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Mini Review

A systematic review of the toxicity of salsolinol and its metabolites

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Abstract

Salsolinol (1-methyl-6,7-dihydroxy-1,2,3,4-tetrahydroisoquinoline) is a tetrahydroisoquinoline compound formed by the condensation reaction between dopamine and acetaldehyde. While it plays a role in normal physiological functions at physiological levels, elevated concentrations have been associated with toxicity. This study aimed to investigate the mechanisms underlying salsolinol toxicity. A literature search was conducted using PubMed and Scopus databases covering the years 2013 to 2023. A total of 6,678 studies were identified using predefined keywords such as "salsolinol," "tetrahydroisoquinoline," "DMDHIQ+," "N-methyl salsolinol," "toxicity," "toxic," and "toxin." Based on predetermined inclusion and exclusion criteria, 6,381 articles were excluded. Seven articles met the eligibility criteria and were critically appraised; all were included in this review. Most of the studies reviewed found that elevated levels of salsolinol in the blood and brain contributed to addiction-like behaviours, particularly in alcoholism, through enhanced dopaminergic signalling in the ventral tegmental area. Two studies examined the oxidative stress effects of salsolinol and its metabolites on neurons and their potential role in cancer development. A novel finding also implicated salsolinol in the degeneration of myenteric neurons, leading to alterations in gut function. Salsolinol and its metabolites exhibit toxic effects in both the central and peripheral nervous systems, primarily through oxidative stress and the modulation of addiction-related pathways. These findings underscore the need for further research to explore potential therapeutic targets to mitigate these pathological effects.

Keywords: DMDHIQ+, N-methyl salsolinol, Salsolinol, tetrahydroisoquinoline, toxic, toxicity, toxin.

1. Introduction

Salsolinol (1-methyl-6,7-dihydroxy-1,2,3,4-tetrahydroisoquinoline) is a tetrahydroisoquinoline compound formed through the condensation of dopamine with acetaldehyde. It was first identified in the urine of Parkinson's disease patients undergoing L-DOPA treatment, suggesting a potential role in disease pathogenesis [1-6], particularly in Parkinson's disease. Salsolinol possesses a catechol structure and contains an asymmetric center at the C1 position, giving rise to two enantiomers: R-salsolinol and S-salsolinol [1]. Figure 1 shows the chemical structure of salsolinol, with the molecular formula $C_{10}H_{10}NO_2$.

There are three pathways involved in the synthesis of salsolinol: i) a non-enzymatic reaction between dopamine with either acetaldehyde or pyruvic acid to form a tetrahydroisoquinoline, followed by decarboxylation and reduction reactions, producing R-salsolinol; ii) a non-enzymatic Pictet-Spengler reaction involving the condensation of dopamine and acetaldehyde, yielding racemic mixtures of R- and S-salsolinol; and iii) the selective enzymatic synthesis of R-salsolinol from dopamine and acetaldehyde via R-salsolinol synthase, whose activity increases following ethanol intake [1,2]. The prevailing hypothesis suggests that salsolinol is synthesized endogenously through these three pathways, as evidenced by studies in rat and human brain tissue. Additionally, several exogenous sources of salsolinol exist, primarily plant- and protein-derived food products such as cheese, cocoa powder, bananas, flour, eggs, beer, and milk, which may elevate blood salsolinol levels. However, these exogenous forms do not cross the blood-brain barrier and therefore do not exert central effects [1,3]. Endogenous synthesis occurs predominantly in brain regions with high dopamine turnover, such as the ventral midbrain and striatum [1,5]. Within the nigrostria-

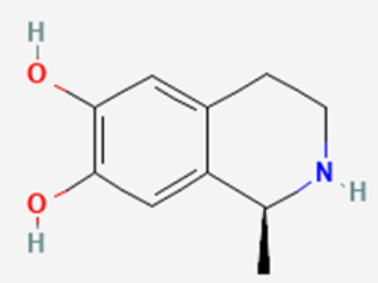

Fig. 1. Chemical structure of salsolinol (1).

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tal system, salsolinol undergoes further methylation by N-methyltransferase, leading to the accumulation of N-methyl-R-salsolinol and its neurotoxic metabolite, 1,2-dimethyl-6,7-dihydroxyisoquinolinium ions (DMDHIQ+) in the substantia nigra [3-5].

At physiological levels, salsolinol plays a role in regulating several functions, including catecholaminergic neurotransmission and neuromodulation of the mesolimbic pathway through interaction with the μ-opioid receptor and acting as a prolactin-releasing factor in the tuberoinfundibular pathway [1,2]. Despite its physiological roles, elevated levels of salsolinol have been associated with toxicity, attributed in part to its chemical structure. Due to its structural similarity to 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), a compound known to cause Parkinsonian symptoms, salsolinol has been hypothesized to exert neurotoxic effects at high concentrations [1,3,5]. A key feature responsible for MPTP toxicity is the N-methyl group. The N-methyl group allows MPTP to penetrate into the cells and be metabolized into the stable toxic metabolite MPP^+, which is taken up by dopaminergic neurons and causes mitochondrial dysfunction leading to cell death. Interestingly, the N-methyl group in salsolinol also contributes to toxicity, but only in the R-enantiomer. N-methyl-R-salsolinol can be further oxidized to form DMDHIQ+, which inhibits mitochondrial enzymes and contributes to cellular damage.

Nevertheless, the role of salsolinol remains enigmatic due to reports of both neurotoxic and neuroprotective effects. Tetrahydroisoquinoline derivatives, including salsolinol, contain both catechol and non-catechol structures. The catechol moiety, in particular, is associated with antioxidant properties [1,2,4,5], though this effect is highly concentration-dependent [1,2,5]. However, more reports have provided evidence of the toxicity of salsolinol rather than its antioxidant capacity.

This systematic review focuses specifically on the toxic effects of salsolinol and its metabolites, with the aim of identifying characteristics that may contribute to the pathophysiology of disease.

2. Materials and Methods

2.1. Search strategy

A systematic literature search was conducted in the PubMed and Scopus databases to identify studies published from 2013 to August 2025 concerning salsolinol, its derivatives, and their toxicological profiles. Both free-text keywords and Boolean operators were applied to enhance search sensitivity and specificity. The search terms included: salsolinol, tetrahydroisoquinoline, DMDHIQ+, N-methylsalsolinol, and their combinations with “toxicity,” “toxic,” or “toxin.” The comprehensive search string was formulated as:

(Salsolinol OR tetrahydroisoquinoline OR DMDHIQ+ OR N-methylsalsolinol) AND (toxicity OR toxic OR toxin).

No language restrictions were applied. Records were screened by title, abstract, and full text when necessary. The search was executed on 10 September 2025, and all retrieved records were documented to ensure transparency and reproducibility. This strategy adhered to established systematic review guidelines to achieve a balance between comprehensiveness and precision.

2.2. Study Selection

The selection of studies for inclusion in this systematic review was performed according to a rigorous and predefined protocol based on explicit inclusion and exclusion criteria aligned with the research objectives.

Initially, all retrieved records were collated, and duplicate entries were systematically identified and removed. The study selection process was conducted in two independent screening phases to ensure reproducibility and minimize bias:

Title and Abstract Screening:

Two reviewers independently screened the titles and abstracts of all identified records against the eligibility criteria. A standardized screening checklist was developed and pilot-tested to guide decision-making and enhance inter-rater reliability. Discrepancies in decisions were resolved through discussion or consultation with a third reviewer when consensus was not reached. Records irrelevant to the research scope were excluded, and the rationale for exclusion was documented.

Full-Text Screening:

Full-text articles of all potentially eligible studies from the first phase were retrieved and independently assessed by the same reviewers for final eligibility. This phase emphasized critical appraisal to confirm that studies addressed the focused research question, employed valid methodology, and reported relevant outcomes. Reasons for exclusion at this stage were recorded comprehensively to maintain transparency.

Throughout the process, inter-rater agreement was evaluated, and any disagreements were addressed by consensus to preserve methodological rigor. Additionally, reference lists of included articles were hand-searched to identify further pertinent studies.

The entire study selection workflow was documented and is presented as a PRISMA flow diagram, detailing the number of articles identified, screened, excluded (with reasons), and ultimately included in the review. This approach follows established systematic review standards to optimize transparency, reproducibility, and comprehensiveness.

2.2.1. Inclusion Criteria

Only original research articles were included to allow for an unbiased evaluation of information based on a standardised criterion, facilitating the identification of high-quality studies. Articles had to be published in reputable journals indexed in PubMed or Scopus, as this was considered a measure of study credibility. Only articles written in English with full-text availability were included. The publication date range was limited to 2013–2025 to capture recent evidence on the growing interest in salsolinol toxicity. Preliminary searches indicated that some earlier literature highlighted the protective effects of salsolinol; therefore, a key inclusion criterion was that studies must focus specifically on its toxic effects.

2.2.2. Exclusion Criteria

Grey literature was excluded due to concerns over credibility and peer review standards. Unpublished studies were also omitted for similar reasons. Articles not written in English or those without full-text access were excluded.

Studies published prior to 2013 were not considered in order to focus on the most current findings and to meet time constraints. Review articles were excluded to avoid the influence of selective reporting and to ensure direct evaluation of primary data. Finally, studies that focused on the therapeutic or protective potential of salsolinol, rather than its toxic effects, were excluded from this review.

2.2.3. Critical Appraisal Step

The final step in the study selection process was the critical appraisal of shortlisted articles following the initial screening. The purpose of this appraisal was to assess the quality of evidence by systematically evaluating the validity, potential biases, and justification of conclusions in each study. Articles that failed to meet the requirements for more than four questions on the appraisal checklist [7] were excluded. This applied to responses marked as "No" or "Unclear," while responses marked as "Not applicable" were not counted. The following are the questions in the appraisal checklist.

i. Is there congruity between the stated philosophical perspective and the research methodology?
ii. Is there congruity between the research methodology and the research question or objectives?
iii. Is there congruity between the research methodology and the methods used to collect data?
iv. Is there congruity between the research methodology and the representation and analysis of data?
v. Is there congruity between the research methodology and the interpretation of results?
vi. Is there a statement locating the researcher culturally or theoretically?
vii. Is the influence of the researcher on the research, and vice versa, addressed?
viii. Are participant, and their voices, adequately represented?
ix. Is the research ethical according to current criteria or, for recent studies, and is there evidence of ethical approval by an appropriate body?
x. Do the conclusions drawn in the research report flow from the analysis or interpretation of the data?

This step served as the final filter in the selection process. In total, 8 out of 8 articles passed the critical appraisal and were included in the review.

3. Results

The detailed steps of the systematic review and their outcomes are presented hierarchically according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flowchart in Figure 2. The final list of included articles is provided in Table 1.

4. Discussion

The toxicity of salsolinol was initially hypothesized to be due to its structural resemblance to the synthetic heroin analog contaminant, MPTP, a potent neurotoxin known to impair mitochondrial function by inhibiting mitochondrial proteins in the electron transport chain [8]. Subsequent *in vitro* and *in vivo* studies led to the identification of salsolinol metabolites and their detrimental effects on various physiological systems, ultimately contributing to the onset of neurological disorders, most notably Parkinson's disease. The results of this systematic review provided insights into research examining specific salsolinol metabolites with structural features that enhance toxicity, as well as the mechanisms underlying their harmful effects. The evidence consolidated in this review highlights the neurotoxic effects of salsolinol, particularly in relation to neuronal degeneration and addictive behaviours [8-14]. Additionally, its alcohol-mediated involvement in breast cancer pathogenesis and its role in myenteric neuronal degradation, affecting gastrointestinal function, have been reported [9].

Discussions surrounding the neurotoxic effects of salsolinol frequently focus on its addictive properties, particularly in the context of alcoholism [9-12, 14]. Salsolinol is formed through the condensation of acetaldehyde, a primary metabolite of ethanol, with dopamine. While earlier research attributed alcohol's addictive effects to ethanol and acetaldehyde, recent studies have highlighted the significantly greater addictive potential of salsolinol. The dose–response relationship of salsolinol exhibits a U-shaped curve, with minimal effects observed at very low (e.g., 0.01 μM) and very high (e.g.,≥0.3 μM) doses, and maximal behavioural effects occurring at intermediate doses. These include addiction-related behaviours such as intracranial self-administration of salsolinol (observed using the intracranial self-administration paradigm), conditioned place preference, and increased locomotor activity in mice (likely driven by the activation of the brain's reward pathway), specifically the ventral tegmental area. Notably, intraperitoneal administration of salsolinol resulted in elevated levels in the neostriatum of *in vivo* models, suggesting its ability to cross the blood-brain barrier. Once in the striatum, salsolinol binds to opioid receptors in the ventral tegmental area, enhancing dopamine release and triggering cravings - a behavioural pattern consistent with psychostimulant addiction.

Another key mechanism of salsolinol toxicity is its ability to induce oxidative stress via mitochondrial dysfunction [8, 9]. In one study, salsolinol was incubated with cytochrome C, a mitochondrial protein, and a time-dependent release of iron ions was observed. This process involved oxidative damage to cytochrome C, leading to amino acid loss and protein aggregation. The altered protein structure could no longer effectively bind iron ions, promoting conditions conducive to the Fenton reaction. This reaction,

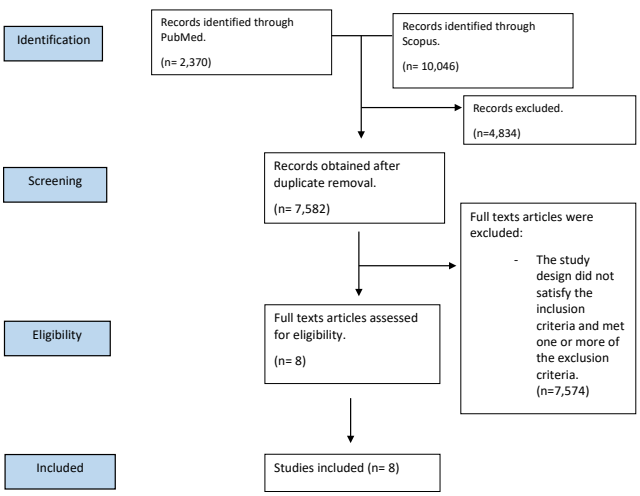

Fig. 2. PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flowchart of the study selection process.

Table 1. Summary of Key Findings from Selected Original Research Articles on the Toxicity of Salsolinol and Its Metabolites.

No	Articles	Main findings	Reference
1.	Salsolinol, a catechol neurotoxin, induces oxidative modification of cytochrome c	Toxicity is associated with the ability of salsolinol to induce oxidative stress through mitochondrial dysfunction, which leads to the denaturation of the mitochondrial protein cytochrome C.	[8]
2.	Oxidative DNA Damage and Mammary Cell Proliferation by Alcohol-Derived Salsolinol	Alcohol intake increases systemic salsolinol levels, contributing to oxidative stress that can damage DNA and exert carcinogenic effects.	[9]
3.	(R)-Salsolinol, a product of ethanol metabolism, stereospecifically induces behavioural sensitization and leads to excessive alcohol intake.	Repeated administration of R-salsolinol has been associated with locomotor sensitization, conditioned place preference, and an increase in binge-like alcohol intake.	[10]
4.	Microinjections of acetaldehyde or salsolinol into the posterior ventral tegmental area increase dopamine release in the nucleus accumbens shell.	When salsolinol and acetaldehyde were administered at intermediate concentrations into the posterior ventral tegmental area, stimulation of dopamine neurons was observed. However, at higher concentrations, secondary effects led to the inhibition of dopamine neuronal activity.	[11]
5.	Salsolinol, free of isosalsolinol, exerts ethanol-like motivational/sensitization effects leading to increases in ethanol intake	Repeated administration of salsolinol to rats resulted in increased locomotor activity and voluntary ethanol consumption.	[12]
6.	The effect of peripheral chronic salsolinol administration on fat pad adipocytes morphological parameters	Systemic administration of salsolinol was also associated with a reduction in adipocyte mass.	[13]
7.	Key role of salsolinol in ethanol actions on dopamine neuronal activity of the posterior ventral tegmental area	When administered directly into the posterior ventral tegmental area, salsolinol excites dopamine neurons, increases dopamine transmission in the nucleus accumbens, and sustains its own self-administration. In contrast, dopamine and acetaldehyde alone do not exhibit such efficacy.	[14]
8.	Biological response and cell death signaling pathways modulated by tetrahydroisoquinoline-based aldoximes in human cells	Tetrahydroisoquinoline-based aldoximes showed time- and cell-dependent cytotoxicity, inducing mitochondrial dysfunction, caspase-dependent apoptosis, oxidative stress, and DNA damage.	[15]

Note: DNA- Deoxyribonucleic acid.

involving the interaction of free iron ions with hydrogen peroxide, generates hydroxyl radicals, thereby increasing oxidative stress. The selective accumulation of these ions in specific brain regions is implicated in the development of neurodegenerative disorders such as Parkinson's and Alzheimer's disease. While this mechanism underpins the fundamental basis of neuronal toxicity, other metabolites of salsolinol, such as N-methyl-(R)-salsolinol and DMD-

HIQ+, also contribute to similar pathological outcomes.

As previously mentioned, the toxicity of salsolinol is largely attributed to its capacity to induce oxidative stress via mitochondrial impairment [8,9]. Oxidative stress is also known to cause deoxyribonucleic acid (DNA) mutations and is therefore associated with cancer development. Alcohol consumption, which increases systemic levels of salsolinol, may elevate oxidative stress and, consequently, the risk of developing cancer - particularly breast cancer. Breast cancer carcinogenesis may be explored through the lens of oxidative damage, particularly in the presence of transition metals such as iron, copper, and magnesium, which are already prevalent in breast tissue. Salsolinol, produced from the condensation of acetaldehyde and dopamine, can undergo oxidation in the presence of Mn, Cu(II), and Fe(III) ions to form semiquinone radicals while reducing the metal ions to Mn(I), Cu(I), and Fe(II). These radicals can subsequently oxidize to form benzoquinone derivatives and superoxide radicals ($O_2^{\bullet-}$), which dismutate into hydrogen peroxide. The reduced metal ions then react with hydrogen peroxide to generate reactive oxygen species, leading to DNA damage, as confirmed in the estrogen-sensitive MCF-7 breast cancer cell line. In the MCF-10A cell line, reactive oxygen species were found to activate the early growth response pathway, namely estrogen receptor α-estrogen response element (ERα-ERE) and reactive oxygen species (ROS)-mediated epidermal growth factor receptor (EGFR) activation, in epithelial cells. Meanwhile, in the MCF-7 cell line, the proliferation of cells exposed to salsolinol was inhibited by tamoxifen (an estrogen receptor antagonist), suggesting that salsolinol binds to estrogen receptors. Therefore, alcohol intake may increase the risk of estrogen-receptor-positive breast cancers.

Beyond its effects on the central nervous system's dopaminergic neurons, salsolinol has also been linked to damage and reduced functionality of the myenteric neurons. Salsolinol reduces adipocyte size, which reflects alterations in adipocyte physiology. Experimental evidence suggests that salsolinol may induce weight loss either by enhancing satiety signalling or through alterations in gut function. The satiety hypothesis is supported by salsolinol's inhibition of dopamine reuptake and degradation, which increases dopamine levels and may mimic satiety signals, thus reducing food intake. However, this was contested by a study comparing food intake and body weight in two groups of mice: one administered salsolinol intraperitoneally via osmotic mini-pumps, and the other receiving physiological saline. Both groups had comparable food consumption throughout the experiment. Despite this, a decrease in body weight was observed in the salsolinol-treated group. Histological analysis using Hematoxylin and Eosin staining of fat tissue revealed reduced adipocyte size based on measurements such as cell area, perimeter, and axis lengths. These findings suggest that the weight loss may be due to peripheral effects (physiological actions outside the central nervous system) rather than changes in appetite or food intake [13].

5. Conclusion

This systematic review demonstrates that salsolinol and its metabolites exert notable toxic effects on both central and peripheral nervous systems, primarily through mechanisms involving oxidative stress and the modulation of addiction-related dopaminergic pathways. Elevated salsolinol levels—either arising endogenously in dopamine-rich brain regions or systemically following alcohol consumption—have been consistently associated with neuronal degeneration, increased addictive behaviors, and potential carcinogenic effects due to oxidative DNA damage. The literature also underscores salsolinol's role in mediating neurochemical changes that underlie alcohol addiction and implicates it in the degeneration of myenteric neurons, which may impact gastrointestinal health. Yet, the precise physiological versus toxicological role of salsolinol remains to be fully clarified, as conflicting reports exist regarding possible dose-dependent neuroprotective effects. Overall, these findings highlight the importance of further targeted research to better elucidate the pathological pathways involved and to explore therapeutic interventions that may mitigate the detrimental impacts of salsolinol and its metabolites.

Abbreviation

DNA: Deoxyribonucleic acid; DMDHIQ+: 1,2-dimethyl-6,7-dihydroxyisoquinolinium ions

Registration and protocol

The review was not registered.

Conflict of interest

The authors declare no conflict of interest.

Consent for publications

All authors have read and approved the final manuscript and consent to its publication.

Ethics approval and consent to participate

Not applicable. This study did not involve human participants, animal subjects, or identifiable personal data, and therefore did not require ethical approval or consent to participate.

Informed Consent

Not applicable. This study did not involve human participants or identifiable personal data.

Availability of data and material

The datasets analyzed during the current study are derived from previously published studies available in PubMed and Scopus. Full references are provided in the manuscript.

Authors' contributions

JKS performed the methodology, formal analysis, investigation, data curation, and original draft preparation. Conceptualization was led by RYK, who also supported methodology development along with BYC and KGLV. Review and editing were conducted by HYW, SS, APKL, SMC, and RYK. Visualization was provided by HYW. Validation and supervision were carried out by BYC, KGLV, and RYK. Additionally, RYK managed formal analysis, resources, project administration, and funding acquisition.

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References

1. Kurnik-Łucka M, Panula P, Bugajski A, Gil K (2018) Salsolinol: an unintelligible and double-faced molecule-Lessons learned from in vivo and in vitro experiments. Neurotox Res 33: 485–514. doi:10.1007/s12640-017-9818-6
2. Kurnik-Łucka M, Latacz G, Martyniak A, Bugajski A, Kieć-Kononowicz K, Gil K (2020) Salsolinol-neurotoxic or neuroprotective? Neurotox Res 37: 286–297. doi:10.1007/s12640-019-00118-7
3. Możdżeń E, Kajta M, Wąsik A, Lenda T, Antkiewicz-Michaluk L (2015) Salsolinol, an endogenous compound triggers a two-phase opposing action in the central nervous system. Neurotox Res 27: 300–313. doi:10.1007/s12640-014-9511-y
4. Naoi M, Maruyama W, Akao Y, Yi H (2002) Dopamine-derived endogenous N-methyl-(R)-salsolinol: its role in Parkinson's disease. Neurotoxicol Teratol 24: 579–591. doi:10.1016/s0892-0362(02)00211-8
5. Wąsik A, Romańska I, Michaluk J, Antkiewicz-Michaluk L (2015) Chronic salsolinol administration prevents the behavioral and neurochemical effects of L-DOPA in rats. Neurotox Res 27: 399–410. doi:10.1007/s12640-015-9523-2
6. Kotake Y, Sekiya Y, Okuda K, Ohta S (2014) Detection of a novel neurotoxic metabolite of Parkinson's disease-related neurotoxin, 1-benzyl-1,2,3,4- tetrahydroisoquinoline. J Toxicol Sci 39: 749–754. doi:10.2131/jts.39.749
7. Lockwood C, Munn Z, Porritt K (2015) Qualitative research synthesis: methodological guidance for systematic reviewers utilizing meta-aggregation. Int J Evid Based Healthc 13: 179–187. doi:10.1097/XEB.0000000000000062
8. Kang JH (2013) Salsolinol, a catechol neurotoxin, induces oxidative modification of cytochrome c. BMB Rep. 46: 119–123. doi:10.5483/bmbrep.2013.46.2.220
9. Murata M, Midorikawa K, Kawanishi S (2013) Oxidative DNA damage and mammary cell proliferation by alcohol-derived salsolinol. Chem Res Toxicol 26: 1455–1463. doi:10.1021/tx400182n
10. Quintanilla ME, Rivera-Meza M, Berríos-Cárcamo P, Cassels BK, Herrera-Marschitz M, Israel Y (2016) (R)-Salsolinol, a product of ethanol metabolism, stereospecifically induces behavioral sensitization and leads to excessive alcohol intake. Addict Biol 21: 1063–1071. doi:10.1111/adb.12268
11. Deehan GA, Jr Engleman EA, Ding ZM, McBride WJ, Rodd ZA (2013) Microinjections of acetaldehyde or salsolinol into the posterior ventral tegmental area increase dopamine release in the nucleus accumbens shell. Alcohol Clin Exp Res 37: 722–729. doi:10.1111/acer.12034
12. Quintanilla ME, Rivera-Meza M, Berrios-Cárcamo PA, Bustamante D, Buscaglia M, Morales P, Karahanian E, Herrera-Marschitz M, Israel Y (2014) Salsolinol, free of isosalsolinol, exerts ethanol-like motivational/sensitization effects leading to increases in ethanol intake. Alcohol 48: 551–559. doi:10.1016/j.alcohol.2014.07.003
13. Aleksandrovych V, Kurnik M, Białas M, Bugajski A, Thor P, Gil K (2016) The effect of peripheral chronic salsolinol administration on fat pad adipocytes morphological parameters. Folia Med Cracov 56: 81–95.
14. Melis M, Carboni E, Caboni P, Acquas E (2015) Key role of salsolinol in ethanol actions on dopamine neuronal activity of the posterior ventral tegmental area. Addict Biol 20: 182–193. doi:10.1111/adb.12097
15. Zandona A, Madunić J, Miš K, Maraković N, Dubois-Geoffroy P, Cavaco M, Mišetić P, Padovan J, Castanho M, Jean L, Renard PY, Pirkmajer S, Neves V, Katalinić M (2023) Biological response and cell death signaling pathways modulated by tetrahydroisoquinoline-based aldoximes in human cells. Toxicol 494. doi: 10.1016/j.tox.2023.153588