Journal of Disease Mechanisms and Genomics
Volume 1; Issue 1; April 2026

JDMG Journal of Disease Mechanisms and Genomics www.jdmg.org

Chromatin Remodeling Complexes in Tumor Progression: From SWI/SNF Dysfunction to Precision Oncology

Asad Khani[a], Nabil Akbar[b]

[a]*Department of Biochemistry, Peshawar University, Peshawar, Pakistan*
[b]*Faculty of Medicine, University of Punjab, Pakistan*

Correspondence: Asadk9720@gmail.com

Article history:
Received 15 February 2026
Accepted 18 March 2026
Published 20 April 2026

Keywords:
Chromatin remodeling; SWI/SNF complex; epigenetics; tumor progression; precision oncology

Abstract

Chromatin remodeling complexes are fundamental regulators of genome accessibility, transcriptional plasticity, DNA repair, and epigenetic homeostasis. Among these, the SWI/SNF (Switch/Sucrose Non-Fermentable) family has emerged as one of the most frequently altered chromatin remodeling systems in human malignancies. Accumulating evidence demonstrates that mutations, deletions, and epigenetic dysregulation of SWI/SNF subunits contribute substantially to tumor initiation, progression, metastatic dissemination, and therapeutic resistance. Dysfunction of key components including ARID1A, SMARCB1, SMARCA4, and PBRM1 reshapes chromatin architecture, perturbs enhancer activity, and promotes oncogenic transcriptional programs across diverse cancer types. Recent advances in next-generation sequencing, epigenomic profiling, and precision oncology have further elucidated the intricate interplay between chromatin remodeling defects and tumor microenvironment dynamics, immune evasion, and lineage plasticity. Importantly, the therapeutic vulnerability associated with SWI/SNF deficiency has stimulated the development of targeted interventions, including EZH2 inhibitors, bromodomain inhibitors, synthetic lethality-based approaches, and immunotherapeutic strategies. This review provides an overview of the molecular architecture of chromatin remodeling complexes, with particular emphasis on SWI/SNF dysfunction in cancer biology and its emerging translational relevance in precision oncology.

1. Introduction

Chromatin The eukaryotic genome is intricately packaged into chromatin, a highly dynamic nucleoprotein structure that regulates DNA accessibility and transcriptional fidelity. Chromatin organization is not static; rather, it undergoes continuous structural remodeling mediated by ATP-dependent chromatin remodeling complexes and epigenetic modifiers that collectively orchestrate gene expression, DNA replication, and genomic stability [1,2]. Aberrations in these regulatory systems have emerged as pivotal drivers of tumorigenesis, contributing to uncontrolled proliferation, impaired differentiation, and

therapeutic resistance across numerous malignancies [3].

Among the major ATP-dependent chromatin remodeling families, the SWI/SNF complex has attracted substantial attention owing to its recurrent genetic alterations in approximately 20–25% of human cancers [4]. Originally identified in yeast as regulators of mating type switching and sucrose fermentation, mammalian SWI/SNF complexes comprise multiple evolutionarily conserved subunits that function cooperatively to reposition nucleosomes and regulate enhancer accessibility [5]. These complexes are broadly categorized into canonical BAF (cBAF), polybromo-associated BAF (PBAF), and non-canonical BAF (ncBAF) assemblies, each possessing distinct structural compositions and biological functions [6].

Genomic studies have identified frequent mutations in core SWI/SNF subunits including ARID1A, SMARCB1, SMARCA4, ARID2, and PBRM1 in diverse tumor types such as ovarian clear cell carcinoma, renal cell carcinoma, hepatocellular carcinoma, rhabdoid tumors, and lung cancer [7,8]. Loss-of-function alterations in these genes disrupt chromatin accessibility landscapes and facilitate oncogenic transcriptional reprogramming. For instance, ARID1A deficiency has been associated with impaired DNA damage repair, inflammatory signaling dysregulation, and enhanced sensitivity to synthetic lethal therapeutic strategies [9]. Similarly, SMARCB1 inactivation represents a hallmark event in malignant rhabdoid tumors and epithelioid sarcomas, underscoring the tumor suppressive role of SWI/SNF complexes [10] (Figure 1).

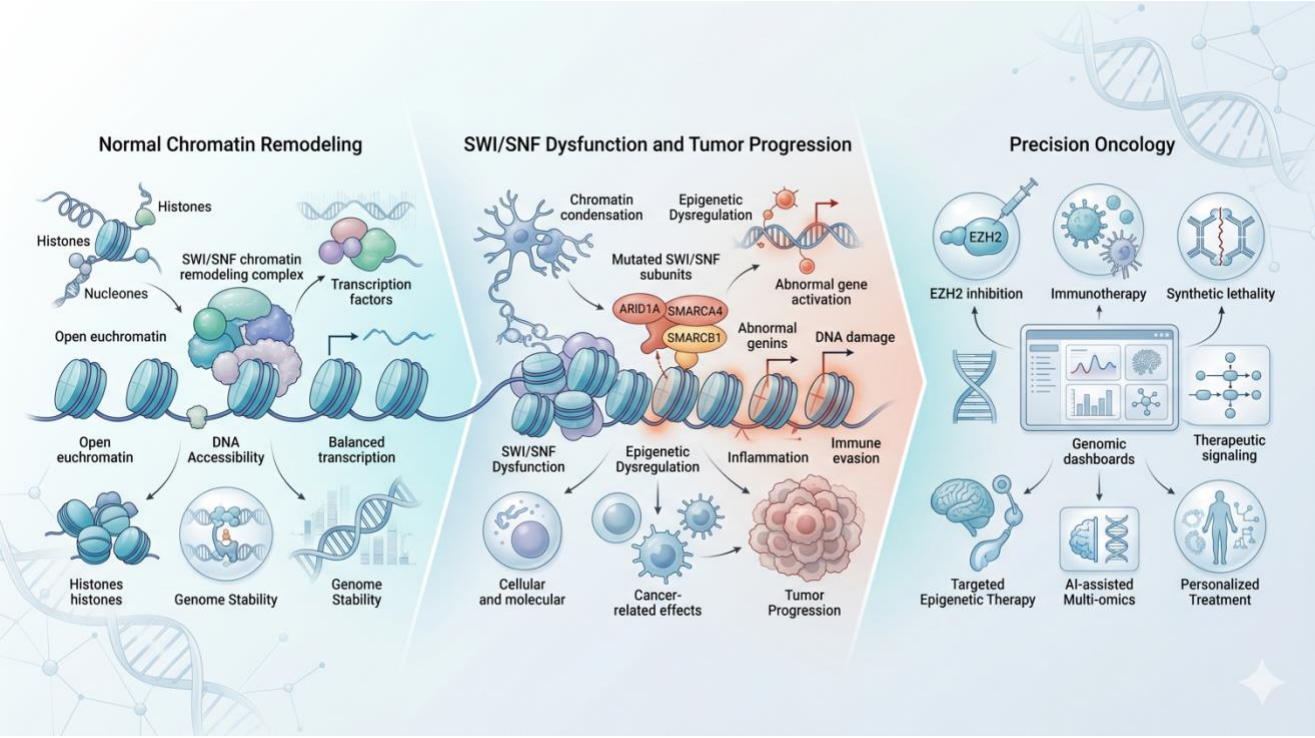

Figure 1: Schematic overview of the role of SWI/SNF chromatin remodeling complexes in tumor progression and precision oncology. The left panel illustrates physiological chromatin remodeling mediated by functional SWI/SNF complexes, which maintain chromatin accessibility, transcriptional regulation, DNA repair, and genomic stability. The central panel depicts the consequences of SWI/SNF dysfunction, including mutations or loss of key subunits such as ARID1A, SMARCA4, and SMARCB1, leading to epigenetic dysregulation, aberrant transcriptional programs, genomic instability, immune evasion, and tumor progression. The right panel highlights emerging precision oncology strategies targeting SWI/SNF-deficient malignancies, including EZH2 inhibition, synthetic lethality-based therapies, immunotherapy, and multi-omics-guided personalized treatment approaches.

Recent advances in epigenomics and single-cell sequencing technologies have expanded the understanding of chromatin remodeling dysfunction beyond classical tumor suppressor paradigms. Emerging evidence suggests that

SWI/SNF aberrations contribute to intratumoral heterogeneity, immune evasion, metabolic rewiring, and lineage plasticity during cancer evolution [11]. Furthermore, antagonistic interactions between SWI/SNF complexes and Polycomb Repressive Complex 2 (PRC2), particularly through enhancer of zeste homolog 2 (EZH2), have generated considerable interest in epigenetic-targeted therapeutics [12]. The clinical success of EZH2 inhibitors in SWI/SNF-deficient malignancies exemplifies the growing translational significance of chromatin remodeling biology in precision oncology [13].

In parallel, integrative multi-omics approaches incorporating genomics, transcriptomics, epigenomics, and proteomics are reshaping the landscape of cancer classification and therapeutic stratification [14]. The identification of chromatin remodeling-associated biomarkers now offers opportunities for personalized therapeutic interventions, including synthetic lethality-based treatments, immune checkpoint blockade combinations, and targeted epigenetic modulation [15].

Collectively, chromatin remodeling complexes occupy a central position at the intersection of epigenetic regulation and cancer biology. Understanding the mechanistic consequences of SWI/SNF dysfunction is therefore essential for elucidating tumor progression pathways and advancing precision oncology strategies.

2. Methodology

This review was conducted through a comprehensive literature survey of peer-reviewed articles indexed in PubMed, Scopus, Web of Science, and Google Scholar databases. Relevant studies published primarily between 2000 and 2026 were identified using combinations of keywords including "chromatin remodeling", "SWI/SNF", "BAF complex", "epigenetics", "tumor progression", "ARID1A", "SMARCA4", "SMARCB1", and "precision oncology". Original research articles, systematic reviews, meta-analyses, and landmark mechanistic studies focusing on chromatin remodeling dysfunction in cancer were prioritised. Additional emphasis was placed on studies investigating translational applications, therapeutic vulnerabilities, and emerging epigenetic-targeted interventions in SWI/SNF-deficient malignancies. Relevant references cited within selected articles were also manually screened to ensure comprehensive coverage of the topic.

3. Molecular Architecture and Biological Functions of SWI/SNF Complexes

ATP-dependent chromatin remodeling complexes regulate chromatin accessibility through nucleosome repositioning, eviction, and restructuring, thereby modulating transcriptional activity and genome stability [16]. Among these complexes, the SWI/SNF family constitutes one of the most evolutionarily conserved chromatin remodeling systems and plays indispensable roles in cellular differentiation, lineage specification, DNA repair, and tumor suppression [17]. Mammalian SWI/SNF complexes are structurally heterogeneous multiprotein assemblies composed of approximately 12–15 subunits organized into distinct functional modules [18]. Based on subunit composition, these complexes are classified into canonical BAF (cBAF), polybromo-associated BAF (PBAF), and non-canonical BAF (ncBAF/GBAF) complexes [19]. Central ATPase activity is mediated by mutually exclusive catalytic subunits SMARCA4 (BRG1) or SMARCA2 (BRM), which hydrolyze ATP to reposition nucleosomes and facilitate chromatin accessibility [20].

The functional specificity of SWI/SNF complexes is largely determined by accessory subunits such as ARID1A, ARID1B, PBRM1, DPF2, BRD7, and BRD9 [21]. These components guide chromatin targeting, enhancer selection, and transcription factor interactions. Importantly, tissue-specific subunit configurations allow SWI/SNF complexes to coordinate context-dependent transcriptional programs during embryogenesis and tissue homeostasis [22]. SWI/SNF complexes frequently antagonize Polycomb Repressive Complex 2 (PRC2), which mediates transcriptional repression through histone H3 lysine 27 trimethylation (H3K27me3) [23]. This epigenetic antagonism maintains balanced chromatin states required for cellular differentiation and genome integrity.

Consequently, disruption of SWI/SNF activity results in aberrant chromatin compaction, transcriptional dysregulation, and oncogenic transformation [24].

4. SWI/SNF Dysfunction in Human Cancers

Genomic sequencing studies have revealed that mutations in the SWI/SNF complex occur in approximately one-quarter of all human malignancies, placing these complexes among the most frequently altered epigenetic regulators in cancer [25]. Alterations include missense mutations, frameshift mutations, deletions, and epigenetic silencing events affecting both catalytic and accessory subunits.

4.1 ARID1A Mutations and Oncogenic Reprogramming

ARID1A is one of the most commonly mutated SWI/SNF subunits in cancer, particularly in ovarian clear cell carcinoma, gastric cancer, hepatocellular carcinoma, colorectal carcinoma, and endometrial carcinoma [26]. Loss of ARID1A disrupts enhancer accessibility and impairs transcriptional regulation of tumor suppressive pathways [27]. Mechanistically, ARID1A deficiency contributes to defective DNA damage repair through impaired homologous recombination and mismatch repair signaling [28]. In addition, ARID1A-mutant tumors frequently exhibit increased inflammatory signaling, altered cytokine production, and enhanced immune checkpoint expression [29]. These molecular changes create therapeutic vulnerabilities that may be exploited using PARP inhibitors, EZH2 inhibitors, and immune checkpoint blockade strategies [30]. Emerging evidence also indicates that ARID1A loss promotes metabolic reprogramming and oxidative stress adaptation, facilitating tumor cell survival within hypoxic tumor microenvironments [31].

4.2 SMARCB1 Deficiency in Aggressive Malignancies

SMARCB1 (INI1/SNF5) functions as a core tumor suppressor subunit within SWI/SNF complexes [32]. Biallelic inactivation of SMARCB1 represents a defining molecular feature of malignant rhabdoid tumors, epithelioid sarcoma, and renal medullary carcinoma [33]. Loss of SMARCB1 results in widespread epigenetic dysregulation, aberrant MYC activation, and uncontrolled cellular proliferation [34]. Furthermore, SMARCB1-deficient tumors exhibit profound dependency on EZH2-mediated transcriptional repression, establishing the biological rationale for EZH2-targeted therapies [35]. The FDA approval of the EZH2 inhibitor tazemetostat for epithelioid sarcoma highlights the translational significance of targeting epigenetic vulnerabilities associated with SWI/SNF deficiency [36].

4.3 SMARCA4 and Tumor Plasticity

SMARCA4 mutations are prevalent in thoracic malignancies, ovarian small cell carcinoma of hypercalcemic type, and subsets of lung adenocarcinoma [37]. Loss of SMARCA4 disrupts enhancer regulation and promotes lineage plasticity, epithelial-to-mesenchymal transition (EMT), and metastatic dissemination [38]. SMARCA4-deficient tumors are frequently associated with aggressive clinical behavior and poor prognosis [39]. Recent studies have demonstrated increased sensitivity of these tumors to bromodomain inhibitors, oxidative phosphorylation inhibitors, and immunotherapy combinations [40].

5. Chromatin Remodeling and Tumor Microenvironment Interactions

Beyond intrinsic tumor cell regulation, SWI/SNF complexes influence tumor progression through modulation of the tumor microenvironment (TME) [41]. Chromatin remodeling abnormalities alter cytokine secretion profiles, immune cell recruitment, antigen presentation pathways, and interferon signaling networks [42]. ARID1A-deficient tumors frequently display increased tumor mutational burden and enhanced PD-L1 expression, potentially sensitizing these cancers to immune checkpoint inhibitors [43]. Similarly, PBRM1 mutations in renal cell carcinoma have been associated with improved responses to immunotherapy [44]. Recent single-cell epigenomic studies further suggest that chromatin remodeling dysfunction contributes to immune evasion through suppression of antigen processing machinery and

induction of exhausted T-cell phenotypes [45]. These findings position SWI/SNF-deficient tumors as promising candidates for combined epigenetic-immunotherapeutic approaches.

6. Synthetic Lethality and Therapeutic Vulnerabilities

Synthetic lethality has emerged as one of the most promising therapeutic paradigms for targeting SWI/SNF-deficient cancers [46]. Because chromatin remodeling complexes operate within interconnected epigenetic networks, loss of one component frequently creates dependency on compensatory pathways. The antagonistic relationship between SWI/SNF complexes and PRC2 has generated considerable interest in EZH2 inhibition [47]. Preclinical studies demonstrated selective sensitivity of ARID1A- and SMARCB1-deficient tumors to EZH2 inhibitors, findings that subsequently translated into clinical applications [48].

Additional synthetic lethal interactions include dependencies involving ATR, PARP, CDK4/6, BRD9, and DNA damage repair pathways [49]. Bromodomain-containing proteins such as BRD9 and BRD4 are increasingly recognized as therapeutic targets in SWI/SNF-mutant malignancies [50]. Furthermore, CRISPR-based functional genomic screens continue to identify novel epigenetic dependencies that may expand therapeutic opportunities in precision oncology [51].

7. Emerging Role of Multi-Omics and Precision Oncology

Recent advances in multi-omics technologies have revolutionized the understanding of chromatin remodeling abnormalities in cancer biology [52]. Integrative analyses combining genomics,

transcriptomics, epigenomics, proteomics, and metabolomics now allow comprehensive characterization of SWI/SNF-deficient tumor states [53]. Single-cell sequencing technologies have further uncovered substantial intratumoral heterogeneity driven by chromatin remodeling defects [54]. These approaches reveal dynamic transitions between transcriptional states associated with metastasis, drug resistance, and immune escape.

Artificial intelligence-assisted bioinformatics platforms are increasingly applied to identify chromatin remodeling-associated biomarkers and therapeutic signatures [55]. Such strategies are expected to enhance patient stratification and facilitate personalized therapeutic interventions in precision oncology. Despite substantial progress, important challenges remain regarding resistance mechanisms, tumor context specificity, and long-term therapeutic efficacy [56]. Continued integration of epigenomics, functional genomics, and translational oncology will therefore be essential for optimizing future chromatin-targeted therapies.

8. Conclusion

Chromatin remodeling complexes represent central regulators of genome organization and transcriptional plasticity in both physiological and pathological contexts. Dysregulation of SWI/SNF complexes profoundly alters chromatin accessibility landscapes, driving oncogenic transformation, tumor progression, immune evasion, and therapeutic resistance. Advances in molecular oncology have significantly expanded the understanding of SWI/SNF biology, revealing multiple translational vulnerabilities exploitable through synthetic lethality-based approaches and epigenetic therapies. The emergence of precision oncology and multi-omics technologies further highlights the clinical relevance of chromatin remodeling dysfunction in cancer management. Future investigations integrating epigenomics, immunotherapy, and functional genomics are expected to accelerate the development of individualized therapeutic strategies for SWI/SNF-deficient malignancies.

References

1. Kornberg RD. Chromatin structure: a repeating unit of histones and DNA. *Science.* 1974;184(4139):868–871.
2. Clapier CR, Iwasa J, Cairns BR, Peterson CL. Mechanisms of action and regulation of ATP-

dependent chromatin-remodelling complexes. *Nat Rev Mol Cell Biol*. 2017;18(7):407–422.
3. Feinberg AP, Koldobskiy MA, Göndör A. Epigenetic modulators, modifiers and mediators in cancer aetiology and progression. *Nat Rev Genet*. 2016;17(5):284–299.
4. Kadoch C, Hargreaves DC, Hodges C, et al. Proteomic and bioinformatic analysis of mammalian SWI/SNF complexes identifies extensive roles in human malignancy. *Nat Genet*. 2013;45(6):592–601.
5. Wang W, Côté J, Xue Y, et al. Purification and biochemical heterogeneity of the mammalian SWI-SNF complex. *EMBO J*. 1996;15(19):5370–5382.
6. Mashtalir N, D'Avino AR, Michel BC, et al. Modular organization and assembly of SWI/SNF family chromatin remodeling complexes. *Cell*. 2018;175(5):1272–1288.
7. Shain AH, Pollack JR. The spectrum of SWI/SNF mutations, ubiquitous in human cancers. *PLoS One*. 2013;8(1):e55119.
8. Helming KC, Wang X, Roberts CWM. Vulnerabilities of mutant SWI/SNF complexes in cancer. *Cancer Cell*. 2014;26(3):309–317.
9. Bitler BG, Aird KM, Garipov A, et al. Synthetic lethality by targeting EZH2 methyltransferase activity in ARID1A-mutated cancers. *Nat Med*. 2015;21(3):231–238.
10. Versteege I, Sévenet N, Lange J, et al. Truncating mutations of hSNF5/INI1 in aggressive paediatric cancer. *Nature*. 1998;394(6689):203–206.
11. Centore RC, Sandoval GJ, Soares LMM, et al. Mammalian SWI/SNF chromatin remodeling complexes: emerging mechanisms and therapeutic strategies. *Trends Genet*. 2020;36(12):936–950.
12. Wilson BG, Wang X, Shen X, et al. Epigenetic antagonism between polycomb and SWI/SNF complexes during oncogenic transformation. *Cancer Cell*. 2010;18(4):316–328.
13. Italiano A, Soria JC, Toulmonde M, et al. Tazemetostat, an EZH2 inhibitor, in relapsed or refractory B-cell non-Hodgkin lymphoma and advanced solid tumours: a first-in-human, open-label, phase 1 study. *Lancet Oncol*. 2018;19(5):649–659.
14. Hoadley KA, Yau C, Hinoue T, et al. Cell-of-origin patterns dominate the molecular classification of 10,000 tumors from 33 types of cancer. *Cell*. 2018;173(2):291–304.
15. Mittal P, Roberts CWM. The SWI/SNF complex in cancer — biology, biomarkers and therapy. *Nat Rev Clin Oncol*. 2020;17(7):435–448.
16. Becker PB, Workman JL. Nucleosome remodeling and epigenetics. Cold Spring Harb Perspect Biol. 2013;5(9):a017905.
17. Narlikar GJ, Sundaramoorthy R, Owen-Hughes T. Mechanisms and functions of ATP-dependent chromatin-remodeling enzymes. Cell. 2013;154(3):490–503.
18. Kadoch C, Crabtree GR. Mammalian SWI/SNF chromatin remodeling complexes and cancer: Mechanistic insights gained from human genomics. Sci Adv. 2015;1(5):e1500447.
19. Michel BC, D'Avino AR, Cassel SH, et al. A non-canonical SWI/SNF complex is a synthetic lethal target in cancers driven by BAF complex perturbation. Nat Cell Biol. 2018;20(12):1410–1420.
20. Wang W, Xue Y, Zhou S, et al. Diversity and specialization of mammalian SWI/SNF complexes. Genes Dev. 1996;10(17):2117–2130.
21. Alpsoy A, Dykhuizen EC. Glioma tumor suppressor candidate region gene 1 (GLTSCR1) and its paralog GLTSCR1-like form SWI/SNF chromatin remodeling subcomplexes. J Biol Chem. 2018;293(10):3892–3903.
22. Ho L, Crabtree GR. Chromatin remodelling during development. Nature. 2010;463(7280):474–484.
23. Stanton BZ, Hodges C, Calarco JP, et al. Smarca4 ATPase mutations disrupt direct eviction of PRC1 from chromatin. Nat Genet. 2017;49(2):282–288.
24. Roberts CWM, Orkin SH. The SWI/SNF complex—chromatin and cancer. Nat Rev Cancer. 2004;4(2):133–142.
25. Hodges C, Kirkland JG, Crabtree GR. The many roles of BAF (mSWI/SNF) and PBAF complexes in cancer. Cold Spring Harb Perspect Med. 2016;6(8):a026930.
26. Wu JN, Roberts CWM. ARID1A mutations in cancer: another epigenetic tumor suppressor? Cancer Discov. 2013;3(1):35–43.
27. Mathur R. ARID1A loss in cancer: towards a mechanistic understanding. Pharmacol Ther. 2018;190:15–23.
28. Shen J, Ju Z, Zhao W, et al. ARID1A deficiency promotes mutability and potentiates therapeutic antitumor immunity unleashed by immune checkpoint blockade. Nat Med. 2018;24(5):556–562.
29. Fukumoto T, Magno E, Zhang R. SWI/SNF complexes in ovarian cancer: mechanistic insights and therapeutic implications. Mol Cancer Res. 2018;16(12):1819–1825.
30. Williamson CT, Miller R, Pemberton HN, et al. ATR inhibitors as a synthetic lethal therapy for

tumours deficient in ARID1A. Nat Commun. 2016;7:13837.
31. Zhang L, Shi L, Zhao X, et al. Metabolic reprogramming mediated by ARID1A deficiency in cancer progression. Cancer Lett. 2021;512:1–10.
32. Biegel JA, Zhou JY, Rorke LB, et al. Germ-line and acquired mutations of INI1 in atypical teratoid and rhabdoid tumors. Cancer Res. 1999;59(1):74–79.
33. Hollmann TJ, Hornick JL. INI1-deficient tumors: diagnostic features and molecular genetics. Am J Surg Pathol. 2011;35(10):e47–e63.
34. Wang X, Sansam CG, Thom CS, et al. Oncogenesis caused by loss of the SNF5 tumor suppressor is dependent on MYC activation. Cancer Res. 2009;69(21):8099–8108.
35. Knutson SK, Warholic NM, Wigle TJ, et al. Durable tumor regression in genetically altered malignant rhabdoid tumors by inhibition of methyltransferase EZH2. Proc Natl Acad Sci USA. 2013;110(19):7922–7927.
36. Gounder MM, Schöffski P, Jones RL, et al. Tazemetostat in advanced epithelioid sarcoma with loss of INI1/SMARCB1: an international, open-label, phase 2 basket study. Lancet Oncol. 2020;21(11):1423–1432.
37. Herpel E, Rieker RJ, Dienemann H, et al. SMARCA4 and SMARCA2 deficiency in thoracic tumors. Virchows Arch. 2017;471(4):481–489.
38. Romero OA, Torres-Diz M, Pros E, et al. The tumour suppressor and chromatin-remodelling factor BRG1 antagonizes Myc activity and promotes cell differentiation in human cancer. EMBO Mol Med. 2012;4(7):603–616.
39. Le Loarer F, Watson S, Pierron G, et al. SMARCA4 inactivation defines a group of undifferentiated thoracic malignancies. Nat Genet. 2015;47(10):1200–1205.
40. Hoffman GR, Rahal R, Buxton F, et al. Functional epigenetics approach identifies BRM/SMARCA2 as a critical synthetic lethal target in BRG1-deficient cancers. Proc Natl Acad Sci USA. 2014;111(8):3128–3133.
41. Topper MJ, Vaz M, Chiappinelli KB, et al. Epigenetic therapy ties MYC depletion to reversing immune evasion and treating lung cancer. Cell. 2017;171(6):1284–1300.
42. Pan D, Kobayashi A, Jiang P, et al. A major chromatin regulator determines resistance of tumor cells to T cell-mediated killing. Science. 2018;359(6377):770–775.
43. Samstein RM, Lee CH, Shoushtari AN, et al. Tumor mutational load predicts survival after immunotherapy across multiple cancer types. Nat Genet. 2019;51(2):202–206.
44. Miao D, Margolis CA, Gao W, et al. Genomic correlates of response to immune checkpoint therapies in clear cell renal cell carcinoma. Science. 2018;359(6377):801–806.
45. Hsu JM, Xia W, Hsu YH, et al. STT3-dependent PD-L1 accumulation on cancer stem cells promotes immune evasion. Nat Commun. 2018;9(1):1908.
46. Ogiwara H, Takahashi K, Sasaki M, et al. Targeting the vulnerability of glutathione metabolism in ARID1A-deficient cancers. Cancer Cell. 2019;35(2):177–190.
47. Kim KH, Roberts CWM. Targeting EZH2 in cancer. Nat Med. 2016;22(2):128–134.
48. Chan-Penebre E, Armstrong K, Drew A, et al. Selective killing of SMARCA2- and SMARCA4-deficient small cell carcinoma cells by inhibition of EZH2. Mol Cancer Ther. 2017;16(5):850–861.
49. Garutti M, Pelizzari G, Bartoletti M, et al. BRD4 inhibition in cancer therapy: mechanisms and clinical implications. Cancers (Basel). 2021;13(3):477.
50. Hohmann AF, Martin LJ, Minder JL, et al. Sensitivity and engineered resistance of myeloid leukemia cells to BRD9 inhibition. Nat Chem Biol. 2016;12(9):672–679.
51. Manguso RT, Pope HW, Zimmer MD, et al. In vivo CRISPR screening identifies Ptpn2 as a cancer immunotherapy target. Nature. 2017;547(7664):413–418.
52. Hasin Y, Seldin M, Lusis A. Multi-omics approaches to disease. Genome Biol. 2017;18(1):83.
53. Argelaguet R, Cuomo ASE, Stegle O, Marioni JC. Computational principles and challenges in single-cell data integration. Nat Biotechnol. 2021;39(10):1202–1215.
54. Suvà ML, Tirosh I. Single-cell RNA sequencing in cancer: lessons learned and emerging challenges. Mol Cell. 2019;75(1):7–12.
55. Ching T, Himmelstein DS, Beaulieu-Jones BK, et al. Opportunities and obstacles for deep learning in biology and medicine. J R Soc Interface. 2018;15(141):20170387.
56. Dawson MA. The cancer epigenome: Concepts, challenges, and therapeutic opportunities. Science. 2017;355(6330):1147–1152.