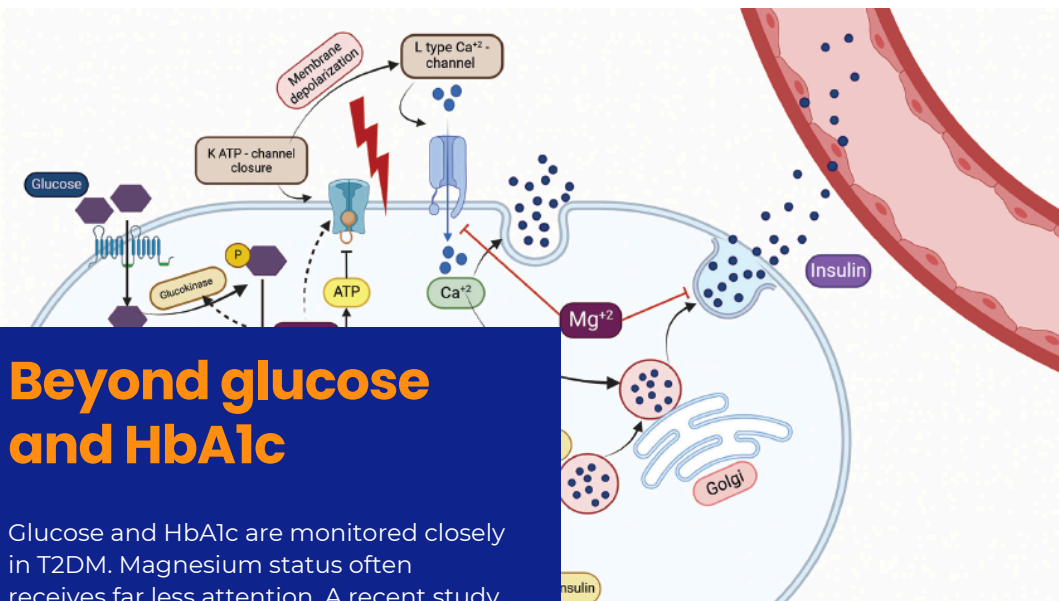


MAGNESIUM AND INSULIN RESISTANCE IN TYPE 2 DIABETES

Could a routine serum magnesium measurement add a piece to the metabolic picture?



Beyond glucose and HbA1c

Glucose and HbA1c are monitored closely in T2DM. Magnesium status often receives far less attention. A recent study in *Advances in Human Biology* examined how serum magnesium relates to insulin resistance in patients with T2DM.

Magnesium (Mg²⁺) is an essential intracellular mineral that plays a key role in glucose metabolism and insulin function. It supports insulin receptor autophosphorylation and signalling, contributes to GLUT-4-mediated glucose uptake in skeletal muscle and adipose tissue

Adequate magnesium is also important for normal insulin synthesis and secretion, while magnesium deficiency may impair insulin signalling and contribute to insulin resistance.

● The Design of Study

This was a single-center, cross-sectional study conducted in an outpatient setting at Holy Family Red Crescent Medical College Hospital, Dhaka, Bangladesh, involving 107 adults with type 2 diabetes or prediabetes (mean age 55.5 ± 11.4 years; 52.3% male).

After a 10–12-hour fast, fasting plasma glucose, fasting insulin, and serum magnesium were measured, with insulin resistance assessed using the HOMA-IR formula (fasting insulin × fasting glucose / 22.5). Regression analyses were performed with adjustment for age and sex.

Headline Findings

The study demonstrated a high prevalence of insulin resistance among participants with type 2 diabetes or prediabetes. Overall, 92.5% (99/107) met the study-defined criteria for insulin resistance, while 34.6% (37/107) had hypomagnesemia. The mean HOMA-IR was 9.94 ± 7.27. Notably, participants with serum magnesium in the lower-normal range had significantly higher insulin resistance compared with those in the normal magnesium range (HOMA-IR: 12.6 ± 6.75 vs. 8.52 ± 7.27; *P* = 0.005). The authors further reported that a 1 mg/dL decrease in serum magnesium was associated with a 47.2% increase in HOMA-IR (*P* = 0.009). However, serum magnesium was not significantly associated with fasting blood glucose or fasting insulin in this study. Women also had significantly higher HOMA-IR than men (11.69 ± 7.25 vs. 8.34 ± 6.96; *P* = 0.016).

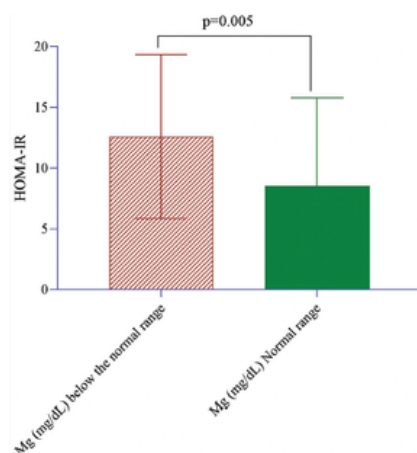


Figure 1 : Association between serum magnesium and homeostasis model assessment of insulin resistance. A multivariate regression model was used to estimate the P value, and the model was adjusted based on age and sex. Illustration Credit: Md Ahsanul Haq.

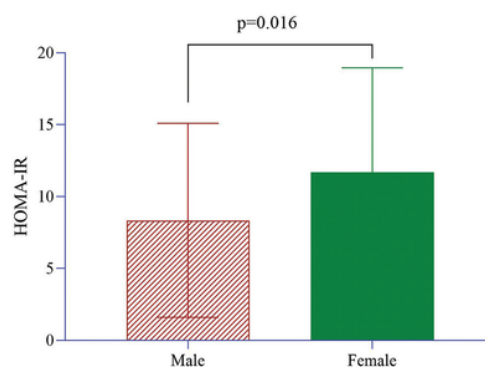


Figure 2 : The mean difference in homeostasis model assessment of insulin resistance between males and females. A univariate regression model was used to estimate the P value. Illustration Credit: Md Ahsanul Haq.

A practical reading of the result

- Magnesium status is relevant enough to consider measuring, rather than assuming it is normal.
- “Normal” is not always reassuring: the lower-normal group had higher HOMA-IR than the normal-magnesium group.
- The result is associative, not causal. The study did not randomize magnesium treatment, and it did not show that correcting magnesium deficiency improves HbA1c, fasting glucose, insulin requirements or cardiovascular outcomes.

What might this change in clinic?

- For patients with type 2 diabetes, the paper provides a rationale for a more deliberate magnesium review when insulin resistance is prominent or difficult to explain.

Consider adding serum magnesium to the assessment when:

- Glycemic control and insulin resistance appear worse than expected.
- There is a history suggesting possible magnesium depletion or reduced intake.
- Electrolyte abnormalities are being investigated.
- A patient is being evaluated for a broader metabolic or nutritional problem.
- Interpret the value in context: laboratory reference intervals differ, and serum magnesium may not perfectly reflect total-body or intracellular magnesium status. A single value should therefore be treated as a clinical clue, not a stand-alone diagnosis.

Clinical Takeaway for Physicians

Low magnesium is common enough in diabetes—and sufficiently linked with insulin resistance in this study—to deserve attention. The strongest clinical message is one of measurement and context: consider magnesium status as part of a structured evaluation, especially when insulin resistance is marked or unexplained. Correct a documented deficiency safely and deliberately, but do not overinterpret this observational result as proof of therapeutic benefit.

In type 2 diabetes, a low or lower-normal serum magnesium level may flag greater insulin resistance; use it to prompt assessment, not to replace comprehensive diabetes care.