Histol Histopathol (2007) 22: 1051-1055

http://www.hh.um.es

Histology and Histopathology

Cellular and Molecular Biology

Review

The function of actin in gene transcription

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Summary. Recent developments in the field of gene transcription regulation have unfolded a key role for actin as an important co-factor for all three eukaryotic RNA polymerases. In this review article we discuss the latest findings on actin in transcription of protein-coding and ribosomal genes, in complex with specific hnRNP proteins and a form of myosin 1ß which is entirely localized to the cell nucleus. Based on these recent studies, we propose a general model where actin may function in basal gene transcription as an allosteric regulator, to recruit transcriptional co-activators on active genes. A future challenge will be the identification of the polymerization state of actin in gene transcription and how it is mechanistically regulated.

Key words: Nuclear actin, Nuclear myosin, Ribonucleoproteins, RNA polymerase, Transcription

Introduction

Actin and myosins have been topics of cutting-edge research for many years. They were originally discovered in the 1860s as the actomyosin complex by the work of W. Kühne (Pederson and Aebi, 2002). In 1941-42, actin was further identified as an individual protein in extracts of rabbit skeletal muscles, almost concomitantly with the discovery of the ATPase activity of myosins (reviewed by Pederson and Aebi, 2002). However, we had to wait approximately forty years before the first convincing reports, discussing the presence of actin in the cell nucleus and a potential function, were published (Clark and Merriam, 1977; Gounon and Karsenti, 1981). Gounon and Karsenti (1981) demonstrated that actin forms a nuclear gel where chromosomes are likely to be embedded and, a few years later, the laboratories of B. Jockush and P. Chambon provided first circumstantial evidence that actin could be involved in gene transcription (Egly et al., 1984; Scheer et al., 1984).

After those initial studies, the possibility that actin could even play a role in nuclear function was put aside. The concept of actin in the cell nucleus was questioned with the argument that cellular actin is very abundant and actin contamination in nuclear protein extracts could not be ruled out. As a consequence of this general scepticism, a relatively long period of time had to pass before actin was identified as a component of certain ATP-dependent chromatin remodelling complexes (Zhao et al., 1998; reviewed by Olave et al., 2002). Almost concomitantly, actin was also found in nascent RNP particles (Percipalle et al., 2001, 2002) and to reside on active transcription sites (Percipalle et al., 2001). A few years after these observations, several independent reports were published demonstrating that actin is associated with all three eukaryotic RNA polymerases and it is directly involved in gene transcription (Fomproix and Percipalle, 2004, Hofmann et al., 2004, Hu et al., 2004; Philimonenko et al., 2004; Kukalev et al, 2005).

In this review, we primarily discuss the molecular mechanisms underlying the role of actin in transcription of protein coding genes and ribosomal genes.

RNA polymerase II-mediated transcription requires actin/RNP complexes

Initial mechanistic clues on the involvement of actin in pol II transcription were obtained using the polytene chromosomes in the salivary glands of the dipteran *Chironomus tentans*. In this model organism it is possible to visualize the large Balbiani ring (BR) pre-mRNPs *in status nascente*, still associated with the chromatin axis, when they are released in the nucleoplasm and finally, during passage through the nuclear pore complex (reviewed by Daneholt, 2001a,b). An affinity purified polyclonal antibody against actin immunostained the transcription sites of the *C. tentans* chromosomes in an RNA-dependent manner and

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Abbreviations: RNP (ribonucleoproteins), hnRNPs (heterogeneous ribonucleoproteins), IEM (immunoelectron microscopy)

labelled nascent BR pre-mRNPs (Percipalle et al., 2001). In addition, IEM with the same anti-actin antibody revealed labelling of mature BR mRNPs in the nucleoplasm, passing through the nuclear pore complex, and detected labelling of tubular structures on the endoplasmic reticulum. Based on the above findings, it was concluded that actin accompanies the BR mRNA from gene to polysomes (Percipalle et al., 2001). These observations also suggested that actin may play a key role throughout the entire gene expression process, including gene transcription (Egly et al., 1984; Scheer et al., 1984), mRNA export (Hofmann et al., 2001) and translation (Stapulionis et al., 1997).

Analysis of the actin-associated proteins in pre-mRNP/mRNP particles provided more insights into the role of actin in mRNA synthesis. DNase I affinity chromatography on nuclear protein extracts combined with mass spectrometry identified a subset of hnRNPs both in *C. tentans* and in higher vertebrates (Percipalle et al., 2001, 2002). The *C. tentans* protein hrp65-2 and the human protein hnRNP U were found to directly interact with actin through a specific novel actin-binding motif that is conserved from insects to humans (Percipalle et al., 2003; Kukalev et al., 2005). *In vivo* disruption of both interactions caused a severe down-regulation of pol II transcription, as monitored by BrUTP incorporation into nascent pre-mRNA transcripts (Percipalle et al., 2003; Kukalev et al., 2005). These results suggested that the function of actin may not be restricted only to the gene promoter, to facilitate assembly of the transcription-competent pol II machinery (Hofmann et al., 2004). Actin, in complex with hrp65-2 or hnRNP U, may also facilitate mRNA elongation by maintaining efficient pol II transcription. Evidence in support of this model comes from IEM performed on the BR gene, where it is possible to visualize proximal, medial and distal regions with respect to the gene promoter (Kiesler and Visa, 2004 and references therein). In these studies, the localizations of actin and hrp65-2 along the gene were statistically correlated with the medial and distal region, suggesting a preferential localization for actin and hrp65-2 along the active gene (Percipalle et al., 2001, 2003). Similarly, in mammalian cells, chromatin immunoprecipitation experiments demonstrated that actin and hnRNP U are located at promoter, as well as along coding region of constitutively active genes (own unpublished observations).

During the elongation phase of pol II transcription, the interaction between actin and specific hnRNPs may be required for recruitment of transcriptional co-activators (Percipalle and Visa, 2006) and, consistent with this model, the actin/hrp65-2 complex was shown to be required for HAT recruitment on active BR genes (Sjölinder et al., 2005). Given that hnRNP U is known to associate with HATs (Martens et al., 2002), a similar scenario can be envisaged for the actin/hnRNP U interaction (for a speculative model, see figure 1). Future analyses are required to identify the putative HAT and the molecular mechanisms required for the actin/hnRNP U-mediated recruitment.

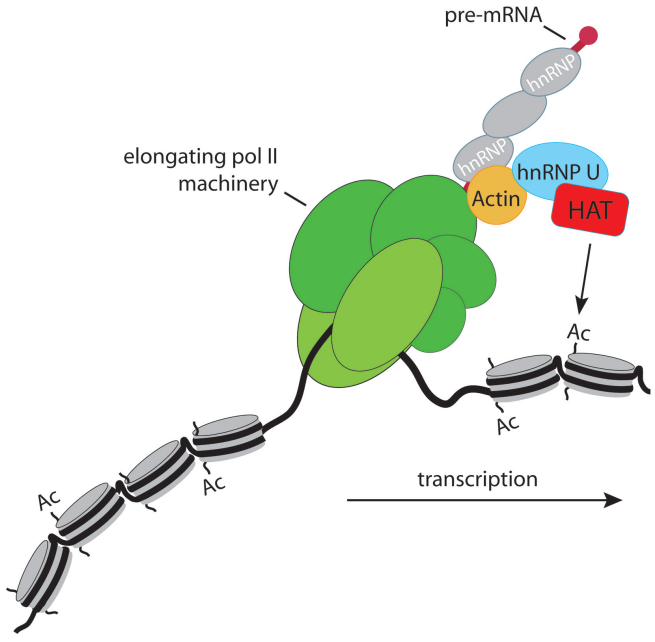

Fig 1. A speculative model for the function of actin/hnRNP complexes in pol II transcription. The specific interaction between actin and hnRNP U may facilitate pol II transcription elongation by recruitment of co-activators, such as histone acetyl transferases (HATs), to the active gene (see text for details).

Transcription of ribosomal genes requires actin and myosin 1ß

In 2000, a form of myosin 1ß entirely localizing to the cell nucleus was discovered in the laboratory of P. de Lanerolle (Pestic-Dragovich et al., 2000). The difference between the nuclear and the cytoplasmic myosin 1ß mainly resides in a short N-terminal amino acid sequence that imparts nuclear localization. The extra sequence, found in the nuclear form of myosin 1ß, appears to be conserved in higher vertebrates, as well as *Xenopus laevis*, but not in lower eukaryotes (Kahle et al., 2007). Analysis of the cellular distribution with a peptide-specific antibody showed that nuclear myosin 1 (NM1) exhibits a preferential nuclear localization, but it is also present in nucleolar foci corresponding to fibrillar centres, the sites where rDNA transcription takes place (Fomproix and Percipalle, 2004; Percipalle et al., 2006). In addition, actin and NM1 were revealed as genuine nucleolar proteins, they could be co-immunoprecipitated with the largest pol I subunit and were found at rDNA promoter and coding region by chromatin immunoprecipitation (Fomproix and Percipalle, 2004; Philimonenko et al., 2004; Leung et al., 2006; Percipalle et al., 2006). Finally, the intranuclear distribution of both actin and NM1 was shown to be sensitive to the transcription state of the cell (Kysela et al., 2005). In

conclusion, the above observations underscored a potential key role for actin and NM1 in pol I transcription.

A direct function for both actin and NM1 in rRNA synthesis was identified in *in vitro* transcription experiments using both naked rDNA templates and pre-assembled chromatin (Philimonenko et al., 2004). Insights into how actin and NM1 may cooperate in pol I transcription were obtained from the discovery that NM1 is a component of a novel chromatin remodelling complex, termed B-WICH, that contains WSTF (William syndrome transcription factor) and the ATPase SNF2h (Cavellan et al., 2006; Percipalle et al., 2006). Antibodies to NM1 and WSTF blocked synthesis of run-off rRNA transcripts from pre-assembled chromatin templates and post-transcriptional gene silencing of both NM1 and WSTF produced a down-regulation of the total amount of the 45S precursor (pre)-rRNA (Philimonenko et al., 2004; Percipalle et al., 2006). In addition, antibodies to NM1 and WSTF did not interfere with the initial phase of pol I transcription but affected only the elongation phase (Philimonenko et al., 2004; Percipalle et al., 2006). Altogether, these results suggested a role for the B-WICH complex in activation and maintenance of the post-initiation phase of pol I transcription (Percipalle et al., 2006; Percipalle and Östlund Farrants, 2006).

The identification of B-WICH as a transcription activator raises several mechanistic questions. One important point is whether B-WICH is already present on the ribosomal gene at the exit of mitosis when ribosomal gene transcription is restarted. Consistent with this hypothesis, recent evidence suggests that NM1 localizes to nucleolar organizer regions (NoRs) in mitotic cells (Fomproix and Percipalle, 2004).

It is possible that NM1 has a concerted role with actin in the pre-assembly of the polymerase I machinery at the promoter (reviewed by Visa, 2005; Grummt, 2006). However, antibodies to actin and NM1 did not affect abortive transcription initiation assays. Therefore, it is plausible that the main function for the actin/NM1 complex is exerted in later events such as promoter clearance or rRNA transcript elongation. One suggestive model, depicted in figure 2, may be that the dynamic actin/NM1 interaction mediates B-WICH recruitment on the ribosomal gene to alter ribosomal chromatin structure for productive pol I transcription (Percipalle and Östlund Farrants, 2006). In this view, the actin/NM1 interaction may work as a molecular switch for the dynamic recruitment of pol I co-factors (Percipalle and Östlund Farrants, 2006).

Allosteric regulation of gene transcription: what is next?

The observation that actin is associated with all three eukaryotic RNA polymerases and is required for RNA synthesis emphasizes a general role in both initiation and maintenance of basal gene transcription.

In association with elongating RNA polymerases, we propose that actin functions as an allosteric regulator of gene transcription, mediating the recruitment of transcriptional co-activators along the gene. Co-activator recruitment is likely to be facilitated through specific interactions of actin with adaptor proteins – NM1 in pol I transcription to recruit B-WICH and hnRNP U in pol II transcription to recruit HATs – through a nucleotide exchange-based mechanism. This model takes into consideration the high degree of conformational plasticity that is a characteristic of the actin molecule, but requires further testing at the molecular level, for instance through the identification and functional characterization of the nuclear actin-binding proteome.

The recent studies discussed above have not yet clarified the conformational state of actin in gene transcription. The transcription-competent actin does not seem to form the classical filamentous structures observed in the cytoplasm (Percipalle and Visa, 2006). It appears to be in a monomeric, globular (G) state or in a short, non-conventional, oligomeric form which is not positive to phalloidin staining (Percipalle and Visa, 2006; own unpublished observation).

Indeed, there is evidence that both monomeric and oligomeric forms of actin are present in the cell nucleus. A low mobility population of nuclear actin has recently been identified in mammalian cells and visualized in *Xenopus* oocytes, where it is believed to be implicated in nuclear structure and stability (McDonald et al., 2006; Bohnsack et al., 2006). Nuclear actin polymerization has also been observed in response to viral pathogens (Goley et al., 2006) and several factors involved in the regulation of the polymerization state of cytoplasmic actin are also present in the cell nucleus and may be involved in nuclear function. N-WASP, which facilitates actin polymerization, has recently been found to play a role in pol II transcription (Rohatgi et al., 1999;

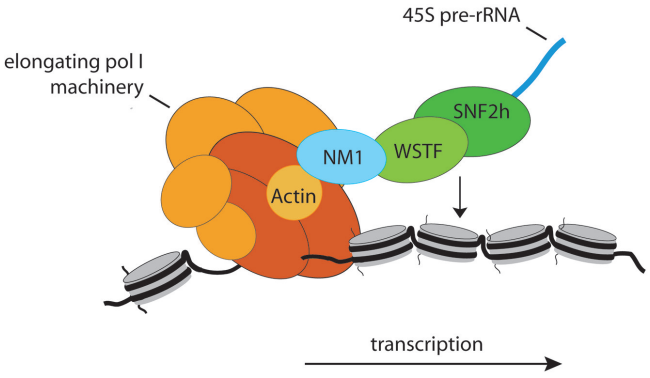

Fig. 2. The suggested model for the action of actin and nuclear myosin 1ß (NM1) in pol I transcription. The dynamic interaction between actin and NM1 mediates recruitment of the ß-WICH chromatin remodelling complex required to activate the post-initiation phase of pol I transcription (see detailed text).

Takenawa and Miki, 2001; Millard et al., 2004; Wu et al., 2006; Vieu and Hernandez, 2006). Similarly, small actin binding proteins, such as ß-thymosin, cofilin and profilin, which stabilize G-actin, have also been discovered in the cell nucleus (Pendleton et al., 2003; Skare et al., 2003; Huff et al., 2004). Altogether, the above observations support the view that monomeric and oligomeric forms of actin co-exist in the cell nucleus (Jockhush et al., 2006). Considering its ability to form a gel (Gounon and Karsenti 1979), nuclear actin may be in a precarious state between an unstable monomeric form and non-conventional oligomeric states, such as the actin dimer (Bubb et al., 2002; Reutzel et al., 2004), known to be required for the nucleation of non-conventional forms of actin (Pederson and Aebi, 2005). Further corroboration to this hypothesis is provided by two studies in which anti-actin monoclonal antibodies were characterized and shown to have high specificity against nuclear actin epitopes which may be hidden in the conventional cytoplasmic F-actin (Gonsior et al., 1999; Schoenenberger et al., 2005). The availability of these antibodies, referred to as conformational antibodies, will probably facilitate our understanding of the polymerization state of the transcription-competent nuclear actin.

In summary, even though it is not yet clear whether gene transcription is fine-tuned through nuclear actin polymerization events, there is now compelling evidence in support of a main function for actin in pol I, II and III transcription. Future investigations are likely to uncover the molecular mechanisms underlying the precise function of actin and will probably redesign the complex network of functional protein-protein interactions required by the transcriptosome for productive gene transcription.

Acknowledgements. Our work is supported by grants from the Swedish Research Council (Vetenskapsrådet) to PP.

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Accepted March 27, 2007