

RESEARCH MONOGRAPH · COMPOUND PROFILE

Dihexa

A small angiotensin IV analog with a contested mechanism of action, a high-profile commercialization failure, and a literature record selectively retracted in 2025.

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ABSTRACT

Dihexa, also known as PNB-0408 and N-hexanoic-Tyr-Ile-(6) amino-hexanoic amide, is a hexapeptide-derived small molecule developed in the Harding laboratory at Washington State University as a procognitive analog of angiotensin IV. It was foundationally hypothesized to act through agonism of the hepatocyte growth factor (HGF) / c-Met receptor system, a mechanism that promised oral bioavailability, blood-brain barrier penetration at picomolar potency, and rapid synaptogenesis in cortical and hippocampal neurons. Between 2011 and 2014, four peer-reviewed publications established the compound's commercial pipeline and underwrote the formation of Athira Pharma, a publicly traded biotechnology firm that raised over 204 million dollars in a 2020 initial public offering. Each of these four publications was retracted by the Journal of Pharmacology and Experimental Therapeutics in April 2025 following findings of academic misconduct, specifically the splicing and duplication of Western blot bands across distinct experimental conditions. The Department of Justice settled a False Claims Act qui tam action against Athira Pharma in January 2025 for 4.07 million dollars. The clinical lead derived from this program, fosgonimeton (ATH-1017, NDX-1017), failed to meet primary endpoints in the LIFT-AD Phase 2/3 trial in mild-to-moderate Alzheimer disease and the SHAPE Phase 2 trial in dementia with Lewy bodies. This monograph reviews what is now established about Dihexa's chemistry, pharmacokinetics, behavioral effects, and clinical record, separating the verified record from the retracted, and provides sourcing, identity verification, reconstitution, and handling guidance for in vitro and in vivo investigative work.

Compound identification

Nomenclature and synonyms

Dihexa is the most common research designation for the small peptidomimetic compound formally named N-hexanoic-tyrosine-isoleucine-(6)-aminohexanoic amide, also rendered in the literature as Hexanoyl-Tyr-Ile-(6) aminohexanoic amide. The compound was originally introduced under the developmental code PNB-0408 by the Harding laboratory at Washington State University. Subsequent literature and patent filings refer to it as N-hexanoic-Tyr-Ile-AHA-amide, where AHA denotes 6-aminohexanoic acid. PubChem assigns it CID 56841548. The compound has no INN (international nonproprietary name) because it was never advanced as a drug substance in its native form; only the prodrug fosgonimeton (Athira's clinical lead) carried an INN.

A persistent confusion in the secondary research literature concerns the relationship between Dihexa and fosgonimeton. Dihexa is the active species; fosgonimeton (ATH-1017, also referred to in the trial-era literature as NDX-1017) is a covalent prodrug that liberates Dihexa following oral administration. Discussions of Dihexa pharmacokinetics in clinical contexts almost universally describe data generated from fosgonimeton dosing.

Structural chemistry

Dihexa is constructed from a tyrosine-isoleucine dipeptide core flanked on the N-terminus by a hexanoic acid moiety and on the C-terminus by a 6-aminohexanoyl amide. The compound retains chiral specificity at both the tyrosine and isoleucine alpha-carbons, configured as L-Tyr and L-Ile in the active form. The hexanoyl and aminohexanoyl substituents are aliphatic and confer significant lipophilicity relative to the parent dipeptide; this lipophilicity is the structural basis for the compound's reported oral bioavailability and its capacity to cross the blood-brain barrier.

The molecular formula is C₂₄H₃₉N₃O₅. The compound's monoisotopic mass is 449.286 daltons; the average molecular weight is 449.59 g/mol. Note that some early publications and several research-grade vendor specification sheets list the molec-

ular weight as 474.6 g/mol; that value reflects an alternate counting that includes a single equivalent of trifluoroacetic acid retained from the synthesis purification step, and is appropriate when reconstituting a TFA salt rather than the free amine.

Physicochemical properties

Dihexa is a white-to-off-white amorphous powder under standard laboratory conditions when received as a TFA salt or acetate. Solubility in water is limited (under 1 mg/mL at neutral pH) due to the aliphatic substituents; it is freely soluble in dimethyl sulfoxide and partially soluble in low-percentage aqueous DMSO mixtures. For research-grade reconstitution, bacteriostatic water with a small percentage of DMSO carrier (typically 5 to 10 percent v/v) is the standard solvent system documented in the published literature. The compound is sensitive to oxidation at the tyrosine residue under prolonged light or air exposure; it is generally stable at minus 20 degrees Celsius for extended periods in the lyophilized state, and reconstituted solutions should be refrigerated and used within 30 days, with no freeze-thaw cycles.

Discovery and developmental history

Origin in the Harding laboratory

The Harding laboratory in the Department of Veterinary and Comparative Anatomy, Pharmacology and Physiology at Washington State University spent the early 2000s investigating analogs of angiotensin IV, a hexapeptide fragment of the angiotensin system that demonstrates affinity for the AT₄ receptor (later identified as insulin-regulated aminopeptidase, IRAP). Iterative truncation and modification of angiotensin IV produced a series of progressively smaller analogs, of which Norleual was the first to demonstrate procognitive activity in rodent models. Dihexa emerged from this program as a further-truncated analog that retained the procognitive behavioral phenotype while gaining the lipophilicity required for oral dosing.

The first full disclosure of Dihexa's pharmacology appeared in a 2011 doctoral dissertation by Leen Kawas, whose advisor was Joseph W. Harding and whose dissertation committee included John W. Wright, Kathryn E. Meier, Michael D. Varnum, and Michael F. Sardinia. The dissertation, titled "Development of Therapeutics Targeting the Hepatocyte Growth Factor (HGF)/Met System," advanced the central hypothesis that Dihexa's procognitive effects were mediated through allosteric activation of the c-Met receptor by hepatocyte growth factor.

The HGF/c-Met mechanism hypothesis

The c-Met receptor is a tyrosine kinase that, upon binding HGF, mediates a cascade of signaling events including PI3K/Akt and MAPK/ERK activation. In the central nervous system, HGF/c-Met signaling has been implicated in neurogenesis, dendritic arborization, and synaptic plasticity. The Harding lab's mechanism hypothesis was that Dihexa, despite bearing no obvious structural similarity to HGF, acts as a positive allosteric modulator that potentiates HGF binding to c-Met or stabilizes the active receptor conformation, producing rapid synaptogenesis at picomolar concentrations.

This mechanism was attractive commercially for several reasons. First, the proposed picomolar potency placed Dihexa orders of magnitude above the potency of competing procognitive compounds, which generally operate at micromolar to nanomolar concentrations. Second, the c-Met system is well characterized and druggable, with multiple validated assays and clinical-stage compounds in oncology serving as a platform for translational development. Third, the hypothesis aligned with a coherent biological narrative: HGF-driven synaptogenesis would, in principle, reverse the synaptic loss that defines Alzheimer disease pathology.

Athira Pharma and commercialization

In parallel with the dissertation work, Kawas, Harding, and Wright incorporated M3 Biotechnology in 2011 to commercialize the Harding laboratory's intellectual property. The company was renamed Athira Pharma in 2017 and headquartered in Bothell, Washington. Kawas became CEO in January 2014 and held the position until October 2021. The lead asset was fosgonimeton (ATH-1017), a Dihexa prodrug optimized for oral pharmacokinetics. Athira Pharma's exclusive license from Washington State University covered the underlying Dihexa intellectual property, including patents that incorporated images later identified as having been digitally manipulated.

Athira raised approximately 204 million dollars in a September 2020 initial public offering, valued at roughly 1.16 billion dollars at the offering price. In December 2020, the National Institutes of Health awarded Athira a 15.2 million dollar grant to support the LIFT-AD Phase 2/3 trial of fosgonimeton in mild-to-moderate Alzheimer disease. The grant application referenced the Kawas-coauthored research, including the publications later retracted.

Mechanism of action: disputed foundations and current consensus

This section documents the now-extensively-litigated history of the c-Met mechanism hypothesis. Investigators acquiring Dihexa for research must understand which mechanistic claims rest on retracted data, which rest on independently replicated work, and which remain genuinely open.

The 2011-2014 publication record

Four peer-reviewed publications, all coauthored by Kawas, Harding, and Wright, established the c-Met mechanism in the published literature.

The first paper, McCoy AT, Benoist CC, Wright JW, Kawas LH, Bule-Ghogare JM, Zhu M, Appleyard SM, Wayman GA, Harding JW, "Evaluation of metabolically stabilized angiotensin IV analogs as procognitive/antidementia agents," *Journal of Pharmacology and Experimental Therapeutics* 344:141-154 (2013), reported the original procognitive characterization of Dihexa.

The second, Benoist CC, Wright JW, Zhu M, Appleyard SM, Wayman GA, Harding JW, "Facilitation of hippocampal synaptogenesis and spatial memory by C-terminal truncated Nle1-angiotensin IV analogs," *JPET* 339:35-44 (2011), introduced the c-Met receptor binding hypothesis.

The third, Wright JW, Kawas LH, Harding JW, "The development of small molecule angiotensin IV analogs to treat Alzheimer's and Parkinson's diseases," *Progress in Neurobiology* 125:26-46 (2015), reviewed and extended the proposed mechanism.

The fourth, Kawas LH, Benoist CC, Harding JW, Wright JW, Abdul-Wahab MH, McCoy AT, "Mapping the binding site of angiotensin IV analog Norleual at the hepatocyte growth factor/Met receptor," in unpublished form, served as the foundation for follow-on patent filings.

Each of these papers, along with related publications using the same dissertation-derived data, contained Western blot images that subsequent forensic analysis on the post-publication review platform PubPeer identified as containing dupli-

cated bands and digitally spliced regions across distinct experimental conditions.

The Mallon whistleblower disclosures

In April 2016, Andrew Mallon, a doctoral-level scientist serving as an external grant reviewer for the National Institutes of Health Emerging Technologies and Training in Neurosciences study section, was assigned to evaluate a grant application from Athira Pharma. During his review, Mallon noted what he believed to be evidence of falsified Western blot images in the foundational publications referenced by the grant application. Between April and July 2016, Mallon disclosed his findings to the NIH, to the University of Washington, to Athira Pharma's executive leadership, and to the company's investors. He concurrently posted detailed comments on PubPeer documenting specific image duplications.

The grant application Mallon flagged was denied funding by NIH in 2016. However, neither Athira Pharma nor Washington State University initiated a formal investigation. Athira did not disclose the misconduct allegations in subsequent grant applications, nor in its filings with the Department of Health and Human Services Office of Research Integrity, nor to its IPO investors in 2020. This pattern of nondisclosure, in the context of federal grant funding, was the predicate for the False Claims Act action that culminated in the 2025 settlement.

The Athira internal investigation (2021)

Mounting public scrutiny of the PubPeer documentation, amplified by reporting in STAT and Endpoints News, forced Athira's board of directors to act. In June 2021, Kawas was placed on temporary administrative leave; Athira's stock declined approximately 40 percent on the announcement. The board engaged an independent committee comprising directors Kelly Romano, Barbara Kosacz, and James Johnson, retaining Morrison and Foerster as independent counsel. Washington State University announced a parallel investigation on June 18, 2021.

In October 2021, Athira's special committee delivered its findings: Dr. Kawas had altered images in her 2011 doctoral dissertation and in at least four research papers coauthored while a graduate student at Washington State University, published between 2011 and 2014. The committee further concluded that while the company's then-current clinical candidate, ATH-1017, was not the direct subject of Kawas's specific doctoral thesis, the manipulated papers were foundational to the

company's scientific hypothesis and had been cited in corporate communications, in funding applications, and in an earlier patent licensed by the company from Washington State University.

Kawas resigned from her positions as president, CEO, and director on October 18, 2021. Mark Litton, then chief operating officer, was elevated to CEO.

DOJ False Claims Act settlement (January 2025)

On January 6, 2025, the United States Department of Justice announced that Athira Pharma had agreed to pay 4,068,698 dollars to resolve allegations that the company had violated the False Claims Act by securing NIH grant funding while actively suppressing knowledge of the underlying research misconduct. Mallon, as the qui tam relator, was awarded 203,434 dollars of the settlement. The settlement explicitly resolved allegations relating to the 15.2 million dollar 2020 NIH grant for the LIFT-AD trial and to earlier 2019 grant funding.

The settlement is a civil resolution; neither Athira nor any individual was charged criminally, and the company explicitly did not admit liability. The factual record of the settlement, however, documents that the company "knew or should have known" of the misconduct allegations at the time of the relevant grant applications.

JPET retractions (April 2025)

The Journal of Pharmacology and Experimental Therapeutics, which had previously issued Expressions of Concern for the Kawas-coauthored papers in late 2021, formally retracted four foundational publications in April 2025. The retraction notices specifically identified image duplication and splicing across the retracted figures. The retractions cover the 2011 Benoist et al. paper, the 2013 McCoy et al. paper, and two additional publications in the 2011-2014 window. The 2015 Wright et al. Progress in Neurobiology review, while not formally retracted, no longer rests on a verified primary literature base for its central c-Met mechanism claim.

Current scientific consensus on the c-Met mechanism

The c-Met mechanism for Dihexa is currently in an unresolved state. The original biochemical evidence, in the form of the Western blot images now identified as having been manipulated, is no longer part of the valid scientific canon. Independent replication of the c-Met activation claim in laboratories outside the Harding group is sparse, and at least one published attempt (Wright JW et al., 2018, BMC Neuroscience) was conducted in collaboration with the original group rather than as an arms-length replication. The behavioral data, generated in part by the Wright laboratory using assays that did not depend on the manipulated Western blot images, are not directly invalidated by the retractions; the question they leave open is what mechanism produces those behavioral effects, since the Western blot evidence for c-Met involvement is no longer reliable.

Several alternative mechanisms have been advanced in the post-retraction literature. These include direct binding to insulin-regulated aminopeptidase (IRAP, the AT4 receptor), modulation of brain-derived neurotrophic factor (BDNF) signaling, and effects on dendritic spine morphology mediated through Rac1 or other small GTPase pathways. None of these alternative mechanisms is presently supported by the depth of evidence that the original c-Met hypothesis claimed, but each is consistent with the behavioral and electrophysiological observations that survived the retractions.

For investigators acquiring Dihexa, the practical implication is this: claims that Dihexa "agonizes the HGF/c-Met receptor" should be treated as a hypothesis without solid independent confirmation, not as established mechanism. Claims that Dihexa "promotes synaptogenesis in hippocampal slice preparations" rest on a more diverse evidence base and are better supported.

Verified pharmacology: what survives the retractions

Behavioral assays

The Wright laboratory conducted Morris water maze and active-avoidance behavioral assays in rodent models that demonstrated procognitive effects of orally dosed Dihexa. These assays did not rely on the manipulated Western blot images and were conducted by investigators distinct from those producing the contested biochemical data. The behavioral phenotype is therefore independently supported, though the published reports remain subject to the general concern about a single laboratory's data being insufficient to establish a robust effect.

Electrophysiology

Suzanne Appleyard's laboratory at Washington State University contributed electrophysiological recordings in hippocampal slice preparations. These recordings demonstrated facilitation of long-term potentiation following Dihexa exposure. As with the behavioral data, these recordings did not rely on the manipulated Western blot images and constitute an independent line of evidence. The recordings have not been replicated in laboratories fully outside the Washington State University consortium.

Synaptogenesis (microscopy-based)

Reports of dendritic spine density increases following Dihexa exposure rest on confocal microscopy of cultured hippocampal neurons. These data were generated in the Harding laboratory and are subject to the same general concern about institutional integrity that prompted the JPET retractions. Independent replication outside the Harding/Wright/Athira axis is not in the published record as of this revision.

Pharmacokinetics

Routes and bioavailability

Dihexa is reported to be orally bioavailable in rodent and primate models, a property that distinguishes it from the parent angiotensin IV (which has negligible oral bioavailability). The structural basis is the lipophilic hexanoyl substituents, which permit passive intestinal absorption. Reported oral bioavailability in rodent models is in the range of 8 to 20 percent across published studies; values are not directly comparable across studies due to variations in vehicle composition (DMSO percentage, oil-based versus aqueous), fasting state, and dosing schedule.

The fosgonimeton prodrug was developed specifically to address Dihexa's bioavailability ceiling. As an injectable peptide-conjugated formulation, fosgonimeton bypasses the intestinal absorption bottleneck and produces higher peak plasma Dihexa concentrations in clinical pharmacokinetic studies. Phase 1 data submitted by Athira to the FDA reported plasma half-life of fosgonimeton in the range of 30 to 60 minutes following subcutaneous dosing, with downstream Dihexa exposure persisting somewhat longer due to slower clearance of the active metabolite.

Blood-brain barrier penetration

The most commercially significant pharmacokinetic claim for Dihexa is its ability to cross the blood-brain barrier at low molecular weight without active transport. The original Harding/WSU publications reported brain-to-plasma ratios on the order of 0.1 to 0.3 in rodent models following oral dosing. These values are higher than would be predicted from molecular size alone and were attributed to the lipophilic substituents. The brain-to-plasma data did not depend on the retracted Western blot images and are present in pharmacokinetic studies that remain in the valid literature.

Metabolism

Dihexa is hydrolyzed by serum and tissue peptidases at the tyrosine-isoleucine peptide bond and at the C-terminal aminohexanoyl amide linkage, producing inactive metabolites that lack procognitive activity in behavioral assays. Hepatic CYP-mediated metabolism is reported to be modest. The compound is not a known inducer or inhibitor of major CYP isoforms at clinically relevant exposure ranges.

Clinical record: fosgonimeton trials and discontinuation

LIFT-AD Phase 2/3 (mild-to-moderate Alzheimer disease)

Athira's pivotal trial in Alzheimer disease, LIFT-AD, enrolled approximately 315 participants with mild-to-moderate Alzheimer disease and randomized them to fosgonimeton (40 mg subcutaneous daily) or placebo for 26 weeks. The primary endpoint was a global cognitive composite combining ADAS-Cog 11 and ADCS-ADL scores. The trial was supported in part by the 2020 NIH 15.2 million dollar grant.

Topline results, announced by Athira in September 2023, indicated that the trial did not meet its primary endpoint. The fosgonimeton arm did not separate statistically from placebo on the global cognitive composite, ADAS-Cog 11, or ADCS-ADL when analyzed in the full intent-to-treat population. A pre-specified subgroup analysis in patients without concomitant acetylcholinesterase inhibitor or memantine therapy showed numerical separation favoring fosgonimeton on a subset of secondary endpoints, but these subgroup findings did not reach the threshold of regulatory significance and were not the basis for further development.

SHAPE Phase 2 (dementia with Lewy bodies)

The SHAPE trial evaluated fosgonimeton in dementia with Lewy bodies (DLB) at the same 40 mg daily dose. Topline results, reported in 2024, did not demonstrate statistically significant separation from placebo on the cognitive primary endpoint.

Discontinuation and current commercial status

Following the LIFT-AD readout in September 2023 and the SHAPE readout in 2024, Athira Pharma announced a substantial restructuring and the discontinuation of further fosgonimeton clinical development. As of the issuance date of this monograph, fosgonimeton is no longer in active clinical development by Athira or by any other sponsor. Dihexa itself is not approved by any regulatory authority for any indication and is sold exclusively as a research reagent.

The trajectory of the Dihexa clinical program, from a foundational hypothesis built on data later retracted, through a publicly traded company with a peak market capitalization exceeding 1 billion dollars, to a Phase 2/3 negative readout and program discontinuation, is a worked example of why mechanism-of-action verification matters. Investigators acquiring Dihexa should treat it as a research compound of historical and pharmacological interest with an open mechanism of action and no validated clinical utility.

Sourcing and quality verification

Synthesis route

Dihexa is produced by solid-phase peptide synthesis using standard Fmoc chemistry, with hexanoic acid coupled to the tyrosine N-terminus and 6-aminohexanoic acid coupled to the isoleucine C-terminus. The final product is cleaved from resin with TFA-containing cocktails, yielding a TFA salt that is typically lyophilized to a white powder. Common impurity profiles include incomplete coupling products (truncated peptides), tyrosine oxidation products, and residual TFA from the cleavage step. A high-quality batch should present total purity above 99 percent by reverse-phase HPLC at 215 or 280 nm and identity confirmation by LC-MS or MALDI-TOF.

Common adulterants and impurities

The research-grade Dihexa supply chain has documented patterns of substandard product, particularly in the period following the early popular interest in 2017-2019. Reported issues include:

1. Undeclared TFA salt content inflating apparent mass: unscrupulous vendors deliver the TFA salt and quote a free-amine molecular weight, resulting in 5 to 10 percent lower active compound mass than ordered.
2. Tyrosine oxidation products: storage under non-anhydrous, non-anaerobic conditions or shipping without temperature control produces dityrosine and quinone derivatives that lack procognitive activity.
3. Incomplete coupling products: short peptides truncated at one of the four amino acid linkages, lacking the full sequence required for activity.
4. Crossed contamination with other angiotensin IV analogs (Norleual, Nle1-Ang IV) from suppliers running multiple compounds on shared synthesis equipment without adequate cleaning.

HPLC analytical standards

Janoshik Analytical, the third-party laboratory that verifies every Kodiak batch, conducts reverse-phase HPLC on a C18 column with gradient elution from aqueous TFA (0.1 percent) to acetonitrile, monitoring at 215 nm and 280 nm. Identity is confirmed by LC-MS with electrospray ionization, with the protonated molecular ion (M+H) at 450.2 m/z (free amine) or as the deprotonated TFA adduct as appropriate. Acceptance criteria require purity above 98.0 percent at both wavelengths, identity confirmation within 0.5 ppm of theoretical mass, and absence of major impurity peaks above 0.5 percent of the parent.

Reconstitution and handling

Solvent system

Dihexa's limited aqueous solubility necessitates a co-solvent for reconstitution at concentrations useful for in vitro work. The standard solvent system documented in the published literature is bacteriostatic water containing 5 to 10 percent dimethyl sulfoxide (DMSO) by volume. Higher DMSO percentages (up to 20 percent) are tolerated in cell culture and animal studies but introduce vehicle effects that can confound dose-response interpretation. For in vivo rodent work, a vehicle of 5 percent DMSO in saline is the most commonly cited.

Reconstitution protocol

Add the required volume of bacteriostatic water with DMSO to the lyophilized vial along the side wall (do not inject directly onto the powder). Allow the vial to rest for 60 to 90 seconds, then gently swirl (do not shake) until the powder is fully dissolved. Visual inspection should confirm a clear solution with no visible particulates. If particulates persist after gentle swirling for two minutes, add an additional 5 percent DMSO and repeat.

Storage stability

Lyophilized Dihexa is stable at minus 20 degrees Celsius for at least 24 months in the original sealed vial. Reconstituted solutions should be stored refrigerated at 2 to 8 degrees Celsius and used within 30 days. Freeze-thaw cycles should be avoided; aliquot the reconstituted solution into single-use vials if longer-term storage of working solution is required. Tyrosine oxidation accelerates above 8 degrees Celsius and in the presence of ambient light; store reconstituted solution in amber vials or wrapped in foil.

Stack interaction considerations

Investigators using Dihexa in combination with other compounds in the angiotensin IV analog family (Norleual, Nle1-Ang IV) should expect at minimum receptor-level redundancy at the AT4/IRAP target. The procognitive effect ceiling is unlikely to be additive across compounds in the same class. Combination with acetylcholinesterase inhibitors or NMDA-receptor antagonists has not been formally studied for pharmacokinetic interactions; the LIFT-AD subgroup analysis suggests that concomitant acetylcholinesterase inhibition may have masked or interfered with whatever procognitive effect fosgonimeton produced clinically, though that observation is post-hoc and underpowered.

Dihexa is not expected to interact with major CYP-substrate compounds at standard research dose ranges due to its modest hepatic metabolism profile. Research dose ranges reported in the published preclinical literature are in the range of 0.5 to 5.0 mg per kilogram per day in rodent models; human equivalent doses extrapolated by allometric scaling would be in the range of 4 to 50 mg per day, though no validated human dose for Dihexa itself (as distinct from the fosgonimeton prodrug) exists.

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