a comorbidity, presented with fever, cough, and diarrhea, but no hypoxia. This patient had been managed with metformin for several years and had undergone bariatric surgery for morbid obesity 16 months before presenting with SARS-CoV-2 infection, but did not have acute glycemic control issues at the time of SARS-CoV-2 diagnosis.

Of the 3 patients with diabetes who required hospitalization, all had systemic symptoms attributable to both SARS-CoV-2 infection, as well as diabetes. One had longstanding type 1 diabetes and prior central nervous system injury secondary to diabetic ketoacidosis/cerebral edema; this patient presented with fever, chest pain, and increased oxygen requirement from baseline. She had a history of good glycemic control (last A1C 7.4%), but had hyperglycemia, without diabetic ketoacidosis at the time of SARS-CoV2 infection. The second patient presented with shortness of breath and chest pain, but no hypoxia, and had concomitant nausea, vomiting, and hypoglycemia. This patient had been diagnosed 1.5 years earlier at an external facility with type 2 diabetes and treated with short-acting insulin rather than oral therapy. However, upon review at our institution, she was deemed to be more consistent with prediabetes rather than diabetes and she was discharged on oral therapy. The third patient had type 2 diabetes with longstanding poor glycemic control despite insulin therapy. She initially presented with fever, chills, nausea, and headache, without respiratory symptoms or acute change in her glycemic control and was managed as an outpatient. However, after improvement in her symptoms, she was admitted 10 days later (after the date of our interim report) with chest pain and shortness of breath, and determined to have pulmonary embolism, as well as significant hyperglycemia (300-400) without diabetic ketoacidosis, requiring initial intravenous insulin therapy. All 3 of these patients had stabilization of their blood glucose levels relatively quickly after hospitalization.

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Body mass index rebound, weight gain in puberty, and risk of cardiovascular disease

To the Editor:

Yuan et al identified 3 groups with distinct trajectories in body mass index (BMI) related to cardiovascular disease risk in adulthood by analyses of a dataset with 30 years of follow-up in a population in northern China.[1] Among the 3 groups characterized as low-, moderate-, and high-increasing, based on the rate of BMI increase, the high-increasing group (11.9% of 2789 total participants) had the highest rate of cardiovascular disease in adulthood. This group had relatively moderate initial BMI levels, but BMI then increased steeply from about age 12 years and exceeded BMI in the moderate-increasing group at age 18 years.

In the pathway to obesity, adolescent obesity forms with acceleration of BMI in childhood following early adiposity rebound, which refers to an early increase in BMI before 4 years of age.[2-4] As these authors mentioned, the present study lacks data on children before age 6 years, which makes it impossible to look at the relationship between timing of adiposity rebound and BMI trajectories into adolescence.[1]

In this study, the high-increasing group had lower average BMI at 6 years of age compared with the moderate group, and their BMI then began to increase after entering adolescence.[1] This change is unlikely to be attributable to early adiposity rebound. This position is supported by previous studies showing that early adiposity rebound is associated with high BMI at 6-8 years of age, and children who develop adolescent obesity have a higher rate of change of BMI from early childhood.[5-7]

Thus, it seems that there is a distinct subgroup in which BMI increases rapidly after onset of puberty, increasing the cardiovascular disease risk owing to increasing adiposity. Pubertal maturation worsens obesity after puberty, but social background is also thought to be involved in the development of obesity.[8-10] Although efforts to alter trajectories that are predictive of adult obesity should commence before preschool age, a prevention strategy is also needed for obesity developing during puberty as a second critical window.

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References

1. Yuan Y, Chu C, Zheng WL, Ma Q, Hu JW, Wang Y, et al. Body mass index trajectories in early life is predictive of cardiometabolic risk. J Pediatr 2020;219:31-7.
2. Hughes AR, Sherriff A, Ness AR, Reilly JJ. Timing of adiposity rebound and adiposity in adolescence. Pediatrics 2014;34:e1354-61.
3. Geserick M, Vogel M, Gausche R, Lipek T, Spielau U, Keller E, et al. Acceleration of BMI in early childhood and risk of sustained obesity. N Engl J Med 2018;379:1303-12.
4. Arisaka O, Ichikawa G, Koyama S, Sairenchi T. Early increase in body mass index and cardiometabolic risk in adolescence. J Pediatr 2019;214:243.
5. Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I, et al. Avon Longitudinal Study of Parents and Children Study Team. Early life risk factors for obesity in childhood: cohort study. BMJ 2005;330:1357.
6. Estevez-Gonzalez MD, Santana Del Pino A, Henriquez-Sanchez P, Pena-Quintana L, Saavedra-Santana P. Breastfeeding during the first 6 months of life, adiposity rebound and overweight/obesity at 8 years of age. Int J Obes 2016;40:10-3.
7. Kato N, Isojima T, Yokoya S, Tanaka T, Ono A, Yokomichi H, et al. Earlier BMI rebound and lower pre-rebound BMI as risk of obesity among Japanese preschool children. Int J Obes (Lond) 2017;42:52-9.
8. Kimm SY, Barton BA, Obarzanek E, McMahon RP, Kronsberg SS, Waclawiw MA, et al. NHLBI Growth and Health Study. Obesity development during adolescence in a biracial cohort: the NHLBI Growth and Health Study. Pediatrics 2002;110:e54.
9. Jasik CB, Lustig RH. Adolescent obesity and puberty: the "perfect storm". Ann NY Acad Sci 2008;1135:265-79.
10. Takaya J, Okawa T. Impact of family income on the lifestyle and physique of schoolchildren in Higashi-Osaka City, Japan. Pediatr Int 2020;62:74-80.

Reply

To the Editor:

Osamu et al identified a distinct increase in body mass index (BMI) after puberty that is probably not related to early adiposity rebound. We consider this to be a valuable point.

We identified 3 BMI trajectories in early life and the high-increasing groups had the greatest risks of cardiovascular disease risks in middle age.[1] It has been reported that early BMI rebound, which is related to being overweight at 6 years of age, is associated with adult obesity.[2] We used the cut-off points of BMI_{24} and BMI_{28} at 18 years of age, and found few children with overweight at 6 years of age in our study.[3,4] We indicated that these data do not fully reflect the relationship between BMI trajectory from birth to middle age and cardiometabolic risks.

Additionally, participants in the high-increasing group experienced moderate initial BMI levels from 12 years of age and exceeded BMI levels in the moderate-increasing group at 18 years of age. Puberty involves a series of physiologic and metabolic changes as well as changing fat distribution.[4] Osamu et al pointed out there is a distinct subgroup in which BMI increases rapidly after onset of puberty, exacerbating the cardiovascular disease risk owing to increasing adiposity. We found that, in the high-increasing group, the relative risk of hypertension is more than 2 times higher for those in puberty compared with those in prepuberty, but participants in prepuberty had higher relative risks of diabetes, high-risk high-density lipoprotein cholesterol levels, and triglycerides than those in puberty (see Table V in the article). Puberty is another important period to focus on obesity prevention, but its relationship with cardiovascular risk needs further analysis in large-scale studies.

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References

1. Kato N, Isojima T, Yokoya S, Tanaka T, Ono A, Yokomichi H, et al. Earlier BMI rebound and lower pre-rebound BMI as risk of obesity among Japanese preschool children. Int J Obes (Lond) 2018;42:52-8.
2. Li H, Zong XN, Ji CY, Mi J. Body mass index cut-offs for overweight and obesity in Chinese children and adolescents aged 2 - 18 years. Zhonghua Liu Xing Bing Xue Za Zhi 2010;31:616-20.
3. Zhao W, Yu K, Tan S, Zheng Y, Zhao A, Wang P, et al. Dietary diversity scores: an indicator of micronutrient inadequacy instead of obesity for Chinese children. BMC Public Health 2017;17:440.
4. Graber JA, Seeley JR, Brooks-Gunn J, Lewinsohn PM. Is pubertal timing associated with psychopathology in young adulthood. J Am Acad Child Adolesc Psychiatry 2004;43:718-26.

Age differentiation in children with asthma treated with intravenous magnesium sulphate

To the Editor:

With great interest we read the article by Johnson et al regarding intravenous magnesium sulphate (IVMg) in children between 2 and 17 years of age with acute asthma.[1] The authors reported that clinicians used IVMg in 10.5% of 60 000 children visiting with asthma. Other findings include highly variable use between centers, mostly in moderate and severe asthma cases, late administration of IVMg in the emergency department, and low return rates of treated children within 72 hours after discharge.

The authors did not report separately their findings for young children between 2 and 5 years of age with acute episodic viral wheezing and children of 6 years and older with acute asthma. Evidence of effect of IVMg in acute episodic viral wheezing, similar to oral corticosteroids and bronchodilators, is limited.[2-6] For example, Pruikkonen