dihexa - mechanistic, possible benefits and risks, no influencers, no blogs, no clinics, no youtube, only use clinical and preclinical studies published in reputable journals

Dihexa is an AngIV-derived, HGF-mimetic oligopeptide that allosterically potentiates HGF/c-Met signaling, driving marked synaptogenesis and procognitive effects in rodent models, with a prodrug (fosgonimeton/ATH-1017) in early human trials but with substantial unresolved safety questions, particularly around tumorigenesis.[1] [2] [3] [4] [5]

Core mechanism

- Dihexa is derived from Angiotensin IV (Nle¹-AngIV) and binds HGF with very high affinity, acting as an allosteric agonist/mimetic rather than a direct c-Met agonist.[2] [5]
- In HEK-293T cells and hippocampal tissue, dihexa and Nle¹-AngIV potentiate HGF-dependent c-Met phosphorylation at subnanomolar concentrations and augment HGF-driven cell scattering and downstream responses, with effects seen at endogenous or subthreshold HGF levels.[5]
- In hippocampal neuron cultures and brain slices, dihexa increases synapse number and functional synaptic connectivity, consistent with an HGF-driven **synaptogenic** mechanism.[5]
- The broader HGF/c-Met axis is pro-survival, pro-angiogenic, anti-inflammatory, and neurotrophic in CNS, supporting neuronal survival, anti-apoptotic signaling, and modulation of neuroinflammation.[6]

Illustration: in the primary 2013 mechanistic paper, dihexa at femto- to picomolar levels shifted HGF dose–response curves leftward and increased c-Met phosphorylation and synaptogenesis in hippocampal preparations, implying a catalytic potentiation of endogenous HGF rather than supraphysiologic receptor overdrive.[5]

Preclinical benefits

Cognitive and synaptic effects

- In rat models of cognitive dysfunction, chronic dihexa administration improves performance in spatial learning and memory tasks and increases synapse number in hippocampal regions implicated in cognition.[1] [5]
- The original “procognitive and synaptogenic” paper showed that dihexa reversed scopolamine-induced deficits and age-related impairments, with associated increases in

dendritic spine density and synaptic markers (e.g., synapsin).[5]

- A later study in a neuroinflammatory/neurodegenerative context (AngIV-analog dihexa study, 2021) reported that dihexa rescued cognitive impairment and restored neuronal structure, at least in part by modulating neuroinflammation and preventing neuronal apoptosis in the hippocampus.[4]

Neuroprotection and disease-model data

Across rodent models (summarizing key themes):

- Dihexa reduced neuroinflammatory markers and preserved neuronal morphology in models of cognitive decline, suggesting combined synaptogenic and anti-inflammatory actions.[4]
- Pharmacology reviews describe robust procognitive activity even at very low doses, with high brain penetrance and long half-life, leading to sustained synaptic and behavioral effects in animals.[1]

Because most detailed datasets are in the primary 2013–2014 AngIV/dihexa work and subsequent Chinese-language or region-specific publications, the picture is: strong, reproducible improvements in rodent learning/memory, increased synapse number, and neuroprotection in inflammatory and degenerative paradigms.[4] [1] [5]

Human data (fosgonimeton/ATH-1017)

The clinically developed entity is fosgonimeton (ATH-1017), a small-molecule prodrug of the dihexa pharmacophore that targets the same HGF/c-Met neurotrophic pathway.[3] [7]

Early-phase trials

- A randomized, double-blind, placebo-controlled phase I trial (healthy young, healthy elderly, and patients with Alzheimer's disease) evaluated safety, PK, and PD of fosgonimeton.[7] [3]
- Fosgonimeton was generally safe and well tolerated in this study, with no serious adverse events and acceptable adverse-event profile over the treatment window.[3] [7]
- In AD subjects, repeated dosing produced statistically significant improvements in ERP P300 latency versus placebo, interpreted as an acute enhancement of synaptic function/neurophysiology; this supported further development.[7] [3]

Later Alzheimer's trial outcomes

- In a 26-week study in Alzheimer's disease, fosgonimeton showed numerically favorable changes versus placebo on global and cognitive measures (GST, ADAS-Cog11, ADCS-ADL23), but differences did not reach statistical significance for primary or key secondary endpoints.[8]
- Despite the lack of efficacy signal at the prespecified thresholds, biomarker data (e.g., NfL, GFAP, plasma phosphorylated tau species, Aβ42/Aβ40 ratio) tended to move in a direction

favoring fosgonimeton, suggesting some disease-modifying or neuroprotective activity may be present but underpowered or dose/timing-dependent.[8]

- Overall, current human data show: short-term safety and tolerability, a plausible PD signal in ERP/synaptic markers, but no clearly demonstrated clinical benefit in phase II-like Alzheimer's endpoints to date.[3] [8] [7]

Risks and unresolved safety issues

Tumorigenesis and c-Met activation

- The c-Met receptor and HGF/c-Met signaling are implicated in multiple cancers; overexpression of HGF and MET correlates with tumorigenesis, metastasis, and poor prognosis in a variety of malignancies.[6] [1]
- The Alzheimer's Drug Discovery Foundation cognitive vitality review explicitly notes that no animal or human studies have evaluated long-term safety of dihexa and raises a theoretical concern that dihexa's potentiation of HGF could augment c-Met signaling in premalignant or malignant tissues, potentially promoting tumor growth or progression.[1]
- This concern is entirely mechanism-based at present: the same HGF/c-Met axis that is neurotrophic and reparative in CNS is also pro-proliferative and pro-invasive in many cancer cells.[6] [1]

Long half-life, tissue persistence, and off-target effects

- Dihexa is designed to be highly stable and to cross the blood–brain barrier, with a long half-life and sustained CNS presence described in preclinical dossiers; this underlies its potent and long-lasting procognitive effects but also raises concerns about chronic pathway activation and cumulative off-target actions.[1]
- Because HGF/c-Met is expressed in multiple peripheral tissues (liver, vasculature, kidney, etc.), chronic systemic exposure could modulate angiogenesis, fibrosis, and metabolic pathways outside the CNS, with unknown net effects in humans.[6] [1]

Neuroplasticity and network-level concerns

- The same structural plasticity that supports recovery from injury and cognitive enhancement could, in principle, produce maladaptive circuitry if applied in inappropriate contexts (e.g., aberrant plasticity in psychiatric conditions, epilepsy, or in the presence of toxic inputs), but this remains speculative; no controlled animal data show seizure or behavioral toxicity at therapeutic ranges yet.[5] [1]

Clinical safety dataset limitations

- For dihexa itself (the peptide), there are no long-term animal carcinogenicity studies or chronic toxicity series reported in the peer-reviewed literature, and no formal human trials.[1]

- For fosgonimeton, early trials indicate good short-term tolerability and absence of serious adverse events, but total exposure time and cohort sizes are modest, and there is no published long-term malignancy surveillance or multi-year safety follow-up.[7] [3]

Structured summary

Aspect	Evidence	Key points
Primary mechanism	Cell, slice, rodent synaptogenic studies [5] [2] [6]	AngIV-derived HGF mimetic; high-affinity HGF binding; potentiates HGF-dependent c-Met activation; drives synaptogenesis and neurotrophic signaling.
Preclinical cognition	Rat cognitive dysfunction models, neuroinflammatory models [5] [4] [1]	Improves learning/memory, increases synapse number, preserves neuronal structure, reduces neuroinflammatory markers.
Human PD signal	Phase I fosgonimeton in AD, ERP/qEEG data [3] [7]	Safe and well tolerated short term; improves ERP P300 latency and qEEG gamma power, consistent with enhanced synaptic function.
Human clinical benefit	26-week AD trial [8]	Numerical but not statistically significant benefit on cognition and function; some favorable biomarker shifts.
Long-term safety	ADDF review, HGF/c-Met oncology literature [1] [6]	No chronic toxicology or carcinogenicity data for dihexa; theoretical cancer risk due to c-Met activation; HGF/MET overexpression linked to worse outcomes in many cancers.
Overall risk–benefit status	Integrated across above [1] [3] [5] [8]	Strong preclinical synaptogenic and procognitive signal; early human safety with mechanistic PD readouts; efficacy in AD not yet proven; oncogenesis and long-term systemic effects remain major unresolved risks.

If you'd like, I can next pull out specific dosing paradigms, durations, and effect sizes from the core rodent papers (e.g., Benoist et al. synaptogenesis work and Sun et al. 2021) and convert them into HED-style exposure ranges for mechanistic benchmarking rather than human translation.

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Dihexa HGF/c-Met cancer risk evidence from preclinical oncology studies

Based on the preclinical oncology literature, there is robust mechanistic and observational evidence that HGF/c-Met pathway activation drives tumor growth, invasion, metastasis, and treatment resistance across many cancer types, but **no published studies have directly tested dihexa itself for carcinogenic effects in animal tumor models.**[16] [17] [18] [19] [20]

HGF/c-Met as a pro-oncogenic pathway

Core oncogenic functions

c-Met is a receptor tyrosine kinase that, when activated by HGF, promotes tumor cell growth, survival, migration, invasion, angiogenesis, and metastasis. Aberrant HGF/c-Met signaling is frequently dysregulated in malignancies and is associated with tumor progression, aggressive disease phenotype, and poor prognosis.[18] [19]

Cancer types with documented HGF/c-Met involvement

- **Hepatocellular carcinoma (HCC):** c-Met activation promotes tumor cell growth, survival, migration, invasion, angiogenesis, and metastasis; overexpression of MET is associated with poor prognosis and more aggressive HCC.[19] [18]
- **Pediatric solid tumors:** In alveolar rhabdomyosarcoma, MET is highly overexpressed in all specimens, and 62% co-express HGF; loss of Ink4a/Arf combined with HGF/c-Met disruption induces rhabdomyosarcoma with high penetrance and short latency in transgenic mice. In neuroblastoma, HGF-induced c-Met phosphorylation drives ligand-induced migration and proliferation.[21] [22]
- **Colorectal and other epithelial cancers:** c-Met overexpression correlates with invasive growth, epithelial-to-mesenchymal transition (EMT), and metastasis; TNF-α-driven inflammation potentiates c-Met signaling to promote invasive growth and metastasis.[17] [20] [16]
- **General solid tumors:** HGF/c-Met is a central mediator of invasive growth, and MET is a transcriptional target of NF-κB, linking chronic inflammation to invasive cancer phenotypes.[20] [16] [17]

Mechanistic basis for oncogenesis

The same HGF/c-Met axis that supports neuronal survival, synaptogenesis, and tissue repair in the CNS is a well-established **"invasive growth" genetic program** in cancer. Activation of c-Met by HGF recruits multiple downstream effectors (PI3K, SHP2, Ras, PLC-γ, STAT3), leading to sustained signaling that drives cell proliferation, anti-apoptotic survival, angiogenesis, and metastatic dissemination.[22] [16] [21] [17] [19] [20]

HGF/c-Met signaling promotes:

- **Epithelial-to-mesenchymal transition (EMT):** A prerequisite for invasive growth and metastasis, driven by TNF-α and other inflammatory mediators acting through c-Met.[20]
- **Angiogenesis:** Tumor vascularization via VEGF and related pathways is augmented by HGF/c-Met activation, supporting tumor expansion and dissemination.[23] [19]
- **Resistance to therapy:** c-Met induction by ionizing radiation confers radioresistance and invasive growth; HGF-driven survival signaling allows cancer cells to resist chemotherapy and targeted therapies.[16] [17]

Therapeutic targeting of HGF/c-Met in oncology

The oncogenic potential of HGF/c-Met has led to development of multiple therapeutic strategies:

- **HGF-neutralizing antibodies (e.g., TAK-701):** Humanized antibody against HGF, tested in preclinical models to block ligand-driven c-Met activation and tumor growth, with demonstrated antitumor activity against pediatric solid tumors and adult cancers.[21] [22]
- **c-Met small-molecule inhibitors (e.g., PHA665752):** These inhibit ligand-induced c-Met phosphorylation, migration, and proliferation in neuroblastoma and other c-Met–positive cancers.[22] [21]
- **Clinical development in HCC and other cancers:** c-Met inhibitors and HGF antagonists are being pursued in hepatocellular carcinoma and other MET-driven malignancies because of the pathway's central role in aggressive tumor behavior.[18] [19]

Absence of dihexa-specific oncogenicity data

Despite the extensive preclinical oncology literature on HGF/c-Met, **no peer-reviewed studies have evaluated dihexa for tumorigenic, carcinogenic, or cancer-promoting effects in animal models.** The safety concern for dihexa is entirely **mechanism-based and extrapolated** from the known role of HGF/c-Met in cancer:

- Dihexa is a high-affinity HGF mimetic that allosterically potentiates HGF/c-Met signaling at subnanomolar concentrations.
- Because the same c-Met pathway that is neuroprotective and synaptogenic in brain is pro-oncogenic in cancer, chronic or systemic dihexa exposure could theoretically amplify c-Met signaling in tissues with pre-malignant lesions, established tumors, or MET overexpression, potentially accelerating tumor growth, invasion, or metastasis.
- No formal carcinogenicity studies (2-year rodent bioassays), chronic toxicology series, or tumor xenograft/implant experiments with dihexa have been published or referenced in the preclinical or clinical literature.

Risk assessment summary

Evidence type	Findings	Implication for dihexa
HGF/c-Met in cancer biology	Pathway drives tumor growth, invasion, metastasis, EMT, angiogenesis, therapy resistance; overexpression correlates with poor prognosis across many cancers[16] [17] [18] [19] [20]	Dihexa's potentiation of HGF/c-Met could plausibly enhance these pro-oncogenic processes if used in individuals with occult or established malignancy.
Pediatric and adult tumor models	HGF/c-Met drives rhabdomyosarcoma, neuroblastoma, HCC, and colorectal cancer in preclinical models; HGF neutralization and c-Met inhibition show antitumor efficacy[21] [22] [18] [19]	Augmenting HGF activity (as dihexa does) runs counter to therapeutic strategies that block this pathway in cancer.

Evidence type	Findings	Implication for dihexa
Inflammation and c-Met cross-talk	TNF-α and inflammatory microenvironment upregulate c-Met and drive invasive growth; MET is a transcriptional target of NF-κB [16] [17] [20]	In inflammatory or injury contexts where dihexa might be used, c-Met pathway amplification could have unintended pro-tumorigenic consequences.
Direct dihexa oncogenicity studies	None published in peer-reviewed preclinical oncology literature	**Critical data gap**: no animal carcinogenicity, chronic toxicity, or tumor-promotion studies for dihexa itself.

The oncogenic risk of dihexa is **theoretical but mechanistically grounded**, derived from decades of HGF/c-Met oncology research, not from direct testing of the compound. For any chronic or widespread use, formal carcinogenicity assessment (including 2-year rodent studies and tumor xenograft safety studies) would be required to quantify this risk.

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fosgonimeton compare to DIHEXA

Fosgonimeton (ATH-1017) is a subcutaneously administered prodrug designed as a clinically viable derivative of dihexa, with modifications to optimize pharmacokinetics, tolerability, and regulatory development, but both compounds act through the same HGF/c-Met potentiation mechanism.[31]

Chemical and pharmaceutical relationship

Fosgonimeton is structurally and mechanistically derived from dihexa, which itself is an orally active, blood-brain barrier-permeable analog of Norleucine¹-AngIV (Nle¹-AngIV). The core pharmacophore—HGF allosteric potentiation—is conserved across the developmental lineage: Nle¹-AngIV → dihexa → fosgonimeton.[31]

Feature	Dihexa	Fosgonimeton (ATH-1017)
Chemical name	N-hexanoic-tyrosine-isoleucine-(6) aminohexanoic amide	Proprietary prodrug form of dihexa pharmacophore
Route	Oral (preclinical)	Subcutaneous injection (clinical)
Development stage	Experimental; no formal clinical trials	Phase I/II clinical trials completed
Regulatory status	Research chemical; not FDA-evaluated	IND-enabled, clinical-stage investigational drug
Mechanism	High-affinity HGF binding (K_d = 65 pM); allosteric potentiation of HGF/c-Met signaling[31]	Same: positive modulator of HGF/c-Met

Fosgonimeton was developed to address the pharmacokinetic and formulation barriers that prevent direct clinical use of dihexa, while retaining its potent HGF-mimetic synaptogenic activity.[31]

Shared mechanism: HGF/c-Met allosteric potentiation

Both compounds bind HGF with high affinity and act as **positive allosteric modulators** of the HGF/c-Met system rather than direct c-Met agonists.[31]

Core mechanistic features (demonstrated for dihexa, presumed conserved in fosgonimeton)

- **High-affinity HGF binding:** Dihexa binds HGF saturably with $K_d = 65$ pM.[31]
- **Inhibition of HGF dimerization but potentiation of c-Met activation:** Despite blocking HGF dimer formation (like HGF antagonists), dihexa and Nle^1-AngIV markedly augment HGF-dependent c-Met phosphorylation at subnanomolar concentrations, suggesting allosteric stabilization of an active HGF conformation that interacts with preformed c-Met multimers on neuronal membranes.[31]
- **Synaptogenic and procognitive effects dependent on HGF/c-Met:** In dissociated hippocampal neurons and organotypic slices, dihexa (10^{-12} M) and HGF (10 ng/ml) both significantly increase dendritic spine density; subthreshold dihexa (10^{-13} M) + subthreshold HGF (2.5 ng/ml) produce maximal spinogenesis, confirming synergistic HGF/c-Met pathway engagement.[31]
- **Blockade by HGF antagonist:** The procognitive effects of orally delivered dihexa in scopolamine-impaired rats (Morris water maze) are abolished by intracerebroventricular administration of an HGF antagonist (Hinge peptide), confirming that dihexa's cognitive benefit requires HGF/c-Met activation.[31]

Fosgonimeton is described as a "positive modulator of HGF/MET" with the same target engagement, supporting the inference that its clinical effects arise from the same allosteric HGF potentiation.

Preclinical data: dihexa

Dihexa has extensive rodent validation but **no formal human trials**:

- **Cognitive rescue:** Orally administered dihexa (2 mg/kg/day) reverses scopolamine-induced spatial learning deficits in rats and improves performance in aged animals.[31]
- **Synaptogenesis and functional connectivity:** Dihexa increases dendritic spine density, presynaptic (synapsin, VGLUT1) and postsynaptic (PSD-95) markers, and miniature excitatory postsynaptic current frequency in cultured hippocampal neurons, indicating formation of functional synapses.[31]
- **HGF/c-Met dependence:** c-Met shRNA knockdown blocks dihexa-induced spinogenesis, and the HGF antagonist Hinge abolishes dihexa's procognitive effects in vivo.[31]
- **BBB penetrance and oral activity:** Dihexa is orally bioavailable and crosses the blood-brain barrier, with sustained CNS effects.[31]
- **Safety data gap:** No published chronic toxicity, carcinogenicity, or oncogenicity studies for dihexa itself.[31]

Key preclinical finding

Dihexa's mechanism was definitively linked to HGF/c-Met in the 2014 *J Pharmacol Exp Ther* paper, which demonstrated that dihexa binds HGF, potentiates c-Met activation, drives synaptogenesis, and produces procognitive effects that are blocked by HGF antagonism. **Note:** This paper has been retracted (2025), but the mechanistic framework (HGF allosteric modulation) is consistent with fosgonimeton's described target engagement.[31]

Clinical data: fosgonimeton (ATH-1017)

Fosgonimeton has completed phase I and exploratory phase II trials, with published safety, PK, and PD data.

Phase I: healthy volunteers and AD subjects (NCT03298672)

- **Design:** Randomized, double-blind, placebo-controlled; single ascending dose (SAD) in healthy young males (n=48; 2–90 mg SC), multiple ascending dose (MAD) in healthy elderly (n=29; 20–80 mg SC × 9 days), and fixed dose in mild-to-moderate AD subjects (n=11; 40 mg SC × 9 days).
- **Safety and tolerability:** Fosgonimeton and placebo were safe and well tolerated across all doses; no serious adverse events or dose-limiting toxicities.
- **Pharmacokinetics:** Dose-proportional PK; no sex effect or accumulation over 9 days; suitable for once-daily SC dosing.
- **Pharmacodynamics (PD):** qEEG showed acute and sustained gamma power induction (marker of synaptic activity); in AD subjects, fosgonimeton (40 mg) produced significant ERP P300 latency normalization versus placebo ($p=0.027$, n=7 vs. n=4), suggesting enhanced synaptic function and potential procognitive effects.

Phase II efficacy trial in Alzheimer's disease (ACT-AD, 26 weeks)

- **Outcome:** Fosgonimeton showed numerically favorable changes on global and cognitive measures (e.g., ADAS-Cog11, ADCS-ADL23) versus placebo, but **did not meet statistical significance for primary or key secondary endpoints.**[32]
- **Biomarkers:** Trends toward favorable effects on NfL, GFAP, plasma phosphorylated tau species, and Aβ42/Aβ40 ratio, suggesting possible disease-modifying or neuroprotective activity, but underpowered or dose/duration-dependent.[32]
- **Interpretation:** Mechanistic PD signal (P300, gamma) is present and consistent with synaptic potentiation, but translation to clinically meaningful cognitive benefit in AD was not demonstrated at the tested dose and duration.[32]

Key differences: dihexa vs. fosgonimeton

Aspect	Dihexa	Fosgonimeton
Formulation	Oral peptide (preclinical)	Subcutaneous prodrug (clinical)

Aspect	Dihexa	Fosgonimeton
Pharmacokinetics	Long CNS half-life, high BBB penetrance (rodent)[31]	Dose-proportional, no accumulation, SC once daily (human)
Human PK/PD data	None	Phase I: safe, well tolerated, dose-proportional; PD: gamma power ↑, P300 latency normalization in AD
Human efficacy data	None	Phase II: no significant cognitive benefit at 26 weeks in AD[32]
Safety profile	No chronic toxicity or carcinogenicity studies published	Short-term human safety established; no serious AEs in phase I; long-term and oncogenicity data pending
Regulatory path	Research chemical; no IND	IND-enabled; completed phase I, exploratory phase II; further development uncertain after ACT-AD neutral result[32]

Mechanistic translation and clinical implications

Both dihexa and fosgonimeton exploit the same **HGF/c-Met neurotrophic axis** to drive synaptogenesis and synaptic potentiation. The preclinical rodent data for dihexa demonstrate robust synapse formation, increased functional connectivity (mEPSC frequency), and procognitive effects that are HGF/c-Met–dependent. Fosgonimeton's phase I PD signals (ERP P300 latency normalization, gamma power induction) are consistent with acute synaptic enhancement and align with dihexa's mechanistic profile.[31]

However, the **failure of fosgonimeton to produce statistically significant cognitive benefit in the 26-week AD trial** highlights a critical translational gap:[32]

- **Dose and duration:** 40 mg SC daily for 26 weeks may be insufficient for sustained, clinically meaningful synaptogenesis in advanced neurodegeneration.
- **Disease stage:** Synaptic potentiation may require viable neuronal substrate; in moderate-to-severe AD, neuronal loss may limit the capacity for new synapse formation.
- **Outcome measures:** Synaptic PD endpoints (P300, gamma) are sensitive to acute effects, but cognitive and functional scales (ADAS-Cog, ADL) may require longer treatment or earlier-stage intervention.
- **HGF/c-Met pathway complexity:** While HGF/c-Met is neurotrophic and synaptogenic in healthy brain and injury models, its efficacy in chronic neurodegenerative conditions with widespread pathology (amyloid, tau, inflammation) remains uncertain.

Unresolved oncogenic risk (applies to both)

As detailed in prior analysis, **HGF/c-Met is a pro-oncogenic pathway** in many cancers, driving tumor growth, invasion, metastasis, and therapy resistance. Neither dihexa nor fosgonimeton has been evaluated in formal carcinogenicity studies (2-year rodent bioassays) or tumor xenograft safety models. The phase I fosgonimeton trial (short duration, small cohorts) did not include long-term malignancy surveillance. For chronic or widespread use, this remains a **critical safety data gap** for both compounds.[33] [34] [35] [36] [37]

Summary: Fosgonimeton is a clinically optimized, subcutaneous prodrug of the dihexa pharmacophore, sharing its mechanism (HGF/c-Met allosteric potentiation) and demonstrating early human PD signals of synaptic enhancement (P300, gamma). However, fosgonimeton has not yet shown clinically significant cognitive benefit in Alzheimer's disease, and both compounds lack long-term safety data, particularly regarding oncogenic risk from chronic HGF/c-Met pathway activation.

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