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REVIEW

Long non-coding RNAs: the tentacles of chromatin remodeler complexes

Bernadette Neve[1] · Nicolas Jonckheere[1] · Audrey Vincent[1] · Isabelle Van Seuningen[1]

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Abstract

Chromatin remodeler complexes regulate gene transcription, DNA replication and DNA repair by changing both nucleosome position and post-translational modifications. The chromatin remodeler complexes are categorized into four families: the SWI/SNF, INO80/SWR1, ISWI and CHD family. In this review, we describe the subunits of these chromatin remodeler complexes, in particular, the recently identified members of the ISWI family and novelties of the CHD family. Long non-coding (lnc) RNAs regulate gene expression through different epigenetic mechanisms, including interaction with chromatin remodelers. For example, interaction of lncBRM with BRM inhibits the SWI/SNF complex associated with a differentiated phenotype and favors assembly of a stem cell-related SWI/SNF complex. Today, over 50 lncRNAs have been shown to affect chromatin remodeler complexes and we here discuss the mechanisms involved.

Keywords ATP-dependent helicase · Nucleosome · Histone and epigenetic regulation

Introduction

Long non-coding RNAs (lncRNAs) are a heterogeneous class of long RNAs without a large open reading frame encoding proteins. To better understand the role of lncRNAs in the formation and recruitment of chromatin remodeler complexes, we will briefly discuss the organization of DNA into chromatin, and then review the chromatin remodeler subfamilies with the involved subunit proteins.

Chromatin is the state of organized DNA condensation in the nucleus. DNA is wrapped into nucleosome structures that are further packed into chromatin fibers and condensed into either "euchromatin" or highly condensed "heterochromatin". Chromatin condensation is a dynamic process resulting in different nuclear sub-compartments, including topologically lamina-associating domains, nucleoli, Cajal bodies, nuclear stress bodies, paraspeckles and non-chromatin bodies like nuclear speckles and PML bodies (reviewed by [1]).

In the fundamental nucleosome unit, ~ 146 base-pairs (bp) of the negatively charged DNA helix are folded ~ 1.7 times around an octamer of positively charged histone proteins. A tetramer consisting of two H3 and two H4 histones binds to the DNA, where after two H2A-H2B dimers join the complex. The first ~ 20 bp of DNA that sort the fold are hold together by the histone H1 protein and a "beads on a string" structure is formed with strings of ~ 15–70 bp free DNA (Fig. 1a, b, [2]).

Nucleosome positioning is part of epigenetic regulation since the condensed DNA is inaccessible for transcription, replication, and repair [3]. Positioning of nucleosomes is not arbitrary; firstly, it depends on the thermodynamic bending properties of different DNA sequences. In particular, the properties of poly(deoxyAdenylic(dA):deoxyThymidylic(dT)) DNA stretches disfavoring nucleosome formation. DNA sequence motifs can also be recognized by sequence-specific factors that may lead to nucleosome remodeling or depletion. Secondly, DNA modifications, such as 5-methylation and 5-hydroxymethylation of cytosines, affect DNA flexibility and nucleosome stability. Thirdly, the DNA-interactions with histone tails may vary, as these are subjected to several posttranslational modifications, which may change electric charges and/or evoke steric hindrance (Fig. 1c). In addition, acetylation of lysine residue 16 of histone 4 (H4K16) weakens its protein–protein

Audrey Vincent and Isabelle Van Seuningen are last co-auteurs.

✉ Bernadette Neve
bernadette.neve@inserm.fr

[1] UMR9020-U1277 - CANTHER - Cancer Heterogeneity, Plasticity and Resistance to Therapies, Univ. Lille, CNRS, Inserm, CHU Lille, 59000 Lille, France

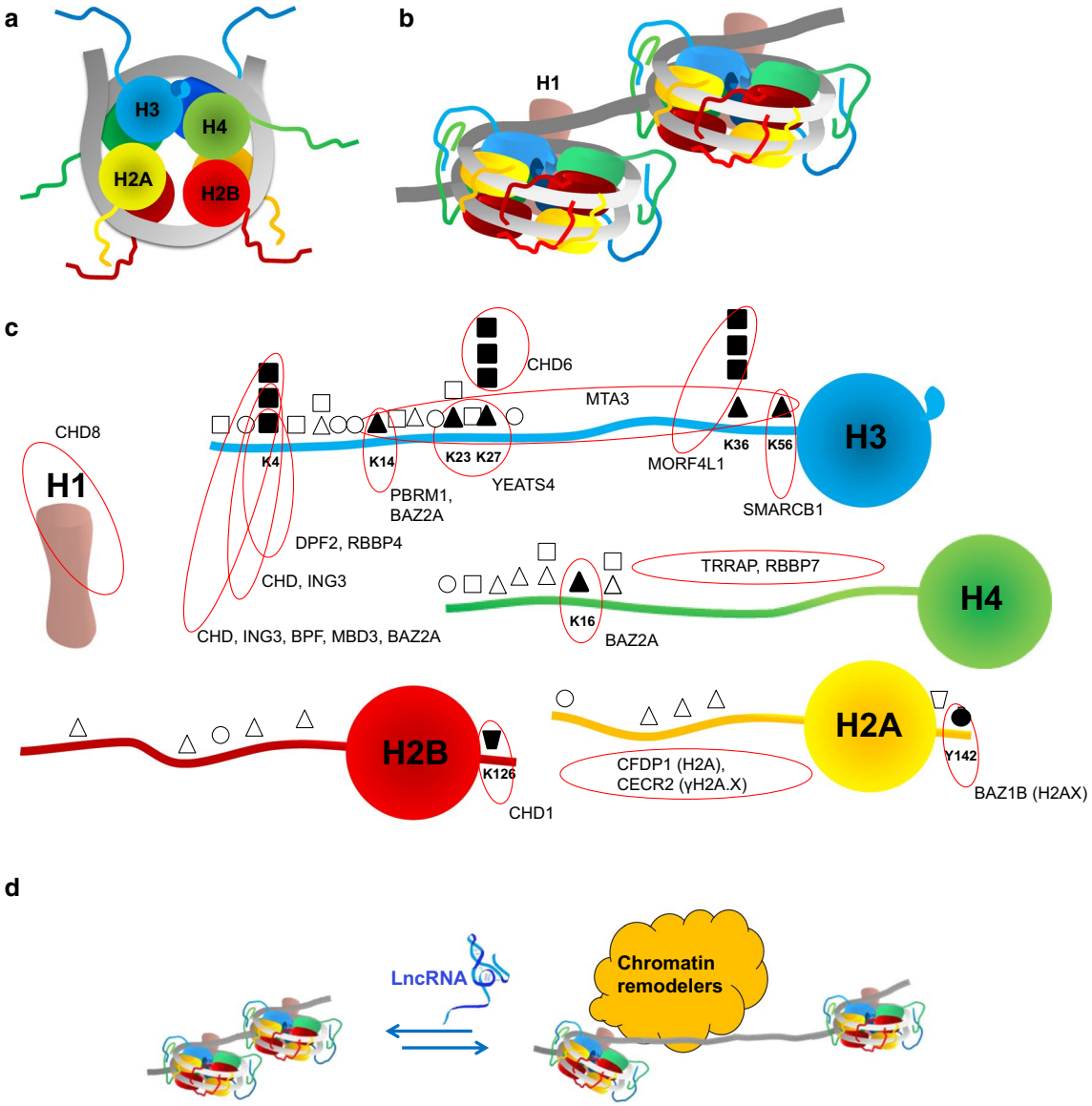

Fig. 1 Organization of chromatin. Depicted is a DNA helix (in grey) with histone proteins H2A, H2B, H3, H4 and H1 (different colors). **a** Schematized top view of a nucleosome with tetramer $(H3\text{-}H4)_2$ and two dimers (H2A-H2B). **b** Nucleosomes side view with histone H1 stabilizing the DNA linker. Methylated histone tails have stronger DNA interactions and then acetylated tails. **c** Presented are common histones modifications, such as methylation (squares), acetylation (triangles), phosphorylation (circles) and ubiquitination (trapeziums). Histones modifications are frequently recognized by chromatin readers. Modifications that are implicated in chromatin remodeling complexes are depicted by solid symbols and their corresponding readers are annotated (red circles). **d** LncRNAs (in blue) can either inhibit or stimulate chromatin remodeling via interference with remodeler complexes

interaction with residues from the H2A-H2B dimer of the adjacent nucleosome and significantly reduces nucleosome stacking [4]. Moreover, modifications like methylation and acetylation of lysine residues are important for the recognition by chromatin reader proteins [5]. These chromatin readers are known to recruit complexes with writers and erasers of chromatin modifications, as well as chromatin remodelers (Fig. 1c).

Rather than modifying DNA or histones to modulate chromatin (like writers/erasers), the remodeler complexes change nucleosome positioning (Fig. 1d). They affect spacing of nucleosomes, regulate nucleosomes transfer and evoke histone variant switching. The kinetic energy needed to modulate the stable nucleosome organization comes from ATP hydrolysis. All chromatin remodeling complexes contain a catalytic unit from the superfamily 2 helicases, which categorizes them into four types of

Table 1 Proteins of human chromatin remodeling complexes

Hugo name	Alternative names	Complex family	Bound complexes	Function	References	Implicated lncRNAs
SWI/SNF subunits (Histone dimer/nucleosome removal, nucleosome sliding)						
1. Catalytic subunit (SMARC subfamily A)						
SMARCA4	BRG1	SWI/SNF	BAF, PBAF	ATP-dependent helicase	[6]	Evf2, UCA1, TUG1, Myheart, CPS1-IT1, NEAT1, MALAT1, LincRNA Cox2 IL-7–AS, LncFZD6, lncTCF7
SMARCA2	BRM	SWI/SNF	BAF			NEAT1, lncBRM
2. SMARC subfamily B subunit						
SMARCB1	SNF5, BAF47	SWI/SNF	BAF, PBAF	Constitutive Core Subunit, chromatin reader binding to H3K56Ac, Enhances DNA integration	[5]	SWINGN, HOTAIR
3–4. SMARC subfamily C dimer (BAF155-155 or 155-170)						
SMARCC1	BAF155	SWI/SNF	BAF, PBAF	Constitutive Core Subunit	[6]	
SMARCC2	BAF170	SWI/SNF	BAF	Core Subunit; mono-ADP-ribosylated by SIRT6	[113]	
5. AT-rich interaction domain subunit						
ARID2	BAF200	SWI/SNF	PBAF	DNA and transcription factor binding	[6]	
ARID1A	BAF250A	SWI/SNF	BAF			HOTAIR, MVIH, DGCR5, LINC00163, CASC15
ARID1B	BAF250B	SWI/SNF	BAF			
6. SMARC subfamily E subunit						
SMARCE1	BAF57	SWI/SNF	cBAF, PBAF	Binding to '4-way' branched DNA (Holliday junction)	[6]	
7–8. Actin-related Subunits						
ACTL6A	BAF53A, Arp4, INO80K	General	BAF,PBAF, INO80, p400/TRRAP	SMARCA/TRRAP binding	[6]	uc.291
ACTL6B	BAF53B	SWI/SNF	ncBAF			
ACTB	(β-Actin)	General	BAF,PBAF, p400/TRRAP			
9. Poly-bromo subunit						
PBRM1	BAF180	SWI/SNF	PBAF	chromatin reader binding to H3K14ac, phosphorylated by ATM	[11, 114]	
10. B-cell lymphoma 11 subunit						
BCL11A		General	BAF/PBAF, NuRD, SIN3A, PRC2 [115]	Enhances DNA integration	[6]	CDKN2B-AS1, uc.57
BCL11B		SWI/SNF	BAF/PBAF			
11. Bromodomain subunit						
BRD7		SWI/SNF	PBAF	Bromo-domain interaction with acetylated lysine.	[116]	
BRD9	LAVS3040	SWI/SNF	ncBAF		[117]	

Table 1 (continued)

Hugo name	Alternative names	Complex family	Bound complexes	Function	References	Implicated lncRNAs
12. B-cell lymphoma 7 subunit						
BCL7A		SWI/SNF	BAF	(B-cell CLL/Lymphoma 7)	[6]	
BCL7B		SWI/SNF	npBAF			
BCL7C		SWI/SNF	npBAF			
13. SMARC subfamily D subunit						
SMARCD1	BAF60A	SWI/SNF	BAF/PBAF	Transcription factor binding [118]		DLEU1
SMARCD2	BAF60B	SWI/SNF	PBAF		[118]	
SMARCD3	BAF60C	General	BAF, MyoD		[119]	
14. PHD-zinc finger subunit						
PHF10	BAF45A	SWI/SNF	PBAF	Phosphorylated protein, complex stability	[6]	
DPF1	BAF45B, NEUD4	SWI/SNF	BAF	(*D*ouble *P*lant homeodomain **F**inger)	[6]	
DPF2	BAF45C	SWI/SNF	BAF	Chromatin reader binding to H3K4me1	[14]	
DPF3	BAF45D	SWI/SNF	BAF		[5]	
15. BRD-interacting subunit						
BICRA	GLTSCR1	SWI/SNF	ncBAF	(*G*lioma *t*umor *s*uppressor *c*andidate region 1), BRD interacting protein	[6]	
BICRAL	GLTSCR1L	SWI/SNF	ncBAF		[6]	
16. Synovial Sarcoma translocation protein Chr18 subunit						
SS18	SSXT/SYT	SWI/SNF	ncBAF	Interaction with BAF47	[120]	
SS18L1	CREST	SWI/SNF	ncBAF	*C*alcium-*res*ponsive transactivator	[120]	
INO80/SWR1 subunits (nucleosome replacement)						
1. Catalytic subunit (ATPase)						
INO80	INO80A, INOC1,	INO80/SWR1	INO80	ATPase	[121]	LCTS5, HAND2-AS1
SRCAP	EAF1, FLHS, SWR1, DOMO1	INO80/SWR1	SRCAP		[121]	LncKdm2b
EP400	p400	INO80/SWR1	p400/TRRAP		[121]	
2–3. Catalytic subunit dimer (helicase)						
RUVBL1	INO80H, RVB1, TIP49	INO80/SWR1	INO80, SRCAP, p400/TRRAP	ATP-dependent helicase	[121]	
RUVBL2	INO80J, RVB2, TIP48	INO80/SWR1	INO80, SRCAP, p400/TRRAP			
4–6. Effector subunit						
YY1	INO80S	General	INO80 , p400/TRRAP, PRC2	Transcription factor (GLI-Krüppel zinc finger), Polycomb proteins recruitment	[122]	ANRIL, Linc-YY1, TUG1, SPAG5-AS1, HOTAIR, LINC00899, LINC00668, Linc01134,
KAT5	TIP60	INO80/SWR1	p400/TRRAP	Lysine (K) acetyltransferase 5	[121]	

Table 1 (continued)

Hugo name	Alternative names	Complex family	Bound complexes	Function	References	Implicated lncRNAs
TRRAP	Tra1	INO80/SWR1	p400/TRRAP	Histone acyltransferase (H4)	[121]	
7–9. Actin-related subunits						
ACTR5	ARP5, INO80M	INO80/SWR1	INO80	INO80 binding	[121]	
ACTR8	ARP8, INO80N	INO80/SWR1	INO80			
ACTL6A	Arp4, INO80K, BAF53A	General	INO80, SRCAP, p400/TRRAP, BAF, PBAF,	TRRAP/SMARCA binding		
ACTB	(β-Actin)	General	p400/TRRAP, BAF, PBAF			
ACTR6	ARP6, CDA12; hARPX, HSPC281, MSTP136	INO80/SWR1	SRCAP	SRCAP binding		
10–17. INO80 subunits						
INO80B	IES2, PAPA-1	INO80/SWR1	INO80	INO80 binding	[121]	
INO80C	IES6	INO80/SWR1	INO80			
TFPT	INO80F, FB1, amida	INO80/SWR1	INO80	Modulated by SUMOylation (increases interaction with INO80E), transcription factor?	[123]	
INO80E	CCDC95	INO80/SWR1	INO80	Binding to TFPT	[121]	
INO80D		INO80/SWR1	INO80	Has two putative DNA-binding domains		CR933609
MCRS1	INO80Q, MSP58, P78	INO80/SWR1	INO80	Has a nuclear and nucleolar localization signal		
NFRKB	INO80G	INO80/SWR1	INO80	NF (nuclear factor) binding to kappa B regulatory elements/winged-helix domains involved in protein-protein interactions, recruitment of EXO1 enhances resection, stimulating homologous recombinase DNA repair (non-essential for the in vitro nucleosome sliding)	[30]	
UCHL5	UCH37	INO80/SWR1	INO80, RPN13	Deubiquitinating enzyme, but inhibited by NFRKB, recruitment of EXO1 (non-essential for the in vitro nucleosome sliding)	[124],	DRAIC
9–12. SRCAP/TRAPP subunits						
VPS72	YL1; CFL1; Swc2; YL-1; TCFL1	INO80/SWR1	SRCAP, p400/TRRAP	Histone chaperone	[125]	

Table 1 (continued)

Hugo name	Alternative names	Complex family	Bound complexes	Function	References	Implicated lncRNAs
DMAP1	SWC4, MEAF2, EAF2, DNMTAP1	General	SRCAP, p400/TRRAP, DNMT1/HDAC complexes	Regulation of transcription by binding to DNMT1, HDAC2 and HAT complexes	[121]	
BRD8	p120	INO80/SWR1	SRCAP, p400/TRRAP	Bromodomain interaction with acetylated lysine (histone H4) and nuclear receptors (TR, RXR)	[121]	RNCR3
YEATS4	GAS41; NUBI-1	INO80/SWR1	SRCAP, p400/TRRAP	Chromatin reader, binding to H3K23acK27ac binding, H3K122suc, homology to transcription factors MLLT 1, 3	[126, 127]	lncAKHE
13–14. SRCAP specific subunits						
CFDP1	Swc5, p97, BCNT	INO80/SWR1	SRCAP	Interacting with H2A	[121]	
ZNHIT1	ZNFN4A1	INO80/SWR1	SRCAP	Interacting with H2AZ	[128]	
13–16. P400/TRAPP specific subunits						
MORF4L1	MRG15	General	p400/TRRAP (MRG15-MRGBP), HDAC Rpd3S/Sin3S (MRG15-Pf1)	Chromatin reader that binds to H3K36me3, H3K4me1 and H3K4me3	[121]	
MRGBP	Eaf7, MRG15BP, URCC4	INO80/SWR1	p400/TRRAP	Interaction with BDR8, MORF4L1	[121]	
ING3	ING2, EAF4	General	p400/TRRAP,	Chromatin reader, binding to H3K4me3, interaction with TP53	[121]	CASC7
MEAF6	EAF6	General	p400/TRRAP, HAT complexes (eg., MOZ/MORF, NuA3)	Interaction with EAF4	[121]	
ISWI (nucleosome spacing, DNA repair)						
1. Catalytic subunit (ATP-dependent helicase)						
SMARCA5	SNF2H	ISWI	WICH, NoRC, RSF, ACF/CHRAC	ATP-dependent helicase	[46]	
SMARCA1	SNF2L	ISWI	NURF, CERF		[46]	DLEU1
SMARCAD1	ADERM, BASNS, ETL1, HEL1, HRZ	?	"CUE"	ATP-dependent helicase, binds to H2A-ubiquitin, CUE-domain binds KAP1; maintaining H3K9me3 marks.	[129]	
HELLS	SMARCA6	?	CHIRRC	ATP-dependent helicase, binds to CDCA7	[48]	BlackMamba
2. Signature subunit						

Table 1 (continued)

Hugo name	Alternative names	Complex family	Bound complexes	Function	References	Implicated lncRNAs
BAZ1B	WSTF	ISWI	WICH	Histone phosphorylation (H2A.X-pY142), interacts with RNA polymerase II, stimulates transcription-coupled homologous recombination	[52]	
BAZ2A	Tip5, NoRC	ISWI	NoRC	Chromatin reader that binds to H3K4, H4K16ac, H3K14ac. Binds also to TCF7L2 and ncRNAs. Responsible for heterochromatin formation at major clusters of repetitive elements	[130, 131]	Lnc pRNA
RSF	HBXAP	ISWI	RSF	Remodeling and spacing factor 1; histone chaperone, involved in homologous recombination DNA repair	[132]	
BAZ1A	ACF1	ISWI	ACF/CHRAC	Dimer ACF complex, RNF20-mediated chromatin relaxation, and KU70 interaction, mediating DNA repair	[55]	NEXN-AS1
BPTF	NURF301	ISWI	NURF	Chromatin reader binding to H3K4me3 and H4K16ac, interacts with AT rich DNA sequences	[57]	NMR (LINC01672)
CECR2	CERF	ISWI	CERF	Chromatin reader binding to acetyl-lysine residues of histone H2A and H3, stimulating γH2A.X formation	[53]	
TRIM28	KAP1	?	"CUE"	Maintaining H3K9me3 marks.	[129]	Paupar
CDCA7	ICF3, JPO1	?	CHIRRC	Nucleosome sliding	[48]	FGD5-AS1
3–4 CHRAC subunits						
CHRAC1	YCL1, CHRAC15	ISWI	ACF/CHRAC	Chromatin accessibility complex subunit 1; histone-fold protein that form a stable complex with POLE3 and binds naked DNA	[76]	
POLE3	CHRAC17, p17, YBL1	General	ACF/CHRAC, DNA POL ϵ complex, HAT GCN5/PCAF complex	DNA polymerase epsilon 3; accessory subunit; histone H3, H4 chaperone	[133]	
3–4 RB binding subunits						

Table 1 (continued)

Hugo name	Alternative names	Complex family	Bound complexes	Function	References	Implicated lncRNAs
RBBP4	NURF55, RBAP48	General	NURF, NuRD , PRC2 complex	RB binding protein 4; histone tetramer deposition; histone H3 and BCL11A binding	[76]	
RBBP7	RbAp46	General	NURF, NuRD, PRC2 complex	Binding to histone H4, histone acetyltransferase HAT1	[76]	
CHD (chromodomain helicase DNA-binding, DNA repair; NuRD, histone deacetylation/demethylase complex)						
1. Catalytic subunit (Chromodomain Helicase DNA-binding)						
CHD1	PILBOS		CHD1/ PAF1, CHD1/ SSRP1, CHD1/ NCoR CHD1/HAT complexes (SAGA/SLIK/ GCN5)	Chromatin reader that binds to H3K4me2/3, stabilization of H2AX at DSBs, stabilization of H2B(tail)K123ub	[67–70, 134]	MATN1-AS1
CHD2	EEOC		CHD2/PARP-1	Deposition of histone variant H3.3 in NHEJ DNA-repair	[72]	CHASERR
CHD6	CHD5, RIGB		CHD6/Polycomb units	Chromatin reader that binds to H3K27me3	[135]	
CHD7	CRG, KAL5		CHD7/SOX2, CHD7/SOX9, CHD7/8, PBAF	SOX2, SOX9, SMARCC2 binding	[79]	LINC01410
CHD8	AUTS18		CHD8/β-catenin, CHD8/CHD7, CHD8/MT complexes (BRD4-NSD3, BACH1-MAFG-DNMT3, KMT2)	Binds to histone H1, H3K36 methyltransferase (MT) complex	[80, 81]	
CHD9	KISH2		CHD2/PPARα, CHD2/PPARγ, BAF	Chromatin reader that binds to H3 (K9me2/3,K27me3), also interaction with PPARα, γ, SMARCA2. Ribosomal gene regulation	[76]	
1. Catalytic subunit NuRD						
CHD3	Mi-2a	NuRD	NuRD, PRC2 complex	Generation of compacted chromatin after DNA replication; isoforms CHD3.1 and CHD3.3: SUMOylated KAP-1 binding; transcription factor interaction	[55]	
CHD4	Mi-2b	NuRD	NuRD, PRC2 complex	Transcription factor interaction, interaction with RNF8	[76, 136]	
CHD5		NuRD	NuRD, PRC2 complex	H3K27me3 binding	[135]	
2. Interacting subunit						
FAM124B			CHD7/8		[86]	
3. GATA-type Zn-finger subunit						

Table 1 (continued)

Hugo name	Alternative names	Complex family	Bound complexes	Function	References	Implicated lncRNAs
GATAD2A	p66a	NuRD	NuRD/CHD5	CHD and MBD2 binding	[75]	
GATAD2B	p68	NuRD	NuRD/CHD5	Protein-protein interaction, deacetylating, methylated nucleosomes		
4–5 Histone deacetylase Subunit						
HDAC1		general	NuRD, PRC2 complex	Histone deacetylase (Kac)	[76]	ANRIL
HDAC2		general	NuRD, PRC2 complex			SNHG15, ARAP1-AS1
6–7 RB binding subunits						
RBBP4	NURF55, RBAP48	general	NURF, NuRD , PRC2 complex	RB binding protein 4; histone tetramer deposition, histone H3 and BCL11A binding	[76]	
RBBP7	RbAp46	general	NURF, NuRD, PRC2 complex	Binding to histone H4 and histone acetyltransferase HAT1		
2. Interacting subunit						
CTBP2		NuRD	NuRD, PRC2 complex	C-terminal binding protein 2; NADH-dependent interaction with p300-Runx2 complex, decoy inhibiting transcriptional activation of this complex	[137]	
3. Methyl-CpG binding subunit						
MBD3		NuRD	NuRD, PRC2 complex	methyl-CpG binding domain protein 3; no binding to methylated DNA, mediates the association of MTA2 with HDAC, H3K4me3 binding	[136]	
MBD2	DMTase	NuRD	NuRD, PRC2 complex	methyl-CpG binding domain protein 2; reposition nucleosomes away from unmethylated CpG-rich regions of the genome	[136]	
4. Metastasis-associated subunit						
MTA1		NuRD	NuRD, PRC2 complex	Metastasis-associated protein 1	[76]	
MTA2		NuRD	NuRD, PRC2 complex	Metastasis-associated protein 2	[76]	HOTAIR
MTA3		NuRD	NuRD, DMT complex LSD1/SIX3, PRC2 complex	Binds to pan-acetylated H3, histone demethylase, opposing action to MTA1, binding to BCL6	[76]	
5. Associated subunit						

Table 1 (continued)

Hugo name	Alternative names	Complex family	Bound complexes	Function	References	Implicated lncRNAs
CDK2AP1	DOC1	NuRD	NuRD/CHD3/4	NuRD recruitment to displace SWI/SNF complex	[75]	

ARID AT-rich interactive domain, *ARP* actin-related protein, *BAF* BRG1-associated factor, *BAZ* bromodomain adjacent to zinc finger domain, *BRD* bromodomain containing, *BPTF* bromodomain PHD finger transcription factor, *CHD* Chromodomain Helicase DNA-binding, *CHIRRC* CDCA7–HELLS ICF-related nucleosome remodeling complex, *EAF* Esa1- associated factor, *HAT* histone acetyltransferase, *HDAC* histone deacetylase, *ISWI* Imitation SWI, *Kac* acetyl-lysine, *Ksuc* succinyl-lysine, *Kub* ubiquitin-lysine, *NURF* nucleosome remodeling factor, *NuRD* Nucleosome remodeling and histone deacetylation, *SMARC* SWI/SNF related, matrix associated, actin-dependent regulator of chromatin, *SNF* Sucrose Non-Fermentable, *SWI* SWItch [128]

chromatin remodeling complexes: (1) the SWI/SNF, (2) the INO80/SWR1, (3) the ISWI and (4) the CHD family. The complexes of each chromatin remodeler family have distinct subunits and different functionalities that are listed in Table 1. Today, 34 lncRNAs have been described to interact with diverse subunits. Another 18 lncRNAs were reported to act as competing endogenous (ce) RNA with miRNA targets. We will summarize the current knowledge of the effect that lncRNAs have on the function of each chromatin remodeler complex family.

Chromatin remodeling complexes of the SWI/SNF family

Subunits of the SWI/SNF complexes

The SWI/SNF proteins were discovered in yeast as important factors regulating mating-type switching (SWI = SWItch) and regulating the use of different energy sources (SNF = Sucrose Non-Fermentable) [6]. The catalytic subunit in these complexes is either the ATP-dependent helicase Brahma (SMARCA2, BRM) or Brahma-related gene-1 (SMARCA4, BRG1). The core subunits are SMARCB1 (SNF5), SMARCC1 (BAF155) and SMARCC2 (BAF170) (Table 1). SWI/SNF remodeler complexes anchors to histone proteins and translocate 1–2 bp of DNA along the surface of the nucleosome, depending on the ATPase activity, this results in nucleosome sliding, or in destabilizing and removal of H2A–H2B dimers or entire histone cores [7], thus creating open chromatin structures.

Depending on subunit composition the SWI/SNF complexes are classified as PBAF (PolyBromo-Associated Factors) and BAF (BRG1- or BRM-Associated Factors). Exclusive subunits of the PBAF complex are ARID2, PBRM1 (BAF180), PHF10, and BRD7, in combination with the helicase SMARCA4. In the embryonic stem cell, the PBAF complex is important for maintaining the stem cell transcriptome [8]. Indeed, SMARCA4 plays an important role in the NANOG signaling pathway [9]. The PBAF complex inhibits growth through down-regulation of cell cycle genes CDK2, CDK4 and CCND1 and interference with the phosphorylated RB-pathway [10]. In addition, the PBAF complex is implicated in transcriptional repression at DNA double-strand breaks [11].

The BAF complex harbors the exclusive subunits ARID1A or ARID1B, BRD9 and SS18. They contain either the helicase SMARCA4 or SMARCA2. The difference between the PBAF and BAF complexes also results in different functionality [10, 12]. The SMARCA4- and SMARCA2-containing complexes have been assigned to ‘actively marked’ and ‘repressively marked’ chromatin

binding complexes, respectively [13]. In line with this, both complexes have differential affinity for histone modifications (PBAF complexes binds to H3K4-monomethylated, BAF binds to H3K4-trimethylated; [14]).

Whereas the PBAF complex is associated with an embryonic stem cell phenotype, BAF complexes seem to be tissue- and cell-type specific [15]. In particular, SMARCD3 subunits defines cardiac progenitors (cBAF complex) and SMARCD1 neuronal progenitors (npBAF complex), and the association with SS18L1 is associated with post-mitotic neurons (nBAF complex) [15].

Long non-coding RNAs and the SWI/SNF family

Amongst the chromatin remodelers, the SWI/SNF complex has frequently been shown to interact with lncRNAs (Table 2, Fig. 2a). In particular, SMARCA4 physically associates with primary transcribed RNA [16], suggesting a *cis*-acting function as shown for the lncRNAs lncFZD6 and lncTCF7. Some lncRNAs (e.g., MANTIS, SChLAP1) do not bind the SWI/SNF complex, but compete for DNA-binding at a particular chromosomal promoter gene region (e.g., MANTIS at the *ICAM-1* gene). SMARCA4-lncRNA interaction may also interfere with recruitment of SWI/SNF complexes at multiple gene regions (*trans*-acting) (e.g. LincRNA Cox2 and IL-7–AS). Recently, also SMARCB1 (SNF5) has been shown to interact with a pool of lncRNAs [17], including SWINGN and HOTAIR. Interestingly, lncBRM seem to inhibit SWI/SNF-SMARCA2 BAF complexes by its association with SMARCA2 (BRM), which in turn favors the assembly of SWI/SNF-SMARCA4 BAF complexes in liver cancer stem cells [18]. The BAF-specific subunit ARID1A has also been shown to bind lncRNAs (HOTAIR, MVIH, DGCR5 and LINC00163) thereby interfering with the transcription of several genes (Table 2 and Fig. 2a). Binding of lncRNA uc.291 with ACTL6A competes with BAF-ACTL6A binding and affects the expression of several differentiation genes in skin keratinocytes [19]. In addition, the MAPK pathway is affected by the interaction of SWI/SNF subunit BCL11A with CDKN2B-AS1 in lymphocytes and lncRNA uc.57 in breast cancer cells [20, 21]. Lots of lncRNAs have been shown to regulate gene expression by competing for miRNA binding [22], and for the SWI/SNF family lncRNA CASC15 and DLEU1 were reported to act as ceRNA for ARID1A and SMARCD1 transcripts, respectively [23, 24]. In addition, lncRNA DSCAM-AS1 may upregulate BCL11A expression [25].

Chromatin remodeling complexes of the INO80/SWR1 family

Subunits of the INO80/SWR1 complexes

The INO80/SWR1 factors were first identified in budding yeast in a screen for regulators of phospholipid biosynthesis (INOsitol requiring) and for switching from glycolytic to oxidative metabolism (SWR1) [26]. This family includes three major complexes, each having a unique ATPase unit (INO80, SRCAP or p400) in combination with the two ATP-driven helicases RUVBL1 and RUVBL2. The "effector" sub-units may recruit other enzymes, such as transcription factors and histone acetyltransferases. In particular, the p400-TRRAP-TIP60 complex mediates acetylation of histone H4 and H2A-tails. This acetylation stimulates the INO80 and SRCAP complexes to replace histone H2A with the 60% homologous histone H2A.Z. The H2A.Z diverges with H2A at two loops interacting with the nucleosomal DNA and at the tail section interacting with histone H3, which may result in a reduced stability or in nucleosome sliding [27]. Nucleosomes with histone H2A.Z are enriched at flanking regions of transcription start sites. In addition, H2A.Z can also be modified by acetylation, methylation, phosphorylation, SUMOylation and ubiquitination [28]. Removal of H2A.Z from DNA by the histone chaperone ANP32E and INO80 is a primary step in DNA repair [29]. The INO80 complex may recruit additional subunits, e.g., NFKRP, implicated in double strand break DNA repair [30].

Long non-coding RNAs and the INO80/SWR1 family

Both the lncRNA LCTS5 and HAND2-AS1 interact with the INO80 subunit, however, they have opposite effects (Fig. 2b). LCTS5 inhibits and HAND2-AS1 stimulates INO80 complex recruitment [31–33]. Similarly, ANRIL and Linc-YY1 both interact with transcription factor YY1 resulting, respectively, in recruitment to promoter loci of IL6/IL8 and eviction of polycomb repressive complex (PRC2) regulated promoters [34, 35]. Interaction of lncAKHE with YEATS4 from the histone acetylation p400-TRRAP complex results in activation of NOTCH2 signaling in hepatocellular carcinomas [36].

Several lncRNAs regulate the expression of INO80 subunits. UCHL5 mediates de-ubiquitination of NFRKB (INO80G), which is prevented by its interaction with lncRNA DRAIC [37]. The subsequent NFRKB ubiquitination-mediated degradation affects resection of DNA in double strand breaks DNA-repair [30]. In addition, lncRNA PTCSC3 inhibits INO80 expression by negatively regulating STAT3 [38]. The lncRNAs CR933609, RNCR3, and CASC7 were reported to act as ceRNAs targeting INO80D, BRD8

Table 2 LncRNAs known to interact with human chromatin remodeling complexes

lncRNA (name UCSC)	Binding to	Type of lncRNA	Action	Refs.
SWI/SNF subunits (Histone dimer/nucleosome removal, nucleosome sliding)				
Evf2 (DLX6-AS1)	SMARCA4	Antisense of DLX6	Competing with transcription factor DLX1 for binding to BRG1	[138]
UCA1	SMARCA4	Intergenic	Prevents binding to p21 promoter UCA1 expression regulated by ARID1A	[139, 140][$]
TUG1	SMARCA4	Intergenic, sharing promoter with MORC2	Inhibiting BRG1 degradation (MORC2 has PARP1-mediated ATPase and chromatin remodeling activities important in the DNA-damage response [141]).	[142]
Myheart (Mhrt)	SMARCA4	Antisense of MYH7	Binding to BRG1 prevents DNA binding of SWI/SNF complex	[143]
CPS1-IT1	SMARCA4	Intronic of CPS1 gene	Prevents binding to Cyr61 promoter region	[144][$]
NEAT1	SMARCA2, SMARCA4	Intergenic	Facilitates the organization of paraspeckle proteins (independent of SMARCA catalytic activity)	[145]
MALAT1	SMARCA4	intergenic	Complex with HDAC9 that represses vascular smooth muscle cell genes	[146]
LincRNA Cox2	SMARCA4	Antisense	Recruiting the SWI/SNF complex to inflammatory-response genes, e.g., Saa3 and Ccl5	[147]
IL-7–AS	p300/SMARCA4	Antisense	Recruiting HAT p300 followed by recruitment of SWI/SNF complex to activate inflammatory genes, (e.g., CCL2, CCL5, and IL-6)	[148]
LncFZD6 (BAALC-AS1)	SMARCA4	Antisense, sharing promoter with FZD6	Recruits SWI/SNF complex to FZD6 promoter,	[149][$]
lncTCF7	SMARCA4	antisense	Recruiting the SWI/SNF complex to the TCF7 promoter	[143]
lncBRM	SMARCA2	Antisense	Associates with BRM and favors assembly of BRG1-BAF complexes in liver cancer stem cells	[18][$]
SWINGN (LINC00565/ LINC00452)	SMARCB1	Intergenic, sharing promoter with GAS6	Transcriptional activation of several genes (e.g., GAS6, PDGFRB and COL1A1).	[17][$]
HOTAIR	SMARCB1, ARID1A	Intergenic, sharing promoter of HOXC11	Affecting expression of transcriptional repressor gene SNAIL	[150][$]
MVIH (AK094613)	ARID1A	Intragenic, overlapping RPS24 exons	Affects CDKN1A transcription	[151][$]
DGCR5	ARID1A	Intergenic	Promotes p21 expression	[152][$]
LINC00163	ARID1A	Intergenic	Stimulation of TCF21 expression	[153][$]
uc.291*	ACTL6A	Intragenic at LRMDA gene	Prevents inhibition by ACTL6A on epidermal differentiation genes	[19]
CDKN2B-AS1	BCL11A	Antisense	Inhibition of MAP4K1	[20]

Table 2 (continued)

lncRNA (name UCSC)	Binding to	Type of lncRNA	Action	Refs.
uc.57*	BCL11A	Intergenic upstream of BCL11A	Inhibition of PI3K/AKT and MAPK signaling pathways	[21]$
Xist	SWI/SNF	Intergenic	Binding to SWI/SNF complex inhibits binding to Xi-genes regions	[154]
MANTIS	DNA	Intragenic, ANXA4 gene	Preventing SWI/SNF complex binding at ICAM-1 promoter region	[155]
SChLAP1	DNA	intergenic	SMARCA4 associates frequently with primary transcripts including SChLAP1, preventing SWI/SNF complex binding	[16, 102, 156]$
CASC15	miR-221	Intergenic	ceRNA ARID1A	[23]$
DLEU1	miR-490-3p	Intergenic sharing promoter of DLEU2	ceRNA CDK1, CCND1 and SMARCD1	[24]$
INO80/SWR1 subunits (nucleosome replacement)				
LCTS5 (AC008610.1)	INO80	Intergenic	Binding to INO80 inhibits binding to enhancer regions near lung cancer associated genes.	[31]
HAND2-AS1 (lncHand2)	INO80	Antisense, sharing promoter with SCRG1	Recruiting the INO80 complex to e.g. the BMP-R1A and Nkx1-2 promoter	[32, 33]$
ANRIL	YY1	Antisense in INK4 Locus	Recruitment of YY1 to promoter loci of IL6 and IL8	[34]
Linc-YY1	YY1	Intragenic of YY	evict YY1/Polycomb repressive complex (PRC2) from target promoters	[35]
lncAKHE (AK056594)	YEATS4	Intragenic of TRIM55	activation of NOTCH2 signaling in hepatocellular carcinoma	[36]$
DRAIC	UCHL5	Intergenic	Promotes NFRKB (INO80G) ubiquitination-mediated degradation	[37]$
CASC7	miR-21	Intergenic (in between CHRAC1-Ago2)	ceRNA ING3	[41]$
RNCR3 (LINC00599)	miR-185-5p	Intergenic	ceRNA BRD8	[40]$
TUG1	miR-145 and YY1	Intergenic, sharing promoter with MORC2	Interference PRC2 complex	[157]$
CR933609	miRNA-5096	Overlapping INO80D 3'UTR	ceRNA INO80D	[39]$
SPAG5-AS1	miR-769-5p	Antisense	ceRNA YY1	[42]
HOTAIR	miR-1, miR-206	Intergenic, sharing promoter of HOXC11	ceRNA YY1	[43]$
LINC00899	miR-744-3p	Intergenic	ceRNA YY1	[44]$
LINC00668	miR-532-5p	Intergenic	ceRNA YY1	[45]$
Linc01134	miR-324-5p	Intergenic	ceRNA IGF2BP1, increased stability YY1 expression	[158]$
LncKdm2b (KDM2B-DT)	SRCAP	Antisense to Kdm2b	SRCAP complex recruitment to Zbtb3 gene, activating expression of this transcription factor	[59]

Table 2 (continued)

lncRNA (name UCSC)	Binding to	Type of lncRNA	Action	Refs.
ISWI (nucleosome spacing, DNA repair)				
LncKdm2b (KDM2B-DT)	SATB1-ISWI	Antisense to Kdm2b	NURF complex directed to Zfp292 promoter via recruitment of chromatin organizer SATB1 (also capable of recruiting SWI/SNF complexes)	[58]
NMR (LINC01672)	BPTF	Intergenic	NURF complex recruitment promoting ERK1/2 signaling pathway	[60]$
DLEU1	SMARCA1	Intergenic sharing promoter of DLEU2	NURF complex recruitment activating of KPNA3	[61]$
NEXN-AS1	BAZ1A	Antisense	Upregulation of NEXN expression, which is associated with atherosclerosis-related diseases	[62]
Lnc pRNA	BAZ2A	At intergenic spacer (IGS) sequences separating rDNA regions	Establishment of heterochromatin at ribosomal RNA genes (repression) e.g. RNA45SN2 (45S pre-rRNA)	[63, 159]
Paupar	TRIM28	Intragenic of PAX6-AS1	promotes KAP1 (H3K9me3 deposition) recruitment at a subset of distal targets, through formation Paupar-KAP1-PAX6 complex	[64]
BlackMamba (–)	HELLS	Intergenic upstream of KCNMA1	Gene expression (e.g., RGS1, CCL17, CCL22, PAK2, and KCNMA1) in anaplastic large cell lymphoma	[65]$
FGD5-AS1	miR-302e	Antisense	ceRNA CDCA7	[66]$
CHD (chromodomain helicase DNA-binding, DNA repair; NuRD, histone deacetylation/demethylase complex)				
CHASERR (LINC01578)	CHD2	Intergenic, upstream of CHD2	Transcriptional interference between Chaserr and CHD2 results in negative feedback loop	[87]
MATN1-AS1	miR-200b/c/429	Antisense	ceRNA CHD1	[91]$
LINC01410	miR-23c	Intergenic	ceRNA CHD7	[92]$
ANRIL	miR-34a	Antisense in INK4 Locus	ceRNA HDAC1	[93]$
SNHG15	miR-490-3p	miRNA host gene	ceRNA HDAC2	[94]$
ARAP1-AS1	miR-2110	antisense	ceRNA HDAC2	[95]$
HOTAIR	miR-326	Intergenic, sharing promoter of HOXC11	ceRNA MTA2	[96]$

*UltraConserved elements gene location at https://genome-test.gi.ucsc.edu/~hiram/hubs/GillBejerano/hg19/hg19.ultraConserved.bb. $Studies showing the role of lncRNA chromatin remodeling in cancer

and ING3, respectively [39–41]. SPAG5-AS1, HOTAIR, LINC00899 and LINC00668 were reported to act as ceRNAs targeting YY1 [42–45].

Chromatin remodeling complexes of the ISWI family

Subunits of the ISWI complexes

Initially discovered in a search for SWI/SNF genes in Drosophila [46], ISWI complexes regulate nucleosome spacing and are implicated in DNA repair. These chromatin

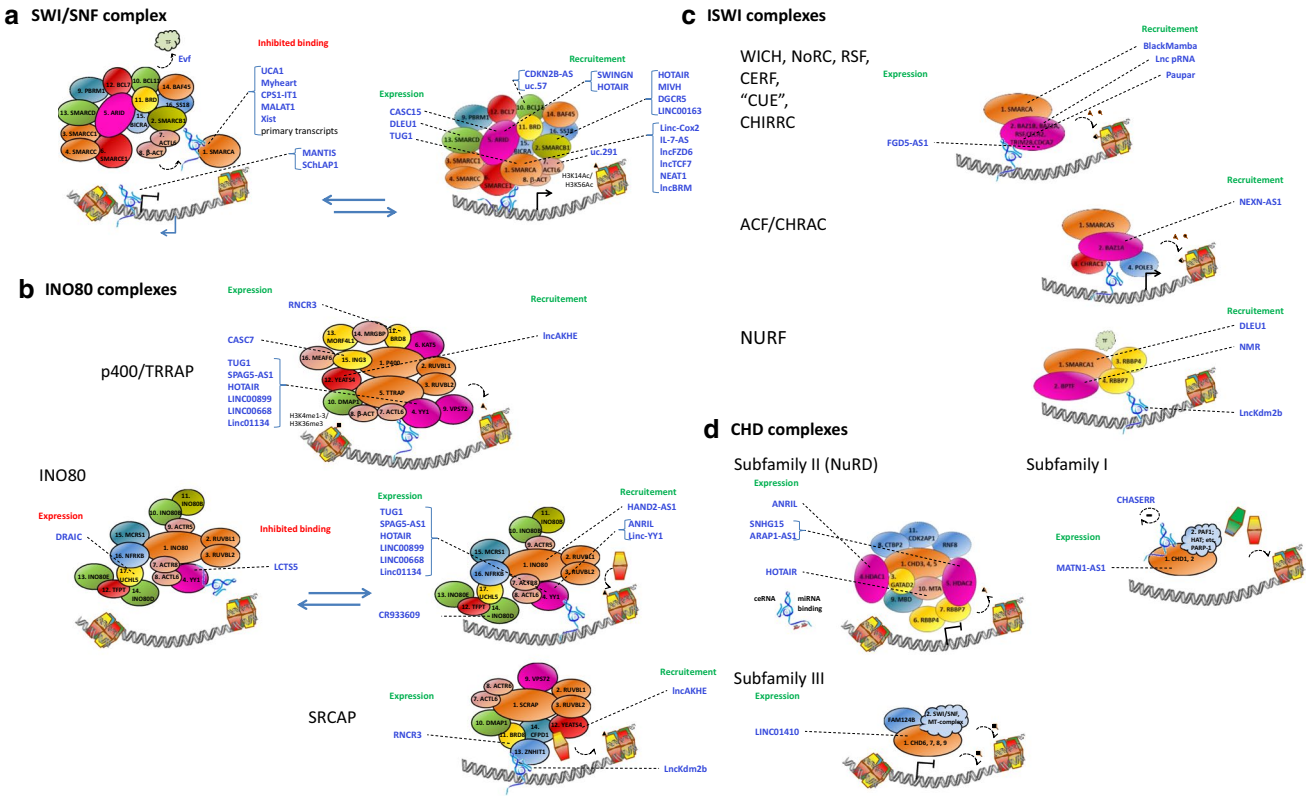

Fig. 2 Effects of long non-coding RNAs on chromatin remodeling complexes. LncRNAs (in blue) affect chromatin remodeling by changing subunit expression (transcription and/or competing for miRNA binding (ceRNAs)), by inhibiting complex binding to or by stimulating the recruitment to a genomic region. **a** Interaction of SWI/SNF complexes with lncRNA results in nucleosome sliding either further apart or closer together. **b** Histone replacement (H2A.Z) and modifications in nucleosomes are affected by lncRNAs that regulate the INO80 complexes. **c** LncRNAs known to regulate the different ISWI complexes are mainly recruiting these complexes. **d** Generally, lncRNAs that function as ceRNAs are known for the CHD family

remodelers are rather small complexes with 2–4 subunits. The best-known ATP-helicases in these complexes are SMARCA5 and SMARCA1. Both SMARCAD1 and SMARCA6 (HELLS) are SWI/SNF genes that can also be classified in this family. SMARCAD1-TRIM28 binds to H2A-ubiquitin and stimulates acetylation [47]. HELLS-CDC7A re-modulates the nucleosome to facilitate access to DNA for DNMT3B [48, 49]. SMARCA3 and SMARCAL1 are also ATP-driven DNA-binding helicases, but they are implicated, respectively, in DNA unwinding, and in the stabilization of the replication fork and they do not interact with nucleosomes [50, 51].

The WICH (SMARCA5-BAZ1B) and CERF (SMARCA1-CECR2) complexes affect the phosphorylation of histone H2A.X (γH2A.X) [52, 53]. This histone variant is highly homologous to H2A, but has a 13 amino acid extended C-tail that includes three phosphorylation sites (T136, S139 and Y142) [54]. Phosphorylation and ubiquitination of H2A.X are key events in the detection and response to double strand breaks of DNA damage.

The ACF (SMARCA5-BAZ1A) complex can either function as a heterodimer complex playing a role in double strand break repair [55], or as the CHRAC complex (ACF interacting with CHRAC1 and POLE3). Both complexes have very similar nucleosome sliding activity [56]. The NURF (SMARCA1-BPTF-RBBP4-RBBP7) complex also mediates nucleosome sliding and enhances recruitment of transcription factors and chromatin insulator protein elements [57].

Long non-coding RNAs and the ISWI family

The LncKdm2b (KDM2B-DT) seems to have a general function in recruiting chromatin remodeler complexes, as it was reported to direct both SRCAP (INO80 family) and NURF (ISWI family) complexes to two different zinc finger protein genes (Zbtb3 and Zfp292) [58, 59] (Fig. 2b, c). The NURF complex is also recruited through binding of lncRNA NMR to BPTF [60] and of DLEU1 to SMARCA1 [61]. Interestingly, DLEU1 has a double function as it seems to inhibit other complexes via miRNA-mediated decay of SMARCD1 (subunit of SWI/SNF complex). The NEXN-AS1-mediated

recruitment of BAZ1A (ACF complex) to NEXN was shown to upregulate its expression [62], which may suggest a *cis*-acting function similar to the recruitment of the SWI/SNF complex to primary transcribed RNA. The "lnc pRNA" has a specific action on ribosomal RNA genes as its interaction with PARP1 and BAZ2A (NoRC complex) represses these genes in particular [63].

Recently, it was shown that Pauper interacts with KAP1 to promote H3K9me3 deposition at a subset of distal targets, through formation of a Paupar-KAP1-PAX6 complex [64]. If the ATP-dependent helicase SMARCAD1, also interacting with KAP1, is part of this complex was not assessed in this study. The CHIRRC complex was reported to be affected by lncRNA BlackMamba via interaction with HELLS and by FGD5-AS1 as ceRNA inhibiting CDCA7 degradation [65, 66].

Chromatin remodeling complexes of the CHD family

Subunits of the CHD complexes

The chromodomain helicase DNA-binding (CHD) proteins that were initially identified as mammalian DNA-binding factors with a SWI-like helicase domain [67], are implicated in DNA repair and in recruitment of histone deacetylase/demethylase enzymes. CHD1 and CHD2 belong to the same subclass I. CHD1 has been shown to promote stabilization of H2A.X and the efficient repair of double strand breaks through homologous recombination [68]. CHD1 has no specific subunit, but has been described to interact with several factors, such as SSRP1 and the NCoR complex [69, 70]. CHD1 is a critical regulator of transcription initiation and elongation stimulating androgen receptor (AR)-mediated regulation and pluripotency gene expression (Oct 4 and Nanog) [67, 71].

The CHD2 protein has been shown to interact with PARP1 complex stimulating histone variant H3.3 deposition in non-homologous end joining (NHEJ) DNA-repair regions [72]. The histone H3.3 has only four amino acid differences compared to H3, introducing a posttranslational phosphorylation site (S31). Phosphorylated H3.3S31p stimulates p300-mediated histone H3K27 acetylation of neighboring nucleosomes [73]. In addition, the combination of histone H3.3 with H2A.Z in nucleosomes enriched near promoters and enhancers also evokes a loose nucleosomal packaging [74].

In the NuRD complex, one of the helicase from subfamily II, CHD3, CHD4 or CHD5 is present where they interact with GATAD2A/B. Interaction of the GATAD2 proteins with MDB2/3 results in recruitment of the NuRD histone deacetylase sub-complex (HDAC1/2, RBBP4/7, CTBP2, MTA1/2/3) [75]. The NuRD complex may also interact with several additional proteins, such as CDK2AP1, SALL1/4 and ZMYND8 that may direct the NuRD complex to specific regions [75]. CHD4 is also binding to RNF8, which is implicated in histone H1-ubiquitinylation and loading of the BRCA1 complex [76].

Subfamily III of CHD remodelers includes CHD6–CHD9. They interact with diverse transcription factors and posttranslationally modified histone H3. For CHD6, CHD7 and CHD8 it has been shown that they bind to the DNA strings in between nucleosomes. CHD6 and CHD7 both bind to short linker DNA, whereas CHD8 requires longer DNA sequences for binding and thus slides nucleosomes further apart [77]. CHD6 rather disrupts nucleosomes and was recently shown to relocalize to sites of DNA damage, suggesting a role in DNA repair [77, 78]. CHD7 and CHD9 have been shown to interact with chromatin remodelers from the SWI/SNF family (PBAF/BAF) [79]. CHD8 can directly bind β-catenin mediating interaction with histone H1 or other factors like the Gfi1b-complex regulating Wnt/β-catenin-dependent gene expression [80, 81]. CHD8 was further shown to interact with diverse methyltransferase complexes, such as BRD4-NSD3 [82], BACH1-MAFG-DNMT3 [83], SET1 (KMT2) [84] and WDR5-ASH2L-RbBP5 [85]. In addition, CHD7 was shown to interact with CHD8/FAM124B [86]. Both CHD7 and CHD8 are implicated in neuronal and developmental disorders (e.g., CHARGE, autism [77]).

Long non-coding RNAs and the CHD family

So far, only a few lncRNAs have been described in the context of CHD complexes (Fig. 2d). Recently, a negative regulation loop beween lncRNA CHASERR and CHD2 has been described. The *CHASERR* gene is located on the same strand (1.7 kb apart) as the *CHD2* gene at chr15q26. The CHASERR transcript can be bound by CHD2 protein and regulate transcription of the *CHD2* gene [87]. Some lncRNAs have been shown to affect transcriptional regulation of CHD subunits. The lncRNA MTA2TR transcriptionally upregulates MTA2 expression [88]. LUCAT1 upregulates MTA1 in cervical cancer and HDAC1 in papillary thyroid cancer [89, 90]. Six lncRNAs have been described as ceRNAs for CHD subunits (MATN1-AS1 ceRNA CHD1 [91], LINC01410 ceRNA CHD7 [92], ANRIL ceRNA HDAC1 [93], SNHG15 and ARAP1-AS1 ceRNA HDAC2 [94, 95] and HOTAIR ceRNA MTA2 [96]).

Concluding remarks

The lncRNAs described in this review often recruit chromatin remodeler complexes at promoter and enhancer regions by binding a particular subunit. Some other lncRNAs, not discussed herein, are related to the chromatin remodeler complex through their position, but they seem to have unrelated functions (lnc-Arid2-IR, lnc-SMARCC2, lnc-MDB2, lnc-Dpf3) [97–100].

A recurrent chromatin regulatory mechanism is recruitment of remodeler complexes by interaction with primary transcribed RNAs, either by antisense lncRNA encoded at genomic proximity, or by *trans*-acting lncRNAs (e.g., LncKdm2b-mediated recruitment of SRCAP and NURF complexes [58, 59]). The interaction of chromatin remodeler subunits with some lncRNAs results in a distinct cellular function. For example, interaction of lncBRM with BRM inhibits the SWI/SNF complex associated with a differentiated phenotype and favors assembly of a stem cell-related SWI/SNF complex [18]. Also lnc-pRNA regulates primarily ribosomal RNA gene expression via the NoRC complex [63]. The interaction between chromatin remodeler subunits, in particular, the flexible and/or successive nature of complex formation in the different processes like NHEJ or HR-mediated DNA repair will be an important asset to further define the role of lncRNAs in these processes. The role of the chromatin remodelers in the different stages of DNA repair is closely related to cancer (e.g., [67, 101–103]). Indeed, from the lncRNAs that affect the chromatin remodeling complexes (Table 2) at least 35 are associated with cancer. Cellular capture-based approaches can be employed to further analyze the interactions of lncRNAs with the chromatin remodeler complexes, using either RNA-specific, DNA-locus specific or genome wide approaches (reviewed by [104]), although non-specific background interaction signals and sequential complex formation remains a major issue. The CRISPR-Cas9 engineering revolution now enables in situ capture of RNA-chromatin interactions by biotinylated dCas9 [104], which may also reveal implication of lncRNAs in the cancer-related nucleosome remodeling evidenced by Druliner et al. [105]. In addition, analyzing CRISPR/Cas9-based gene perturbations (e.g., lncRNAs invalidation/activation) combined with single cell sequence analysis (recently reported by [106–108]), may unravel the specific actions of lncRNAs, that are measured as low expressed in bulk-analyzing experiments but can be abundantly expressed in individual, and phenotype-specific cells (e.g., shown in neuronal cells and cardiomyocytes [109, 110]). Although today still presenting a huge technical challenge, these analyses could be combined with studies of chromatin organization and protein interactions in single cells [111]. Identifying the lncRNAs that contribute to epigenetic regulation by controlling the specific chromatin modifications associated with disease, may result in interesting novel targets for e.g. oligonucleotide-based therapy [112]. In particular, DNA-Gapmer antisense oligonucleotides, of which several have received therapeutic FDA-approval, are suitable for targeting lncRNAs as they can evoke RNASEH1-mediated transcript degradation both within the nucleus and cytosol.

In conclusion, the description of lncRNAs interacting with chromatin remodeler complexes in this review, intends to highlight the importance of lncRNA-mediated chromatin remodeling via remodeler complexes in physiological and carcinogenic conditions.

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