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Original Research Article

Serum Total Carnitine Response and Autism Severity after L-Carnitine Supplementation: A Randomized Double-Blind Placebo-Controlled Trial

Ali Mohsen Saleh[1]*, Adheed Khalid Sharrad Al-Sharrad[1], Hayder Kadhim Jabbar[2]

[1]Dept. of Clinical Pharmacy, College of Pharmacy, University of Basrah, Basrah, Iraq
[2]Pediatric Neurology, Al-Sayyab Teaching Hospital, Basra Health Directorate, Basrah, Iraq

***Corresponding Author:** Ali Mohsen Saleh
Dept. of Clinical Pharmacy, College of Pharmacy, University of Basrah, Basrah, Iraq

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Abstract: ***Background*:** Autism spectrum disorder (ASD) is a heterogeneous neurodevelopmental condition characterized by persistent difficulties in social communication and interaction together with restricted or repetitive patterns of behavior. ***Aim*:** A study of changes in total carnitine levels was associated with clinical improvement after oral carnitine supplementation in children with autism spectrum disorder. ***Methodology*:** 120 treatment-naive children aged 3–7 years with Childhood Autism Rating Scale (CARS) scores of 35–50 were allocated equally to L-carnitine syrup (50 mg/kg/day in two divided doses) or matching placebo for six months. ***Results*:** CARS and serum total carnitine were assessed at baseline and month 6, and all participants completed follow-up. CARS decreased from 40.83±5.19 to 34.05±5.45 in the L-carnitine group and from 41.12±4.14 to 39.85±4.29 with placebo. The between-group difference in change was −5.52 points (95% CI −6.72 to −4.31; $p<0.001$; Cohen's d = −1.66). Serum total carnitine increased from 29.10±9.25 to 50.22±7.24 µmol/L with L-carnitine but remained unchanged with placebo (28.82±6.20 to 28.98±6.19 µmol/L); the between-group difference in change was 20.96 µmol/L (95% CI 19.03 to 22.89; $p<0.001$). Within the active group, greater carnitine elevation was strongly associated with greater CARS reduction (Pearson r= −0.86, $p<0.001$). A predefined response of at least a 4-point CARS reduction occurred in 73.3% versus 5.0% of participants. Adverse events were mild and no participant discontinued treatment. ***Conclusion*:** Six months of L-carnitine improved serum total carnitine and autism severity relative to placebo, with a strong exposure-response association. Larger multicenter trials are required.

Keywords: Autism Spectrum Disorder, Clinical Trial, L-Carnitine, Placebo.

INTRODUCTION

Autism spectrum disorder (ASD) is a heterogeneous neurodevelopmental condition characterized by persistent difficulties in social communication and interaction together with restricted or repetitive patterns of behavior. Clinical presentation varies widely in language, cognition, sensory responses, adaptive functioning and associated medical conditions, which makes treatment response difficult to predict (American Psychiatric Association, 2022; Lord *et al*., 2018). Current management is multidisciplinary and is directed mainly toward developmental support, behavioral intervention and treatment of associated symptoms rather than a single disease-modifying biological target (Hyman *et al*., 2020).

Evidence from clinical, biochemical and cellular studies suggests that mitochondrial dysfunction may be relevant in a subgroup of children with ASD. Reported findings include altered respiratory-chain activity, increased oxidative burden, abnormal lactate or pyruvate handling and distinctive acylcarnitine patterns (Giulivi *et al.,* 2010; Rossignol & Frye, 2012; Frye, 2020). These abnormalities are not universal and should not be interpreted as a single explanation for ASD, but they provide a rationale for testing metabolically directed adjunctive interventions in carefully characterized participants. Carnitine is required for the transport of long-chain fatty acids into mitochondria and contributes to the maintenance of the intramitochondrial acyl-CoA/free-CoA balance. Its availability therefore influences fatty-acid oxidation

and cellular energy production (Longo *et al*., 2016; Rebouche, 2004). Reduced carnitine status and altered acylcarnitine profiles have been described in subsets of individuals with ASD, although the frequency and clinical importance of these findings remain uncertain (Filipek *et al*., 2004; Frye *et al*., 2013; Barone *et al*., 2018). Small clinical trials have examined L-carnitine as an adjunctive intervention in ASD. Geier *et al*. reported improvement in several clinical outcomes after levocarnitine at 50 mg/kg/day for three months, while Fahmy *et al*. observed improvement in CARS scores and increased circulating carnitine fractions after six months at a higher dose (Geier *et al*., 2011; Fahmy *et al*., 2013). Later trials of L-carnitine added to risperidone also reported behavioral benefits, but differences in background treatment, dose and outcome measures limit direct comparison (Shakibaei & Jelvani, 2023; Nasiri *et al*., 2024). Reviews consequently describe the evidence as promising but still insufficient for routine universal use (Malaguarnera & Cauli, 2019; Kepka *et al*., 2021).

Most earlier studies were small, and few directly examined whether the biochemical response to supplementation was related to the magnitude of clinical change. Measuring serum total carnitine before and after treatment can confirm systemic exposure and may help distinguish children who achieve a substantial biochemical response from those who do not. A clinical-biochemical association would not by itself establish causality, but it would strengthen the plausibility of an exposure-response relationship. The present report focuses on two linked outcomes from a six-month randomized trial: autism severity measured with the Childhood Autism Rating Scale (CARS) and serum total carnitine. The objectives were to compare changes in CARS and serum total carnitine between L-carnitine and placebo groups, determine the proportion of participants reaching a predefined CARS response threshold and examine the relationship between carnitine elevation and clinical improvement.

Experimental Section

Study Design and Setting

A prospective, randomized, double-blind, placebo-controlled clinical trial was conducted at the Pediatric Neurology Outpatient Clinic of Al-Sayyab Teaching Hospital, Basra, Iraq. Recruitment and data collection were undertaken from March to December 2025. Each participant received the assigned intervention for six months. An a priori calculation using G*Power version 3.1.9.7 indicated that at least 52 participants were required under the assumptions used for the parent trial. To improve precision and allow balanced group comparison, 120 children were enrolled and randomized in a 1:1 ratio.

Participants

Children were eligible when they were newly diagnosed with ASD by a consultant pediatric neurologist, were 3–7 years old, were treatment-naive with respect to autism-related medication and L-carnitine supplementation, had a baseline CARS score between 35 and 50 and had written consent provided by a parent or legal guardian. Both boys and girls were eligible. Exclusion criteria were epilepsy or recurrent seizures, cerebral atrophy, severe physical or sensory disability that would interfere with assessment, major intellectual disability preventing reliable follow-up, hereditary hematological disease, clinically significant endocrine disease, renal insufficiency or active kidney disease and baseline CARS outside the prespecified range.

Randomization, Blinding and Intervention

A pre-prepared randomization code assigned participants equally to active treatment or placebo. Study bottles were identical, unlabelled amber containers identified only by code. Parents, participants, the consultant pediatric neurologist who performed CARS scoring and laboratory personnel remained unaware of allocation until clinical and laboratory procedures were completed. The active group received berry-flavored L-carnitine syrup at 50 mg/kg/day, divided into equal morning and evening doses, for six months. The syrup concentration was 40 mg/mL. The placebo group received an identical weight-matched volume formulated to resemble the active syrup in appearance, viscosity and taste. Families were asked to maintain the child's usual diet, routine and background behavioral or educational interventions and to avoid starting non-essential L-carnitine or antioxidant supplements. Adherence and tolerability were reviewed by telephone and at outpatient visits every two months.

Clinical Assessment

CARS was administered at baseline and after six months by the same blinded consultant pediatric neurologist. The scale contains 15 items rated from 1 to 4, producing a total score from 15 to 60; higher scores indicate greater autism severity (Schopler *et al*., 1980; Schopler *et al*., 2010). The primary clinical variable in this focused analysis was the change in total CARS score, calculated as month-6 minus baseline value. Negative values therefore represented improvement. A responder analysis used a prespecified threshold of at least a 4-point reduction in total CARS.

Blood Sampling and Serum Total Carnitine Assay

Approximately 2 mL of venous blood was obtained before treatment and at month 6. Samples were collected into gel and clot-activator tubes, allowed to clot at room temperature and centrifuged at 1500 rpm. Serum was aliquoted and stored at −80 °C. Baseline and follow-up samples were retained until completion of the clinical phase and analyzed under

the same laboratory conditions to reduce batch variation. Serum total carnitine was quantified with a Human L-Carnitine ELISA Kit (ELK Biotechnology, Wuhan, China; catalog ELK9821) according to the manufacturer's instructions. Optical density was read at 450 nm with a BioTek 800TS microplate reader, and concentrations were obtained from the assay standard curve.

Statistical Analysis

Data were organized in Microsoft Excel 2019 and analyzed with SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Distributional assumptions were examined using Kolmogorov–Smirnov and Shapiro–Wilk tests. Continuous variables are presented as mean ± standard deviation. Baseline and month-6 values were compared within groups using paired-samples t tests, and independent-samples t tests were used for between-group comparisons. Change scores were compared between groups to estimate treatment effects beyond the change observed with placebo. Mean differences are reported with 95% confidence intervals (CIs), and Cohen's d was used to describe standardized effect magnitude. Responder proportions were compared with Fisher's exact test. Within the active group, Pearson and Spearman correlations examined the association between change in serum total carnitine and change in CARS. All tests were two-tailed, and $p < 0.05$ was considered statistically significant.

RESULTS AND DISCUSSION

Participant Flow and Baseline Comparability

All 120 enrolled children completed the six-month intervention and were included in the analysis (Figure 1). Sixty participants received L-carnitine and 60 received placebo. No participant was lost to follow-up and no treatment was discontinued. At baseline, the groups were similar in age, body size, CARS score and serum total carnitine (Table 1). Mean CARS values were 40.83±5.19 in the active group and 41.12±4.14 in the placebo group (p= 0.7415). Mean serum total carnitine values were 29.10±9.25 and 28.82±6.20 µmol/L, respectively (p= 0.8463). This balance reduces the likelihood that post-treatment differences arose from initial disparities in symptom severity or circulating carnitine.

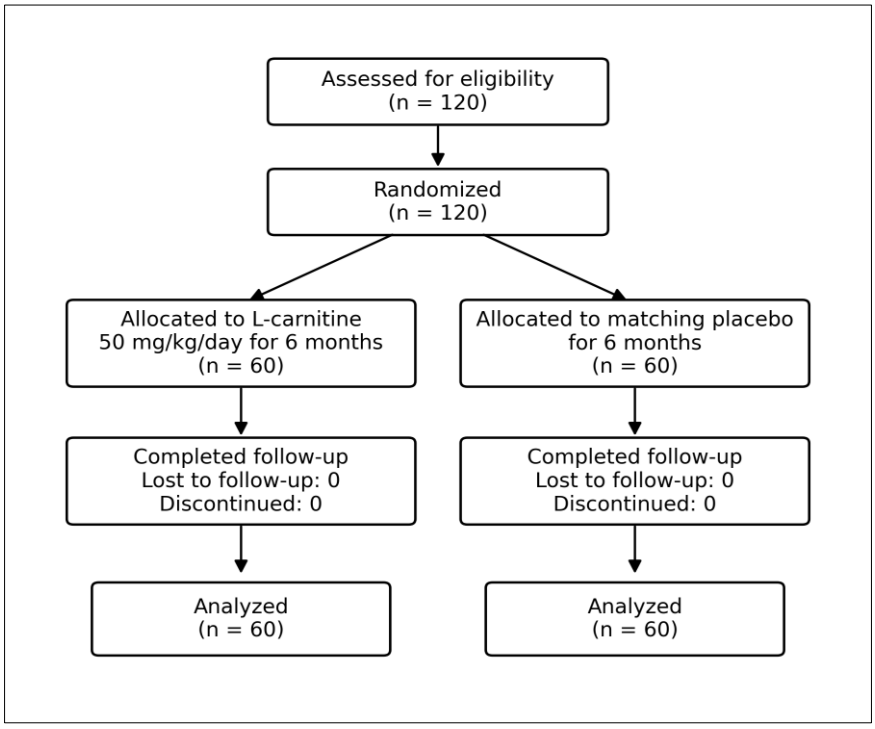

Figure 1: Participant flow through enrollment, randomization, six-month follow-up and analysis

Table 1: Baseline demographic, clinical and biochemical characteristics

Variable	L-carnitine (n = 60)	Placebo (n = 60)	p-value
Age (months)	48.65±7.23	48.23±6.79	0.7455
Weight (kg)	20.26±2.34	20.47±2.39	0.6415
Height (cm)	111.50±5.20	113.01±5.45	0.1238
CARS score	40.83±5.19	41.12±4.14	0.7415
Serum total carnitine (µmol/L)	29.10±9.25	28.82±6.20	0.8463

Values are mean ± standard deviation. Between-group comparisons used independent-samples t tests. CARS, Childhood Autism Rating Scale

Effect on Autism Severity

After six months, mean CARS declined by 6.78 points in the L-carnitine group, from 40.83±5.19 to 34.05±5.45 ($p < 0.001$). The placebo group also showed a smaller reduction of 1.27 points, from 41.12±4.14 to 39.85±4.29 ($p < 0.001$) (Table 2). The comparison of change scores favored L-carnitine by 5.52 points (95% CI 4.31 to 6.72 points in magnitude; $p < 0.001$), with a large standardized effect (Cohen's d = 1.66) (Table 3). The small improvement observed with placebo is not unexpected in pediatric ASD trials. Repeated clinical contact, developmental maturation, stable behavioral support, increased caregiver engagement and expectancy can all contribute to change over time (Siafis *et al*., 2020). For this reason, the between-group difference is more informative than within-group significance alone. The active-placebo contrast indicates that the CARS reduction associated with L-carnitine exceeded the nonspecific change observed during the same period. The direction of the clinical finding is consistent with earlier controlled studies. Geier *et al*., reported improvement after 50 mg/kg/day of levocarnitine for three months, and Fahmy *et al*., observed a reduction in CARS after six months of supplementation (Geier *et al*., 2011; Fahmy *et al*., 2013). The present study included a larger sample than those early trials and used the same clinician for baseline and follow-up scoring under blinded conditions. Differences in dose, duration, baseline severity and concurrent treatment prevent direct numerical comparison, but the overall pattern supports further investigation of carnitine-responsive ASD subgroups. A prespecified reduction of at least four CARS points was reached by 44 of 60 children receiving L-carnitine (73.3%) and 3 of 60 receiving placebo (5.0%; Fisher's exact $p < 0.001$). This threshold should be interpreted as a pragmatic responder definition rather than a universally established minimal clinically important difference for CARS. Nevertheless, the marked separation between groups shows that the mean result was not produced solely by a small number of extreme responders.

Table 2: Within-group changes in CARS and serum total carnitine

Outcome	Group	Baseline	Month 6	Mean change (95% CI)	Paired p-value
CARS score	L-carnitine	40.83±5.19	34.05±5.45	−6.78 (−7.94 to −5.62)	<0.001
CARS score	Placebo	41.12±4.14	39.85±4.29	−1.27 (−1.63 to −0.90)	<0.001
Total carnitine (µmol/L)	L-carnitine	29.10±9.25	50.22±7.24	21.12 (19.19 to 23.05)	<0.001
Total carnitine (µmol/L)	Placebo	28.82±6.20	28.98±6.19	0.16 (−0.14 to 0.46)	0.2890

CARS change was calculated as month 6 minus baseline; negative values indicate clinical improvement. CI, confidence interval.

Effect on Serum Total Carnitine

Serum total carnitine increased by 21.12 µmol/L in the active group, from 29.10±9.25 to 50.22±7.24 µmol/L ($p< 0.001$), representing a 72.57% increase. The placebo group remained essentially unchanged, with a mean increase of 0.16 µmol/L ($p = 0.2890$). The between-group difference in change was 20.96 µmol/L (95% CI 19.03 to 22.89; $p < 0.001$; Cohen's d = 3.92). This large biochemical effect is expected because circulating carnitine is the direct exposure marker of the administered intervention. It confirms that weight-based dosing produced a measurable systemic response and provides an objective complement to the behavioral outcome. Fahmy *et al*., similarly reported increased free and total carnitine after supplementation, while studies of ASD metabolic phenotypes have described baseline heterogeneity in carnitine and acylcarnitine profiles (Fahmy *et al*., 2013; Frye *et al*., 2013; Barone *et al*., 2018). Total carnitine alone cannot characterize the full carnitine cycle, but it is clinically useful for documenting exposure when free carnitine and detailed acylcarnitine profiling are unavailable.

Table 3: Between-group comparison of change scores

Outcome	L-carnitine change	Placebo change	Difference in change (95% CI)	p-value	Cohen's d
CARS score	−6.78±4.49	−1.27±1.42	−5.52 (−6.72 to −4.31)	<0.001	−1.66
Total carnitine (µmol/L)	21.12±7.47	0.16±1.15	20.96 (19.03 to 22.89)	<0.001	3.92

Values are mean ± standard deviation unless otherwise stated. Between-group comparisons used independent-samples t tests

Relationship between Carnitine Response and CARS Change

Within the L-carnitine group, the rise in serum total carnitine was strongly associated with the reduction in CARS (Pearson $r = −0.86$, $p < 0.001$; Spearman rho = −0.80, $p < 0.001$). Change in CARS was defined as month 6 minus baseline, so a more negative value represented greater clinical improvement. The negative coefficient therefore means that larger carnitine increases tended to occur in children with larger CARS reductions. This association is compatible with an exposure-response relationship, but it should not be interpreted as proof that the serum concentration itself caused behavioral improvement. Greater serum increase may reflect better adherence, absorption, distribution or underlying metabolic need. Oral L-carnitine has dose-dependent and saturable pharmacokinetics, and between-child variation in intestinal transport and renal handling can influence circulating concentrations (Evans & Fornasini, 2003; Longo *et al*., 2006). Future studies should evaluate whether baseline free carnitine, acylcarnitine patterns, diet or pharmacokinetic measures predict the magnitude of clinical response. The strong correlation differs from Fahmy *et al*., who did not identify

a clear relationship between carnitine fractions and CARS improvement (Fahmy *et al*., 2013). The discrepancy could reflect sample size, assay method, dose, participant selection or the wider range of biochemical response in the present cohort. Replication is essential because correlations measured within one treatment arm may be influenced by adherence and other unmeasured variables.

Safety and Tolerability

Six children in the active group (10.0%) and two in the placebo group (3.3%) reported at least one mild adverse event; the difference was not statistically significant (Fisher's exact p = 0.272). Active-group events included abdominal discomfort, loose stool or mild diarrhea, transient irritability and fish-like body odor (Table 4). No serious adverse event or treatment discontinuation occurred. The observed events are consistent with the known tolerability profile of oral L-carnitine. Gastrointestinal symptoms may be related to incomplete intestinal absorption, while trimethylamine formation by intestinal microbiota can contribute to fish-like odor (Evans & Fornasini, 2003; Goin-Kochel *et al*., 2019). Dividing the daily dose may reduce intestinal load, although this study was not designed to compare dosing schedules. The findings support short-term tolerability at 50 mg/kg/day under clinical monitoring but cannot exclude uncommon or delayed adverse effects.

Table 4: Reported adverse events during the six-month intervention

Adverse event	L-carnitine (n = 60)	Placebo (n = 60)	Outcome
Any adverse event	6 (10.0%)	2 (3.3%)	Mild and self-limited
Abdominal discomfort	2 (3.3%)	1 (1.7%)	Resolved without withdrawal
Loose stool/mild diarrhea	2 (3.3%)	0	Transient
Mild irritability/restlessness	1 (1.7%)	1 (1.7%)	Self-limited
Fish-like body odor	1 (1.7%)	0	No discontinuation
Serious adverse event	0	0	None reported
Treatment discontinuation	0	0	None

Adverse events were reported by parents during scheduled follow-up. Fisher's exact p = 0.272 for any adverse event

Strengths and Limitations

Important strengths include random allocation, double blinding of families, clinician and laboratory personnel, balanced baseline values, complete follow-up, repeated assessment by the same blinded clinician and objective confirmation of biochemical exposure. Reporting both change scores and effect sizes also helps distinguish statistical significance from treatment magnitude (Vickers & Altman, 2001; Sullivan & Feinn, 2012). The study also has limitations. It was conducted at one center, which restricts generalizability. Follow-up ended at six months, so persistence of benefit after discontinuation is unknown. CARS provides a global severity score but does not fully characterize language, adaptive functioning, social responsiveness or caregiver-reported quality of life. Serum total carnitine was measured without free carnitine, acylcarnitines or the acyl/free ratio, and dietary intake, gastrointestinal symptoms and background behavioral interventions were not quantified in detail. The correlation analysis was exploratory and may be affected by adherence and unmeasured confounding. Finally, the responder threshold was pragmatic and should be validated in future trials.

CONCLUSION

Six months of oral L-carnitine at 50 mg/kg/day produced a larger reduction in CARS score and a marked increase in serum total carnitine compared with placebo in children with ASD. The strong inverse association between carnitine change and CARS change supports a possible exposure-response relationship, although causality cannot be established from correlation alone. L-carnitine was generally well tolerated, with only mild, self-limited adverse events. These findings support further multicenter, biomarker-informed trials incorporating free and acylcarnitine profiling, broader developmental outcomes and longer follow-up before routine clinical use is recommended.

Ethical Considerations

The protocol was approved by the Research Ethics Committee of the Basra Health Directorate and the Ethical Committee of the College of Pharmacy, University of Basrah (2017 at 7/Jan/2025). Administrative approval was obtained from Al-Sayyab Teaching Hospital. Parents or legal guardians provided written informed consent after receiving information about study procedures, potential benefits and possible adverse effects. Participant data and samples were coded to preserve confidentiality.

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Conflict of Interest: The authors declare no conflict of interest.

Data Availability: De-identified data supporting the findings may be available from the corresponding author on reasonable request and subject to ethical and institutional approval.

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