EXHIBIT “B”

UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF NEW YORK

ROBERT SUTHERS and NIWANA MARTIN,	Docket No.:05 Civ. 4158 (PKC)
Plaintiffs,	
v.	
AMGEN, INC., a Delaware Corporation,	
Defendant.	A Civil Action

CERTIFICATION OF DON M. GASH, PH.D., JOHN T. SLEVIN M.D., AND GREG GERHARDT, PH.D.

1. We stand by the statements in our Affidavit of 20 April 2005. Here, we will provide additional information on our expertise in neuroscience, neurology and neurosurgery. We will also comment on issues that have been raised.

2. Dr. Gash received his Ph.D. from Dartmouth College in 1975. He holds the Alumni Chair at the University of Kentucky, where he is Professor and Chair of Anatomy and Neurobiology and Director of the Magnetic Resonance Imaging and Spectroscopy Center. As a pioneer in research on neural regeneration, the company that discovered GDNF (Synergen) asked him to begin research on the properties of the trophic factor in nonhuman primates in 1993. Since then, Dr. Gash and his colleagues have published 22 papers on GDNF. Dr. Gash initiated the IND for FDA approval for the Kentucky GDNF Clinical Trial. The IND was later transferred to Amgen, who assumed the costs of the study. In addition to the scientific reports on GDNF, he has published another 160 papers, invited reviews and book chapters. His expertise is on trophic factors, Parkinson's disease and other age-associated brain dysfunctions. His research

is funded through the National Institutes of Health, including a project on the Morris K. Udall Parkinson's Disease Research Center of Excellence at the University of Kentucky.

3. John T. Slevin, M.D., a professor in the Departments of Neurology and Molecular & Biomedical Pharmacology, is the Director of The Movement Disorder Clinic at the University of Kentucky. An experienced physician-investigator who has devoted his entire career to translational research, Dr. Slevin received undergraduate training at Johns Hopkins University and graduated from West Virginia University School of Medicine where he completed an Internal Medicine internship. He completed a residency in neurology at the University of Virginia and then returned to Johns Hopkins for fellowship training in neuropharmacology. He is certified in Neurology by the American Board of Psychiatry & Neurology, Inc. For over twenty years he has been the recipient of continuous VA Merit Review funding to evaluate basic mechanisms of epileptogenesis and has participated in both NIH and industry-sponsored clinical drug trials of anticonvulsants. He has participated in numerous NIH and industry-sponsored clinical drug trials for Parkinson's disease. In addition to preparing IND applications, he has experience in developing Phase 1 and 2 clinical trials. He has published more than 130 abstracts, peer-reviewed papers and book chapters.

4. Dr. Gerhardt is Professor and Director of the Morris K. Udall Parkinson's Disease Research Center of Excellence at the University of Kentucky Chandler Medical Center. His primary academic appointment is in Anatomy & Neurobiology with joint appointments in Neurology, and Psychiatry. He is also the Director of the Center for Sensor Technology (CenSeT) and he has been Editor-in-Chief (Americas and Australasia) of the Journal of Neuroscience Methods since 1999. He has received numerous awards including a recent Level II Research Scientist Development Award from NIMH (2000-2005) and he has published over 200

original papers and book chapters. His research focuses on Parkinson's disease, normal aging and the repair of damaged dopamine neurons in the basal ganglia of the brain using growth factors such as GDNF. In addition, his laboratory develops technologies to directly measure chemical communication in the living brain.

5. In our opinion, the intraputamenal toxicology study on rhesus monkeys was seriously compromised by procedures that resulted in the euthanasia of 13 out of 72 animals before the study's conclusion. The problems with procedures have introduced confounds into the toxicology study that could seriously affect the predictive value of other results. For example, if taken at face value, the toxicology study would predict a morbidity/mortality rate of 18% in human studies. This prediction was not realized; the safety profile of patients receiving intraputamenal GDNF therapy for up to three years has been excellent.

6. If the cerebellar lesions are not an artifact resulting from the procedural problems (and the lesions seen in the study have not been replicated), a complete and independent assessment of the factors underlying the development of cerebellar lesions in the four monkeys cannot be made until all the data are made public. In our opinion, GDNF withdrawal is the other leading candidate as the mechanism of action producing the lesions and is consistent with confidential information which Amgen has not released.

7. Contrary to the statements provided by some other individuals, the cerebellum can be closely monitored for tissue loss by MRI. Techniques are available and used for the patients in the Kentucky study to detect the loss of as little as 0.5% of cerebellar tissue. It is generally considered that tissue loss/injury compromising more than 25% of the cerebellum is required before clinical symptoms emerge. It should be stated again that cerebellar lesions from GDNF therapy have not been found in patients.

8. Contrary to the statements provided by some other individuals, the situation of neutralizing GDNF antibodies is similar to that of neutralizing antibodies to beta interferon. Up to 45% of the patients treated with beta interferon develop antibodies, without clinical manifestations. One reason is that other related proteins in the body can substitute for beta interferon. GDNF also has closely related proteins that can substitute for it. An example is neurturin, which is found in overlapping brain areas with GDNF. Other proteins related to GDNF are found outside of the brain in the body. It should be stated again that clinical manifestations to GDNF antibodies have not been documented in patients receiving GDNF therapy. The muscle weakness in one patient receiving GDNF has since been reported as being due to other causes.

9. Two members of the Kentucky team (DMG and JTS) were investigators attending the FDA meeting on January 11, 2005. The FDA report on the meeting has not been released by Amgen and the versions given by non-investigators are incomplete in our opinion. The presentations by the investigators on the indications of efficacy and paucity of clinically manifest side effects in the two phase 1 trials and the phase 2 trial were accepted by the FDA. The investigators also presented analyses of the safety issues, specifically the issues of cerebellar toxicity and antibody production to GDNF, and how they could be effectively managed. At the end of our presentation, the FDA did not see any reason why the current patients could not continue to receive drug in extended treatment. The conditions for the FDA to approve an extended study included that it be well organized and coordinated, with responsible oversight. Another condition was that the extended study should include more extensive monitoring of the patients than normal. The investigators considered the FDA requirements to be reasonable. We

were fully prepared to work with Amgen and the FDA to provide current patients, with proper informed consent, the opportunity for receiving extended GDNF therapy.

10. Based on our peer-reviewed published results of the 10 Parkinson's disease patients in the Kentucky phase 1 trial and the peer-reviewed published results on 5 Parkinson's disease patients in the Bristol phase 1 trial by Steven Gill and his colleagues, we hold that there is significant evidence for the safety and efficacy of intraputamenal infusion of GDNF. Our preclinical research posits that the efficacy of intraputamenal GDNF therapy is directly related to dose and tissue distribution. These parameters were sufficiently optimized in the two Phase 1 trials to where 15 out of 15 treated patients showed significant clinical improvements. We believe that they were on the bubble of achieving efficacy in the Amgen Phase 2 trial. Dose and delivery procedures need to be optimized for any phase 2 patients who elect to go back on GDNF therapy. The simplest approach would be to increase the dose.

11. I certify under penalty of perjury under the laws of the United States of America that the foregoing statements are true and based upon my personal knowledge. I am aware that if any of the foregoing statements are false, I may be subject to punishment.

Dated: Monday, May 23, 2005

Don M. Gash, Ph.D.

Dated: Monday, May 23, 2005

John T. Slevin, M.D.

Dated: Monday, May 23, 2005

Greg Gerhardt, Ph.D.