August 23, 2011

Margaret A. Hamburg, M.D.
Commissioner
U.S. Food and Drug Administration
Department of Health and Human Services
WO 2200
10903 New Hampshire Avenue
Silver Spring, MD 20993-0002

Dear Dr. Hamburg,

I am a 68 year old male in generally good health, and a journalist and editor by profession. I am writing to you as a long-time sufferer of severe gastroesophageal reflux disease (GERD), which was worsened and prolonged by my dependence on proton pump inhibitors (PPIs). My personal experience with PPI therapy is testament to the life-long dependence on the drugs, due to the strong rebound symptoms, that many patients experience.

In 1993, after moderate off-and-on ordinary heartburn, I had a severe bout of either heartburn or esophageal spasm, which sent me to the emergency room. The pain, concentrated just below my breastbone, was unbearable. After heart problems were ruled out, I was referred to a gastroenterologist who put me on a low dose of Pepcid (famotidine). I continued to have occasional flare-ups, and he soon put me on what became a daily maintenance 20 mg dose of Prilosec (omeprazole).

I settled into a pattern where I had to be progressively more careful with diet. Over time, the GERD episodes, including ones just like the attack that sent me to the ER, were more easily triggered. Gradually, I went off coffee, then off caffeinated tea. I stopped eating cheese, butter, or anything with cream. I ate relatively small meals. Moderate alcohol did not set it off, but I found that even modest departures from this diet regime would trigger a very painful and prolonged sharp pain, which seemed to be some kind of spasm. I found that immediate and high doses of over-the-counter (OTC) antacid and chewable Pepcid (30-50 mg) would break the spasm and sometimes head it off if I could sense it coming. I had to carry these around with me.

Whenever these bouts occurred—maybe six or eight times a year--my doctor recommended tripling the dose of Prilosec for a week or two to calm things down, then gradually tapering back to my normal dose. When I tried to taper the dose more abruptly, I experienced repeat attacks, more frequent and sometimes stronger than before. Over time, I became progressively reliant on higher doses for longer periods of time. At no point did I attempt to stop the PPI “cold turkey” for fear of even stronger reactions.

The doctor and I concluded that all this was a somewhat inexplicable, worsening propensity to GERD, possibly due to less efficient containment of stomach acid. We did not address the possibility of chronic rebound effects from the medication.

I had endoscopies after two of these recurrent bouts, which showed mild to moderate esophagitis; further routine endoscopies every five years to rule out more serious conditions have consistently shown a totally normal esophagus. We ruled out surgery as not worth the risk.

In 2009-2010, I had a period of several months of chronic heartburn, and the higher dose of Prilosec calmed things down only temporarily but did not change the pattern of worsening sensitivity to GERD and the need for progressively higher doses. At my gastroenterologist's suggestion, I was taking as much as 80 mg of Prilosec a day.

In 2010, after Dr. Sidney Wolfe shared with me studies showing rebound effects, I concluded that the Prilosec might be paradoxically responsible for a progressive worsening of my condition. I was referred by my internist to another gastroenterologist who agreed to work with me to end my dependence on Prilosec.

We devised an initial regimen where I would keep to a strict diet, begin 20 mg of Pepcid in the morning, and 40 mg before bed, plus OTC antacid, then reduce the Prilosec to 20 mg once daily--and after a few symptom-free weeks stop the Prilosec altogether. The premise was that Pepcid plus OTC antacid would reduce the acid and the symptoms, with far less rebound effect, and enable me to stop using PPI medication.

My doctor said that once I stopped the PPI, I might suffer some symptoms for a while due to the rebound effect, but to treat them with OTC antacid. In fact, due to this alternative step-down regimen of an H2A (Pepcid) and antacid, getting off Prilosec turned out to be surprisingly easy. After two weeks with no Prilosec, I was able to drop the morning dose of Pepcid. By January 2011, I was symptom-free.

Since then, to my very pleasant surprise, I am now able to drink caffeinated tea, eat modest amounts of butter and cheese, and even occasional ice cream. I have had no episodes whatsoever of the very painful esophageal spasms (or acute heartburn) that I suffered for nearly 20 years. Very occasional mild sour stomach is easily calmed with one or two chewable OTC antacid tablets. And I have cut the bedtime preventive dose of Pepcid to 30 mg, and will soon try reducing it to 20 mg.

I am not a clinician, though I was a medical writer for four years as national policy correspondent for the New England Journal of Medicine, but here is a working hypothesis and some informed conjectures of a layman:

My experience certainly seems to confirm the pattern of PPI medication causing--or in my case, seriously aggravating--the condition that it supposedly treats. In my case, the PPI seemed to have primed my system to produce increasing amounts of acid so that over time I was more prone to more attacks triggered by ever more minor departures from a

very low fat diet. The ever increasing amounts of PPI helped only temporarily and required dependence on even higher doses, and so on, over several cycles.

Only getting off the PPI reversed what seemed to be a chronic and progressive condition.

I strongly support the petition for Black Box Warnings on the rebound effects of PPIs, and the multiple serious safety concerns (of which I was never aware when taking such high PPI doses) identified in Public Citizen's petition. My conclusion from my own experience is that the use of Prilosec or other PPIs as initial therapy for chronic GERD should be reassessed. In my case, safer H-2 blockers (Pepcid), in moderately high and then diminishing doses, combined with OTC antacid use when needed for breakthrough attacks, have contained GERD far more effectively, and have reversed what was a steady worsening of the condition. It is possible, but surely unlikely, that mine is a unique case.

I would be happy to share additional information, and urge you to act on Public Citizen's recommendations.

Thank you,

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