Acetylation of histones H4 and H3 targeted to promoters/enhancers is linked to the activation of transcription, whereas widespread, long range acetylation of the same histones has been linked to the requirement for open chromatin at transcriptionally active loci and regions of V(D)J recombination. Using affinity-purified polyclonal antibodies to tetra/tri-acetylated histone H2B in chromatin immunoprecipitation (ChIP) assays with mononucleosomes from 15-day chicken embryo erythrocytes, a high resolution distribution of H2B acetylation has been determined and compared with that of H4 and H3 at the same genes/loci. At the β-globin locus, the H2B acetylation is high throughout and in general mirrors that of H3 and H4, consistent with the observation of co-precipitation of hyperacetylated H4 together with the hyperacetylated H2B. In contrast, at the weakly expressed genes glyceraldehyde 3-phosphate dehydrogenase (GAPDH) and Gas41 (housekeeping) and carbonic anhydrase (tissue specific), very little or no hyperacetylated H2B was found despite the presence of acetylated H4 and H3 at their promoters and proximal transcribed sequences. At the inactive lysozyme and ovalbumin genes essentially no acetylation of H2B, H3, or H4 was observed. Acetylation of H2B appears to be principally a feature of only the most actively transcribed genes/loci.

The N-terminal tail of mammalian H2B can be defined as the trypsin-sensitive 26 residues (1) that precede the extremely basic run of eight trypsin-insensitive residues that passes through the gyres of the DNA superhelix in the crystal structure of the core particle (2). This flexible N-terminal segment of H2B could not be modeled in the crystal structure of the core particle (2). This flexible N-terminal segment of H2B could not be modeled in the crystal structure of the core particle and contains four sites of lysine acetylation (K5, K12, K15, and K20). Only for yeast H2B, which has a very different N-terminal sequence from that in higher eukaryotes, has any connection been made between the acetylation of H2B and the activation or repression of transcription. It was shown (3) that disruption of the deacetylase gene HDA1 leads to hyperacetylation of H2B (and H3) at the ENA1 promoter. In higher eukaryotes there is only circumstantial evidence; we have noticed that, when conducting ChIP experiments with antibodies specific for hyperacetylated H4 that precipitate chromatin highly enriched in active sequences, the proteins in the antibody-bound fraction are enriched in acetylated species not only of H4 but also H2B (e.g. Ref. 4). Although this suggests there is substantial cohabitation of acetylated H2B and H4 in the same nucleosomes, this does necessarily occur at transcriptionally active regions of the genome, because there may be other functionally important regions that harbor both modified histones. To shed light on this problem we generated antibodies specific to tetra/tri-acetylated H2B so as to test their association with transcriptionally active and inactive regions. Such antibodies are used here in ChIP assays with mononucleosomes from 15-day chicken erythrocytes, cells for which a significant amount of data have already accumulated for the acetylation of histones H4 and H3 (4–6).

**EXPERIMENTAL PROCEDURES**

Preparation and Affinity Purification of Antibodies—Anti tetra/tri-acetylated histone H2B serum was prepared by immunizing rabbits with chemically acetylated H2B, as described for histone H4 in Ref 4. After purification of total Ig on protein A-agarose, antibodies were affinity purified using a tetra-acetylated N-terminal peptide, residues 1–24, of H2B (Alta Bioscience) coupled to controlled pore glass (CPG) beads. Specific antibodies were eluted with 3.5 mM KSCN and immediately desalted on a G25 column equilibrated in 10 mM sodium bicarbonate, pH 8.0 (7). Preparation and characterization of the anti-hyperacetylated H4 and H3 antibodies have already been described (8).

**ELISA Testing of Serum and Western Blotting**—ELISA screening of sera was performed as described (5). Histones were resolved at 15% SDS-PAGE or 15% acetic acid/urea (AU)– or acetic acid/urea/Triton (AUT)–PAGE (9). For Western blotting, SDS gels were equilibrated and electrophoretically transferred to 15 µm glycine, 20 µm Tris, 0.1% SDS, and 20% methanol using a Bio-Rad Trans-Blot apparatus (400 mA, 90 min at 10 °C) onto nitrocellulose (Amersham Biosciences ECL membrane). Proteins from 15% AU-PAGE gels were transferred onto polyvinylidene difluoride membranes (Immobilon P) after equilibration in 5% acetic acid and 10% methanol followed by washing and transfer in 0.1% acetic acid and 10% methanol (200 mA, 16 h at 4 °C). Membranes were blocked in 4% (w/v) Marvel (nonfat milk) in 1× phosphate-buffered saline for 1 h, washed in 1× phosphate-buffered saline and 0.1% (v/v) Tween 20 and incubated with diluted serum for 1 h as indicated in the Fig. 1 legend. After further washing with 1× phosphate-buffered saline and 0.1% Tween, chemiluminescent detection was performed using an ECL kit (Amersham Biosciences).

Preparation of Nucleosomes and Immunoselection of Chromatin—Mononucleosomes were prepared from 15-day chicken embryo erythrocytes, and ChIP assays were performed as described (4, 8). Typically, 400 µg of input mononucleosomes (as DNA) was immunoprecipitated with 100 µg of affinity-purified antibody. The histones and DNA from

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The on-line version of this article (available at http://www.jbc.org) contains Supplemental Figs. A and B and Supplemental Table 1.† These authors contributed equally to this work.

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1 The abbreviations used are: ChIP, chromatin immunoprecipitation; ELISA, enzyme-linked immunosorbent assay; AU, acetic acid/urea; AUT, acetic acid/urea/Triton; BSA, bovine serum albumin; GAPDH, glyceraldehyde 3-phosphate dehydrogenase; CA, carbonic anhydrase; 1, input fraction; B, bound fraction; U, unbound fraction; MAP, multiple antigen peptide; LCR, locus control region.
the input (I), unbound (U), and bound (B) fractions were recovered, and UV quantified as described (4).

Quantitative Real Time PCR—Differences in DNA sequence content from the bound and input fractions were determined by real-time PCR using the ABI Prism 7900 sequence detector following the PE Applied Biosystems TaqMan Universal PCR Master Mix protocol. PCR was carried out in triplicate using 20 ng of template DNA (quantified by UV absorption) from each fraction at 50 °C for 2 min and 95 °C for 10 min, followed by 40 cycles of 95 °C for 15 s and 60 °C for 1 min. Primer and TaqMan probe concentrations were optimized using the Applied Biosystems protocol. In brief, the threshold (CT) was set at a point such that the fluorescence signal was above the baseline noise but as low as possible in the exponential amplification phase. A standard curve for each amplicon was constructed by plotting the cycle number at which the fluorescence crossed the threshold (the crossing value) against increasing amounts of genomic DNA template. The crossing values of the input and bound fractions were used to obtain the target sequence content from the standard curve. The enrichment of a given target sequence achieved by the antibody (the indicator of its acetylation level) was determined as the ratio of the sequence content in equal weights of the bound fraction and the input fraction; fold enrichment is B/I. If the sequence content in the bound fraction was less than that in the input, the ratio of input to bound (I/B) is given; this is fold depletion. Primers and TaqMan probes were selected from the chicken β-globin locus using PE Applied Biosystems Primer Express software. Primers and TaqMan probe sequences together with absolute enrichments and errors obtained for each of the amplicons are shown in supplemental Table I (in the on-line version of this article). Typical B/I errors were 5–10%. All amplicons were designed to be substantially shorter than 147 bp (the size of a nucleosome monomer). PCR quantification of the sequence content of the U fraction was also routinely checked.

To be sure that there is a nucleosome at the ampiclon of interest, the input signal was always compared with that from genomic DNA to check for under-representation. Typically, the signal from the input mononucleosomes was ~35% of that obtained from an equal weight of genomic DNA. Checking the input DNA also has the advantage that if the ampiclon is present elsewhere in the genome, then a relative over-representation will be apparent. It should be noted that enrichments at different ampiclons can be directly compared for a given ChIP, as can enrichments measured at different genes using DNA from the same ChIP. In contrast, relative acetylation levels of H2B, H3, and H4 at a given ampiclon do not follow directly from comparing enrichments obtained in the three different ChIPs.

Reverse Transcriptase PCR—Total RNA was extracted from 15-day chicken erythrocytes using an RNAqueous kit (Ambion) and was DNase I treated (1 unit/mg, 30 min, 37 °C). First strand cDNA was prepared using random hexamer primers (Promega) and SuperScript II enzyme (Invitrogen) at 95 °C for 3 min followed by 300 units of SuperScript II at 37 °C for 60 min. Products were amplified using gene-specific primers spanning an intron, and products were quantified by real-time PCR.

RESULTS

Generation and Testing of Serum—Antibodies to hyperacetylated H2B were obtained by immunizing rabbits with chemically acetylated chicken H2B. The sera obtained were first tested by ELISA (Fig. 1), from which it is clear that there is a very strong response to the immunogen (AcH2B) and also to a MAP peptide (MAP-H2B) consisting of eight copies of the first 24 residues of H2B with all four natural sites acetylated (K5, K12, K15, and K20), synthesized on a branched poly-lysine core. Importantly, there was no ELISA response to chicken erythrocyte H2B, which carries very little acetylation (CE H2B), or to fully chemically acetylated histone H4 (AcH4) or chemically acetylated BSA (AcBSA). These results imply that the antiserum is specific for acetylated H2B and, in particular, does not recognize acetylated forms of H4. Furthermore, because AcBSA is not recognized, there is no activity against the epitope ε-acetyl-lysine as was reported in earlier studies with an antiserum raised against chemically acetylated H4 (7).

SDS-PAGE Western blotting was then used with a total histone extract from HeLa cells grown in butyrate (high levels of acetylation in all four core histones), and Fig. 2A shows that the antiserum is specific to H2B and does not recognize any of the other histones. An AU-PAGE Western blot (Fig. 2B) of an H2A/H2B mixture from butyrate-treated HeLa cells then showed that the antiserum recognizes the tetra-acetylated (Ac4) species of H2B predominantly, with a smaller response to tri-acetylated (Ac3) but very poor responses to the other forms of H2B, which are present in much greater quantities. This antiserum is therefore referred to as “anti tetra/tri-acetylated H2B.”

Analysis of Immunoprecipitated Proteins—Mononucleosomes were generated from nuclei of 15-day chicken embryo erythrocytes using micrococcal nuclease (MNase), followed by sucrose gradient purification. ChIP experiments were then performed with affinity-purified anti-tetra/tri-acetylated H2B antibodies using previously described methods (see “Experimental Procedures”). Because these antibodies have not been used previously for ChIP experiments, parallel experiments were performed using our already characterized anti-hyperacetylated H4 and H3 antibodies that were used in ChIP experiments to monitor acetylation of these histones at most of the genes and loci studied here (using manual methods of quantitative PCR; Ref. 8). To make further checks on the efficacy and specificity of the anti-tetra/tri-acetylated H2B antibodies, proteins were extracted from the immunoprecipitated mononucleosomes and compared with those of the input and unbound mononucleosomes using AUT gels, Fig. 3. From this gel it is clear that there is substantial enrichment of the most acetylated species of H2B in the bound chromatin relative to the input, showing the effectiveness of the antibodies in selecting nucleosomes carrying hyperacetylated H2B.

Strikingly, however, there is also significant enrichment of the most acetylated species of H4 in the antibody-bound mononucleosomes. Because the present series of ChIP experiments compares the genomic distributions of acetylated forms of H2B with those of H4 and H3, we also analyzed the proteins in the antibody-bound chromatin from the H4 ChIP experiments conducted in parallel (Fig. 4). It is clear that, in addition to the expected high level of H4(Ac4) in the bound mononucleosomes, there is also a substantial increase in hyperacetylated H2B species. Because the ChIP experiments were conducted with pure mononucleosomes, it follows that there must be considerable cohabitation of hyperacetylated H2B with hyperacetylated H4 at various points in the genome.

Acetylation Mapping at the β-Globin Locus—Fig. 5A shows
the distribution of tetra/tri-acetylated H2B across the chicken \( \beta \)-globin locus in 15-day embryos, which can be compared with that of H3 and H4 in Fig. 5, B and C, respectively. At 15 days the \( \beta \)-adult (\( \beta^a \)) gene is strongly active, the \( \beta^H \) (hatching) gene is active (though at a lower level than \( \beta^a \)), but the embryonic genes \( \beta^e \) and \( \beta^o \) are inactive. It is immediately evident that there is a close parallel between the distribution of acetylation of all three core histones, with substantial acetylation at intergenic regions in addition to the four genes themselves. The acetylation of H3 shows the greatest degree of modulation across the locus, whereas that of H2B is very similar to that of H4.

The boundaries of the locus are well defined by reduction of the acetylation to essentially zero at both ends. The upstream edge is at the core element (250 bp) of the 5'-insulator that coincides with HS4, the constitutive hypersensitive site. No acetylation is found at 13350 (in the numbering system used here) within the heterochromatin region (16 kb in all) that
Fig. 5. Distribution of acetylation of the three core histones (H2B, H3, and H4). A, a map of tetra/tri-acetylated histone H2B levels across the chicken β-globin locus in 15-day embryo erythrocytes, obtained by immunoprecipitating purified mononucleosomes with the affinity-purified antibodies described in “Results.” DNA sequence content was determined by real time TaqMan PCR after quantifying the DNA from the I, U, and
separates the locus from that of the folate receptor gene, the transcriptional start of which is at 5395 (10, 11). The downstream (3') end of the β-globin locus is also characterized by a constitutive hypersensitive site, 3HS (12), and here the enrichment drops to unity shortly beyond this point. At this end of the locus there is no intervening heterochromatin, and it immediately abuts an olfactory receptor gene, the ATG of which is at 53056.

The distribution of acetylation is also strongly asymmetric, the most intense being at the upstream end within the 5'-insulator at HS4 and also at all three tissue-specific hypersensitive sites, HS3, HS2, and HS1, that together presumably represent the LCR. In fact, the acetylation is very high for about 8 kb at the 5'-end of the locus, with a reduction only between HS3 and HS2, as noted in an earlier study (11). With the exception of the H9252 A promoter, the long-range acetylation tails off toward the 3'-boundary.

**Acetylation Mapping at Other Chicken Genes**—Our previous observations (8) showed that in 15-day erythrocytes the widespread acetylation of H3 and H4 at the H9252-globin locus was not a feature of housekeeping genes nor of weakly expressed tissue-specific genes. In those cases, the H3 and H4 acetylation was restricted to the promoters. We therefore wondered if the same is true for H2B acetylation and thus analyzed the DNA from the acetylated H2B ChIP at the glyceraldehyde 3-phosphate dehydrogenase (GAPDH), carbonic anhydrase (CA), and ovalbumin genes. H2B acetylation was also monitored at the lysozyme locus (13, 14), a well defined region of ~21 kb that contains two genes, namely lysozyme (active only in oviduct and mature macrophages and silent in erythrocytes) and the gene Gas41 (active in all cell types tested). The Gas41 gene codes for an essential protein (15) that interacts with the nuclear matrix protein NuMA (16) and with the SWI/SNF chromatin-remodeling complex (17). Its very broad expression pattern means that the Gas41 gene can be regarded as a housekeeping gene, and it contains a CpG island. The transcriptional status of the GAPDH, CA, and ovalbumin genes in 15-day erythrocytes was established previously (8), but not that of the Gas41 gene. Reverse transcriptase PCR experiments were therefore performed, using a total RNA extract as template, to compare the Gas41 mRNA level with that of the highly active β^A gene and the inactive lysozyme gene. Primers for the 16-kb heterochromatin were also used (the most upstream amplicon in Fig. 5). Whereas essentially no signal was obtained from the lysozyme and the 16-kb heterochromatin amplicons, a significant response was obtained at the Gas41 amplicon, implying that it is transcribed in 15-day erythrocytes. Quantification of the PCR products showed that the Gas41 mRNA level is ~30,000× less than for the β^A gene, i.e. transcription is at a low level. This large difference can be seen in the context of previous reverse transcriptase PCR estimates (8) in which the GAPDH message pool was shown to be several...
orders less than that from the β^A^ gene, i.e. the Gas41 mRNA level is significantly less than that of GAPDH.

The DNA from all three ChIPs was analyzed at the above five genes using TaqMan probes. At the GAPDH gene (Fig. 6A), substantial H4 acetylation was seen at the promoter and some within the gene itself (Exon V), though the H3 acetylation was limited to the promoter. In contrast, no enrichments were found for H2B acetylation, either at the promoter or at Exon V. At the carbonic anhydrase gene (Fig. 6B), substantial H4 and H3 acetylation was found within the CpG island region but none within the gene (Exon VII). As with GAPDH, no H2B acetylation was found within the CpG island region, and little or none was found within the gene at Exon VII. At the active Gas41 gene (Fig. 7), substantial H4 and H3 acetylation was found within the CpG island region and the adjacent transcribed sequences rather as at the GAPDH gene. However, no enrichments for H2B were observed at any point within the Gas41 gene or at the promoter of the silent lysozyme gene for any of the three core histones. Furthermore, neither the −2.4 kb silencer (−2.4S) nor the proximal promoter (P) affected the expression of the gene in macrophages (reviewed in Ref. 13) exhibited any enrichments. As a further negative control, we monitored the ovalbumin gene (silent in erythrocytes) and found substantial depletions in all three ChIPs, especially within Exon VII (I/B, −1 for H2B). As regards H4 and H3, this is in good agreement with the earlier observations (8).

The results make it clear that in these 15-day erythrocytes there is very little acetylation of H2B at the three weakly expressed genes monitored here, in contrast to the situation at the β-globin locus where intense and widespread H2B acetylation is present, even at the inactive embryonic β^A^/β^B^ genes.

**DISCUSSION**

Overall, the H2B acetylation at the β-globin locus is similar to that of H4 and is similarly modulated, i.e. is present at both active and inactive genes as well as at intergenic regions. If this situation were repeated at many points in the genome it would explain the high level of cohabitation of the hyperacetylated histones H2B and H4. However, the intensive acetylation of...
H2B at the β-globin locus is not characteristic of the other genes studied here, wherein essentially no H2B acetylation is seen despite GAPDH, CA and Gas41 being active. Because GAPDH and Gas41 are housekeeping genes (a category that includes about half of all genes) and carry acetylated H4 at their promoters and some distance into the transcribed regions but no acetylated H2B, cohabiting acetylated H4 and H2B does not appear to come from this subset of genic regions and must derive from loci of the β-globin type or from other chromatin subsets.

If one compares the two genes inactive in these erythrocytes, ovalbumin and lysozyme, neither shows evidence of H2B acetylation, but a difference is nevertheless noted in that whereas substantial and reproducible depletions (up to 3.5 ×) are seen at the ovalbumin gene, B/B ratios of ~ 1 were found at the enhancer and promoter of the lysozyme gene. This difference could be due to the fact that the lysozyme gene is in a locus where there is an immediately adjacent active gene (Gas 41).

From the present data it seems that H2B acetylation is a feature of only the most active genes/loci, but it will require investigation of other highly active genes/loci to decide this question definitively. If this hypothesis is correct, then, for example, in macrophages and oviduct tissue where the lysozyme gene is highly active, the acetylation of H2B will be substantial and widespread.

The distinction between locus-wide histone acetylation and more targeted enhancer/promoter modifications is re-emphasized by the present data on H2B acetylation in that it covers a large part of the chicken β-globin locus, i.e. at intergenic regions as well as at both active and inactive genes and across the whole 8 kb of the LCR. In this respect H2B acetylation follows that of H4 and H3, but, in contrast, acetylated H2B is not found even at the promoters of active housekeeping genes.

The presence of widespread acetylation at active loci has been proposed as a requirement for maintaining the overall transcriptional competence of a locus (4, 21–23), for example, resisting the formation of the 30-nm supercoil conformation by acetylation and tracking models in which acetylation is initiated at the LCR and then spreads along the locus (25, 32–34).

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Acetylation of Histone H2B Mirrors that of H4 and H3 at the Chicken β-Globin Locus but Not at Housekeeping Genes
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