

Non-Medication Stimulant Usage in Pediatrics and its Effect on Bone Health

Garrett Miedema, BS¹, Nicholas Mulder, BS¹, Abdoul Mbengue, BS¹, Lucas Kopp, BS¹, William Woodhams BA¹ Alicia Knickerbocker BS¹, Karim Mhanna BS¹, Darby Dean BS¹, Camryn McIntyre, BS¹, Taylor Wesley, BS¹, William Galardi¹, Muaaz Wajahath¹, Alison Tenfelde, MD¹

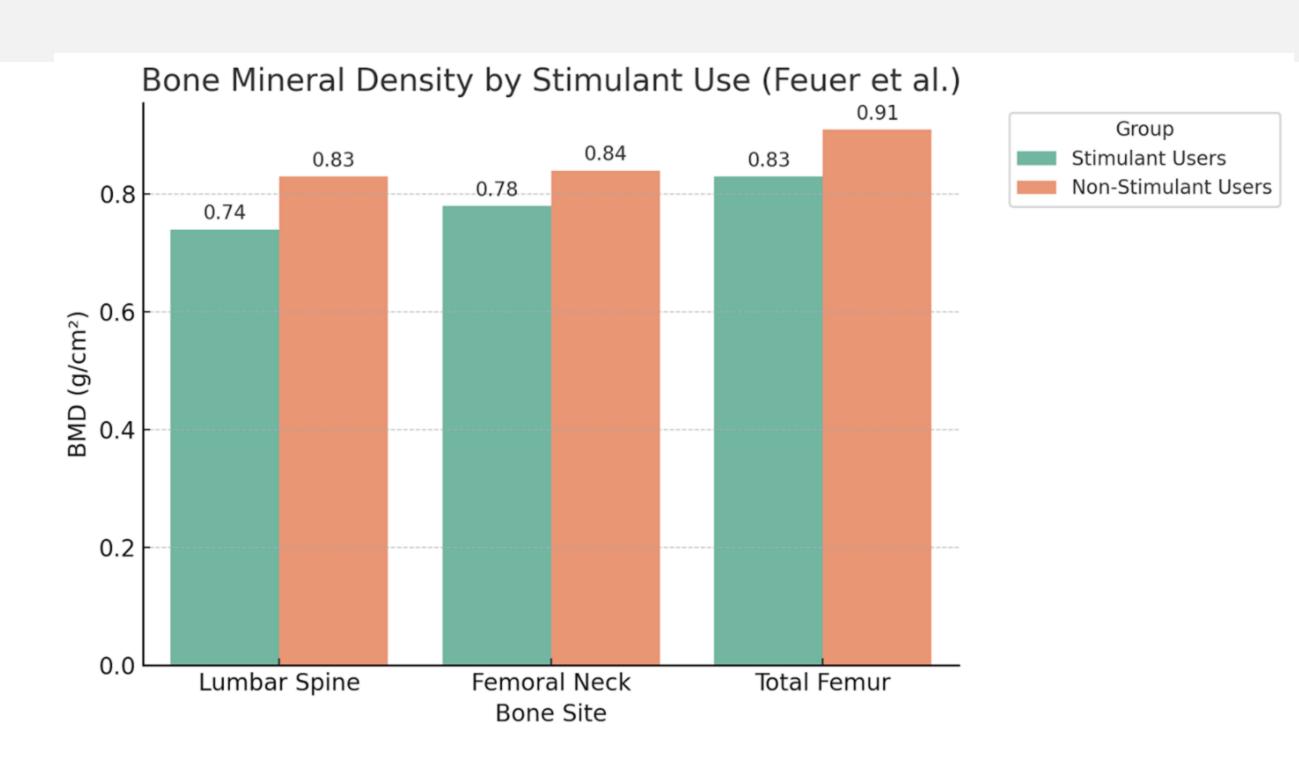
Introduction

Stimulant prescriptions for adolescents, primarily for ADHD, have increased in recent years, with ~10% reporting medical use and ~17% reporting nonmedical use^{1,2}. While effective for symptom control, stimulants are also widely misused for academic and athletic performance². Beyond recognized side effects such as insomnia and cardiovascular changes³, emerging evidence links stimulant use with reduced bone mineral density and impaired bone healing^{4,5}. This is concerning given that over half of U.S. children participate in organized sports, where fracture risk is already elevated⁶. Interference with bone accrual during adolescence may compromise peak bone mass, increasing the likelihood of fragility fractures later in life^{4,5}. Despite these risks, research on the long-term skeletal effects of stimulant use and misuse remains limited. This study systematically reviews the evidence to clarify potential consequences and future clinical counseling.

Methods

The study was performed via a systematic review. The study was conducted using the search engine Endnote, investigating primary resources and articles through PUBMed and EMBase. 111 initial studies were analyzed and were assessed using keywords "pediatrics", "bone", "stimulants", "osteoporosis", "health". Stimulants were defined as prescription medications primarily used in the treatment of attention-deficit/hyperactivity disorder (ADHD), including amphetamine-based drugs (e.g., Adderall, Dexedrine) and methylphenidate-based drugs (e.g., Ritalin, Concerta). Primary articles which did not contain these search criteria were excluded. Based upon exclusion criteria, 44 primary articles remained. Independent variables such as stimulant dosage, frequency, patient age, and previous medical history were tracked and assessed. Dependent variables such as bone mineral cortex (BMC), bone mineral density (BMD), and osteoporosis were measured and assessed accordingly. Of the 44 articles, 6 articles were included for analysis following secondary exclusion criteria with measurements of the independent and dependent variables mentioned.

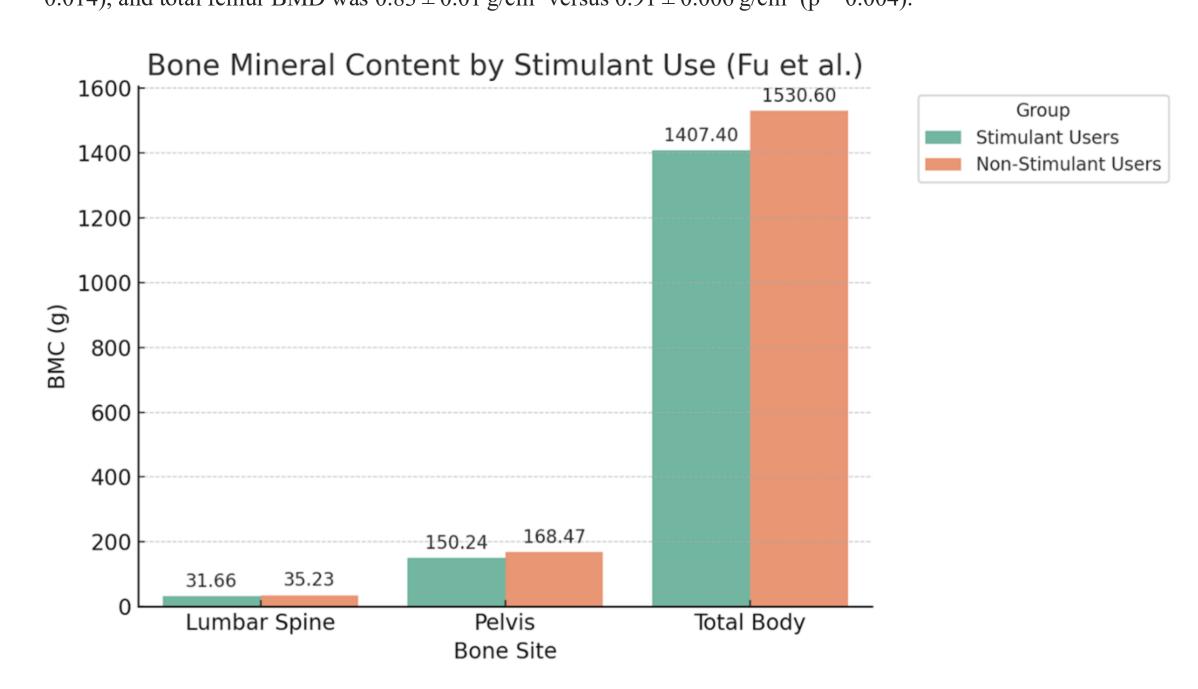
Results



Data from NHANES 1999-2004 youth aged 8-20 years (N=6754).

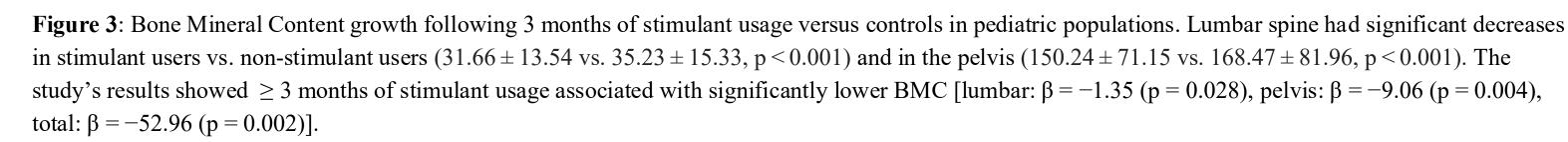
Source: Feuer et al., Journal of Pediatrics, 2016;178:196-202.e1.

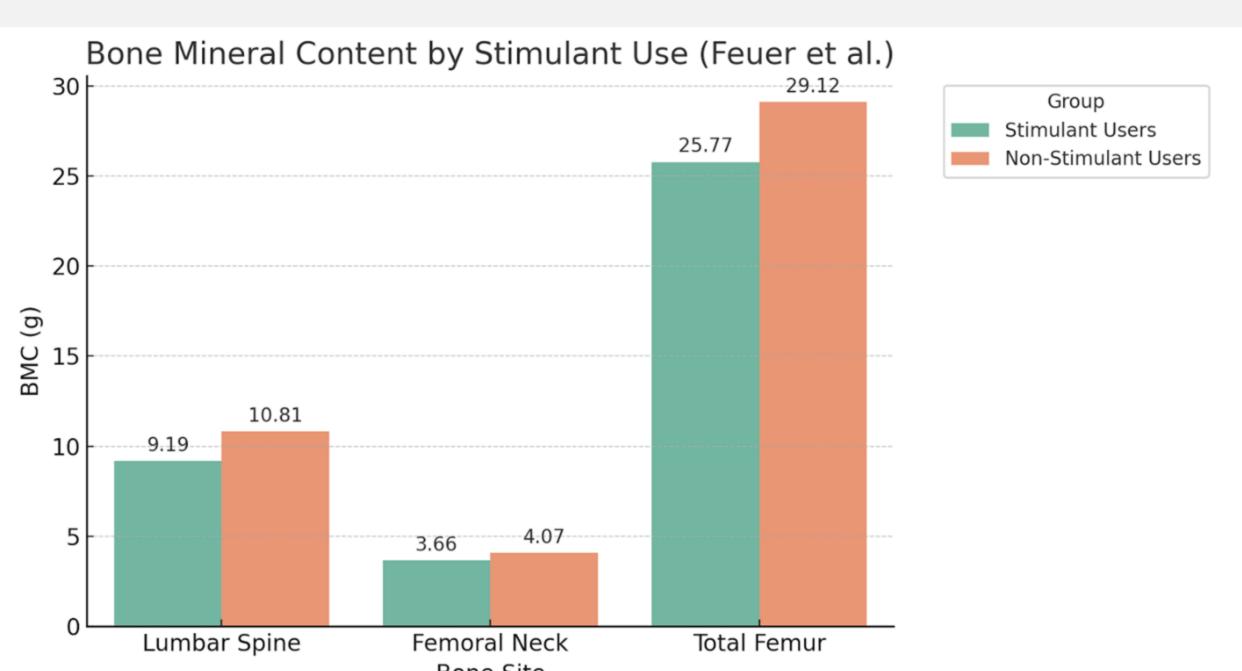
Figure 1: Stimulant users had significantly lower BMD compared to non-users across all measured skeletal regions. Specifically, lumbar spine BMD was 0.74 ± 0.01 g/cm² in stimulant users versus 0.83 ± 0.005 g/cm² in non-users (p = 0.004). Femoral neck BMD was 0.78 ± 0.01 g/cm² versus 0.84 ± 0.006 g/cm² (p = 0.014), and total femur BMD was 0.83 ± 0.01 g/cm² versus 0.91 ± 0.006 g/cm² (p = 0.004).



Data from a cross-sectional study of 5–17 year olds with ADHD (N=5472).

Source: Fu et al., European Journal of Pediatrics, 2021;180:2221–2231.

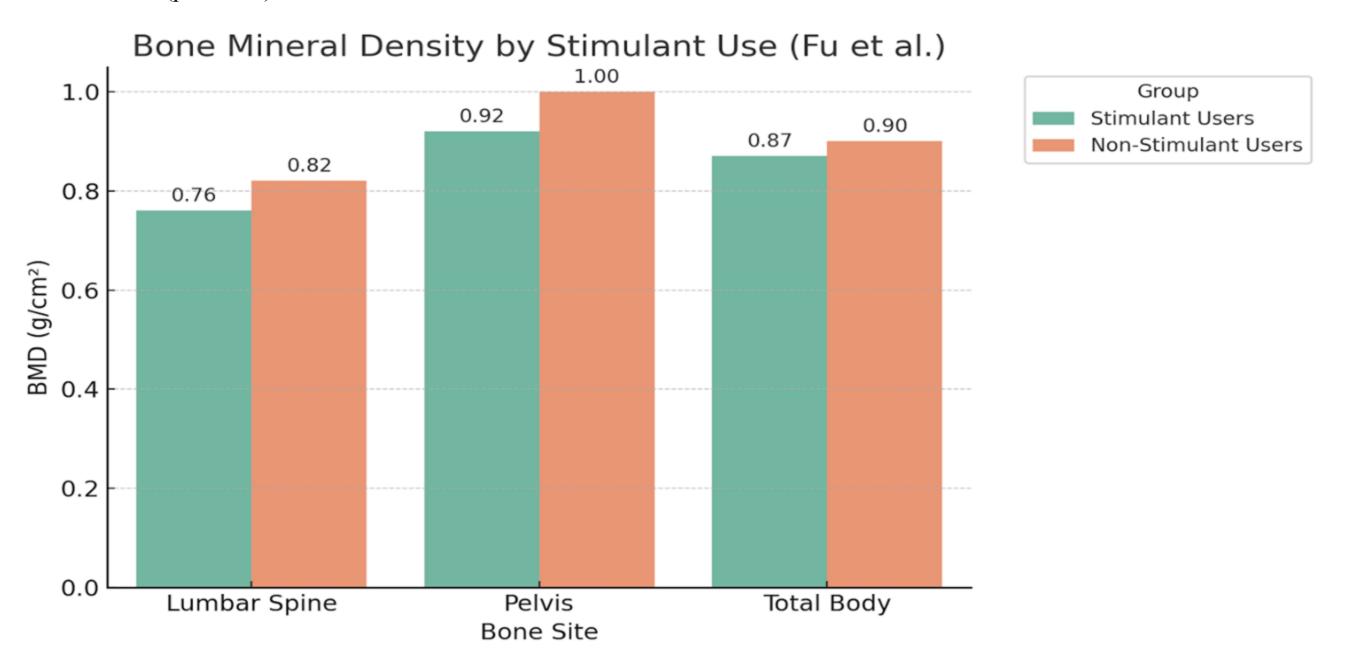




Data from NHANES 1999-2004 youth aged 8-20 years (N=6754).

Source: Feuer et al., Journal of Pediatrics, 2016;178:196-202.e1.

Figure 2: Bone Mineral Content in Stimulant vs. Non-Stimulant Users in Pediatric populations. Stimulant users had significantly lower BMC across all measured bone sites compared to non-users. Lumbar spine BMC was 9.19 ± 0.27 in stimulant users versus 10.81 ± 0.14 in non-users (p < 0.001). Femoral neck BMC was 3.66 ± 0.10 compared to 4.07 ± 0.06 (p = 0.013), and total femur BMC was 25.77 ± 0.73 versus 29.12 ± 0.40 (p = 0.001)



Data from a cross-sectional study of 5–17 year olds with ADHD (N=5472).

Source: Fu et al., European Journal of Pediatrics, 2021;180:2221–2231.

Figure 4: BMD between stimulant vs. non-stimulant users in pediatric populations. There was a significantly decreased BMD measured at the level of the lumbar spine $(0.76 \pm 0.16 \text{ vs. } 0.82 \pm 0.17, P < 0.001)$, pelvis $(0.92 \pm 0.21 \text{ vs. } 1.00 \pm 0.22, P < 0.001)$, and total body $(0.87 \pm 0.12 \text{ vs. } 0.90 \pm 0.14, P < 0.001)$.

Conclusions

Stimulant use in adolescents is rising both for ADHD treatment and nonmedical performance enhancement, creating an underrecognized threat to skeletal health during a period of rapid musculoskeletal development^{1,2,10}. Our synthesis of the available evidence demonstrates consistent reductions in bone mineral density (BMD) and bone mineral content (BMC) at clinically significant sites, including the lumbar spine, femoral neck, and pelvis⁴⁻⁹. Even relatively short exposures (>3 months) were associated with impaired bone accrual and diminished healing after fracture, suggesting that stimulant effects extend beyond transient growth suppression and may alter fundamental processes of skeletal remodeling^{7,8}.

Mechanistic studies support these clinical findings: stimulants may impair osteoblast differentiation, disrupt calcium balance, and compromise trabecular and cortical bone microarchitecture, offering a biologically plausible pathway to long-term fragility⁴⁻⁹. These risks are especially concerning in youth athletes, over half of whom participate in organized sports where fracture risk is already elevated⁶. Left unaddressed, these changes could translate into premature osteoporosis and higher lifetime fracture burden.

Clinically, our findings highlight the importance of proactive counseling for families, careful monitoring of bone health in stimulant-treated youth, and the consideration of protective strategies such as optimizing vitamin D, calcium intake, and weight-bearing exercise. At the systems level, these data emphasize the need for preventive education targeting the rising normalization of stimulant misuse in both academic and athletic environments^{2,10}.

Future research should directly assess fracture incidence in stimulant-treated versus untreated children using large-scale electronic health record (EHR) data, which will provide crucial evidence to guide prescribing practices and inform targeted interventions.

Referenced Literature

- Danielson ML, Bitsko RH, Ghandour RM, et al. Prevalence of ADHD diagnosis and treatment in U.S. children, 2016. *J Clin Child Adolesc Psychol*. 2018;47(2):199-212.
 McCabe SE, West BT, Veliz P, Boyd CJ. Prescription stimulant use and misuse among adolescents. *J Am Acad Child Adolesc Psychiatry*. 2017;56(3):226-233.e4.
 Storebø OJ, Pedersen N, Ramstad E, et al. Adverse events of methylphenidate in children and adolescents with ADHD. *Cochrane Database Syst Rev*. 2018;5:CD012069.
 Feuer AJ, Thai A, Demmer RT, Vogiatzi MG. Stimulant use and bone mass in children with ADHD. *JAMA Pediatr*. 2016;170(12):e162804.
- Feuer AJ, Thai A, Demmer RT, Vogiatzi MG. Stimulant use and bone mass in children with ADHD. *JAMA Pediatr*. 2016;170(12):e162804
 Fu Y, Wang G, Liu J, et al. Stimulant use and bone health in U.S. youth: NHANES analysis. *Eur J Pediatr*. 2022;181(4):1633-1642.
- Black L, Terlizzi E, Vahratian A. Organized sports participation among children aged 6–17 years, U.S. 2020. NCHS Data Brief. 2022;(441):1-8.
 Feuer AJ, Thai A, Demmer RT, Vogiatzi MG. Stimulant use and bone mass in children with ADHD. JAMA Pediatr. 2016;170(12):e162804.
 Fu Y, Wang G, Liu J, et al. Stimulant use and bone health in US youth: NHANES analysis. Eur J Pediatr. 2022;181(4):1633-1642.
- Poulton AS, Bui Q, Melzer E, Evans R. Stimulant effects on growth and bone age in ADHD. *Int Clin Psychopharmacol*. 2016;31(2):93-99.
 Ortiz LM, O'Malley N, Blum K, Hadjiargyrou M, Komatsu DE, Thanos PK. ADHD psychostimulants and bone density after fracture. *J Pediatr Orthop B*. 2023; Online ahead of
- 11. Howard AL, Flibotte J, Koszycki D, Hechtman L, Elman LB. Stimulant use and bone mass in ADHD youth. *J Pediatr*. 2017;188:210-215.e1.
- 12. Veliz P, Boyd C, McCabe SE. Adolescent athletics and nonmedical Adderall use. J Stud Alcohol Drugs. 2017;78(3):500-509.

Acknowledgements

We would like to thank Michigan State University College of Human Medicine and Dr. Allison Tenfelde, MD for their time and efforts in this project.