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Fragranced Products and VOCs

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In the article "Scented Products Emit a Bouquet of VOCs," Potera (2011) gave a broad overview of the work of Steinemann et al. (2010) regarding the quantification of volatile organic compounds (VOCs) from fragranced products. Unfortunately, crucial facts were omitted about the materials cited and the use of alternative substances.

Potera (2011) quoted Steinemann et al. (2010), noting that some of the VOCs detected "are classified as toxic or hazardous by federal laws" and "a single fragrance in a product can . . . react with ozone in ambient air to form dangerous secondary pollutants." Potera stated that limonene reacts with ozone to form formaldehyde but failed to mention that both limonene and pinene are naturally occurring materials found in citrus fruits and pine trees, respectively (Wei and Shibamoto 2007). Fragrance materials are naturally volatile; otherwise, they would not be detectable (Cometto-Muñiz et al. 1998). Langer et al. (2008) showed that exposure to limonene from peeling an orange is far greater than using limonene-scented cleaning products. These authors further showed that secondary organic pollutants formed from cleaning products exist in the lowest range of exposure and that a higher concentration of particulates is formed by peeling an orange.

Potera (2011) quoted Steinemann et al. (2010), noting that "133 unique VOCs [were] identified among 25 products"; however, not all of the 133 VOCs are used as fragrance materials. For example, the highest reported concentration of *d*-limonene was 135 mg/m^3 (unidentified air freshener) in an experiment using conditions completely atypical of consumer use (Steinemann et al. 2010).

Although, the U.S. Environmental Protection Agency does not issue safe exposure limits, they report those from other agencies [National Institute for Occupational Safety and Health (NIOSH), Occupational Safety and Health Administration (OSHA), and American Conference of Governmental Industrial Hygienists (ACGIH)]. As of today, none of these agencies has issued a limit value for *d*-limonene. Germany (NIOSH 2005) and Sweden (International Agency for Research on Cancer 1999) have established limits for *d*-limonene of 110 mg/m^3 and 150 mg/m^3, respectively. Even under the adverse testing conditions reported by Steinemann et al. (2010), the *d*-limonene concentration of 135 mg/m^3 still falls within safe exposure.

Potera (2011) cited a telephone survey by Caress and Steinemann (2009) that attributed consumer health problems to the use of scented products; however, the percentages were not in context with the total population surveyed. Of those surveyed, 19% reported unspecified health problems and 11% noted irritation, all of which were subjectively ascribed to the use of scented laundry products (Caress and Steinemann 2009). While consumer complaints should be taken seriously, one may question the investigators' acceptance of these self-assessments in the absence of objective confirmation by medical testing.

Potera (2011) quoted Claudia Miller, who stated that "we need to find unscented alternatives ...." The fact is a variety of scented and unscented consumer products exist; thus, it is unnecessary to use potentially dangerous home mixtures, such as vinegar (acetic acid) and baking soda (sodium bicarbonate), which was recommended as a replacement for commercial cleaning products (Potera 2011). However, the safe exposure level for acetic acid, according to the ACGIH, NIOSH, and OSHA, is 25 mg/m^3 over 8 hr (OSHA 2007), which suggests a higher toxicity than for limonene. Health effects resulting from inhalation exposure to acetic acid include respiratory irritation, coughing, headache, and dizziness (Iowa State University 2000).

In addition, symptoms include pulmonary edema, chest pain, and hypotension; in contrast, *d*-limonene has not been associated with the development of any of these symptoms. Lacking published inhalation safety information for sodium bicarbonate, NIOSH recommends using a respirator when working with the dry particulate form (Mallinckrodt Baker Inc. 2009).

Potera (2011) ended her article by quoting Claudia Miller's statement that "the best smell is no smell." This is a very subjective assessment and cannot be characterized as an objective, science-based conclusion supported by available data.

All authors are employed by the Research Institute for Fragrance Materials, a nonprofit scientific organization that determines safe use levels for fragrances.

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Breast Cancer Environment Centers and Advocacy

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As Breast Cancer and Environment Research Center (BCERC) project leaders, we would like to address what we believe represents inaccuracies and omissions in the recent article by Baralt and McCormick (2010). Using self-citations, the authors asserted that genes and environment were not included in breast cancer research before advocacy efforts emerged. Yet the environment has long been implicated in breast cancer etiology; for example, for > 50 years the laboratory model of mammary carcinogenesis has involved administration of environmental chemicals (Medina 2007). Further, the Long Island Breast Cancer Study Project (LIBCSP) was not the first environment–breast cancer grant, as suggested by Baralt and McCormick. The National Institute of Environmental Health Sciences issued such grants as early as 1991, including "Environmental Factors and Breast Cancer in High-Risk Areas" [Request for Applications (RFA) CA/ES-93-024] in 1993.

The LIBCSP has been enormously productive, continuing even now, with > 100 scientific publications and $21 million in grant funding using LIBCSP resources.

Baralt and McCormick's (2010) criticism of the LIBCSP ignores the rigorous review process of National Institutes of Health grants, requiring an undeniable hypothesis, scientific plausibility, and high probability of success. What Baralt and McCormick described is the dissatisfaction of some (but not all) advocates with that research process during the initial years of the LIBCSP.

Baralt and McCormick (2010) used the word "frustration" 16 times, without noting the impressive contributions of the BCERC Community Outreach and Translation Cores (COTC) projects. Advocacy and COTC in the BCERC since 2003 have resulted in extensive and innovative dissemination of knowledge and new ideas (Breast Cancer and the Environment Research Program 2011). Mutual learning was facilitated by the participation of advocates and research staff in weekly staff meetings, monthly epidemiology and COTC calls, 16 subcommittee meetings and calls, and organizing calls for the biannual meetings. Coordinated COTC, advocate, and scientific sessions were part of the biannual BCERC meetings. Rather than "frustration," the past 7 years could be better summarized as an ongoing, interactive, collaborative, critical process of science and advocacy–indeed a new paradigm of scientific method.

As noted by Baralt and McCormick (2010), the 2002 RFA for BCERC did not require adherence to principles of community-based participatory research. The BCERC COTC members represented a range of experience in community-based participatory research; few had training in basic science. Each center developed different COTC models of community involvement and engagement, not included by Baralt and McCormick in their article. The Bay Area COTC incorporated the principles of community-based, participatory research and used those principles to evaluate the extent to which the approach was participatory and to ascertain the benefits and challenges of the participatory aspects of the project as perceived variously by community, advocacy, and scientific partners (Van Olphen et al. 2009). Other centers used quite different models of community engagement and, accordingly, should be evaluated in a different fashion. Thus, it would have been appropriate for Baralt and McCormick (2010) to assess which model most effectively met the aims stated in the 2002 RFA. Another difference between centers was that, except for the Bay Area, the COTCs were part of a research or academic institution. Thus, we faced multiple challenges on how to effectively involve communities and advocates in research. Over the first 7 years, centers developed a continuum of strategies to create partnerships with the basic scientists and epidemiologists involved in BCERC.

Baralt and McCormick (2010) omitted important details describing their methodology from the article. Specifically, in their Table 1 they included demographics about the sex and race/ethnicity of the investigators from BCERC centers, but no similar table characterized the participants in their study. [The Bay Area BCERC COTC included an African-American member, not four whites as Baralt and McCormick showed in their Table 1.] In addition, the authors did not discuss the involvement of advocates compared with nonadvocates in activities of COTCs at the four centers. It was unclear whether survey participants included only scientists and advocates formally connected with the centers (e.g., those listed in their Table 1) or if they included non-BCERC scientists and advocates who attended the conferences. Also, if the respondents in 2005 and 2007 were completely different, as suggested, it was not appropriate to pool the data nor to report any changes over time. We support advocate participation in research, and we recognize that methods for quantifying their contributions require unique approaches.

J.B. is employed by Zero Breast Cancer, a nonprofit organization. The authors declare that they have no actual or potential competing financial interests.

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Breast Cancer Environment Centers and Advocacy: Baralt and McCormick Respond

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Our findings regarding the Breast Cancer and Environment Research Centers (BCERCs) in which Wolff and Barlow are involved represent a broad overview of all four centers and are meant to portray several dimensions of the collaborative aspects of the work. In our article (Baralt and McCormick 2010), we aimed to advance the types of community-based participatory research projects exemplified by these centers. With this aim, we presented an analysis of the collaborative process to understand ways in which future funding can be better specified in the area of breast cancer and the environment. We sought to clarify how agencies can facilitate deepened participation in examining the potential underlying issues that may affect participatory research projects, particularly with regard to a lack of understanding of and training in community-based participatory research on the part of many scientists and advocates, as well as potentially divergent priorities or desired outcomes regarding the research.

The findings reported in our article (Baralt and McCormick 2010) show a need to further articulate participatory methods. We sought to make it clear throughout the article that our analysis provides a unique contribution to the dialogue about improving the collaborative process of participatory research projects. To this end, in a supplement to the article we provided recommendations that elaborated on the need for participants' commitment to a participatory research approach, participatory research training for advocates and scientists, clearly defined roles for advocates in research, clearly defined decision-making processes, and deliberation and agreement on the allocation of funds. These recommendations were based on our findings and what we heard from both advocates and scientists when we asked them about how the process could be improved upon in the future.

Our analysis (Baralt and McCormick 2010) does not represent our review of the scientific merits of the research being done in the centers or the entirety of environmental breast cancer research, which has been in existence for many decades. The environmental breast cancer research to date has been of critical importance to science, policy making, and advocates who have also played an important role in advancing environmental breast cancer research beginning in the 1990s. The rigorous National Institutes of Health review processes necessary for each center to be funded assured that the BCERCs are innovative and compelling.

Our research (Baralt and McCormick 2010) provided a useful overview perspective on the collaborative process within the BCERCs, highlighting the strengths of the Bay Area BCERC, with the goal of improving similar projects in the future. We did not conduct ethnographic research in each center, which would have demonstrated more about the specific nature of collaboration in each location, as we noted that Van Olphen et al. (2009) nicely did with the Bay Area BCERC COTC. Rather, we provided an overview of

the collaborative process (not the outcomes of the scientific research or COTC translation and dissemination activities) by assessing the perceptions of the collaboration by advocates and scientists who responded to our survey and were interviewed. This overview of the centers demonstrated, as noted by Wolff and Barlow, that the centers varied with regard to their experience with community-based participatory research. Wolff and Barlow note in their letter that the Bay Area COTC was the one center that incorporated the principles of community-based research and, based on our research, provided the best example of successful advocate–scientist collaboration among the BCERCs. Therefore, throughout our article (Baralt and McCormick 2010) we used the Bay Area BCERC (as well as in the Supplemental Material, in which we elaborated on a number of recommendations for future breast cancer–environment research collaborations) as a model for future collaborative projects. Additionally, we were careful in noting the limitations of our methods, acknowledging that our findings reflect only our sample of possible respondents and therefore may not be generalizable to all center advocates and scientists.

Our article (Baralt and McCormick 2010) and recommendations are both in the spirit of furthering the work of the BCERCs and projects like the BCERCs that engage in the "ongoing, interactive, collaborative, critical process of science and advocacy," as Wolff and Barlow describe their work with the BCERCs over the past 7 years. We encourage other researchers to continue investigating how environmental breast cancer research and other types of participatory projects can best serve the interests of science, advocates and policy-makers.

The authors declare they have no actual or potential competing financial interests.

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Redefining Low Lead Levels

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In the January 2011 issue of *EHP*, Giddabasappa et al. (2011) reported that gestational lead exposure (GLE) of C57BL/6 mice produced selective nonmonotonic increases in the numbers of rods and cone bipolar cells (BCs) in the adult retina. Interestingly, this increase was characterized by an inverted U-shaped dose–response curve. Moreover, findings of this study showed that GLE increases and prolongs proliferation of retinal progenitor cells (RPCs) without decreasing apoptosis. Consequently, this phenomenon produced an adult retina with normal lamination and a selectively increased number of rods and BCs. These results should be considered to define a more adequate risk assessment at low levels of lead exposure. In fact, other published articles have indicated that lead induced a biphasic dose–response relationship (Calabrese and Baldwin 2003).

In experiments in Swiss mice using low-level lead exposures similar to and lower than those used by Giddabasappa et al. (2011), we observed an increase in the number of red blood cells, in female gestational parameters, and in Th1 cytokine levels (Iavicoli et al. 2003, 2004, 2006a, 2006b). For this reason, it would be interesting if Giddabasappa et al. could verify this increase in the number of neurons in the rod-signaling pathway at even lower blood lead levels (< 10 μg/dL). The findings of our studies were also implicated over several generations.

In any case, we agree with Giddabasappa et al. (2011) that their findings, as ours, raise complex issue for toxicologists, pediatricians, public health regulators, and risk assessors who need to incorporate the occurrence of such U-shaped dose responses in the hazard and risk assessment process. In this context, these findings could be explained by the hormesis phenomenon, which is a dose–response relationship characterized by low-dose stimulation and high-dose inhibition (Calabrese 2008, 2009).

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Editor's note: In accordance with journal policy, Giddabasappa et al. were asked whether they wanted to respond to this letter, but they chose not to do so.