

Potential Human-Health Implications of Ractopamine and Methionine in Poultry Products: An Expanded Substance-Specific Narrative Review

Noor A Abdulrazzaq [1], Mustafa F Hameed [2*]
[1-2] Directorate General of Education of Basrah, Ministry of Education, Basrah, Iraq

* Corresponding Author: **Mustafa F Hameed**

Article Info

P-ISSN: 3051-3405
E-ISSN: 3051-3413
Volume: 02
Issue: 02
Received: 06-08-2026
Accepted: 04-09-2026
Published: 02-10-2026
Page No: 36-42

Abstract
Background: Ractopamine and methionine are sometimes classified as a single umbrella term "growth promoters" but the two are quite different in chemical class, biological activity.
Objective: To discuss, in this narrative review (which is expanded to include additional relevant extra information), the possible implications of ractopamine and methionine in poultry products in terms of human health.
Methodology: Narrative synthesis of peer-reviewed studies and toxicological reviews and regulatory assessments. The evidence was categorized as regulatory, human experimental, human observational, animal experimental, mechanistic, or nutritional.
Results: Species-specific residue depletion, tissue concentration, dietary exposure and pharmacodynamic endpoints are required for Ractopamine. Differences in data interpretation and risk-management approaches by national/international authorities have led to different regulatory conclusions. Methionine is not a long lasting veterinary drug residue. It is metabolically relevant based on the total food intake, folate and vitamin B status, renal function, genetic factors, and one-carbon metabolism. Experimental methionine loading has been shown to increase plasma concentrations of homocysteine, but this alone does not prove that habitual poultry consumption is responsible for cardiovascular disease. Evidence for association of either agent with gynecomastia, precocious puberty, infertility, cancer or chronic metabolic disease is not yet sufficient.
Conclusion: Residue monitoring for ractopamine in poultry should be continued and monitoring of methionine should be viewed in the broad context of overall nutrient and metabolic considerations. There is no evidence yet that ordinary consumption of compliant poultry products is linked to major chronic, developmental, reproductive or cardiovascular diseases, unless it is measured and clinically verified.

DOI: https://doi.org/10.54660/IJABRN.2026.2.2.36-42

Keywords: Ractopamine, Methionine, Poultry Products, Homocysteine, Nutritional Metabolism, Food Safety

1. Introduction

The advancement of practices in poultry production has been achieved through the genetic selection, improved housing conditions, vaccination, biosecurity, precision nutrition, as well as select feed additives and veterinary medicines. Although frequently grouped into a single category in public discussion, these do not represent a single toxicological category. Different biological endpoints specific to different antimicrobial agents, beta adrenergic agonists, amino acids, hormones, and environmental contaminants exist, as do their sample purposes and pathways of exposure [1]. Ractopamine is a synthetic phenethanolamine which has been applied to some food animal systems to alter nutrient partitioning and enhance lean tissue deposition. This use has brought with it scientific, legal and trade-related disputes as it is a pharmacological activity that must be evaluated as a veterinary drug and not as an ordinary feed nutrient [2]. Methionine, however, is an essential amino acid that

is needed for protein biosynthesis and the transfer of methyl groups. It has been added to poultry diets regularly where a balanced amino-acid profile is not provided through the diet. This review is focused on the differences between the two substances.

The assessment of Ractopamine is based on residue depletion, tissue distribution, dose, pharmacodynamic response and maximum residue limits. The evaluation of methionine includes total dietary intake, amino-acid balance, vitamin status, renal function, and one-carbon metabolism. Treat methionine as adding a drug residue and concerns may be overstated, yet treat ractopamine as a regular nutrient and pharmacological risks may be underestimated.

2. Review Scope and Methodology

The current article is a structured narrative review, and not a systematic review or meta-analysis. The literature that is peer-reviewed and pertinent regulatory documents were studied regarding ractopamine pharmacology, veterinary-drug residues, species-specific applicability, cardiovascular endpoints, methionine nutrition, homocysteine metabolism, endocrine interpretation, and human dietary exposure of ractopamine. All evidence was grouped into the following categories: regulatory evidence, human experimental evidence, human observational evidence, animal experimental evidence, mechanistic or in-vitro evidence and nutritional evidence. Greater weight was assigned to evidence that had measured exposures, clinically valid outcomes, appropriate controls, plausible temporality, species-specific evidence and realistic dose-response relationship. The steps in the chain of causation applied in the interpretation are the use (in the animal), the concentration in edible tissue, human dietary intake, internal dose, biological endpoint and clinically verified outcome. A receptor effect can indicate disease occurred in consumers, and a residue detection can serve as a surveillance finding, but neither can be used singly to indicate disease in consumers. Evidence is heterogeneous therefore a pooled estimate is not calculated. This is done intentionally, as the sources included vary in several aspects such as animal species, route of exposure, magnitude of exposure, tissue studied, comparator, definition of outcome, and regulatory context. Being able to merge all of these studies into a statistical model would achieve a degree of numerical precision that the actual evidence in each of the studies does not justify.

3. Chemical and Biological Classification

Ractopamine hydrochloride (Fig. 1) is a synthetic phenethanolamine and beta-adrenergic agonist. At appropriate levels of exposure, its activity can modify energy metabolism, heart rate and vascular tone, lipolysis and protein deposition. Methionine (Fig. 1) is another sulfur containing amino acid, containing a thioether unit and playing a central role in protein synthesis, methylation, transsulfuration, and metabolism in relation to antioxidants. The two substances have thus distinct assessment considerations. For ractopamine the question is, can a consumer receive a pharmacologically relevant dose from a particular animal species and tissue? In the case of methionine, the critical point is whether methionine level of total diet influences nutritional condition or metabolic disease secondarily in a clinically meaningful manner.

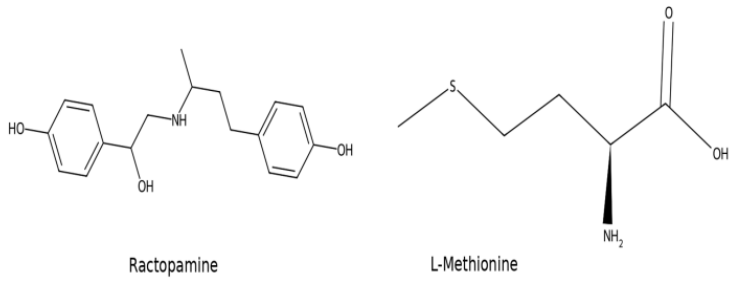


Fig 1: Chemical structures of ractopamine and methionine

4. Ractopamine Use and Poultry-Specific Applicability

The use of the ractopamine is largely tested in varieties of industrially reared food producing animals such as pig and cattle, and its status of regulation differs across jurisdictions. According to EFSA, ractopamine falls in the group of beta-agonists which are not allowed in the EU for use in food-producing animals under the specific legal framework [3]. The use of ractopamine is forbidden outside the EU. The regulatory record is not a record of the presence of the substance in ALL poultry products or of all exposures causing disease. Specific mention that evidence of ractopamine is applicable or not must be given for each: chicken, turkey, pig and cattle. Parameters such as absorption, metabolism, tissue distribution, depletion kinetics, approved dose, withdrawal period and maximum residue limits are species and jurisdiction specific. The JECFA assessment resulted in ADI 0–1 µg/kg bw/day, and acute cardiovascular effects in humans were considered as the critical endpoint [4]. The evaluation also provided residue values of cattle and pigs, an extrapolation of which to chicken and turkey tissues is not necessarily valid. Weaknesses were subsequently highlighted in the supporting data for the JECFA assessment and EFSA did not agree to base the European maximum residue level on this assessment [5]. This disagreement is a reflection of both the weaknesses in the quality and management of data and a division not between "safe" and "unsafe" science, but of that represented by the disagreement.

5. Ractopamine Pharmacology

Ractopamine affects the beta-adrenergic and related receptor systems. Experimental receptor studies have confirmed its potential to interact with trace amine-associated receptor 1 and other pathways of interest for adrenergic signalling [6]. The mechanisms of action by which ractopamine promotes lipolysis also have been studied with reference to the beta-adrenergic receptor (BAR) subtypes [7]. These conclusions provide pharmacological evidence of plausibility but not clinical efficacy of the typical food intake. In animal research, ractopamine has demonstrated an ability to affect protein and

lipid deposition to a greater degree, particularly during the finishing stages when controlled amounts of ractopamine are administered [8]. These studies are helpful for the understanding of production effects, tissue biology and dose-response relationships. They should not be directly stated to imply that when consumer consumes poultry, consumers will develop cardiovascular, metabolic, reproductive or developmental disease. Factors involved in human exposure involve concentrations in edible tissues, portion size, body weight, intake rates, bioavailability, adherence to withdrawal periods and individual susceptibility. A risk estimate will therefore have to relate measured tissue concentration to dietary intake and internal dose. The presence of a residue alone does not imply clinical toxicity and a concentration below a legal limit does not imply that all biological effect is ruled out.

6. Residue Monitoring and Dietary Exposure

Assessment of veterinary-drug residue assessment necessitates validation and analytical procedures, representative sampling, traceability, and species-specific depletion data. Maximum residue limits for ractopamine in various tissues and species are documented in the Codex [9] and are substance-, tissue-, and animal-specific, instead of universal values. The difference between residue compliance and clinical toxicity are different but related. A concentration below an MRL is considered compliant with the MRL, under the exposure assumptions used for the MRL. The presence of a concentration above the limit does not mean non-compliance, and although indicative of clinical poisoning, the concentration must still be used to investigate. Likewise, the lack of a residue in a small sample cannot be used to infer the lack of residue everywhere. Particular dietary-exposure calculation takes body weight, portion size and frequency of consumption, concentration in the tissue of interest and proportion of diet from the food item into account. The fact that a child receives a higher dose per kg of body then adult from the same dose of medicine does not mean that the child is more susceptible or HARMED.

7. Regulatory Risk Assessment

This makes it possible for different regulatory decisions to be made, both on the assumptions about human exposure and on the uncertainty factors, pivotal endpoints, legal thresholds and the application of the precautionary principle. The European legislation applicable to the case bans the use of beta-agonists in food producing animals including ractopamine [10]. Solutions that do not reflect fully or ideally developed residue data shall include a description of uncertainty. In regulatory toxicology guidance, uncertainty of the data arising from different factors such as data quality, identity of residues, tissue distribution, reliability of analytical methods and level of ingested residues have been highlighted as key to informing the risk conclusion [11]. Reviews of antibiotic residues in poultry tissues also point out that the notions of being a residue and being responsible for human risk are not synonymous [12]. The review should include a description of the substance, treated species, tissue, measured concentration, withdrawal period, regulatory standard and endpoint for the comparison to be defensible between jurisdictions. Different regulatory opinion should not be interpreted as a clinical statement suggesting that poultry would lead to disease.

8. Cardiovascular Effects of Ractopamine

The only definitive pharmacodynamic issue identified in humans in ractopamine assessment is cardiovascular response. JECFA evaluation employed alterations of the heart rate, cardiac output and associated cardiovascular parameters as endpoints [13]. These are effects with a biological plausibility, and it is based on the activation of beta-adrenergic receptors. There is no evidence that regular consumption of compliant poultry leads to chronic hypertension, arrhythmia, heart failure or cardiovascular disease. A harmful stochastic outcome can be identified at high-level exposure in experimental pharmacology and/or controlled dosing and these results cannot replace measured exposure in a consumer population and observed outcomes. Importantly, the majority of data available on the residue issues surrounding ractopamine are from controlled animal studies or from observations from humans pharmacologically exposed to ractopamine, as opposed to from prospective cohorts of consumers of poultry products. The conclusion should therefore be limited to pharmacological plausibility and/or exposure related risk and not to findings of proven disease causation. Established causes of palpitations, tachycardia, or blood-pressure abnormalities (e.g., cardiovascular disease, thyroid disease, stimulant use, medications, anxiety, anemia, electrolyte disorders) should be considered during any clinical assessment of palpitations, tachycardia, or blood-pressure abnormality. However, such results cannot be attributed to ractopamine solely on the basis of the amount of poultry consumed.

9. Metabolic and Endocrine Interpretation of Ractopamine

Beta-adrenergic signalling may affect glucose and lipid metabolism, energy output, vascular tone and adipose tissue metabolism. They do not prove that insulin resistance, metabolic syndrome, type 2 diabetes or obesity is the result of the consumption of ordinary chicken, but are effects that suggest a mechanism for the investigation of metabolic endpoints. It should also be noted that evidence is lacking to describe ractopamine as an estrogenic compound. There is no regulatory or pharmacological available literature in terms of beta-adrenergic activity or cardiovascular endpoints that document this mechanism of excess estrogens. Therefore, it is not known if exposure to ractopamine from poultry products leads to gynecomastia clinically. The association would have to be confirmed, with evidence of a documented alteration in the androgen-to-estrogen ratio, common causes excluded, and clinically-proven associations. If food-producing animals are exposed to hormones, this poses an additional hazard identification and exposure assessment problem. Review of application of hormones recognize and differentiate naturally occurring hormones from synthetic agents, and again stress that hazard identification is not by itself an indication of risk for the diet [14]. Regulatory evaluations also emphasize the need to know the identity, concentration, absorption, duration, and cancer/endocrine effects of the residues before a claim of cancer/endocrine activity can be made [15].

10. Developmental and Reproductive Claims Related to Ractopamine

Caution is needed with developmental and reproductive claims as the data in production studies are usually wrongly

extrapolated to human health outcomes. Because the findings from a study in an animal demonstrating that growth, body composition, tissue response has been altered is not proof of producing infertility, precocious puberty or impaired neurodevelopment, or impaired sperm quality in human. There is no literature available that resulted in the conclusion that there is no evidence of precocious puberty occurring with ordinary consumption of compliant poultry products. The conclusions of all reviews on EDCs are varied and highlight the roles of adiposity, genetics, developmental timing and combined exposures to different environmental factors [16]. That is, these general endocrine effects are not necessarily ractopamine, in the absence of compound-specific measurements. Similar findings reported for systematic reviews of EDCs in relation to male reproductive function, for which there are inconsistent findings and high degrees of confounding [17]. This observation does not provide evidence to prove ractopamine does not make animals infertile. Rather, age, obesity, medications, work history, and hormonal tests or sperm testing should be used when evaluating the situation.

11. Methionine in Poultry Nutrition

Methionine, an amino acid which is essential for normal protein synthesis. As per nutritional reviews, methionine requirements are age, feed type, health and bio-availability dependent [18]. In a poultry operation, an amino-acid deficiency can be remedied through the addition of supplemental DL-methionine to help keep protein deposition under control. The reason for this is that the presence of methionine in poultry meat cannot be viewed as the presence of a pharmacologically active veterinary drug. Methionine is both incorporated into normal proteins or metabolized via physiological routes. For the relevant exposure variable, it is not a continuously present residue concentration specifically caused by poultry intake, but rather the total dietary intake from all foods. The interconversion of methionine, homocysteine, cystathionine and cysteine is part of sulfur-amino-acid metabolism. These pathways are responsible for the transfer of methyl groups, redox balance and the biosynthesis of sulfur-containing metabolites [19]. The nutrition role of Methionine and Cysteine has been outlined in detail in human and animal nutrition [20].

12. Homocysteine Metabolism

The methionine passed into the cell is then converted to S-adenosylmethionine, which is the principal donor of cellular methyl groups (Fig. 2). Demethylation results in the generation of S-adenosylhomocysteine and then homocysteine. Homocysteine can be remethylated to methionine or become part of the transsulfuration pathway to generate cystathionine and cysteine. Folate, B12, B6 metabolism, renal function, genetics, medications, age and nutrition are all important influences. Vitamin B12 deficiency is a known cause of deranged homocysteine metabolism, and should be considered when clinically appropriate [21]. Folate and vitamin B12 are involved in interrelated pathways related to regulation of homocysteine clearance [22]. The physiology of folate and vitamin B12 suggests that plasma homocysteine concentration is only partially dependent on methionine [23]. In the literature, high levels of homocysteine have been correlated to vascular disease, but correlation does not establish causation in the case of any given food or amino acid [24].

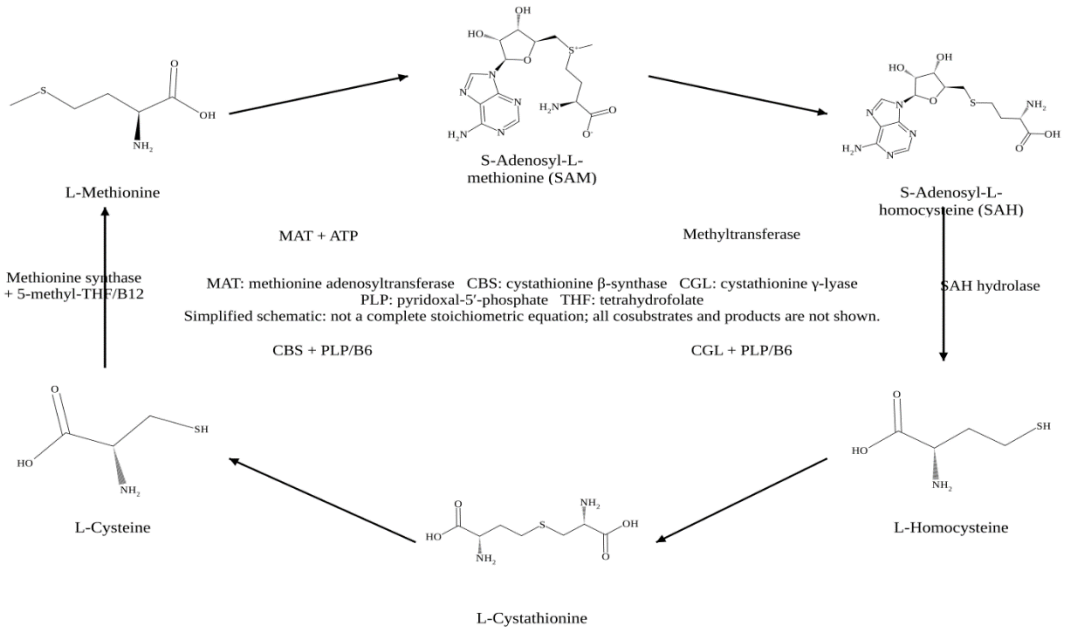


Fig 2: Methionine–homocysteine metabolic pathway

13. Methionine Loading and Human Cardiovascular Interpretation

The plasma homocysteine may be acutely elevated by large experimental oral methionine challenges especially in patients who have reduced clearance or a deficiency in vitamins [25]. These loading studies are a pharmacological or experimental challenge and not a model of the methionine dose that can be consumed from a typical serving of chicken. Homocysteine has been studied as a potential vascular risk factor, biomarker or mediator. Reviews have focused on the complexity of the relationship and the fact that a decrease in a biomarker does not necessarily mean that the disease process has been abated. Randomised evidence of folic-acid therapy has demonstrated that reduction of homocysteine

levels is not always associated with reduced cardiovascular events [26]. Although meta-analytic data show beneficial effects of folic acid on reducing blood homocysteine levels, biochemical improvement does not necessarily translate to clinical benefit [27]. Accordingly, it is not justified to describe regular chicken consumption a demonstrated cause for hyperhomocysteinemia and cardiovascular disease. Clinical interpretation should reflect total diet, renal function and known risk factors and vitamin status. The methionine loading study and the circulating homocysteine study provide different information from the usual poultry intake studies and cannot be used interchangeably. An experimental biochemical response cannot be considered to be a clinically important response to a normal food.

14. Methionine Toxicity and Safety Limits

The toxicity of methionine is **p**rimarily dependent on dose and context. The experimental conditions of use of feeds are different from the normal nutritional setting and different from the toxicity of a species to a particular dose of substance(s). However, the possibility of toxicity becomes more credible at high, atypical concentrations or in the presence of a nutritional imbalance, or when clearance is reduced [28]. Inherited abnormalities of sulfur-amino-acid metabolism may affect the metabolism of methionine and homocysteine but should not be interpreted to imply that a normal amount of poultry is detrimental for the general population [29]. Therefore, the clinical evaluation should be centered around the patient's history, diet, renal function tests, vitamin, medication use and laboratory results. That methionine-containing foods should be avoided is not a scientifically tenable public-health stance. Instead, consider a diet adequate for needs, correct deficiencies that are documented and consider the high levels of supplements in a clinical context.

15. Ractopamine and Methionine in Children and Adolescents

If dose is expressed in units of body weight, children and adolescents might need age-specific exposure assessment, depending on the stage of their development differential physiology. Age-specific vulnerability, however, is not indicative of toxicity to the substance associated with age. Before crediting any claim that takes issue with the idea of ractopamine increasing a bird’s heart rate and causing a child's endocrine system to be disrupted and developmental diseases, controlled exposures and proven outcomes are needed. Requirements for methionine are also different for different life-stages. Assessment of nutrition should include focus on total protein consumption, growth, renal function, folate, vitamin B12, vitamin B6 and the overall nutritional pattern. Dietary restriction cannot be recommended based on poultry intake alone and homocysteine level testing is for a child who would have indications for such testing. There is no evidence that normals consumption of compliant poultry products is associated with gynecomastia, precocious puberty, neurologic or developmental defects, or reproductive diseases in children. There are established and potential determinants for these outcomes and causal attribution needs more than dietary history.

16. Adults, Renal Function, and Metabolic Vulnerability

Ractopamine assessment in adults is directed towards pharmacological exposure/cardiovascular response only. If a person is exposed and a history of cardiovascular disease or the use of interacting stimulants exists, then special clinical consideration may be warranted, but an ordinary dose is not a meaningful one from the point of view of consumption of ordinary poultry. The nutritional status and renal function have a strong influence on methionine interpretation in adults. Factors that can affect homocysteine levels include impaired clearance, lack of folate, lack of vitamin B12, lack of vitamin B6, and possibly some genetic disorders. These factors should be evaluated separately from the assumptions pertaining to the poultry. A sound clinical practice will therefore not overlook a history of adequate exposure or a history of failure to comply, but neither will it consider asymptomatic, or chronic disease, to be a result of ractopamine and methionine given no objective evidence to support the reviewers' conclusions.

17. Research Priorities

When further studies are conducted with ractopamine, they should be designed to produce representative poultry species and tissue residue information for the various production systems, doses, and withdrawal times for the poultry. Dietary exposure models should be reported on a body-weight basis, in number of serving size(s) consumed, frequency of consumption, and as a range of uncertainty. Clinically meaningful cardiovascular endpoints should be used in human studies and differential effects of experimental loading and common food exposure should be outlined. Future methionine trials should measure total dietary intake of methionine, and study interactions with folate, vitamins B6 and B12, renal function, age, sex, variation in genetic polymorphisms and metabolic disease. Studies should indicate if methionine is part of the diet, added to the diet or an experiment. Both research programmes have to be able to refine source attribution, employ validated analytical approaches and differentiate a measured exposure from a clinically proven effect. Such priorities would minimize speculations and make future human-health recommendations more relevant.

18. Evidence Boundaries and Causal Attribution

Biochemical association is not synonymous with dietary causation and caution should be used in interpreting methionine/homocysteine. Considerations for the interpretation of observational associations have been highlighted in reviews of homocysteine and cognition [30]. Previous research on plasma homocysteine also gives an example to dispel the automatic assumption that a vascular association is evidence of the role of a specific food in disease [31]. The metabolism of methionine is also affected by enzymes and genes. Sulphur containing amino acid metabolism in mammals reveals that dietary intake will have different effects due to clearance, cofactors and disease status [32]. Another explanation for the inter-individuals difference in the level of homocysteine is the differences in the physiology of folate and vitamin B12 [33]. These sources favour a metabolic explanation for methionine over a drug-residue explanation. For any statements pertaining to endocrine or reproductive or developmental effects, evidence must be provided for the compound. Hazard/exposure assessment reviews on hormonal residues highlight the need to carry out these assessments separately for naturally occurring hormones and synthetic compounds [34]. The hazard identification is reinforced with a general toxicological

review for risk assessment for consumption, which shows that hazards are insufficient to define risks of dietary consumption [35]. Therefore, ractopamine and methionine should not be considered as being causative agents of gynecomastia, infertility, precocious puberty or cancer unless there is documented exposure history and clinical evidence for the development of these conditions.

Other nutrition reviews report on methionine and cysteine, and methyl group metabolism in health and disease [36]. All reviews of methionine toxicity differentiate normal dietary amounts with experimental methionine loading versus high and unusual supplemental exposures [37]. Practically speaking, in the clinical context, a reduction in a biomarker does not necessarily mean reduction of the disease itself [38]. These findings underscore the importance of distinguishing between biochemical plausibility and adverse health outcomes in consumers. The key uncertainty for ractopamine is species-specific residue data, assumptions on food use and a revised relationship between the observed cardiovascular effects observed in the experimental animals and realistic diet human exposure [39]. Comparatively little regulatory literature has been published on ractopamine, and it is mainly discussed in terms of beta adrenergic pharmacology or the evaluation of residues, but not in terms of estrogenic or antiandrogenic activity [40]. The residue and pharmacology is also recorded in a separate JECFA monograph.

19. Limitations

This is a narrative review and does not include any pooled effect estimate or quantitative risk of bias score. There are variations in evidence by species, dose, tissue, regulatory jurisdiction and definition of outcome. For ractopamine or methionine, few studies are available that have directly conducted human exposure assessments with poultry products. Based on some mechanistic and animal evidence, it can be said that some of the evidence is related to the context of the consumer. A full list of risks for all production systems and jurisdictions can also not be determined during the review. Regulatory information, feed practice, residue monitoring and withdrawal compliance varies. The review does not state that ractopamine is the same for all poultry products and that there is a similar residue profile or exposure. Nor does it state that methionine consumption has the same metabolic effects on everyone. While these limitations can facilitate conservative interpretation and clear communication, they are not sufficient reason to make a direct cause-and-effect association between chronic disease and regular consumption of poultry, as reported by consumers and adjudicated by the clinician, without quantitative and calculable exposure and noted clinical effects.

20. Conclusion

Ractopamine and methionine should not be in a single toxicological category. Ractopamine is a pharmacologically active beta-adrenergic veterinary drug with consumer risk variables relating to animal species, tissue residues, dietary residue, compliance with withdrawal periods and cardiovascular pharmacology. Methionine is an essential amino acid and its role is related to the overall diet, amino acid balance, one-carbon metabolism, vitamin levels, kidney function and susceptibility. There is no current evidence for chronic cardiovascular or cardiovascular-metabolic disease, developmental disease, reproductive disease, or endocrine disease resulting from ordinary consumption of poultry products meeting the standard. The most defensible approach is: species specific residue monitoring for ractopamine, nutrition interpretation based on measured methionine exposure, and ongoing research relying on measured exposure and clinically demonstrated results.

References

1. Gadde U, Kim WH, Oh ST, Lillehoj HS. Alternatives to antibiotics for maximizing growth performance and feed efficiency in poultry: a review. Anim Health Res Rev. 2017;18(1):26-45.
2. Abbas K, Raza A, Vasquez RD, *et al*. Ractopamine at the center of decades-long scientific and legal disputes: a lesson on benefits, safety issues, and conflicts. Biomolecules. 2022;12(10):1342.
3. European Food Safety Authority. EFSA evaluates safety of ractopamine in feed. Parma: European Food Safety Authority; 2009. Available from: https://www.efsa.europa.eu/en/news/efsa-evaluates-safety-ractopamine
4. Joint FAO/WHO Expert Committee on Food Additives. Ractopamine hydrochloride. WHO Food Additives Series 53. Geneva: World Health Organization; 2004. Available from: https://apps.who.int/food-additives-contaminants-jecfa-database/chemical.aspx?chemID=1064
5. European Food Safety Authority. Safety evaluation of ractopamine. EFSA J. 2009;7(4):1041.
6. Liu X, Grandy DK, Janowsky A. Ractopamine, a livestock feed additive, is a full agonist at trace amine-associated receptor 1. J Pharmacol Exp Ther. 2014;350(1):124-9.
7. Mills SE, Spurlock ME, Smith DJ. β-Adrenergic receptor subtypes that mediate ractopamine stimulation of lipolysis. J Anim Sci. 2003;81(3):662-8.
8. Dunshea FR, King RH, Campbell RG. Interrelationships between dietary protein and ractopamine on protein and lipid deposition in finishing gilts. J Anim Sci. 1993;71(11):2931-41.
9. Codex Alimentarius Commission. Ractopamine hydrochloride: maximum residue limits. Veterinary Drugs Database. Rome: FAO/WHO; 2012. Available from: https://www.fao.org/fao-who-codexalimentarius/codex-texts/dbs/vetdrugs/veterinary-drug-detail/en/?d_id=79
10. Council of the European Union. Council Directive 96/22/EC concerning the prohibition on the use in stockfarming of certain substances having a hormonal or thyrostatic action and of beta-agonists. Off J Eur Communities. 1996;L125:3-9. Available from: https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX:31996L0022
11. Chicoine A, Erdely H, Fattori V, *et al*. Assessment of veterinary drug residues in food: considerations when dealing with sub-optimal data. Regul Toxicol Pharmacol. 2020;118:104806.
12. Donoghue DJ. Antibiotic residues in poultry tissues and eggs: human health concerns? Poult Sci. 2003;82(4):618-21.
13. Joint FAO/WHO Expert Committee on Food Additives. Ractopamine (addendum). WHO Food Additives Series 53. Geneva: World Health Organization; 2004. Available from:

https://www.inchem.org/documents/jecfa/jecmono/v53je08.htm
14. Qaid MM, Abdoun KA. Safety and concerns of hormonal application in farm animal production: a review. J Appl Anim Res. 2022;50(1):426-39.
15. Jeong SH, Kang D, Lim MW, Kang CS, Sung HJ. Risk assessment of growth hormones and antimicrobial residues in meat. Toxicol Res. 2010;26(4):301-13.
16. Papadimitriou A, Papadimitriou DT. Endocrine-disrupting chemicals and early puberty in girls. Children (Basel). 2021;8(6):492.
17. Martínez MÁ, Marquès M, Salas-Huetos A, *et al*. Lack of association between endocrine disrupting chemicals and male fertility: a systematic review and meta-analysis. Environ Res. 2023;217:114942.
18. Elango R. Methionine nutrition and metabolism: insights from animal studies to inform human nutrition. J Nutr. 2020;150(Suppl 1):2518S-23S.
19. Stipanuk MH. Sulfur amino acid metabolism: pathways for production and removal of homocysteine and cysteine. Annu Rev Nutr. 2004;24:539-77.
20. Brosnan JT, Brosnan ME. The sulfur-containing amino acids: an overview. J Nutr. 2006;136(6 Suppl):1636S-40S.
21. O'Leary F, Samman S. Vitamin B12 in health and disease. Nutrients. 2010;2(3):299-316.
22. Stover PJ. Physiology of folate and vitamin B12 in health and disease. Nutr Rev. 2004;62(6 Pt 2):S3-S12.
23. Finkelstein JD. Inborn errors of sulfur-containing amino acid metabolism. J Nutr. 2006;136(6 Suppl):1750S-4S.
24. Selhub J. Homocysteine metabolism. Annu Rev Nutr. 1999;19:217-46.
25. Refsum H, Ueland PM, Nygård O, Vollset SE. Homocysteine and cardiovascular disease. Annu Rev Med. 1998;49:31-62.
26. Huo Y, Li J, Qin X, *et al*. Efficacy of folic acid therapy in primary prevention of stroke among adults with hypertension in China: the CSPPT randomized clinical trial. JAMA. 2015;313(13):1325-35.
27. Homocysteine Lowering Trialists' Collaboration. Dose-dependent effects of folic acid on blood concentrations of homocysteine: a meta-analysis of randomized trials. Am J Clin Nutr. 2005;82(4):806-12.
28. Baker DH. Comparative species utilization and toxicity of sulfur amino acids. J Nutr. 2006;136(6 Suppl):1670S-5S.
29. Finkelstein JD, Martin JJ. Methionine metabolism in mammals: distribution of homocysteine between competing pathways. J Biol Chem. 1984;259(15):9508-13.
30. McCaddon A, Miller JW. Assessing the association between homocysteine and cognition: reflections on Bradford Hill, meta-analyses, and causality. Nutr Rev. 2015;73(10):723-35.
31. Ueland PM, Refsum H. Plasma homocysteine, a risk factor for vascular disease: plasma levels in health, disease, and drug therapy. J Lab Clin Med. 1989;114(5):473-501.
32. Carmel R, Green R, Rosenblatt DS, Watkins D. Update on cobalamin, folate, and homocysteine. Hematology Am Soc Hematol Educ Program. 2003;2003(1):62-81.
33. Forges T, Monnier-Barbarino P, Alberto JM, Guéant-Rodriguez RM, Daval JL, Guéant JL. Impact of folate and homocysteine metabolism on human reproductive health. Hum Reprod Update. 2007;13(3):225-38.
34. Kamaly HF, Sharkawy AA. Hormonal residues in chicken and cattle meat: a risk threat to present and future consumer health. Food Chem Toxicol. 2023;182:114172.
35. Zhang Q, *et al*. Risk assessment of veterinary drug residues in meat products. Curr Drug Metab. 2020;21(10):779-89.
36. Fukagawa NK. Sparing of methionine requirements: evaluation of human data takes sulfur amino acids beyond protein. J Nutr. 2006;136(6 Suppl):1676S-81S.
37. Garlick PJ. Toxicity of methionine in humans. J Nutr. 2006;136(6 Suppl):1722S-5S.
38. Lyon P, Strippoli V, Fang B, Cimmino L. B vitamins and one-carbon metabolism: implications in human health and disease. Nutrients. 2020;12(9):2867.
39. You SH, Lee CN. Dietary exposure and risk assessment of beta-agonist residues in commercial beef and pork in Taiwan. Foods. 2023;12(22):4052.
40. Centner TJ, Alvey JC, Stelzleni AM. Beta agonists in livestock feed: status, health concerns, and international trade. J Anim Sci. 2014;92(9):4234-46.

How to Cite This Article
Abdulrazzaq NA, Hameed MF. Potential human-health implications of ractopamine and methionine in poultry products: an expanded substance-specific narrative review. International Journal of Advanced Biochemistry Research Noosphere. 2026;2(2):36-42.
doi:10.54660/IJABRN.2026.2.2.36-42.

Creative Commons (CC) License
This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution NonCommercial-ShareAlike 4.0 International (CC BY-NC-SA 4.0) License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.