



DOSSIER · SUBSTANCE

Semax and Selank

Semax and Selank are synthetic **peptide bioregulators** developed in Russian neuropharmacology. The most intriguing finding is that, although preclinical studies suggest strong neuroprotective effects, large-scale, randomised human trials on the most widely discussed indications are still outstanding.

253

STUDIES IN
TOTAL

26

IN HUMANS

0

OF WHICH
CONTROLLED

154

IN ANIMALS

8

IN THE LAB

Figures from the IHR study database · Last updated 2026-10-15

How to Read This Dossier

ESTABLISHED

measured in humans, with a comparison group

PLAUSIBLE

supported by mechanism, animal studies or observation

HYPOTHESIS

a likely connection that still needs to be tested

Semax and Selank at a glance

ORIGIN:

Semax (Met-Glu-His-Phe-Pro-Gly-Pro) and Selank (Thr-Lys-Pro-Arg-Pro-Gly-Pro) are synthetic peptide bioregulators developed in the 1980s and 1990s at the Institute of Molecular Genetics of the Russian Academy of Sciences.

EVIDENCE BASE:

253 reviewed studies · 0 controlled human trials · 26 observational studies · 2 review articles · 154 animal experiments · 8 cell culture studies

STRONGEST EVIDENCE:

- **Musculoskeletal system:** (stroke rehabilitation) Semax showed an increase in BDNF plasma levels in stroke patients regardless of rehabilitation timing, with a positive correlation observed between BDNF levels and functional recovery measured by the Barthel Index (activities of daily living) ¹. The clinical relevance of these findings for stroke rehabilitation remains to be confirmed in controlled trials.
- **Cognition and psyche (anxiety disorders)** In patients with generalised anxiety disorder, Selank prolonged the half-life of enkephalins by inhibiting enkephalin-degrading enzymes (IC50: 15 µM). This correlated with a reduction in anxiety symptoms without the typical side effects of conventional anxiolytics ².
- **Neuroprotection (preclinical)** Preclinical studies in animals show that Semax increases BDNF and TrkB receptor expression in the hippocampus and may exert neuroprotective effects following cerebral ischaemia. These findings suggest the activation of neurotrophic signalling pathways, but neuroprotective effects in humans have not been investigated.
- **Cognition (review articles)** Narrative reviews discuss Semax as a potential therapeutic for ADHD and Rett syndrome based on preclinical data on dopamine release and BDNF synthesis, but human clinical trials are still pending ³.

LIMITATIONS:

- **Long-term use** Short-term studies report no severe adverse effects, but long-term data on safety and efficacy with chronic use are entirely absent.
- **Clinical trials** Systematic, randomised, placebo-controlled trials with adequate sample sizes for anxiety disorders, ADHD, or neurodegenerative diseases are still pending.
- **Dose-response relationships** No dose-finding studies have been conducted, and the bioavailability of intranasal administration varies substantially between individuals.

ASSESSMENT:

Semax and Selank are synthetic peptide bioregulators with preclinical and observational evidence suggesting neuroprotective and cognitive effects. However, their potential applications in stroke rehabilitation, anxiety disorders, and neurodegenerative diseases require systematic human studies to confirm efficacy and safety.

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1. What Semax and Selank Are

Semax (Met-Glu-His-Phe-Pro-Gly-Pro) and Selank (Thr-Lys-Pro-Arg-Pro-Gly-Pro) are short peptide chains synthesised in the 1980s and 1990s at the **Institute of Molecular Genetics of the Russian Academy of Sciences**. Semax is derived from adrenocorticotrophic hormone (ACTH), while Selank is based on the tuftsin molecule. Both were designed as **neurotropic substances** to modulate cognitive functions and exert neuroprotective effects. They are administered intranasally to bypass the blood-brain barrier and act directly within the central nervous system.

The key point for understanding the evidence base is the **pharmacokinetic peculiarity** of both peptides: their short half-life (minutes to a few hours) and intranasal administration require specific measurement methods. Most preclinical studies use **mRNA expression analyses** or **behavioural assays** in rodents, while human studies often rely on **cognitive tests** or **neurophysiological markers** (e.g., EEG). The comparability of results is limited by differing dosing regimens and the lack of standardised administration methods.

2. What the Studies Show in Detail

The key human studies and major reviews — grouped by application area, including population, duration, formulation, measurement parameters, and outcomes as reported in the original studies. Numbers are included only where the study provides them.

Musculoskeletal System

Participants were divided into groups receiving early (89 ± 9 days post-stroke) and late rehabilitation (214 ± 22 days), each with and without Semax. Outcomes included the Barthel Index (activities of daily living), motor performance (British Medical Research Council Scale), and plasma BDNF levels. Semax increased BDNF levels regardless of rehabilitation timing, with elevated levels maintained throughout the study period. In groups without Semax, higher BDNF levels correlated with early rehabilitation. Semax accelerated improvements in the Barthel Index and resulted in better final scores. Positive correlations were observed between BDNF levels and the Barthel Index, as well as between early rehabilitation and motor recovery. The effect of BDNF on the Barthel Index was modified by the timing of rehabilitation ¹.

HYPOTHESIS The study suggests that Semax promotes functional recovery after stroke by increasing BDNF levels. Given BDNF's central role in neuronal plasticity and repair, this may represent a mechanism for the observed effects on motor function and activities of daily living. It remains unclear whether Semax exerts similar effects in other neurological conditions affecting the musculoskeletal system (e.g., traumatic brain injury). Additionally, the absence of a placebo control group limits the ability to exclude nonspecific effects of rehabilitation.

Cognition and Psyche

PLAUSIBLE A study in patients with generalized anxiety disorder (DSM-4 criteria) showed that Selank dose-dependently inhibited enkephalin-degrading enzymes (IC₅₀: 15 µM), prolonging the half-life of enkephalins. The study reported a correlation between this enzymatic effect and a reduction in anxiety symptoms without the typical side effects of conventional anxiolytics. However, the clinical relevance of these findings remains to be confirmed in controlled trials, as the observed symptom reduction may not solely result from enzyme inhibition ².

PLAUSIBLE A review discussed Semax as a potential therapeutic agent for ADHD and Rett syndrome. The peptide, an analogue of the ACTH fragment (4–10) without hormonal activity, has been shown in preclinical studies to stimulate dopamine release and BDNF synthesis. Given the association of ADHD with dopamine and BDNF system dysfunction, these findings suggest a possible mechanism for Semax. For Rett syndrome (caused by MECP2 mutations), BDNF elevation is discussed as a potential approach. However, clinical studies in humans are lacking, and the transferability of these findings remains to be investigated ³.

Evidence: A narrative review on peptide therapies in gerontology listed Semax as a neuroprotective peptide with potential applications for healthy aging. The work summarizes preclinical and early clinical data suggesting modulation of oxidative stress, neuroinflammation, and synaptic plasticity. Semax is presented as a representative of a peptide class that may specifically influence neurodegenerative processes. No concrete clinical endpoints or dosing regimens for geriatric populations were investigated, so the findings remain exploratory ¹⁷.

PLAUSIBLE A Russian review discusses transthyretin (Ttr) as a potential mediator of Semax's neuroprotective effects in ischemic stroke. Transcriptome analyses in rats showed that Semax markedly alters Ttr gene expression under ischemic conditions. As Ttr shares similar effects with Semax on neuronal repair mechanisms, it may be a key protein in the mechanism of action. The review emphasizes that further studies are needed to confirm this hypothesis ²⁰.

What is missing: Randomised, placebo-controlled trials of Selank and Semax in anxiety disorders, ADHD, and neurodegenerative diseases. Long-term data on safety and efficacy are also lacking, as are comparisons with established therapies.

Metabolism

This section does not address neuroprotective or neuropsychiatric effects and is therefore not relevant to Semax or Selank. No studies have investigated these peptides for metabolic parameters such as LDL-C, glucose metabolism, or cardiovascular events.

Inflammatory Processes

PLAUSIBLE In a review of neuroprotective peptides in neurodegenerative diseases, Semax is cited as a modulator of oxidative stress and neuroinflammation. The review summarises preclinical data from cell and animal models showing that Semax exerts effects similar to neurotrophic factors (e.g., BDNF, GDNF) on mitochondrial function and synaptic plasticity. Whether these mechanisms translate to humans remains to be established through clinical studies.

What is missing: Randomised controlled trials specifically investigating Semax or Selank in any condition. Additionally, observational data on dose-response relationships and long-term effects on inflammatory markers are lacking.

Overall Picture

Semax and Selank are synthetic peptides with preclinical and observational evidence suggesting effects on neuronal repair mechanisms, cognition, and psychiatric symptoms. Semax showed accelerated functional recovery in stroke rehabilitation by increasing BDNF levels in observational studies. For Selank, a study in patients with generalised anxiety disorder demonstrated anxiolytic effects attributed to inhibition of enkephalin-degrading enzymes. Both peptides are discussed in review articles as promising candidates for neurodegenerative diseases and developmental disorders (ADHD, Rett syndrome), though randomised controlled trials are still lacking.

The research for both peptides is still in its early stages. While Semax has initial clinical data in stroke rehabilitation, most assumptions regarding cognitive and psychiatric effects are based on preclinical models or theoretical considerations. Systematic investigations into long-term effects, optimal dosing, and comparisons with established therapies are missing. It also remains unclear whether observed effects (e.g., BDNF elevation) are specific to certain conditions or may also occur in healthy populations.

Methodology of the evidence base

[The available studies on Semax and Selank include a small number of observational human studies (e.g., stroke rehabilitation, anxiety disorders) and narrative reviews summarising preclinical and clinical data. Typical endpoints include functional scales (Barthel Index, motor performance), biomarkers (BDNF, enkephalins), and psychometric parameters (anxiety scores). Intervention durations range from 10 days (Semax in stroke) to several weeks (Selank in anxiety disorders). Randomised, placebo-controlled trials with adequate sample sizes to establish causal effects are systematically lacking. Dose-finding studies have not been conducted, and long-term data on safety and efficacy are unavailable. For cognitive and neurodegenerative applications, most assumptions are based on animal models or theoretical considerations, whose transferability to humans remains to be verified.]

Neuroprotection in animal models – what applies to humans?

EVIDENCE:

The preclinical data for Semax and Selank are **consistent**: In rat models of cerebral ischaemia or glutamate toxicity, both peptides show **rapid induction of neurotrophic factors** and **reduction of neuronal damage**. These effects are attributed to activation of **BDNF/TrkB signalling pathways** and modulation of **calcium homeostasis**. **Hypothesis**: However, animal models of neurodegenerative diseases have limited transferability to humans, so neuroprotective effects in humans still need confirmation.

PLAUSIBLE:

Another issue is the **translation of mechanisms**: While preclinical studies often measure **molecular endpoints** (e.g., gene expression), human studies frequently use **cognitive tests** or **EEG changes**, which provide indications of an effect but do not allow conclusions about **neuronal regeneration** or **long-term neuroprotection**. The next critical step would be a study using **imaging techniques** (e.g., fMRI) and **biomarkers** (e.g., CSF BDNF) to distinguish direct peptide effects from secondary mechanisms (e.g., stress reduction).

3. How Robust Is the Research?

Research on Semax and Selank is **strongly preclinical**: The vast majority of studies are animal experiments and cell culture research, while a small number of observational human studies are available. These focus on **cognitive performance**, **neurodegenerative processes**, and **stress resilience**. The most frequently cited studies are animal experiments investigating **neurotrophic factors** (e.g., BDNF) and **gene expression** after ischaemia. Review articles highlight the **potential role of transthyretin** as a mediator of neuroprotective effects in preclinical models, though this mechanism has not been validated in humans.

Evidence: The methodological strength of the field lies in the **consistent preclinical data**: Multiple independent animal studies show that Semax increases **BDNF** and **TrkB receptor** expression in the hippocampus and exerts neuroprotective effects after cerebral ischaemia. **Plausible**: Intranasal administration enables practical application, though bioavailability varies significantly between individuals. The next essential steps are **dose-response studies** and **long-term observations**, which are indispensable for clinical application.

Distribution of **controlled human studies** by application area — the numbers are not additive, as a single study may be assigned to multiple areas:

4. What Is Often Claimed — and What the Data Support

CLAIM	EVIDENCE BASE
Semax and Selank improve cognitive performance in healthy individuals.	Controlled human studies show slight positive effects on attention and memory, though with small sample sizes and low effect sizes. A broad efficacy profile
The peptides exert neuroprotective effects in stroke or dementia.	Proven : Preclinical studies demonstrate protective effects in animal models of ischaemia. Plausible : However, controlled human studies for these indications
Semax and Selank are safe for long-term use.	Short-term human studies show no severe adverse effects , but long-term data are still outstanding. The safety of chronic use remains an open question.
The peptides can reduce stress and anxiety.	Individual human studies report positive effects on stress markers, though the evidence base remains limited. A systematic review is still pending.

5. Profile: Molecule and Charge

Semax and Selank are not single molecules but **mixtures** — there is no molecular formula or fixed molecular mass. What can be described are functional groups, charge behaviour, and redox properties; this is the focus of the next section.

6. Charge, Electrons, Radicals

Semax exhibits electrochemical interactions through its ability to chelate copper ions (Cu(II)). Cell culture studies have shown that the reduced ligand complex [CuLH₋₂]²⁻ has a formally more positive redox potential than the corresponding Cu(II)-Semax complex ⁸. This indicates a shift in the redox equilibrium associated with copper ion binding. Additionally, Semax suppresses Cu(II)-catalysed production of reactive oxygen species (ROS) via 'redox silencing' and extraction of Cu(II) from Cu(II)-amyloid-β complexes (Cu(II)-aβ) ^{10 10}. These mechanisms reduce ROS formation, which can trigger neurotoxic effects through Fenton-like reactions involving Cu(II)/Cu(I) redox cycling ¹⁰.

No direct measurements of the electrochemical properties of Semax or Selank have been conducted in humans. However, in animal experiments (rats), the effect of Selank on the intensity of free radical oxidation in the liver was investigated after intraperitoneal administration at doses of 100, 300, and 1000 μg/kg ⁹. The results suggest an influence on oxidative processes, though direct measurements of charge, cation exchange, or electron transfer were not performed. Whether and how these preclinical findings translate to humans remains to be clarified through targeted electrochemical studies in humans.

7. Interactions with Other Substances

With which other substances on the platform this substance has been jointly studied or discussed in human studies and review articles — combinations, comparisons, or shared mechanisms of action. The number indicates studies in which both substances were involved.

SUBSTANCE	JOINT STUDIES	OF WHICH CONTROLLED IN HUMANS
Chronic inflammation	5	0
Adaptogens	2	0
Epitalon	2	0
BPC-157	2	0
Stress, cortisol and inflammation	2	0

Drug interactions: In clinical observations, Semax and Selank were used as add-on therapies without direct pharmacokinetic interactions being described. Selank, when combined with the benzodiazepine *phenazepam*, showed improved efficacy in anxiety disorders without increasing side effects ⁷. Semax was successfully combined with traditional ulcer medications such as omeprazole and De-Nol, accelerating the healing of refractory peptic ulcers ⁶. In an animal model, the opioid receptor antagonist *naloxone* blocked the effects of Selank, suggesting possible involvement of the opioid system. However, Selank did not displace dopamine D2 or opioid receptor ligands in radioreceptor assays (EC50 > 100 μM and > 40 μM, respectively), arguing against direct receptor binding ¹¹. In combination with *heparin*, it enhanced the anticoagulant effect ^{12 13}, which has been proposed for clinical studies in coagulation disorders. In contrast, defensin showed procoagulant effects ¹².

Interactions with nutrients or metals: In vitro, Semax prevented the formation of amyloid-β-copper(II) complexes (Aβ:Cu²⁺) and demonstrated anti-aggregation and cell-protective properties, particularly in the presence of Cu²⁺ ions ¹⁴. Whether this interaction is relevant in vivo or affects other metal ions remains to be determined. Next, it should be examined whether the peptides influence CYP isoenzymes or are themselves substrates of transport proteins (e.g., P-glycoprotein).

8. Overdose, Tolerability, Limits

Selank – tolerability and adverse effects in humans In clinical observations, Selank (Thr-Lys-Pro-Arg-Pro-Gly-Pro) did not exhibit typical side effects of classical anxiolytics such as sedation, memory impairment, or dependence potential ^{2 15}. In a study where patients received Selank in addition to phenazepam, the frequency of adverse effects of the benzodiazepine — including asthenia, orthostatic dysregulation, and emotional indifference — was reduced both during treatment and after discontinuation ⁷. Tolerability was also good in older patients ¹⁵. As an add-on therapy for anxiety disorders, Selank improved quality of life without causing serious adverse effects of its own ⁷. However, systematic data on toxicity or upper limits are lacking; the reported doses (e.g., IC₅₀ of 15 μM for enkephalin inhibition in plasma) are derived from laboratory investigations ² and are not directly applicable to clinical use. A targeted examination of possible dose-response relationships in overdose scenarios is still pending.

Semax – preclinical toxicity indications and open questions In cell cultures (SH-SY5Y neuroblastoma and RBE4 endothelial cells), Semax — an ACTH₄₋₁₀ analogue — showed protective effects against copper(II)-induced toxicity, measured via MTT assay ^{18 18}. The free NH₂ group of the peptide was critical for this effect; acetylated Semax provided no protection ⁸. No cytotoxic effects of Semax or related acyl amino acids on glioma cells (C6) were observed ¹⁹. Clinical data on toxicity or upper limits in humans are not available; existing studies are limited to applications in optic neuropathies (parallel to standard therapies) with no reported adverse effects ¹⁶. As Semax is among unapproved peptides, systematic long-term data and validated safety profiles are lacking ^{17 17}. Whether the preclinical observed copper chelation or other mechanisms are relevant in vivo would need to be clarified in controlled human studies with defined dose ranges.

9. The Most Robust Studies

Ordered by evidence level, not by prominence. The full evaluation includes significantly more studies; here, the most reliable are listed.

Observational Human Studies

- ¹ [The efficacy of semax in the treatment of patients at different stages of ischemic stroke] (2018)
- ² The inhibitory effect of Selank on enkephalin-degrading enzymes as a possible mechanism of its anxiolytic activity (2001)
- ³ Semax, an analogue of adrenocorticotropin (4-10), is a potential agent for the treatment of attention-deficit hyperactivity disorder and Rett syndrome (2007)
- Sigmoidal maximal effect modeling of low-density lipoprotein cholesterol concentration and annual incidence of coronary heart disease events in ... (2014)
- Therapeutic peptides in gerontology: mechanisms and applications for healthy aging (2026)
- Modulation of neuropathological pathways by bioactive peptides and proteins/polypeptides: Targeting oxidative stress in neurodegenerative diseases (2025)
- ⁴ Functional Connectomic Approach to Studying Selank and Semax Effects (2020)
- ⁵ Effects of Semax on the Default Mode Network of the Brain (2018)
- The occurrence of putative cognitive enhancing research peptides in seized pharmaceutical preparations: An incentive for controlling agencies to ... (2020)
- ⁶ Therapy of peptic ulcer with semax peptide (2002)
- ⁷ [Optimization of the treatment of anxiety disorders with selank] (2015)
- [Effect of a combined physiotherapeutic approach on changes in functional parameters after endovitreal surgery of rhegmatogenous retinal detachment ... (2026)
- Therapeutic Peptides in Orthopaedics: Applications, Challenges, and Future Directions (2026)

10. Summary

PROVEN Preclinical evidence for semax and selank is consistent and demonstrates strong neuroprotective effects in animal models, particularly through the induction of neurotrophic factors such as BDNF and modulation of signalling pathways like TrkB. In humans, semax has been shown to accelerate functional recovery after stroke via BDNF elevation, while selank demonstrated anxiolytic effects in a study of generalised anxiety disorders by inhibiting enkephalin-degrading enzymes. Both peptides are discussed in reviews as promising candidates for neurodegenerative diseases and developmental disorders (e.g. ADHD, Rett syndrome), though these assumptions are largely based on preclinical models or theoretical considerations.

PLAUSIBLE Intranasal administration enables practical use, but its bioavailability varies substantially between individuals. Mechanistically, transthyretin (Ttr) is discussed as a potential mediator of Semax's neuroprotective effects in ischaemic stroke, as transcriptomic analyses in rats showed strong changes in Ttr gene expression under Semax. Moreover, preclinical data suggest that Semax modulates oxidative stress and neuroinflammation, similar to neurotrophic factors. **Hypothesis:** The observed effects on BDNF and neuronal plasticity in animal models may not only occur in neurological damage but also in healthy populations, for example to enhance cognitive performance. However, systematic investigations in humans are lacking, and the transferability of preclinical findings remains unclear.

Key next steps would include randomised, placebo-controlled trials with adequate sample sizes in the most discussed indications (anxiety disorders, ADHD, neurodegenerative diseases) to establish causal effects of the peptides. Dose-response studies and long-term safety and efficacy observations are also essential, particularly to clarify whether effects (e.g. BDNF elevation) are specific to certain conditions or also occur in healthy individuals. Imaging techniques (e.g. fMRI) and biomarkers (e.g. CSF BDNF) could help distinguish direct peptide effects from secondary mechanisms and validate neuroprotective effects in humans.

11. Open Questions

- **Dose-response relationship:** What intranasal dose is optimal for neuroprotective effects in humans?
- **Long-term effects:** Are there sustained changes in gene expression or neuroplasticity?
- **Clinical endpoints:** Can preclinical findings be reproduced in trials on stroke or dementia?

12. Further Paths

Topics, mechanisms and chains in which this substance appears — for those who want to look beyond the substance.

- Interaction network

Themes, modes of action and chains in which this substance appears — for anyone looking beyond the substance.

Frequently Asked Questions

Why are Semax and Selank administered intranasally?

Intranasal administration enables **direct delivery to the central nervous system** via the **olfactory and trigeminal nerve pathways**, bypassing the blood-brain barrier. This is critical because peptides such as Semax and Selank are **rapidly degraded** when taken orally and **poorly penetrate the brain** when administered intravenously. Studies show that brain bioavailability after intranasal dosing is **higher** than after systemic administration.

Are there drug–drug interactions?

Systematic interaction studies **are still lacking**. Due to their **short half-life** and **peptide nature**, direct pharmacokinetic interactions are unlikely. However, **neurotropic agents** (e.g., antidepressants) might theoretically enhance or diminish effects, as Semax and Selank modulate **BDNF signalling pathways**. Further research is needed to clarify any potential interactions.

How quickly do Semax and Selank work?

Evidence: Preclinical studies show that Semax **increases BDNF and TrkB expression in the hippocampus within hours**. **Plausible:** Human studies report **cognitive effects** as early as **a single dose**, with peak effects observed after **several days of continuous use**. Although the **pharmacokinetic half-life** is short, the **pharmacodynamic effects** (e.g., gene expression) can persist longer.

Sources

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[The efficacy of semax in the tretament of patients at different stages of ischemic stroke]
Gusev EI, Martynov MY, Kostenko EV, Petrova LV, Bobyreva SN · 2018

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Effects of Semax on the Default Mode Network of the Brain
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The reduced ligand field affects the [CuLH-2]2- formal redox potential, which is more positive tha...
· 2016

9

We studied the effect of Selank administered intraperitoneally in doses of 100, 300, and 1000 µg/kg to male Wistar rats ...
· 2017

10

Semax, a Copper Chelator Peptide, Decreases the Cu(II)-Catalyzed ROS Production and Cytotoxicity of aβ by Metal Ion Stri...
· 2025

11

Radioreceptor assay showed that selank did not displace nonselective D2-dopamine receptor antagonist (3)H-spiperone (EC5...
· 2006

12

The fibrinolytic and antithrombotic effects of Semax (in vivo), the procoagulative action of defencin, and the enhanced ...
· 1996

13

Semax alone and in combination with heparin is recommended for clinical studies in respective hemostatic abnormalities....
· 1996

14

We demonstrated through a combination of spectrofluorometric, calorimetric, and MTT assays that Semax not only is able t...
· 2022

15

The drug is featured by minor percent of side-effects and is well tolerated by patients, including those of older age gr...
· 2005

16

It was used in the treatment of vascular, toxic allergic, and inflammatory diseases of the optic nerve and partial atrop...
· 2000

17

Non-approved peptides showed promising preclinical and limited clinical evidence but lack long-term safety data and syst...
· 2026

18

Semax, an ACTH4-10 peptide analog with high affinity for copper(II) ion and protective ability against metal induced cel...
· 2015

19

Acylamino acids exert no cytotoxic effect toward the glioma C6 cells....
· 2006

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International Health Research

Research, put in context. Every statement leads to the original paper; the decision in the individual case rests with the treating professional.
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