Transcription-induced Chromatin Remodeling at the c-myc Gene Involves the Local Exchange of Histone H2A.Z*

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The post-translational modification of histones and the incorporation of core histone variants play key roles in governing gene expression. Many eukaryotic genes regulate their expression by limiting the escape of RNA polymerase from promoter-proximal pause sites. Here we report that elongating RNA polymerase II complexes encounter distinct chromatin landscapes that are marked by methylation of lysine residues Lys4, Lys36, and Lys79 of histone H3. However, neither histone methylation nor acetylation directly regulates the release of elongation complexes stalled at promoter-proximal pause sites of the c-myc gene. In contrast, transcriptional activation is associated with local displacement of the histone variant H2A.Z within the transcribed region and incorporation of the major histone variant H2A. This result indicates that transcribing RNA polymerase II remodels chromatin in part through coincident displacement of H2A.Z-H2B dimers and incorporation of H2A-H2B dimers. In combination, these results suggest a new model in which the incorporation of H2A.Z into nucleosomes down-regulates transcription; at the same time it may act as a cellular memory for transcriptionally poised gene domains.

RNA pol II encounters nucleosomes and other multiprotein complexes during transcription. Conceptually, any mechanism that destabilizes the DNA-histone interaction or removes part or all of the histone octamer potentially increases the elongation efficiency of RNA polymerases. Protein complexes such as FACT, Elongator, and Swi/Snf have been proposed to alleviate barriers that are imposed by chromatin (1–3). For instance, the protein complex FACT assists RNA polymerase during elongation by altering the nucleosomal conformation and destabilizing the interaction between the H2A/H2B dimer and the (H3/H4)2 tetramer (4). After the transcription complex has passed the nucleosome, FACT participates in the reassembly of the nucleosome. In addition to the FACT-mediated catalytic remodeling of nucleosomes, the post-translational modification of histones, and/or the incorporation of histone variants also may facilitate the passage of the transcription complex through nucleosomes (5–7). For example, the identification of the acetyltransferase activity in the Elp3 subunit of the RNA pol II-associated Elongator complex suggests that histone acetylation is an important component in the regulation of transcription elongation (8, 9). Indeed, previous studies demonstrated that the yeast Elongator complex is associated with nascent transcripts (2). In addition to histone acetylation, methylation of H3 lysine residues Lys4, Lys36, and Lys79 also has been reported to play a role in transcript elongation (10, 11). In yeast, the Set1, Set2, and Dot1p histone methylation transferase complexes are recruited by the RNA polymerase complex to catalyze methylation of Lys4, Lys36, and Lys79 of histone H3 within the coding region. The recent isolation of the Lys4-specific histone demethylase LSD1 suggests a model in which RNA polymerase II elongation is regulated by the antagonistic action of histone methyltransferases and histone demethylases (12). Alternatively, a replication-independent histone exchange pathway in which variant forms of histones are incorporated during active transcription may provide an opportunity to continuously exchange histones with either hyper- or hypomethylated forms in order to stimulate or inhibit transcriptional elongation.

In combination, these results suggest that genes regulated at the level of elongation are characterized by changes in histone N-terminal tail modification and/or incorporation of histone variants. Here we have tested these predictions at the mammalian c-myc gene.

MATERIALS AND METHODS

Antibodies—Antibodies detecting the histone variant H2A.Z were a kind gift from the laboratory of David Tremethick (Australian National University, Canberra) or were purchased from Abcam. Both antibodies specifically immunoprecipitate the histone variant H2A.Z, but not the major H2A form (13) (Abcam). Methylated Lys9 (H3)-specific antibodies were generously provided by Dan Gottschling and Fred van Leeuwen (Fred Hutchinson Cancer Research Center, Seattle, WA). Antibodies specific for acetylated (Lys9,Lys14) histone H3 and pan-acetylated H4, methylated (Lys9) histone H3, and RNA polymerase II were purchased from Upstate Biotechnology. Antibodies directed against di- and trimethyl Lys9 (H3), acetylated (Lys9) histone H3, and acetyl H2B (Lys12,Lys15) were obtained from Abcam.

Cell Lines—The human promyelocytic cell line HL60 was grown in RPMI, 10% fetal calf serum. Differentiation was induced by addition of dimethyl sulfoxide (Me2S O) for 2ha t a final concentration of 1.5%. The IL-2-dependent CTLL-2 cell line was grown and induced as described (14).

Chromatin Immunoprecipitation—Chromatin immunoprecipitation (ChIP) experiments were performed as described (15). The total amount of antibody used in one chromatin immunoprecipitation was 10 μg.

PCR Analysis—DNA recovered from immunoprecipitations was analyzed by duplex PCR using gene-specific primers and reference primers (β-globin). The reactions were performed with the FailSafe™ Taq polymerase in a solution containing 1× buffer F (Epicenter) and 10 pmol of each c-myc primer in the presence of [32P]dCTP. Under these conditions, the amplification of fragments was linear. After electro-
phoresis on a native 6% acrylamide gel, the signals were quantified with the Cyclone Phosphor System. The $\frac{\text{enrichment}}{\text{input}}$ in each immunoprecipitation was determined by calculating the ratio of the signal obtained with c-myC primers to that of the reference primer divided by the ratio of signals obtained with the same primers in the input DNA reaction. Primers sequences used to detect immunoprecipitated DNA are available upon request.

**Nuclear Run-on**—Nuclear run-on experiments were performed as described (16).

**RNA Analysis**—Nuclease S1 protection assays were performed as described previously (15). Northern analysis followed the previously published protocol (14).

## RESULTS

### Histone Subdomains across Transcribed Regions of Mammalian Genes

The interaction of histone methyltransferase Set1 and Set2 with RNA pol II in yeast requires the transcription elongation complex PAF, suggesting that histone modifications may regulate pol II elongation (11, 17–19). We set out to define the distribution of the modifications mediated by these complexes across the transcribed region in several mammalian genes. To determine the pattern of histone methylations across the coding region, we used ChiPs. To maximize the resolution of the ChiP experiment, we tested the *bcl-2* and *bcl-6* oncogenes in which the transcribed portion extends over 35 and 180 kb, respectively (Fig. 1). Both genes are constitutively expressed in the human promyelocytic leukemia cell line HL60 (data not shown). Formaldehyde-cross-linked chromatin prepared from HL60 cells was immunoprecipitated with antibodies specific for methylated Lys$^4$, Lys$^{79}$, or Lys$^{36}$ of histone H3. Enrichment of specific gene regions was determined by quantitative duplex PCR in which signals obtained by primer sets were normalized to the signal obtained from the reference primer set. As shown in Fig. 1, the methylation of H3 at residues Lys$^4$, Lys$^{79}$, and Lys$^{36}$ of histone H3 is non-uniform over these genes and creates distinct regions characterized by different combinations of histone modifications. For instance, the promoter and the 5' portion of the transcribed sequences of both the *bcl-2* and *bcl-6* genes are characterized by Lys$^4$-dimethylated histone H3. However, this modification is undetectable in regions further downstream, consistent with a previous report in which dimethylation of Lys$^4$ was detected in 5'- but not 3'-regions of transcribed genes in chicken erythrocytes (20).

By contrast, methylation of lysine 79 is restricted to the transcribed region, and its intensity decreases in the direction 5'– to 3'. The decrease in the level of histone lysine 79 methylation is accompanied by an increase in the level of lysine 36 methylation. Lysine 36 methylation is undetectable in the 5'-region and peaks in the 3'-region (Fig. 1, primer pairs b6-2E to b6-11). These observations demonstrate that transcription elongation complexes encounter distinct chromatin subdomains that differ in the level of specific histone modifications.

### Histone Modifications and Transcription Elongation

Several studies in *Drosophila* and in mammalian cell lines have shown that RNA polymerase II complexes pause shortly after transcription is initiated (16, 21, 22). The frequency of pol II release from promoter-proximal pause sites contributes to the regulation of c-myC RNA levels. To test whether histone modifications contribute to the release or stall of pol II within promoter-proximal regions, we have performed ChiP assays using the HL60 cell line in which c-myC RNA levels are rapidly (≤2 h) reduced when cells are induced to differentiate into granulocytes following treatment with Me$_2$SO. C-myC down-regulation is due to the failure of pol II to resume transcription...
elongation after pausing 30 nucleotides downstream of the transcription initiation site (23). Thus, induction of differentiation by Me2SO leads to a drastic decrease in pol II density in regions downstream of the pause site even though transcription initiation and recruitment of RNA polymerase II complexes within the P2 promoter region remains unchanged (Fig. 2A). In contrast to HL60 cells, the down-regulation of c-myc gene expression in the IL-2-dependent murine T-cell line CTLL-2 is regulated at the level of transcription initiation. When IL-2 is removed from the cell culture media, the recruitment of RNA pol II complexes to the c-myc promoter is substantially reduced (Fig. 2A). The addition of IL-2 to cytokine-deprived CTLL-2 cells triggers a rapid induction of c-myc expression within 1 h. The high level c-myc expression persists for more than 9 h.

To determine whether the induction of the block to elongation correlates with dynamic changes in histone modification, we performed ChIP experiments with antibodies specific for acetylated or methylated lysine residues on histone H3 (acetylated Lys9:Lys14, dimethylated Lys4, trimethylated Lys4, dimethylated Lys79, and dimethylated Lys36) and for pan-acetylated H4. Enrichment of histone modifications within specific c-myc gene regions was determined by quantitative duplex PCR. Consistent with previous results (15), a high level of
histone H3 and H4 acetylation is observed both within the promoter region and in the 5′-portion of transcribed sequences. However, histone acetylation is relatively low within exon 3 sequences. Importantly, H3 and H4 hyperacetylation persists even after the onset of the transcription elongation block and remains largely unaffected across the entire c-myc gene in MeSO-treated HL60 cells (Fig. 2B, DMSO). Similar results were obtained in cells that were induced to differentiate for 24 h (data not shown). This indicates that the regulation of transcription elongation at the c-myc gene is not directly coupled to histone acetyltransferase activity catalyzing hyperacetylation of all or a subset of histones to promote elongation.

We next analyzed the pattern of histone methylation of the c-myc gene. ChIP assays using methyl group-specific antibodies confirmed that the Lys4-, Lys79-, and Lys36-methylated histones create distinct subdomains within a gene locus (compare Figs. 1 and 2, C and D). The promoter and the transcribed region of the c-myc gene are enriched in Lys4-dimethylated histone H3. Similarly, Lys4-trimethylated H3, a modification previously linked to active transcription (24), was detectable in both the promoter and transcribed sequences and closely correlated with the pattern of Lys4-dimethylated H3. In contrast, methylation of Lys79 in histone H3 was detected preferentially within transcribed sequences. Lys36-methylated histone H3 was restricted to the 3′-end of the transcribed sequences, further confirming observations made at the bcl-2 and bcl-6 genes.

To test whether the observed histone methylation pattern directly correlates with transcription elongation, the level of histone methylation in proliferating HL60 cells was compared with that in MeSO-induced cells. Lys4 di- and trimethylation as well as Lys79 and Lys36 methylation are only minimally affected by the rapid change in pol II occupancy (Fig. 2C). Thus, although the tested histone methylation marks are characteristic of transcriptionally active genes, they do not invariably correlate with gene activity or RNA pol II density in mamalian cells. Consistent with previous observations (25), the methylation of N-terminal tails remains stable for an extended period, suggesting that only the initial round of transcription requires the association of methyltransferase complexes with the elongating RNA polymerases. The elongation of reinitiated transcription complexes may be independent of the recruitment of histone methyltransferases.

In contrast to HL60 cells, the IL-2-dependent murine T-lymphocyte cell line CTLL-2 down-regulates c-myc expression through a decrease in pol II recruitment to the c-myc promoter rather than by inhibition of transcription elongation of promoter-proximal transcription complexes. Resting CTLL-2 cells cultures do not express c-myc, because of the lack of IL-2-cytokine in the culture media. Stimulation of cells by IL-2 increases RNA pol II recruitment 20-fold upon addition of IL-2, as shown by the results of nuclear run-on and ChIP experiments (Fig. 2A, and data not shown). Despite the dramatic difference in pol II occupancy associated with these physiological conditions, the levels of Lys4, Lys36, and Lys79 methylation in the promoter region and in the transcribed regions of the c-myc gene do not change significantly (Fig. 2D). Growth stimulation by IL-2 only minimally affects the abundance of these methylation marks, and the observed changes are well within the range of experimental variation. In combination, these data suggest that histone modifications by histone acetyltransferases or methyltransferases occur in distinct regions but are not sufficient to regulate the block to transcription elongation.

C-myc Transcription and Histone Variants—Several studies have indicated that histone variants are important regulators of transcriptional activity (5, 7, 26–28). The histone variant H2A.Z (Htz1 in yeast) has been detected at both transcribed and non-transcribed regions. Whole genome microarray analysis in the yeast htz mutant identified genes that are either positively or negatively regulated in the absence of Htz (29). The genetic interaction of Htz with Dst, which encodes the transcription elongation factor TFIIIS, suggests that H2A.Z may also play a role in transcription elongation (30). Structural studies of H2A.Z-containing nucleosomes demonstrated that the incorporation of H2A.Z results in a local destabilization of the docking domain, which is known to interact with the N terminus of histone H4 (31). Recent studies using fluorescence resonance energy transfer (FRET) concluded that this H2A.Z-mediated alteration does not result in an overall destabilization of the nucleosome; rather, it stabilizes the interaction of the H2A.Z/H2B dimer with the (H3/H4)2 tetramer (32). In addition, genetic and ChIP studies have suggested that yeast Htz antagonizes silencing and functions to prevent the spreading of heterochromatin into euchromatic transcriptionally active domains (29). Indeed, biophysical evidence supports a role of H2A.Z in the establishment of domains poised for transcriptional activation (13). To address the role of H2A.Z in the regulation of the mammalian c-myc gene, we performed ChIP assays with chromatin derived from both resting and IL-2-induced CTLL-2 cells using an antibody specific for the C terminus of H2A.Z (Fig. 3). The results demonstrate that H2A.Z associates with the c-myc promoter in both resting and stimulated CTLL-2 cells. H2A.Z decorates the entire promoter region (~2.5 kb) up to the binding site of CTCF, which we have shown to be part of the c-myc insulator element MINE located upstream of the transcription start site (15). Thus, consistent with the hypothesis that H2A.Z inhibits the spreading of silent heterochromatin, this histone variant demarcates the c-myc gene domain. Interestingly, H2A.Z has recently been identified as one of the components in CTCF-containing cellular complexes (33). This suggests that CTCF exerts its boundary function through its association with H2A.Z in the c-myc gene domain.

In addition to the c-myc promoter region, the coding region also associates with H2A.Z, with the exception of the 3′-end of the c-myc gene (exon 3, Fig. 3A). Importantly, IL-2-induced transcriptional activation of the murine c-myc gene depletes H2A.Z within the coding sequences, whereas H2A.Z levels within the promoter region remain unaffected. In contrast, the level of acetylated H2B remains almost unchanged during IL-2-mediated transcriptional activation throughout the c-myc gene domain, suggesting that the regional loss of H2A.Z is not due to a general loss of nucleosomes, as previously observed in a whole genome study in yeast (34). In addition, the depletion of H2A.Z correlates with an increase in the Lys5-acetylated form of major histone H2A within the transcribed region. The increase in H2A concurrent with a decrease in H2A.Z suggests that the nonvariant histone H2A replaces the minor histone variant H2A.Z during mitogenic induction of T-lymphocytes. Consistent with a role of H2A.Z in the suppression of transcriptional elongation, the coding region of the constitutively transcribed GAPDH gene does not associate with H2A.Z, although this histone variant is clearly detectable in the promoter region of GAPDH (Fig. 3B). This demonstrates that the H2A.Z/H2A exchange observed after induction of c-myc transcription is not simply the consequence of the greater availability of histone H2A when cells approach the S phase of the cell cycle.

**DISCUSSION**

Both the modification of histones and the incorporation of histone variants into nucleosomes contribute to the structural and functional organization of chromatin. Studies in *Tetrahymena* and *Saccharomyces cerevisiae* indicate a function for the histone variant H2A.Z in gene activation (5, 35, 36). However, immunofluorescence analyses of chromatin in mouse embryos and of polytene chromosomes in *Drosophila* have revealed an

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This text discusses the role of histone modifications and histone variants in transcription elongation. It highlights the importance of histone H3 and H4 acetylation and the role of histone methylation, particularly Lys4, Lys79, and Lys36 methylation, in regulating transcription elongation. The text also explores the role of histone variant H2A.Z in transcription elongation, especially in the context of IL-2 induction. The integration of histone modifications and histone variants is found to be crucial in the regulation of gene expression.
enrichment of H2A.Z in untranscribed pericentric heterochromatin (37, 38). Structural studies of nucleosomal arrays in vitro indicated a role for the histone variant H2A.Z in the formation of compacted 30-nm fibers while subsequently inhibiting fiber-fiber interactions and the formation of highly condensed chromatin domains. Thus, H2A.Z serves multiple functions in both transcription and the maintenance of genomic stability. Our observation that H2A.Z is depleted from the transcribed region but not from the promoter region suggests that the histone variant H2A.Z has a dual function. Through its association with the promoter region, it demarcates active genes and maintains them in a transcriptionally poised status. At the same time, the incorporation of H2A.Z into the coding region inhibits transcriptional activity, presumably through increasing the nucleosomal stability, which in turn decreases both transcription elongation and recruitment of preinitiation complexes. These features reconcile the previously reported conflicting results that H2A.Z induces a stronger interaction with the (H3-H4) tetramer and, at the same time, is present in transcriptionally active regions (5, 32). The repressive effect of H2A.Z on transcription may depend on its ability to facilitate the formation of 30-nm fibers within the transcribed region while simultaneously inhibiting the formation of highly condensed chromatin domains by blocking fiber-fiber interactions (13). However, the H2A.Z-mediated formation of...
transient removal of H2A-H2B dimers (40, 41). Detailed studies have established a role of the heterodimeric complex in the transient destabilization and the reassociation of a histone H2A-H2B dimer during transcription (4, 42). Thus, the observation that transcriptional activity involves the dynamic exchange of histone variants in nucleosomes may represent the first in vivo evidence of FACT histone exchange activity in higher eukaryotes.

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