Review

The diversity of acetylated proteins
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Published: 30 April 2002
Genome Biology 2002, 3(5):reviews0006.1–0006
The electronic version of this article is the complete one and can be found online at http://genomebiology.com/2002/3/5/reviews/0006
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Modification of proteins extends the range of possible molecular structures beyond the limits imposed by the 20 encoded amino acids and, if reversible, gives a means of control and signaling. Many proteins are acetylated, both co- and post-translationally, and at least for eukaryotic proteins, acetylation is the most common covalent modification out of over 200 types that have been reported. Acetylation of proteins is catalyzed by a wide range of acetyltransferases that transfer acetyl groups from acetyl-coenzyme A to either the ε-amino group of amino-terminal residues or to the ε-amino group of lysine residues at various positions. (The ε-amino group designates the position of the central carbon atom of amino acids, whereas the ε-amino group of lysine residues designates the position of a carbon atom in the side chain.) As shown in Table 1, amino-terminal acetylation occurs cotranslationally on the bulk of acetylated eukaryotic proteins [1-3] and post-translationally on prokaryotic ribosomal proteins [4,5] and on processed eukaryotic regulatory peptides [6]. Amino-terminal acetylation is one of the most common protein modifications in eukaryotes, occurring on approximately 85% of eukaryotic proteins, but is rare for prokaryotic proteins [1-3]. Furthermore, ε-lysine acetylation occurs post-translationally on histones, high mobility group (HMG) proteins, transcription factors, nuclear receptors, α-tubulin [10]. Acetylation affects many protein functions, including enzymatic activity, stability, DNA binding, protein-protein interaction, and peptide-receptor recognition, and occurs on numerous and diverse proteins.

Amino-terminal acetylation

Cotranslational amino-terminal acetylation of eukaryotic proteins

Studies in vitro indicate that amino-terminal acetylation of eukaryotic proteins takes place when there are between 20 and 50 residues protruding from the ribosome [1,11]. Proteins susceptible to amino-terminal acetylation have a variety of different amino-terminal sequences, with no simple consensus motifs and no dependence on a single type of residue [1,3,12]. Proteins with serine and alanine termini are the most frequently acetylated, and these residues, along with methionine, glycine, and threonine, account for over 95% of the amino-terminal acetylated residues [1,2]. Only subsets of proteins with any of these amino-terminal residues are acetylated, however, and none of them guarantees acetylation [3].

The complexity of the termini that are acetylated is due to the presence of multiple N-acetyltransferases (NATs; Tables 1,2), each acting on different groups of amino-acid sequences and whose specificity is determined by two or more residues at the amino-terminal positions [13]. Unlike the situation for histones and other proteins with acetylated ε-lysine residues, amino-terminal modifications are irreversible.

Studies with the yeast Saccharomyces cerevisiae have revealed three amino-terminal acetyltransferases, NatA, NatB, and NatC, that act on different groups of substrates; each group of substrates has a different degenerate motif recognized by the NAT [3]. As shown in Table 2, all
amino-terminal acetylated proteins are substrates for one of NatA, NatB or NatC. Furthermore, we do not know of any acetylated proteins in yeast that could not reasonably be a NatA, NatB or NatC substrate. Nevertheless, it remains to be seen if there are other NATs that act on rarer substrates. The similarity in the pattern of amino-terminal acetylation of the proteins from higher eukaryotes and *S. cerevisiae* and the presence of genes orthologous to those encoding the three amino-terminal acetyltransferases in mammals and plants (our unpublished observations) suggest that the same systems may operate in all eukaryotes.

The biological significance of amino-terminal modification varies; some proteins require acetylation for function whereas others that are acetylated do not absolutely require the modification. The viability of yeast mutants lacking the catalytic subunits (*ard1*-Δ, *mak3*-Δ or *nat3*-Δ) or other subunits (*nat1*-Δ) of NATs suggests that the function of acetylation may be subtle and not absolute for most proteins [13]. It is possible that only a subset of proteins actually requires this modification for activity or stability, whereas the remainder are acetylated only because their termini fortuitously correspond to consensus sequences. Amino-terminal acetylation does not necessarily protect proteins from degradation, as has often been supposed, nor does it play any obvious role in protection of proteins from degradation by the ‘N-end rule’ pathway that determines whether to degrade proteins according to their amino-terminal residue.

**Amino-terminal acetylated proteins in prokaryotes**

There are few examples of amino-terminal acetylated proteins in prokaryotes; they include the *Escherichia coli* ribosomal
proteins S5, S18 and L12 [4,5] and mycobacterial ribosomal protein L12. These modifications probably occur post-translationally (Table 1). The corresponding E. coli NAT genes, rimI, rimJ, and rimL, have been identified but it is still unclear how related they are - structurally, functionally and evolutionarily - to eukaryotic cotranslational NATs. These E. coli NATs are analogous to eukaryotic NatAs, which also acetylate α-Ser and α-Ala residues of ribosomal proteins.

**Amino-terminal acetylation of processed regulatory peptides and hormones**

Most eukaryotic regulatory peptides, hormones, and neurotransmitters are synthesized in the cell as larger precursor proteins, which are biologically inactive and must undergo a variety of post-translational processing steps to yield the active peptides [14]. After translation, the precursor is generally cleaved by an endopeptidase; this is followed by peptide modification, for example by carboxy-terminal amidation, sulfation, phosphorylation, glycosylation or amino-terminal acetylation [6]. These modifications frequently have a profound influence on the biological activity of the peptide; for example, both sulfation on tyrosine and carboxy-terminal amidation are obligatory for full biological activity of the octapeptide cholecystokinin, a gastrointestinal hormone. Importantly, more than one biologically active peptide can be produced from the same precursor and there may be variations in the pathways of processing at any of several different levels, so that different cells expressing a particular gene may give rise to different peptide products [15,16].

The finding that amino-terminal acetylation, occurring post-translationally, increases the pigment-producing (melanotropic) effects of α-melanocyte-stimulating hormone (α-MSH) and enhances its activity in behavioral tests represents the clearest example of the biological importance of this modification [16,17]. Amino-terminal acetylation also causes a greater than 50-fold increase in the potency of growth hormone releasing factor. In contrast, amino-terminal acetylation of β-endorphin, which takes place in the ‘storage’ form of this peptide hormone markedly reduces its opioid activity compared with the form with a free amino terminus [18]. Acetylation can also affect protein stability: there is evidence that the half-life of nonacetylated α-MSH in rabbit plasma is one-third of that of the acetylated form [19], and the stability of acetylated synthetic peptide MART-1 (a peptide derived from human melanoma-associated Melan-A antigen) is higher than that of MART-1 with a free amino terminus. Importantly, the expression of α-MSH and β-endorphin peptides is physiologically regulated and can be induced, but little is known about the factors that govern the cell-type-specific patterns of processing and modification of regulatory peptides; elucidation of these factors is currently a major challenge.

Similarly, little is known about the regulatory peptide acetyltransferases. Although a peptide acetyltransferase activity has been partially characterized [20], the corresponding gene has not been identified. It is unlikely, but cannot be excluded in certain cases, that NATs acting cotranslationally can modify regulatory peptides or their precursors. The amino-terminal residue of β-endorphin is tyrosine, however, which is not a substrate for cotranslational NATs; this tyrosine is nevertheless normally acetylated in the storage form of β-endorphin. Also, peptide acetyltransferases probably act in cooperation with peptide secretion, in contrast to cotranslational NATs that are likely to be associated with cytoplasmic polysomes. It is possible that neuropeptides and hormones may also be modified after secretion in ways that change their biological activities. Finally, it would be interesting to identify the genes encoding acetylpeptide hydrolases, which presumably deacetylate and thus activate β-endorphin from its acetylated storage form in mammals. Studies of the regulation of function of both peptide acetylases and peptide acetylhydrolases may be of great importance for the pharmacology and molecular genetics of human diseases.

**Acetylation on internal lysines**

**Acetylation of histones**

The most studied proteins that are acetylated on ε-lysine residues include histones H2A, H2B, H3, and H4, in which the modification occurs at multiple sites in the amino-terminal tail domains, and the HMGP proteins, which are found in a variety of eukaryotes from yeast to humans [7]. The important feature of acetylation of ε-lysine residues is that it is reversible. Histones are frequently subjected to post-translational modifications that include acetylation, methylation, and phosphorylation of specific arginine, lysine, histidine, serine and threonine residues [21]. These modifications, many of which are also reversible, all decrease the positive charges of histone tail structures, thereby significantly altering histone-DNA binding, and interactions between nucleosomes and between histones and regulatory proteins. The discoveries of Gcn5p, the first nuclear histone acetyltransferase (HAT), and of the first histone deacetylase (HDAC),

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**Table 2**

| Substrates | NatA | NatB | NatC |
|------------|------|------|------|
| α-Ser-     | α-Met-Glu- | α-Met-Ile- |      |
| α-Ala-     | α-Met-Asp- | α-Met-Leu- |      |
| α-Gly-     | α-Met-Asn- | α-Met-Trp- |      |
| α-Thr-     | α-Met-Met- | α-Met-Phe- |      |

*Acetylation occurs on all proteins with α-Met-Glu- and α-Met-Asp- termini but only on subclasses of proteins with the other termini.*
established that acetylation of histones is an important controlling step in transcription [22]. Some of the nuclear HATs are also well known and extensively characterized as transcription factors. Not surprisingly, histone acetylation appears to influence other processes, including cell-cycle progression, chromosome dynamics, DNA replication, recombination and repair, silencing, and apoptosis [23]. Despite significant accumulation of information on HATs, understanding of the precise molecular role of histone acetylation in the assembly of chromatin, the accessibility of transcription factors and nucleosome remodeling is still elusive.

There are over 20 HATs that fall into several families, listed in Table 1. All HATs act in a site-specific and histone-specific manner, and specificity may differ in vivo and in vitro; such diversity may help to explain why there are so many HATs. Remarkably, some HATs are associated with other HATs and coactivators, suggesting a layer of complexity that is not yet understood. It is important to note, however, that the steady-state balance of histone acetylation appears to exert different effects on different genes in different settings. Alignment of the amino-acid sequences surrounding modified lysines in acetylated proteins and mutagenesis of the human importin-α protein Rchi suggest that the HAT recognition motif may be GKKK (in the single-letter amino-acid code, with the acetylated ε-lysine residue in bold) [24].

**Histone deacetylases**

A large number of HDACs have now been identified, many of which act as corepressors of transcription [23]. The yeast deacetylases Rpd3p and Hda1p are recruited by repressor proteins to promoters, causing a localized deacetylation of chromatin [25]. Specialized regions of chromatin, including telomeres, centromeres, and silent yeast mating-type loci, are transcriptionally inactive and form hypoacetylated heterochromatin-like (tightly packed) domains. Heterochromatin formation in yeast is mediated by the silencing proteins Sir2p, Sir3p, and Sir4p; Sir2p has been found to have HDAC activity. Interestingly, deacetylases are detected in some chromatin-remodeling complexes, which regulate changes in chromatin structure, together with HATs. Little is known about the specificity of HDACs, although it has been found that HDAC1 can deacetylate not only histones but also the transcription factor E2F1 [26].

**Acetylation of HMG proteins**

HMG proteins are a heterogeneous family of non-histone chromosomal proteins whose function is still not completely understood, despite their abundance and ubiquity. A subset of these proteins contains the HMG domain, a DNA-binding motif that recognizes bent DNA or induces bending in linear duplex DNA. Two post-translational modifications, namely phosphorylation and acetylation, influence the DNA-binding properties of HMG1. This protein is reversibly acetylated at conserved lysines at positions 2 and 11 [27], and it has been shown that monoacetylation at lysine 2 of HMG1 increases the binding affinity of the protein for some types of distorted DNA [28]. This indicates the possible involvement of HMG1 in DNA repair, separate from its ‘architectural’ role in nucleoprotein complexes. Also, HMG1 and HMG2 have been implicated in protein-protein interactions and have been shown to facilitate the specific binding of regulatory proteins - such as steroid hormone receptors, Hox and POU-domain proteins (developmental transcription factors), p53 (a tumor suppressor), and the TATA box-binding basal transcription factors - to their target DNA sequences [29].

**Acetylation of transcription factors**

In the nucleus, DNA is tightly packaged into several orders of structure with no easy accessibility for the transcription machinery. Acetylation of lysine residues within histones, histone-like proteins, and non-histone proteins (such as transcription factors) has recently emerged as a major mechanism used by the cell to overcome repressed chromatin states [8,9]. Several transcription factors have been identified as substrates for HATs, particularly for the HATs CREB-binding protein (CBP) and its close homolog p300, which are cofactors of nuclear-receptor-activated gene transcription, and p300/CBP-associated factor (PCAF). These substrates include the transcriptional activators E2F-1-3 (involved in progression through G1/S cell-cycle transition), p53, c-Jun (a transcription factor involved in the response to mitogens), the erythroid Krüppel-like transcription factor (EKLF), the transcriptional coactivator GATA1 that is required for megakaryocyte and erythrocyte differentiation, the muscle-specific differentiation regulator MyoD, the product of the proto-oncogene c-myb, the HMG protein HMG1(Y), the T-cell factor regulated transcription activator TCF (which is downstream of Wnt signaling proteins), hepatocyte nuclear factor HNF-4, the general transcription factors TFIIIEβ and TFIIIF, erythocyte transcription factor NF-E2(MafG), and the steroid hormone nuclear receptor coactivator ACTR ([9,21,24-32] and references therein). The list of the new HAT substrates is growing rapidly. Acetylation of transcription factors can alter their ability to bind DNA (in the cases of E2F1, p53, EKLF, GATA1, and HNF-4), to interact with other proteins (c-Jun, TCF, ACTR, and HNF-4), or to remain in the nucleus (HNF-4). In addition, PCAF, p300 and CBP can autoacetylate, facilitating intramolecular rearrangements between the bromodomain (which binds acetyl-lysine) and the acetylated lysine(s); this interaction may be important for HAT activity and for recruitment of remodeling complexes to acetylated chromatin [33].

The effect of acetylation on DNA-binding-protein function depends on the location of the modified site within the protein. In case of the transcription factors p53, E2F1, EKLF, and GATA-1, the acetylation site is located directly adjacent to the DNA-binding domain, and acetylation stimulates DNA binding [26,30,34]. In contrast, the lysines acetylated within HMG1(Y) are within the DNA-binding domain and
result in disruption of DNA binding. Thus, acetylation does not always stimulate transcription.

Acetylation also affects protein–protein interactions. For example, the association of nuclear steroid hormone receptors with their coactivator ACTR is inhibited by acetylation [31]. Apparently, histone acetylation generates a recognition site for the bromodomain, a motif conserved in many proteins, including HATs [33]. Histone acetylation may precede the recruitment of ATP-dependent chromatin-remodeling activities during transcriptional activation. In particular, the HAT Gcn5p is involved in stabilizing binding of the SWI/SNF chromatin-remodeling complex to a promoter, and this interaction seems to be mediated through the Gcn5p bromodomain [21]. There is some evidence, exemplified by the transcription factor E2F1, that acetylation increases the half-life of the protein [26].

Acetylation of nuclear import factors
HATs can also target other nuclear proteins. A screen of a large set of proteins involved in different cellular processes resulted in the identification of two nuclear import proteins, Rch1 and importin-α7, as substrates for the acetyltransferase CBP [9]. The reaction seemed to be specific because another nuclear import factor, importin-α3, was not a substrate for CBP. Both p300 and CBP can mediate acetylation of Rch1 and importin-α7 in vivo, most likely in the nucleus [9]. The acetylated residue, ε-Lys22, lies within the binding site in Rch1 for the other nuclear import factor, importin-β, and acetylation of the site promotes interaction with importin-β in vitro [9]. Thus, it is possible that nuclear import may be regulated by acetylation, mediated by the p300/CBP HATs.

The targeting of HAT enzymes to their substrates is likely to be important and may play a role in regulation by other signaling pathways, as indicated by the finding that phosphorylation of p53 stimulates its acetylation, probably by increasing the association of p53 with p300 [35]. Some evidence indicates that the activity of HATs is regulated by proliferation and differentiation signals [23], via phosphorylation or hormonal signaling. For example, the HAT activity of CBP is stimulated at the G1-S phase boundary of the cell cycle, and hormone-induced acetylation of ACTR represses nuclear receptor function. Together, these results have led to the hypothesis that acetylation is a regulatory modification that may rival phosphorylation in cell signaling [36].

Acetylation of tubulin
Microtubules are cylindrical cytoskeletal structures that are found in almost all eukaryotic cell types and are involved in a great variety of cellular processes, including mitosis, ciliary and flagellar motility, intracellular transport of vesicles and organelles, and possibly in determining morphology of certain cells [37]. The structural subunit of microtubules is the 100 kDa protein tubulin, which consists of α and β isoforms that form heterodimeric complexes and associate head-to-tail to form profilaments and then laterally to make up the walls of cylindrical microtubules. Several types of post-translational modification affect tubulin function, including acetylation, phosphorylation, polyglutamation, polyglycylation, and detyrosination [10]. Most of these modifications are reversible and all, except acetylation, occur at the highly variable carboxyl termini of tubulin α and β subunits.

The first evidence for acetylation of tubulins was obtained with a flagellar tubulin from the unicellular alga Polytomella [38]. Tubulin acetylation has since been observed in vertebrates, insects, nematodes and plants, in all of which the acetyl group is attached to the ε-amino group of lysine 40. The α-tubulin acetyltransferase was purified from the flagellated unicellular alga Chlamydomonas and from mammalian brain and was shown to have molecular mass of 62-67 kDa [39]. During purification of the enzyme from Chlamydomonas, evidence was obtained for a tubulin deacetylase and for an inhibitor of α-tubulin acetyltransferase. In Chlamydomonas, the tubulin acetyltransferase exhibits a two-fold preference for polymerized over soluble tubulin, but in HeLa cells the acetylation occurs mainly after polymerization [40]. Generally, acetylation can happen quickly - almost immediately - and acetylated tubulin therefore does not necessarily demarcate old microtubules. Some correlation has been found between α-tubulin acetylation and microtubule stability [40]. Acetylated microtubules commonly resist drug-induced disassembly but not cold-induced disassembly, although in some cells a subset of acetylated microtubules is cold-resistant [41]. It is still unclear, however, how the intracellular spatial organization of acetylated microtubules is determined. There may be some factors limiting acetyltransferase enzyme activity to certain cellular microtubules and to restricted regions: candidates for such factors include the microtubule-associated proteins MAP1B, MAP2 and γ, which either enhance or inhibit the interaction of the acetyltransferase with microtubules [40]. Another possibility is that the interplay of microtubules with other cytoskeletal elements or organelles regulates acetyltransferase enzyme activity.

The role of acetylated microtubules in cells remains an important unanswered question. Acetylated tubulin is not required for survival, and a mutant of the ciliate Tetrahymena with lysine 40 replaced with arginine is indistinguishable from the wild type [41]. Cloning and analysis of the 62-67 kDa α-tubulin acetyltransferase mentioned above will be critical for understanding the role of α-tubulin acetylation.

Diversity of acetylated proteins
Acetylated proteins are varied, and acetylation can have a range of effects on protein function. Rapidly accumulating new results of functional analysis on HATs allowed
Kouzarides [36] to suggest that, like phosphorylation, acetylation can regulate such different essential processes in the cell as transcription, nuclear import, microtubule function, and hormonal response. At the moment, there is no clear evidence that acetyltransferases act in cascade, although there are other striking similarities between phosphorylation and acetylation. Both autoacetylation [33] and autophosphorylation are known to occur, and both phosphorylation and acetylation [21,23] can be reversible. Thus, acetylation may rival phosphorylation in cell signaling. Although both phosphorylation and acetylation alter the charge of the modified protein or protein domain, the effect of acetylation is ‘milder’. The spectrum of substrates for acetylation is much broader than that of phosphorylation, however, and includes proteins and polypeptides from almost all cellular compartments and involves both amino-terminal and internal ε-lysine modifications. The biological role of protein acetylation is diverse, reflecting the different acetyltransferases that have evolved to meet the requirements of individual proteins or protein families.

Acknowledgements

We thank Jeffrey J. Hayes (University of Rochester) for useful comments. This work has been supported by National Institute of Health Grant R01 GM12702. B.P. dedicates this work to the memory of his father, Wasył Polewoja, who had an extraordinary mind and a full life.

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