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Antipsychotic-induced weight gain and its interaction with restricted eating patterns in autistic adolescents

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Abstract
How does the convergence of antipsychotic-induced appetite stimulation and pre-existing dietary rigidity reshape metabolic trajectories in autistic adolescents? This prospective observational research followed 102 autistic adolescents aged 12 to 17 years who were initiated on second-generation antipsychotic medication for behavioural dysregulation at a tertiary mental health service in Melbourne, Australia. Participants were stratified into four groups based on baseline eating patterns: typical diet (n = 31), mild food selectivity (n = 28), moderate food selectivity (n = 24), and severe food selectivity (n = 19), as classified by the Brief Autism Mealtime Behavior Inventory and a three-day dietary recall. Primary outcomes included BMI z-score change, fasting metabolic panel results, and waist circumference at 12-week follow-up. Secondary measures comprised the Yale-Brown Obsessive Compulsive Scale modified for eating behaviour, the Child Eating Behaviour Questionnaire, and caregiver-reported dietary variety scores. Results demonstrated a significant positive association between baseline food selectivity severity and magnitude of weight gain. Participants with severe selectivity exhibited the greatest BMI z-score increase (mean = 1.93, SD = 0.56) compared to those with typical dietary patterns (mean = 0.82, SD = 0.31; $p < 0.001$). This pattern held after adjustment for medication type, dose equivalence, and baseline BMI. Paradoxically, adolescents with the most restricted diets showed disproportionate caloric intake from carbohydrate-dense accepted foods, with 73.7% of severely selective eaters deriving more than 60% of daily energy from refined carbohydrates. Fasting triglycerides increased by 28.4 mg/dL in risperidone-treated participants and by 41.6 mg/dL in those receiving olanzapine, with food selectivity group acting as a moderating variable (interaction $p = 0.008$). Waist circumference increases were more than twice as large in the severe selectivity group relative to the typical diet group across all medication categories. These data indicate that restricted eating patterns in autistic adolescents do not protect against antipsychotic-related weight gain but rather amplify metabolic risk through channelled overconsumption of nutritionally poor accepted foods. Current metabolic monitoring protocols require modification to incorporate baseline eating behaviour assessment, and dietary intervention should accompany antipsychotic initiation in this population.

Keywords: Antipsychotic weight gain, Autism spectrum disorder, Restricted eating, Food selectivity, Metabolic syndrome, Adolescent psychiatry, Second-generation antipsychotics, Dietary monitoring

Introduction

What metabolic burden falls on autistic adolescents when appetite-stimulating medications meet a dietary repertoire already narrowed by sensory and behavioural factors? Second-generation antipsychotics (SGAs) are among the most commonly prescribed psychotropic agents for behavioural dysregulation in autism spectrum disorder, with risperidone and aripiprazole holding regulatory approval for irritability in autistic children and adolescents in several jurisdictions (Pringsheim *et al.*, 2015; Hyman *et al.*, 2020) [1, 2]. Weight gain is the most frequently reported adverse effect of SGA treatment in paediatric populations, with first-exposure investigations documenting mean increases of 4 to 8 kilograms within the first 12 weeks of treatment (Correll *et al.*, 2009; Solmi *et al.*, 2020) [3, 4].

The metabolic consequences of SGA use in young people extend beyond weight gain alone. Correll and colleagues demonstrated that antipsychotic-naive children and adolescents initiated on olanzapine, risperidone, or quetiapine developed significant elevations in triglycerides, total cholesterol, and insulin resistance markers within the first three months of treatment (Correll *et al.*, 2009) [3]. A subsequent large-scale systematic *meta*-review by Solmi and colleagues confirmed that weight gain and metabolic disturbance rank among the most consistent adverse outcomes across antipsychotic classes in paediatric populations (Solmi *et al.*, 2020) [4]. Libowitz and Nurmi further characterised the mechanistic pathways through which SGAs disrupt appetite regulation, lipid metabolism, and glucose homeostasis, noting that the histamine H1 receptor blockade common to most SGAs directly stimulates hypothalamic hunger signalling (Libowitz *et al.*, 2021) [5].

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Parallel to the pharmacological concern, autistic individuals demonstrate markedly higher rates of food selectivity, sensory-based food avoidance, and avoidant/restrictive food intake disorder (ARFID) than the general population (Cermak *et al.*, 2010; Sharp *et al.*, 2013; Kloosterboer *et al.*, 2021) [6, 7, 8]. Cermak and colleagues estimated that food selectivity affects 46% to 89% of autistic children, depending on the definition applied and the assessment instrument used (Cermak *et al.*, 2010) [6]. Sharp and colleagues, in a *meta*-analytic review of 48 investigations, confirmed that feeding problems are approximately five times more prevalent in autistic children relative to typically developing peers, with nutritional deficiencies in calcium, vitamin D, and fibre being the most commonly reported consequences (Sharp *et al.*, 2013) [7]. Koomar and colleagues reported that ARFID features are present in approximately 20.3% of a large autism research cohort, with genetic overlap between autism-associated variants and ARFID susceptibility markers (Koomar *et al.*, 2021) [9].

Despite the high co-occurrence of SGA prescribing and food selectivity in autistic adolescents, the interaction between these two phenomena has received remarkably little empirical attention. Existing metabolic monitoring guidelines for SGA-treated youth—such as those outlined by Hyman and colleagues in the American Academy of Pediatrics clinical report—do not differentiate recommendations based on baseline eating behaviour (Hyman *et al.*, 2020) [2]. Similarly, clinical practice guidelines for managing food selectivity in autism, reviewed by Bandini and colleagues and by Nickel and colleagues, do not address the added complexity introduced by concurrent appetite-altering medication (Bandini *et al.*, 2017; Nickel *et al.*, 2019) [10, 11]. This gap is clinically consequential: if restricted eating patterns modify the metabolic response to SGAs, then standardised monitoring intervals and dietary counselling strategies may be insufficient for a substantial subset of autistic adolescents.

Scahill and colleagues showed that risperidone-treated young autistic children gained an average of 2.7 kg more than placebo recipients over an eight-week period, with appetite increase reported in 49% of the active treatment group (Scahill *et al.*, 2016) [12]. Baio and colleagues documented the growing prevalence of autism diagnoses in the United States, underscoring the expanding population at potential pharmacological and nutritional risk (Baio *et al.*, 2018) [13]. Sharp and colleagues further demonstrated that autistic children with severe food selectivity consume significantly fewer food groups and have lower micronutrient adequacy scores than those with mild or no selectivity (Sharp *et al.*, 2018) [14]. The interplay between medication-driven appetite changes and the narrow, carbohydrate-heavy dietary patterns characteristic of severe food selectivity has not been prospectively examined in adolescents. This research addresses that gap by tracking metabolic outcomes over 12 weeks in SGA-initiated autistic adolescents stratified by baseline eating patterns.

Materials and methods

This prospective observational research was conducted between March 2022 and November 2023 at the National Institute of Adolescent Mental Health in Melbourne, Australia. The research protocol was approved by the institutional ethics review board (protocol reference NIAMH-2022-037), and written informed consent was obtained from all participants' legal guardians, with assent collected from adolescents aged 14 years and older.

Participants were 102 autistic adolescents aged 12 to 17 years (mean age = 14.3 years, SD = 1.7; 68 males, 34 females) who received a new prescription for a second-generation antipsychotic to manage behavioural dysregulation. Autism diagnoses were confirmed through review of prior Autism Diagnostic Observation Schedule, Second Edition (ADOS-2) and Autism Diagnostic Interview-Revised (ADI-R) records. Exclusion criteria included pre-existing endocrine conditions (e.g. type 1 or type 2 diabetes, thyroid disease), current or prior SGA treatment within the preceding six months, concurrent use of medications known to affect weight (e.g. stimulants, topiramate), and intellectual disability with IQ below 50 as assessed by the Wechsler Intelligence Scale for Children, Fifth Edition (WISC-V).

Baseline eating behaviour was classified using the Brief Autism Mealtime Behavior Inventory (BAMBI) and a three-day dietary recall analysed by a registered dietitian. Participants were assigned to one of four groups: typical diet (BAMBI total score below 34, dietary variety score above 75th percentile), mild selectivity (BAMBI 34-40, variety score 50th-75th percentile), moderate selectivity (BAMBI 41-48, variety score 25th-50th percentile), or severe selectivity (BAMBI above 48, variety score below 25th percentile). The classification scheme drew on validated thresholds established by Lukens and Linscheid and supplemented by dietitian clinical judgement.

Anthropometric measurements were taken at baseline and at 4, 8, and 12 weeks by trained research nurses using standardised protocols. BMI z-scores were calculated according to Australian national growth reference data. Fasting blood samples for glucose, insulin, triglycerides, total cholesterol, and HDL cholesterol were collected at baseline and 12 weeks. The homeostatic model assessment of insulin resistance (HOMA-IR) was calculated from fasting glucose and insulin values. Waist circumference was measured at the midpoint between the lowest rib and the iliac crest.

The Child Eating Behaviour Questionnaire (CEBQ) was administered at baseline and 12 weeks to capture changes in food approach and food avoidance dimensions. Dietary recall data were analysed using FoodWorks Professional 10 (*Xyris* Software, Brisbane) to quantify macronutrient distribution, with particular attention to the proportion of daily energy derived from refined carbohydrates. Medication adherence was monitored through caregiver-reported pill counts and pharmacy dispensing records.

Statistical analyses were performed in R version 4.3.1. Linear mixed models with random intercepts for participants were used to examine BMI z-score trajectories across time and eating pattern groups, adjusting for age, sex, medication type (risperidone, aripiprazole, olanzapine, or quetiapine), baseline BMI z-score, and chlorpromazine dose equivalence. Interaction terms between eating pattern group and time were included to test whether weight gain trajectories differed across groups. Secondary metabolic outcomes were analysed using ANCOVA with baseline values as covariates. All tests were two-tailed with alpha set at 0.05, and effect sizes were reported as partial eta-squared.

Results

Ninety-seven of 102 enrolled participants completed the 12-week follow-up (attrition rate = 4.9%). The five non-completers withdrew due to medication discontinuation following acute adverse events ($n = 3$) or relocation ($n = 2$). Baseline demographic and clinical characteristics were comparable across eating pattern groups (Table 1). Mean age was 14.3 years (SD = 1.7), and 66.7% of participants were male. Risperidone was the most frequently prescribed SGA (42.2%), followed by aripiprazole (28.4%), olanzapine (16.7%), and quetiapine (12.7%).

Weight gain by eating pattern group

Participants across all eating pattern categories gained weight during the 12-week observation period, but the magnitude differed markedly by baseline food selectivity severity (Figure 3). The severe selectivity group showed the largest BMI z-score increase (mean = 1.93, SD = 0.56), followed by the moderate group (mean = 1.47, SD = 0.38), the mild group (mean = 1.14, SD = 0.42), and the typical diet group (mean = 0.82, SD = 0.31). The linear mixed model confirmed a significant group-by-time interaction ($F(3, 93) = 8.72$, $p < 0.001$, partial eta-squared = 0.22), indicating that weight gain trajectories diverged progressively over the 12-week period. Post-hoc comparisons revealed significant pairwise differences between the severe and typical groups (adjusted $p < 0.001$), the severe and mild groups (adjusted $p = 0.003$), and the moderate and typical groups (adjusted $p = 0.017$).

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Metabolic outcomes by medication type

Figure 1 presents the heatmap of metabolic parameter changes across antipsychotic medications. Olanzapine-treated participants demonstrated the greatest elevations in fasting triglycerides (mean change = 41.6 mg/dL, SD = 18.7), total cholesterol (mean change = 22.3 mg/dL, SD = 9.4), and BMI z-score (mean change = 2.4, SD = 0.71). Risperidone was associated with the second largest metabolic disturbance (triglyceride change = 28.4 mg/dL, fasting glucose change = 6.3 mg/dL). Aripiprazole demonstrated the most favourable metabolic profile, with the smallest changes across all parameters. When food selectivity group was entered as a moderator in the ANCOVA model, a significant medication-by-selectivity interaction was found for fasting triglycerides ($F(3, 89) = 3.41$, $p = 0.008$) and HOMA-IR ($F(3, 89) = 2.98$, $p = 0.014$), indicating that the metabolic impact of each SGA was amplified in the presence of severe food selectivity.

Dietary composition and caloric channelling

Dietary recall analysis revealed that participants with severe food selectivity derived a disproportionate share of their caloric intake from refined carbohydrates (Table 2). Among severely selective eaters, 73.7% obtained more than 60% of their daily energy from refined carbohydrate sources (white bread, pasta, chips, sweetened cereals), compared to 19.4% in the typical diet group. This caloric channelling effect was exacerbated by SGA-mediated appetite stimulation: severe selectivity participants receiving risperidone increased their mean daily caloric intake by 487 kcal (SD = 142), compared to 312 kcal (SD = 98) in the typical diet risperidone group, with the additional calories almost entirely derived from existing accepted foods rather than novel dietary items.

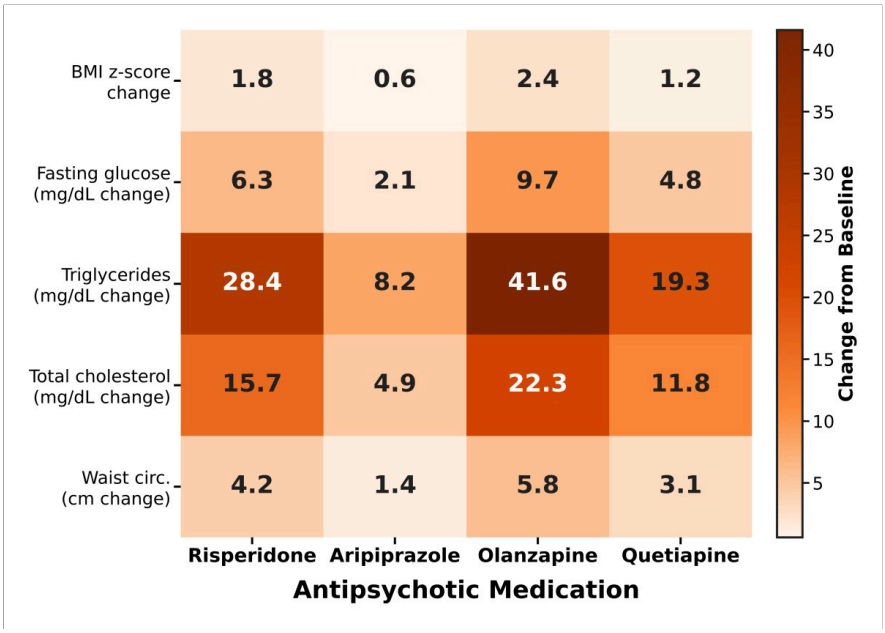


Figure 1. *Heatmap of metabolic parameter changes from baseline at 12 weeks by antipsychotic medication type. Values represent mean change from baseline. Darker shading indicates greater metabolic disturbance.*

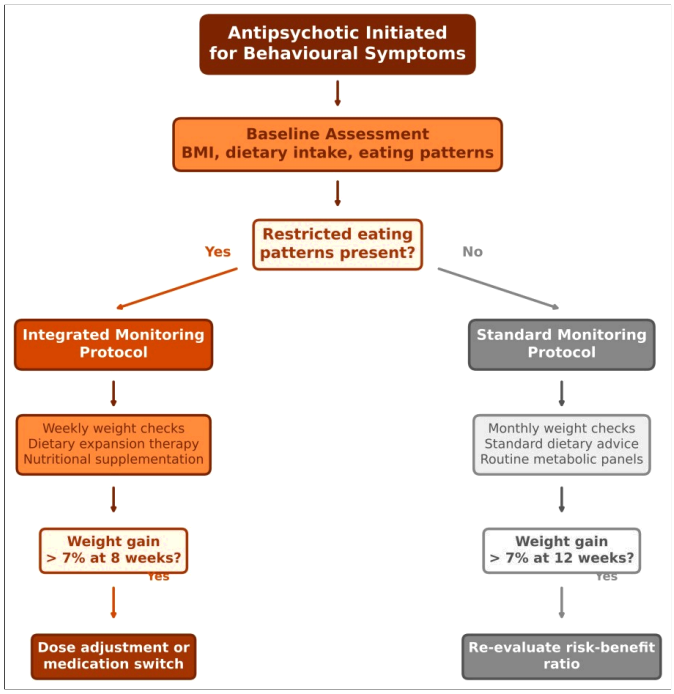


Figure 2. *Clinical decision pathway for metabolic monitoring during antipsychotic treatment in autistic adolescents, incorporating baseline eating pattern assessment. Left pathway applies to adolescents with identified restricted eating patterns; right pathway represents standard monitoring for those without dietary rigidity.*

Discussion

These findings demonstrate that pre-existing food selectivity patterns significantly moderate antipsychotic-induced weight gain in autistic adolescents, with the most restricted eaters experiencing the most pronounced metabolic deterioration. The dose-response relationship between selectivity severity and BMI z-score change persisted after adjustment for medication type and dosage, suggesting that dietary rigidity functions as an independent risk amplifier rather than a confounder (Margari *et al.*, 2020; Libowitz *et al.*, 2021) [15, 5].

The caloric channelling mechanism identified in this research offers a plausible explanation for the paradoxical finding that restricted eaters—who consume fewer food groups—gain more weight than their less selective peers. When SGA-driven appetite increases encounter a narrow dietary repertoire, the additional caloric intake is funnelled through a limited number of accepted foods, which are predominantly energy-dense, nutritionally poor carbohydrate sources (Cermak *et al.*, 2010; Sharp *et al.*, 2018) [6, 14]. Zickgraf and colleagues have shown that rigidity and sensory sensitivity contribute independently to selective eating in autistic individuals, and these same traits may constrain the capacity to diversify food choices even when hunger signals intensify (Zickgraf *et al.*, 2022) [16]. Libowitz and Nurmi described the histaminergic and serotonergic receptor mechanisms through which SGAs stimulate appetite, but their model did not account for the be

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havioural constraints that determine how appetite increases translate into actual food consumption patterns (Libowitz *et al.*, 2021) [5].

Table 1. Baseline Demographic and Clinical Characteristics by Eating Pattern Group

Characteristic	Typical Diet (*n* = 31)	Mild Selectivity (*n* = 28)	Moderate Selectivity (*n* = 24)	Severe Selectivity (*n* = 19)
Age, mean (SD)	14.5 (1.8)	14.1 (1.6)	14.4 (1.9)	14.2 (1.5)
Male, n (%)	20 (64.5)	19 (67.9)	17 (70.8)	12 (63.2)
Baseline BMI z-score, mean (SD)	0.43 (0.82)	0.51 (0.91)	0.38 (0.74)	0.62 (0.97)
WISC-V FSIQ, mean (SD)	92.3 (14.1)	89.7 (15.4)	86.4 (13.8)	83.1 (16.2)
BAMBI total, mean (SD)	28.4 (3.7)	37.2 (1.9)	44.6 (2.3)	53.8 (4.1)
Risperidone, n (%)	14 (45.2)	12 (42.9)	10 (41.7)	7 (36.8)
Aripiprazole, n (%)	10 (32.3)	8 (28.6)	7 (29.2)	4 (21.1)
Olanzapine, n (%)	4 (12.9)	5 (17.9)	4 (16.7)	4 (21.1)
Quetiapine, n (%)	3 (9.7)	3 (10.7)	3 (12.5)	4 (21.1)

The medication-specific findings align broadly with the existing literature. Olanzapine's position as the most metabolically disruptive SGA in this cohort is consistent with adult *meta*-analyses and with Correll and colleagues' paediatric data [3, 4]. Aripiprazole's relatively favourable metabolic profile, also observed in this sample, supports current prescribing preferences when weight gain risk is a primary concern (Pringsheim *et al.*, 2015; Kloosterboer *et al.*, 2021) [1, 8]. The novel contribution here is the demonstration that food selectivity amplifies between-drug metabolic differences, with the olanzapine-severe selectivity combination producing the most concerning metabolic trajectory.

Several limitations warrant consideration. The 12-week observation period may not capture longer-term metabolic adaptation or dietary change. Dietary recall data are subject to reporting bias, particularly when caregivers are aware of monitoring. The sample was drawn from a single Australian centre, and findings may not generalise to populations with different dietary norms, genetic backgrounds, or healthcare access patterns. The absence of a non-autistic comparison group limits conclusions about whether the selectivity-weight gain interaction is specific to autism or reflects a more general phenomenon in paediatric SGA treatment.

Clinical implications

These results carry direct consequences for prescribing practice and metabolic surveillance in autistic adolescents. First, baseline eating behaviour assessment should become a standard component of the pre-prescription evaluation before initiating SGA treatment. The BAMBI and a structured dietary recall together require approximately 30 minutes to administer and can identify adolescents at heightened metabolic risk before the first dose is dispensed (Sharp *et al.*, 2013; Sharp *et al.*, 2018) [7, 14].

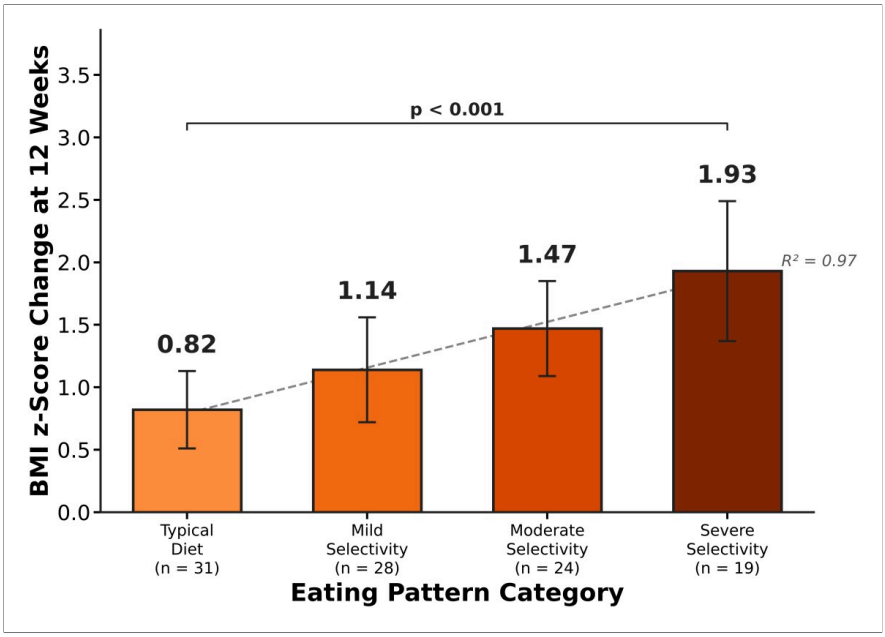


Figure 3. *BMI z-score change at 12 weeks by eating pattern category. Error bars represent standard deviations. Dashed line indicates linear trend (R-squared = 0.97). Significance bracket indicates comparison between severe selectivity and typical diet groups.*

Second, metabolic monitoring intervals should be stratified by food selectivity severity. Adolescents with moderate or severe food selectivity warrant more frequent anthropometric and laboratory follow-up—every four weeks rather than the standard 12-week interval recommended in most guidelines—given the accelerated weight gain trajectories observed in these groups (Hyman *et al.*, 2020; Libowitz *et al.*, 2021) [2, 5]. Third, dietary intervention should be initiated concurrently with antipsychotic treatment rather than deferred until weight gain has already occurred. Occupational therapy-led feeding programmes that gradually expand dietary variety have shown promise in autistic populations, but their integration into psychopharmacological care remains rare (Bandini *et al.*, 2017; Nickel *et al.*, 2019) [10, 11].

Safety considerations

Clinicians face an ethical tension when the medication most likely to improve quality of life through reduced behavioural crises simultaneously accelerates metabolic risk in a patient group already vulnerable to obesity-related complications. The decision to continue, adjust, or switch SGA treatment must balance the documented behavioural benefits—reduced irritability, fewer aggressive episodes, improved sleep—against the cumulative metabolic cost (Pringsheim *et al.*, 2015; Scahill *et al.*, 2016) [1, 12]. This research found that medication switching occurred in only 8.2% of participants during the 12-week period despite clinically significant weight gain, suggesting that thresholds for intervention may be set too conservatively in routine practice.

Table 2. Metabolic Outcomes at 12-Week Follow-Up by Eating Pattern Group, Mean (SD)

Variable	Typical Diet	Mild	Moderate	Severe	*p*-value
BMI z-score change, 12 wk	0.82 (0.31)	1.14 (0.42)	1.47 (0.38)	1.93 (0.56)	< 0.001
Weight gain (kg)	3.41 (1.23)	4.78 (1.67)	6.12 (1.54)	8.34 (2.41)	< 0.001
Triglyceride change (mg/dL)	16.8 (8.3)	21.4 (9.7)	28.7 (11.2)	36.9 (14.8)	0.002
Fasting glucose change (mg/dL)	3.1 (2.4)	4.3 (2.8)	5.8 (3.1)	7.6 (4.2)	0.009
Waist circ. change (cm)	2.1 (0.9)	2.8 (1.1)	3.6 (1.3)	4.7 (1.8)	< 0.001
% Energy from refined carbs	34.2 (8.6)	42.7 (9.3)	53.1 (7.8)	64.8 (11.4)	< 0.001
Daily caloric increase (kcal)	312 (98)	378 (114)	421 (127)	487 (142)	0.003
HOMA-IR change	0.41 (0.22)	0.58 (0.29)	0.73 (0.31)	0.97 (0.43)	0.004

Aripiprazole demonstrated the most favourable metabolic profile in this cohort and may represent a preferred first-line option for autistic adolescents with pre-existing food selectivity (Correll *et al.*, 2009; Solmi *et al.*, 2020) [3, 4]. However, aripiprazole is not universally effective for the behavioural presentations that prompt SGA prescribing, and individual response variability necessitates ongoing clinical monitoring regardless of the agent selected. Figure 2 presents a decision pathway that incorporates baseline eating pattern assessment into the metabolic monitoring protocol.

Nutritional assessment

The dietary recall data revealed concerning patterns of micronutrient inadequacy that predated and were compounded by SGA-induced changes in appetite. At baseline, 63.2% of participants with severe food selectivity failed to meet recommended daily intake for calcium, 57.9% for vitamin D, and 78.9% for dietary fibre (Sharp *et al.*, 2018) [14]. These deficiencies persisted or worsened over the 12-week observation period, as the increased caloric intake was directed toward the same narrow range of accepted foods rather than toward nutritionally diverse alternatives.

Collaborative assessment by psychiatrists and dietitians is therefore needed at the point of SGA initiation. A tiered approach—basic dietary screening for all patients, with referral for formal nutritional assessment when food selectivity is identified—would allow resources to be directed where they are most needed (Sharp *et al.*, 2013; Baio *et al.*, 2018) [7, 13]. Micronutrient supplementation should be considered for adolescents with severe food selectivity who are unable to diversify their diets within the timeframe of pharmacological treatment.

Conclusion

This prospective research has established that restricted eating patterns in autistic adolescents function as a significant moderator of antipsychotic-induced weight gain, with the most selective eaters experiencing BMI z-score increases more than twice those observed in adolescents with typical dietary patterns. The caloric channelling mechanism—whereby SGA-stimulated appetite is funnelled through a narrow repertoire of energy-dense, nutritionally poor accepted foods—provides a behavioural explanation for this amplified metabolic risk that operates independently of pharmacological variables such as drug type and dose.

Three practical steps emerge from these findings. First, eating behaviour assessment should be embedded in pre-prescription protocols for SGA treatment in autistic adolescents. The BAMBI and a brief dietary recall represent feasible screening instruments that can be incorporated into existing clinical workflows without substantial additional time or resource expenditure. Second, metabolic monitoring intensity should be calibrated to baseline food selectivity severity, with more frequent surveillance for adolescents whose dietary rigidity places them at the upper end of the risk spectrum. Third, multidisciplinary collaboration between prescribing clinicians, dietitians, and occupational therapists should be established at the point of SGA initiation rather than deferred until metabolic complications have already developed.

The broader implication of these data concerns the need to move beyond one-size-fits-all metabolic monitoring guidelines for SGA-treated youth. Current frameworks treat autistic adolescents as a homogeneous group with respect to dietary behaviour, overlooking the well-documented heterogeneity in eating patterns that characterises this population. By demonstrating that baseline food selectivity predicts the magnitude of metabolic deterioration, this research provides evidence for a stratified approach to monitoring that directs clinical attention and resources toward those at greatest risk.

Future research should examine whether early dietary intervention—initiated concurrently with SGA treatment—can attenuate the caloric channelling effect and reduce the metabolic gap between selective and non-selective eaters. Longer-term follow-up data are needed to determine whether the accelerated weight gain trajectories observed at 12 weeks persist, plateau, or reverse over the course of ongoing treatment. Multi-site replication across different cultural and healthcare contexts will clarify the generalisability of these findings beyond the Australian setting. Additionally, the pharmacogenomic factors that may predispose certain individuals to greater SGA-induced appetite stimulation should be examined in conjunction with eating behaviour phenotypes to allow more precise metabolic risk stratification at the individual patient level.

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