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Review

Clinical-Phenotypic Mapping of Autism Without Intellectual Disability and with Fluent Functional Language: A Heuristic Framework for Multidimensional Clinical Formulation

Leonardo Zoccante [1], Marco Zaffanello [2,*], Mattia Zene [3] and Roberto Keller [4]

[1] Ulss 9 Scaligera Company, 37122 Verona, Italy; leonardo.zoccante@aulss9.veneto.it
[2] Department of Surgery, Dentistry, Gynecology and Pediatrics, University of Verona, 37129 Verona, Italy
[3] Department of Human Sciences, University of Verona, Lungadige Porta Vittoria 17, 37129 Verona, Italy; mattia.zene@univr.it
[4] Adult Autism Centre, Mental Health Department, ASL Città di Torino, 10138 Turin, Italy; roberto.keller@aslcittaditorino.it
* Correspondence: marco.zaffanello@univr.it

Highlights

What are the main findings?

- The review proposes a descriptive multidimensional matrix that maps ten clinical-phenotypic profiles by construct/diagnostic status, function, developmental relevance, and clinical implications, with sensory reactivity treated as cross-cutting.
- The map distinguishes cognitive, communicative, and neuropsychological profiles from co-occurring conditions and adaptive, psychopathological, or identity-related trajectories; it is not a diagnostic classification.

What are the implications of the main findings?

- The framework may help clinicians structure multidimensional case formulation and identify areas requiring further assessment, particularly when fluent language or intellectual ability may obscure adaptive difficulties.
- Its clinical utility and applicability across developmental stages have not been established and require empirical evaluation.

Abstract

This conceptual review proposes a descriptive, heuristic framework for clinical formulation in autism spectrum disorder (ASD) without intellectual disability and with fluent functional language. Based on a purposive, non-systematic literature review, it organises ten selected, heterogeneous profiles into three parallel domains: cognitive, communicative, and neuropsychological functional profiles, co-occurring conditions and diagnostic relationships, and adaptive, psychopathological, or identity-related trajectories and associations. Sensory reactivity is represented as a cross-cutting dimension. The framework's contribution is to make these differing conceptual statuses explicit in one formulation matrix and to prompt inquiry into strengths, adaptive functioning, context, and support needs that may be obscured when fluent language or intellectual ability is treated as evidence of adequate everyday functioning. It introduces no diagnostic criteria, and its clinical utility remains untested. Developmental salience may vary across age and context; applicability across paediatric, adolescent, transitional, and adult care requires empirical evaluation.

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1. Introduction

The condition historically described as Asperger syndrome (AS) is now subsumed under autism spectrum disorder (ASD). In DSM-5 and DSM-5-TR, the previous categories of autistic disorder, Asperger disorder, and pervasive developmental disorder not otherwise specified were merged into a single diagnosis [1]. In ICD-11, the profile closest to the former Asperger diagnosis corresponds to category 6A02.0, defined as "Autism spectrum disorder without disorder of intellectual development and with mild or no impairment of functional language" (World Health Organization, ICD-11 MMS, release 2025-01) [2]. The eponym has also become a matter of debate following archival documentation of Hans Asperger's role in Nazi-era Vienna, which supports a historically informed and context-aware use of the term, and following the Pediatric Research editorial board's recommendation to discontinue its use [3–5]. For these reasons, the term does not appear in the title and is used in the manuscript only in a historical and descriptive sense—including to denote the area of overlap between high-functioning autism and the former Asperger profile (organising domain A4)—not as a separate diagnostic category. The target population comprises autistic people without intellectual disability and with fluent functional language. These characteristics do not, by themselves, determine the DSM-5-TR support level. In this review, "fluent functional language" is a review-specific descriptive term for routine production of connected, spontaneous spoken utterances that can serve everyday communication; no standardised score or cut-off is implied. It describes expressive language only and does not imply intact receptive-language comprehension (e.g., understanding vocabulary, sentence-level meaning, or instructions) or pragmatic competence (e.g., adapting communication to context, conversational reciprocity, and turn-taking, and interpreting non-literal or inferred meaning). These dimensions are considered separately and may remain areas of difficulty [6].

Recognition of this population is influenced by diagnostic bias. Autism is diagnosed more frequently in males, but the traditionally reported male-to-female ratio of 4:1 is likely to be an overestimate: the meta-analysis by Loomes and colleagues placed the true ratio at approximately 3:1, with a male-to-female odds ratio of 3.25 (95% CI 2.93–3.62) in population-screening studies compared with 4.56 (95% CI 4.10–5.07) in studies based on already established diagnoses [7,8]. The difference between these estimates is consistent with diagnostic bias. Possible contributors include less immediately identifiable presentations, supported by social compensation strategies such as masking or camouflaging, with interests that are more socially contextualised and, therefore, less readily identified as atypical [9–11].

A major clinical concern in this population is the underestimation of support needs when fluent language or intellectual ability is taken as evidence of adequate adaptive functioning [12]. This risk extends across the intellectual range: borderline intellectual functioning may go unrecognised because fluent language conceals it, and higher intellectual functioning may sustain more effective compensation and further delay recognition. This may lead to late recognition, environmental demands calibrated to apparent competence, and a psychopathological burden that services often identify before the underlying condition itself: anxiety, depression, eating disorders, social withdrawal, and fatigue [13].

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Clinical formulation is further complicated by the heterogeneity of the presentation, which may include executive difficulties, psychopathological vulnerability, co-occurring neurodevelopmental conditions, food selectivity, social fatigue, and other adaptive difficulties. A purely categorical reading, such as a simple distinction between "mild" and "severe" forms, does not account for this variability [14,15]. The literature has approached this heterogeneity through data-derived subgrouping and transdiagnostic dimensional models. Subgrouping searches for recurring patterns in samples, whereas dimensional approaches characterise graded or cross-diagnostic variation. These approaches address important research and assessment questions, but they do not have the same purpose as an individual case formulation: they do not, by themselves, specify how to integrate observations that belong to different conceptual levels—a functional profile, a co-occurring condition, an adaptive trajectory, a historical descriptor, or an area of ability. For the target population, the practical question is how clinicians can keep these domains visible, distinguish their status, and consider them together without treating them as equivalent or as ASD subtypes.

This purposive, non-systematic review addresses an organisational need identified in the literature consulted: a case-level approach to bringing selected profiles of different conceptual status into a single formulation for autistic people without intellectual disability and with fluent functional language. The map is intended to complement dimensional models and guideline-based assessment by keeping functional abilities and challenges, co-occurring conditions, and developmental or adaptive trajectories visible together. Its distinctive contribution is the explicit arrangement of these selected constructs; whether it adds value beyond usual assessment remains to be evaluated.

Aims

This article aims to provide a descriptive and heuristic clinical-phenotypic framework for autistic people without intellectual disability and with fluent functional language. It presents ten selected profiles in a descriptive multidimensional matrix across construct/diagnostic status, functional domain, developmental relevance, and clinical implications. For navigation, the profiles are arranged within three broad organising domains: (A) cognitive, communicative, and neuropsychological functional profiles, including areas of ability and historical ASD/Asperger overlap, (B) co-occurring neurodevelopmental conditions, and (C) adaptive, psychopathological, or identity-related trajectories and associations. Sensory reactivity is cross-cutting. The proposal is intended as a narrative tool for clinical formulation, interdisciplinary discussion, and future empirical validation, with attention to developmental periods in which profiles may become clinically recognisable, especially childhood and adolescence.

2. Methods

2.1. Methodological Approach

This conceptual review was not conducted as a systematic or scoping review. No protocol was registered, records were not counted at identification or selection, and no formal risk-of-bias assessment was undertaken. AI assistance during manuscript preparation and proof correction is described in the Acknowledgments.

For each profile, several aspects were considered, including the nature of the clinical construct, the age or period of onset or recognition, the main phenotypic characteristics, possible developmental trajectories, and the relationship with current diagnostic classifications. The profiles included in the table do not necessarily belong to the same conceptual level: some refer to cognitive, communicative, or neuropsychological domains, including areas of exceptional ability and overlapping ASD/Asperger presentations,

others indicate co-occurring neurodevelopmental conditions, and others describe adaptive strategies and psychopathological or identity-related clinical associations.

2.2. Targeted Literature Review

To support the proposal, a targeted literature review was conducted to identify publications relevant to the profiles on the map. PubMed/MEDLINE, Scopus, Web of Science, PsycINFO, and Google Scholar were searched, together with the reference lists of selected articles. The initial searches were completed by 30 June 2026, and selected publications identified during manuscript revision were subsequently considered. Search terms included those referring to the clinical profile of interest—"Asperger syndrome", "autism spectrum disorder", "ASD level 1", "high-functioning autism", "autism without intellectual disability", and "fluent language"—and those referring to the specific domains of the map: "pragmatic language", "social pragmatic communication disorder", "nonverbal learning disorder/disability", "ADHD", "Tourette", "tic disorders", "pathological demand avoidance", "demand avoidance", "feeding difficulties", "eating disorders", "ARFID", "gender dysphoria", "gender incongruence", "camouflaging", "autistic burnout", "social fatigue", "sensory processing", "sensory reactivity", "adaptive functioning", "savant skills", and "circumscribed abilities". The term "ASD level 1" was retained solely as a search term to retrieve publications using that terminology; it is not used as an operational definition or a synonym for the target population, nor does it specify DSM-5-TR support needs.

The population terms listed above were combined with profile-specific terms. Representative concept combinations were as follows: (population terms) AND ("pragmatic language" OR "social pragmatic communication disorder"), population terms AND (ADHD OR Tourette OR "tic disorders"), and population terms AND terms for demand avoidance, feeding/eating difficulties including ARFID, camouflaging/autistic burnout/social fatigue, sensory processing/reactivity, adaptive functioning, and savant/circumscribed abilities. These are concept-level combinations derived from the terms reported above, not verbatim database-specific query strings. The search terms were in English, but the cited literature includes a German-language review. Language restrictions and a publication-date cut-off were not reported—cited sources span 1994–2026. Initial searches were completed by 30 June 2026, with selected publications considered during revision. The exact date of a separate search update was not recorded.

The ten profiles were retained through purposive, iterative selection rather than specified in advance or derived from clustering. Each addresses a distinguishable clinical-phenotypic focus relevant to multidimensional formulation within the target population; sensory reactivity was retained separately as a cross-cutting dimension, not as an eleventh profile. The number ten reflects this author-led synthesis, not a data-derived threshold or claim of exhaustiveness. Inclusion indicates conceptual and clinical relevance, not equivalent evidential support, and the empirical basis varies by profile and is summarised in Supplementary Table S5. The literature consultation was non-systematic, and the resulting set remains open to revision.

Because the literature consultation was purposive rather than systematic, source counts are bibliographic, not quantitative indicators of evidence volume or strength. Reviews may summarise primary studies cited separately, so distinct publications do not necessarily represent independent samples or non-overlapping evidence. This conceptual review does not estimate unique cohorts. Sources were selected to document the constructs and the main areas of clinical or conceptual uncertainty.

Supplementary Table S5 provides an illustrative map of principal sources for each profile, including study design and methods, samples, main reported findings, and

limitations on interpretation; it is neither exhaustive nor a formal evidence-quality appraisal.

Peer-reviewed articles, narrative or systematic reviews, meta-analyses, observational studies, clinical-conceptual contributions, and methodological papers were included when considered relevant to (a) describing profiles observable in autistic people without intellectual disability and with fluent functional language, (b) clarifying relevant co-occurrences or diagnostic overlaps, (c) distinguishing established clinical constructs from debated descriptors without established diagnostic status, and (d) informing paediatric, neuropsychiatric, or neuropsychological clinical formulation.

The following were excluded: papers not relevant to the target clinical presentation, studies focused exclusively on ASD with intellectual disability or absence of functional language, purely neurobiological contributions without clinical-phenotypic implications, non-peer-reviewed articles, commentaries lacking an explicit empirical or clinical basis, and profiles that were not sufficiently represented in the literature or were not distinguishable from domains already included in the map.

Candidate constructs emerged from the targeted searches and selected reference lists and were considered against the stated population scope and profile-level criteria. Alternatives encountered in those sources were not retained as separate profiles when they fell outside the scope, were too sparsely represented for a meaningful profile-level account, or did not add a distinguishable focus on formulation. No exhaustive candidate list or systematic exclusion log was created; therefore, the review cannot claim that every possible alternative was identified or compared.

Exclusion reflected the review's scope, the available literature, or insufficient conceptual distinctiveness, rather than a judgement that an omitted construct was clinically unimportant. The three organising domains differ in conceptual and diagnostic status—they imply neither a hierarchy nor a causal relationship. Profiles may coexist, with sensory reactivity considered across domains.

The review was not designed to estimate prevalence or effect sizes, grade evidence, or establish diagnostic categories. Selection was partly circular: profiles were chosen in part for their anticipated usefulness within the map. The resulting composition is, therefore, neither exhaustive nor data-derived and requires external evaluation, as outlined in Section 4.13.

Findings were synthesised narratively. Where the cited sources provided sufficient detail, differences in population, developmental stage, design, construct definitions, and measures informed interpretation of discrepant findings. Otherwise, uncertainty was retained. No prespecified evidence-grading or formal adjudication procedure was used.

3. The Clinical-Phenotypic Map

Table 1 presents a descriptive multidimensional matrix of clinical-phenotypic profiles in autistic people without intellectual disability and with fluent functional language. Each row represents a profile, and columns map its predominant construct/diagnostic status, functional domain, developmental relevance/period of recognition, clinical marker, and implications/cautions. The A–C labels denote broad organising domains: (A) cognitive, communicative, and neuropsychological functional profiles, including areas of ability and historical overlap, (B) co-occurring neurodevelopmental conditions, and (C) adaptive, psychopathological, or identity-related trajectories and associations. These domains differ in status and do not imply hierarchy, causal relation, developmental sequence, or mutual exclusivity—the matrix does not replace DSM-5-TR/ICD-11 diagnostic assessment. Sensory reactivity remains cross-cutting.

Table 1. Descriptive multidimensional clinical-phenotypic matrix of profiles in autistic people without intellectual disability and with fluent functional language.

Domain/Construct Status	Profile	Function	Development/Recognition	Clinical Marker	Implications/Cautions
(A) Functional profiles	A1. Savant-like abilities	Areas of ability/strengths	Childhood or adolescence	Circumscribed or exceptional abilities in specific domains	Specific abilities do not amount to global giftedness and do not exclude adaptive vulnerabilities
	A2. Pragmatic-language profile	Pragmatic-communicative dysfunction	Childhood; more evident as reciprocal-conversational demands increase	Formally fluent language, but difficulties with contextual, inferential, and reciprocal use of language	Consider social (pragmatic) communication disorder (SCD) only if ASD Domain B criteria are not met; SCD and ASD should not be treated as overlapping subtypes
	A3. Nonverbal learning disorder/nonverbal learning disability	Visuo-spatio-temporal	School age; more evident in adolescence	Visuospatial deficits, motor, mathematical, and organisational difficulties, and difficulty interpreting nonverbal information	May mimic or coexist with ASD, but should not be automatically assimilated to the autistic profile
	A4. HFA—overlapping ASD/Asperger profile	ASD/Asperger overlap	Childhood, sometimes late recognition	ASD without intellectual disability and with functional language; intellectual functioning from borderline to superior	IQ is not a proxy for adaptive functioning or support needs; recognition, compensation, and needs may differ across the intellectual range
(B) Co-occurring conditions	B1. ASD with ADHD co-occurring features	Neurodevelopmental/co-occurring	Early childhood or school age	Inattention, impulsivity, hyperactivity, and executive dysregulation	Distinguish ADHD co-occurrence from ADHD-like features that may accompany rigidity, anxiety, or sensory overload
	B2. ASD with tic/Tourette-related features	Neurodevelopmental/differential	Childhood, with a fluctuating course	Motor/vocal tics, compulsive phenomena, or complex tics	Differentiate tics, autistic stereotypies, OCD-like compulsions, and ASD restricted/repetitive behaviours
(C) Adaptive and associated trajectories	C1. Social fatigue/camouflaging profile	Adaptive/compensatory	Adolescence or adulthood	Apparent social competence may be supported by reported camouflaging or other demanding compensatory strategies; fatigue and possible burnout are separate features to assess	Document strategies and context, fatigue/recovery, and functional change separately; visible performance may understate support needs

C2. Demand avoidance/PDA-like features	Adaptive/controversial construct	Childhood or adolescence	Rigid avoidance of demands, need for control, anxiety, rigidity, sensory, or executive overload	Use as a provisional functional descriptor; assess anxiety, trauma, rigidity, sensory/executive overload, and family/school context
C3. Feeding behaviours/possible eating disorder co-occurrence	Descriptive feeding features/possible co-occurring disorder	Food selectivity may be present in childhood; diagnosable EDs may be recognised later	Food selectivity, food refusal, sensory sensitivities, rigidity, or food-related rituals; ED requires separate criteria-based assessment	Feeding behaviours alone do not establish an eating disorder; assess diagnostic criteria and nutritional/medical risks separately
C4. Gender incongruence and dysphoria	Identity-related clinical association	Childhood, adolescence, or young adulthood	Possible co-occurrence between ASD/autistic traits and GD/GI	Assess gender incongruence and any distress/impairment individually and in context; avoid automatic causal assumptions

Abbreviations. ADHD, attention-deficit/hyperactivity disorder; ASD, autism spectrum disorder; ED, eating disorder; GD/GI, gender dysphoria/gender incongruence; HFA, high-functioning autism; IQ, intelligence quotient; NLD, nonverbal learning disorder; NVLD, nonverbal learning disability; OCD, obsessive-compulsive disorder; PDA, pathological demand avoidance; SCD, social (pragmatic) communication disorder. Note. Table 1 is a descriptive matrix, not a diagnostic taxonomy. The first column identifies the broad organising domain, and profile labels and cautions indicate each entry's predominant construct or status. Other columns summarise function, the period during which a feature may be recognised (not necessarily its onset), clinical markers, and implications. Domains A–C differ in conceptual, diagnostic, and evidential standing; they are non-hierarchical and imply neither a shared mechanism nor a developmental sequence. Profiles may coexist and are not ASD subtypes—interpret them with DSM-5-TR/ICD-11 criteria, developmental history, and individual functional assessment. Sensory reactivity is cross-cutting.

Terminology used throughout: the framework is the overall heuristic proposal, the map is its representation, shown in matrix form, domains are broad organising categories, and profiles are individual entries. "Cluster" refers only to a possible data-derived subgroup in future analyses and does not name the domains shown here.

A more detailed description of the profiles, including the period of recognition, clinical-phenotypic features, and possible clinical implications, is provided in Supplementary Table S1.

Supplementary Table S2 synthesises the three organising domains and characteristics that may span more than one profile. It is descriptive and does not replace Table 1, whose columns map each profile by predominant status, functional domain, developmental relevance, clinical marker, and implications/cautions. The domains distinguish functional profiles, co-occurring neurodevelopmental conditions/diagnostic relationships, and adaptive, psychopathological, or identity-related trajectories/associations—profiles may overlap.

4. Discussion

The Discussion considers the conceptual basis and evidence for the profiles (Sections 4.1–4.8), proposed clinical uses (Section 4.9), assessment and methodological limitations (Sections 4.10 and 4.11), the framework's contribution (Section 4.12), and future evaluation (Section 4.13).

This work proposes a descriptive clinical-phenotypic framework to organise the heterogeneity of autism without intellectual disability and with fluent functional language. The profiles included in Table 1 belong to different conceptual levels and can be combined within the same individual; the intention is to avoid both reducing the picture to a generic 'mild autism' or 'high functioning' and fragmenting it into unvalidated diagnostic subtypes [14,15].

4.1. Relationship with Existing Frameworks

The proposal should be situated within a body of work addressing the same problem—the heterogeneity of autism—using different tools. Lombardo and colleagues distinguish supervised approaches, which start from theoretically defined a priori hypotheses, from unsupervised approaches, which derive subgroups from data and propose model utility as the evaluation criterion, that is, the capacity of a model to account for clinical or mechanistic phenomena [16]. In these terms, the present proposal is an a priori, theory-informed clinical framework rather than a data-derived model. Its intended utility is organisational and communicative rather than mechanistic or predictive.

Astle and colleagues argue that taxonomies that assign individuals to discrete categories are poorly suited to explaining variability in neurodevelopmental disorders because they do not map reliably onto genetics, neurobiology, or cognition [17]. This concern also applies here: even a non-hierarchical map may encourage readers to treat named profiles as established entities. The intended level of application is, therefore, important. The domains of the map are not proposed as natural entities or explanatory categories, but as entries within a clinical observation framework. Their value should, therefore, be assessed in terms of inter-rater agreement and usefulness for clinical case formulation, rather than biological validity. In this sense, the proposal is complementary to, and not an alternative to, dimensional and transdiagnostic approaches.

Mottron and Bzdok offer a contrasting view: fragmenting the phenotype into dimensional traits and sub-profiles may produce a partly artefactual heterogeneity, and research should instead preserve a line of enquiry focused on prototypical autism [15]. We consider this argument especially relevant to aetiological research and less directly

applicable to the clinical formulation of an individual case, where observed heterogeneity is the starting point. This remains a substantive difference in perspective.

On the empirical side, HiTOP organises psychopathological co-occurrence into spectra derived from observed symptom covariances [18], whereas RDoC and HiTOP are discussed as distinct but potentially complementary dimensional approaches in recent autism-focused work [19]. These approaches aim to characterise dimensions that cross diagnostic boundaries and, depending on the model, relations among symptoms, functional constructs, and mechanisms. Their scope is different from that of the present proposal: they do not aim to provide a case-level index that juxtaposes mixed-status observations, such as functional profiles, co-occurring diagnoses, historical descriptors, adaptive trajectories, and areas of ability. This is a difference in purpose, not a shortcoming of the dimensional models. The proposed map cannot estimate symptom covariation, identify mechanisms, or derive population subgroups. Its provisional role is to make the conceptual status of observations explicit during individual formulation while remaining compatible with dimensional measures and data-derived models. Its incremental clinical usefulness has not been established.

Finally, there are direct clinical precedents. Lai and Baron-Cohen described the "lost generation" of autistic adults without intellectual disability, addressing camouflaging, psychiatric co-occurrences, female underdiagnosis, and the diagnostic process [20]. More recently, Bostwick and McKean re-examined the contemporary clinical relevance of presentations historically referred to as "high-functioning autism" [21]. The specific contribution of the present paper, compared with these precedents, does not lie in identifying new constructs but in explicitly arranging them across distinct conceptual levels and formulating testable predictions about the resulting structure. This conceptual contribution is distinct from practical usefulness: any benefit for assessment, case formulation, or support planning remains hypothetical until it is evaluated empirically.

4.2. Empirical Subgroups: What the Data Show

Existing data-derived subgroups provide an important basis for comparison within this a priori framework. In autistic adults with average or above-average intellectual functioning, community detection applied to cognitive, psychological, and well-being variables identified two subgroups that were replicated across independent samples [22]; the same subgroups proved stable at two and five years and predictive of psychological difficulties and quality of life [23]. Neither corresponds to the domains of the present map.

Similarly, a latent profile analysis conducted in autistic adults without intellectual disability found that a substantial proportion of the sample showed fully normative socio-cognitive performance [24]. This illustrates variability in social cognition, although performance on these tasks should not be equated with pragmatic-language competence. It follows that the map should not be presented as an alternative to empirical subgroups, but as a tool of a different order: an observation framework for case formulation, whose expected value lies in the systematic coverage of domains to be explored, rather than in the discovery of latent partitions within the population. If future analyses identify empirical subgroups, their validation could be guided by the SUVAC checklist [25], which allows the proposal to state transparently which requirements it meets and which it does not.

4.3. Functional Profiles: Cognitive, Communicative, and Neuropsychological Domains (Organising Domain A)

Organising domain A groups profiles that describe cognitive, communicative, and neuropsychological functioning: circumscribed abilities, pragmatic-language features, the visuo-spatio-temporal NLD/NVLD profile, and historical ASD/Asperger overlap. Their

shared placement reflects a functional level of description—how the person processes information, communicates, and learns—and a related assessment focus, not a common diagnosis, mechanism, or evidential status. In formulation, these profiles are explored through communicative and neuropsychological assessment.

4.3.1. Savant-Like Abilities (A1)

Strengths are represented first in domain A: the savant-like/circumscribed exceptional abilities profile (A1) describes circumscribed or exceptional abilities that may support motivation, learning, and educational planning. However, such abilities should not be confused with global giftedness, nor interpreted as evidence of good overall adaptive functioning [26–28]. Clinical formulation should, therefore, integrate both vulnerabilities and resources, avoiding both global pathologisation and the idealisation of specialised abilities. To reduce the risk of inappropriate overlap between distinct constructs, Supplementary Table S3 summarises, for each profile, the appropriate relationship to the target population described in this review, as well as the main critical point for differential diagnosis or clinical formulation.

4.3.2. Pragmatic-Language Profile (A2)

Within this framework, the pragmatic-language profile (A2) denotes difficulty using and interpreting language in social contexts, including conversational reciprocity, contextual cues, and inference about implied or non-literal meaning. When marked, these difficulties can be captured by standardised measures of social communication, and their degree is clinically informative. Pragmatic-language difficulties are not universal across the target population.

The pragmatic-language profile is descriptive, not an autonomous subtype; it may represent a communicative feature or an area for differential assessment. In DSM-5-TR, distinguishing ASD from social (pragmatic) communication disorder (SCD) requires applying the full diagnostic criteria for each condition and reviewing developmental history. ASD requires all social communication and interaction criteria, at least two of the four restricted/repetitive behaviour categories (currently or by history), symptoms in the early developmental period, and clinically significant impairment. An isolated current or historical restricted/repetitive behaviour does not establish ASD. SCD should be considered only when its own full criteria are met and the presentation is not better explained by ASD or another condition [1,6].

4.3.3. Nonverbal Learning Disorder/Nonverbal Learning Disability (NLD/NVLD) (A3)

Nonverbal learning disorder/nonverbal learning disability (A3), by contrast, represents a neuropsychological profile centred on the visuo-spatio-temporal domain, with visuospatial, motor, mathematical, organisational, and nonverbal interpretation vulnerabilities. The available prevalence estimate is 3–4% in North American children and adolescents (2.78%, 95% CI 2.03–3.52; 3.10%, 95% CI 1.93–4.27; 3.9%, 95% CI 1.96–5.78, across the three samples examined) [29,30]. The profile may phenotypically overlap with the target ASD presentation or co-occur with ASD, but it should not be automatically assimilated into the autistic profile. The severity and pattern of visuospatial difficulties should inform clinical formulation but should not be used alone to distinguish NVLD from ASD. Social difficulties associated with visuospatial or pragmatic-contextual vulnerabilities are not, on their own, sufficient to infer a diagnosis of ASD, which requires developmental assessment of socio-communicative criteria and restricted and repetitive behaviours and interests [31].

4.3.4. HFA as an Overlapping ASD/Asperger Profile, and Theory of Mind (A4)

HFA is retained in the map as a historical-descriptive shorthand for the area of overlap between high-functioning autism and the former Asperger profile, not as a separate diagnostic category. It is, therefore, placed within organising domain A rather than treated as an autonomous subtype. Its use requires caution, because intelligence quotient is not a sufficient indicator of adaptive functioning. Fluent language and an average or above-average IQ do not necessarily ensure good social, educational, occupational, or psychological adaptation [12]. This point is particularly relevant because the main clinical risk in autistic people without intellectual disability is the underestimation of support needs.

A further reason for caution is that the label obscures a wide range of intellectual functioning. The target population may include people with borderline intellectual functioning, conventionally described as an IQ of approximately 70–84. However, this range should not be used alone to establish or exclude intellectual disability—intellectual and adaptive functioning should be assessed together. For clinical formulation, fluent language should not be taken as sufficient evidence of adequate everyday functioning.

Greater compensation has been associated with higher verbal IQ, better executive functioning, and higher self-reported anxiety [32,33]. These findings suggest that observable social performance may not fully reflect the effort required to achieve it. Intellectual level is, therefore, relevant to the map as a source of individual variation, rather than as a substitute for assessment of adaptive functioning and support needs.

Similarly, difficulties with theory of mind (inferring mental states) should not be used as the sole defining feature of the overlapping profile referred to as HFA. The "double empathy problem" perspective suggests that many communication difficulties between autistic and non-autistic people may also arise from mutual misunderstanding and differences in communication styles, rather than solely from an intrinsic socio-cognitive deficit in the autistic person. This clarification is consistent with a dimensional and contextual understanding of social functioning in ASD and reduces the risk of reductionist or stigmatising interpretations [34,35].

4.4. Co-Occurring Neurodevelopmental Conditions and Diagnostic Relationships (Organising Domain B)

The matrix's organising domain B records co-occurring neurodevelopmental conditions, namely, ADHD (B1) and tic/Tourette-related features (B2), which should not be interpreted as autism-specific variants. They may modify the clinical presentation, adaptive burden, psychopathological risk, and developmental trajectory. Their placement reflects a diagnostic relationship: these conditions have their own diagnostic criteria and may co-occur with ASD, rather than describing the autistic presentation itself. Recognising them changes the clinical formulation and support plan.

4.4.1. ASD with ADHD Co-Occurring Features (B1)

Co-occurrence or overlap between ASD and ADHD may complicate the differential diagnosis between inattention, executive dysregulation, sensory overload, rigidity, and social difficulties [36,37]. Co-occurring ADHD should, therefore, be distinguished from ADHD-like manifestations secondary to rigidity, anxiety, or sensory overload.

4.4.2. ASD with Tic/Tourette-Related Features (B2)

Similarly, tics, stereotypies, compulsions, and restricted repetitive behaviours require careful phenomenological distinction [38,39]. The literature reports symptomatic overlap between Tourette syndrome and ASD, and with it a risk of diagnostic confusion:

the relationship is one of overlap and possible co-occurrence and requires careful differential assessment and does not imply progression from one condition to the other.

4.5. Adaptive, Psychopathological, and Identity-Related Trajectories and Associations (Organising Domain C)

Organising domain C includes adaptive, psychopathological, and identity-related trajectories or associations: social fatigue/camouflaging, demand avoidance, feeding and eating difficulties, and GD/GI. Their shared placement reflects their relation to adaptation, developmental change, or other clinical phenomena, not a common diagnosis, mechanism, or causal pathway. Several may become more clinically evident in adolescence or young adulthood. Feeding and eating difficulties and the ASD–GD/GI association may be relevant in discussions of female presentations, but they are not female subtypes of ASD.

These entries should not be read as equivalent developmental phenomena. C1 describes reported compensatory strategies and their possible subjective costs; fatigue is recorded separately, and possible autistic burnout is treated as a broader, provisional state. C2 is a debated behavioural descriptor; the sources reviewed do not establish a uniform construct, developmental course, or distinct diagnostic status. C3 includes descriptive feeding behaviours as well as possible diagnosable feeding/eating disorders, which require separate criteria-based assessment. Their shared placement in domain C is organisational only. Statements about adolescent or young-adult recognition apply to the specific features and evidence discussed for each profile—they do not imply a common onset or developmental sequence.

4.5.1. Social Fatigue, Camouflaging, and Autistic Burnout (C1)

Among the adaptive trajectories, the social fatigue/social adaptation/camouflaging profile (C1) is particularly relevant. Apparent social competence may be sustained by cognitively and emotionally demanding compensatory strategies, and fatigue, anxiety, stress, sleep disturbance, burnout, or depressive symptoms may also be reported [33,40,41]. This profile helps distinguish observable performance from the person's internal functioning: good social performance in structured contexts does not necessarily imply low support needs.

Camouflaging nevertheless has conceptual and measurement limitations. Fombonne questioned its construct and measurement validity, also noting, among other issues, the intentionality implied by the term [42]. In their response, Lai and colleagues acknowledged that camouflaging is not necessarily a characteristic of autism in females; Ai and colleagues examined camouflaging in the general population [43,44]. The systematic review by Cook and colleagues shows that the two main measurement approaches—the discrepancy between external presentation and internal characteristics, and self-report assessment using the CAT-Q—are not equivalent and do not yield the same results [45]. It has also been proposed that camouflaging may be reconceptualised as a particular case of impression management and, therefore, as not specific to autism [46]. Levels of camouflaging and mental health correlates have also been documented in the general population [44]. Accordingly, in the map, this profile should be understood as a prompt to document compensatory strategies and assess any associated subjective costs separately, not as a diagnostic marker or as a trait specific to a subgroup.

Autistic burnout has been described as a broader state of exhaustion accompanied by reduced functioning and reduced tolerance to stimuli. The initial qualitative definition referred to episodes typically lasting three months or longer [47]. Subsequent research has not established a consistent duration criterion, and further work is needed to clarify how it differs from depression and occupational burnout [48,49]. Conceptual models of risk

and protective factors have also been proposed [50]. These findings raise concern that interventions focused primarily on concealing autistic characteristics may add to the burden of compensation. This is a clinical implication of the available evidence, rather than a demonstrated causal effect of masking-focused interventions. Clinical formulation should, therefore, consider environmental adjustments, subjective effort, and support needs, rather than focusing solely on observable social performance.

For the purposes of this heuristic map, the three C1 components are documented separately. Camouflaging refers to the strategies the person reports using to manage social presentation and the contexts in which they use them. The CAT-Q is a self-report measure of camouflaging and does not assess the separate C1 dimensions of reported fatigue or possible autistic burnout; therefore, it cannot capture the full C1 profile. Fatigue is recorded as the person's reported effort or tiredness, including its timing, social or sensory context, effect on everyday functioning, and recovery; it is not inferred from camouflaging strategies or observable performance, and is not presumed to be caused by camouflaging. Possible autistic burnout is considered separately as a broader state by asking about longitudinal change from the person's baseline in exhaustion, functioning or skills, and tolerance to stimuli, while considering differential or co-occurring explanations, such as depression and occupational burnout. These are prompts for clinical formulation, not validated diagnostic criteria or a validated C1 assessment protocol.

4.5.2. Demand Avoidance and PDA-Like Features (C2)

The construct of demand avoidance/PDA-like features (C2) should be retained as a cautious and controversial clinical descriptor, not as an autonomous diagnosis or as a validated subtype of autism. Rigid avoidance of demands should be interpreted primarily in functional terms, as a possible expression of intense anxiety, a need for control, sensory or executive overload, reduced tolerance for unpredictability, and interactions between individual vulnerabilities and environmental demands. Clinical formulation should, therefore, explore anxiety, trauma, oppositionality, cognitive rigidity, sensory profile, executive functions, and family/school context, while avoiding the diagnostic reification of PDA as a separate syndrome [51,52].

The debate remains open and has evolved since 2021. In 2024, the journal that had published Green's review also published a contribution calling for priority to be given to establishing the prevalence and causes of the construct, together with the related replies [53]. A German-language critical review concluded that PDA is neither a diagnostic entity nor a subtype of autism, but rather a behavioural profile [54]. A more recent systematic review documented, across twelve studies, a problem of circular logic in the definition and measurement of the construct [55]. Within the present framework, PDA poses a risk that it must avoid: a provisional descriptor that, if treated as an established diagnosis, may alter referral practices and communication between professionals and families before it has been validated.

4.5.3. Feeding/Eating Difficulties and Eating Disorders (C3)

Feeding difficulties and eating disorders are grouped in C3 because both may affect clinical presentation, adaptive burden, and support needs. In this map, food selectivity refers to a restricted range of accepted foods; other feeding difficulties include food refusal, sensory sensitivities, rigidity, or food-related rituals. These behaviours are descriptive features and do not, by themselves, establish an eating disorder. A diagnosable feeding/eating disorder (including avoidant/restrictive food intake disorder (ARFID), anorexia nervosa, bulimia nervosa, or binge-eating disorder) requires assessment against the relevant diagnostic criteria. ARFID warrants specific discussion within C3. Under the DSM-5-TR, avoidance or restriction must be associated with at least

one of the following: significant weight loss or faltering growth, significant nutritional deficiency, dependence on enteral feeding or oral nutritional supplements, or marked interference with psychosocial functioning. Sensory-based avoidance, low interest in eating, and concern about aversive consequences are possible drivers; these may overlap with autistic food selectivity, but food selectivity alone—even when sensory-related—does not establish ARFID. The criteria also distinguish ARFID from restriction associated with weight or shape disturbance and require consideration of food availability, culturally sanctioned practices, medical conditions, and other mental disorders [1]. The reviewed literature describes overlap or co-occurrence between eating disorders, ASD, and ADHD, but is heterogeneous and does not establish a simple causal relationship [56–58]. These issues may be relevant in some female presentations but should not be used to define a sex-specific ASD subtype.

4.5.4. Gender Incongruence and Gender Dysphoria (C4)

Gender identity is not, by itself, a psychopathology or maladaptive outcome. In ICD-11, gender incongruence is classified in the chapter on conditions related to sexual health, outside the chapter on mental and behavioural disorders (World Health Organization, ICD-11 MMS, release 2025-01) [2]. Under DSM-5-TR, gender dysphoria is diagnosed only when the relevant criteria are accompanied by clinically significant distress or impairment [1]—gender identity or incongruence alone is not sufficient for that diagnosis. Clinical formulation should distinguish identity/incongruence from any associated distress or impairment and consider the person's family, cultural, and clinical context. The association reviewed below should not be interpreted as evidence that gender incongruence is a psychopathology or maladaptive outcome, or as evidence of a causal relationship with ASD.

The meta-analysis by Kallitsounaki and Williams estimated a pooled prevalence of ASD diagnoses of 11% (95% CI 8–16%) across samples examining people described as having gender dysphoria or gender incongruence, with substantial heterogeneity ($I^2 \approx 97\%$) and a prediction interval of 1–54%. In the cumulative meta-analysis, restricting the analysis to the 10 largest studies, each with more than 200 participants, yielded an estimate of 8% (95% CI 4–13%). Subgroup analyses by study design yielded estimates of 8% in clinical samples and 20% in population-based samples [59]. These estimates were obtained across different age groups, recruitment settings, and methods of identifying ASD diagnoses. They should not be regarded as interchangeable across populations or as estimates specific to children or to the target population. Given this heterogeneity, the pooled estimate should not be applied directly to an individual patient or used alone to guide clinical decisions. Assessment should remain individualised and avoid causal interpretations.

4.6. ASD-Like Phenotypic Convergence

Here, "ASD-like phenotypic convergence" is used provisionally to describe a similar observable clinical pattern across people with different developmental or diagnostic histories. It refers to resemblance at the level of presentation; it does not identify a shared cause, developmental sequence, or separate diagnostic category. Its limited contribution is to name this presentation-level resemblance and prompt consideration of distinct explanations without collapsing them. These may include (i) co-occurrence of established ASD and another condition, (ii) diagnostic overlap, where similar behaviours may arise from different conditions and require differential assessment, (iii) late recognition of ASD that was present from early development, or (iv) non-autistic adaptive or compensatory strategies that produce some similar observable behaviours. These explanations are not interchangeable, may coexist, and cannot be inferred from surface features alone

[32,36,37]. The term does not imply a mechanism by which ADHD, NLD/NVLD, tic/Tourette-related features, eating disorders, or other profiles develop into ASD. It is a heuristic prompt to review developmental history, context, and functioning against full diagnostic criteria, not a basis for reclassification [60,61]. The distinction between late-recognised ASD, ASD-like adaptation, co-occurrence, diagnostic overlap, and diagnostic conversion is summarised in Supplementary Table S4.

The cited studies report co-occurrences, symptomatic overlap, or compensatory strategies; they do not test "ASD-like phenotypic convergence" as a unified construct. Its ability to distinguish explanations or improve formulation remains untested.

Potentially informative observations are prompts for assessment, not stand-alone discriminators. For late-recognised ASD, review developmental history, including collateral accounts or records when available, for evidence that defining features were present in the early developmental period despite later recognition [1,60]. For co-occurrence, assess whether each proposed condition meets its own full diagnostic criteria; for apparent diagnostic overlap, examine the form, function, timing, and context of the behaviour and whether the full ASD criteria are met or another condition better accounts for it [36,37]. For non-autistic adaptation or compensation, document the strategies described, the contexts in which they are used, and any reported effort or fatigue. Such observations may contextualise apparent social performance but are not specific to autism. For diagnostic conversion, document the change in diagnosis and the evidence prompting reassessment; a revised label records a change in classification and does not establish causal transformation. These observations may guide reassessment, but explanations may coexist, and their ability to distinguish them has not been established.

4.7. The So-Called Female Autism Phenotype

A further implication concerns accounts sometimes grouped under the label "female autism phenotype". This label does not denote a distinct subtype or a uniform set of features. The evidence reviewed here describes differences in early concerns and diagnostic predictors in some child samples, and social adaptation in a small qualitative sample of adult women; these observations are specific to their samples and developmental periods. Their clinical relevance is to consider whether visible social performance reflects the person's actual functioning, effort, and support needs, rather than to presume camouflaging or delayed recognition. The age-stratified evidence below does not establish a single developmental trajectory.

Within the map, social fatigue and camouflaging are explored when relevant to the individual, rather than inferred from sex. Assessment considers any gap between observable performance and the demands of adaptation [7,41].

In a retrospective caregiver survey, carers of school-age children later diagnosed with ASD recalled sex-related differences in preschool concerns, social-navigation strategies, and restricted interests [10]. Among consecutively referred children aged 2.5–10 years, parent-reported restricted and repetitive behaviour symptoms were less predictive of ASD diagnosis for girls than for boys, whereas emotional and behavioural problems were more predictive; the predictive value of overall autistic impairment, sensory symptoms, and cognitive functioning did not differ by sex [8]. These findings concern recognition in specific samples; they do not imply fewer core features in girls or establish a general female symptom profile. The sources considered here provide limited evidence specific to female adolescents. A mixed-sex cohort of 114 autistic participants aged 15–25 years examined sleep, fatigue, social well-being, and depressive symptoms and included sex comparisons [40]. These outcomes may inform assessment in older adolescence and young adulthood, but the study cannot isolate adolescent girls or establish sex-specific manifestations or change with age. As a clinical assessment inference, school-stage

transitions and changing peer and autonomy demands may alter which difficulties are reported or visible, and this requires direct study.

In adult women, qualitative interviews with ten Japanese autistic women aged 28–50 years (diagnosed between ages 17 and 36) described social adaptation in relation to accumulated negative experiences. Some participants reported misdiagnosis, difficulty obtaining an appointment, or being misunderstood because they did not fit a typical autistic image [41]. These accounts identify possible recognition and access barriers in that selected adult sample; they do not estimate frequency or establish onset or paediatric manifestations. Current functioning should be considered alongside developmental history, drawing on early-childhood caregiver reports, school-age peer, educational, and adaptive functioning, and adolescent autonomy, well-being, and transitions. Reported effort, fatigue, and support needs should be explored when relevant, without presuming camouflaging or delayed recognition from sex alone. Onset, recognition, and functional impact should be distinguished: Table 1 records recognition, not onset, and functional impact may vary with age-related demands and supports.

The domains considered in this map should not be interpreted as female subtypes of ASD or as evidence of causal relationships. In clinical formulation, we suggest taking account of potential barriers to diagnostic recognition [7–9], together with the possible discrepancy between observable social performance and the demands of compensation [32,41]. This assessment should include a detailed developmental history, sensory load, and real-world adaptive functioning. Feeding and eating difficulties should also be considered: overlapping eating behaviours, including food selectivity, have been described in female samples with ASD or anorexia nervosa (AN) [57].

Camouflaging should not be regarded as exclusive to females or as a defining feature of a female autism subtype [43]. It has also been conceptualised as a form of impression management shaped by the social context [46]. The distinction between sex assigned at birth and gender identity should be maintained, particularly when discussing GD/GI. Findings from sex-based comparisons should not automatically be interpreted as differences associated with gender identity.

4.8. Sensory Reactivity as a Cross-Cutting Dimension

Sensory reactivity represents a cross-cutting dimension of the model. In DSM-5/DSM-5-TR, hyper- or hypo-reactivity to sensory input and unusual interest in sensory aspects of the environment are included among the domain B criteria for ASD; therefore, sensory reactivity should not be interpreted as a separate variant, but as a core and cross-cutting component of the ASD clinical presentation. Within the present framework, sensory load is hypothesised to contribute to social fatigue and other adaptive difficulties. This proposed cross-cutting role requires empirical testing. Assessment of social fatigue should, therefore, include not only masking and camouflaging, but also the sensory profile and environmental load [39,62–65].

An empirical caveat should be noted regarding this choice. Factor mixture modelling applied to large samples suggests that sensory features are organised both along continuous dimensions and into latent classes [66]. The placement of sensory reactivity as a single cross-cutting dimension is, therefore, an operational simplification adopted for clinical formulation, not a claim about the construct's latent structure.

The map retains sensory reactivity as its one explicit cross-cutting dimension because, as summarised above, it forms part of the ASD diagnostic description and is treated here as potentially relevant across functional domains rather than as a separate subtype. This was a heuristic choice of scope—the literature review did not systematically compare or rank candidate cross-cutting dimensions. Other potentially cross-cutting influences, including adaptive functioning, executive demands, anxiety or mood

symptoms, fatigue, and environmental context, are discussed within relevant profiles or clinical formulation rather than represented as additional overlays. Their omission as overlays does not imply that they are unimportant, and sensory reactivity is not claimed to be the only or most influential cross-domain factor. Whether further dimensions should be represented as cross-cutting remains open to participatory and empirical revision.

These sources describe sensory features and group differences—they do not establish that sensory load causes social fatigue or adaptive difficulties. The proposed cross-domain contribution remains a hypothesis.

Figure 1 summarises the three organising domains and their profiles, as presented in Table 1. Its lower bands add a shared developmental/contextual lens and clarify relationships: profiles may coexist, while their salience may vary across age and setting—sensory reactivity is cross-cutting. The figure does not assign severity or imply a developmental sequence or causal pathway. It remains a formulation representation rather than a second classification.

DESCRIPTIVE MULTIDIMENSIONAL MATRIX FOR CLINICAL FORMULATION

A. Functional profiles: cognitive, communicative, and neuropsychological	B. Co-occurring neurodevelopmental conditions/diagnostic relationships	C. Adaptive, psychopathological, and identity-related trajectories/associations
A1. Savant-like/circumscribed exceptional abilities: not synonymous with global giftedness. A2. Pragmatic-language profile: fluent formal language with difficulties in contextual use, inference, and reciprocity. A3. NLD/NVLD: profile centred on the visuo-spatio-temporal domain. A4. HFA as an overlapping ASD/Asperger presentation: IQ is not a proxy for adaptive functioning.	B1. ASD with ADHD co-occurring features. B2. ASD with tic/Tourette-related features. These require differential formulation, not subtype assignment.	C1. Social fatigue/social adaptation/camouflaging. C2. Demand avoidance/PDA-like features. C3. Feeding/eating difficulties and ED profile. C4. ASD–gender association: distinguish identity/incongruence from dysphoria-related distress; not an ASD subtype.
DEVELOPMENT/CONTEXT—Consider how age-related transitions, learning and peer demands, home/school/community settings, environmental expectations, and available support may shape what is observed and its functional significance; record the source and context of each account.		
RELATIONSHIPS/CROSS-CUTTING—Profiles may co-occur and overlap. Domains have no severity order, developmental sequence, or causal pathway; sensory reactivity is represented across A–C.		

Figure 1. Descriptive multidimensional clinical-phenotypic matrix for formulation. Columns A–C summarise the organising domains and selected profiles. The lower bands invite consideration of developmental transitions, home, school, and peer contexts, environmental expectations, and supports, and state that profiles may co-occur without implying a hierarchy, developmental sequence, or causal pathway. Sensory reactivity is represented as cross-cutting. The matrix complements Table 1 and should be interpreted alongside DSM-5-TR or ICD-11 diagnostic assessment.

4.9. Clinical Implications

The risk highlighted at the outset—that observable competence may be mistaken for adaptive functioning and that support needs may consequently be underestimated—provides a rationale for a structured and multidimensional approach to clinical observation. The heterogeneity described in autism without intellectual disability supports consideration of communicative, neuropsychological, co-occurring, adaptive, and ability-related domains within individual clinical formulations [15]. The proposed map is intended to organise these domains and to identify areas that may warrant further assessment, including pragmatic communication, cognitive-neuropsychological functioning, neurodevelopmental or psychopathological co-occurrences, camouflaging, sensory load, social fatigue, and circumscribed strengths. Its clinical utility has not yet been established and requires empirical evaluation.

For children and young people under 19, NICE guideline CG128 already recommends a multidimensional diagnostic assessment, including a profile of strengths, skills, impairments, and needs considered in family and educational context, assessment of relevant domains, such as intellectual ability, learning and academic skills, speech, language, and communication, adaptive behaviour, mental and emotional health, sensory sensitivities and socialisation, and consideration of coexisting conditions [67]. The proposed map is consistent with this approach but has a narrower conceptual role: it organises selected clinical-phenotypic profiles by domain and conceptual status to support clinical discussion and formulation. It does not replace NICE-recommended diagnostic or needs-based assessment, add diagnostic criteria, or claim demonstrated incremental clinical utility. This comparison applies to paediatric and adolescent assessment within CG128; the map's applicability to adults requires separate evaluation.

The cited paediatric ASD study found that IQ alone was an imprecise predictor of adaptive abilities, particularly among children without intellectual disability [12]. Intended users include paediatricians, child and adolescent psychiatrists, psychologists and neuropsychologists, speech and language therapists, and occupational therapists working in paediatric and adolescent autism services. For established ASD, the map may support formulation and support planning. During diagnostic assessment, it serves only as a prompt for further assessment; it cannot diagnose ASD or replace comprehensive evaluation. It does not measure adaptive functioning or support needs, and its clinical utility remains untested.

The matrix arranges potentially coexisting profiles by conceptual status, with sensory reactivity considered across domains. These entries prompt formulation rather than assign diagnoses or prescribe an assessment sequence. Table 2 links domains to indicative tools and formulation outputs. The map is intended to support individualised discussion but does not assign a severity score or prescribe interventions. In keeping with NICE CG128, it may inform a profile of strengths, skills, impairments, and needs for needs-based planning [67]. Whether it improves planning or outcomes remains to be evaluated. When informants disagree or evidence is uncertain, record each account separately by source, setting, timeframe, and whether it is observed or reported; note whether differences are context-specific or unresolved. Do not average accounts or treat missing information as absence. This documentation guidance is a formulation prompt, not a validated adjudication rule.

Table 2. Clinical assessment checklist for autism without intellectual disability and with fluent functional language.

Assessment Axis	Clinical Focus	Indicative Tools	Practical Output
Core ASD clinical presentation	Functional language, social reciprocity, pragmatics, RRB, restricted interests, predictability, and sensory profile	ADOS-2, ADI-R, SRS-2, Sensory Profile 2, or equivalents	Description of the ASD clinical presentation and support needs
Cognitive, neuropsychological, and adaptive profile	IQ, unevenness of the profile, executive functions, visuospatial abilities, learning, and adaptive functioning	WISC-V/WAIS-IV, Vineland-3, targeted neuropsychological batteries	Strengths, vulnerabilities, and IQ-adaptive functioning discrepancy
Co-occurrences/identity-related concerns	ADHD, tic/Tourette-related features, OCD-like features, anxiety/depression, eating disorders, and other feeding difficulties (assess separately); gender identity/incongruence and any distress or impairment (assess separately, in context)	Conners/ADHD-RS (ADHD); YGTSS (tic severity); EDE/EDE-Q (eating disorder psychopathology); PARDI (suspected ARFID; see note); specialist clinical interview	Separate diagnostic assessment and individualised, context-sensitive formulation and support planning
Adaptation, sensory load, and context	Camouflaging strategies and contexts; reported fatigue (timing, context, impact, and recovery); sensory load; school/family/work context	CAT-Q (self-reported camouflaging); clinical interview/history (fatigue and recovery); Sensory Profile; contextual reports	Separate description of strategies, fatigue, sensory contributors, recovery, and support needs
Strengths and clinical risks	Possible burnout, longitudinal change in skills/functioning, isolation, depression, and suicide risk	Longitudinal clinical/functional history; targeted psychopathological assessment	Individualised, strengths-based plan responsive to functional change and clinical risk

Note. This checklist is intended as a structured aid to multidimensional clinical formulation and initial assessment. It is not a validated diagnostic scale, screening instrument, prognostic tool, or therapeutic algorithm. The listed tools are domain-specific examples: Conners/ADHD-RS for ADHD symptoms, YGTSS for tic severity, and EDE/EDE-Q for eating disorder psychopathology. The EDE is a semi-structured clinician interview, whereas EDE-Q is self-report; the two may diverge, and EDE-Q should not be treated as interchangeable with the interview or as a diagnosis on its own [68,69]. Neither standard measure is a general assessment of food selectivity, sensory feeding difficulties, or ARFID. Suspected ARFID requires separate assessment against DSM-5-TR criteria [1]. A dedicated interview such as the PARDI may be considered where age-appropriate and available; the cited validation evidence is from children, adolescents, and young adults and does not establish validity in the target population [70]. The examples should be selected according to age, clinical context, assessment objectives, local availability, and clinical judgement. Abbreviations. ARFID, avoidant/restrictive food intake disorder; ADI-R, Autism Diagnostic Interview-Revised; ADOS-2, Autism Diagnostic Observation Schedule, Second Edition; ADHD, attention-deficit/hyperactivity disorder; ADHD-RS, ADHD Rating Scale; ASD, autism spectrum disorder; CAT-Q, Camouflaging Autistic Traits Questionnaire; ED, eating disorder; EDE, Eating Disorder Examination; EDE-Q, Eating Disorder Examination Questionnaire; IQ, intelligence quotient; PARDI, Pica, ARFID and Rumination Disorder Interview; OCD, obsessive-compulsive disorder; RRB, restricted and repetitive behaviours; SRS-2, Social Responsiveness Scale, Second Edition; WAIS-IV, Wechsler Adult Intelligence Scale, Fourth Edition; WISC-V, Wechsler Intelligence Scale for Children, Fifth Edition; YGTSS, Yale Global Tic Severity Scale.

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One flexible workflow is to clarify the referral question, developmental history, and context, review diagnostic status and co-occurring or differential conditions using their own criteria, map strengths and functional needs across domains, then prioritise further assessment and support with the young person and family. Overlap does not imply causation—keep discrepancies linked to source, setting, and timeframe.

Educational, peer, and family context. For children and adolescents, the formulation should describe participation in classroom learning, transitions, peer interactions, and home routines, alongside individual strengths and needs. Record the young person's account and caregiver/educator observations separately, linking each to setting, timeframe, and environmental expectations, such as task structure, sensory or social demands, and available support. Contextual differences may help explain why a need is visible in one setting but not another—they do not establish causation. Family circumstances should be recorded when relevant to access to support or the feasibility of a plan, without being treated as an explanation for autistic features. Peer interactions should be interpreted in context and as reciprocal, consistent with the discussion of communication between autistic and non-autistic people in Section 4.3. These are proposed formulation prompts that extend the family and educational context already described in the NICE CG128 discussion above; their effects on educational participation or family outcomes have not been tested.

Interdisciplinary communication and accommodations. The matrix may offer a shared summary for discussion among autism clinicians, the young person, family members, and, when relevant, school staff or other educators. Such a summary could communicate strengths and needs, the context and source of each account, uncertainty, questions for further assessment, and priorities for support, allowing clinical and educational perspectives to be considered alongside one another. In discussion with the young person and family, and, where appropriate, with education staff, the team may identify environmental changes to consider, such as written task steps, transition preparation, or a quieter recovery space, as illustrated in the hypothetical examples below. Any accommodation remains an individualised proposal to be reviewed with the person and the relevant team; the map has not been shown to improve communication, accommodation use, or outcomes.

Fictional examples (illustrative only; not validation evidence). The scenarios below are hypothetical, not patient material, and show formulation/support-planning questions rather than evidence of validity or utility.

Example 1. A 12-year-old with established ASD and fluent speech does well in a preferred subject yet reports difficulty organising multistep school tasks and recovering after noisy group work; caregivers describe similar needs, while school staff mainly see confident verbal participation. Formulation could record strengths alongside executive/adaptive demands, sensory context, and differences between accounts. Follow-up might assess adaptive functioning and consider written task steps or transition preparation, with the student's views guiding review.

Example 2. A 16-year-old with established ASD and fluent functional language reports rehearsing social exchanges and fatigue after school; family members observe fewer difficulties at home. The clinician records each account by source, setting, and timeframe, then considers pragmatic demands, reported fatigue/recovery, and sensory load as related but distinct formulation domains—camouflaging is assessed rather than inferred. Possible support needs to explore include difficulty with unstructured group tasks and recovery after demanding school periods. Written task steps or access to a quieter recovery space could be discussed with the adolescent and reviewed individually.

4.10. Assessment Tools and Measurement Limitations

ADOS-2 and ADI-R findings should be integrated with developmental history and clinical assessment rather than interpreted in isolation, particularly when evaluating adults without intellectual disability [71]. The CAT-Q measures self-reported camouflaging; however, observational and self-report approaches to the construct are not equivalent and may yield different results [45,72]. The discrepancy between IQ and Vineland scores may indicate a gap between cognitive ability and everyday adaptive functioning but is not diagnostic in itself. Instruments should be selected according to age, clinical context, assessment aim, local availability, validation in the target population, and clinical judgement. The checklist has not been validated and is intended as a structured aid to clinical formulation rather than as a diagnostic scale; the requirements for its future empirical evaluation are described in the section "Future research directions and empirical validation".

4.11. Methodological and Conceptual Limitations

The principal limitation is methodological. This is a purposive descriptive-narrative review informed by indexed literature and clinical experience; it did not use systematic screening, prespecified eligibility coding, or formal risk-of-bias or evidence-certainty assessment. It, therefore, cannot provide prevalence, prognostic, or diagnostic-accuracy estimates. The profiles are non-mutually exclusive and describe constructs at different conceptual levels. The framework's reliability, validity, and incremental clinical utility remain untested, and the proposed matrix should be treated as a formulation hypothesis open to participatory and empirical revision.

A further methodological limitation concerns potential selection bias in the choice of included profiles. Since selection was based on clinical relevance, recurrence in the literature, and usefulness for multidimensional formulation, more established or widely described profiles, such as ADHD, camouflaging/social fatigue, or eating disorders, may be more strongly represented than emerging, controversial, or less-studied profiles that may nevertheless be relevant in clinical practice. In addition, the narrative nature of the review does not exclude the possibility that other clinical-phenotypic domains may be identified in future systematic reviews, Delphi studies, or empirical analyses of large clinical samples.

Evidence must be distinguished from the organisation proposed here. Supplementary Table S5 summarises source-specific empirical findings by design, studied population, and limitations, and these findings support claims only within the samples and measures used. The selection of ten profiles, their arrangement in a multidimensional matrix, and suggested relationships across domains are the authors' conceptual synthesis and have not been tested as a whole. The literature varies in directness and strength, particularly for debated constructs such as PDA/demand avoidance—source counts do not indicate evidence quality. No formal risk-of-bias or certainty grading was conducted.

Across the mapped studies, convergence is limited to a broad assessment implication: single indicators or isolated behaviours may not resolve functional impact, diagnostic status, or the need for further assessment. The evidence does not converge on a single phenotype or developmental mechanism. Apparent contradictions are difficult to interpret because the studies address different constructs and populations: SRS-defined ASD-related traits in Tourette samples are not clinical ASD diagnoses and may overlap with tic/OCD symptoms, eating behaviour overlap cannot distinguish co-occurrence from shared or look-alike features, studies of demand-avoidant/PDA-like features do not establish a stable independent construct, and sensory group differences do not test the proposed cross-domain role of sensory load. As summarised in Supplementary Table S5,

the sources also vary in design, recruitment, informant, measures, age, and fit to the target population. Their results cannot be pooled or reconciled by counting studies, and the review did not conduct formal risk-of-bias or evidence-certainty grading. The appropriate implication is to use the profiles as prompts for individualised assessment and formulation, while retaining uncertainty about their boundaries, developmental relations, and comparative support—the map does not show that all profiles are equally established or mutually exclusive.

Age coverage and directness to the target group also vary by domain. The most direct paediatric/adolescent samples among the mapped principal sources concern A3 (ages 6–19) and A4 (ages 1–18), although neither was defined by the target-population combination. C2's review mainly included autistic children and adolescents, but its studies were heterogeneous, and the sensory evidence includes a cross-sectional child sample aged 5–17. In B2, the child–adult contrast concerns SRS-defined probable ASD features in a Tourette sample, not clinician-confirmed ASD diagnoses. Adult evidence includes A1's follow-up cohort (mean age 24 at assessment, with a broad intellectual range) and C1's qualitative online study of adults. Neither provides age-stratified evidence for the defined target population, and C1 cannot establish childhood/adolescent course. A2 and B1 do not provide age-stratified primary cohorts. The principal sources for C3 do not support an age-stratified comparison across the full range of feeding/eating difficulties, and C4 pools mixed populations without age-stratified inference. These differences describe age coverage and design in selected sources, not comparative certainty: evidence was not formally graded by domain, and the principal-source map is non-exhaustive. Adult findings may inform questions for later-life assessment but should not be extrapolated as evidence of childhood manifestations; paediatric findings likewise do not establish adult applicability.

Representing sensory reactivity as a single cross-cutting dimension is a simplification. Future work should examine its relationships with fatigue, avoidance, food selectivity, and adaptive functioning.

The selection process also risks circularity, as discussed in Section 2.2. Independent assessment is needed to determine whether the map offers value beyond the clinical assumptions that shaped it.

Several elements are, therefore, especially provisional and open to revision: the completeness of the selected profile set, the allocation of profiles to three organising domains, the choice and interpretation of the matrix axes, the current representation of sensory reactivity as a single cross-cutting dimension, the inclusion and interpretation of demand-avoidant/PDA-like features, the grouping of camouflaging and social fatigue within C1, and "ASD-like phenotypic convergence" as a descriptive, unvalidated proposal. These points concern selection and conceptual organisation and do not imply equal diagnostic or evidential status across the underlying phenomena. Participatory and empirical review may lead to revised definitions, axes, groupings, or exclusions.

A different kind of limitation concerns the absence of direct participation by autistic people. The map was developed by clinicians, for clinicians, without participatory involvement or systematic consultation of first-person sources. This asymmetry is particularly relevant because first-person accounts and autistic scholarship have contributed to concepts such as autistic burnout and monotropism [47,73]. The literature on terminology also shows that community preferences do not always coincide with academic usage [74,75]. A subsequent version of the proposal should include a participatory consultation phase before any Delphi study among clinicians.

Finally, it should be acknowledged that some authors question whether descriptive subgrouping has adequately resolved autism's heterogeneity [76]. The present proposal does not dispute that assessment at the level of aetiological research; rather, it

acknowledges it and explicitly limits its claim to the level of individual clinical formulation.

4.12. Distinctive Conceptual Contribution

The framework's conceptual contribution lies in arranging selected, previously described profiles into a single descriptive matrix: functional profiles, co-occurring conditions and diagnostic relationships, and adaptive, psychopathological, or identity-related trajectories and associations. This makes their differing roles visible during case-level synthesis while preserving their non-hierarchical status. The framework introduces no new diagnosis or causal mechanism; its conceptual coherence and completeness remain open to empirical and participatory review.

The matrix (Figure 1), checklist, and supplementary tables have complementary roles: Table 1 provides profile-level descriptors, Figure 1 summarises domain relationships and developmental/contextual prompts, Table 2 lists indicative assessment tools, and the supplements provide expanded descriptions, conceptual distinctions, and an evidence map. Their anticipated value as a shared language for clinicians, autistic people, families, and schools remains to be tested. Section 4.9 describes possible clinical uses, and Section 4.13 specifies future validation questions.

4.13. Future Research Directions and Empirical Validation

For the proposal to be testable, and not merely plausible, future studies should prespecify the domains, outcomes, and criteria by which its reliability and clinical usefulness will be evaluated. Poor inter-rater agreement in assigning domains would indicate that the current operational definitions require revision. Associations between specific domains and external measures should be examined using prespecified outcomes and interpreted based on effect estimates, confidence intervals, and precision, rather than statistical significance alone.

Hypotheses concerning individual domains should be tested at the level to which they apply. For example, associations between camouflaging and discrepancies between cognitive ability and adaptive functioning would primarily inform the validity of the social fatigue/camouflaging profile, rather than domain C as a whole. Similarly, the incremental contribution of sensory measures should be evaluated across relevant outcomes, such as fatigue, avoidance, food selectivity, and adaptive functioning, rather than inferred from a single outcome.

Candidate operational indicators should be recorded by profile, with the information source, context, and functional impact noted, rather than collapsed into a total score. For domain A, examples include profile-specific evidence of pragmatic communication, cognitive or neuropsychological strengths and difficulties, and their everyday adaptive implications. For domain B, record whether each proposed co-occurring condition meets its own diagnostic criteria and how it affects functioning. For domain C, document reported camouflaging strategies separately from fatigue or possible burnout, the context and function of demand-avoidant behaviour, feeding/eating features separately from any criteria-based diagnosis, and gender identity/incongruence separately from associated distress or impairment. Sensory reactivity may be recorded across domains by modality, setting, impact, and report source. These are candidate descriptive indicators for future operationalisation, not validated items, scores, or thresholds.

Evidence that the proposed domains largely collapse onto a general severity dimension, or that the framework provides little clinically meaningful information beyond already replicated empirical subgroups, would call into question the added value of the map. Such conclusions should, however, be based on adequately powered studies,

predefined criteria for clinically meaningful incremental value, and, where possible, replication across independent samples [22,23].

Stage 1—Participatory refinement. Begin with accessible consultation involving autistic people and family members, with separate opportunities to contribute where appropriate; use this input to refine domain content, definitions, and clinical relevance. A subsequent Delphi study conducted according to transparent standards could assess expert consensus and content validity [77].

Autistic participation should inform content decisions, not only acceptability, and family input should complement, not replace, autistic participants' own perspectives. Consultation should invite autistic people to assess each domain's relevance and distinctiveness, identify omissions or overlaps, propose additions, removals, splits, or mergers, shape definitions and examples, identify strengths and support priorities, and review terminology. Family members can contribute contextual observations about support needs and change across settings, particularly for children and adolescents—record these reports separately from first-person accounts. Autistic people and family representatives should also be invited to interpret feasibility findings and the usefulness of resulting support plans. Include varied communication and support needs and record disagreement rather than claiming consensus. These are proposed procedures; they were not undertaken in the present review.

Stage 2—Feasibility. After participatory refinement, pilot the checklist in its intended use as a structured aid to clinical formulation rather than as a psychometric scale. Clinicians should trial the workflow, with autistic people and family members invited, where relevant, to comment on clarity, burden, acceptability, and relevance. Examine completion rates, missing items, additional time required, and fit with clinical workflow. Principles from measurement-property frameworks may be adapted where appropriate to the type of information collected and the intended clinical use of the checklist [78], without assuming a total score, a single latent factor, or internal consistency across heterogeneous domains.

Stage 3—Reliability and domain-specific measurement properties. After feasibility work, assess inter-rater agreement by asking independent clinicians to apply the framework to the same clinical cases and using agreement statistics appropriate to the type of data generated. Test–retest stability should be considered only for domains expected to remain relatively stable over the assessment interval, whereas convergent validity should be assessed separately for individual domains using independent external measures. Criteria for subtype validation in psychiatry [25] would become relevant only if future empirical work identifies discrete and reproducible subgroups. This approach preserves the heuristic and formulation-oriented nature of the proposal while avoiding premature treatment of the checklist as a validated diagnostic or psychometric scale.

Stage 4—Structural examination. After feasibility and reliability have been assessed, examine the empirical structure of the framework in multicentre clinical samples of autistic people without intellectual disability and with fluent functional language, integrating developmental, diagnostic, neuropsychological, adaptive, sensory, and psychopathological data. Factor-analytic approaches could be used to examine the structure of measured dimensions, whereas cluster analysis, latent class analysis, or latent profile analysis could be used to investigate whether reproducible subgroups of individuals emerge from the data. These analyses address different questions and should not be treated as interchangeable [16]. The incremental contribution of sensory load should be tested separately using a prespecified clinical outcome and a multivariable model including camouflaging and other theoretically justified covariates. Effect estimates, confidence intervals, and model performance should be considered when evaluating added value. Comparison with empirically replicated subgroups could then

assess whether the proposed framework provides clinically relevant information beyond existing data-derived classifications. The analytical strategy described here represents a proposal for future validation and should not be attributed directly to the cited methodological literature.

Factor-analytic and subgroup analyses may inform the framework's empirical structure and the reproducibility of patterns, but they do not, by themselves, establish added clinical value in practice. That requires a separate clinical utility evaluation against the usual multidimensional assessment, as specified below.

Stage 5—Clinical utility. The framework's ability to identify clinically relevant differences in support needs must be evaluated empirically rather than assumed. Incremental utility should be assessed against usual multidimensional assessment, not against the absence of structured assessment. A study could compare case formulations based solely on guideline-concordant usual assessment with those based on the same assessment supplemented by the map. For children and young people, the comparator could follow the multidimensional approach described in NICE CG128 (see Clinical Implications); adult services would require a separately specified, age-appropriate comparator. The question is whether the map provides clinically meaningful information beyond routine assessment, not whether it can replace routine assessment. Candidate outcomes include clinically relevant needs identified beyond usual assessment, completeness and consistency of formulations, appropriateness of referrals or support recommendations, additional assessment and formulation time, and the perceived usefulness of support plans. Definitions and assessment criteria should be prespecified, with independent reviewers assessing needs and recommendations where feasible. Autistic people and family members should contribute to interpreting the usefulness of support plans. These are proposed outcomes, not validated measures; improved process ratings alone would not establish better clinical outcomes.

5. Conclusions

The matrix proposed in this review organises selected clinical-phenotypic profiles into three parallel conceptual domains and makes their differing status explicit: functional profiles, co-occurring conditions and diagnostic relationships, and adaptive, psychopathological, or identity-related trajectories and associations. Sensory reactivity is cross-cutting. The framework is intended to structure discussion of strengths, everyday functioning, context, and further assessment when observable competence may obscure support needs. It adds no diagnostic criteria and does not define ASD subtypes.

"ASD-like phenotypic convergence" remains a provisional descriptive label for presentation-level resemblance across different developmental or diagnostic histories. Within the framework, it prompts review of developmental history, context, and everyday functioning—it establishes neither a separate category nor a shared causal process.

Independent studies should assess content validity, reliability, and incremental usefulness beyond usual multidimensional assessment for case formulation and support planning. Until such evidence is available, the anticipated clinical benefit remains unestablished. Poor reliability or an absence of clinically meaningful added value would warrant revision before wider use.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/children13121667/s1. Supplementary Table S1: Expanded profile descriptions, developmental/recognition periods, and possible implications. Supplementary Table S2: Domain-level summary of the three organising domains and cross-cutting characteristics. Supplementary Table S3: Profile-specific relationship to the target population and critical points.

Supplementary Table S4: Conceptual distinctions among late-recognised ASD, ASD-like adaptation, co-occurrence, diagnostic overlap, and diagnostic conversion. Supplementary Table S5: Selected evidence map of principal sources by profile, design, population, findings, and limitations. References [6,12,26,29,30,32,33,36,38,39,51,52,56–59,79] are cited in the supplementary materials.

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Abbreviations

ADHD, attention-deficit/hyperactivity disorder; ADI-R, Autism Diagnostic Interview-Revised; ADOS-2, Autism Diagnostic Observation Schedule, Second Edition; AS, Asperger syndrome (historical use); ASD, autism spectrum disorder; CAT-Q, Camouflaging Autistic Traits Questionnaire; DSM-5-TR, Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision; ED, eating disorder; GD/GI, gender dysphoria/gender incongruence; HFA, high-functioning autism (historical use); IQ, intelligence quotient; NLD/NVLD, nonverbal learning disorder/disability; OCD, obsessive-compulsive disorder; PDA, pathological demand avoidance; RRB, restricted and repetitive behaviours; SCD, social (pragmatic) communication disorder.

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