Proceedings of the Nutrition Society

cambridge.org/pns

The Nutrition Society of Australia 2025 was held at the Gold Coast Convention & Exhibition Centre on 3rd - 5th December 2025

Nourishing Generations: 50 years of the Nutrition Society of Australia

Symposium 10 – Nutrigenomics and personalised nutrition

Review Article

Cite this article: Hui PSC, Reed DR, and Hwang L-D (2026). Genetics of sensory nutrition. *Proceedings of the Nutrition Society*, page 1 of 8. doi: 10.1017/S0029665126105400

Received: 31 March 2026 UTC
Revised: 29 July 2026 UTC
Accepted: 30 July 2026 UTC

Keywords:
Genetics; Taste; Smell; Sensory nutrition

Corresponding author:
Liang-Dar Hwang;
Email: d.hwang@uq.edu.au

© The Author(s), 2026. Published by Cambridge University Press on behalf of The Nutrition Society. This is an Open Access article, distributed under the terms of the Creative Commons Attribution licence (https://creativecommons.org/licenses/by/4.0/), which permits unrestricted re-use, distribution and reproduction, provided the original article is properly cited.

Genetics of sensory nutrition

Phoebe S.C. Hui[1], Danielle R. Reed[2] and Liang-Dar Hwang[1,2]

[1]Institute for Molecular Bioscience, The University of Queensland, Australia and [2]Monell Chemical Senses Center, USA

Abstract

Sensory nutrition is an emerging research area that examines how chemosensory perception, particularly taste and smell, shapes dietary behaviours, nutritional status, and disease risk. Variation in how individuals perceive the same foods may help explain differences in diet quality and responsiveness to behavioural dietary interventions, yet chemosensory phenotypes are rarely measured at the population level. Genetic variation contributes to this perceptual diversity and provides a framework for investigating sensory determinants of diet using genomic approaches. This review summarises evidence linking chemosensory genetics to perception and dietary behaviours, and discusses applications for causal inference and for precision and personalised nutrition. Twin studies reveal moderate to high heritability for bitter taste traits, with more modest and phenotype-dependent estimates for sweetness, sourness, saltiness, fat-related traits, and olfactory measures. Genome-wide association studies have identified loci in taste and olfactory receptor genes associated with specific chemosensory traits as well as liking and intake of various foods, although the evidence remains concentrated on bitter taste and populations of European ancestry. These genetic variants have been used in Mendelian randomisation, a genetics-based approach that strengthens causal inference, to test whether sensory traits influence dietary behaviour. For precision nutrition, evidence for taste genotype-stratified interventions remains limited and mixed. Realising the promise of sensory nutrition will require scalable and standardised chemosensory phenotyping, Findable, Accessible, Interoperable, and Reusable (FAIR) data infrastructure, expanded research in diverse populations, and integration with broader biological and sociocultural determinants of dietary intake.

Chemosensory perception – encompassing taste (gustation), smell (olfaction), and chemesthesis (detection of chemical irritants) – shapes how we perceive food flavours and, consequently, our food preferences and dietary intake[1,2]. Individuals can vary substantially in their perception of the same food. For example, coriander is described as foul and soapy by some but fresh and zesty by others[3], while some bitter compounds are intensely unpleasant to some people but barely detectable to others[2]. While chemosensory perception is acknowledged as an individual-level barrier to dietary changes in clinical settings, it is seldom systematically measured or considered at the population level. These perceptual differences are in part genetically determined, which may offer an explanatory layer for why individuals respond differently to the same dietary advice, beyond what conventional measures and social determinants can account for. Moreover, genetic variants influencing chemosensory perception may offer a novel approach to understanding the causal effects of dietary exposures on disease risk, leveraging techniques from genetic epidemiology such as Mendelian randomisation (MR). This review summarises evidence from genomic approaches on the relationships between chemosensory perception and dietary intake, and explores applications of sensory genetics for causal inference, and for precision and personalised nutrition. An overview of the topics covered is presented in Fig. 1.

Taste, smell, and chemesthesis

Chemosensory perception arises when chemical compounds from the environment interact with specialised receptors in the oral and nasal cavities, initiating neural signals that the brain interprets as taste, smell, and chemesthetic sensations[4]. These three systems operate through distinct molecular and anatomical pathways but together translate chemical information from food and beverages into the unified percept of flavour[1,4].

Taste buds, the primary sensory organs for gustation, are onion-shaped clusters comprising 50–100 specialised cells located within gustatory papillae on the tongue's surface. These cells detect the five basic taste modalities: bitter, salty, sour, sweet, and umami[4], with emerging evidence for fattiness and starchiness as additional tastes[2,5]. Salty and sour tastes are mediated by ion channels that detect, respectively, sodium and protons (acids), while bitter, sweet, and umami tastes are detected by G-protein-coupled receptors, also called taste receptors[4]. In both cases, stimulus detection initiates intracellular signalling cascades that lead to cell depolarisation

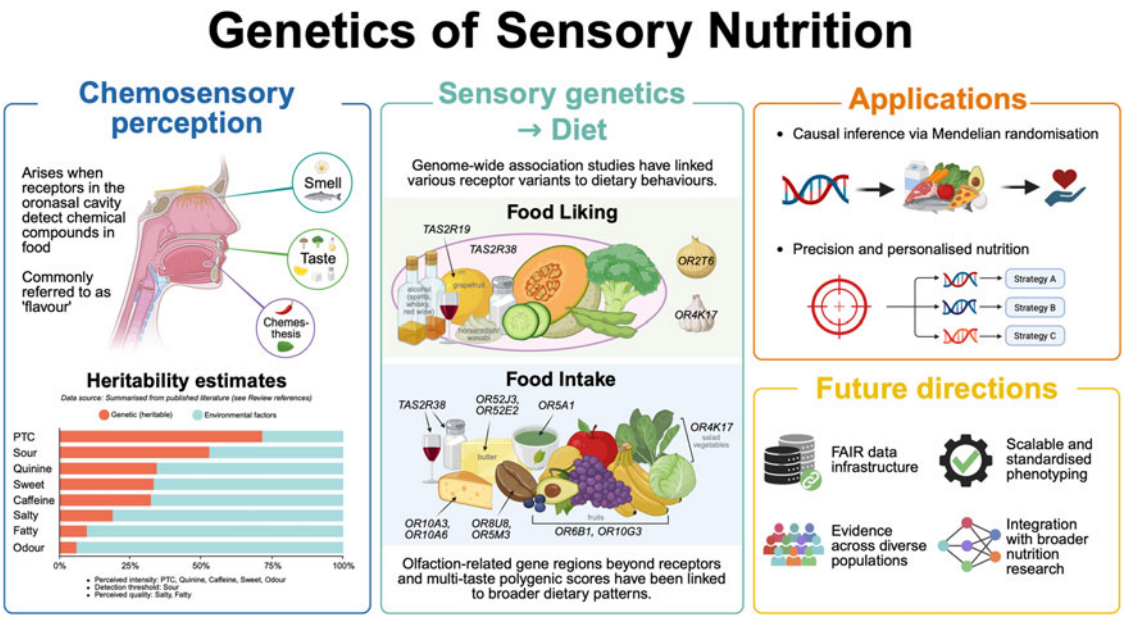


Fig. 1. Chemosensory genetics in nutrition: heritability, dietary associations, applications, and future directions. Heritability represents the proportion of trait variation attributable to genetic factors. TAS2Rs denote taste receptors (family 2), and ORs denote olfactory receptors. FAIR, Findable, Accessible, Interoperable, Reusable; PTC, phenylthiocarbamide. Created in BioRender.

and the release of neurotransmitters[4]. This taste information is then transmitted to the brain via three cranial nerves (CN): facial (CN VII), glossopharyngeal (CN IX), and vagus (CN X)[4].

Taste receptors comprise two families. The TAS1R family has three members, in which the common subunit TAS1R3 pairs with TAS1R2 for sweet taste and with TAS1R1 for umami taste[6]. The human TAS2R family, specialised for bitter taste perception, has approximately 25 functional members[6,7]. Each TAS2R receptor exhibits varying degrees of specificity, with some narrowly tuned to specific chemical classes. For instance, TAS2R38 responds to thiourea compounds, including propylthiouracil (PROP; a synthetic bitter compound widely used in taste research) and isothiocyanates found in cruciferous vegetables when chopped or chewed, while TAS2R16 detects β-glucopyranosides such as amygdalin (e.g., apricot kernels, peach pits)[6]. Others, such as TAS2R14, respond broadly to structurally diverse bitter compounds[6]. Meanwhile, many bitter compounds can activate multiple TAS2Rs, with quinine (a flavouring agent in tonic water) activating nine different receptors[7]. Recent evidence suggests that *TAS2R2*, previously classified as a pseudogene, encodes a 26th functional bitter receptor in some populations, suggesting that the exact number of functional bitter receptors may vary across individuals due to ancestry-specific polymorphisms[8].

Olfactory perception occurs when volatile chemical compounds (odorants) bind to olfactory receptors, expressed on the membrane of olfactory sensory neurones (OSNs) within the nasal epithelium[4,9]. Each OSN only expresses one type of olfactory receptor, and the OSN itself serves as both the receptor cell and the primary sensory neurone, with its axons collectively forming the olfactory nerve (CN I)[4]. Humans possess approximately 400 functional olfactory receptors, which are also G-protein-coupled receptors[9]. Each receptor can be activated by diverse odorants, while individual odorants can also activate multiple different receptors[4,9]. This allows a combinatorial coding pattern through which a limited repertoire of receptors can discriminate between thousands of distinct smells[4,9]. Upon receptor activation, signals from OSNs expressing the same receptor type converge in the olfactory bulb before being transmitted to the olfactory cortex for processing odour intensity and to higher-order regions for recognition, reward, and memory association[4].

Chemesthesis refers to the activation of somatosensory pathways by specific chemical stimuli, producing sensations often described as irritation, heat, cooling, or tingling[10]. Dietary examples include the cooling sensation of menthol (mint), burning from capsaicin in chilli peppers, and the pungency of mustard[10]. Chemesthesis is mediated by mechanoreceptors, nociceptors, and thermoreceptors located in the oronasal cavity, which transmit these chemical signals to the brain primarily via the trigeminal nerve (CN V), and via the glossopharyngeal nerve (CN IX) from the posterior third of the tongue[10]. Taken together, variation in perception can arise at multiple stages, including chemical-receptor binding[2,6,9], intracellular signal transduction within the sensory cells[4,9], and subsequent neural processing in the brain[1,4,10].

Genetic determinants of taste and smell perception

Variation in perception mechanisms can manifest as differences in the chemosensory experiences of the same stimuli, including sensitivity thresholds (minimum concentration required for detection), suprathreshold perceived intensity (subjective strength at a given concentration), identification, and hedonic responses (perceived pleasantness or liking). These differences are, in part, driven by genetic variation, including small genetic differences in taste and smell receptor genes, the extent of which has been reviewed extensively elsewhere[2,5]. While these phenotypes are relatively well established for taste and smell, chemesthesis encompasses a broad range of sensations for which trait definitions and standardised measurement remain less developed[1]. These measurement challenges are an active area of methodological development, with ongoing work seeking rapid, scalable, and ecologically relevant approaches to assessing chemesthetic function[1,11].

https://doi.org/10.1017/S0029665126105400 Published online by Cambridge University Press

Evidence for genetic contributions to taste and smell comes in part from twin studies, which compare trait similarities between monozygotic (identical) and dizygotic (fraternal) twins to estimate heritability (h^2), the proportion of individual variation in the trait attributable to genetic factors. As monozygotic twins share approximately 100% of their genome while dizygotic twins share on average 50%, greater trait similarity between monozygotic twins compared to dizygotic twins suggests genetic influences on the trait.

Heritability estimates for the perceived intensity of bitter compounds vary by stimulus. PROP and the structurally similar phenylthiocarbamide (PTC) consistently showe high heritability ($h^2 = 0.71–0.73$)[12–14], followed by quinine ($h^2 = 0.34–0.40$)[12–14], caffeine ($h^2 = 0.30–0.34$)[13,14], and sucrose octa-acetate ($h^2 = 0.28–0.40$)[13,14], which show moderate heritability. Sweet taste perception falls within a similar range ($h^2 = 0.30–0.36$ for sugars and high-potency sweeteners)[14,15]. Multivariate modelling further suggests that the perception of sweetness and bitterness shares a common genetic factor that explains 17–37% of the variance in non-PROP bitter perception and 8% of the variance in sweet perception, particularly through post-receptor transduction mechanisms common to taste G-protein-coupled receptors[14]. Heritability of sour taste recognition threshold has been estimated at 0.53 using citric acid[16]. Heritability estimates for saltiness (sodium chloride) vary by phenotype, ranging from no detectable genetic contribution for recognition thresholds[16] to modest estimates for perceived saltiness ($h^2 = 0.19$) and liking ($h^2 = 0.38$)[17]. Similarly, for fat taste, heritability appears higher for liking (up to $h^2 = 0.29$) than for perceived fattiness ($h^2 = 0.05–0.15$)[17]. For olfactory traits, heritability estimates appear less established than for taste. One twin study estimated heritability of odour pleasantness at 0.38–0.40, while odour intensity showed markedly lower heritability at 0.03–0.09[18].

While heritability estimates quantify the genetic influences, they do not inform which specific genes are responsible for this variation. Genome-wide association studies (GWAS) have since enabled a hypothesis-free approach to identifying these genetic contributors, systematically testing associations between genetic variants across the entire genome, typically single-nucleotide polymorphisms (SNPs), and traits of interest[19]. By comparing the frequencies of each variant across individuals exhibiting different levels of the trait, GWAS can identify loci associated with the trait of interest without prior assumptions or knowledge of biological function[19].

Bitter taste perception has been extensively studied at the genome-wide level, with multiple GWAS ($n = 141–1,999$) revealing associations for various bitter compounds within bitter taste receptor clusters on chromosomes 7 (PROP, denatonium benzoate) and 12 (quinine, caffeine, sucrose octa-acetate)[20–22]. The bitter receptor gene cluster on chromosome 12 has also been associated with perceived bitterness of acesulfame potassium, an artificial sweetener that can produce a bitter aftertaste[23]. Cross-ancestry comparisons reveal both shared and population-specific genetic architecture for bitter taste perception. *TAS2R38* has been consistently associated with PROP or PTC perception across populations, though both the frequency of taster haplotypes and the distributions of taster phenotypes vary considerably across ancestries[24,25].

GWAS findings for sweet taste perception remain relatively inconsistent. Hwang et al.[26] identified several suggestive associations in Australian ($n = 1,757$ adolescents) and US ($n = 686$ adults) samples regarding perceived sweet taste intensity, but these associations failed to replicate across the adolescent and adult cohorts. Previously reported associations with sweet and umami receptors (*TAS1R2*, *TAS1R3*, and *GNAT3*) also failed to replicate in both GWAS and candidate gene approaches in these European-ancestry cohorts, with some associations appearing to reflect population stratification rather than within-ancestry associations[26]. Between-ancestry variation in allele frequencies in these genes may therefore contribute to differences in sweet taste traits between ancestral populations (e.g., European versus African ancestry), whereas within-ancestry genetic variation appears to explain little variation in sweet taste perception within European populations. Similarly, GWAS on self-reported preference for sweet taste ($n = 425$ individuals with metabolic syndrome) did not replicate these receptor associations, but instead identified an association with variants in *PTPRN2*, which requires independent replication[27]. More broadly, disentangling ancestry-specific genetic associations in sweet taste perception remains an active area of research, including ongoing examination of how environmental sweetness exposure may interact with genetic predispositions for sweet liking in populations of African and East Asian ancestry[28].

For smell perception, GWAS have examined both identification and sensitivity to specific odorants. For odour identification, the largest meta-analysis to date, involving 21,495 European individuals, identified several age-adjusted associations within olfactory receptor gene clusters, as well as novel associations with *GSX2* (involved in olfactory bulb neurogenesis) and *ADCY2* (involved in G-protein-coupled receptor signalling pathways), with some showing sex-specific associations[29]. SNP-based heritability estimates (the proportion of variance explained by common genetic variants measured on genotyping arrays used in the respective GWAS) for odour identification ranged from 8–12%, depending on the odorant and sex[29]. Earlier work on odour identification in African American older adults identified loci linked to neurodegenerative (Parkinson's disease, Alzheimer's disease) or neuropsychiatric (schizophrenia, epilepsy) diseases[30], consistent with the consensus that olfactory dysfunction is an early symptom of neurodegenerative diseases. Notably, these associations were not observed in European American participants from the same cohorts, highlighting potential ancestry-specific contributions to the genetic architecture of the sense of smell. For odour sensitivity, GWAS have identified associations near or within olfactory receptor gene clusters, particularly for perceived intensity of androstenone[31] and detection thresholds of food-related odours, including 2-heptanone (blue cheese), isobutyraldehyde (malt), and β-damascenone (apple)[32]. Genetic variation in olfactory receptor genes has also been associated with specific odorants such as cis-3-hexen-1-ol (green/grassy; *OR2J3*)[33] and β-ionone (fruity/floral; *OR5A1*)[34], while sensitivity to these odorants has been associated with preference for foods containing them[34,35]. A missense variant in *TAAR5*, a trace amine-associated receptor expressed in the olfactory epithelium, has been associated with reduced perceived intensity of fish odour[36].

Other chemosensory modalities remain understudied. Using food liking as proxies for taste preferences, Haydar et al.[37] identified a genome-wide significant variant on chromosome 5 associated with umami liking, albeit with no obvious biological pathway, and confirmed associations between *TAS2R38* and

https://doi.org/10.1017/S0029665126105400 Published online by Cambridge University Press

bitter-tasting foods (rocket salad) in a Danish population. GWAS evidence for fatty, salty, sour, and starchy tastes remains limited to date and is virtually non-existent for chemesthesis.

From sensory perception to dietary behaviours

The influence of chemosensory perception on dietary intake is well-acknowledged across multiple frameworks of food choice[38]. From an evolutionary perspective, this relationship conferred survival advantages, with sweet and umami tastes indicating energy-dense and protein-rich foods, and bitterness warning against potentially toxic compounds[39,40]. While less is understood about the two remaining tastes, it is thought that saltiness helps signal the presence of essential minerals, and sourness helps detect spoilage or unripe fruits[40]. In present-day contexts, chemosensory perception continues to shape food choice and intake through its influence on palatability. For example, children who are highly responsive to bitter and sour tastes (i.e., higher perceived intensity) demonstrate lower preferences for fruits and vegetables[41], while adults with lower oligofructose detection thresholds tend to have higher carbohydrate and overall energy intake[42]. In terms of smell, individuals with olfactory dysfunction showed poorer diet quality, characterised by higher energy density and higher intakes of saturated fats and added sugars, potentially due to compensatory behaviours to reduce perceived flavour enjoyment, such as seeking more intense basic tastes[43]. Beyond individual physiology, the food industry actively exploits chemosensory cues, such as scented product packaging, to influence consumer behaviour and intake[44].

Consistent with observational findings, genetic studies have linked variation in chemosensory perception to individual differences in dietary traits, as reviewed elsewhere[2,5,24]. However, this evidence remains uneven across chemosensory modalities and populations. Candidate gene studies have predominantly focused on taste receptor variants, especially bitter taste receptors, and many have used small samples with limited power to detect the generally modest associations with complex dietary traits[2,5,24]. Although *TAS2R38* has accumulated the most evidence linking genetic variation in taste to food preferences, including vegetables, alcohol, and sweet foods[2,5,24], findings have often been inconsistent or unreplicated, and publication bias may inflate the apparent consistency of associations.

Recent large-scale genomic studies have expanded both the scale and breadth of this evidence, although most studies have been conducted in populations of European ancestry, limiting the generalisability of their findings. A large phenome-wide association study (PheWAS) in the UK Biobank demonstrated that bitter-sensitive *TAS2R38* alleles were associated with reduced liking for alcoholic beverages, grapefruit, horseradish, and salty foods, and with lower intake of added salt and red wine, though several previously reported associations, such as coffee, Brussels sprouts, and pickles, were not replicated[45]. An early GWAS in 23andMe cohort also identified variants within an olfactory receptor gene cluster associated with coriander perception (soapy taste) and dislike[3]. While *OR6A2* and *OR10A2* were proposed as the most plausible candidates, these associations were not replicated in a subsequent UK Biobank study, which instead identified an independent association with *OR10A6*[46].

This UK Biobank study systematically examined non-synonymous variants across 425 taste and olfactory receptor genes in relation to 140 food liking traits and identified 700 associations across 117 genes[46]. Among the strongest signals were *OR4K17* with garlic liking, *TAS2R19* with grapefruit liking, and *OR2T6* with onion liking. Notably, some variants exhibited broad tuning (i.e., being associated with multiple food-liking traits), whereas others showed narrower, food-specific associations[46]. When food-liking items were categorised empirically based on genetic correlations, associations with taste and olfactory receptor genes were concentrated in acquired-taste categories (including bitter beverages, alcoholic drinks, and pungent foods) and low-calorie foods (vegetables, fruits, wholegrains) rather than highly palatable foods such as processed, fatty, or sweet items[47]. The pattern suggests that chemosensory receptor variants may be more relevant to learned or aversion-driven preferences than to reward-driven liking[47].

In a GWAS of 85 food intake traits in the UK Biobank[48], several olfactory receptor gene regions emerged among the 814 associated loci identified, including *OR6B1* and *OR10G3* with fruit intake, a cluster including *OR5A1* with tea intake, a nearby but distinct cluster containing *OR8U8* and *OR5M3* with coffee intake, *OR52J3* and *OR52E2* with butter, *OR4K17* with salad vegetables, and *OR10A3* and *OR10A6* with cheese intake. When examining associations between nonsynonymous chemosensory receptor variants and dietary patterns derived from the same food items, however, associations were weaker and far fewer variants reached genome-wide significance[49]. In contrast, a GWAS of adherence to a traditional Japanese dietary pattern identified a locus on chromosome 14, annotated to genes involved in olfaction (*BCL2L2*, *SLC22A17*), suggesting that genetic variation in broader olfactory pathways, beyond receptor variation alone, may influence dietary behaviours[50].

Beyond individual SNPs, polygenic scores (PGS) – which aggregate the effect estimates of many variants into a single value representing an individual's genetic predisposition to a specific trait – have been used to examine whether cumulative genetic predisposition to taste perception is associated with dietary behaviours. Gervis et al.[51] derived taste-specific PGS for bitter, sweet, umami, and sour perception in over 6,200 participants from the Framingham Heart Study using 33 taste-related SNPs from prior taste perception-related GWAS, finding that higher bitter perception scores were associated with lower wholegrain intake and higher umami perception scores were associated with lower vegetable intake. Similarly, a sweet taste PGS constructed from 38 SNPs was inversely associated with the Dietary Approaches to Stop Hypertension score in Puerto Rican older adults, with higher genetic predisposition to sweet taste sensitivity associated with lower wholegrain and vegetable intake[52]. However, the predictive accuracy of PGS is theoretically bounded by the SNP-based heritability of the underlying trait and, in practice, by the sample size and quality of the discovery GWAS[19]. Given the inconsistent replication and sparse GWAS evidence across several chemosensory modalities, validating chemosensory PGS against directly measured perception phenotypes in large, diverse cohorts remains an important methodological priority.

Nutritional status and disease risk are important potential outcomes downstream of dietary behaviours. For genetic variation in chemosensory perception to affect these outcomes through diet, these genetic differences would need to produce changes in the relevant dietary exposure – whether specific foods or nutrients, or broader dietary patterns – that are sufficiently large and sustained to affect nutritional status and disease risk. The current evidence base supports associations between some variants related to chemosensory perception and specific dietary phenotypes, but studies generally did not examine downstream nutritional or health consequences. Nonetheless, the *TAS2R38* PheWAS described

https://doi.org/10.1017/S0029665126105400 Published online by Cambridge University Press

above reported associations of *TAS2R38* variants with renal function markers and bipolar disorder[45]. While parallel dietary and health associations for the same perception-related variants do not establish a diet-mediated pathway, these findings provide hypotheses for future studies examining potential downstream health implications of chemosensory variation.

Leveraging sensory genetics for causal inference

Despite evidence linking chemosensory perception to dietary intake, observational associations cannot definitively establish causality due to confounding and reverse causation. Sodium restriction interventions have been shown to shift salt taste preferences towards lower concentrations within three months[53], while reduced simple sugar intake has been shown to increase perceived intensity of both low- and high-sugar foods[54]. However, a larger six-month trial varying sweet taste exposure found no changes in sweet taste liking or intensity perception[55], suggesting that the plasticity of taste preferences may be more limited than indicated by shorter-term studies. Perception also changes in response to events throughout the life course, such as illness, medical treatment, or environmental exposures, including tobacco smoking[1]. Moreover, unlike dietary intake, which can be modified through randomised interventions, chemosensory perception cannot be easily manipulated in the way randomised controlled trials (RCTs) require. This limits our ability to draw causal conclusions about the downstream effects of chemosensory perception itself through traditional experimental approaches.

MR uses genetic variants as proxies for modifiable exposures to create natural experiments analogous to RCTs to infer causality[56,57]. Using SNPs in bitter taste receptor genes as proxies for perceived bitterness – namely rs1726866 in *TAS2R38*, and rs10772420 and rs2597979 in the bitter receptor cluster on chromosome 12, which account for 46%, 6%, and 2% of variance in perceived intensity of PROP, quinine, and caffeine, respectively[21,22,58] – MR analyses revealed that higher perceived caffeine intensity causally increased coffee intake and the likelihood of being a heavy coffee drinker (>4 cups/day)[59]. In contrast, higher perceived intensity of PROP and quinine decreased coffee intake[59]. Higher PROP bitterness was also associated with lower alcohol consumption, particularly red wine[59]. These findings suggest that bitter taste perception may causally shape the consumption of certain bitter beverages, though effects vary by the bitter stimulus used to measure perception and by beverage type, and effect sizes are modest[59].

The same challenge of establishing causality extends to diet-disease relationships in nutritional epidemiology more broadly, where the number of MR studies examining food and beverage exposures has grown rapidly, with 178 MR studies identified since 2002, of which 43% were published in 2024 alone[60]. Coffee was the most studied exposure, followed by tea, alcohol, and overall diet (defined as more than 5 food groups in one publication). However, applying MR to dietary exposures presents methodological challenges. Variants identified in dietary GWAS may reflect behavioural responses to health status rather than primary dietary preferences[46]. For example, *FTO* variants are associated with both higher BMI and healthier dietary behaviours such as low sugar intake[26] (possibly reflecting reverse causation), meaning that if used as a proxy for diet, any observed association with a health outcome could reflect *FTO*'s direct effects on adiposity and metabolism rather than the lower sugar intake itself. Chemosensory receptor variants may provide more biologically interpretable instruments than variants selected solely on the basis of genome-wide significance, as they have a clearer mechanistic link to perception and dietary behaviours and are less likely to capture behavioural or disease-related confounding[46,49]. Nonetheless, biological plausibility alone does not inherently establish instrument validity, as gene-diet associations may still be weak, and extra-oronasal receptor expression may introduce pathways to disease outcomes that do not operate through perception and dietary intake. Instrument strength and potential pleiotropy must therefore always be evaluated for each causal question.

Leveraging sensory genetics for precision and personalised nutrition

While population-wide dietary guidelines and policies have been successful at preventing nutritional deficiencies, chronic disease prevention presents greater complexity and greater individual variability in response to dietary interventions[61]. This has driven increasing interest in precision and personalised nutrition strategies, which leverage technological advances in genomics and other data sources to provide highly tailored interventions[61]. In particular, precision nutrition targets population subgroups with shared characteristics, while personalised nutrition refers to recommendations tailored to individuals[61].

The potential for taste genetics to guide dietary recommendations and improve adherence was proposed nearly two decades ago[62]. As discussed earlier, sweet taste preferences show resistance to modification through dietary exposure, with participants spontaneously returning to baseline sweet food intake levels after the intervention[55]. This suggests that individual differences in taste preferences may be partly driven by factors less amenable to environmental influences. More broadly, sensory genetics may inform the design and delivery of dietary interventions by explaining heterogeneity in responses to interventions. In a randomised controlled trial aimed at promoting vegetable intake, Calancie et al.[63] examined whether genetic variation in bitter taste sensitivity, indexed by the three *TAS2R38* SNPs associated with PROP taster status, moderated the intervention's effectiveness. While all participants receiving intensive dietary counselling increased vegetable intake regardless of genotype, PROP tasters showed smaller increases than intermediate tasters and non-tasters[63]. In contrast, among participants receiving minimal counselling, vegetable consumption declined over time only among PROP tasters[63]. These findings suggest that genetically predisposed bitter taste sensitivity may hinder sustained dietary change, and that individuals with this sensory profile may require more intensive or tailored behavioural support.

Beyond *TAS2R38*-mediated PROP sensitivity, however, evidence for genotype-stratified dietary interventions across other bitter compounds and receptors (e.g., quinine and caffeine) and other chemosensory modalities remains absent. While genotypes are less susceptible to measurement error and remain stable over time, future intervention studies might also consider whether direct chemosensory phenotyping (e.g., using tastant solutions)[11] offers complementary stratification to genotyping alone, given that genotype explains only a proportion of the variation in chemosensory perception. In dietary counselling, the more immediate application of the evidence reviewed herein may lie in recognising chemosensory barriers to dietary behaviour change, especially in the absence of routine genotyping. In an online study of 218 adults, Briazu et al.[64] found that participants receiving healthy-eating advice tailored to profiles combining questionnaire-assessed sensory

https://doi.org/10.1017/S0029665126105400 Published online by Cambridge University Press

preferences with cognitive and sociodemographic characteristics reported stronger intention to change diet than those receiving generic advice, although the contribution of sensory tailoring could not be isolated and changes in dietary behaviour were not assessed. A reported aversion to the taste or smell of a recommended food should therefore inform how advice is individualised, consistent with broader principles of patient-centred care that emphasise responsiveness to lived experiences[65]. Recognising such an aversion as a genuine barrier that may not always be readily overcome does not require attributing the aversion to a particular genotype.

Future directions and conclusions

Realising the potential of sensory genetics to inform precision and personalised nutrition fundamentally depends on the quality and scale of sensory phenotype data available for genetic association studies. However, chemosensory science currently lags behind other disciplines in data sharing, standardisation, and infrastructure[66]. Transitioning towards the FAIR principles – ensuring data are Findable, Accessible, Interoperable, and Reusable – is essential for the field to achieve the same level of success seen in genomics for other phenotypes, where standardised repositories have enabled meta-analyses across millions of individuals that drive breakthroughs in understanding complex traits[66]. Chemosensory research would require a similar transformation, moving from small, isolated studies to coordinated, large-scale data collection that can support genetic discovery and enable validation across diverse populations[66]. Critically, establishing shared repositories and adopting common data standards (e.g., naming conventions, structured file formats) will facilitate computational analyses and integration across studies[66]. Similarly, developing and adopting consistent, scalable chemosensory phenotyping protocols across taste, smell, and chemesthesis would help ensure that data combined across studies reflect genuine variation in perception rather than methodological artefacts[11]. Fostering a culture of data contribution across research groups will be as critical as these technical foundations in building the evidence base needed to integrate sensory genetics into nutrition research and practice[66].

The vast majority of GWAS and MR studies examining chemosensory genetics have been conducted in populations of European ancestry, as with most genomic publications. It is important to distinguish between genomic ancestry (genetic similarity reflecting shared evolutionary history) and ethnicity or race (sociocultural constructs) as both may independently influence dietary behaviours through different mechanisms. With respect to genomic ancestry, variants influencing chemosensory perception may differ in the specific variants involved, their frequencies, and their effect sizes across diverse ancestries. This has direct implications for the predictive accuracy of PGS, which deteriorates with increasing ancestral distance from the population in which it was derived[67], and for the validity of MR findings, as the strength and specificity of genetic instruments may differ across ancestral populations[57]. With respect to cultural diversity, dietary research should continue to strive for inclusivity in its design and measurement tools to ensure that foods and dietary practices from diverse sociocultural contexts are adequately represented in the data[68].

In summary, chemosensory perception is a fundamental yet often under-measured determinant of food choice and intake. The application of genomic tools has clarified the biological basis for individual differences in taste and smell perception, with genetic variation in chemosensory receptors being associated with the consumption of specific foods and beverages. Sensory genetics has the potential to enhance the rigour of nutritional epidemiology through techniques such as MR for causal inference, or PGS to study chemosensory-diet associations at scale. Beyond informing intervention design, accounting for individual differences in chemosensory perception provides an additional lens for evaluating heterogeneity in intervention responses and effectiveness. Integrating sensory genetics into nutrition research supports a more nuanced path towards improving long-term dietary adherence and, ultimately, chronic disease prevention.

Acknowledgements. None.

Author contributions. PSCH: Writing – Original Draft, Visualization; DRR: Writing – Review & Editing, Supervision; LDH: Conceptualization, Writing – Review & Editing, Supervision, Funding acquisition. All authors reviewed and approved the final manuscript.

Financial support. PSCH is supported by a UQ Research Training Scholarship from the University of Queensland (UQ). DRR is supported by the Monell Institutional Fund. LDH is supported by an Australian Research Council (ARC) Discovery Early Career Researcher Award (DE240100014). UQ, ARC, and the Monell Chemical Senses Center had no role in the design, analysis, or writing of this article.

Competing interests. The authors declare no conflicts of interest.

References

1. Reed DR, Alhadeff AL, Beauchamp GK, *et al.* (2021) NIH workshop report: sensory nutrition and disease. *Am J Clin Nutr* **113**, 1, 232–245. doi: 10.1093/ajcn/nqaa302.
2. Feeney EL, McGuinness L, Hayes JE, *et al.* (2021) Genetic variation in sensation affects food liking and intake. *Curr Opin Food Sci* **42**, 203–214. doi: 10.1016/j.cofs.2021.07.001.
3. Eriksson N, Wu S, Do CB, *et al.* (2012) A genetic variant near olfactory receptor genes influences cilantro preference. *Flavour* **1**, 22. doi: 10.1186/2044-7248-1-22.
4. Jaime-Lara RB, To L & Joseph PV (2021) Anatomy, physiology, and neurobiology of olfaction, gustation, and chemesthesis. In *Sensory Science and Chronic Diseases* [Internet], pp. 3–20 [PV Joseph, VB Duffy, editors]. Cham: Springer International Publishing. doi: 10.1007/978-3-030-86282-4_1.
5. Diószegi J, Llanaj E & Ádány R (2019) Genetic background of taste perception, taste preferences, and its nutritional implications: a systematic review. *Front Genet* **10**, 1272. doi: 10.3389/fgene.2019.01272.
6. Behrens M & Schaefer S (2025) Bitter taste receptors. *Chem Senses* **50**, bjaf064. doi: 10.1093/chemse/bjaf064.
7. Meyerhof W, Batram C, Kuhn C, *et al.* (2010) The molecular receptive ranges of human TAS2R bitter taste receptors. *Chem Senses* **35**, 2, 157–170. doi: 10.1093/chemse/bjp092.
8. Lang T, Di Pizio A, Risso D, *et al.* (2023) Activation profile of TAS2R2, the 26th human bitter taste receptor. *Mol Nutr Food Res* **67**, 11, 2200775. doi: 10.1002/mnfr.202200775.
9. Wu C, Xu M, Dong J, *et al.* (2024) The structure and function of olfactory receptors. *Trends Pharmacol Sci* **45**, 3, 268–280. doi: 10.1016/j.tips.2024.01.004.
10. Linne BM & Simons CT (2021) Integration of taste, smell, and chemesthesis: clinical implications. In *Sensory Science and Chronic Diseases* [Internet], pp. 105–124 [PV Joseph, VB Duffy, editors]. Cham: Springer International Publishing. doi: 10.1007/978-3-030-86282-4_5.
11. Munger SD, Zhao K, Barlow LA, *et al.* (2025) Towards universal chemosensory testing: needs, barriers, and opportunities. *Chem Senses* **50**, bjaf015. doi: 10.1093/chemse/bjaf015.

https://doi.org/10.1017/S0029665126105400 Published online by Cambridge University Press

12. Knaapila A, Hwang LD, Lysenko A, *et al.* (2012) Genetic analysis of chemosensory traits in human twins. *Chem Senses* **37**, 869–881. doi: 10.1093/chemse/bjs070.
13. Hansen JL, Reed DR, Wright MJ, *et al.* (2006) Heritability and genetic covariation of sensitivity to PROP, SOA, quinine HCl, and caffeine. *Chem Senses* **31**, 403–413. doi: 10.1093/chemse/bjj044.
14. Hwang LD, Breslin PAS, Reed DR, *et al.* (2016) Is the association between sweet and bitter perception due to genetic variation? *Chem Senses* **41**, 737–744. doi: 10.1093/chemse/bjw083.
15. Hwang LD, Zhu G, Breslin PAS, *et al.* (2015) A common genetic influence on human intensity ratings of sugars and high-potency sweeteners. *Twin Res Hum Genet* **18**, 361–367. doi: 10.1017/thg.2015.42.
16. Wise PM, Hansen JL, Reed DR, *et al.* (2007) Twin study of the heritability of recognition thresholds for sour and salty taste. *Chem Senses* **32**, 749–754. doi: 10.1093/chemse/bjm042.
17. Lin C, Colquitt L, Wise P, *et al.* (2020) Studies of human twins reveal genetic variation that affects dietary fat perception. *Chem Senses* **45**, 467–481. doi: 10.1093/chemse/bjaa036.
18. Hunter SR, Lin C, Nguyen H, *et al.* (2024) Effects of genetics on odor perception: Can a quick smell test effectively screen everyone? *Chem Senses* **49**, bjae025. doi: 10.1093/chemse/bjae025.
19. Uffelmann E, Huang QQ, Munung NS, *et al.* (2021) Genome-wide association studies. *Nat Rev Methods Primer* **1**, 59. doi: 10.1038/s43586-021-00056-9.
20. Hwang LD, Gharahkhani P, Breslin PAS, *et al.* (2018) Bivariate genome-wide association analysis strengthens the role of bitter receptor clusters on chromosomes 7 and 12 in human bitter taste. *BMC Genom* **19**, 678. doi: 10.1186/s12864-018-5058-2.
21. Ledda M, Kutalik Z, Souza Destito MC, *et al.* (2014) GWAS of human bitter taste perception identifies new loci and reveals additional complexity of bitter taste genetics. *Hum Mol Genet* **23**, 259–267. doi: 10.1093/hmg/ddt404.
22. Reed DR, Zhu G, Breslin PAS, *et al.* (2010) The perception of quinine taste intensity is associated with common genetic variants in a bitter receptor cluster on chromosome 12. *Hum Mol Genet* **19**, 4278–4285. doi: 10.1093/hmg/ddq324.
23. Kan M, Saraiva LR, Hwang LD, *et al.* (2025) Genome-wide association study of the taste and hedonic ratings of the low-calorie sweetener acesulfame potassium. *Sci Rep* **15**, 22121. doi: 10.1038/s41598-025-05739-x.
24. Robino A, Mezzavilla M, Pirastu N, *et al.* (2014) A population-based approach to study the impact of PROP perception on food liking in populations along the silk road. Simon SA, editor. *PLoS ONE* **9**, 3, e91716. doi: 10.1371/journal.pone.0091716.
25. Yang Q, Williamson AM, Hasted A, *et al.* (2020) Exploring the relationships between taste phenotypes, genotypes, ethnicity, gender and taste perception using Chi-square and regression tree analysis. *Food Qual Prefer* **83**, 103928. doi: 10.1016/j.foodqual.2020.103928.
26. Hwang LD, Lin C, Gharahkhani P, *et al.* (2019) New insight into human sweet taste: a genome-wide association study of the perception and intake of sweet substances. *Am J Clin Nutr* **109**, 1724–1737. doi: 10.1093/ajcn/nqz043.
27. Fernández-Carrión R, Sorlí JV, Coltell O, *et al.* (2021) Sweet taste preference: relationships with other tastes, liking for sugary foods and exploratory genome-wide association analysis in subjects with metabolic syndrome. *Biomedicines* **10**, 79. doi: 10.3390/biomedicines10010079.
28. Cheung MM, Hubert PA, Reed DR, *et al.* (2024) Understanding the determinants of sweet taste liking in the African and East Asian ancestry groups in the U.S.–A study protocol. *PLoS ONE* **19**, 4, e0300071. doi: 10.1371/journal.pone.0300071.
29. Förster F, Emmert D, Horn K, *et al.* (2025) Genome-wide association meta-analysis of human olfactory identification discovers sex-specific and sex-differential genetic variants. *Nat Commun* **16**, 5434. doi: 10.1038/s41467-025-61330-y.
30. Dong J, Wyss A, Yang J, *et al.* (2017) Genome-wide association analysis of the sense of smell in U.S. older adults: identification of novel risk loci in African-Americans and European-Americans. *Mol Neurobiol* **54**, 8021–8032. doi: 10.1007/s12035-016-0282-8.
31. Knaapila A, Zhu G, Medland SE, *et al.* (2012) A genome-wide study on the perception of the odorants androstenone and galaxolide. *Chem Senses* **37**, 541–552. doi: 10.1093/chemse/bjs008.
32. McRae JF, Jaeger SR, Bava CM, *et al.* (2013) Identification of regions associated with variation in sensitivity to food-related odors in the human genome. *Curr Biol* **23**, 1596–1600. doi: 10.1016/j.cub.2013.07.031.
33. McRae JF, Mainland JD, Jaeger SR, *et al.* (2012) Genetic variation in the odorant receptor OR2J3 is associated with the ability to detect the "grassy" smelling odor, cis-3-hexen-1-ol. *Chem Senses* **37**, 585–593. doi: 10.1093/chemse/bjs049.
34. Jaeger SR, McRae JF, Bava CM, *et al.* (2013) A Mendelian trait for olfactory sensitivity affects odor experience and food selection. *Curr Biol* **23**, 1601–1605. doi: 10.1016/j.cub.2013.07.030.
35. Jaeger SR, Pineau B, Bava CM, *et al.* (2012) Investigation of the impact of sensitivity to cis-3-hexen-1-ol (green/grassy) on food acceptability and selection. *Food Qual Prefer* **24**, 230–242. doi: 10.1016/j.foodqual.2011.11.005.
36. Gisladottir RS, Ivarsdottir EV, Helgason A, *et al.* (2020) Sequence variants in TAAR5 and other loci affect human odor perception and naming. *Curr Biol* **30**, 23, 4643–4653.e3. doi: 10.1016/j.cub.2020.09.012.
37. Haydar S, Karlsson CC, Linneberg A, *et al.* (2025) A genome-wide association study of taste liking in the Danish population. *J Nutr* **155**, 2591–2601. doi: 10.1016/j.tjnut.2025.06.001.
38. Chen PJ & Antonelli M (2020) Conceptual models of food choice: influential factors related to foods, individual differences, and society. *Foods* **9**, 12, 1898. doi: 10.3390/foods9121898.
39. Breslin PAS (2013) An evolutionary perspective on food and human taste. *Curr Biol* **23**, R409–418. doi: 10.1016/j.cub.2013.04.010.
40. Callaway E (2012) Evolutionary biology: the lost appetites. *Nature* **486**, S16–17. doi: 10.1038/486S16a.
41. Ervina E, Almli VL, Berget I, *et al.* (2021) Does responsiveness to basic tastes influence preadolescents' food liking? Investigating taste responsiveness segment on bitter-sour-sweet and salty-umami model food samples. *Nutrients* **13**, 8, 2721. doi: 10.3390/nu13082721.
42. Costanzo A, Settapramote N, Utama-ang N, *et al.* (2021) Carbohydrate taste is associated with food intake and body mass in healthy Australian adults. *Nutrients* **13**, 11, 3844. doi: 10.3390/nu13113844.
43. Rawal S, Duffy VB, Berube L, *et al.* (2021) Self-reported olfactory dysfunction and diet quality: findings from the 2011–2014 National Health and Nutrition Examination Survey (NHANES). *Nutrients* **13**, 12, 4561. doi: 10.3390/nu13124561.
44. Spence C (2015) Leading the consumer by the nose: on the commercialization of olfactory design for the food and beverage sector. *Flavour* **4**, 31. doi: 10.1186/s13411-015-0041-1.
45. Brito Nunes C, Lim AWY, Dy Q, *et al.* (2025) Phenome-wide investigation of dietary and health outcomes associated with bitter taste receptor gene TAS2R38. *Eur J Nutr* **64**, 5, 218. doi: 10.1007/s00394-025-03718-6.
46. Hwang LD, Lin C, Evans DM, *et al.* (2026) A biologically informed framework for instrument selection in dietary Mendelian randomization using chemosensory genetics. *BMC Med* **24**, 359. doi: 10.1186/s12916-026-04966-x.
47. May-Wilson S, Matoba N, Wade KH, *et al.* (2022) Large-scale GWAS of food liking reveals genetic determinants and genetic correlations with distinct neurophysiological traits. *Nat Commun* **13**, 2743. doi: 10.1038/s41467-022-30187-w.
48. Cole JB, Florez JC & Hirschhorn JN (2020) Comprehensive genomic analysis of dietary habits in UK Biobank identifies hundreds of genetic associations. *Nat Commun* **11**, 1467. doi: 10.1038/s41467-020-15193-0.
49. Hui PS, Devlin BL, Evans DM, *et al.* (2026) Comparing genome-wide significant and chemosensory variants as instruments for dietary patterns in Mendelian randomisation. *Int J Epidemiol.* In press. doi: 10.1093/ije/dyag186.
50. Suzuki H, Nakamura Y, Matsuo K, *et al.* (2021) A genome-wide association study in Japanese identified one variant associated with a preference for a Japanese dietary pattern. *Eur J Clin Nutr* **75**, 937–945. doi: 10.1038/s41430-020-00823-z.
51. Gervis JE, Ma J, Chui KKH, *et al.* (2023) Bitter- and umami-related genes are differentially associated with food group intakes: the Framingham heart study. *J Nutr* **153**, 483–492. doi: 10.1016/j.tjnut.2022.11.005.
52. Julián-Serrano S, Pomares-Millan H, Mangano KM, *et al.* (2026) Sweet taste genetic risk score and alignment to diet quality among Puerto Rican

https://doi.org/10.1017/S0029665126105400 Published online by Cambridge University Press

adults in Massachusetts. *J Nutr* **156**, 3, 101369. doi: 10.1016/j.tjnut.2026.101369.
53. Bobowski N (2015) Shifting human salty taste preference: potential opportunities and challenges in reducing dietary salt intake of Americans. *Chemosens Percept* **8**, 112–116. doi: 10.1007/s12078-015-9179-6.
54. Wise PM, Nattress L, Flammer LJ, *et al.* (2016) Reduced dietary intake of simple sugars alters perceived sweet taste intensity but not perceived pleasantness. *Am J Clin Nutr* **103**, 50–60. doi: 10.3945/ajcn.115.112300.
55. Čad EM, Mars M, Pretorius L, *et al.* (2026) The sweet tooth trial: a parallel randomized controlled trial investigating the effects of a 6-month low, regular, or high dietary sweet taste exposure on sweet taste liking, and various outcomes related to food intake and weight status. *Am J Clin Nutr* **123**, 101073. doi: 10.1016/j.ajcnut.2025.09.041.
56. Davey Smith G & Ebrahim S (2003) 'Mendelian randomization': can genetic epidemiology contribute to understanding environmental determinants of disease?. *Int J Epidemiol* **32**, 1–22. doi: 10.1093/ije/dyg070.
57. Sanderson E, Glymour MM, Holmes MV, *et al.* (2022) Mendelian randomization. *Nat Rev Methods Primer* **2**, 6. doi: 10.1038/s43586-021-00092-5.
58. Kim U-K, Jorgenson E, Coon H, *et al.* (2003) Positional cloning of the human quantitative trait locus underlying taste sensitivity to phenylthiocarbamide. *Science* **299**, 1221–1225. doi: 10.1126/science.1080190.
59. Ong JS, Hwang LD, Zhong VW, *et al.* (2018) Understanding the role of bitter taste perception in coffee, tea and alcohol consumption through Mendelian randomization. *Sci Rep* **8**, 16414. doi: 10.1038/s41598-018-34713-z.
60. Sutton KJ, Gervis J, Jatoi M, *et al.* (2025) Dietary intake Mendelian randomization: assessment and development of methods for instrument selection and robust inference. medRxiv [Preprint]. doi: 10.1101/2025.06.26.25330002.
61. Livingstone KM, Ramos-Lopez O, Pérusse L, *et al.* (2022) Precision nutrition: a review of current approaches and future endeavors. *Trends Food Sci Technol* **128**, 253–264. doi: 10.1016/j.tifs.2022.08.017.
62. Tanaka T, Reed D & Ordovas JM (2008) Taste as the gatekeeper of personalized nutrition. In *Personalized Nutrition: Principles and Applications*, pp. 115–132 [F Kok, L Bouwman and F Desiere, editors]. Boca Raton: CRC Press.
63. Calancie L, Keyserling TC, Taillie LS, *et al.* (2018) *TAS2R38* predisposition to bitter taste associated with differential changes in vegetable intake in response to a community-based dietary intervention. *G3 Genes Genomes Genet* **8**, 2107–2119. doi: 10.1534/g3.118.300547.
64. Briazu RA, Bell L, Dodd GF, *et al.* (2024) The effectiveness of personalised food choice advice tailored to an individual's socio-demographic, cognitive characteristics, and sensory preferences. *Appetite* **201**, 107600. doi: 10.1016/j.appet.2024.107600.
65. Sladdin I, Ball L, Bull C, *et al.* (2017) Patient-centred care to improve dietetic practice: an integrative review. *J Hum Nutr Diet* **30**, 453–470. doi: 10.1111/jhn.12444.
66. Parma V, Mainland JD, Hwang LD, *et al.* (2026) FAIR chemosensory data: unlocking AI for flavour, food and health. *Trends Food Sci Technol* **169**, 105545. doi: 10.1016/j.tifs.2026.105545.
67. Martin AR, Kanai M, Kamatani Y, *et al.* (2019) Clinical use of current polygenic risk scores may exacerbate health disparities. *Nat Genet* **51**, 584–591. doi: 10.1038/s41588-019-0379-x.
68. Tucker KL & Noel SE (2025) Cultural considerations in assessing dietary intake in racially, ethnically, or socioeconomically diverse populations. *Annu Rev Nutr* **45**, 299–317. doi: 10.1146/annurev-nutr-111424-112604.

https://doi.org/10.1017/S0029665126105400 Published online by Cambridge University Press