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Clinical Presentation of Chronic Abacterial Prostatitis Shows No Association with TAS2R38 Taster Status

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Keywords
Chronic prostatitis · Chronic pelvic pain syndrome · Microbiology · Taste receptor family 2 isoform 38 protein · Taste receptor family 2

Abstract
Introduction: Chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS) is a relatively common disease and shows an association with urogenital infections. Tuft cells in general have been identified at various entry points into the body (respiratory tract, gastrointestinal tract, and urogenital tract) and are seen as guardians against invading threats. Urethral tuft cells utilizing canonical taste transduction cascade to detect of microbial products and initiating reflex micturition and neurogenic inflammation as a protective mechanism in response. Impaired chemoreception of the T2R38 taste receptor predisposes individuals to upper respiratory tract infections. Therefore, it is very likely that impaired chemoreception has a comparable effect on bacterial urogenital infections, whereas nonbacterial urogenital infections should remain unaffected. The aim of this study was to investigate the influence of TAS2R38 receptor functionality, as measured by a taste test, on the clinical presentation of patients with chronic abacterial prostatitis type III. ***Methods:*** From 2016 to 2025, a total of 252 patients with diagnosed CP/CPPS received a comprehensive andrological work-up including a taste test for the functionality of the TAS2R38 receptor. Complete semen analysis was performed according to WHO 2021 recommendations including the determination of inflammatory parameters in the ejaculate as well as microbiological examination of first-void urine, post-prostate massage urine and ejaculate. ***Results:*** The proportion of tasters was 55.95%, while nontasters accounted for 44.05%. No significant differences could be found between tasters and nontasters with CP/CPPS with regard to symptom burden measured using questionnaires, various ejaculate parameters, prostate-specific antigen (PSA), and microbiological results. Only seminal elastase and serum C-reactive protein (CRP) levels showed a significant difference, but with higher values in the taster group, which, in view of our initial hypothesis, is more likely a statistical coincidence. ***Conclusion:*** The results of our studies show that the taste status of TAS2R38 in patients with

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chronic abacterial prostatitis type III had no association with symptom severity, the ejaculate parameters examined, or the serum levels of PSA and CRP.

Introduction

The immune system uses a variety of chemoreceptors to detect potential pathogens and initiate a targeted defense response against them [1]. In some cases, the immune and taste systems use the same chemoreceptors, in particular the bitter taste receptors from the taste receptor family 2 (T2R) [2]. These are G protein-coupled receptors, which were originally found in the type 2 receptor taste cells of the tongue, but have since been found in numerous other tissues, especially the respiratory tract [2, 3]. Chemosensory tuft cells are seen as key contributors to this process [4–6]. They act as sentinels at entry sites, using canonical taste transduction pathways to detect harmful stimuli and trigger protective responses [5, 7–9].

Recent studies have identified T2Rs as a novel mechanism of innate pathogen recognition in the airways [10–12]. These receptors are expressed in various respiratory cells and regulate innate immune defense processes in both mice and humans [13–16]. Within this family, the T2R isoform 38 protein (T2R38), mainly found in the cilia of the nose and paranasal sinuses, plays a key role in antigen detection [16, 17]. Sensitivity to the bitter compounds phenylthiocarbamide and 6-propyl-2-thiouracil (PTU/PROP) is linked to TAS2R38 and varies between individuals. Three single nucleotide polymorphisms form two common haplotypes, PAV and AVI [18, 19]. The PAV haplotype is associated with tasting phenylthiocarbamide/PTU, while AVI is linked to nontasting, resulting in three genotypes: PAV/PAV ("super taster"), PAV/AVI ("taster"), and AVI/AVI ("non-taster"). Taste strip tests are commonly used to determine this status [20, 21].

TAS2R38 had a significant influence on pathogen detection, the course of an upper respiratory tract infection, and the development of chronic sinusitis [16, 17]. Further studies have shown that a failure of chemorecognition of TAS2R38, were associated with a higher risk of chronic sinusitis and nasal polyps [22–24]. TAS2R38 was particularly involved in the detection of gram-negative bacteria such as *Pseudomonas aeruginosa* by detecting bacterial quorum sensing molecules and other metabolites at mucosal interfaces and modulating the immune response to these pathogens [25, 26].

Further studies have shown increased expression of TAS2R38 in the bladder urothelium and detrusor, suggesting a potential link to recurrent urinary tract infections (UTIs) and overactive bladder symptoms [27, 28]. TAS2R38 in epithelia also contributes to the recognition of *Escherichia coli* and regulation of immune responses via receptor-specific mechanisms [29].

Although comprehensive studies on its role in UTIs are limited, involvement seems likely, particularly given the role of urethral tuft cells in defending against uropathogenic bacteria and regulating urogenital inflammation [26]. Expression of multiple T2R receptors has been identified in cholinergic urethral brush cells, where activation by heat-inactivated uropathogenic *E. coli* triggers the micturition reflex and induces neurogenic inflammation [9, 30, 31].

Chronic prostatitis (CP) is a common disorder with lifetime prevalences ranging from 1.8 to 8.2% [32, 33]. Conditions causing neuropathic pain and those increasing susceptibility to UTIs are considered risk factors [34]. Individuals with factors enabling retrograde bacterial ascent into the urethra and prostate, as well as a history of urethritis due to sexually transmitted infections (STIs), appear more prone to developing CP [34–36]. It is important to distinguish prostatitis from other causes of pelvic pain, such as benign prostate hyperplasia, interstitial cystitis, and other sources of dysuria [37, 38].

The National Institutes of Health (NIH) has divided the condition into four categories: acute bacterial prostatitis (category I), chronic bacterial prostatitis (category II), chronic nonbacterial prostatitis/chronic pelvic pain syndrome (CP/CPPS) (category III), and asymptomatic inflammatory prostatitis (category IV) [38]. Type IIIA, which has inflammatory features in the ejaculate, and type IIIB, which does not, are further divisions of category III [38]. CP/CPPS accounts for about 90% of cases of CP. At least 3 months of prostatic pain without conclusive microbiological results are required [32, 38–40]. It is clinically important to accurately differentiate between type II and type III, as the former necessitates antibiotic treatment while the latter requires nonantibiotic, symptom-focused management [39].

As mentioned, urogenital infections contribute significantly to CP/CPPS pathogenesis [34–36]. The UPOINTS classification system of the European Association of Urology (EAU) enables phenotypic stratification across seven domains (urinary, psychosocial, organ-specific, infection, neurological/systemic, tenderness, sexual) and supports individualized management [41–43]. This study aims to evaluate the association

between TAS2R38 taster status and CP/CPPS, including symptom severity, recurrent UTIs, and microbiological findings.

Materials and Methods

Study Population

As part of our specialized consultation for pelvic pain/CP, this prospective study investigated 360 patients referred to our tertiary university department from December 2016 to October 2025 due to suspected CP.

Prior to inclusion, each patient provided his written informed consent to participate in our study. The Institutional Ethics Committee of Justus-Liebig-University Giessen also granted a positive approval (protocol code 55/13, date of approval: 4 November 2013). Men who did not fulfill the diagnostic criteria for CP/CPPS (n = 108) were excluded from the study population. The study group was made up of 252 individuals with CP/CPPS. The control group consisted of 79 age-matched subjects without a history of current or previous prostate diseases and without recurrent UTIs.

Clinical Investigations

Every participant underwent a comprehensive andrological assessment, which involved a systematic review of their medical history, validated questionnaires for lower urinary tract symptoms (International Prostate Symptom Score, IPSS), erectile dysfunction (International Index of Erectile Function, IIEF), and CP (National Institutes of Health Chronic Prostatitis Symptom Index, NIH-CPSI), a physical examination, analysis of sex hormones, a 2-glass urine test, and further semen analysis [39, 40]. Phenotyping was accomplished by using the categories of UPOINTS system [41]. Ultrasonography was employed to evaluate the volumes of the prostate and testicles, as per clinical recommendations [44, 45]. Irregularities were also documented.

Laboratory Methods

Routine blood draws were performed on every patient to assess serum levels of prostate-specific antigen (PSA), estrogen, testosterone (normal range: 300–1,000 ng/dL), C-reactive protein (CRP), and estradiol. If a lower testosterone level was discovered, the levels of prolactin, sex hormone-binding globulin, albumin, follicle-stimulating hormone, luteinizing hormone, and albumin were measured simultaneously using standard laboratory procedures in the central laboratory of Giessen University Hospital (ADVIA and ADVIA Centaur, Siemens Health Care). Leukocyturia was detected using a urine dipstick and an automated quantitative urine particle analyzer (Cobas u 411, Roche Diagnostics GmbH). The individual conducting the tests was unaware of the source of the material.

Routine Microbiological Tests and Semen Analysis

To rule out a bacterial pathogen as the source of pelvic pain, conventional cultures and PCR tests for sexually transmitted pathogens were conducted on first-void urine, post-prostatic massage urine, and ejaculate samples. Bacteriospermia was defined as a bacterial count exceeding 1,000 colony-forming units (CFUs) per milliliter of ejaculate [46]. In the case of urine samples, a bacterial count of at least 1,000 CFUs per ml was considered significant [47].

A blind analysis of the semen was performed within an hour of collection, following WHO 2010 and WHO 2021 guidelines, with the methodologies for basic semen parameters remaining consistent [48, 49]. After disinfecting the glans and foreskin and adhering to patient instructions, samples were gathered at the clinic by means of masturbation into a sterile container. All patients underwent testing of their urine (first-void urine, urine post-prostatic massage) and semen for STIs (*Mycoplasma genitalium*, *Mycoplasma hominis*, *Ureaplasma urealyticum*, *Ureaplasma parvum*, *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, and *Trichomonas vaginalis*) and received bacterial cultures to exclude the presence of infections [50].

During standard processing, the concentration of leukocytes that tested positive for peroxidase was assessed (Leucoscreen, FertiPro). Furthermore, in each semen sample (Demeditec Diagnostics GmbH), polymorphonuclear elastase, which indicates local inflammation, was quantified in cell-free seminal plasma with an enzyme-linked immunoassay. Concentrations of the inflammatory cytokine interleukin-8 (IL-8) were measured using the cytometric bead array method (BD Biosciences, San Jose, CA, USA). Neutral α-glucosidase and fructose (total enzymatic activity) amounts were measured using spectrophotometric techniques, as described in [51].

Taste Test

The presence of a functional TAS2R38 receptor was determined by a taste strip test. For this test, the subject first received an untreated cellulose strip, which served as a negative control and elicited no taste sensation. The patient then received a strip containing 6-n-propylthiouracil (Eisco Labs, Honeoey Falls, NY, USA), which, as a natural ligand, promptly elicited a bitter taste sensation in the presence of a functional receptor [52, 53]. The division into tasters and nontasters was soley based on taste perception, without any further subdivision in the

Table 1. One-way ANOVA power analysis

	N[a]	Actual power[b]	Power	Effect size[c]	Significance
Overall test[d]	210	0.950	0.95	0.25	0.05

[a]Total sample size across groups. [b]Based on noncentral F-distribution. [c]Cohen's f. [d]Test the null hypothesis that population mean is the same for all groups.

intensity of taste perception. A further distinction of the super-tasters was not performed, as the indication of the intensity of the bitter taste would be too subjective.

Statistical Analysis

The statistical analysis was conducted using SPSS 30 for Windows (IBM GmbH, Ehningen, Germany). An a priori size calculation was performed using a one-way ANOVA power analysis. A p value <0.05 was considered significant. The data were tested for normal distribution using the Kolmogorov-Smirnov test and Quantile-Quantile plot.

Since most data were not normally distributed, the Mann-Whitney U test was applied. Mann-Whitney U test was used to compare patients depending on their taster status with various parameters. A value of $p < 0.05$ was considered statistically significant. Since multiple clinical and laboratory parameters were compared between the groups, the Holm-Bonferroni correction was applied as an adjustment for multiple comparisons.

Multivariate regression modeling was used to examine the association between the results of the taste test and various seminal inflammatory parameters, sperm concentration, CRP and PSA, and the CPSI total score. Only non-missing data were included in the modeling exercise using a forward stepwise process. A value of $p < 0.05$ was considered statistically significant.

Results

A Priori Sample Size Calculation

To calculate a sufficient number of participants to reliably detect relevant differences between tasters and nontasters, an a priori sample size calculation was performed (Table 1). We conducted a one-way ANOVA with a single power of 0.95, a value of 0.25, and a significance level of 0.05. This resulted in a minimum study population size of 210 participants, demonstrating that the study was sufficiently powered.

Demographics

The detailed clinical and demographic findings are shown in Table 2. With a median age of 39, the patients were aged between 20 and 84 years. Type IIIB CP was found in 85.7% of the patients, while 14.3% had type IIIA CP. Among those assessed, the proportion of tasters was 55.95%, while nontasters accounted for 44.05%.

Questionnaires

The study group exhibited a medium level of lower urinary tract symptoms, indicated by a median score of 10 points on the International Prostate Symptom Score (IPSS). The median score for the International Index of Erectile Function (IIEF) was 27 points, falling within the normal range. The Chronic Prostatitis Symptom Index (NIH-CPSI) from the National Health Institute indicated a moderate symptom burden associated with CP, with median scores of 12 points for pain (CPSI-I), 3 points for urinary tract symptoms (CPSI-II), and 9 points for the impact on quality of life (CPSI-III). Unfortunately, not all patients were able to complete the questionnaires due to language barriers. Although a validated questionnaire in the patient's native language was used where available, these were not available for some of our patients (especially Kurdish and Pashto speakers). Additionally, those who did not engage in sexual activity were not able to complete the IIEF-5 questionnaire in a meaningful way. Missing data did not lead to exclusion from the study.

Phenotyping according to UPOINTS

Among our study population, the organ-specific domain was affected most frequently (96.42%), followed by the urinary (78.17%) and sexual (57.93%) domains. In contrast, recurrent urogenital infections were identified in only a minority of patients (4.76%). The median number of impacted domains was three from a total of seven.

Andrological Results

The mean testicular volume measured was 15.0 mL, which falls within the normal range [44]. The median prostate volume, which was 21.0 mL [45], is similarly true.

The laboratory parameters were within normal limits, with median values of total testosterone at 436 ng/dL, PSA at 0.69 ng/mL, estradiol at 30 pg/mL, and C-reactive protein (CRP) at 0.5 mg/L.

Table 2. Demographic, andrological findings, taster status, and affected UPOINT(S) domains of the study population

Parameter	Median (IQR) or *n* (%)	Number of patients
Age, years	39 (31–48)	252
Type of prostatitis		252
Type IIIA	14.3%	36
Type IIIB	85.7%	216
IPSS (points)	10 (6–17)	229
IIEF (points)	27 (21–29)	156
CPSI-I (points)	12 (9–14)	236
CPSI-II (points)	3 (2–6)	236
CPSI-III (points)	9 (7–11)	236
CPSI total score (points)	24 (18–30)	236
Total testosterone, ng/dL	436 (335–558)	249
PSA, ng/mL	0.69 (0.47–1.07)	248
Estradiol, pg/mL	30 (25–37)	249
CRP, mg/L	0.5 (0.5–1.8)	250
Testicular volume, mL	15.0 (12–18)	250
Prostate volume, mL	21.0 (17–27)	251
Taster status		
Taster	55.95%	141
Nontaster	44.05%	111
UPOINT(s)		252
Urinary (U)	78.17%	197
Psychosocial (P)	34.92%	88
Organ specific (O)	96.42%	243
Infection (I)	4.76%	12
Neurological (N)	23.41%	59
Tenderness (T)	19.44%	49
Sexual (S)	57.93%	146
Number of positive domains	3 (2–4)	252

IQR, interquartile range.

Table 3 presents the WHO lower reference limits for the basic semen variables alongside the semen parameters of the patients. The study population showed no signs of inflammatory processes, as demonstrated by the median values of all evaluated semen parameters within the cohort falling within the normal range, especially for the seminal markers of inflammation IL-8, elastase, and peroxidase-positive leukocytes. Due to the fact that the ejaculate volume was sometimes insufficient, not every parameter could be assessed in every case.

Microbiology

Each sample type (first-void urine, post-prostatic massage urine, ejaculate) underwent microbiological analysis using conventional culture and STI-PCR. There were only $n = 4$ patients with a positive STI in our study population. In 2 patients, *Ureaplasma urealyticum* and *Mycoplasma hominis* were detected simultaneously, while a co-infection with *Mycoplasma hominis* and *Mycoplasma genitalium* was detected once. All STI-positive patients received appropriate antibiotic therapy with subsequent eradication testing via STI-PCR and a follow-up visit after 6–12 months. In all affected patients, the symptoms of CP persisted despite successful pathogen eradication, ultimately leading to a diagnosis of type III prostatitis.

Online supplementary Table S1A presents the pathogens identified in the first-void urine, categorized by microbiological culture and STI-PCR. In our study cohort, which had undergone extensive antibiotic pretreatment, the conventional culture yielded positive results for only 51 patients. The identified pathogens

Table 3. Semen parameters of the study population compared with WHO 2021 reference values [49]

Parameter	Patients with CP/CPPS (n = 228)	WHO 2021 reference values	Number of patients
Volume	2.3 (1.2–3.5)	1.4[a]	231
pH value	7.7 (7.5–8.0)	≥**7.2**[b]	231
Sperm concentration (10^6/mL)	52.7 (23.9–112.9)	**16**[a]	230
Total sperm count (10^6/ejaculate)	117.5 (33.2–278.4)	39[1]	230
Progressive motility, %	51 (36–56)	30[a]	212
Sperm vitality, %	63 (64–76)	58[a]	63
Normal forms, %	11 (7–15)	4[1]	212
α-glucosidase, mU/ejaculate	45.5 (20.6–72.0)	≥**20/ejaculate**[b]	223
Fructose, µmol/ejaculate	25.8 (8.7–46.2)	≥**13/ejaculate**[b]	221
Zinc, µmol/ejaculate	6.7 (3.0–15.3)	≥**2.4/ejaculate**[b]	107
Peroxidase-positive leukocytes (10^6/mL)	0.2 (0–0.6)	<1[b]	228
Elastase, ng/mL	37.5 (12–117)	<250[c]	226
IL-8, pg/mL	3,770.0 (2,031–6,774)	<100,00[c]	214

[a]Lower reference limit based on 5th percentile. [b]Consensus-based reference values. [c]Threshold levels established in the Giessen Andrology laboratory.

were primarily either contaminations or constituents of the normal flora, exhibiting a predominantly low bacterial count ranging from 100 to 1,000 CFU per milliliter.

In the same way, online supplementary Table S1B presents the microbiological findings from urine collected after prostatic massage. The same patients showed a positive STI-PCR as in the first-void urine analysis, with two STI pathogens detected simultaneously in 1 patient.

The results for the ejaculate are shown in online supplementary Table 1C. Again, only very few pathogens were identified by culture (n = 49) and STI-PCR (n = 4). This time, in 3 patients with positive STI-PCR double infections could be detected: *Mycoplasma hominis* and *Ureaplasma urealtyicum* were simultaneously detected twice, *Mycoplasma hominis* and *Mycoplasma hominis* once.

Comparison Taster and Nontaster

Table 4 compares the clinical characteristics between tasters and nontasters. To test for significant differences, both groups were compared using the Mann-Whitney U test; a $p < 0.05$ was considered significant.

No significant differences were found between the two groups with regard to age, blood biochemistry, questionnaire results, or most ejaculate parameters. Only seminal elastase and serum levels of CRP showed a significant difference, with elevated levels in the tasters. After applying the Holm-Bonferroni correction, no result remains significant ($\alpha = 0.05$). The initially found significant differences therefore should be considered a type I error. This also seems plausible, as higher values for both parameters were found in the tasters, which would contradict the initial hypothesis.

To compare the microbiological results, patients with at least one positive culture and at least one positive STI-PCR in one of the three sample types (first-void urine, post-prostate massage urine, ejaculate) were compared. Again, no statistically significant differences were found between the two groups.

Table 5 shows a univariate and multivariate analysis between taster status and sperm concentration as an important fertility marker, the parameters of seminal inflammation (leukocytes in ejaculate, levels of elastase and IL-8), the CPSI total score as a marker for symptom burden, microbiological results and the PSA and CRP levels in serum. A positive microbiological culture in at least one of the three sample types (first-void urine, post-void urine, ejaculate) again leads to classification in the "positive culture" group. The same procedure was followed in the case of a positive STI test. PSA and CRP were chosen as surrogate parameters for

Table 4. Comparison of clinical characteristics between tasters and nontasters

Parameter	Taster (n = 141)	Nontaster (n = 111)	Number of patients (Taster/Nontaster)	Reference value	p value[a]	Holm-adjusted p values
Age, years	39 (31–47)	39 (31–52)	141/111	n.a.	0.371	1.000
Patients with type IIIA CP	20	16		n.a.	0.731	1.000
Affected domains	3 (2–4)	3 (2–4)	141/111	n.a.	0.670	1.000
IPSS score	10 (6–17)	11 (6–15)	130/99	n.a.	0.672	1.000
CPSI total score	24 (17–29)	24 (18–30)	135/101	n.a.	0.217	1.000
IIEF score	27 (22–29)	27 (21–29)	90/66	n.a.	0.861	1.000
PSA, ng/mL	0.69 (0.49–1.15)	0.68 (0.46–0.99)	139/109	<4.0	0.401	1.000
CRP, mg/L	0.69 (0.5–2.2)	0.5 (0.5–1.2)	140/110	<0.5	0.011*	0.176
Sperm concentration (10^6/mL)	50.3 (23.6–107.5)	56.3 (24.0–113.3)	135/95	16[b]	0.637	1.000
Total sperm count (10^6/ejaculate)	121.9 (42.0–304.9)	103.2 (30.2–250.0)	135/95	39[b]	0.404	1.000
Progressive motility, %	51 (39–56)	50 (34–56)	117/95	30[b]	0.639	1.000
Peroxidase-positive leukocytes (10^6/mL)	0.2 (0–0.6)	0.1 (0–0.3)	133/95	<1[c]	0.537	1.000
Elastase, ng/mL	50 (16–139)	28 (10–85)	131/95	<250[d]	0.040*	0.600
IL-8, pg/mL	3,770 (2241–7,141)	3,665 (1,834–6,618)	125/89	<100,00[d]	0.467	1.000
Positive culture	50	45	134/105	n.a.	0.510	1.000
Positive STI-PCR	2	2	134/105	n.a.	0.658	1.000

n.a., not applicable. [a]Mann-Whitney U test comparing tasters and nontasters with CP type IIIA and IIIB. [b]Lower reference limit based on 5th percentile according to WHO 2021 Manual. [c]Consensus-based reference values. [d]Threshold levels established in the Giessen Andrology laboratory. *$p < 0.05$.

prostatic inflammation. There was no significant association with any of the parameters examined, except for seminal plasma elastase and serum CRP levels in the univariate analysis. A $p < 0.05$ was considered statistically significant.

Comparison between Patients with Type III Prostatitis and the Control Group

Table 6 compares the parameters collected from patients with CP to those of the control group. The Mann-Whitney U test was used to test for significant differences; a p value <0.05 was considered significant. As expected, a significant difference was found in the scores of the IPSS and CPSI questionnaires, with higher scores in the prostatitis group. No significant differences were found regarding the other biochemical and seminal parameters. In particular, the percentage of nontasters based on the TAS2R38 was almost identical in both groups.

Discussion

As a continuation of our previous publications [54], our study is the first to investigate the role of TAS2R38 receptor functionality in the clinical presentation of CP NIH type III. Previous studies have shown that nontaster status or lack of chemoreception of the receptor is associated with a higher risk of upper respiratory tract infections and the development of chronic sinusitis [16, 17, 22, 23]. Evidence indicates that the TAS2R38 receptor is particularly involved in the detection of *Pseudomonas aeruginosa* and thus performs a function in the antigen recognition of the innate immune system [25, 26]. Another study was also able to demonstrate the chemodetection of *Escherichia coli* by the receptor, which makes it interesting for a potential role in UTIs [31]. At mucosal interfaces in the digestive system and the urogenital tract, it has been demonstrated that various bitter receptors of the TAS2R family can detect

Table 5. Association of taster status (n = 239) with CPSI total score, sperm concentration, inflammatory seminal parameters, PSA, and CRP

	Correlation coefficient r	p value (univariate)	Confidence interval (95%)	Correlation coefficient ß	p value (multivariate)	Confidence interval (95%)
CPSI total score	0.080	0.281	−0.047 to 0.205	0.100	0.147	−0.027 to 0.224
Sperm concentration	−0.028	0.674	−0.154 to 0.099	−0.034	0.621	−0.160 to 0.093
Leukocytes in ejaculate	0.120	0.070	−0.007 to 0.243	−0.019	0.780	−0.146 to 0.108
Seminal plasma elastase	0.137	0.040*	0.010 to 0.259	−0.044	0.542	−0.170 to 0.083
Seminal plasma IL-8	0.050	0.468	−0.077 to 0.176	0.041	0.575	−0.086 to 0.167
PSA, ng/mL	0.053	0.402	−0.074 to 0.179	0.032	0.640	−0.095 to 0.158
CRP, mg/L	0.161	0.011*	0.035 to 0.282	0.111	0.107	−0.016 to 0.235
Positive culture	0.045	0.512	−0.082 to 0.171	0.037	0.588	−0.090 to 0.163
Positive STI-PCR	0.045	0.500	−0.082 to 0.171	0.059	0.395	−0.068 to 0.185

Multivariate analysis: univariate and multivariate regression analysis between taster status and CPSI, seminal parameters, CRP and PSA, and microbiological results, n = 239.

Table 6. Comparison between patients with type III prostatitis and the control group

Parameter	Patients with CP/CPPS (n = 252)	Control group (n = 79)	p value[a]
Age	39 (31–48)	41 (31–52)	0.206
IPSS (points)	10 (6–17)	3 (0–5)	<0.001*
IIEF (points)	27 (21–29)	26 (19–30)	0.771
CPSI total score (points)	24 (18–30)	8 (7–10)	<0.001*
Total testosterone, ng/dL	436 (355–558)	497 (384–597)	0.089
PSA, ng/mL	0.69 (0.47–1.07)	0.72 (0.52–1.23)	0.303
CRP, mg/L	0.5 (0.5–1.8)	0.53 (0.5–1.66)	0.626
Nontaster	44.05%	43.03%	0.968
Sperm concentration (10^6/mL)	52.7 (23.9–112.9)	65.3 (29.4–163.9)	0.082
Total sperm count (10^6/ejaculate)	117.5 (33.2–278.4)	126.7 (54.9–394.7)	0.304
Progressive motility, %	51 (36–56)	53 (38–59)	0.558
Normal forms, %	11 (7–15)	11 (5–16)	0.592
Peroxidase-positive leukocytes (10^6/mL)	0.2 (0–0.6)	0.3 (0–0.8)	0.065
Elastase, ng/mL	37.5 (12–117)	46 (23–128)	0.226
IL-8, pg/mL	3,770.0 (2031–6,774)	3.934 (1,847–7,404)	0.314
Positive culture	37.69%	34.27%	0.568

[a]Mann-Whitney U test comparing tasters and nontasters with CP type IIIA and IIIB. *$p < 0.05$.

quorum sensing molecule and other bacterial metabolites and lead to activation of the innate immune system [25, 26]. UTIs are considered a risk factor for the development of chronic abacterial prostatitis [34], and thus a connection with the functionality of the TAS2R38 receptor is conceivable. Increased expression of TAS2R38 in the urothelium and detrusor muscle was also detected, which, in addition to activation of the immune system and local inflammation, also provides direct protection against the penetration of pathogens into the urogenital system by triggering the micturition reflex [26–28]. However, assuming that the TAS2R38-mediated defense reactions are associated with urethral tuft cells, it is rather unlikely that abacterial processes are influenced by the taster status.

In our study population, 44% of the subjects in the prostatitis group were nontasters and 43% in the control group; this essentially corresponds to the expected distribution between tasters and nontasters in a Western European cohort. Other studies have found a similar distribution in the general population of European populations, with nontaster rates varying between 24.8 and 53.5% [55–57].

Based on our study data, we could not detect an association between the functionality of the TAS2R38 receptor and the clinical presentation of CP type III. Neither the symptom burden of the questionnaires used, nor the examined ejaculate parameters and PSA values, nor the results of the microbiology showed a significant difference between tasters and nontasters. Only seminal elastase and serum CRP levels showed a significant difference between the two groups, but with higher values in the taster group, which, in view of our initial hypothesis, is more likely a statistical coincidence. After Holm-adjustment of the p values, no corresponding significance could be demonstrated, suggesting a type I error. Also, no significant differences in biochemistry and seminal parameters were found between the prostatitis group and the control group, except for the IPSS and CPSI scores. Univariate analysis only showed a difference between tasters and nontasters for seminal elastase and serum CRP levels; however, this difference was no longer detectable in multivariate analysis. Taken together, this provides little evidence of a link between the functionality of the taste receptor and the clinical presentation of chronic abacterial prostatitis.

A prominent feature of our population is the low rate of positive conventional bacterial culture and STI-PCR results in the samples examined: the STI-PCR showed no pathogen detection in 98% of all three sample types (first-void urine, post-prostate massage urine, ejaculate), and the classical culture also showed no pathogen growth in 79–87%. If a bacterium was detected, it was usually due to contamination with the skin or urethral flora. Only very rarely was a potentially uropathogenic bacterium such as *Escherichia coli* or *Klebsiella oxytoca* detected, and then only in low numbers of around 1,000 bacteria/mL, so that even these detections were considered insignificant. One explanation for this could be the frequent prior treatment by the referring physician. Although not systematically recorded by us, the vast majority of patients had received antibiotic therapy in their history, so that an initial link to a urogenital infection was no longer detectable at the time of presentation. This factor could have had a significant impact on the detection rate of a pathogen and represents a clear limitation of our study in the interpretation of the microbiological results. Another limitation stems from the fact that we did not test for all potential sexually transmitted pathogens. Our panel deliberately focused on pathogens that are readily detectable in urine and ejaculate and are described in the literature as typical STIs associated with chronic bacterial prostatitis [33–35]. Testing for other sexually transmitted pathogens, such as syphilis or the human immunodeficiency virus, was not included. Based on the UPOINTS classification, only 4.76% of the subjects showed an affected infectious domain, indicating that recurrent UTIs play only a minor role in the cohort, at least with regard to phenotyping.

A clear limitation of our study is the single-center data collection and the lack of systematic recording of prior antibiotic treatment. Furthermore, testing for the functionality of the TAS2R38 receptor was only based on the taste test and not via genetic testing. While the taste test is very good at detecting a complete loss of chemoreception [52], it cannot objectively distinguish between reduced sensitivity and normal function. This could lead to differences in the corresponding subgroups that could not be investigated using this method. While the division into tasters and nontasters is a pragmatic approach and easy to apply in practice, it does also not allow for a distinction between heterozygous and homozygous genotypes. This lack of differentiation could prevent the detection of subtle genotype-phenotype associations, potentially influencing the negative study result. Follow-up studies addressing the same research question could employ genetic testing.

The aim of our study was to investigate whether abacterial prostatitis is associated with the taster status; therefore, our study cohort consisted exclusively of patients with chronic type III prostatitis.

While an acute urogenital infection can be identified in the medical history of many patients with this condition as the initial onset of pelvic pain, by definition no relevant pathogens are detected during the course of the disease. Since abacterial prostatitis is promoted by many different factors (UPOINTS), it is unlikely to depend an acute urogenital infection alone. In our opinion, however, only bacterial UTIs may be influenced by TAS2R38, possibly through urethral tuft cells. Therefore, it is not surprising that we found no correlation between taster status and abacterial prostatitis. From a clinical perspective, routine testing for receptor functionality cannot be recommended.

However, in a population with chronic bacterial prostatitis type II, a corresponding correlation is much more likely in future studies in which a relevant pathogen is found when the patient is presented. For such patient population, a repeat study with a similar design would be worthwhile in the future, possibly expanding the testing beyond the taste test to include human genetic analysis to determine the genotype. As mentioned above, recruiting a suitable cohort from the patients referred to us was not possible, presumably due to the extensive antibiotic pretreatment by the referring physicians, as hardly any cases of chronic bacterial prostatitis were found. Similarly, the connection we suspect between the functioning chemoreception of the TAS2R38 receptor and UTIs, analogous to respiratory tract infections, is plausible but has not yet been confirmed by several independent studies.

Conclusions

The results of our studies show that the taste status of TAS2R38 in patients with chronic abacterial prostatitis type III had no association with symptom severity, the ejaculate parameters examined, or the serum levels of PSA and CRP.

Statement of Ethics

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Institutional Ethics Committee of Justus-Liebig-University Giessen (protocol code 55/13, date of approval: 4 November 2013). Written informed consent was obtained from all subjects involved in the study.

Conflict of Interest Statement

Florian M. Wagenlehner was a member of the journal's Editorial Board at the time of submission. The remaining authors have no conflicts of interest.

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Author Contributions

Jens Rosellen: formal analysis, investigation, data curation, and writing – original draft. Adrian Pilatz: data curation, investigation, and writing – review and editing. Hans-Christian Schuppe and Undraga Schagdarsurengin: investigation and writing – review and editing. Lea Hofmann: conceptualization, formal analysis, investigation, writing – original draft, and visualization. Klaus Deckmann: conceptualization, formal analysis, investigation, data curation, writing – original draft, visualization, and project administration. Florian Wagenlehner: conceptualization, writing – review and editing, and supervision. All authors have read and agreed to the published version of the manuscript.

Data Availability Statement

The data that support the findings of this study are not publicly available due to their containing information that could compromise the privacy of research participants but are available from the corresponding author [J.R.] (jens.rosellen@uk-gm.de) upon request.

References

1 Bedford FL. The missing sense modality: the immune system. Perception. 2011;40(10):1265–7. https://doi.org/10.1068/p7119

2 Yamamoto K, Ishimaru Y. Oral and extra-oral taste perception. Semin Cell Dev Biol. 2013;24(3):240–6. https://doi.org/10.1016/j.semcdb.2012.08.005

3 Mennella JA, Spector AC, Reed DR, Coldwell SE. The bad taste of medicines: overview of basic research on bitter taste. Clin Ther. 2013;35(8):1225–46. https://doi.org/10.1016/j.clinthera.2013.06.007

4 Kaske S, Krasteva G, König P, Kummer W, Hofmann T, Gudermann T, et al. TRPM5, a taste-signaling transient receptor potential ion-channel, is a ubiquitous signaling component in chemosensory cells. BMC Neurosci. 2007;8(1):49. https://doi.org/10.1186/1471-2202-8-49

5 Krasteva G, Canning BJ, Papadakis T, Kummer W. Cholinergic brush cells in the trachea mediate respiratory responses to quorum sensing molecules. Life Sci. 2012;91(21–22):992–6. https://doi.org/10.1016/j.lfs.2012.06.014

6 Ogura T, Krosnowski K, Zhang L, Bekkerman M, Lin W. Chemoreception regulates chemical access to mouse vomeronasal organ: role of solitary chemosensory cells. PLoS One. 2010;5(7):e11924. https://doi.org/10.1371/journal.pone.0011924

7 Schütz B, Ruppert AL, Strobel O, Lazarus M, Urade Y, Büchler MW, et al. Distribution pattern and molecular signature of cholinergic tuft cells in human gastro-intestinal and pancreatic-biliary tract. Sci Rep. 2019;9(1):17466. https://doi.org/10.1038/s41598-019-53997-3
8 Zheng X, Tizzano M, Redding K, He J, Peng X, Jiang P, et al. Gingival solitary chemosensory cells are immune sentinels for periodontitis. Nat Commun. 2019;10(1):4496. https://doi.org/10.1038/s41467-019-12505-x
9 Deckmann K, Filipski K, Krasteva-Christ G, Fronius M, Althaus M, Rafiq A, et al. Bitter triggers acetylcholine release from polymodal urethral chemosensory cells and bladder reflexes. Proc Natl Acad Sci U S A. 2014;111(22):8287–92. https://doi.org/10.1073/pnas.1402436111
10 Finger TE, Bottger B, Hansen A, Anderson KT, Alimohammadi H, Silver WL. Solitary chemoreceptor cells in the nasal cavity serve as sentinels of respiration. Proc Natl Acad Sci U S A. 2003;100(15):8981–6. https://doi.org/10.1073/pnas.1531172100
11 Gulbransen BD, Clapp TR, Finger TE, Kinnamon SC. Nasal solitary chemoreceptor cell responses to bitter and trigeminal stimulants in vitro. J Neurophysiol. 2008;99(6):2929–37. https://doi.org/10.1152/jn.00066.2008
12 Saunders CJ, Christensen M, Finger TE, Tizzano M. Cholinergic neurotransmission links solitary chemosensory cells to nasal inflammation. Proc Natl Acad Sci U S A. 2014;111(16):6075–80. https://doi.org/10.1073/pnas.1402251111
13 Tizzano M, Cristofoletti M, Sbarbati A, Finger TE. Expression of taste receptors in solitary chemosensory cells of rodent airways. BMC Pulm Med. 2011;11(1):3. https://doi.org/10.1186/1471-2466-11-3
14 Tizzano M, Gulbransen BD, Vandenbeuch A, Clapp TR, Herman JP, Sibhatu HM, et al. Nasal chemosensory cells use bitter taste signaling to detect irritants and bacterial signals. Proc Natl Acad Sci U S A. 2010;107(7):3210–5. https://doi.org/10.1073/pnas.0911934107
15 Lee RJ, Kofonow JM, Rosen PL, Siebert AP, Chen B, Doghramji L, et al. Bitter and sweet taste receptors regulate human upper respiratory innate immunity. J Clin Invest. 2014;124(3):1393–405. https://doi.org/10.1172/JCI72094
16 Lee RJ, Xiong G, Kofonow JM, Chen B, Lysenko A, Jiang P, et al. T2R38 taste receptor polymorphisms underlie susceptibility to upper respiratory infection. J Clin Invest. 2012;122(11):4145–59. https://doi.org/10.1172/JCI64240
17 Lee RJ, Cohen NA. Role of the bitter taste receptor T2R38 in upper respiratory infection and chronic rhinosinusitis. Curr Opin Allergy Clin Immunol. 2015;15(1):14–20. https://doi.org/10.1097/ACI.0000000000000120
18 Kim UK, Jorgenson E, Coon H, Leppert M, Risch N, Drayna D. Positional cloning of the human quantitative trait locus underlying taste sensitivity to phenylthiocarbamide. Science. 2003;299(5610):1221–5. https://doi.org/10.1126/science.1080190
19 Bufe B, Breslin PA, Kuhn C, Reed DR, Tharp CD, Slack JP, et al. The molecular basis of individual differences in phenylthiocarbamide and propylthiouracil bitterness perception. Curr Biol. 2005;15(4):322–7. https://doi.org/10.1016/j.cub.2005.01.047
20 Mathew SR, P SS, Rao ES, Rizwana, Agarwal A. Phenylthiocarbamide (PTC) taste perception: a study correlating the sensivity to bitter taste and the influence of various demographic and psychographic parameters. J Appl Biol Sci. 2023;17(3):407–16. https://doi.org/10.71336/jabs.1203
21 Risso D, Carmagnola D, Morini G, Pellegrini G, Canciani E, Antinucci M, et al. Distribution of TAS2R38 bitter taste receptor phenotype and haplotypes among COVID-19 patients. Sci Rep. 2022;12(1):7381. https://doi.org/10.1038/s41598-022-10747-2
22 Dżaman K, Piskadło-Zborowska K, Czerwaty K, Jowik R, Stachowiak M, Sarnowska E. The impact of the bitter taste receptor on the predisposition to chronic rhinosinusitis. Acta Otorhinolaryngol Ital. 2025;45(2):116–23. https://doi.org/10.14639/0392-100X-N3032
23 Zborowska-Piskadło K, Stachowiak M, Rusetska N, Sarnowska E, Siedlecki J, Dżaman K. The expression of bitter taste receptor TAS2R38 in patients with chronic rhinosinusitis. Arch Immunol Ther Exp. 2020;68(5):26. https://doi.org/10.1007/s00005-020-00593-3
24 Yilmaz G, Eyigor H, Gur OE, Kalkan T, Gur N, Selcuk OT, et al. The role of TAS2R38 genotype in surgical outcomes and culturable bacteria in chronic rhinosinusitis with or without nasal polyps. Rhin. 2022;0(0):0–60. https://doi.org/10.4193/Rhin22.118
25 Pifferi M, Boner A, Maj D, Michelucci A, Donzelli G, Cangiotti AM, et al. Impact of TAS2R38 polymorphisms on nasal nitric oxide and *Pseudomonas* infections in primary ciliary dyskinesia: relation to genotype. Thorax. 2024;79(11):1069–76. https://doi.org/10.1136/thorax-2024-221396
26 Rudolph E, Dychtenberg H, Pozniak A, Pundir P. Bitter taste receptors in bacterial infections and innate immunity. Immun Inflamm Dis. 2025;13(7):e70232. https://doi.org/10.1002/iid3.70232
27 Ulett GC, Totsika M, Schaale K, Carey AJ, Sweet MJ, Schembri MA. Uropathogenic Escherichia coli virulence and innate immune responses during urinary tract infection. Curr Opin Microbiol. 2013;16(1):100–7. https://doi.org/10.1016/j.mib.2013.01.005
28 Schmidt P, Pfeil U, Lafee M, Petersen S, Perniss A, Keshavarz M, et al. Tuft cells trigger neurogenic inflammation in the urethra. Cell Rep. 2025;44(10):116370. https://doi.org/10.1016/j.celrep.2025.116370
29 Smith TA, Moore BN, Matoso A, Berkowitz DE, DeBerry JJ, Pluznick JL. Identification of novel bladder sensory GPCRs. Physiol Rep. 2021;9(8):e14840. https://doi.org/10.14814/phy2.14840
30 Zhai K, Yang Z, Zhu X, Nyirimigabo E, Mi Y, Wang Y, et al. Activation of bitter taste receptors (tas2rs) relaxes detrusor smooth muscle and suppresses overactive bladder symptoms. Oncotarget. 2016;7(16):21156–67. https://doi.org/10.18632/oncotarget.8549
31 Liszt KI, Wang Q, Farhadipour M, Segers A, Thijs T, Nys L, et al. Human intestinal bitter taste receptors regulate innate immune responses and metabolic regulators in obesity. J Clin Invest. 2022;132(3):e144828. https://doi.org/10.1172/JCI144828
32 Suskind AM, Berry SH, Ewing BA, Elliott MN, Suttorp MJ, Clemens JQ. The prevalence and overlap of interstitial cystitis/bladder pain syndrome and chronic prostatitis/chronic pelvic pain syndrome in men: results of the RAND Interstitial Cystitis Epidemiology male study. J Urol. 2013;189(1):141–5. https://doi.org/10.1016/j.juro.2012.08.088
33 Krieger JN, Lee SW, Jeon J, Cheah PY, Liong ML, Riley DE. Epidemiology of prostatitis. Int J Antimicrob Agents. 2008;31(Suppl 1):S85–S90. https://doi.org/10.1016/j.ijantimicag.2007.08.028
34 Sharp VJ, Takacs EB, Powell CR. Prostatitis: diagnosis and treatment. Am Fam Physician. 2010;82(4):397–406.
35 Nickel JC, Alexander RB, Schaeffer AJ, Landis JR, Knauss JS, Propert KJ; Chronic Prostatitis Collaborative Research Network Study Group. Leukocytes and bacteria in men with chronic prostatitis/chronic pelvic pain syndrome compared to asymptomatic controls. J Urol. 2003;170(3):818–22. https://doi.org/10.1097/01.ju.0000082252.49374.e9
36 Nickel JC, Ardern D, Downey J. Cytologic evaluation of urine is important in evaluation of chronic prostatitis. Urology. 2002;60(2):225–7. https://doi.org/10.1016/s0090-4295(02)01733-8
37 Rees J, Abrahams M, Doble A, Cooper A; Prostatitis Expert Reference Group PERG. Diagnosis and treatment of chronic bacterial prostatitis and chronic prostatitis/chronic pelvic pain syndrome: a consensus guideline. BJU Int. 2015;116(4):509–25. https://doi.org/10.1111/bju.13101
38 Krieger JN, Nyberg L, Nickel JC. NIH consensus definition and classification of prostatitis. JAMA. 1999;282(3):236–7. https://doi.org/10.1001/jama.282.3.236
39 Nickel JC, Shoskes D, Wang Y, Alexander RB, Fowler JE, Zeitlin S, et al. How does the prostate massage and post-massage 2-glass test compare to the Meares-Stamey 4-glass test in men with chronic prostatitis/chronic pelvic pain syndrome? J Urol. 2006;176(1):119–24. https://doi.org/10.1016/s0022-5347(06)00498-8

40 Domes T, Lo KC, Grober ED, Mullen JB, Mazzulli T, Jarvi K. The incidence and effect of bacteriospermia and elevated seminal leukocytes on semen parameters. Fertil Steril. 2012;97(5):1050–5. https://doi.org/10.1016/j.fertnstert.2012.01.124
41 Fall M, Baranowski AP, Elneil S, Engeler D, Hughes J, Messelink EJ, et al. EAU guidelines on chronic pelvic pain. Eur Urol. 2010;57(1):35–48. https://doi.org/10.1016/j.eururo.2009.08.020
42 Magri V, Wagenlehner F, Perletti G, Schneider S, Marras E, Naber KG, et al. Use of the UPOINT chronic prostatitis/chronic pelvic pain syndrome classification in European patient cohorts: sexual function domain improves correlations. J Urol. 2010;184(6):2339–45. https://doi.org/10.1016/j.juro.2010.08.025
43 Cai T, Alidjanov J, Palagin I, Medina-Polo J, Nickel JC, Wagenlehner FME. Chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS): look to the future. Prostate Cancer Prostatic Dis. 2024;27(2):239–41. https://doi.org/10.1038/s41391-023-00645-7
44 Tsili AC, Bougia CK, Pappa O, Argyropoulou MI. Ultrasonography of the scrotum: revisiting a classic technique. Eur J Radiol. 2021;145:110000. https://doi.org/10.1016/j.ejrad.2021.110000
45 Lotti F, Frizza F, Balercia G, Barbonetti A, Behre HM, Calogero AE, et al. The European Academy of Andrology (EAA) ultrasound study on healthy, fertile men: Prostate-vesicular transrectal ultrasound reference ranges and associations with clinical, seminal and biochemical characteristics. Andrology. 2022;10(6):1150–71. https://doi.org/10.1111/andr.13217
46 Rana N, Vaid RK, Phulia SK, Singh P. Assessment of bacterial diversity in fresh bubaline semen. Indian J Anim Sci. 2012;82(6):596–8.
47 Kocer D, Sarıguzel FM, Karakukcu C. Cutoff values for bacteria and leukocytes for urine sediment analyzer FUS200 in culture-positive urinary-tract infections. Scand J Clin Lab Invest. 2014;74(5):414–7. https://doi.org/10.3109/00365513.2014.900189
48 World Health Organization. WHO laboratory manual for the examination and processing of human semen. 5th ed. Geneva, Switzerland: WHO; 2010.
49 World Health Organization. WHO laboratory manual for the examination and processing of human semen. 6th ed. Geneva, Switzerland: WHO; 2021.
50 Henkel R, Solomon M. Semen culture and the assessment of genitourinary tract infections. Indian J Urol. 2017;33(3):188–93. https://doi.org/10.4103/iju.iju_407_16
51 Qiu Z, Chu Q, Zhang W, Luo C, Quan S. Level of neutral alpha-1,4-glucosidase in seminal plasma of Chinese men. Andrologia. 2018;50(3):e12948. https://doi.org/10.1111/and.12948
52 Cossu G, Melis M, Sarchioto M, Melis M, Melis M, Morelli M, et al. 6-n-propylthiouracil taste disruption and TAS2R38 nontasting form in Parkinson's disease. Mov Disord. 2018;33(8):1331–9. https://doi.org/10.1002/mds.27391
53 Chupeerach C, Tapanee P, On-Nom N, Temviriyanukul P, Chantong B, Reeder N, et al. The influence of TAS2R38 bitter taste gene polymorphisms on obesity risk in three racially diverse groups. Biomedicine. 2021;11(3):43–9. https://doi.org/10.37796/2211-8039.1175
54 Rosellen J, Dittmar F, Hauptmann A, Diemer T, Schuppe HC, Schagdarsurengin U, et al. Impaired Semen quality in patients with chronic prostatitis. J Clin Med. 2024;13(10):2884. https://doi.org/10.3390/jcm13102884
55 Keller M, Liu X, Wohland T, Rohde K, Gast MT, Stumvoll M, et al. TAS2R38 and its influence on smoking behavior and glucose homeostasis in the German Sorbs. PLoS One. 2013;8(12):e80512. https://doi.org/10.1371/journal.pone.0080512
56 Wooding S, Kim UK, Bamshad MJ, Larsen J, Jorde LB, Drayna D. Natural selection and molecular evolution in PTC, a bitter-taste receptor gene. Am J Hum Genet. 2004;74(4):637–46. https://doi.org/10.1086/383092
57 Guo SW, Reed DR. The genetics of phenylthiocarbamide perception. Ann Hum Biol. 2001;28(2):111–42. https://doi.org/10.1080/03014460151056310