COMMONWEALTH OF MASSACHUSETTS
EXECUTIVE OFFICE OF ENVIRONMENTAL AFFAIRS
DIVISION OF ADMINISTRATIVE LAW APPEALS

IN THE MATTER OF
DEPARTMENT OF CONSERVATION AND RECREATION
Docket No. DEP-04-919
DEP FILE #233-547
NATICK

TESTIMONY OF HARLEE STRAUSS, Ph.D.

I, Harlee Strauss, Ph.D., on oath depose and say that I have personal knowledge of the facts stated herein.

1. I am President of H. Strauss Associates, Inc., a firm specializing in risk assessment and toxicology consulting, that I founded in 1988. I have been a consultant in the areas of human health risk assessment and toxicology since 1984. My clients include governmental agencies, private sector businesses, non-profit organizations, and attorneys.

2. I have been a resident of Natick MA since 1986 and an elected member of the Natick Town Meeting for the last several years. I have also been a member (Community) of the Restoration Advisory Board (RAB), Soldier Systems Center (Natick Labs), U.S. Army from 1999 to the current time. This facility is located adjacent to Lake Cochituate and the RAB has addressed multiple issues related to the Lake.

3. I received a Ph.D. in Molecular Biology from the University of Wisconsin-Madison in 1979, and an A.B. in Chemistry from Smith College in 1972. I was a post-doctoral fellow in the biology department at Massachusetts Institute of Technology from 1979-1981, and a Congressional Science Fellow (serving in the US House of Representatives) from 1981-1983.

4. I have been retained by the Petitioners in this matter to determine the risk to human health posed by the pesticides diquat dibromide ("Reward") and dipotassium endothall ("Aquathol K") as a result of the treatment of Lake Cochituate with those pesticides as proposed in the April, 2003, Notice of Intent Application, approved with conditions by the Town of Natick and the Massachusetts Department of Environmental Protection. I have undertaken this work on a *pro bono publico* basis. My opinions, as set forth herein, are stated to a reasonable degree of scientific certainty.

5. I have reviewed the following materials in preparing this affidavit:

- Notice of Intent Application, Aquatic Management Program, Lake Cochituate, MA, April 2003, Prepared by Aquatic Control Technology, Inc.

- Lake Cochituate Long Term Vegetation Management Plan, Draft; February 2004. Prepared by Aquatic Control Technology, Inc.

- Lake Cochituate Long Term Vegetation Management Plan. Prepared by Aquatic Control Technology, Inc. Final Draft May 2004.

- Letter to Rachel Freed, DEP from Peter Webber, DCR regarding the Lake Cochituate Field Investigation re: DEP file #233-547. Dated October 15, 2003

- Letter to Rachel Freed, DEP from Martin Levin, Stern Shapiro Weissberg & Garin. RE: Wetlands/Natick DEP file #233-547 Lake Cochituate, dated October 24, 2003.

- Letter to Mr. Martin Levin and Ann Karnofsky from James Sprague, DEP RE: Wetlands/Natick, with Superseding Order of Conditions. Dated March 9, 2004.

- Friesz, P.J. and Church. P.E. 2001. Pond-Aquifer Interaction at South Pond of Lake Cochituate, Natick MA.; USGS Water Resources Investgation Report 01-4040.\

Diquat references

- Syngenta Material Safety Data Sheet (MSDS) for Reward Landscape and Aquatic Herbicide (diquat dibromide).

- US EPA Consumer Factsheet on Diquat (www.epa.gov/safewater/dwh/c-soc/diquat.

- US EPA IRIS Profile Diquat (downloaded 1/12/2005).

- USEPA, 2001. IRIS Screening Level Literature for Diquat, Summary of Available Toxicity Information, Available from the EPA IRIS hotline.

- US EPA Pesticides: Reregistration. Diquat Dibromide TRED Facts. May 2002. www.epa.gov/oppsrrd1/REDs/factsheets/diquat_tred_fs.htm

- US EPA Memorandum from Becky Daiss OPPTS Health Effects Division to Tyler Lane and Betty Shackelford. OPPTS, Special Revew and Reregistration Division. March 6, 2002. Diquat dibromide HED Risk Assessment for Tolerance Reassessment Eligibilty Document (TRED).

- Extoxnet file for diquat dibromide (published 9/93). Pmep.cce.cornell.edu/profiles/extoxnet/dienochlor-glyphosate

- European Commission. April 5, 2000.. Opinion of the Scientific Committee on Plants Regarding Inclusion of Diquat in Annex 1 of directive 91/414/EEC Concerning the Placing of Plant Protection Products on the Market.

- European Commission. January 14, 2002. Opinion of the Scientific Committee on Plants on an Additional Question from the Commission Concerning the Evaluation of Diquat in the Context of Council Directive 91/414/EEC.

- Food and Agriculture Organization (FAO)/World Health Organization (WHO), Food additives. 1970s Evaluation of Some Pesticide Residues in Food – Diquat Monograph. www.inchem.org/documents/jmpr/jmpmono/v070pr11.htm.

- Greenlee, A.R. TM Ellis, and RL Berg. 2004. Low-Dose Agrochemicals and Lawn Care Pesticides Induce Developmental Toxicity in Murine Preimplantation Embryos. Environmental Health Perspectives 112:703-709.

- Gupta, S. RC Husser, RS Geske, SE Welty, and CV Smith. 2000. Sex Difference in Diquat Induced Hepatic Necrosis and DNA Fragmentation in Fischer 344 rats. Toxicological Sciences 54:203-211.

- Rogers, LK, S Gupta, SE Welty, TN Hansen, CV Smith. 2002. Nuclear and Nucleolar Glutathione Reductase, Peroxidase, and Transferase Activities in Livers of Male and Female Fischer 344 Rats. Toxicolgoical Sciences 69:279-285.

- Ran, Q. H. Liang, M. Gu, et al. 2004. Transgenic Mice Overexpressing Glutathione Peroxidase 4 Are Protected against Oxidative Stress-induced Apoptosis. J. Biol. Chem. 55137-55146.

Endothall references

- Cerexagri, Inc. Material Safety Data Sheet for Aquathol K Aquatic Herbicide (dipotassium endothall) dated 8 Jul 2002.

- US EPA Consumer Factsheet on endothall (www.epa.gov/cgi-bin/epaprintonly.cgi

- US EPA. Integrated Risk Information System profile for Endothall, downloaded on 3/31/05.

- USEPA, 2001. IRIS Screening Level Literature for Endothall, Summary of Available Toxicity Information, Available from the EPA IRIS hotline.

- Office of Environmental Health Hazard Assessment (OEHHA), California EPA. December 1997. Public Health Goal for Endothall in Drinking Water. http://www.oehha.ca.gov/water/phg/pdf/endo_c.pdf

Background

6. In April 2003, the Massachusetts Department of Conservation and Recreation (DCR) filed a notice of intent (NOI) application to apply two herbicides, Reward and Aquathol K, to control milfoil growth along the shoreline and coves of the South Pond of Lake Cochituate, and near the State Park swim beach on the Middle Pond of the Lake. Both locations are in the Town of Natick, MA. The NOI application states that the herbicide use is for short term control, with treatment proposed for two consecutive years with the possibility that herbicide use would also be a component of its long term control strategy. The selection of aquatic herbicides was limited because Natick water supply wells are located adjacent to the Lake and Lake Cochituate is designated as a Zone II wellhead protection area. As an added safeguard, the NOI states that no treatment will occur within 200 feet of the public wells. The Order of Conditions issued by the Natick Conservation Commission on June 5, 2003 required a 1000 foot setback.

7. The herbicide Reward is composed of 37.3% diquat dibromide and 62.7% unnamed "inert" chemical compounds (Syngenta MSDS for Reward). According to the NOI, Reward is to be applied at 1-1.5 pounds diquat cation per acre, at the low end of the 1-2 pounds diquat cation per acre maximum printed on the MSDS. This application rate is anticipated to result in surface water concentrations of diquat cation of 0.19-0.30 parts per million (ppm) immediately after application. The NOI suggests that the diquat concentration in the water will decrease over the course of several days as the diquat binds to sediments. It is anticipated that milfoil control would last for only the year of application and that repeated yearly treatments would be required (ACT 2004).

8. The herbicide Aquathol K is composed of 40.3% endothall-potassium and 59.7% unnamed other compounds (Cerexagri MSDS). According to the NOI, "Aquathol K would be applied to the area for control of nuisance vegetation at the application rate of

2-3 parts per million." Presumably, the 2-3 ppm is the intended water concentration of endothall immediately after application of some number of gallons of Aquathol K per acre treated. The number of gallons is dependent upon the depth treated, which is not specified in the NOI. Endothall is currently proposed for use only in Middle Pond of Lake Cochituate, not in South Pond.

9. The Final Draft Aquatic Vegetation Management Plan (ACT, May 2004) also recommends that South Pond be treated with Fluridone (Sonar) in the first year of the program. However, as this recommendation was not included in the NOI that is the subject of this proceeding, I do not address the toxicological issues that may be raised by Fluridone treatment of the Lake. The May 2004 proposal also allows for spot treatment with Reward in the South Pond, and use of Reward and Aquathol K in Middle Pond.

The use of the herbicides diquat, and endothall[1] in the South Pond of Lake Cochituate may result in chronic exposure to children and adults who reside in Natick because of the potential of these herbicides to enter into the town water supply.

10. The Natick Springvale public water supply wellfield is located south of Route 9, approximately 200 feet from the South Pond of Lake Cochituate. According to a recent US Geological Services Study (Friesz and Church 2001), "water levels from the pond and underlying sediments indicate a downward vertical gradient and the potential for infiltration of pond water near the wellfield." Moreover, it was estimated that 64 +/- 15 percent of the water withdrawn from the public water supply wells was derived from pond water. "The rate of infiltration of pond water into the aquifer and discharging to the wellfield was 1.0 million gallons per day at the average pumping rate (Friesz and Church 2001)."

11. The flow of water from the pond to the groundwater is indicated by the by piezometer studies showing a downward vertical gradient of the water (Friesz and Church 2001). The sediments along the shoreline of South Pond are primarily coarse grained, and it is likely that pond water infiltrates into the aquifer predominantly in the shoreline regions (Friesz and Church 2001). Friesz and Church (2001) estimate the time of travel from the pond to the water supply wells ranges from days to more than one year because the wells draw water from multiple locations around the pond.

12. Diquat binds strongly to clay particles because of its cationic properties (it has a charge of 2+), but is not as strongly bound to organic matter. Organic matter predominates the shore areas of Lake Cochituate, and it is not known how much of the diquat will remain soluble in the water, and thus enter into Natick's water supply wells. In addition small particles, such as those in clay may be entrained in the pore water during its migration to

[1] Endothall has not been proposed for use in the South Pond of Lake Cochituate.

Natick's water supply wells and diquat may enter the drinking water supply bound to small particles.

13. Repeated applications of diquat, as suggested in the NOI, may lead to a buildup of diquat in the sediments of the lake because of its slow degradation rate once associated with sediments.

14. Endothall is soluble in water and can enter the groundwater leading to the Springvale water supply wells along the shores of the lake. The transfer from pond water to groundwater will begin immediately in the coarse sediments along the shoreline. Although endothall is degraded in surface waters over the course of a few days or weeks because of the presence of oxygen and/or light, it is not rapidly degraded in groundwater (OEHHA, 1997; Cornell University 1986).

The use of the herbicides diquat and endothall in Lake Cochituate may result in exposure to adults and children who swim or waterski in the Lake.

15. Adults and children swimming or waterskiing in Lake Cochituate soon after the application of the herbicides may come into contact with diquat and endothall dissolved in the water. The contact may result in internal doses of the herbicide either through dermal contact or inadvertent ingestion of the water.

Recent data indicate that diquat is toxic to humans at lower doses than observed in the studies relied upon by EPA and other regulatory agencies.

16. EPA and other regulatory agencies use a value known as a reference dose (RfD) to quantify the (noncancer) toxicity of a substance. EPA (IRIS database) defines an oral RfD to be "an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime."

17. EPA has published an oral RfD for diquat of 0.0022 mg/kg-d. The RfD is based on a study conducted in 1985 by Chevron Chemical Company in which 50 male and 50 female Sprague-Dawley rats were fed diets containing various doses of diquat for two years. The principal toxic effects were lens opacity/cataracts in eyes of males and females and possible kidney effects in males. To obtain the RfD, the lowest dose at which no effect was observed in this study (0.22 mg/kg-d, an average of the male and female doses) was divided by an "uncertainty factor" of 100. The use of the uncertainty factor is intended to account for interspecies (rats vs humans) and intraspecies (the range of sensitivities of humans) differences.

18. The last significant revision of this RfD was dated July 22, 1986. EPA conducted a screening level literature review under the IRIS program (the EPA program that develops

and publishes RfDs) from 1994 to May 1, 2001. EPA concluded that "The literature published since the RfD for diquat was derived (1986) may include study data that could potentially produce a change in the RfD." (USEPA, 2001)

19. Subsequent to the publication of EPA's RfD evaluation and screening level literature review, further scientific studies have been published in peer reviewed literature related to: 1) additional toxic effects that occur at low doses, 2) strain and gender differences in diquat effects, and 3) mechanisms of toxicity. As highlighted below, these additional studies suggest that the current RfD for diquat may not be adequately protective against adverse reproductive effects and perhaps other toxic effects.

20. **Low dose reproductive and developmental effects**. Diquat was among 13 agrochemicals tested for preimplantation toxicity in a toxicity testing system known as the mouse embryo assay (Greenlee et al 2004). This assay is being developed to supplement animal testing protocols for developmental and reproductive toxicants. The concentrations tested in by Greenlee et al. (2004) are equivalent to the RfD, the dose anticipated to be without toxic effects. The results of the test showed that incubating mouse embryos with 0.0022 mg/L diquat led to a statistically significant increase in programmed cell death (apoptosis). As pointed out by Greenlee et al (2004): "Embryos cleaving to blastocyst yet undergoing cellular death at a higher rate could result in embryos composed of fewer cells. Unless repair mechanisms overcome cellular loss, exposures during this period could result in embryonic demise, implantation failures or alterations in physiologic processes underlying maternal recognition of pregnancy. Findings from animal dosing studies are consistent with these possibilities." In other words, the results of this test suggest that low doses of diquat could interfere with the establishment of a pregnancy.

21. **Strain and gender differences**. Diquat is used as a test material to understand the mechanism of toxic injury due to reactive oxygen species (ROS) in cellular systems (Gupta et al. 2000; Rogers et al. 2002). Reactive oxygen species damage DNA, proteins, and cell membranes, thus harming cells and tissues. Some ROS form naturally in cells, and cells have developed methods to reduce this form of toxicity, known as oxidative damage. Some methods include "disarming" the ROS by enzymes such as glutathione peroxidase. Anti-oxidants such as Vitamins C and E also reduce oxidative damage. Oxidative damage accumulates in human tissues with age and contributes to a number of diseases, including heart disease and cancer.

22. Administration of diquat to two strains of rats, Fischer 344 and Sprague-Dawley rats, markedly increases ROS in their livers, which can interact with DNA, proteins and lipids to damage the liver cells. However, diquat results in acute liver damage in one strain of test rats, Fischer 344 rat,s while another strain, Sprague-Dawley rats, are more resistant to this damage. In addition, in both strains, males appear to be more sensitive to the effects than females. The Chevron Chemical Company study that forms the basis of the RfD

published by EPA utilized Sprague-Dawley rats, the strain that is more resistant to the effects of diquat. The use of a study based on Fischer rats likely would have resulted in effects at lower doses and thus a lower RfD. In addition, the RfD was based on averaging the doses provided to male and female rats although the data indicate that male rats are more sensitive to the effects of diquat, consistent with the subsequent data (Gupta et al. 2000; Rogers et al. 2002). The use of the data from only the male rats would have resulted in a somewhat lower RfD.

23. **Mechanisms of toxicity.** The extrapolation/applicability of the toxicity results from rodent studies to humans will ultimately be based on an understanding of the mechanism(s) of toxicity. There is general scientific acceptance that reactive oxygen species contribute to cell and tissue damage, but the molecular mechanisms responsible for the initiation and progression of oxidant-mediated cell damage remain poorly understood (Rogers et al. 2002). Thus, it is not clear whether or not the 10 fold uncertainty factor to extrapolate between the rat and human studies that is incorporated into the RfD is appropriate, inadequate, or overprotective. In addition, it appears that glutathione S-transferase (GST), glutathione reductase, and glutathione peroxidase contribute to anti-oxidant defense systems (Rogers et al. 2002). GST in particular is important in the detoxification of many environmental chemicals and thus co-exposure of diquat with other environmental toxicants could produce additive or even more than additive effects. These uncertainties contribute to the imprudence of intentionally introducing a chemical into Lake Cochituate where it will become associated with the sediments and potentially enter the public water supply.

The data used to derived reference doses do not include an evaluation of the effect of the chemical formulation on toxicity or of the interaction with other toxic chemicals.

24. The references doses for diquat and endothall are based on pure (or technical grade) chemicals and not the pesticide mixture as formulated (EPA IRIS profiles). According to the manufacturers' MSDSs, the commercial products contain approximately 60% "inert" ingredients, meaning they don't have herbicidal properties. However, it does not mean they are nontoxic. In addition, inert ingredients can change some of the characteristics that lead to the toxicity of a substance, such as how much is absorbed through the skin, or how long a substance remains soluble in water. These changes can lead to enhanced toxicity. There are few data regarding the toxicity of the formulated compounds, and the combination of effects. This lack of data on "real life" conditions results in uncertainty regarding the health effects that may occur under conditions of use.

Based on the foregoing evidence, it is my professional opinion that:

The uncertainties associated with the human toxicity of diquat, even at the low concentrations that would occur in Lake Cochituate after application and its potential intrusion into the Natick

public drinking water supply, render it imprudent to use this herbicide in the South Pond of Lake Cochituate.

Signed under the pains and penalties of perjury this 30th day of April 2005.

Harlee S. Strauss, Ph.D.
President
H. Strauss Associates, Inc.