RESEARCH LETTER

Scientific Publications on Firearms in Youth Before and After Congressional Action Prohibiting Federal Research Funding

In January 1996, Congress passed an appropriations bill amendment prohibiting the US Centers for Disease Control and Prevention (CDC) from using “funds made available for injury prevention . . . to advocate or promote gun control.”1 This provision was triggered by evidence linking gun ownership to health harms,1 created uncertainty among CDC officials and researchers about what could be studied,2 and led to significant declines in funding.3 We evaluated the change in the number of publications on firearms in youth compared with research on other leading causes of death before and after the Congressional action. We focused on children and adolescents because they disproportionately experience gun violence and injury.4

Methods | We identified 10 leading causes of death among children and adolescents aged 1 to 17 years using CDC data on mortality between 1991 and 2010.5 Each cause was then matched to a Medical Subject Heading, and PubMed was searched from 1991-2010 using causes of death and child or adolescent to determine the annual number of publications. Publications of all types and on all outcomes were included for each cause. To explore funding trends, we identified federal and private or nonfederal funding sources for firearms studies as reported by PubMed.

To quantify the change in annual publications, we used a quasi-experimental differences-in-differences design implemented with log-linear regression models.4 This approach assessed change in the volume of firearms publications before and after a year, relative to the change in volume of publications on other causes before and after the same cut-point year. The null hypothesis was that the percentage change in volume of firearms publications would have been the same as the percentage change in volume of nonfirearms publications despite the events of 1996.6

Because the Congressional action may have had a delayed effect on publications, we allowed for a lag between the amendment and changes in annual firearms publications of 1 to 6 years. For each year, our model was specified as: publication volume = f(causes of death, firearms, post–cut-point year, firearms × post–cut-point year), in which post–cut-point year is an indicator variable for whether the data were drawn from before or after the year in question. The interaction term coefficient was the parameter of interest. Funding trends were categorized into prelag, perilag, and postlag periods.

Analyses were conducted using Stata version 12.0 (StataCorp Inc). Two-tailed P value of less than .05 was considered significant.

Results | Between 1991 and 2010, there were 310,203 deaths among youth from the 10 leading causes and 301,475 publications. Firearms accounted for 12.6% of deaths, but less than 0.3% of publications. There were 25 publications on firearms in 1991, 61 in 1999, and 33 in 2009 (Figure). In contrast, publications on neoplasms, which are responsible for approximately the same number of deaths, increased from 5519 to 9707. Using the differences-in-differences model and 1999 as the cut point, the volume of publications on firearms was 24.5% (P = .001) lower than it may have otherwise been compared with publications not on firearms (Table). Using different lag times did not significantly alter the results. The estimate was
similar when considering only publications associated with a single cause of death (87%; differences-in-differences estimate, −19.9%; P = .03). In 1991-1996, 1997-2002, and 2003-2010, 33, 43, and 41 firearms publications reported federal funding, respectively; 25, 63, and 86 reported private or nonfederal funding.

Discussion | We only found modest increases in the number of scientific publications on firearms between 1991 and 2010, in contrast to other leading causes of death in youth. The change in number of publications on firearms was lower than anticipated compared with publications not on firearms. There was not a discrete point identified at which the pattern of publications changed. Therefore, whether the Congressional action or other events were responsible is unclear. Important limitations include use of a single database (PubMed) and lack of information on study inception. The effect on publications after President Obama’s January 2013 memorandum directing the CDC to conduct or support research on the causes of gun violence and approaches to prevent it should be evaluated.

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Author Contributions: Dr Ladapo had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Ladapo, Rodwin. Acquisition of data: Ladapo, Rodwin. Analysis and interpretation of data: Ladapo, Ryan, Trasande, Blustein. Drafting of the manuscript: Ladapo, Blustein.
negative outcomes emerged from a recent study,2 in which the retrospective cohort of 1849 patients from 13 centers worldwide in patients with papillary thyroid cancer (PTC) in a large retrospective study. This evidence extends and confirms the results from the study by Xing et al1 that the BRAF V600E mutation and mortality in patients with papillary thyroid cancer. JAMA. 2013;309(6):549-550.

In Reply I disagree with Dr Ciarrocchi and colleagues that BRAF V600E does have a significant association with mortality, which likely occurs through promoting aggressive tumor behaviors and would be misleadingly lost using conventional multivariable models. Further efforts are needed to define how to specifically use the prognostic value of BRAF V600E clinically.

Some studies have not shown an association of BRAF V600E with aggressive behaviors of PTC. However, most have, as shown in large meta-analyses.1-3 Many factors could bias the conclusion about the prognostic value of BRAF V600E. For example, in a large study of 631 patients with PTC that failed to show an association of aggressiveness of PTC with BRAF V600E,3 many patients (41.5%) had only partial thyroidectomy, making thorough pathological characterization difficult. In addition, the majority of the patients in this study did not receive radioiodine ablation, potentially masking an effect of BRAF V600E on clinical outcomes given that BRAF mutation-negative PTC is more sensitive to radioiodine ablation.1,2

Ciarrocchi and colleagues’ statement that a previous study4 showing no aggressive role of BRAF V600E in PTC confirms our study is incorrect. However, the 2 studies are not comparable; the former was a single institution study focused on a small number of highly selected cases and the latter was a large multicenter study of consecutive cases.

The study by Sancisi et al4 selected patients with distant metastases who only had well-differentiated primary cancer;