National Eating Disorder Information Centre

BULLETIN

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Improving Body Image and Preventing Eating Disorders: Should we be linking these quite so tightly?

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Like many eating disorder organizations, in addition to providing support to individuals living with or affected by eating disorders (EDs), the National Eating Disorder Information Centre (NEDIC) devotes resources and time to initiatives focusing on raising awareness of and preventing eating disorders, and promoting healthy body image and self-esteem. Also similar to many ED organizations, NEDIC has received criticism for including body image among its program priorities as opposed to focusing exclusively on EDs. Earlier this year, I published an academic paper directed at the controversy regarding the linking of body image intervention efforts and ED prevention. In this article, my goal is to summarize key points from that longer paper to encourage discussion among NEDIC's audience. Readers interested in the full discussion should see Becker (2016).

STARTING AT SQUARE ONE: CAN WE PREVENT EATING DISORDERS?

In order to answer this question, it is important to first explain what it means to "prevent" a disorder. In public health, prevention typically refers to preventing the onset of a disorder. Thus, if I say "we can prevent lung cancer", this is interpreted as meaning we have an intervention that will reduce the onset of new cases of lung cancer. Over time, this should reduce the number of people who develop lung cancer. Using this definition, the answer for EDs is "Yes – we can prevent some EDs". More specifically, we have several randomized clinical trials that have demonstrated that we can reduce the onset of some ED cases relative to a control condition that allowed cases to develop naturally (i.e., Stice et al., 2008; Taylor et al. 2006; Martinsen et al. 2014).

Now, this is great news, because prior to 2006 we did not have a scrap of scientific evidence to suggest that we could prevent any EDs. So, we have seen real progress! But before we celebrate too much, I must highlight some serious limitations in this area. First, all of the above mentioned trials targeted females who were in high school or college. In other words, we do not have a single trial that has prevented the development of early-onset anorexia nervosa, nor do we have evidence that we know how to do this at this point. Thus, I would argue that a program targeting elementary or middle school-aged children cannot be appropriately labelled an "ED prevention program" as we have no compelling evidence that we are capable of preventing a single ED in this age group at this time. In addition, to date only four programs have been shown to reduce the onset of any ED in high school and college-aged females, one of which is not generalizable beyond elite athletes. So, if you are not delivering the Body Project, the Healthy Weight Intervention or Student Bodies (Stice et al., 2008; Taylor et al. 2006), currently you have no evidence to suggest that you might actually prevent an ED.

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THEN WHAT ARE WE DOING IN ALL OF THOSE OTHER "EATING DISORDER PREVENTION" EFFORTS?

This is where things get both interesting and confusing, secondary to one of the primary factors that limits our ability to find true prevention effects – namely money. More specifically, it is very expensive to conduct research that can show an intervention has prevented the onset of a disorder. There are numerous reasons for this but at the top of the list is the amount of time it takes to show prevention effects. Using my lung cancer example, if I want to show that an intervention can prevent lung cancer, I will need to randomly assign a large number of people to either the intervention or a control condition. Why the large numbers? Because only a small number of people naturally develop lung cancer, and I need to show that significantly fewer people in the intervention group develop lung cancer compared to the control condition. So, I need to have enough people in the control condition to produce a meaningful number of lung cancer cases. If there are only 50 people, it is unlikely that anyone in the control condition will develop lung cancer. As such, I have little chance of demonstrating that the intervention can comparatively reduce cases of lung cancer. I also need to follow people long enough such that some might develop lung cancer. Needless to say, it is expensive to run trials involving large numbers of individuals who are followed for a long period of time. Funding agencies do not give researchers that much money unless they have preliminary evidence to suggest that their intervention might actually work. As a result, throughout public health, researchers focus on an intermediate step by trying to reduce risk factors.

Returning to our lung cancer example, as most people know, smoking is a significant risk factor for the development of lung cancer. Note that smoking does not predestine someone to lung cancer – plenty of smokers never develop the disease. Further, non-smokers also get lung cancer. But, if you smoke, your risk of developing lung cancer significantly increases. Thus, many lung cancer prevention endeavours target smoking – a causal risk factor for the development of lung cancer. A helpful aspect of focusing on risk factors is that it is much easier to demonstrate that an intervention has reduced the *rate of a behaviour* (e.g. smoking) in a population compared to the *onset of a disease* (e.g. lung cancer). As smoking is more common than lung cancer, it is easier to show a decrease in the intervention group compared to the control condition. And, it might take only take a few months to demonstrate a reduction in smoking, while it could take many years before an impact on the number of new lung cancer cases becomes apparent.

Let us now go back to the ED field. Body dissatisfaction is one of the most well established risk factors for the development of EDs (Jacobi & Fittig, 2010). However, just like smoking, not everyone with body dissatisfaction will develop an ED and not everyone with an ED starts with body dissatisfaction. Nonetheless, it is reasonable to attempt to reduce body dissatisfaction in the hope that it can prevent some EDs eventually. Indeed, research for all three of the prevention programs listed above started by investigating if the programs could reduce risk factors such as body dissatisfaction. Ultimately, this evidence was used to convince funding agencies to provide the resources for a true prevention trial.

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Although this is a reasonable line of research, it is inaccurate to assume that all programs that *aim* to reduce body dissatisfaction will in fact prevent a single ED. For instance, I might develop a new program aimed at reducing body dissatisfaction, but my program might be ineffective. As it turns out, it is a lot harder to reduce body dissatisfaction than many people think and many well-meaning endeavours likely have little impact. For instance, Stice et al. developed the cognitive dissonance-based *Body Project* because first generation ED prevention programs, which were largely didactic and psychoeducational in nature, were unsuccessful in reliably reducing ED risk factors, including body dissatisfaction (Mann et al., 1997; Stice et al., 2002; Stice & Shaw, 2004). Meta-analytic research also indicates that didactic psychoeducational programs rarely reduced ED risk factors (including body dissatisfaction) despite sometimes influencing knowledge

(see Stice & Shaw, 2004 for additional information). This finding is consistent with a broader literature in other areas showing little behavioural change following participation in psychoeducational programs despite changes in knowledge (Stice & Shaw, 2004). And yet, I still regularly encounter professionals at schools and in the ED field who report wanting or giving (sometimes on a fairly large scale) psychoeducational lectures on body image and EDs with the aim of improving body image and preventing EDs. Indeed, even third generation programs designed by experienced body image researchers sometimes fail to significantly improve body image in the target population at longer follow-up intervals despite producing other positive effects (e.g., Diedrichs et al., 2016). Thus, if I do not have good research showing my new approach actually reduces body dissatisfaction, I may just be wasting resources.

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In addition, even if my new program does in fact reduce body dissatisfaction, it may not reduce it sufficiently to actually impact the onset of EDs. Going back to smoking, it might be that smokers need to stop smoking altogether to reduce the onset of lung cancer whereas my program only decreases the amount they smoke. This is still good – less smoking will help participants in other ways, but it may be insufficient to stop the onset of lung cancer in those who are also biologically vulnerable. In summary, one cannot assume in *the absence of controlled research* that a body image program will in fact prevent any ED. This is one of the key criticisms that comes from some constituencies in our field (see Becker, 2016 for more discussion). Namely that we are seriously inaccurate when we broadly promote all body image intervention efforts, including those that are more advocacy based, as serious ED prevention interventions. It is particularly a problem for the vast majority of untested or poorly tested body image interventions and advocacy campaigns.

For this reason, I argue that we need to stop conflating all body image intervention efforts with ED prevention. Instead, the term ED prevention should optimally be reserved for programs that have been shown to reduce the onset of EDs in studies sufficiently well designed and funded to be able to demonstrate true prevention effects. All other studies and programs should more accurately state that the goal is to reduce an ED risk factor rather than ED onset. For instance, the *Healthy Weight Intervention* developed by Stice and colleagues has been shown to reduce the onset of new cases of EDs at three-year follow-up (2008); thus it can be described as an ED prevention program. In contrast, the *Female Athlete Body Project*, a derivative program designed to address some of the unique needs of female athletes (e.g., more complex body image concerns and the Female Athlete Triad) and be implemented by peer-leaders, has only been shown to reduce ED risk factors to date; thus it should be described as an ED risk factor reduction program (Becker et al., 2012). I would like to point out that there is trial is currently ongoing that is designed to determine if the *Female Athlete Body Project* does reduce ED onset at 18-month follow-up. If we get positive findings, then we would be able to start calling it an ED prevention program. In summary, we need to be more careful with our language and how we talk about what we do. Note that we are not the only area in public health to struggle with this. Physicians have noted that one cannot say that statins *prevent* myocardial infarction, stroke or death in women; evidence suggests that statins can lower risk factors for these conditions in women but there is not data to support the contention that they prevent those endpoint disorders (Mora, Glynn, Hsia, MacFadyen, Genest, Ridker, 2010; Rabin, 2014).

SO IS THIS THE ONLY CRITICISM/PROBLEM?

Critics of the linking of body image to ED prevention have also argued that this approach runs the risk of trivializing EDs, which are already widely misunderstood and believed by many to be problems that sufferers can solve simply by eating. As a body image researcher who believes that the literature clearly demonstrates a causal association between body dissatisfaction and the onset of *some* EDs (see Becker, 2016 for more detail), I am loath to call for an entire separation of the body image and ED fields. However, given that body dissatisfaction increases risk for a host of negative outcomes (Kilpela et al., 2015),

I think there is a compelling case to be made that body dissatisfaction deserves to be targeted as an end in and of itself. More specifically, people now realize there are many reasons to reduce smoking, and decreasing smoking is simply viewed as generally good for one's health. This is where I think we need to move with body dissatisfaction, particularly given that the vast majority of people who suffer from body dissatisfaction will never go on to develop an ED. Yet they will still suffer. The good news is that much of the world is already on board with this. For instance, in my experience disseminating the *Body Project* on university campuses, many say that their primary concern is *not* reducing EDs because the prevalence of EDs is too low to justify large scale programs (though they are obviously happy if they prevent some ED cases). Rather, the primary aim is to reduce body dissatisfaction, which they perceive as making many students miserable in a wide variety of ways. Further, tying body image programming to EDs anecdotally can reduce participation in body image interventions because people often do not perceive themselves at risk for an ED and are more willing to admit to being uncomfortable with their body. For this reason, I argue we should be very strategic in linking body image interventions to ED prevention and do so sparingly.

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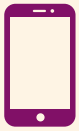
TO CONCLUDE

In summary, I propose that it would be beneficial for us to work towards the following steps. First, we should be more transparent and accurate by consistently labeling studies as "risk factor reduction" instead of "prevention" studies when they are only designed to detect reductions in risk factors. Second, we should increase precision when we talk about the association between body image and EDs by noting that body dissatisfaction is a risk factor for some, but not all, EDs. Third, we should more regularly connect body image to a range of negative outcomes beyond EDs to make the case that body image contributes to overall health and wellbeing. Finally, I recommend substantially reducing discussion of EDs when promoting programs that target body image. In my opinion, taking these steps would enhance discourse in our field, reduce unnecessary tension, and expand the community of people working towards reducing body dissatisfaction and preventing some EDs.



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REFERENCES

1. Basso, C., Perazzolo Marra, M., Rizzo, S., De Lazzari, M., Giorgi, B., Cipriani, A., … Illiceto, S. (2010). Arrhythmic mitral value prolapse and sudden cardiac death. *Circulation, 121*(9), 556-566. doi: 10.1161/CIRCULATIONAHA.109.906479
2. Becker, C. B. (2016). Our critics might have valid concerns: Reducing our propensity to conflate. *Eating Disorders, 24*(1), 79-89. doi: 10.1080/10640266.2015.1113833
3. Becker, C. B., McDaniel, L., Bull, S., Powell, M., & McIntyre, K. (2012). Can we reduce eating disorder risk factors in female college athletes? A randomized exploratory investigation of two peer-led interventions. *Body Image, 9,* 31-42.
4. Diedrichs, P. C.; Atkinson, M. J.; Garbett, K. M.; Williamson, H.; Halliwell, E.; Rumsey, N.; Leckie, G.; Sibley, . G.; Barlow, F. K. (2016). Randomized controlled trial of an online mother-daughter body image and well-being intervention. *Health Psychology, 35,* 996-1006.
5. Jacobi, C. & Fittig, E. (2010). Psychosocial risk factors for eating disorders. In: W. S. Agras (Ed.) Oxford Handbook of Eating Disorders (pp. 123-135). Oxford University Press: New York.
6. Jacobi, C., Hayward, C., de Zwann, M., Kraemer, H. C., & Agras, W. S. (2004). Coming to terms with risk factors for eating disorders: Application of risk terminology suggestions for general taxonomy. *Psychological Bulletin*, *130*(1), 19-65.
7. Kilpela, L. S., Becker, C. B., Wesley, N., & Stewart, T. (2015). Body image in adult women: Moving beyond the younger years. *Advances in Eating Disorders: Theory, Research and Practice, 3,* 144-164.
8. Mann, T., Nolen-Hoeksema, S., Huang, K., Burgard, D., Wright, A., & Hanson, K. (1997). Are two interventions worse than none? Joint primary and secondary prevention of eating disorders in college females. *Health Psychology, 16*(3), 215.
9. Martinsen, M., Bahr, R., Borrensen, R., Holme, I., Pensgaard, A. M., & Sundgot-Borgen, J. (2014). Preventing eating disorders among young elite athletes: A randomized controlled trial. *Medicine & Science in Sports & Exercise, 46*(3), 435-447.
10. Mora, S., Glynn, R. J., Hsia, J., MacFadyen, J. G., Genest, J., & Ridker, P. M. (2010). Statins for the primary prevention of cardiovascular events in women with elevated high-sensitivity c-reactive protein or dyslipidemia. Results from the justification for the use of statis in prevention: An intervention trial evaluating rosuvastitin (JUPITER) and meta-analysis of women from primary prevention tools. *Circulation, 121,* 1069-1077.
11. Stice, E., Marti, C. N., Spoor, S., Presnell, K., & Shaw, H. (2008). Dissonance and healthy weight eating disorder prevention programs: Long-term effects from a randomized efficacy trial. *Journal of Consulting and Clinical Psychology, 76*(2), 329-340.
12. Stice, E., Mazotti, L., Weibel, D., & Agras, W. S. (2000). Dissonance prevention program decreases thin-ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms: A preliminary experiment. *International Journal of Eating Disorders, 27*(2), 206-217.
13. Stice, E., & Shaw, H. (2004). Eating disorder prevention programs: a meta-analytic review. *Psychological bulletin, 130*(2), 206.
14. Taylor, C. B., Bryson, S., Luce, K. H., Cunning, D., Doyle, A. C., Abascal, L. B., Rockwell, R., Dev, P., Winzelberg, A. J., & Wilfley, D. E. (2006). Prevention of eating disorders in at- risk college-age women. *Archives of General Psychiatry, 63*(8), 881-888. doi:10.1001/archpsyc.63.8.881