 plus Dbf4

Cdc7

Kinase Subunit

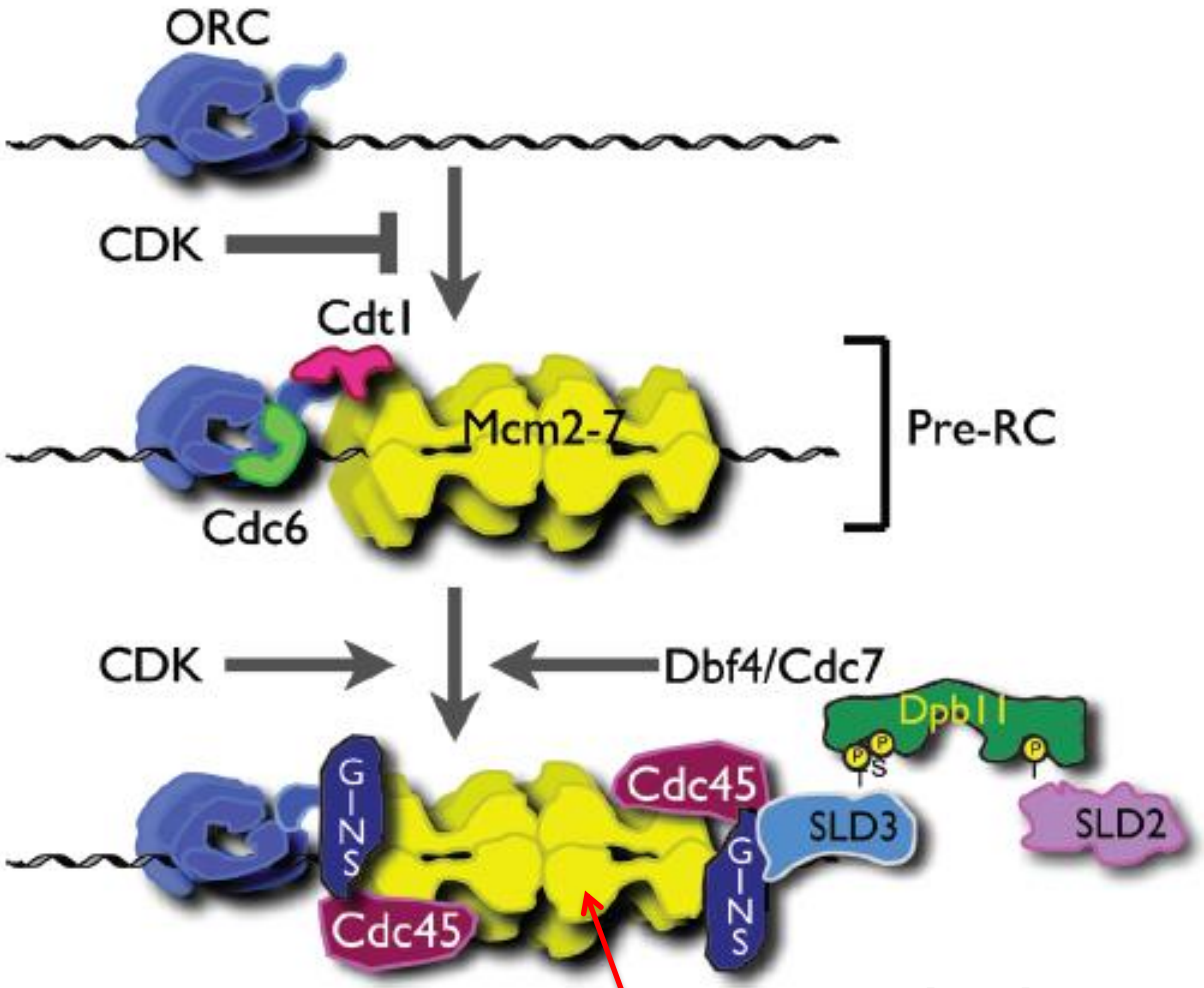
Dbf4

Regulatory Subunit



Hughes et al., 2012

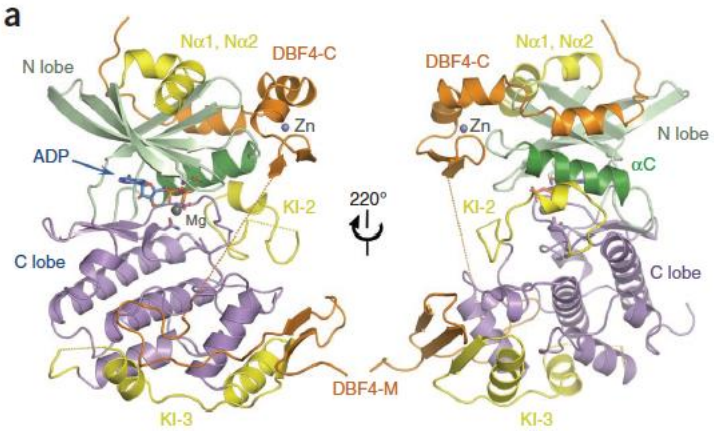
DDK activates MCM helicase by loading Cdc45 onto chromatin



J. Diffley, CSHSQB 2011

mcm5-bob1 mutation bypasses DDK

DDK regulates chromatin loading



MCM	Rev7	Mer2
Cdc45 loads	Polζ loads	Spo11 loads
Replication	TLS	Recombination

Presentation by Bob Sclafani

Happy Retirement, Joan!

From left to right: Bob Sclafani, Dan Rossbach, Rebecca Ferguson, Sunitha Siriwardana and Joan Betz.
(not shown) Josh Ramey and Jim Dutchik

Presentation by Judith Jaehning

Stalking Joan Betz: My quest to have Joan join my lab.

Despite her very full schedule, Joan was always happiest working in the lab.

We met at the Molecular Biology Retreat in 1994. I spent the next several years trying to turn her into a yeast person.

I finally convinced her to join the lab.
Only took me 4 years!

Joan Betz Mei-Ping Chang Stephanie Porter
1998-2006 1993-1999 1999-2004

Presentation by Judith Jaehning

Attending National and International Meetings

Abstract Submitted to Yeast 2004 Meeting

Presentation Type: Poster
Research Topic: *Transcription*

Abstract Reference Number: 0159

Over-expression of Cln2 or Cln3 suppresses loss of the Paf1/RNA polymerase II complex.

Joan L. Betz (1), Bao T. Nguyen (1), Judith A. Jaehning (2)
(1) Biology, Regis University, 3333 Regis Blvd., Denver, CO, 80221, USA; (2) Department of Biochemistry and Molecular Genetics, University of Colorado HSC, Denver

The Paf1 complex associated with yeast RNA polymerase II (pol II) defines a form distinct from the Srb/Mediator holoenzyme, minimally containing Paf1, Ctr9, Rtf1, Leo1 and Cdc73. Mutation of Paf1 factors is not lethal, but causes pleiotropic phenotypes including slow growth, sensitivity to temperature and caffeine, and alterations in cell-cycle regulated genes. Deletion mutants exhibit distinctive patterns of sensitivity to compounds affecting cell wall biosynthesis, and nucleic acid metabolism. Loss of Paf1 or Ctr9 results in identical, severe, phenotypes, and loss of either protein results in loss of the other Paf1 factors. In contrast, loss of Rtf1 results in modest phenotypes, does not cause loss of other Paf1 factors, but does cause disassociation of these factors from pol II. To identify downstream targets of the Paf1 complex, we tested genes known to be involved in some of the altered metabolic pathways, and also selected high-copy suppressors from a yeast library. We found that *RNR1* suppressed sensitivity to hydroxyurea, and over-expression of *PAF1* or *CTR9* partially compensated for loss of the other component. The strongest suppressors of a *paf1 ctr9* double mutant contained the G1/S-phase cyclins *CLN2* or *CLN3*. Expression of *CLN2*, but not *CLN3*, is reduced in a *paf1* mutant. Over-expression of either cyclin partially suppressed phenotypes of *paf1* or *ctr9* including the enlarged cell phenotype. We are currently investigating the molecular basis of this suppression.

This abstract was just submitted for review.

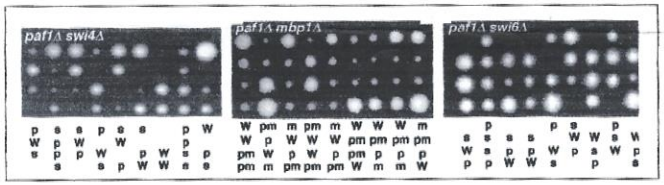
Presentation by Judith Jaehning

Bringing Regis Undergrads to work in the lab.

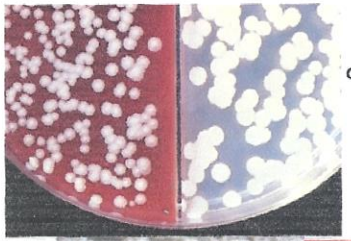
HONORS-IN-BIOLOGY THESIS DEFENSE

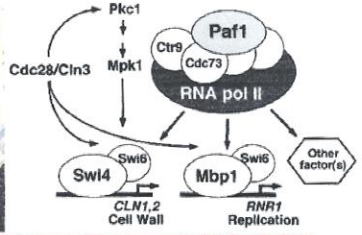
Characterization of Suppressors of Deletion Mutants of the Paf1 Complex Associated with RNA Polymerase II in *Saccharomyces cerevisiae*

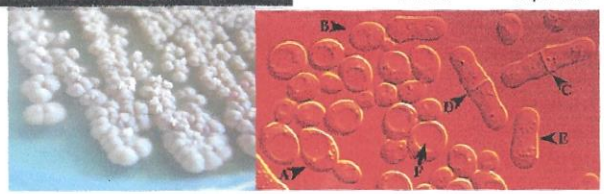
Robyn Nguyen
Regis University



In partial fulfillment of the requirements of the Honors-in-Biology Major.







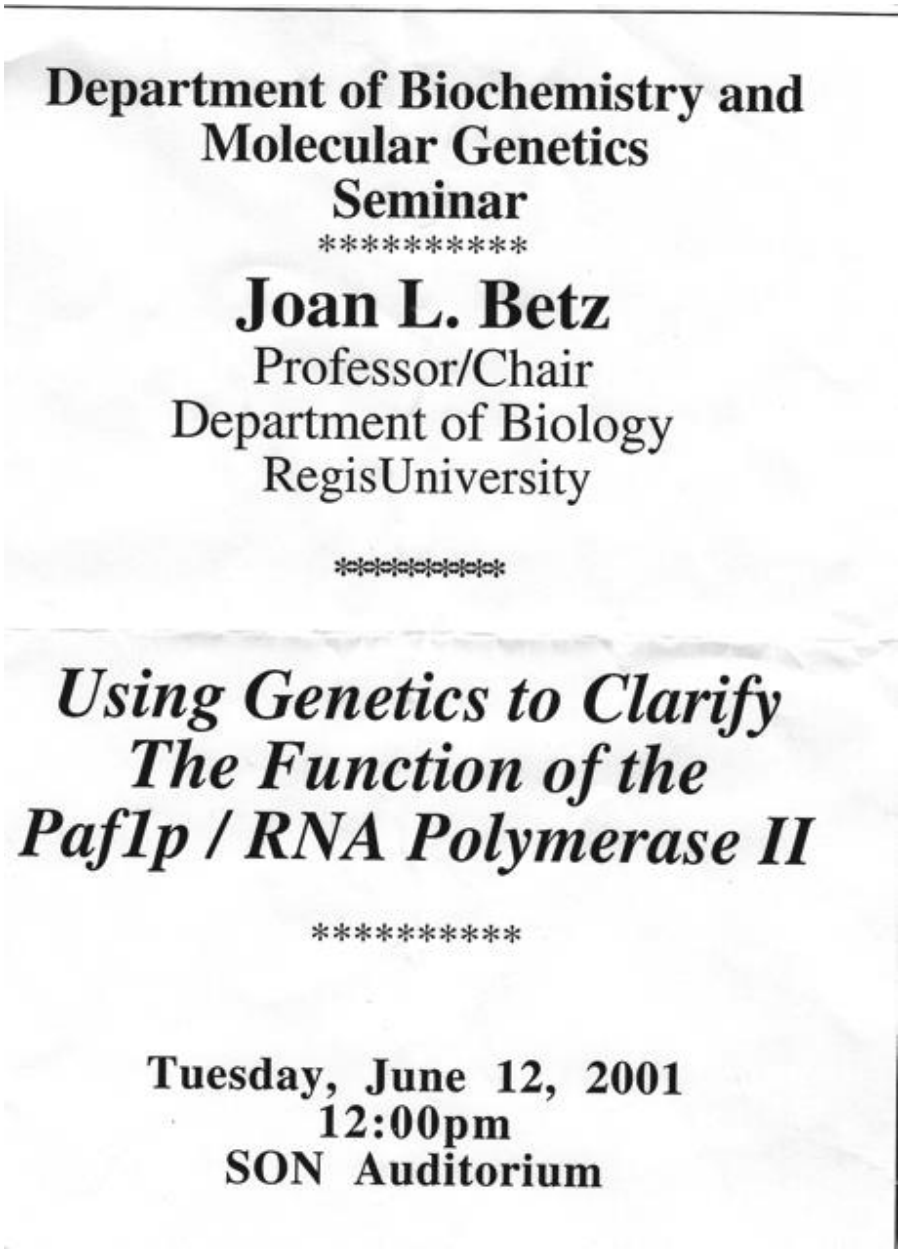
2:00-2:50 PM Tuesday April 20th

Science Building Room 312

Refreshments provided at reception following defense (SCI 210).

Presentation by Judith Jaehning

Participating in the CU BMG Department



Department of Biochemistry and Molecular Genetics Seminar

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Joan L. Betz

Professor/Chair
Department of Biology
RegisUniversity

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Using Genetics to Clarify The Function of the Paf1p / RNA Polymerase II

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Tuesday, June 12, 2001
12:00pm
SON Auditorium

Presentation by Judith Jaehning

Publications

ORIGINAL PAPER

J.L. Betz · M. Chang · T.M. Washburn · S.E. Porter
C.L. Mueller · J.A. Jaehning

Phenotypic analysis of Paf1/RNA polymerase II complex mutations reveals connections to cell cycle regulation, protein synthesis, and lipid and nucleic acid metabolism

Received: 28 April 2002 / Accepted: 15 August 2002 / Published online: 12 September 2002

Abstract Paf1 is an RNA polymerase II-associated protein in yeast, which defines a complex that is distinct from the Srb/Mediator holoenzyme. The Paf1 complex, which also contains Ctr9, Cdc73, Hpr1, Ccr4, Rtf1 and Leo1, is required for full expression of a subset of yeast genes, particularly those responsive to signals from the Pkc1/MAP kinase cascade. We have extensively characterized the pleiotropic phenotypes of deletion mutants for factors present in the Paf1 complex, identifying more than a dozen new phenotypes, and, in some cases, establishing possible molecular explanations for the growth defects. For example, *paf1*Δ causes sensitivity to hydroxyurea; this phenotype correlates with a reduction in *RNR1* transcript abundance and is suppressed by over-expression of *RNR1*. In contrast, the resistance of *paf1*Δ cells to the transcription elongation inhibitors 6-azauracil and mycophenolic acid correlates with its ability to derepress the *IMD2* transcript. We tested the hypothesis that Paf1 communicates with some promoters through the DNA-binding factors Swi4, Mbp1 or Rlm1. The phenotypes of mutations in Paf1 complex components are exacerbated in the *swi4*Δ background, suggesting that the complex acts in a pathway parallel to that controlled by Swi4. Conversely, the fact that *mbp1*Δ and *rlm1*Δ mutations do not enhance the phenoty suggests that the Paf1 complex may function in the sa regulatory pathway(s) with Mbp1 and Rlm1.

Keywords Regulation of transcription · Paf1 complex · Swi4 · Mbp1 · RNA polymerase II

Introduction

Regulation of eukaryotic mRNA transcription requi various cofactors to communicate between DNA-bi ing activators or repressors and RNA polymerase (pol II) and the general transcription factors (GTI which include TBP, TFIIB, TFIIE, TFIIF and TFI (reviewed by Lee and Young 2000). These cofactors clude the TAFs that associate with TBP to form TFIID complex (for review, see Naar et al. 2001), a the SRB and Mediator proteins that bind to pol II form the holoenzyme (Kim et al. 1994; Koleske a Young 1994; Chang and Jaehning 1997; Naar et 2001), as well as coactivator and corepressor comple whose associated enzymatic activities modulate and model chromatin structure (Lee and Young 2000; Na et al. 2001). Intricate regulatory patterns of eukaryo

Presentation by Judith Jaehning

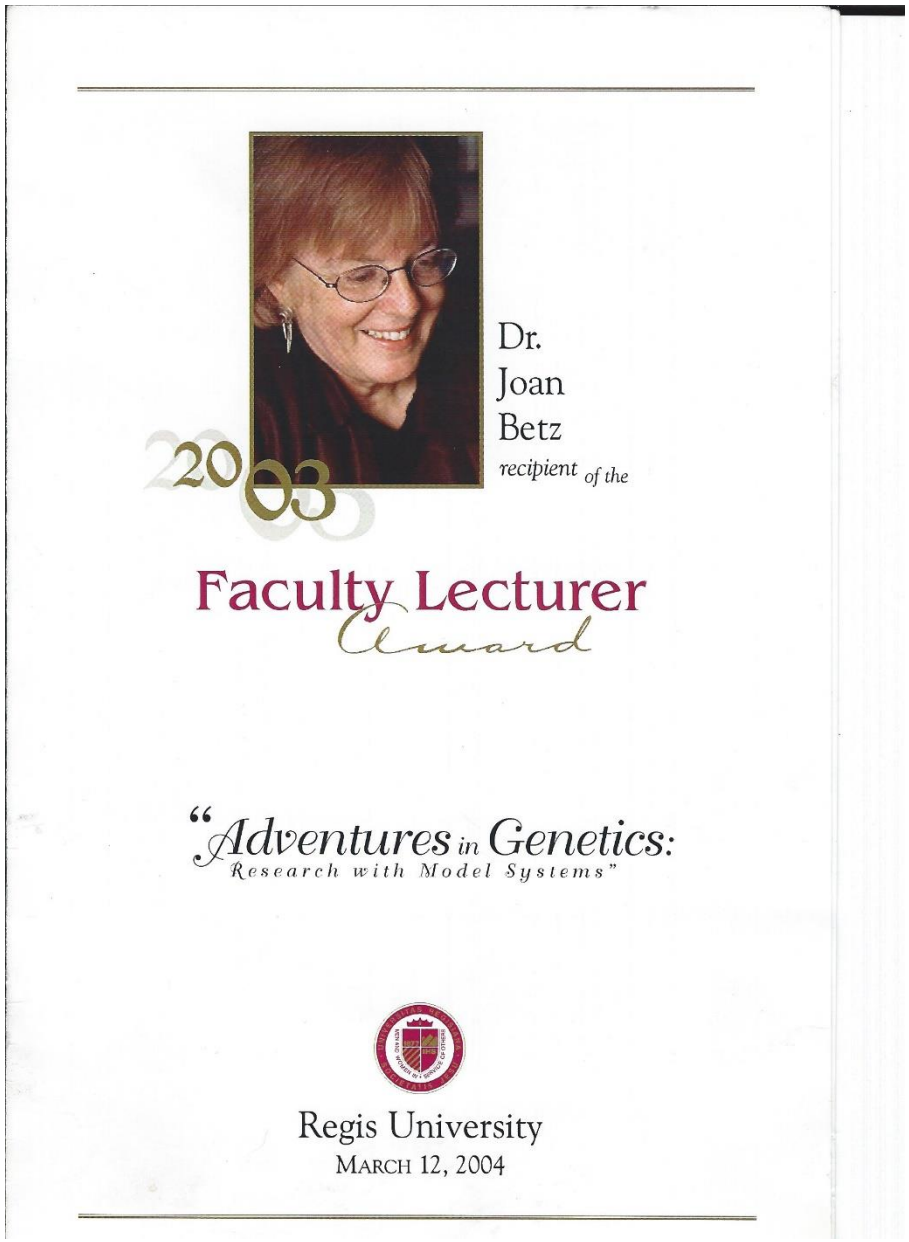
Formidable Organizational Skills!

Table 1. *Saccharomyces cerevisiae* strains used

Strain	Genotype
YJJ576	*MATa leu2Δ1 his3Δ200 ura3-52 paf1Δ::HIS3*
YJJ577	*MATα leu2Δ1 his3Δ200 ura3-52 paf1Δ::HIS3*
YJJ662	*MATa leu2Δ1 his3Δ200 ura3-52*
YJJ664	*MATa leu2Δ1 his3Δ200 ura3-52 paf1Δ::HIS3*
YJJ665	*MATa leu2Δ1 his3Δ200 ura3-52 cdc73Δ::HIS3*
YJJ681	*MATα leu2Δ1 his3Δ200 ura3-52 cdc73Δ::HIS3*
YJJ755	*MATa bar1 his6 his7 leu2 ura3 pep4 prb1 trp1*
YJJ756	*MATa bar1 his6 his7 leu2 ura3 pep4 prb1 trp1 paf1Δ::TRP1*
YJJ879	*MATa leu2Δ1 his3Δ200 ura3-52 ccr4Δ::URA3*
YJJ898	*MATa leu2Δ1 his3Δ200 ura3-52 hpr1Δ::HIS3*
YJJ899	*MATα leu2Δ1 his3Δ200 ura3-52 hpr1Δ::HIS3*
YJJ932	*MATα leu2Δ1 his3Δ200 ura3-52 ccr4Δ::URA3*
YJJ935	*MATα leu2Δ1 his3Δ200 ura3-52 srb5Δ::URA3*
YJJ940	*MATα leu2Δ1 his3Δ200 ura3-52 hpr1Δ::HIS3 srb5Δ::URA3*
YJJ958	*MATa leu2Δ1 his3Δ200 ura3-52 srb5Δ::HIS3*
YJJ962	*MATa leu2Δ1 his3Δ200 ura3-52 cdc73Δ::LEU2 hpr1Δ::HIS3*
YJJ1000	*MATa leu2Δ1 his3Δ200 ura3-52 swi4Δ::URA3*
YJJ1001	*MATα leu2Δ1 his3Δ200 ura3-52 swi4Δ::URA3*
YJJ1035	*MATa/α leu2Δ1/leu2Δ1 his3Δ200/his3Δ200 ura3-52/ura3-52 PAF1/paf1Δ::HIS3*
YJJ1065	*MATα leu2Δ1 his3Δ200 ura3-52 rlm1Δ::kanMX4*
YJJ1068	*MATa leu2Δ1 his3Δ200 ura3-52 mbp1Δ::kanMX4*
YJJ1070	*MATα leu2Δ1 his3Δ200 ura3-52 paf1Δ::HIS3 mbp1Δ::kanMX4*
YJJ1098	*MATa leu2Δ1 his3Δ200 ura3-52 ccr4Δ::URA3 mbp1Δ::kanMX4*
YJJ1100	*MATα leu2Δ1 his3Δ200 ura3-52 ccr4Δ::URA3 rlm1Δ::kanMX4*
YJJ1104	*MATα leu2Δ1 his3Δ200 ura3-52 cdc73Δ::HIS3 rlm1Δ::kanMX4*
YJJ1106	*MATα leu2Δ1 his3Δ200 ura3-52 cdc73Δ::URA3 mbp1Δ::kanMX4*
YJJ1119	*MATα leu2Δ1 his3Δ200 ura3-52 srb5Δ::HIS3 rlm1Δ::kanMX4*
YJJ1123	*MATα leu2Δ1 his3Δ200 ura3-52 hpr1Δ::HIS3 mbp1Δ::kanMX4*
YJJ1131	*MATα leu2Δ1 his3Δ200 ura3-52 swi4Δ::URA3 rlm1Δ::kanMX4*
YJJ1142	*MATa leu2Δ1 his3Δ200 ura3-52 paf1Δ::HIS3 rlm1Δ::kanMX4*
YJJ1149	*MATα leu2Δ1 his3Δ200 ura3-52 hpr1Δ::HIS3 rlm1Δ::kanMX4*
YJJ1150	*MATa leu2Δ1 his3Δ200 ura3-52 cdc73Δ::HIS3 BIK1::URA3*
YJJ1151	*MATa leu2Δ1 his3Δ200 ura3-52 hpr1Δ::HIS3 BIK1::URA3*
YJJ1152	*MATa leu2Δ1 his3Δ200 ura3-52 BIK1::URA3*
YJJ1153	*MATa leu2Δ1 his3Δ200 ura3-52 paf1Δ::HIS3 BIK1::URA3*
YJJ1160	*MATa leu2Δ1 his3Δ200 ura3-52 srb5Δ::HIS3 mbp1Δ::kanMX4*
YJJ1165	*MATa leu2Δ1 his3Δ200 ura3-52 mbp1Δ::kanMX4 rlm1Δ::kanMX4*
YJJ1199	*MATa leu2Δ1 his3Δ200 ura3-52 ctr9Δ::kanMX4*
YJJ1202	*MATα leu2Δ1 his3Δ200 ura3-52 paf1Δ::HIS3 ctr9Δ::kanMX4*
YJJ1208	*MATa leu2Δ1 his3Δ200 ura3-52 rpb9Δ::kanMX4*
YJJ1209	*MATα leu2Δ1 his3Δ200 ura3-52 rpb9Δ::kanMX4*
YJJ1225	*MATa leu2Δ1 his3Δ200 ura3-52 paf1Δ::kanMX4*
YJJ1227	*MATa leu2Δ1 his3Δ200 ura3-52 rpb9Δ::kanMX4 hpr1Δ::HIS3*
YJJ1229	*MATα leu2Δ1 his3Δ200 ura3-52 rpb9Δ::kanMX4 cdc73Δ::HIS3*
YJJ1232	*MATa leu2Δ1 his3Δ200 ura3-52 swi4Δ::kanMX4*
YJJ1234	*MATα leu2Δ1 his3Δ200 ura3-52 swi4Δ::kanMX4*
YJJ1252	*MATa leu2Δ1 his4-912δ lys2-128δ*
YJJ1254	*MATα leu2Δ his4-912δ lys2-128δ ura3Δ paf1Δ::URA3*
YJJ1256	*MATa leu2Δ1 his3Δ200 ura3-52 rpb9Δ::kanMX4 BIK1::URA3*
YJJ1257	*MATa leu2Δ1 his3Δ200 ura3-52 rlm1Δ::kanMX4 BIK1::URA3*
YJJ1259	*MATa leu2Δ1 his3Δ200 ura3-52 swi4Δ::kanMX4 BIK1::URA3*
YJJ1260	*MATα leu2Δ1 his3Δ200 ura3-52 srb5Δ::URA3 swi4Δ::kanMX4*
YJJ1299	*MATa leu2Δ1 his3Δ200 ura3-52 swi4Δ::kanMX4 cdc73Δ::HIS3*
YJJ1301	*MATα leu2Δ1 his3Δ200 ura3-52 hpr1Δ::HIS3 swi4Δ::kanMX4*
YJJ1303	*MATa leu2Δ1 his3Δ200 ura3-52 rtf1Δ::kanMX4*
YJJ1305	*MATa leu2Δ1 his3Δ200 ura3-52 rtf1Δ::kanMX4 BIK1::URA3*
YJJ1324	*MATα leu2Δ1 his3Δ200 ura3-52 cdc73Δ::HIS3 paf1Δ::kanMX4*
YJJ1326	*MATa leu2Δ1 his3Δ200 ura3-52 rtf1Δ::kanMX4 paf1Δ::HIS3*
YJJ1328	*MATα leu2Δ1 his3Δ200 ura3-52 ctr9Δ::kanMX4*
YJJ1335	*MATa leu2Δ1 his3Δ200 ura3-52 leo1Δ::kanMX4 BIK1::URA3*
YJJ1336	*MATα leu2Δ1 his3Δ200 ura3-52 leo1Δ::kanMX4*
YJJ1345	*MATa leu2Δ1 his3Δ200 ura3-52 rtf1Δ::kanMX4cdc73Δ::HIS3*
YJJ1346	*MATa leu2Δ1 his3Δ200 ura3-52 rtf1Δ::kanMX4 hpr1Δ::HIS3*
YJJ1359	*MATα leu2Δ1 his3Δ200 ura3-52 cdc73Δ::HIS3 srb5Δ::URA3*
YJJ1361	*MATa leu2Δ1 his3Δ200 ura3-52 leo1Δ::kanMX4 paf1Δ::HIS3*
YJJ1362	*MATa leu2Δ1 his3Δ200 ura3-52 rtf1Δ::kanMX4 srb5Δ::URA3*
YJJ1365	*MATa leu2Δ1 his3Δ200 ura3-52 rtf1Δ::kanMX4 ctr9Δ::URA3*
YJJ1403	*MATa leu2Δ1 his4-912δ lys2-128δ paf1Δ::kanMX4*
YJJ1462	*MATα leu2Δ1 his3Δ200 ura3-52 ctr9Δ::kanMX4 BIK1::URA3*

Presentation by Judith Jaehning

RECOGNIZED FOR HER EXTRAORDINARY CONTRIBUTIONS



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Helping to wrap up the last details.

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Direct Interactions between the Paf1 Complex and a Cleavage and Polyadenylation Factor Are Revealed by Dissociation of Paf1 from RNA Polymerase II[▽]

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The Paf1 complex (Paf1, Ctr9, Cdc73, Rtf1, and Leo1) is normally associated with RNA polymerase II (Pol II) throughout the transcription cycle. However, the loss of either Rtf1 or Cdc73 results in the detachment of the Paf1 complex from Pol II and the chromatin form of actively transcribed genes. Using functionally tagged forms of the Paf1 complex factors, we have determined that, except for the more loosely associated Rtf1, the remaining components stay stably associated with one another in an RNase-resistant complex after dissociation from Pol II and chromatin. The loss of Paf1, Ctr9, or to a lesser extent Cdc73 or Rtf1 results in reduced levels of serine 2 phosphorylation of the Pol II C-terminal domain and in increased read through of the *MAK21* polyadenylation site. We found that the cleavage and polyadenylation factor Cft1 requires the Pol II-associated form of the Paf1 complex for full levels of interaction with the serine 5-phosphorylated form of Pol II. When the Paf1 complex is dissociated from Pol II, a direct interaction between Cft1 and the Paf1 complex can be detected. These results are consistent with the Paf1 complex providing a point of contact for recruitment of 3′-end processing factors at an early point in the transcription cycle. The lack of this connection helps to explain the defects in 3′-end formation observed in the absence of Paf1.

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Thanks Joan!

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Dear Joan,
Thanks for sharing a small part of your amazing career with me. Congratulations on your retirement-I know it will be just as action packed as your working years.

One piece of advice-it took me almost 5 years after retirement to quit going in to work. Run for it now and just say no to all those requests from your colleagues!

Your Friend,
Judith