send to its customers a letter indicating that it had decided to the exit the CISP business. After the Acquisition, Charlotte Pipe destroyed the CISP production equipment that it acquired from Star Pipe.

D. Conditions of Entry

Entry into the relevant markets would not be timely, likely, or sufficient in magnitude, character, and scope to deter or counteract the anticompetitive effects of the Acquisition.

E. Effects

The effects of Charlotte Pipe's acquisition of Star Pipe's CISP business have been a substantial lessening of competition in the relevant markets. Specifically, the Acquisition has: eliminated actual, direct, and substantial competition between Charlotte Pipe and Star Pipe in the relevant markets; substantially increased the level of concentration in the relevant markets; eliminated a maverick firm; increased the ability of Charlotte Pipe unilaterally to exercise market power; and prevented Star Pipe and certain Star Pipe employees from re-entering the CISP products market for a period of six years.

II. The Proposed Order

Paragraph II of the Proposed Order requires Charlotte Pipe to provide prior notification to the Commission of an acquisition of any entity engaged in the manufacture and sale of CISP products in or into the United States. This paragraph also requires Charlotte Pipe to comply with premerger notification procedures and waiting periods similar to those found in the HSR Act.

This provision is necessary because Charlotte Pipe has previously acquired several firms in the CISP products market in non-reportable transactions. The Proposed Order affords the Commission an appropriate mechanism to review all proposed acquisitions by Charlotte Pipe in the CISP products market to guard against future anticompetitive transactions.

Paragraph III of Proposed Order prevents Charlotte Pipe from enforcing the Confidentiality and Non-Competition Agreement. This frees Star Pipe, and its current and former employees, to enter and compete against Charlotte Pipe in the United States, Canada, or Mexico.

Paragraphs IV–VII impose reporting and other compliance requirements. In particular, Charlotte Pipe is required to send a letter to its customers and to maintain a link on its Web site relating to the Acquisition and Charlotte Pipe's other non-reportable transactions, including Matco-Norca in 2009, DWV Casting Company (''DWV'') in 2004, and Richmond Foundry, Inc. (''Richmond Foundry'') in 2002. This provision is appropriate because Charlotte Pipe's confidential acquisitions are not widely known in the CISP industry and have given rise to a perception among distributors and end-users that importers of CISP products are transient and unreliable operations. The proposed order serves to inform market participants about Charlotte Pipe's role in the exit of Star Pipe, Matco-Norca, DWV, and Richmond Foundry from the CISP industry.

The Proposed Order will expire in 10 years.

By direction of the Commission.

Donald S. Clark,

Secretary.

[FR Doc. 2013–08217 Filed 4–8–13; 8:45 am]

BILLING CODE 6750–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Office of the Secretary

Findings of Research Misconduct

AGENCY: Office of the Secretary, HHS.

ACTION: Notice.

SUMMARY: Notice is hereby given that the Office of Research Integrity (ORI) has taken final action in the following case:

Andrew Aprikyan, Ph.D., University of Washington: Based on the report of an investigation conducted by the University of Washington (UW), the UW School of Medicine Dean's Decision, the Decision of the Hearing Panel at UW, and additional analysis conducted by ORI, ORI found by a preponderance of the evidence that Dr. Andrew Aprikyan, former Research Assistant Professor, Division of Hematology, UW, engaged in research misconduct in research supported by National Cancer Institute (NCI), National Institutes of Health (NIH), grant CA89135 and National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), NIH, grant DK18951, and applies to the following publications and grant applications:

- *Blood* pre-published online on January 16, 2003 (''NEM'')
- *Experimental Hematology* 31:372–381, 2003 (''CMA'')
- *Blood* 97:147–153, 2001 (''ISB'')
- R01 CA89135–01A1
- R01 HL73063–01
- R01 HL79615–01

Blood pre-published online on January 16, 2003, has been retracted and *Experimental Hematology* 31:372–381, 2003, has been corrected.

Specifically, ORI finds that by a preponderance of the evidence, Respondent falsified and/or fabricated results relating to the above publications and grants. Specifically, Respondent:

1. Falsely reported sequencing data in the NEM manuscript to strengthen the hypothesis that NE mutations contributed to the phenotype observed in severe congenital neutropenia (SCN) patients. Specifically:

a. Respondent falsely reported in Figures 2A and 3 that patient 3 had the R191Q neutrophil elastase (NE) mutation, when the majority of the sequencing experiments showed that the mutation was not present.

b. Respondent fabricated text (p. 12) reporting that sequencing of RT–PCR products confirmed the expression of the NE mutants in the SCN patients and that no mutations were present in the granulocyte colony stimulating factor receptor (G–CSFR) gene and the Wiskott-Aldrich Syndrome (WAS) gene in SCN patients, when based on the lack of original records the experiments were not performed. The false claim for G–CSFR sequencing was also reported in CA89135–03.

2. Falsely reported a two-fold increase in apoptosis of human promyelocytic (HL–60) cells transfected with NE mutants compared to wild type NE in Figure 4A, NEM, Figure 6A, CMA, Figure 8, HL73063–01, and Figure 7, HL79615–01. Respondent used arbitrary flow cytometry data files to generate histograms with the desired result. The false results supported the hypothesis that the NE mutations were sufficient for impaired survival of human myeloid cells.

3. Falsified NE and ß-actin Western blots in Figure 4B *Blood,* pre-published online January 16, 2003, Figure 5B of the manuscript initially submitted to *Blood* April 2002, and Figure 6B *Experimental Hematology* 31:372–381, 2003, by falsely labeling lanes to support the hypothesis that accelerated apoptosis in mutant NE transfect HL–60 cells was due to the mutation and not the level of protein present. Specifically:

a. Respondent used portions of a single NE Wester blot to represent: Figure 4B as HL–60 cells transfected with L92H, R191Q, and wtNE, when the cells were transfected with R191Q, P110L, and D145–152; Figure 5B as HL–60 transfected with wtNE, mutNE, and EGFP when they were cells transfected with NE mutants, P110L, D145–152, and 194

b. Respondent used portions of a single ß-actin Western blot to represent: Figure 4B as HL–60 cells transfected

with L92H, R191Q, and wtNE, when they were cells transfected with I31T, P110L, and G185R mutants; Figure 5B as HL–60 cells transfected with wtNE, mutNE, and EGFP, when they were cells transfected with P110L, I31T, and INE; Figure 6B as HL–60 cells transfected with G185R, mock, D145–152, and P110L NE mutants, when they were cells transfected with I31T, P110L, G185R, and 32. The false ß-actin Western blot in Figure 6B was also included in HL73063–01, Figure 8 (where the I31Tlane was labeled correctly), and HL79615–01, Figure 7.

4. Falsified the reported methodology for flow cytometry experiments in Figure 4A, NEM, Figures 1 and 2, and Tables 2 and 3, CMA, and Figures 4, 5, and 6, ISB, to validate the key hypothesis showing accelerated apoptosis in SCN and CN patients. The methodology claimed that flow cytometry experiments were gated for GFP+ populations, or that cell purity was greater than 96%, when based on the available original records, the experiments were not performed as stated.

5. Falsified Figure 2, CMA, Figure 2, HL73063–01, Figure 3, HL79615–01, and Figure 5, CA89135–01A1, demonstrating that the overnight cultures of CD34+ and CD33+ bone marrow cells from SCN/AML patients showed normal cell survival, and only the CD15+ overnight cultures showed accelerated apoptosis, when the actual record available contradicted this result. Respondent used flow cytometry data files to generate histograms with the desired result to support the hypothesis that the progression from SCN to leukemia (AML) involves acquired G–CSFR mutations that override the pro-apoptotic effect of the NE mutations in primitive progenitor cells.

Dr. Aprikyan has entered into a Settlement Agreement in which he denied ORI's findings of research misconduct based on the UW Faculty Adjudication Hearing Panel decision. The settlement is not an admission of liability on the part of the Respondent. Respondent entered into the Agreement solely because contesting the findings would cause him undue financial hardship and stress, lead to lengthy and costly appellate proceedings, and he wished to seek finality. Respondent agreed not to appeal the ORI findings of research misconduct set forth above. He has agreed, beginning on March 12, 2013:

(1) If within two (2) years from the effective date of the Agreement, Respondent receives or applies for U.S. Public Health Service (PHS) support, Respondent agreed to have his research supervised for a period of two (2) years; Respondent agreed that prior to the submission of an application for PHS support for a research project on which his participation is proposed and prior to his participation in any capacity on PHS-supported research, Respondent shall ensure that a plan for supervision of his duties is submitted to ORI for approval; the supervision plan must be designed to ensure the scientific integrity of his research contribution; Respondent agreed that he shall not participate in any PHS-supported research until such a supervision plan is submitted to and approved by ORI; Respondent agreed to maintain responsibility for compliance with the agreed upon supervision plan;

(2) If within two (2) years from the effective date of the Agreement, Respondent receives PHS support, Respondent agreed that for two (2) years, any institution employing him shall submit, in conjunction with each application for PHS funds, or report, manuscript, or abstract involving PHS-supported research in which Respondent is involved, a certification to ORI that the data provided by Respondent are based on actual experiments or are otherwise legitimately derived and that the data, procedures, and methodology are accurately reported in the application, report, manuscript, or abstract; and

(3) Respondent agreed not to serve in any advisory capacity to PHS including, but not limited to, service on any PHS advisory committee, board, and/or peer review committee, or as a consultant for a period of two (2) years beginning with the effective date of the Agreement.

FOR FURTHER INFORMATION CONTACT: Director, Office of Research Integrity, 1101 Wootton Parkway, Suite 750, Rockville, MD 20852, (240) 453–8200.

David E. Wright,

Director, Office of Research Integrity.

[FR Doc. 2013–08207 Filed 4–8–13; 8:45 am]

BILLING CODE 4150–31–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Centers for Disease Control and Prevention

[30Day–13–12MX]

Agency Forms Undergoing Paperwork Reduction Act Review

The Centers for Disease Control and Prevention (CDC) publishes a list of information collection requests under review by the Office of Management and Budget (OMB) in compliance with the Paperwork Reduction Act (44 U.S.C. Chapter 35). To request a copy of these requests, call (404) 639–7570 or send an email to *omb@cdc.gov.* Send written comments to CDC Desk Officer, Office of Management and Budget, Washington, DC 20503 or by fax to (202) 395–5806. Written comments should be received within 30 days of this notice.

Proposed Project

Research to Inform the Prevention of Asthma in Healthcare—New—National Institute for Occupational Safety and Health (NIOSH), Centers for Disease Control and Prevention (CDC).

Background and Brief Description

Healthcare is the largest industry in the United States and performs a vital function in society. Evidence from both surveillance and epidemiologic research indicates that healthcare workers have an elevated risk for work-related asthma (WRA) associated with exposure to groups of agents such as cleaning products, latex, indoor air pollution, volatile organic compounds (VOCs) and bioaerosols. Recent epidemiologic studies of WRA among healthcare workers have utilized job exposure matrices (JEMs) based on probability of exposure, however, specific exposures/etiologic agents are not well characterized and quantitative exposure measurements are lacking. In this project, NIOSH will augment the existing JEM with quantitative exposure data, which will significantly enhance the existing JEMs and develop a survey questionnaire for asthma in healthcare.

Since asthma continues to be a problem among healthcare workers, the overall goal of this project is to prevent work-related asthma among healthcare workers. The primary objective is to identify modifiable occupational risk factors for asthma in healthcare that will inform strategies for prevention. Specific Aims that support the Primary Objective are:

Aim 1. Measure frequency of asthma onset, related symptoms, and exacerbation of asthma in selected healthcare occupations

Aim 2. Assess associations between asthma outcomes and exposures to identify modifiable risk factors

In order to accomplish the goal and aims of this project NIOSH has developed a survey designed to collect information about work history, workplace exposures and asthma health from workers in the healthcare industry. Aim 1 of this project will be completed using data exclusively from this survey. While aim 2 will be completed using asthma outcome data from the survey and exposure data from the JEM