could not find studies on the relationship between a physics requirement and aspects of performance in medical school or medical practice. However, the justification for a background in chemistry or physics is the same as it is for one in the behavioral and social sciences — it allows professors to move directly into more advanced topics. Given the conceptual breadth and strong clinical relevance of the behavioral and social sciences, it seems unrealistic to expect medical schools to adequately train students when many schools are unable to address advanced topics because their curriculum time must be used to remediate poor preparation by some students.

Although we appreciate Dienstag's perspective, we remain convinced that solid preparatory training and evaluation in the behavioral and social sciences is essential for training tomorrow's physicians.

Robert M. Kaplan, Ph.D.
National Institutes of Health
Bethesda, MD
robert.kaplan@nih.gov

Jason M. Satterfield, Ph.D.
University of California
San Francisco, CA

Raynard S. Kington, M.D., Ph.D.
Grinnell College
Grinnell, IA

The views expressed in this letter are those of the authors and do not necessarily represent those of the National Institutes of Health.

Since publication of their article, the authors report no further potential conflict of interest.

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# MIP-1α, MCP-1, and Desensitization in Anaphylaxis from Cow's Milk

**TO THE EDITOR:** Monocyte chemotactic protein 1 (MCP-1), also known as chemokine (C-C motif) ligand 2, has chemotactic activity for monocytes and basophils and causes the degranulation of basophils and mast cells.[1] Another chemokine, macrophage inflammatory protein 1α (MIP-1α), also called chemokine (C-C motif) ligand 3, is produced by macrophages, dendritic cells, and lymphocytes.[2] We have observed that 12 children with an allergy to cow's milk protein,[3] the most prevalent food allergy in children, have significantly lower levels of MCP-1 (mean, 11.1 pg per milliliter) and MIP-1α (12.1 pg per milliliter) than children with atopy who do not have this allergy (MCP-1, 28.4 pg per milliliter; and MIP-1α, 29.8 pg per milliliter).

Before desensitization was performed, oral and written informed consent was obtained from all patients or from their parent or guardian. Compilation of the data was recorded in accordance with European standards of data protection, and the study was approved by the clinical research ethics committee of the Hospital Universitario N.S. Candelaria. The study protocol is available with the full text of this letter at NEJM.org.

Twelve children (10 boys and 2 girls) who were 2 to 15 years of age (median, 6 years) with persistent allergy to cow's milk protein, severe recurrent episodes of grade 2 or 3 anaphylaxis, and multiple visits to the emergency department after accidental ingestion of cow's milk protein despite an appropriate restrictive diet underwent a 2-day rapid protocol of desensitization in the pediatric critical care unit of Hospital Universitario N.S. Candelaria. All patients had positive skin-prick tests and specific IgE antibodies to cow's milk protein (mean serum level of specific IgE to cow's milk protein, >100 kU per liter; mean serum level of specific IgE to casein protein, >100 kU per liter).

Thereafter, a second phase was scheduled in the outpatient clinic for the children to receive increasing doses of undiluted milk for a 6-week period. The goal was for the children to be able to consume 250 ml every 12 hours after this 6-week period. In less than 10 weeks, all 12 children were able to consume 250 ml of milk.

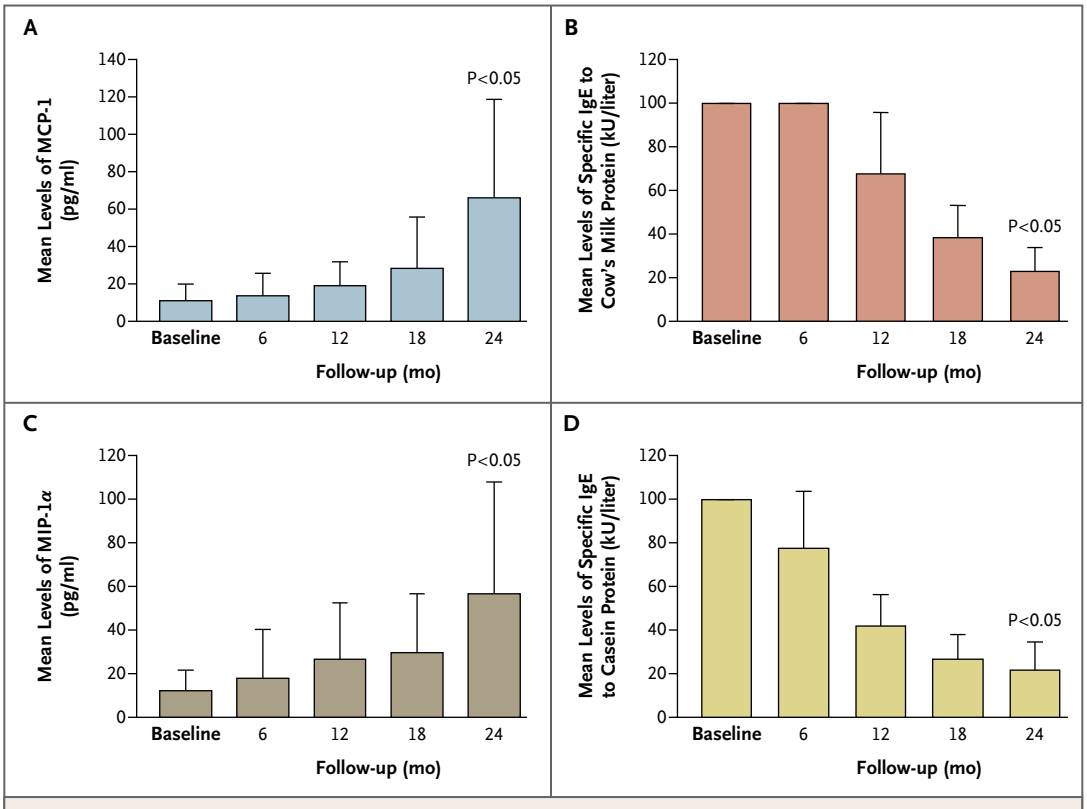


**Figure 1. Mean Serum Levels of Chemokines and Specific IgE to Both Cow's Milk Protein and Casein Protein in 12 Children after Desensitization.**

The T bars indicate 95% confidence intervals. MCP-1 denotes monocyte chemotactic protein 1, and MIP-1$\alpha$ macrophage inflammatory protein 1$\alpha$.

After 2 years, all the children still consumed a glass of milk every day, and their MCP-1 and MIP-1$\alpha$ levels measured by means of flow cytometry (Fig. 1A and 1C) were significantly higher than those in children with persistent allergy to cow's milk protein ($P<0.05$ by the Mann–Whitney U test). However, there were no significant changes in serum levels of serum cytokines such as interleukins 2, 4, 5, 6, 8, 10, 13, and 17; interferon-$\gamma$; eotaxin; RANTES (regulated upon activation normal T-cell expressed and secreted); and tumor necrosis factor $\alpha$.

Although allergy to cow's milk protein resolves in 70% of affected children by 3 years of age, its presence still leads to some deaths, life-threatening anaphylaxis, and many concerns in parents and guardians. With the therapeutic intervention described here, children with suspected allergy to cow's milk protein and anaphylaxis appeared to have less stress[4] and to be able to enjoy an unrestricted diet.

Elevated levels of MCP-1 and MIP-1$\alpha$ could reflect an ongoing subclinical response to food substances and to mast-cell degranulation and differential inflammatory-cell recruitment in response to the antigen-specific continuous challenge. We speculate that measurements of levels of MCP-1[2] and MIP-1$\alpha$[5] might be useful as markers of a successful protocol for milk protein desensitization.

Paloma Poza-R. Glez, M.D., Ph.D.

Hospital del Tórax
Tenerife, Spain

Yvelise Barrios-A. Franco, M.D.

Hospital Universitario de Canarias
Tenerife, Spain

Víctor Matheu, M.D., Ph.D.

Hospital Universitario N.S. Candelaria
Tenerife, Spain
victor.matheu@gmail.com

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## CORRECTIONS

Eltrombopag and Improved Hematopoiesis in Refractory Aplastic Anemia (July 5, 2012;367:11-9). Because of a printer's error, Figure 2 (page 17) was rendered incorrectly in the print issue. The figure is reprinted correctly here. We regret the error. The article is correct at NEJM.org.

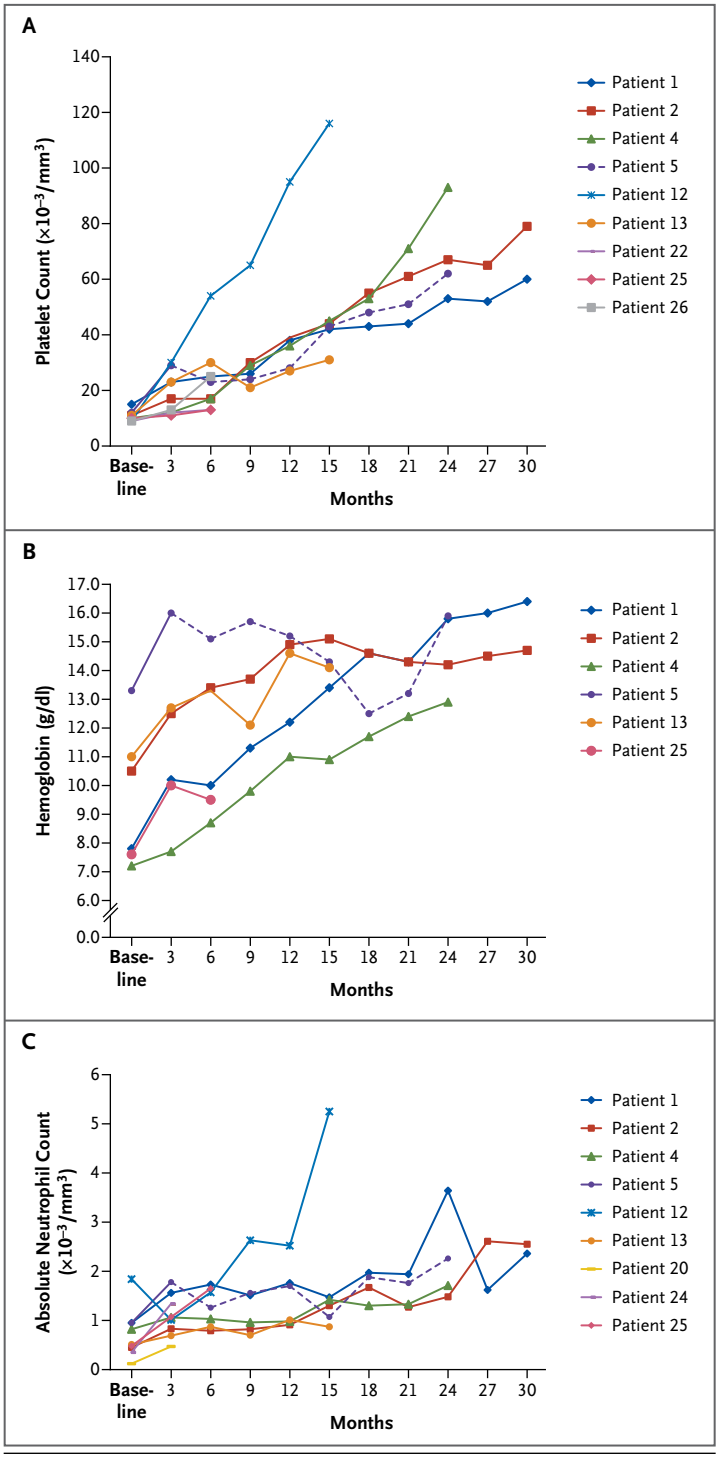


**Figure 2. Longitudinal Hematologic Improvements in Patients Who Received Eltrombopag.**

Longitudinal blood counts for all 11 patients who met response criteria in one or more lineages at 12 weeks are shown. Panel A shows platelet counts in the 9 patients who met the criteria for a platelet response at 12 weeks or later. Panel B shows hemoglobin responses in 6 patients who met the criteria for a hemoglobin response at 12 weeks or later. Panel C shows neutrophil responses in 9 patients who met the criteria for a neutrophil response at 12 weeks or later. The dashed portion of the line for Patient 5 indicates the time after which eltrombopag was discontinued because of putative cataract formation. This patient received monthly therapeutic phlebotomy to treat iron overload between months 6 and 18.