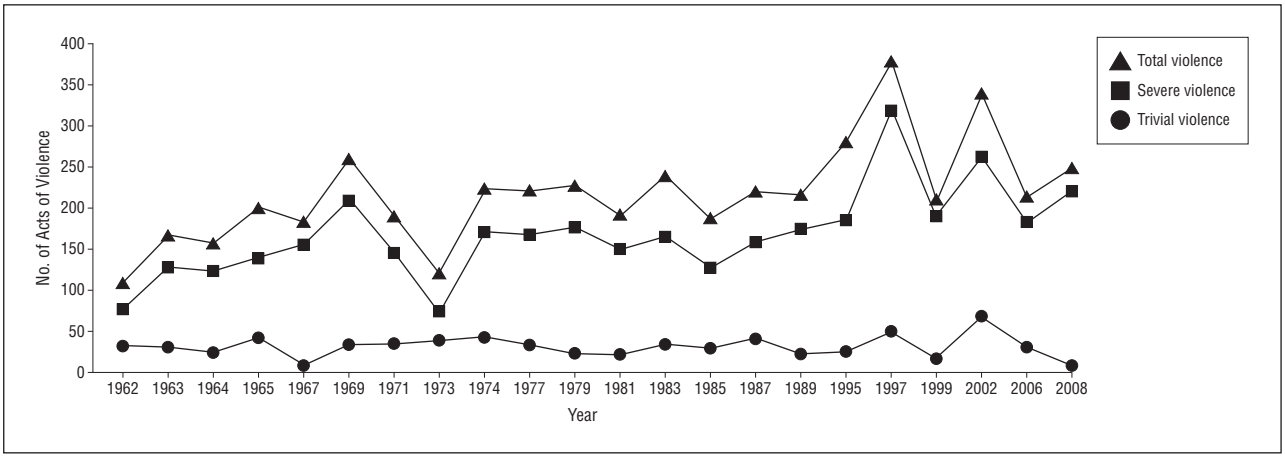

Figure. Violence in Bond films by year of release, showing counts of severe and trivial violence as well as total violence. Total violence is higher than the sum of severe and trivial violence because it includes an adjustment for mass scenes of violence.

Comment. In this review of 22 films spanning almost half a century, portrayals of violence increased such that rates of violence in 2008 were double those observed in 1962. This was due to an increase in severe rather than trivial violent imagery. The findings support our hypothesis that movies, in general, have become more violent. Others have made similar observations in studies of more diverse samples of films.[4,5]

All Bond films were rated as suitable for children or adolescents with parental guidance. The Bond films will therefore have been seen by many young people and the increase in violent content of these movies represents a general increase in the exposure of young people to media violence. Of further concern is that because young people frequently access R-rated films,[5] they are likely to be exposed to even higher rates of violence in these films. The increase in children and adolescents' exposure to media violence is a matter of concern, given that there is good evidence to suggest that viewing violence has associations with violent behavior.[6] Ratings systems should be reviewed so that there is a greater emphasis on levels and severity of violence.

Helena M. McAnally, PhD
Lindsay A. Robertson, MPH
Victor C. Strasburger, MD
Robert J. Hancox, MD

Published Online: December 10, 2012. doi:10.1001/jamapediatrics.2013.437
Author Affiliations: Departments of Psychology (Dr McAnally) and Preventive and Social Medicine (Ms Robertson and Dr Hancox), University of Otago, Dunedin, New Zealand; and University of New Mexico School of Medicine, Albuquerque (Dr Strasburger).
Correspondence: Dr McAnally, Department of Psychology, University of Otago, PO Box 56, Dunedin 9054, New Zealand (helena.mcanally@otago.ac.nz).
Author Contributions: *Study concept and design:* McAnally, Robertson, Strasburger, and Hancox. *Acquisition of data:* McAnally, Robertson, and Hancox. *Analysis and interpretation of data:* McAnally and Hancox. *Drafting of the manuscript:* McAnally, Strasburger, and Hancox. *Critical revision of the manuscript for important intellectual content:* McAnally and Robertson. *Statistical analysis:* McAnally and Hancox. *Obtained funding:* Hancox. *Administrative, technical, and material support:* Robertson.
Conflict of Interest Disclosures: None reported.
Funding/Support: This research was funded by a grant from the Deputy Vice-Chancellor of Research at the University of Otago.
Role of the Sponsors: The funder had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; or preparation, review, or approval of the manuscript.
Additional Contributions: We thank Rob McGee, PhD, for helpful input into the early phases of planning this research.

1. Ash R. *Top 10 of Everything: 2010*. London, England: Hamlyn; 2009.
2. Anderson CA, Bushman BJ. Effects of violent video games on aggressive behavior, aggressive cognition, aggressive affect, physiological arousal, and prosocial behavior: a meta-analytic review of the scientific literature. *Psychol Sci*. 2001;12(5):353-359.
3. *National Television Violence Study*. Vol 1. Santa Barbara: Center for Communication and Social Policy, University of California; 1997.
4. Jamieson PE, More E, Lee SS, Busse P, Romer D. It matters what young people watch: heath risk behaviors portrayed in top-grossing movies since 1950. In: Jamieson PE, Romer D, eds. *The Changing Portrayal of Adolescents in the Media Since 1950*. New York, NY: Oxford University Press; 2008:105-131.
5. Worth KA, Gibson Chambers J, Nassau DH, Rakhra BK, Sargent JD. Exposure of US adolescents to extremely violent movies. *Pediatrics*. 2008;122(2):306-312.
6. Council on Communications and Media. From the American Academy of Pediatrics: policy statement. media violence. *Pediatrics*. 2009;124(5):1495-1503.

COMMENTS

Genetic Risk in Childhood Obesity: Implications for Clinical Practice

We wish to comment on the recently published editorial by Fernandez[1] in relation to an article by Belsky et al[2] that validates a gene-based risk tool for obesity in a birth cohort followed up prospectively.

First, given the steeply rising rates of obesity over the last few decades, it is clear that changes in lifestyle (ie, the environment) are "pivotal" to the development of obe-

sity.[1] While the genes underlying obesity have been present for centuries, it is their effect that has recently emerged in societies where "lifestyle" has dramatically changed. This means regardless of genetic predisposition, there need be no concern about "genetic determinism" in the public health community. Instead there should be enthusiasm for the potential to identify those at greatest risk for targeted and cost-effective interventions. This must be preferable to (over)generalized health messages that are inconsequential for the wider population. The best example of the success of the targeted approach comes from the enormous drop in death rates from coronary artery disease following several decades of cholesterol-lowering treatment (see later).

Second, while the genetic risk score described by Belsky et al[2] may explain only a fraction of the overall genetic risk of obesity, this does not reduce its potential to provide pediatricians with a novel tool to engage their high-risk patients to achieve meaningful outcomes. After all, primary care physicians have been using serum cholesterol measurement for decades to help identify and engage patients in risk reduction for coronary artery disease. Serum cholesterol accounts for a tiny proportion of the overall risk for coronary artery disease.[3] It is therefore possible that combining personalized genetic information with clinical variables might have an equally positive effect where there are no other predictive risk tools available to the clinician.[4] The important issue is to correctly weight the gene-based risk information against other important nongenetic risk variables.[5] In a developmental condition like obesity, with its roots in early life, the study by Belsky et al strongly supports the view that genetic information is helpful in identifying those at highest risk of obesity. That family history provided additional predictive utility over and above that of their genetic risk score is consistent with the majority of other studies of complex diseases.[5] This additional predictive utility of the single-nucleotide polymorphisms[2] is likely to reflect population-wide effects on obesity (from genome-wide association studies) in contrast to family-specific effects represented in the family history (eg, rare variants).

Robert P. Young, BMedSc, MBChB, DPhil, FRACP, FRCP
Raewyn J. Hopkins, BN, MPH

Author Affiliations: Schools of Biological Sciences and Health Sciences, University of Auckland, Auckland, New Zealand.
Correspondence: Dr Young, Respiratory Genetics Group, PO Box 26161, Epsom 1344, Auckland, New Zealand (roberty@adhb.govt.nz).
Author Contributions: *Study concept and design:* Young and Hopkins. *Acquisition of data:* Young. *Analysis and interpretation of data:* Young. *Drafting of the manuscript:* Young. *Critical revision of the manuscript for important intellectual content:* Young and Hopkins. *Administrative, technical, and material support:* Young and Hopkins.
Conflict of Interest Disclosures: None reported.
Funding/Support: Dr Young, and the funding of his research, has been supported by grants from the University of Auckland, Health Research Council of New Zealand, and Synergenz BioSciences Ltd.

1. Fernandez JR. Translating developmental genetic findings into obesity-related clinical practice: are we there yet? *Arch Pediatr Adolesc Med*. 2012; 166(6):576-577.
2. Belsky DW, Moffitt TE, Houts R, et al. Polygenic risk, rapid childhood growth, and the development of obesity: evidence from a 4-decade longitudinal study. *Arch Pediatr Adolesc Med*. 2012;166(6):515-521.
3. Milne RJ, Gamble GD, Whitlock G, Jackson RT. Discriminative ability of a risk-prediction tool derived from the Framingham Heart Study compared with single risk factors. *N Z Med J*. 2003;116(1185):U663.
4. Hopkins RJ, Young RP, Hay B, Gamble GD. Gene-based lung cancer risk score triggers smoking cessation in randomly recruited smokers [abstract]. *Am J Respir Crit Care Med*. 2011;183:A5441.
5. El-Zein RA, Young RP, Hopkins RJ, Etzel CJ. Genetic predisposition to chronic obstructive pulmonary disease and/or lung cancer: important considerations when evaluating risk. *Cancer Prev Res (Phila)*. 2012;5(4):522-527.

In reply

The letter by Young and Hopkins provides an interesting perspective into the much-needed ongoing conversation regarding the use of genetic testing in clinical practice to evaluate risk for complex diseases. Certainly, the use of an obesity risk-prediction measure based on genetic information is of great clinical interest and promises great utility in identifying high-risk patients. However, although the work of Belsky and colleagues[1] is certainly a scientifically sound attempt in that direction, its actual application within the clinical setting raises controversy.

Two aspects need to be considered when facing such controversy. First, it is evident that, in obesity and its related traits, our understanding of the interaction between genetic susceptibility and the obesogenic environment continues to be limited and is furthermore scientifically hindered by the difficulties and limitations of defining and measuring "environment." In the case of obesity, the interaction with the environment is of great relevance, given that any behavioral approach targeted to prevention will be greatly influenced by an individual's surroundings. Second, the identification of high-risk individuals based on genetic susceptibility requires a predictive test, and any predictive test is limited by its accuracy and dependability. The concern of applying a genetic risk score, as described by Belsky et al,[1] lies mainly in the dependability of a test that, using 16 single-nucleotide polymorphisms, explains less than 2% of the variation in body mass index. This limitation does not reduce the relevance and importance of the measure; however, it questions its clinical applicability as a predictive tool for obesity risk at the present time.

The scientific community is enthusiastically motivated to continue the exploration and identification of molecular and statistical methods to improve the accuracy and dependability of genetic prediction in obesity-related and body composition parameters. A good example is the application of whole-genome prediction approaches that explain 45% of the variance for height[2], compared with the 3.7% of the population variation explained by a genome-wide association study approach,[3] proving to be a more comprehensive predictive tool. However, the dependability of a genetic predictive tool will always be relative, given that heritability estimates for BMI are not constant across the life span.[4]

Implementing healthy behaviors that will reduce obesity risk in the pediatric population is paramount in achieving meaningful outcomes in high-risk individuals. In reality, if overall health in the pediatric population is the goal, the lifestyle recommendations for children at lower risk for obesity should not differ much from those at high risk. Un-

til research uncovers mechanisms that reliably link obesity genetic predisposition to obesity clinical prevention (either through pharmacological or behavioral approaches), translating an undependable research tool to clinical practice should not be biased by the novelty of an approach and should be weighted by ethical implications of potential stereotyping that could impair health care delivery. Perhaps, prior to applying such a tool to clinical settings, educational conversations and interactions should occur that involve scientists, clinicians, patients, research participants, and policy makers.

Jose R. Fernandez, PhD

Author Affiliation: Department of Nutrition Sciences, University of Alabama at Birmingham, Birmingham.
Correspondence: Dr Fernandez, Department of Nutrition Sciences, University of Alabama at Birmingham, Webb 443A, 1530 Third Ave S, Birmingham, AL 35294-3360 (jose@uab.edu).
Conflict of Interest Disclosures: None reported.

1. Belsky DW, Moffitt TE, Houts R, et al. Polygenic risk, rapid childhood growth, and the development of obesity: evidence from a 4-decade longitudinal study. *Arch Pediatr Adolesc Med*. 2012;166(6):515-521.
2. Yang J, Benyamin B, McEvoy BP, et al. Common SNPs explain a large proportion of the heritability for human height. *Nat Genet*. 2010;42(7):565-569.
3. Gudbjartsson DF, Walters GB, Thorleifsson G, et al. Many sequence variants affecting diversity of adult human height. *Nat Genet*. 2008;40(5):609-615.
4. Poveda A, Jelenkovic A, Salces I, Ibañez ME, Rebato E. Heritability variations of body linearity and obesity indicators during growth. *Homo*. 2012;63(4):301-310.

Comparative Effectiveness Research

Costs and Benefits of Male Circumcision

Kacker et al[1] present an economic model to justify Medicaid covering newborn male circumcision (MC) in the United States. However, their model uses highly flawed assumptions and projections. About 79% of their savings are MC-attributed reductions in human immunodeficiency virus (HIV) sexual transmission (only beginning about 12 years after enhanced MC coverage), but their base case lifetime cost of treating HIV/AIDS ($388 754) derives from estimates with highly active antiretroviral therapy (HAART) using brand name prices. In 12 years, most of these HAART medications will be generic.

Of particular importance, Truvada, recently Food and Drug Administration–approved for prophylaxis to prevent HIV sexual transmission in both adult and pediatric subjects 12 years or older,[2] will be available generically at pennies per day (by federal law with the lowest possible prices provided to Medicaid and other public programs). Truvada prophylaxis reduces HIV transmission by 40% to more than 80% (depending on medication adherence),[2] while Kacker et al claim that even a nationwide 69% reduction in MC would only increase US HIV prevalence by 12.2%. Moreover, Truvada and other HAART medications may have preventive benefit for all routes of HIV transmission, including intravenous drug use and maternal-infant transmission, while MC can only prevent some fraction of male penetrative sexually transmitted HIV. Although it is unclear how Kacker et al assessed it, human papillomavirus vaccines already exist and are currently recommended by the Centers for Disease Control and Prevention for boys and girls at age 12 years.[3] Herpes simplex and HIV vaccines and/or more effective treatments for all of the relevant infectious diseases may also be available in the next 12 years before any enhanced MC program could even begin to show benefit.[4,5]

Kacker et al cite the Centers for Disease Control and Prevention estimate of 19 million new sexually transmitted diseases per year, but their MC program would circumcise 1.38 million male newborns each year to prevent only 1.6% of these sexually transmitted diseases. Moreover, by their estimates, to prevent each case of HIV transmission, the program would circumcise 285 newborns.

In addition to the ethical concerns surrounding the imposition of a permanent and irreversible MC surgical procedure on male newborns who are incapable of granting informed consent, this antiquated technology is already dominated in terms of net economic benefits by existing preventive treatments (eg, human papillomavirus vaccine and Truvada) for most US population cohorts and is likely to be rendered totally obsolete for its purported sexually transmitted disease benefits early in the lifetimes of those newborns who will be involuntarily subjected to MC. Male circumcision is a poorly targeted and economically unjustified way to prevent infectious disease in the US population.

Joel W. Hay, PhD

Author Affiliations: Department of Clinical Pharmacy, Pharmaceutical Economics, and Policy and Leonard Schaeffer Center for Health Policy and Economics, University of Southern California, University Park Campus, Los Angeles.
Correspondence: Dr Hay, University of Southern California, University Park Campus, UGW–Unit A, Los Angeles, CA 90089-7273 (jhay@usc.edu).
Conflict of Interest Disclosures: None reported.

1. Kacker S, Frick KD, Gaydos CA, Tobian AA. Costs and effectiveness of neonatal male circumcision [published online August 20, 2012]. *Arch Pediatr Adolesc Med*. 2012;166(10):910-918.
2. Truvada FDA label. http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm?fuseaction=Search.Label_ApprovalHistory#labelinfo. Updated July 16, 2012. Accessed August 17, 2012.
3. HPV vaccine information for clinicians: fact sheet. Centers for Disease Control and Prevention website. http://www.cdc.gov/std/hpv/stdfact-hpv-vaccine-hcp.htm. Accessed August 17, 2012.
4. Whitley RJ, Roizman B. Herpes simplex viruses: is a vaccine tenable? *J Clin Invest*. 2002;110(2):145-151.
5. HIV Vaccine Trials Network website. http://www.hvtn.org/. Accessed August 17, 2012.

Comparative Effectiveness Research

Male Circumcision Cost-effective Articles Ignore Methodological Problems and Ethical Concerns

I read the article by Kacker et al[1] and the accompanying editorial[2] with dismay. The authors have attempted to apply the results of randomized controlled trials of voluntary adult male circumcision (MC) and human immunodeficiency virus, herpes simplex vi-