Minnesota Center for Environmental Advocacy Comments on the Minnesota Department of Agriculture Special Registration Review of Atrazine

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Executive summary

The Minnesota Center for Environmental Advocacy (MCEA) has prepared this submittal in response to the Minnesota Department of Agriculture (MDA) request for public comments on its January 2010 *Special Registration Review of Atrazine*. In that review, MDA along with the Minnesota Department of Health (MDH) and the Minnesota Pollution Control Agency (MPCA) have jointly declined to propose any changes to the regulation of atrazine. MCEA does not share the view that existing regulations applicable to atrazine are sufficiently protective of health and environment. Specifically, the drinking water limit for atrazine should be adjusted downward to ensure protection of public health.

To date, state and federal agency health risk assessments of this heavy-use herbicide have not accounted for the highly concerning pattern of serious, costly birth defects and other adverse developmental effects associated with atrazine in several epidemiological studies. These human epidemiological data should be integrated into any comprehensive human health risk assessment of atrazine. Inability to incorporate relevant epidemiological data into the risk assessment framework does not equate to a basis for reporting that current regulations are appropriately protective of public health and the environment.

MCEA also provides comment herein on the MDA economic analysis included in the atrazine registration review. While MCEA does not dispute MDA's right to raise the intriguing scenario of a prohibition on the use of atrazine, MCEA cautions against incomplete economic cost/benefit analysis of such a scenario, as it could yield erroneous conclusions. MCEA recommends instead that the agency conduct an economic analysis of a more discrete scenario, e.g., a cost/benefit breakdown of implementation of a more health-protective standard for atrazine in drinking water.

Atrazine human health risk assessment

In a 2010 multi-agency report, the Minnesota Department of Agriculture (MDA) along with the Minnesota Department of Health (MDH) and the Minnesota Pollution Control Agency (MPCA) have jointly declined to recommend any changes to the regulation of the heavy-use herbicide atrazine.[1] The Minnesota Center for Environmental Advocacy (MCEA) does not share the view that existing regulations applicable to atrazine are sufficiently protective of public health and the environment. Specifically, the drinking water limit (Maximum Contaminant Level or MCL; Health Risk Limit or HRL) of 3 parts-per-billion (ppb) of atrazine should be adjusted downward to ensure protection of public health. A drinking water limit of 1 ppb or lower for the combined concentration of atrazine and degradates is necessary to protect public health.

The MDH atrazine health risk assessment[2] relies mainly on laboratory toxicology study data and thus does not incorporate the worrisome pattern of developmental effects reported in several epidemiological studies published in recent years.[3-7] These epidemiological data should be integrated into any comprehensive public health risk assessment of atrazine. Inability to incorporate relevant epidemiological data into the risk assessment framework designed by MDH[8] does not equate to a basis for stating that current regulations are adequate.

Epidemiological studies of adverse developmental impacts associated with atrazine in drinking water[3-7] cumulatively represent a body of scientific evidence of harm. A dangerous pattern has emerged and it would be prudent to act immediately to protect public health. This is especially urgent because the reported health effects have been observed among particularly susceptible population sub-groups (infants, fetuses, pregnant women).

There is uncertainty as to the minimum concentration of atrazine in water that will predictably lead to specific adverse health effects in individuals, yet the consistency and seriousness of the reported human health effects warrants an enhanced health-protective regulatory stance. Due to the nature of scientific inquiry and ethical considerations in human epidemiological research, absolute certainty on these questions may not be attainable. We should not, however, rest in inaction simply because these risks cannot yet be precisely quantified. Failure to take precautionary action based upon what we already know about atrazine could place large numbers of people at risk for serious, costly health impacts.

Endocrine, reproductive, and developmental health risks of atrazine

Toxicological studies have repeatedly demonstrated that atrazine is an endocrine disruptor in mammals.[9-13] These hormonal perturbations cause a variety of reproductive and developmental impacts in mammals.[14] Mammalian toxicology studies are the established method for anticipating similar affects in humans. Studies of human cell lines lend further biological plausibility to the risk for significant health impacts in humans.[15] Endocrine and reproductive effects of atrazine have also been observed in both amphibians[16-18] and fish[18-20] which of course are less biologically similar to humans but the data provide additional support for the position that enhanced regulatory protections are warranted.

While toxicological data are a critical component of chemical risk assessment, data from epideimological studies should not be disregarded, particularly in cases where researchers report impacts that are strikingly similar to those reported in mammalian toxicology experiments. As far back as 1997, a peer-reviewed study had been published indicating that drinking water concentrations below the 3 ppb limit are associated with adverse birth outcomes in human populations.[3] More recently, other studies[4-7] have also reported associations between atrazine in surface water and adverse developmental effects. Taken as a whole, these epidemiological studies represent a very worrisome pattern suggesting that the drinking water limit of 3 ppb may not be sufficiently protective of public health.

Munger *et al*.[3] conducted a study among Iowa residents to assess the correlation between atrazine in drinking water and developmental effects. The principal endpoint of interest was intrauterine growth restriction (IUGR), a condition which elevates risk for neonatal health complications and fetal death.[21] Atrazine was the primary contaminant in the water supply serving the Iowa study communities. Atrazine was detected at a mean concentration of 2.2 µg/L, compared to 0.6 µg/L in other Iowa water supplies. The rate of births with IUGR was 80% higher in the study community: Relative risk = 1.8; 95% CI = 1.3 - 2.7. On their own, the data from this ecologic study could be considered a preliminary finding, yet if a statistically significant increase in adverse developmental effects is likely to occur at a population exposure level of 2.2 µg/L, a drinking water standard of 3 µg/L would not be adequately protective of public health, particularly for susceptible sub-populations such as infants and pregnant women.

More recently, Ochoa-Acuña *et al*.[4] studied the potential for atrazine in Indiana drinking water supplies to adversely impact fetal and neonatal development. The developmental indicator applied in this study was small for gestational age (SGA), a sign of impaired fetal or infant development closely related to IUGR.[21] The authors found that SGA was significantly

more prevalent among infants born to women in communities where the water supply contained > 0.644 μg/L atrazine (adjusted PR = 1.14; 95% CI, 1.03 - 1.24) compared to the control group where the mean atrazine concentration was < 0.179 μg/L. When the analysis was restricted to Ft. Wayne, IN, the largest municipality studied, a similar elevation in SGA prevalence was still found (adjusted PR = 1.11; 95% CI, 1.00 - 1.24) when the high exposure group (> 0.742 μg/L) was compared with the reference group (< 0.320 μg/L). Ochoa-Acuña *et al*. also evaluated atrazine drinking water levels just during the third trimester of pregnancy, a critical phase of fetal development, and found a 17-19% increase in the prevalence of SGA associated with concentrations > 0.1 μg/L. These results complement those reported by Munger *et al*.[3] and the atrazine concentrations linked to health effects are of a similar magnitude, indicating that a drinking water standard of 1.0 ppb or lower should be implemented to ensure that infants are protected from adverse developmental outcomes.

Villanueva *et al*.[22] conducted a study of atrazine in French water supplies, to determine if atrazine lingering in water resources after the herbicide was banned was associated with developmental effects. No significant increases in risk for SGA or other developmental impairments were found, except when the timing of the third trimester of pregnancy was considered. The lack of association with SGA does not necessarily contradict the results reported by Munger[3] or Ochoa-Acuña,[4] because atrazine concentrations in the French water supplies were very low relative to the U.S. studies—Even in the highest exposure group studied by Villanueva, the cutoff was 0.075 μg/L atrazine in raw water, and 0.036 μg/L in treated water.

Other epidemiological studies have investigated the potential for atrazine-contaminated water to increase risk for birth defects. Mattix *et al*.[5] found a significant correlation ($p = 0.0125$) between monthly concentrations of atrazine in Indiana surface waters and incidence of congenital abdominal wall defects. The specific effects studied were gastroschisis and omphalocele. It is worth considering the seriousness of these birth defects when deciding whether to enhance health-protective regulatory measures to prevent them. For infants born with a gastroschisis, the intestines protrude outside the body through a rupture in the abdominal wall.[21] An omphalocele is similar; the protruding intestines (and in some cases, other organs such as the liver and spleen) are covered with a membrane. Both of these birth defects typically require surgery, as they are serious and potentially life-threatening conditions. With a larger abdominal rupture, the infant will require general anesthesia before the surgery.[21]

Subsequent to the release of the MDA atrazine review,[1] results from a separate study of atrazine and abdominal wall defects were presented at the annual conference of the Society for Maternal-Fetal Medicine. Waller *et al*.[6] conducted a retrospective case-control study of the

relationship between agricultural chemicals in Washington surface waters and the occurrence of gastroschisis. They reported a significantly increased risk (OR = 1.6; 95% CI, 1.1 - 2.3) for gastroschisis among infants born to women residing in proximity to atrazine-contaminated waters.

These studies of atrazine and gastroschisis may be especially timely because the prevalence of this birth defect has increased precipitously in recent years.[23-25] As with many birth defects, the causes of gastroschisis are as yet unclear,[26] yet researchers believe that the trend data implicate environmental factors such as chemical teratogens rather than genetic factors or shifting diagnostic criteria.[23,27]

Gastroschisis and omphalocele are also very costly conditions, requiring major surgery followed by a lengthy hospital stay. The average hospital stay for infants with surgically repaired gastroschisis is 41.0 days; for omphalocele it is 32.5 days.[28] These are the two longest for any birth defects. The mean costs-per-case (hospital charges) are:

$155,629.00 per case for gastroschisis, and,

$141,724.00 per case for omphalocele.

The total U.S. costs of treatment for 2003 were estimated at:

$218,516,169.00 for gastroschisis, and,

$ 54,905,010.00 for omphalocele.

As these figures were calculated in 2003 dollars, one can reasonably assume that costs-per-case are higher in 2010 due to inflation. Similarly, the cumulative U.S. costs for treatment of these birth defects are also likely to be higher in 2010, not only due to inflation but also due to the reported increase in prevalence.

In a broader study of atrazine and birth defects, Winchester *et al.*[7] investigated the correlation between conceptions occurring in the spring months when pesticide water concentrations are highest and various types of birth defects. Nationwide elevations in the rates of several types of birth defects, including gastroschisis, were significantly associated with peak water levels of atrazine and other agricultural chemicals. The limitations of this study design are evident, yet when considered along with the more focused studies of adverse birth outcomes[3-6] it lends further support to the case for strengthening regulation of atrazine to protect public health.

In 2008, an expert committee of the National Research Council (NRC) warned: “Risk assessment…is at a crossroads, and its credibility is being challenged.”[29] To renew the credibility and usefulness of health risk assessment, regulatory agencies must incorporate all reliable scientific evidence of harm into promulgation of standards to assure protection of public

health and the environment, and not fail to act simply due to the existence of scientific uncertainty. In the case of atrazine, MCEA strongly urges state and federal government agencies to take preventive action to reduce the risk of adverse developmental effects and serious, costly birth defects that have repeatedly been shown to be associated with atrazine in water resources. With specific regard to the State of Minnesota multi-agency review of atrazine,[1] MCEA also recommends that MDH bear in mind its explicit statutory obligation to develop water quality standards that protect the health of susceptible sub-populations such as infants and children from chemical pollutants linked to adverse developmental, endocrine, and reproductive health impacts, among others.[30]

Carcinogenic potential of atrazine

Based on current science, the endocrine, reproductive, and developmental effects of atrazine appear to be the gravest threats to public health. It would be premature, however, to conclude that carcinogenicity is not a potential outcome as well. Although atrazine is not known to be mutagenic, studies have indicated that atrazine's demonstrated capacity to disrupt hormone system function can create conditions ripe for carcinogenesis.[31-33]

Fenton[32] described how exposure to atrazine and other endocrine-disrupting compounds during critical early periods of development can lead to cancer later in life. In that paper, Fenton identified a mechanistic pathway for breast cancer causation whereby atrazine's hormonal effects delay differentiation of terminal end buds, with the result that the mammary gland is "vulnerable to the effects of carcinogens for a significantly longer developmental period."[32] In other words, atrazine can increase cancer risk by influencing hormonal controls over the number and/or duration of structures that are susceptible to carcinogenesis, which elevates risk for cancer later in life. In an earlier publication,[31] Birnbaum and Fenton explained the significance of their laboratory data:

> Atrazine exposure *in utero* not only delays early mammary gland development in female offspring, but may also confer an extended window of sensistivity to potential carcinogens after sexual maturity.

Birnbaum and Fenton also raised questions on whether the appropriate studies on the carcinogenic potential of atrazine have even been conducted:

> All of these studies have demonstrated that prenatal exposure to endocrine-disrupting chemicals can alter the hormonal milieu, reproductive tissue development, and susceptibility to potential carcinogen exposure in the adult. These compounds are not genotoxic, yet can have significant adverse health

> outcomes. We must ask these questions: Are the appropriate, sensitive animal strains being used to test for endocrinologically based diseases, such as breast cancer? Are many of the adult rodents whose brain and endocrine function are fully developed relatively insensitive when exposed to endocrine-disrupting chemicals as adults? There have been epidemiologic studies investigating the association of environmental chemicals, including both organochlorines, such as PCBs and atrazine, with breast cancer incidence. These particular studies have measured the levels of exposure of these chemicals in adult women who develop breast cancer. Could we be trying to correlate exposure and effect at the wrong time? If it is prenatal or early life-stage exposure that is critical to disease susceptibility, why are we measuring environmental chemicals in people once they have developed breast cancer? The critical exposure window may have occurred much earlier.[31]

If these researchers are correct, and atrazine does act as a promoter, it would be a mistake for regulatory agencies to disregard atrazine's carcinogenic potential. In 2008, the NRC warned against precisely this approach, stating that one problem with the current risk assessment framework is that there is too often "undue focus on 'complete' carcinogens, ignoring contributions to ongoing carcinogenesis processes and the multifactorial nature of cancer."[29] The problem with this overly narrow focus, according to the NRC expert panel, is that:

> Chemicals that may increase human cancer risk by contributing to an underlying process are handled essentially as non-carcinogens even though they may be integral to the carcinogenic process.[29]

Since experimental data have shown that atrazine does have the potential to increase cancer risk by triggering such underlying processes, it is not realistic to conclude that atrazine is non-carcinogenic and exclude its carcinogenic potential from regulatory risk assessment.

Economic cost-benefit analysis

As part of its atrazine review, MDA conducted an analysis of the economic costs that could result if the registration of atrazine were cancelled.[34] MCEA recognizes the difficulties inherent to such an analysis, and would strongly caution against publishing an incomplete and potentially unbalanced cost/benefit breakdown. In an incomplete analysis, significant costs or benefits could be left out, yielding skewed or erroneous conclusions.

The MDA analysis primarily considers potential costs to corn producers if atrazine were no longer registered for use. The MDA approach does not fully account for costs of continued registration of atrazine. For example, a comprehensive cost/benefit analysis should encompass human health impacts associated with atrazine, such as costly birth defects.[5-7] As noted above, atrazine in surface water is associated with increased prevalence of gastroschisis, a birth defect

that costs the U.S. economy over $200,000,000.00 annually.[28] Similarly, the costs of ecological impacts appear to have been overlooked by MDA. Rohr *et al*.[18] published a meta-analysis and found consistent confirmation of atrazine's capacity to cause hormone disruption and related impacts in fish and amphibians. These authors warned that these impacts "must be weighed against any of [atrazine's] benefits and the costs and benefits of alternatives to atrazine use."[18]

Even on the more limited question of costs to corn producers, careless or incomplete citation from economic studies can be misleading. On page 4 of 8 of its assessment, MDA states: "Ackerman's combined assessment of Coursey, Fawcett, and EPA states that these economic studies roughly agree that the cost of banning atrazine is between $26 – 28 per acre."[34] This citation is incomplete and out of context, giving the reader the impression that Ackerman concluded that cancelling the registration of atrazine would be very costly, and, further, that all available major studies had concurred on this point and on the magnitude of the economic effects on corn production.

MDA neglects to mention that Ackerman also evaluated a fourth study of atrazine published by the U.S. Department of Agriculture (USDA), which reached significantly different conclusions. Ackerman believes the USDA study to be of superior quality to the other three, which Ackerman describes as "deficient":[35]

> EPA, Coursey, and Fawcett do not include the full range of economic impacts that were (appropriately) included in the USDA study—some of which represent increases in farm income, partially or wholly offsetting the losses.

MDA did not incorporate any of the USDA data into its analysis. Ackerman, however, did:

> Of the four studies discussed, USDA estimated the ban would reduce corn yields by about 1%, and would result in a slight gain to producers, but a larger loss to consumers due to higher corn prices.[35]

Ackerman's own conclusions are also quite different from what one might believe based on the MDA report. On the impacts of an atrazine ban, Ackerman writes:

> If…the yield impact is on the order of 1%, as USDA estimated, or close to zero, as suggested by the newer evidence discussed here, then the economic consequences become minimal.[35]

MCEA does not dispute MDA's right to raise the intriguing scenario of a prohibition on the use of atrazine, but an incomplete economic cost/benefit analysis of such a scenario could be misleading and yield erroneous conclusions. Unless MDA is seriously considering cancellation of the registration of atrazine, it would perhaps be of greater benefit for the agency

to conduct an economic analysis of a more limited and discrete scenario, e.g., a cost/benefit breakdown of a Minnesota standard of 1.0 or 0.5 ppb for atrazine in drinking water.

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