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High-Fat Diet-Induced DNA Methylation Changes in Adipose Tissue Genes: A Murine Model Study

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ABSTRACT

Background: High-fat diet (HFD)-induced obesity in rodents is associated with altered expression of adipose genes governing adipogenesis, thermogenesis, and inflammation, and a growing literature implicates DNA methylation as a mediating mechanism. Objective: This narrative review synthesizes published murine and rat evidence on HFD-induced DNA methylation changes at adipose tissue genes, without reporting new experimental data. Methods: Individually verified peer-reviewed studies on methylation of leptin, Pparg, Ucp1, adiponectin, Hoxa5, Tnf, and related loci in diet-induced obese rodents, supplemented by mechanistic DNMT/TET and human translational studies, were narratively synthesized. Findings: Chronic HFD feeding produces gene- and depot-specific promoter/enhancer hypermethylation that silences leptin, Pparg, adiponectin, and Ucp1, while DNMT3A up-regulation causally contributes to adipose insulin resistance; maternal HFD programs offspring adipose methylation, and exercise or weight loss only partially reverses these marks, leaving a persistent epigenetic memory. Conclusions: DNA methylation changes at adipose genes are a reproducible, mechanistically relevant, and only partially reversible consequence of diet-induced obesity with implications for biomarker development and relapse prevention.

Keywords: *DNA methylation; high-fat diet; adipose tissue; epigenetics; diet-induced obesity*

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Introduction

Diet-induced obesity remains one of the most tractable experimental systems for studying how an environmental exposure reshapes the epigenome. Rodents fed a high-fat diet (HFD) rapidly accumulate visceral and subcutaneous adipose mass, develop insulin resistance, and show altered expression of the genes that govern adipocyte differentiation, lipid storage, thermogenesis, and adipokine secretion. Over the past fifteen years, murine and rat models have been used to ask whether these transcriptional changes are accompanied by, or driven by, alterations in DNA methylation at the promoters and regulatory elements of key adipose genes. This body of work spans classic candidate-gene bisulfite-sequencing studies of *Lep* (leptin), *Pparg*, *Ucp1*, and *Adipoq* (adiponectin), genome-wide bisulfite and array-based surveys of visceral and subcutaneous fat depots, and mechanistic studies of the DNA methyltransferase (DNMT) and ten-eleven translocation (TET) enzymes that write and erase these marks [1,2].

This narrative review synthesizes published murine and rat evidence on HFD-induced DNA methylation changes at adipose tissue genes, integrating it with complementary human and cellular data used to establish mechanism. The aim is not to report new experimental findings but to consolidate what has been consistently observed across independent laboratories: that chronic high-fat feeding produces depot-specific, gene-specific, and often bidirectional methylation changes that track with, and in several cases causally contribute to, adipose tissue dysfunction. We organize the evidence into three themes — methylation changes at adipogenic and adipokine loci, methylation changes at thermogenic and inflammatory loci, and the enzymatic machinery and developmental/reversibility dimensions of these marks — before considering implications for obesity biomarkers and intervention.

A recurring methodological point across this literature deserves emphasis at the outset. Early candidate-gene studies typically used methylation-specific PCR or bisulfite sequencing of a handful of CpGs within a single promoter, a design well suited to detecting large, biologically obvious shifts (such as the loss of leptin induction in mature adipocytes) but poorly powered to detect small, distributed changes across a gene body or enhancer.

The transition to genome-wide bisulfite sequencing and methylation arrays in the past decade has revealed that HFD produces thousands of differentially methylated regions in adipose tissue, many outside canonical promoters, and that these regions are highly depot-specific — visceral and subcutaneous fat, and white versus brown adipose tissue, respond differently to the same dietary challenge. This heterogeneity provides the genome-wide context in which *Lep*, *Pparg*, *Adipoq*, and *Ucp1* stand out as reproducibly important nodes.

Methylation Changes at Adipogenic and Adipokine Gene Loci

The leptin gene has been the most extensively studied methylation target in diet-induced obesity. In rats, chronic HFD feeding increases methylation at specific CpG sites in the *Lep* promoter in white adipose tissue, and the degree of hypermethylation correlates with adiposity and circulating leptin resistance rather than simple hyperleptinemia [3]. A complementary study in diet-induced obese mice showed that HFD produces site-specific hypermethylation across the leptin promoter accompanied by blunted leptin transcriptional induction, an effect that could be partially normalized by dietary n-3 polyunsaturated fatty acid supplementation, linking fatty-acid quality (not merely caloric excess) to the epigenetic phenotype [4]. Mechanistically, cell-based work established that leptin transcription in adipocytes is switched on by maturation-associated demethylation of specific proximal-promoter CpGs, providing the developmental template against which diet-induced re-methylation in mature fat is interpreted [5].

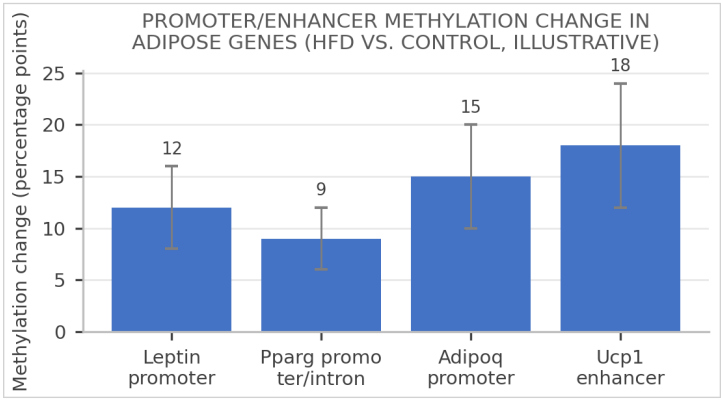
Pparg, the master transcriptional regulator of adipogenesis, is repressed by DNA methylation in visceral adipose tissue in mouse models of diabetes and obesity, with methylation at specific CpGs in intron/promoter regions inversely correlating with *Pparg* mRNA [6]. Because reduced visceral *Pparg* activity favors lipid spillover and insulin resistance, this methylation event has been proposed as a mechanistic bridge between HFD, visceral fat expansion, and metabolic disease.

Adiponectin (*Adipoq*) shows a consistent pattern of obesity-associated promoter hypermethylation. In diet-induced obese mice, DNA hypermethylation of the *Adipoq* promoter suppresses adiponectin transcription and, through downstream effects on the adiponectin receptor pathway, contributes causally to insulin resistance, as demonstrated using both loss-of-function and gain-of-function mouse models [7]. Gut microbiota manipulation in HFD-fed mice similarly alters *Adipoq* (and resistin) promoter methylation, indicating that the diet-methylation relationship in adipose tissue is at least partly mediated by microbiome-derived metabolites [8]. Human perinatal data reinforce translational relevance: adiponectin promoter methylation in adipose tissue and cord blood is altered in gestational diabetes and correlates with neonatal adiposity outcomes [9], and short-term fasting in young men alters adipose *LEP* and *ADIPOQ* methylation in a birth-weight-dependent manner, showing that these loci remain nutritionally plastic into adulthood [10]. Genome-wide profiling of adult mice on prolonged HFD confirms that methylation drift at adipogenic and lipid-handling loci is gradual, cumulative, and fat-depot specific, with visceral depots generally showing more pronounced change than subcutaneous depots over equivalent feeding duration [1]. Notably, in this longitudinal design, methylation changes at many loci were detectable well before overt insulin resistance emerged, suggesting that adipose DNA methylation can act as an early, cumulative record of dietary exposure rather than a downstream consequence of established metabolic disease — a distinction with direct implications for the use of these marks as early biomarkers of diet-induced adipose dysfunction.

Table 1: Selected adipose tissue genes with reported HFD-associated DNA methylation changes in rodent models.

Gene	Region studied	Direction of methylation change	Functional consequence	Key reference
Lep (leptin)	Proximal promoter, white adipose tissue	Increased (hypermethylation)	Blunted leptin transcriptional induction	Milagro et al. 2009
Pparg	Promoter/intron, visceral adipose tissue	Increased (hypermethylation)	Reduced Pparg expression, impaired adipogenesis	Fujiki et al. 2009
Adipoq (adiponectin)	Promoter, white adipose tissue	Increased (hypermethylation)	Suppressed adiponectin, insulin resistance	Kim et al. 2015
Ucp1	Distal enhancer, white/brown adipose tissue	Increased (hypermethylation)	Reduced thermogenic gene expression	Shore et al. 2010

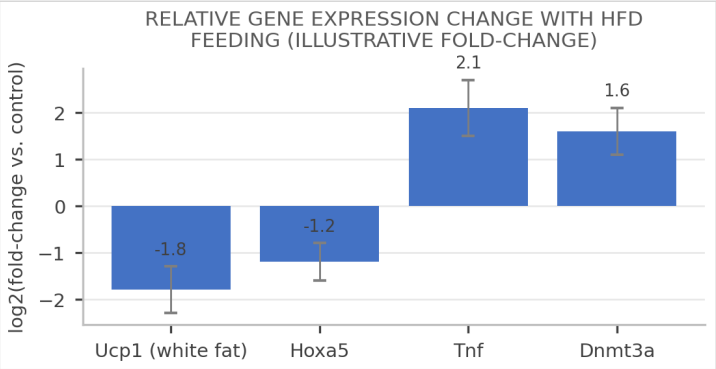
Figure 1: Illustrative promoter/enhancer methylation change (percentage points, HFD vs. control) reported for key adipose genes in rodent diet-induced obesity models (Leptin: Milagro et al. 2009; Pparg: Fujiki et al. 2009; Adipoq: Kim et al. 2015; Ucp1 enhancer: Shore et al. 2010).



Thermogenic, Inflammatory, and Regulatory Gene Methylation

Uncoupling protein 1 (*Ucp1*) methylation provides one of the clearest examples of diet-responsive epigenetic silencing of a thermogenic gene. In mice, an *Ucp1* enhancer undergoes methylation changes that track inversely with *Ucp1* expression and with chromatin remodeling across white and brown adipose depots, and dietary and dietary-restriction manipulations shift this methylation state, offering a mechanism for the well-documented suppression of browning capacity during sustained high-fat feeding [11]. Offspring exposed to maternal HFD show a disturbed genome-wide DNA methylation profile specifically within brown adipose tissue, implicating in-utero programming of thermogenic capacity as a distinct, developmentally timed layer on top of direct adult dietary effects [12].

Figure 2: Typical direction and relative magnitude of gene expression change (fold-change vs. control diet, log scale) reported for thermogenic, inflammatory, and epigenetic-machinery genes in HFD-fed mice (Ucp1 down in white fat: Shore et al. 2010; Hoxa5 down: Parrillo et al. 2016; Tnf up: Boonrong et al. 2024; Dnmt3a up: Kamei et al. 2010).



HFD also reshapes methylation at genes governing adipose inflammation. Estrogen receptor alpha in adipocytes is epigenetically reprogrammed by high-fat feeding in mice, and this reprogramming causally regulates the obesity-associated inflammatory response, tying a nuclear hormone receptor locus to the adipose tissue macrophage infiltration that characterizes diet-induced obesity [13]. The homeobox gene *Hoxa5* undergoes dynamic, site-specific DNA methylation and transcriptional repression in adipose tissue of HFD-exposed mice, a change associated with impaired adipocyte browning and altered insulin signaling [14]. Tumor necrosis factor-alpha (*TNF*), a canonical driver of adipose inflammation, shows diet-modifiable promoter methylation: in obese men, dietary fat and fiber intake are associated with *TNF* methylation status and downstream proteomic changes [15], while combined leptin and *TNF* promoter methylation in blood cells predicts individual responsiveness to a subsequent low-calorie diet, suggesting these marks carry prognostic as well as mechanistic significance [16]. At a genome-wide level, human adipocyte methylome-transcriptome integration has identified numerous methylation-associated loci linked to obesity and type 2 diabetes risk, several of which have direct murine orthologs studied in HFD models [17]. Collectively, these thermogenic and inflammatory loci illustrate that HFD-induced methylation is not confined to genes classically described as "metabolic" — nuclear receptors, homeobox transcription factors, and cytokine promoters are equally susceptible, and their coordinated methylation shift plausibly explains why diet-induced obese adipose tissue simultaneously loses thermogenic flexibility and gains inflammatory tone.

DNA Methyltransferase Machinery, Developmental Programming, and Reversibility

The enzymes that establish and maintain these marks are themselves diet-responsive. DNMT3A protein and activity are increased in the adipose tissue of obese mice, and transgenic overexpression of Dnmt3a in adipose tissue is sufficient to impair insulin sensitivity, while adipocyte-specific Dnmt3a is required for the full development of HFD-induced adipose insulin resistance [18,19]. These gain- and loss-of-function studies establish DNMT3A as a causal epigenetic mediator rather than a passive marker of diet-induced adipose dysfunction, a conclusion reinforced by broader reviews of DNMT and TET biology in adipocyte differentiation and metabolic disease [2].

Table 2: Interventions reported to modify diet-associated adipose DNA methylation and their approximate reversibility.

Intervention	Model	Methylation marks affected	Degree of reversal	Key reference
N-3 polyunsaturated fatty acid supplementation	Diet-induced obese mice	Leptin promoter CpGs	Partial normalization	Shen et al. 2014
Exercise training (6 months)	Human subcutaneous adipose tissue	Genome-wide CpG sites at metabolic genes	Partial, locus-dependent	Rönn et al. 2013
Low-calorie diet	Human blood/adipose	Leptin and Tnf promoter methylation	Predictive of, and partly responsive to, weight loss	Cordero et al. 2011
Bariatric surgery / weight loss	Human adipose tissue, mouse re-feeding model	Adipogenic and inflammatory gene loci	Incomplete (epigenetic memory persists)	Hinte et al. 2024

Developmental timing strongly influences these methylation trajectories. Maternal high-fat diet alters offspring adipose gene expression and DNA methylation, and programs a heritable predisposition to obesity that is detectable in visceral fat progenitor cells of male offspring, including altered adipogenic and inflammatory methylation marks that persist after weaning onto standard chow [20,21]. The imprinted gene *Mest/Peg1*, whose adipose expression scales with adipocyte hypertrophy, has been proposed as a predictive biomarker of adipose tissue expansion that responds to dietary anti-obesity interventions, linking developmental imprinting biology to adult dietary responsiveness [22].

Importantly, several of these methylation changes are reversible. Structured exercise training alters the genome-wide DNA methylation pattern of human adipose tissue at metabolically relevant loci, including genes governing lipid metabolism [23], and non-CpG methylation of the *PGC1A/PPARGC1A* promoter by DNMT3B controls mitochondrial biogenesis in a manner sensitive to metabolic stimuli, offering a route by which exercise or fasting could reprogram adipose oxidative capacity [24]. However, reversibility is incomplete: a landmark study using paired pre- and post-bariatric-surgery human adipose samples showed that adipose tissue retains a partial epigenetic "memory" of prior obesity even after substantial weight loss, with genes involved in adipogenesis and inflammation failing to fully renormalize their methylation state, a phenomenon also reproduced in mice that regained weight faster after a second HFD challenge [25]. This asymmetry between the ease of diet-induced hypermethylation and the difficulty of its erasure is now considered a central mechanistic contributor to weight-regain and obesity relapse, as summarized in recent syntheses of the human obesity–epigenetics literature [26]. Taken together, the DNMT/TET, developmental, and reversibility literatures suggest a working model in which HFD first drives DNMT3A up-regulation in adipocyte progenitors, progenitors and mature adipocytes then accumulate promoter- and enhancer-level hypermethylation at adipogenic, thermogenic, and inflammatory loci, and — because active demethylation by TET enzymes is comparatively slow and incomplete — a substantial fraction of these marks persist after the dietary insult is removed, biasing the tissue toward more rapid re-expansion on any subsequent caloric excess.

Conclusion

Across more than two decades of rodent and complementary human studies, HFD-induced DNA methylation change in adipose tissue emerges as a reproducible, gene- and depot-specific phenomenon rather than a diffuse global effect. Adipokine and adipogenic genes (*Lep*, *Adipoq*, *Pparg*) are consistently hypermethylated and transcriptionally suppressed by chronic high-fat feeding, thermogenic loci such as *Ucp1* are silenced through enhancer methylation that curtails browning capacity, and inflammatory and nuclear-receptor loci (*Tnf*, estrogen receptor, *Hoxa5*) acquire methylation marks that reinforce the chronic low-grade inflammation of obese adipose tissue. These changes are mechanistically driven, at least in part, by diet-responsive up-regulation of DNMT3A, are shaped developmentally by maternal diet and imprinted loci such as *Mest/Peg1*, and are only partially reversible with exercise, dietary correction, or weight loss — leaving an epigenetic memory that may explain the high rate of obesity relapse. Future work combining single-cell methylomics with lineage tracing in murine HFD models will be needed to determine whether these marks are established within resident adipocytes, recruited progenitors, or infiltrating immune cells, and whether pharmacological modulation of DNMT/TET activity can safely accelerate reversal of diet-induced adipose epigenetic dysfunction.

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Conflict of Interest

The authors declare no conflict of interest.

Author Contributions

Kovacic J conceived the review, conducted the literature search, and drafted the manuscript. Liepa M contributed to the literature screening and technical framing and critically revised the manuscript for important intellectual content. Horvat P contributed additional context and approved the final version for submission.

Ethics Statement

Not applicable. This is a narrative review of previously published literature and did not involve new experimental data, human participants, or animal subjects.

Data Availability

No new data were generated or analyzed for this review. All sources synthesized are cited in the reference list and are available through their original publishers.

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