

**SOAH DOCKET NO. 582-08-0202
TCEQ DOCKET NO. 2007-1426-MWD**

**IN THE MATTER OF THE
APPLICATION FOR PERMIT
NO. WQ0014293001 OF HAYS
COUNTY WATER CONTROL
AND IMPROVEMENT
DISTRICT NO. 1**

§
§
§
§
§
§
§
§

**BEFORE THE STATE OFFICE
OF
ADMINISTRATIVE HEARINGS**

**DIRECT TESTIMONY OF
DAVID CREWS, Ph.D.**

1 Q: IDENTIFY YOURSELF AND STATE THE PROFESSIONAL CAPACITY IN WHICH
2 YOU ARE APPEARING IN THIS PROCEEDING.

3 A: My name is Dr. David Crews and I am the Director of the Center for Behavioral
4 Neuroendocrinology and an Ashbel Smith Professor of Zoology and Psychology at the
5 University of Texas. I am appearing on behalf of Hays County.

6 Q: PLEASE DESCRIBE YOUR ACADEMIC BACKGROUND.

7 A: I received a B.A. from the University of Maryland in 1969 and, then, in 1973, I received
8 a Ph.D. from the Institute of Animal Behavior at Rutgers University, where I was a
9 NIMH Predoctoral Trainee in Psychobiology.

10 Q: HAVE YOU HAD OTHER FORMAL EDUCATION THAT BEARS ON YOUR
11 QUALIFICATIONS?

12 A: I was a Trainee of the Summer Training Institute in Behavioral Genetics at the Institute
13 for Behavioral Genetics, University of Colorado, Boulder in the summer of 1975 and also
14 of the Summer Course in Embryology, Marine Biological Laboratories, Woods Hole,
15 Massachusetts in the summer of 1980.

1 Q: PLEASE DESCRIBE YOUR EMPLOYMENT BACKGROUND.

2 A: Since obtaining my Ph.D. from Rutgers University in 1973, I have held various research
3 and faculty positions. Before coming to UT, I was a postdoctoral fellow at the University
4 of California at Berkeley and Assistant and Associate Professor at Harvard University. I
5 have been a professor of Zoology and Psychology at the University of Texas since 1984,
6 becoming an Ashbel Smith Professor of Zoology and Psychology in 1998. Additionally,
7 I have acted as the Director of the Center for Behavioral Neuroendocrinology (nee
8 Institute of Reproductive Biology) at the University of Texas since 2001.

9 Q: WHAT IS THE CENTER FOR BEHAVIORAL NEUROENDOCRINOLOGY?

10 A: This organization is an Organized Research Unit of the University of Texas that has been
11 in existence at the University of Texas for the past 3 decades. It was originally called the
12 Institute of Reproductive Biology. It was renamed the Center for Behavioral
13 Neuroendocrinology in early 2000 to reflect the change in participating faculty. At that
14 time, I took the position of Director from Professor Frank Bronson, who had been the
15 Director of the Institute for many years. Historically, the goal of the Institute/Center has
16 been to provide primary training in the area of reproductive biology, in particular,
17 behavioral neuroendocrinology. The research conducted by its participants is focused on
18 developing principles that apply broadly to the relationship between behavior and the
19 neuroendocrine system in animals, including, in some cases, in humans. The relationship
20 between neuroendocrines and behavior is of fundamental importance in understanding
21 how animals relate to their environments. Our intent is to use a wide variety of animal
22 models to explore the relationship between neuroendocrine activity and behavior at all

1 levels of organization – from the purely molecular to that of an evolving population. The
2 relationship between behavior and neuroendocrine activity is two-directional and our
3 interests span both directions; behavior can be controlled by neuroendocrines and
4 neuroendocrine activity can be regulated by behavior. We expect to discover
5 fundamentally important principles, many of which will be generalizable to humans or to
6 the environmental interests of humans.

7 Q: CAN YOU TELL THE JUDGE BRIEFLY OF ANY ORGANIZATIONS TO WHICH
8 YOU BELONG AND ANY RESEARCH IN WHICH YOU HAVE PARTICIPATED
9 THAT TEND TO ESTABLISH YOUR CREDENTIALS TO HELP THE JUDGE
10 EVALUATE THE POTENTIAL, IF ANY, FOR ENDOCRINE DISRUPTION IN
11 AQUATIC SPECIES?

12 A: My students and I have conducted original research in the area of endocrine disruption
13 using the red-eared slider turtle as a model system. This work has included over 60
14 refereed papers that have been highly cited (referenced) by researchers in this area and
15 have contributed our understanding of endocrine disrupting chemicals as environmental
16 toxicants and how they should be assessed. I have served on several expert review
17 panels, including the Environmental Agency, Government of Japan (2000), Workshop to
18 Examine the Effects of Endocrine Disruptors on Child Development for a National
19 Longitudinal Study (2000), and, most recently, as part of a panel to evaluate EPA's
20 Endocrine Disruptor Screening Program (EDSP) Tier 1 Screen "Amphibian
21 Metamorphosis Assay."

22 Q: HOW ABOUT PEER HONORS? HAVE YOU BEEN RECOGNIZED BY YOUR
23 PEERS IN ANY SIGNIFICANT WAYS THAT BEAR ON YOUR PROFESSIONAL
24 COMPETENCE?

25 A: I have been privileged to receive a few honors over time, including a Research Scientist
26 Award (1977-1997) and a MERIT Award from the National Institutes of Health. An

1 earlier paper on the phenomenon of temperature-dependent sex determination in red-
2 eared slider turtle as selected as an "Example Of Excellence in the Field of
3 Environmental Contamination and Toxicology", *Quintessence, Journal of Excellence in*
4 *Environmental Contamination and Toxicology 1995, Volume 1, Number 3*. My most
5 recent paper on endocrine disruption was #22 of the "100 Top Science Stories of 2007."
6 *Discover: Science, Technology, and The Future* (January 2008: 40). I am also an elected
7 fellow of the American Academy of Arts and Sciences and several other scientific
8 societies.

9 Q: HAVE YOU PREPARED A RESUME OF YOUR PROFESSIONAL
10 QUALIFICATIONS?

11 A: Yes, it is attached as Exh. HC-5.

12 Q: WHAT IS THE PURPOSE OF YOUR TESTIMONY IN THIS PROCEEDING?

13 A: Hays County requested that I address whether, if there were endocrine disruptors in Hays
14 County WCID No. 1's effluent discharge, the endocrine disruptors would be expected to
15 cause more than a *de minimis* degradation of water quality in Bear Creek north of FM
16 1826. I also explain various matters related to endocrine disruptors and their impacts.

17 Q: CAN YOU EXPLAIN BRIEFLY FOR THE JUDGE WHAT AN ENDOCRINE
18 DISRUPTOR IS?

19 A: A definition that I feel is accurate is that used by the Environmental Protection Agency,
20 namely: "An endocrine disruptor is an exogenous agent that interferes with the synthesis,
21 secretion, transport, binding, action, or elimination of natural hormones in the body that
22 are responsible for the maintenance of homeostasis, reproduction, development, and/or
23 behavior." An "exogenous" agent is simply an agent originating outside the body.
24 "Homeostasis" is a state of equilibrium among interacting agents, compounds or the like.

1 The terms “estrogen mimics”, “environmental estrogens”, and “xenoestrogens” –
2 terms one often encounters in this field – actually refer to a subset of a wide variety of
3 natural and synthetic compounds that exert actions upon hormonally sensitive pathways,
4 resulting in endocrine and/or reproductive dysfunctions; these are known collectively as
5 “endocrine-disrupting chemicals (EDCs)”. There are numerous industrial chemicals,
6 pesticides, fungicides, plasticizers, pharmaceuticals, phytoestrogens, and other
7 compounds that may cause endocrine disruption. Although the mechanisms of their
8 actions vary, they may include any or all of the following: nuclear hormone receptors,
9 membrane hormone receptors, enzymes involved in the biosynthesis/metabolism
10 /degradation of hormones, co-regulatory factors, and through neurotransmitter systems in
11 the brain that control neuroendocrine functions, among others (such examples are
12 reviewed in Gore, 2007 and Gore et al., 2006).¹

13 An example of a prototypical EDC is provided by polychlorinated biphenyls
14 (PCBs), a family of highly stable compounds that were used in industry for decades until
15 they were banned in 1977 in the United States. Ironically, it is the very structural
16 properties of PCBs and similar compounds that made them useful in industry that cause
17 them to be EDCs. Their lipophilic, biphenolic structures enable PCBs to interact with
18 nuclear hormone receptors, including the estrogen receptor (ER), thyroid receptor,
19 androgen receptor, and the aryl hydrocarbon receptor. PCBs also exert actions on other
20 steroid-regulatory or sensitive pathways and, of my own particular expertise, sex
21 determination, gonadal differentiation, and neuroendocrine systems that mediate

¹ Gore, A. C., 2007 *Endocrine-disrupting chemicals: From basic research to clinical practice*. Humana Press, Totowa, NJ; Gore, A. C., Heindel J J, Zoeller R T 2006 Endocrine disruption for endocrinologists (and others). *Endocrinology* 147, S1-3.

1 reproductive physiology and behavior. For example, PCBs can disrupt specific aspects of
2 reproductive physiological and behavioral functions controlled by the hypothalamus-
3 preoptic area.

4 Q: CAN YOU TELL THE JUDGE SOMETHING OF YOUR BACKGROUND IN THE
5 STUDY OF ENDOCRINE DISRUPTORS?

6 A: I have worked in the area of endocrine disruption for about 15 years. This work began in
7 collaboration with Dr. John McLachlan, then at the National Institute of Environmental
8 Health Sciences. I had been working for several years on the process of sex
9 determination in the red-eared slider turtles, demonstrating that this normally involves
10 estrogens, and I became interested in the newly developing field of how a variety of
11 natural and man-made chemicals can mimic or antagonize the actions of natural
12 endogenous steroid hormones, resulting in impaired reproductive performance or even
13 sterility in adulthood. This animal subsequently proved to be a very sensitive bioassay
14 for environmental quality, and we have developed this animal model system as a
15 biomarker of potential contamination by environmental estrogens. Our publication
16 (190)² establishing that polychlorinated biphenyls (PCBs) act at minute concentration
17 levels and, in mixtures, can synergize to exert even greater effects than when acting in
18 isolation (that is, mixtures of compounds in ecologically relevant concentrations have
19 different effects on organisms than do single compounds in ecologically-relevant
20 concentrations) is considered a landmark study in the field. See, also, my publication
21 with Dr. Willingham (265), attached as Exh. HC-6. Another study, with Dr. Daniel
22 Sheehan, was the first study to empirically test the No Adverse Effect Level (NOAEL)

² The parenthetical references are to the numbers associated with research papers listed on my vitae.

1 concept, a cornerstone in risk assessment in environmental toxicology. We demonstrated
2 in a power analysis, hypothesis-driven experiment using 2400 individuals that the
3 concept of a threshold dosage does not apply for estrogen-mediated endocrine disruptors
4 (243). Finally, I have examined endocrine disrupting compounds as modifiers of the
5 epigenome of animals, documenting transgenerational epigenetic effects of the common
6 fungicide Vinclozolin on mate preference (324). This last paper has received a fair
7 amount of acclaim (e.g., it was rated among the top 100 science papers of 2007), and its
8 insights to transgenerational impacts offer a sound basis for caution, so it, too, is attached,
9 here, as Exh. HC-7.

10 Q: DO I CORRECTLY UNDERSTAND YOU TO BE SAYING THAT THE LAST TWO
11 PAPERS REFERENCED DEMONSTRATED, RESPECTIVELY, THAT (1) THERE IS
12 NO CONCENTRATION LEVEL FOR ESTROGEN-LIKE ENDOCRINE
13 DISRUPTORS BELOW WHICH THERE ARE NO ADVERSE EFFECTS ON
14 EXPOSED INDIVIDUALS AND (2) THAT AT LEAST SOME ENDOCRINE
15 DISRUPTORS HAVE ADVERSE EFFECTS IN THE FUTURE GENERATIONS OF
16 INDIVIDUALS THAT ARE EXPOSED?

17 A: Yes, that is generally correct.

18 Q: YOU HAD PROBABLY BETTER EXPLAIN FOR THE JUDGE AND AT LEAST
19 SOME OF THE COUNSEL WHAT AN “ESTROGEN-MEDIATED” ENDOCRINE
20 DISRUPTOR IS.

21 A: Estrogen-mediated biological processes involve in some manner the stimulation and/or
22 inhibition of the estrogen receptor(s). These events are fundamental to normal biological
23 function and occur at all life stages. For example, during early development, this process
24 is important in the formation of both types of gonads (ovaries and testes). During early
25 life these estrogen-mediated processes are also important in the differentiation of the
26 brain so as adults behavioral and physiological processes essential to reproduction are
27 supported. Estrogen-mediated responses are important in puberty, and are essential to

1 normal menstrual cycling and its control. Finally, in old age and senescence, they have
2 been demonstrated to play a role of cognitive processes.

3 Q: PLEASE EXPLAIN FOR HIM HOW ENDOCRINE DISRUPTORS INTERACT WITH
4 IN-STREAM ORGANISMS.

5 A: Endocrine-disrupting chemicals, i.e., “EDCs,” interact with in-stream organisms to in
6 some way affect their normal development or composition.

7 Q: WHAT DIFFERENT KINDS OF EFFECTS MAY EDCs HAVE ON ORGANISMS?

8 A: It is first important to distinguish between the various categories of effects of EDCs. The
9 first, and most obvious, are those gross effects on development, whether on specific
10 structures or on systems. Examples would include the development of split and/or
11 duplicated limbs in frogs, stunted growth resulting in smaller individuals, abnormal or
12 erratic escape behaviors from predators or hostile conditions, etc. Such individuals are
13 more likely to die early in life than are those individuals that are not affected.

14 A second category consists of effects on animals that may appear outwardly to be
15 normal but are grossly abnormal internally, either structurally or in physiology.
16 Examples of this are hermaphroditic fish with ovotestes and/or abnormal accessory sex
17 structure development, delayed or absent puberty, high vitellogenin levels, etc. (In most
18 non-mammals, vitellogenin is a liver protein that provides nutrition to the embryo.) Such
19 individuals usually are sterile.

20 The third category consists of effects that leave exposed individuals capable of
21 breeding but blocks them from engaging in normal courtship and copulatory behavior
22 with opposite sex individuals. Examples are individuals that do not respond to species-
23 typical courtship signals, are unable to produce the appropriate signals, or cannot

1 properly integrate the necessary sensory and motor information necessary for successful
2 copulation. This would include birds that form pair-bonds, but that do so with members
3 of the same-sex. Taken together these individuals could be said to be exhibiting effects
4 of EDC exposure, and, though they may live as long as normal, unexposed individuals,
5 they collectively are an agent of evolutionary species death, because they do not
6 reproduce.

7 Q: WHAT ARE SOME OF THE FACTORS THAT DETERMINE THE KIND OF
8 EFFECTS OF ENDOCRINE DISRUPTION?

9 A: Several key topics have emerged in an effort to reconcile the disparate effects of EDCs
10 and to understand their mechanisms. In general, consequences of EDC exposure are
11 dependent upon both the properties of the chemical(s) to which an organism is exposed,
12 together with the individual's genetic susceptibility and developmental or life stage. It is
13 necessary to consider sex, mixtures, timing of exposure, assaying a variety of potential
14 mechanisms, testing low-dose, ecologically-relevant levels of exposure, and considering
15 effects beyond the F1 (i.e., exposed) generation, in the evaluation of exposure effects.
16 For instance, developing organisms will be more greatly affected than adult organisms,
17 one sex is at times more susceptible than another, and effects can also depend on the
18 mixture of chemicals to which the organism is exposed.

19 Q: ARE THERE CUMULATIVE EFFECTS OF ENDOCRINE DISRUPTION?

20 A: There is no question that cumulative experiences throughout an individual's life history
21 interact with genetic predispositions to shape the individual's physiology and behavior.
22 Accordingly, repeated exposure to EDCs will have cumulative effects. Recent evidence
23 suggests that events in past generations may also influence how an individual responds to

1 events in his or her own life history, and there is unequivocal evidence that EDCs can
2 influence not only the exposed individual, but also subsequent generations. Epigenetics
3 is the study of how the environment can affect the genome of the individual during its
4 development, as well as affect the development of its descendants, all without changing
5 the DNA sequence. It is important to distinguish the difference between Context-
6 Dependent vs. Germline-Dependent Epigenetic Modifications. In Context-Dependent
7 epigenetic modifications, we are dealing with transmission within a generation, while in
8 Germline-Dependent epigenetic modifications, we are dealing with transmission across
9 generations. (See, Exh. HC-7, paper number 324).

10 The best examples of Context-Dependent epigenetic modifications are those that
11 either have an effect early in life, such as exposure to endocrine disrupting compounds *in*
12 *utero*. In the first instance, the onset of disease manifests itself later or the deleterious
13 effects decline with time. The extent to which the modification is perpetuated in future
14 generations is determined by the persistence of the environmental factors that bring about
15 the epigenetic modification. That is, in each generation, individuals are exposed to the
16 same environmental conditions. For example, if the diet or environmental toxicant
17 continues to be present in the environment, then the epigenetic modification will be
18 manifest in each generation.

19 Germline-Dependent epigenetic modifications are fundamentally different from
20 Context-Dependent epigenetic modification, in that the epigenetic imprint (i.e., the
21 modification) has become independent of the original causative agent. Here, the
22 epigenetic modification is transferred to subsequent generations, because the change in

1 the epigenome has been incorporated into the germline. Thus, the effect is manifest in
2 each generation, without the need for re-exposure.

3 Q: DR. CREWS, I THINK MOST LAY PEOPLE WOULD INTERPRET GERMLINE-
4 DEPENDENT EPIGENETIC MODIFICATIONS TO BE THOSE THAT ARE
5 CAUSED BY ACTUAL CHANGES TO THE GENES OF THE INDIVIDUAL'S
6 SPERM OR EGGS. IS THAT THE CASE, OR IS THAT AN INACCURATE WAY TO
7 THINK OF THESE MODIFICATIONS?

8 A: That is inaccurate, I am afraid. The term "epigenetics" is derived from epi (meaning
9 above) and genetics. It refers to a process whereby experiences can modify the genome
10 without altering DNA structure itself. If this is incorporated into the germline, the
11 modification can be transmitted across generations. Thus, epigenetics refers to the
12 interactions between the environment and the genome in ways that gene expression is
13 modified independent of genetic mutation.

14 Q: DOES IT TAKE LARGE AMOUNTS OF ENDOCRINE DISRUPTING CHEMICALS
15 TO CREATE THESE EFFECTS?

16 A: No. Especially at an early age, very low-dosages can cause critical effects. The
17 fetal/embryonic basis of adult disease hypothesis postulates that early exposures to sub-
18 toxic levels of EDCs may not have any immediate apparent effects, but can predispose an
19 organism to the latent development of a disease or disorder. Studies with a variety of
20 animal models consistently demonstrate that low-dose exposures of fetuses/embryos to
21 EDCs often have no discernible effects at birth, but result in infertility, abnormalities, and
22 cancers much later in life. Also, some species, many fish and amphibians, for example,
23 are more susceptible to endocrine disrupters than are others.

24 Q: HAVE THERE BEEN STANDARDS OR STUDIES TO CREATE STANDARDS TO
25 DETERMINE A THRESHOLD EXPOSURE LEVEL BELOW WHICH EXPOSURE
26 TO EDCs WILL NOT CAUSE, IN EXPOSED POPULATIONS, BEHAVIORAL OR
27 PHYSICAL ABNORMALITIES IN INDIVIDUALS OR MALFUNCTIONS IN

1 REPRODUCTION IN INDIVIDUALS OF THE POPULATION OR IN THEIR
2 OFFSPRING?

3 A: There are no set standards, but there have been attempts at creating such standards.
4 Traditionally toxicological studies have focused on the dosage/concentration of
5 substances administered that result in mortality and/or disease. Indeed, risk assessments
6 for virtually all chemicals, except genotoxic chemicals (i.e., chemicals that damage
7 DNA), assume that, for any substance, there exists a threshold dose below which
8 exposure is safe. This assumption underlies the concept of a “no observed adverse effect
9 level” (NOAEL), a commonly used approach in risk assessment. As in other common
10 risk assessment approaches, the NOAEL relies on standard toxicology dose-response
11 studies to designate the “lethal dosage low” (LD_{Lo}) as the dosage at which mortality or
12 disease is still detectable (as compared to the LD₅₀, or the dosage at which 50% of a
13 group of test animals die or manifest disease when administered as a single exposure at a
14 specific time period). It is the dose tested below that of the LD_{Lo}, or the dose at which
15 the experimental group shows no significant difference between it and the control, or no-
16 dose, group, that becomes the basis for setting the acceptable exposure risk. This value is,
17 then, typically divided by 100, or a factor of 10 for variability in test responses and
18 another factor of 10 for species extrapolation (i.e., to account for the possibility of
19 interspecies variations in sensitivity).

20 Q: IN YOUR OPINION, IS THERE A THRESHOLD EDC DOSE BELOW WHICH IT IS
21 SAFE TO ASSUME THE ALTERATIONS ABOUT WHICH I JUST ASKED WILL
22 NOT OCCUR IN AQUATIC SPECIES?

23 A: No. Remember the paper Sheehan and I published on the absence of NOAELs for EDCs;
24 I cited it, earlier. Acceptable exposure levels for EDCs that are considered safe are rarely

1 tested empirically because of the number of dosages needed to test, as well as the number
2 of individuals required to detect significant and valid deviations, is prohibitively large.
3 As a consequence, safety standards for most chemicals depend on the validity of the
4 threshold assumption, meaning the standards assume there is some safe threshold level.
5 Yet, this is not necessarily true. It has been demonstrated that, for endocrine-mediated
6 processes, such as sexual development, the individual during any stage of its normal
7 development is already producing the levels of the hormone necessary for that process.
8 In other words, the individual is already at its own threshold, and any additional added
9 ligand (i.e., hormone mimic) from the internal or the external environment is necessarily
10 above threshold and adverse.

11 Early development is a particularly fragile life stage, because it is when the cells
12 that will make up the body are differentiating and there are few of them, that is, relative
13 to the adult body. During this period the fetus/larvae are particularly vulnerable because
14 protective systems are not yet developed, and the “stem cells”, or those cells that will
15 give rise to organs, etc., are tightly regulated. Any deviation from the normal hormonal or
16 genetic constitution will influence their path, and can result in absence of a structure,
17 malformation, or dysfunction. Hormones are a major class of chemicals that help cells
18 communicate and coordinate during this developmental process. Our work with the red-
19 eared slider turtle shows that for a sensitive process like the formation of the appropriate
20 and normal gonad, any amount of exogenous estrogen will disrupt this process.

21 Q: COULD YOU EXPLAIN TO US WHAT HAPPENS TO AN INDIVIDUAL WHEN IT
22 IS EXPOSED TO HORMONES, OR LIGANDS THAT MIMIC HORMONES, WHEN
23 IT IS ALREADY AT ITS THRESHOLD?

1 A: In the case of the red-eared slider turtle, the temperature of the incubating egg determines
2 the gonadal sex that will develop. This is different from the case where sex
3 chromosomes inherited from the parents establish the gonadal sex, as occurs in mammals.
4 Unlike such situations when at fertilization the union of sperm and egg fix the type of
5 gonad that will form, in many turtles there is a narrow window of time during
6 embryogenesis in which temperature acts. This environmental trigger sets into motion the
7 complex cascade of events that, under normal circumstances, ensures that the gonads
8 (testes or ovaries) will form along with the appropriate sex ducts (vas deferens or
9 oviduct/uterus). If, in addition to this temperature trigger, the embryo is exposed to an
10 agent that mimics hormones, an intersex gonad (ovo-testes) and inappropriate set of sex
11 ducts will form. This situation means that the individual, when it reaches adulthood, will
12 not be able to breed and instead be sterile.

13 Q: IF ONE POSTULATED THAT THERE ARE EDCs IN THE DISCHARGES OF
14 WASTE WATER FROM MUNICIPAL WASTE WATER TREATMENT PLANTS,
15 ARE SOME RECEIVING AQUATIC ENVIRONMENTS OF MORE CONCERN
16 THAN ARE OTHERS?

17 A: Yes. Consider effluent-dominated ecosystems. Effluent-dominated or dependent
18 ecosystems are streams or rivers that receive effluent discharges such that the instream
19 flows of these systems are predominately return flows from waste water reclamation
20 plants. Thus, upstream dilution of the discharge effluent is limited, particularly in dry
21 seasons (e.g., summer months in Texas); periodically, the entire flows of some streams
22 and rivers are effluent. I understand Bear Creek in Hays County, Texas, is predicted to
23 become an effluent-dominated (and at many times during the year, an effluent-dependent)
24 wadeable stream, if the proposed discharge of 500,000 gpd (0.77 cfs) enters the stream.

1 Q: WHY ARE EFFLUENT DOMINATED STREAMS OF CONCERN?

2 A: Due to hydrological dynamics, effluent-dominated or dependent streams and rivers are
3 considered “worst case” scenarios for examining the potential impacts to aquatic life of
4 endocrine disrupting and modulating compounds found in pharmaceuticals, personal care
5 products and other industrial chemicals, because the degradation rate of these substances
6 may be exceeded by their rate of introduction to the receiving system, leading, at least for
7 periods of time, to ever-increasing doses of EDCs to the aquatic species in the streams or
8 rivers.

9 Q: I WILL REPRESENT TO YOU THAT, IN SOME CIRCUMSTANCES, TEXAS LAW
10 PROHIBITS DEGRADATION OF WATER QUALITY IN CREEKS, SUCH AS BEAR
11 CREEK SOUTH OF ASPEN DRIVE, BY MORE THAN A *DE MINIMIS* EXTENT. IS
12 THERE A GENERALLY UNDERSTOOD CONNOTATION AMONG
13 TOXICOLOGISTS AS TO WHAT A *DE MINIMIS* EXTENT OF DEGRADATION
14 MIGHT BE?

15 A: I think most toxicologists would agree that increases in levels of EDCs in a water body
16 that caused in any species found there any of the three categories of effects I described,
17 earlier, (i.e., gross external effects, internal and not-so-obvious effects, and abnormal
18 courtship or copulatory effects) would degrade the water to more than a *de minimis*
19 extent. Certainly, increases that would lead, in time, to a population crash of a species in
20 the water body would degrade the water to more than a *de minimis* extent.

21 Q: HAVE YOU READ DR. MCHUGH’S DEPOSITION TESTIMONY, IN WHICH HE
22 STATES THAT THERE ARE NO DOCUMENTED ADVERSE HEALTH OR
23 ENVIRONMENTAL IMPACTS THAT HAVE BEEN SHOWN TO BE CAUSED BY
24 EFFLUENT-BORNE EDCs OR OTHER EMERGING CONTAMINANTS IN
25 SURFACE OR GROUNDWATER?

26 A: Yes. He is incorrect in this assessment.

27 Q: PLEASE EXPLAIN FOR THE JUDGE WHY YOU BELIEVE THAT ASSESSMENT
28 IS INCORRECT.

1 A: Several papers based on work at my own laboratory, as well as many other papers in the
2 peer-reviewed literature, describe experiments that show conclusively that EDCs
3 produced adverse effects on individuals and populations in the systems studied. I should
4 point out also that these studies used ecologically-relevant dosages; that is, the
5 experimenter replicated the dosages of compounds found in nature, including
6 streams/rivers supplemented by wastewater effluent.

7 Q: THE TCEQ, IN ITS RESPONSE TO COMMENTS ON THE DRAFT PERMIT,
8 STATES THAT THE EPA IS INVESTIGATING PHARMACEUTICAL AND
9 PERSONAL CARE PRODUCTS (“PPCPs”) AND HAS STATED THAT SCIENTISTS
10 HAVE NOT FOUND EVIDENCE OF ADVERSE HUMAN HEALTH EFFECTS
11 FROM PPCPs IN THE ENVIRONMENT. WHAT IS YOUR TAKE ON THAT
12 STATEMENT?

13 A: Well, as always, I would like to see the actual EPA statement. I find it hard to believe
14 EPA would say, at least, any time in the last 10 years, that scientists have not found such
15 evidence linking environmental PPCPs and human health effects. I have attached a rather
16 recent paper from researchers at Tufts University School of Medicine that demonstrates
17 convincingly there are adverse human health effects associated with exposure to very low
18 (i.e., environmentally-relevant) doses of EDCs. See, Exh. HC-8.

19 Even if EPA’s alleged statement were true, however, I do not see that it has any
20 bearing on a linkage between environmental PPCPs, especially EDCs, and adverse
21 effects on aquatic species. There is just too much scientific research establishing the
22 opposite to allow anyone to analogize an absence of impacts on aquatic species from an
23 absence of impacts (were there, in fact , an absence of impacts) on humans.

24 Q: DO YOU AGREE WITH THE CONCLUSIONS REACHED BY DR. MCHUGH AND
25 THE TCEQ STAFF REGARDING PPCPS?

1 A: No. As I have already discussed at length, PPCPs include EDCs that negatively impact
2 aquatic species. Unlike what Dr. McHugh and the TCEQ staff indicate, an array of
3 recent research has shown that EDCs will also negatively impact human health.

4 Q: DR. MCHUGH ALSO STATES THAT THERE IS NO EVIDENCE LINKING PPCPs
5 TO ANY ADVERSE ENVIRONMENTAL IMPACT AT CONCENTRATIONS
6 ASSOCIATED WITH TREATED WASTEWATER DISCHARGE. DO YOU AGREE
7 WITH THIS STATEMENT?

8 A: No. The scientific community has repeatedly demonstrated that levels of PPCPs in fact
9 found in waste-water-impacted streams have adverse environmental effects. I am aware
10 of Dr. McHugh's belief the longer retention times and greater density of solids planned
11 for the WCID#1 plant, as compared to those characteristics at less sophisticated plants,
12 will reduce PPCP concentrations in the plant's discharge to lower-than-typical levels.
13 That seems logical and finds some support in the published literature. However, Dr.
14 McHugh does not contend all PPCPs will be removed from the plant's discharge and,
15 given the weight of scientific understanding that there is no safe threshold for exogenous
16 EDC exposure, at least, it simply cannot be said there will be no adverse impacts to the
17 aquatic environment from the plant's discharge.

18 Q: DR. MCHUGH, PER HIS DEPOSITION TESTIMONY, BELIEVES THAT THE
19 WASTEWATER TREATMENT PLANT WILL BE MORE EFFECTIVE AT
20 REMOVING PPCPs THAN A TRADITIONAL WASTEWATER TREATMENT
21 PLANT. EVEN IF THIS WERE TRUE, DO YOU BELIEVE THAT EDCs RELEASED
22 IN THE EFFLUENT WILL HAVE ONLY *DE MINIMUS* EFFECTS ON CREEK
23 WATER QUALITY?

24 A. It is true that there are no numeric criteria associated with PPCP concentrations.
25 However, as I have just said, it is my opinion that, based on the best available
26 information, the proposed discharge will likely discharge compounds containing EDCs
27 that will negatively impact aquatic organisms in ways I have already discussed. The

1 literature on EDCs is heavily weighted to the proposition that there is no safe
2 concentration threshold below which there will not be adverse effects on species exposed
3 to exogenous EDCs.

4 Q: DOES THIS CONCLUDE YOUR TESTIMONY?

5 A: Almost. Before concluding, I would offer the judge a good survey article on this
6 complicated field. The article acknowledges and elaborates briefly on the uncertainties
7 still adhere to many facets of the interaction of EDCs and the natural aquatic
8 environment. It also provides numerous examples of adverse impacts that have been
9 documented in one circumstance or another. I believe it might help the judge evaluate
10 the conflicting testimony he will likely hear. Please see, Exh. HC-9. It concludes my
11 testimony.